Hemorheologic Changes in Elderly post Aerobic Training Program

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ABSTRACT

Background: *Regular physical activity is associated with reduced risk of coronary heart disease, stroke, and cardiovascular mortality in middle age and older age, although the mechanisms are unclear. Because physical activity has to be current and continuous to confer protection, the benefit may be at least partly due to a short-term effect, possibly through influences on blood coagulation, fibrinolysis and platelet aggregation, viscosity, and inflammatory markers. Prospective studies have linked several of these variables, including fibrinogen, viscosity to the risk of coronary heart disease. Therefore, in this study, the effect of 16 weeks of submaximal aerobic training on the hemorheologic factors, including hematocrit, whole blood viscosity, plasma viscosity, and fibrinogen concentration in elderly men was examined.* ***Methods and Results:*** *The effect of 16 weeks of submaximal aerobic training was studied on 15 old sedentary men (training) and 15 nontraining matched controls. After training, a more pronounced decrease in hematocrit, whole blood viscosity, plasma viscosity, and fibrinogen concentration was seen in the training group. The control group demonstrated no difference between the baseline and after 16 weeks measurements for all variables.* ***Conclusion:*** *sixteen weeks of submaximal aerobic training program resulted in a significant decrease in the hemorheologic factors in elderly men.*

Key words: *Submaximal Aerobic Training Program, Elderly Men, Hematocrit, Whole Blood Viscosity, Plasma Viscosity, Fibrinogen Concentration*.

INTRODUCTION

Haemostasis is achieved through a delicate equilibrium between the coagulation and fibrinolytic cascades. Aging is associated with unfavorable changes in coagulation and fibrinolytic components that may play a role in the enhanced risk for thrombotic events[1,33]. Epidemiological studies have shown that certain haemostatic and rheological factors (e.g. fibrinogen, viscosity, haematocrit) are associated with incident cardiovascular events. Possible causal mechanisms include effects on thrombogenesis and ischemia. Elevated blood viscosity may promote atherosclerotic development by increasing platelet adhesion to the subendothelium, by increasing protein infiltration into the arterial wall, and by altering local shear forces at sites of atherogenesis[19,35].

The total peripheral resistance to blood flow is regulated by the caliber of the resistant vessels and the viscous characteristics of the blood. These viscous properties govern the rheological behavior of the blood, which is regulated by a complex interaction of several factors. Amongst these factors are: whole blood viscosity, plasma viscosity, haematocrit, red blood cell deformability, red blood cell aggregation, and fibrinogen concentration[9,12].

Increased blood and plasma viscosity have been associated with an increased risk of coronary heart disease. Expansion of blood

volume has been documented as a consequence of endurance exercise training[4]. Exercise training improves blood rheology in patients with coronary heart disease [9,19]. This improvement may contribute to the increased functional capacity and reduced morbidity and mortality that are associated with participation in exercise programs[3,28].

Plasma viscosity is one of the main determinants of whole blood viscosity. The viscosity of plasma increases with increasing protein concentration. A logarithmic linear relationship between blood viscosity and haematocrit has been reported. Fibrinogen plays a pivotal role in the blood haemostatic mechanism and has been described as one of the main determinants of blood rheology via its effect on the aggregation of red blood cells. Lower plasma fibrinogen concentrations may reduce the risk of atherosclerosis and thrombosis by favorably altering blood viscosity and platelet adhesion and aggregation and by limiting intravascular fibrin formation and deposition 7. Therefore, it has the greatest influence on plasma viscosity[12.]

Disturbance of blood rheology is recognized not only as a coronary risk factor, but also as a risk predictor of cardiovascular disease[22]. Short-term maximal exercise causes an increase in whole blood viscosity, mainly due to an increase in haematocrit and plasma viscosity[2]. The increases in plasma viscosity and haematocrit have been ascribed to exercise-induced haemoconcentration as a result of fluid transfer from the blood to the interstitial spaces[9,28].

It was reported that the fitter patients not only had lower whole blood viscosity, but also had lower plasma viscosity, lower fibrinogen concentrations, and lower red blood cell aggregation than unfit patients. A significant decrease in whole blood viscosity, haematocrit, fibrinogen concentration, and mean corpuscular haemoglobin concentration occurred after 3 weeks of training. This coincided with a significant increase in red blood cell deformability and red blood cell flexibility, which could be added to the list of favorable effects of physical conditioning[25]. These findings may imply that training shortens the life span of red blood cells because of a higher rate of haemolysis and replacement of older cells with younger ones. These changes are favorable because younger and larger red blood cells transport oxygen more efficiently and are more flexible with a greater ability to change shape[6,12,28].

Low red blood cell (RBC) counts and haemoglobin concentration are more likely is due to a training-induced increase in plasma volume which causes haemodilution[1,12,21].

Pathological and clinical studies have indicated that platelets play an important role in the pathogenesis and progression of cardiovascular diseases[36]. Moderate-intensity aerobic training improves fibrinolytic activity and reduces platelet aggregation and blood viscosity[5]. It has been suggested that the clotting activity was sustained while the fibrinolytic activity declined after exercise[14]. Chronic aerobic exercise training may decrease coagulation potential and increase fibrinolytic potential in both healthy individuals and cardiovascular diseased patients[34]. Therefore, chronic aerobic exercise training may cause favourable adaptations that could contribute to decreased risk of ischemic event, both at rest and during physical exertion[39].

Plasma fibrinogen is a major determinant of platelet aggregation and blood viscosity[23,32]. Physical activity might have a protective effect against both arterial and venous thrombosis by reducing platelet count, cofactors in platelet adhesion/aggregation(haematocrit, fibrinogen),

coagulation factors (VIII: antihemophilic A factor, IX: antihemophilic B factor), and fibrin turnover[38]. There are also a reduction in activated partial thromboplastin time, a reduction in fibrinogen, and an increase in fibrin D – dimers[30,31].

In principle, there should not be any increase of blood viscosity factors (plasma viscosity, blood viscosity, and fibrinogen) with aging in a population with good quality of life. The pathological causes of the increase in the blood viscosity factors often observed in the elderly could be ascribed to the following: use of drugs (e.g. cigarette smoking), lack of exercise, unbalanced diet, psychological states, presence of diseases such as heart disease[8,18].

The purpose of the present study was to evaluate the effect of 16 weeks of submaximal aerobic training program on hemorheologic factors in elderly men.

SUBJECTS AND PROCEDURES

Subjects

Thirty sedentary, elderly men were enrolled in the study after they had given their informed consent and understood the experimental procedures. These subjects were randomly divided into control (n = 15) and training (n = 15) groups. Their anthropometric data were (mean + standard deviation): age, 66.7 + 3.7 years; height, 170.4 + 8.4 cm; and weight, 65.4 + 7.4 kg for the control and training groups. None of these subjects was engaged in any regular physical activity before the study. All of the subjects were nonsmokers. All subjects were assessed by a medical specialist and they were free of overt disease as assessed by medical history and physical examination. The subjects were further evaluated for clinical evidence of coronary artery disease with electrocardiograms and blood pressure at rest and during exertion. All subjects did not take any medication and continued to abstain from any medication throughout the training program. This study was carried out in the Geriatrics Medicine Unit, Faculty of Medicine, Ain Shams University.

Procedures

Familiarization session

Before the actual study, subjects in the training group were familiarized with exercise on an electronically bicycle ergometer in the upright position (EN-Cycle, Delft Instruments, The Netherlands) to eliminate the novel effects of a new experience.

BLOOD COLLECTION

All blood collection and analysis procedures were done by a clinical laboratory specialist. To avoid the diurnal variation in hemorheologic factors, all blood samples were collected between 7 a.m. and 10 a.m. after a 12-hour overnight fasting. After the subjects had arrived at the clinic and rested for 30 minutes, 15 ml of blood samples were drawn from the antecubital vein for baseline data on hemorheologic factors (whole blood viscosity, plasma viscosity, blood haematocrit, and fibrinogen concentration in plasma). The first 2 ml was discarded; then the remaining blood sample was used for the measurement of hemorheologic factors. Blood was anticoagulated with K_2-EDTA (1.5 mg/mL) for measurement of haematocrit and whole blood and plasma viscosity at 37^0C in a semiautomated capillary viscometer (Act-Diff - Coulter Electronics, Krefeld - Germany). Blood was also anticoagulated with 0.5 mL of trisodium citrate for measurement of fibrinogen. For quantitative determination of fibrinogen, the Microsample Coagulation Analyzer, Model MCA 210, of Bio/Data

(Horsham, PA, USA) was used. This instrument electronically transforms results to calculate the first derivative of the rate of turbidity increase change that is proportional to fibrinogen concentration in plasma based on the kinetic fibrinogen assay method. This procedure was performed for all subjects at baseline and repeated again after 16 weeks.

Exercise test

After blood collection, a progressive exercise test was performed for subjects in the training group to determine maximal oxygen consumption ($VO_{2\ max}$).

The exercise test was performed under supervision of a cardiologist. Subjects in the training group performed an incremental graded exercise on a cycle ergometer in a sitting position (step test, start 25 Watt, every 3 minutes an increase of 25 Watt until volitional exhaustion) to measure $VO_{2\ max}$. The $VO_{2\ max}$ was measured at 30-seconds intervals using an open spirometric system (Oxycon beta – Mijnhardt, The Netherlands), which was calibrated before and after each test.

Exercise training

After completion of baseline measurements, subjects in the training group participated for 16 weeks in supervised aerobic training sessions on an electronically bicycle ergometer (EN-Cycle, Delft Instruments, The Netherlands). Subjects exercised three times a week for 30 minutes at a constant submaximal level (70% of $VO_{2\ max}$). The subjects were instructed to "warm-up" at 15 Watt for 8 minutes, then to increase the resistance setting until 70 % $VO_{2\ max}$ for 14 minutes, and then the subjects would "cool down" for 8 minutes at 15 Watt. The controls were not trained during the experimental period.

Statistical Analysis:

Statistical analysis was performed with Superior Performing Software Systems (SPSS-PC, version 10.0 statistical software). Results at the beginning and the end of the study period were compared. Intragroup differences were compared by paired Student's "t" test and intergroup differences by unpaired Student's "t" test. A value of $P < 0.05$ was considered statistically significant. Data are presented as mean + S.D. (Standard deviation).

RESULTS

All subjects completed the study period. No medical complications or training-related adverse events were observed during the test and the training period.

Submaximal aerobic training program induced a decreased in hemorheologic factors in the training group. While, the control group did not demonstrate any change in these variables.

Hematocrit

There was no significant difference in baseline plasma hematocrit between both training and control groups, while, there is significant difference in plasma hematocrit between both training and control groups after sixteen weeks ($p<0.05$).The plasma hematocrit decreased significantly in the training group after exercise training ($p<0.05$), compared with the pre-exercise level. [Table (1), Figure (1)]

Table (1): Mean levels of hemorheologic factors before and after sixteen weeks in both trained and control groups

Variable	Training Group (n = 15)		Control Group (n = 15)	
	Pre	Post	Pre	Post
Hematocrit (%)	49.2 ± 3.9	41.7 ± 1.9	49.8 ± 3.2	49.8 ± 2.5
Blood Viscosity (mPa .s)	5.45 ± 0.56	3.25 ± 0.11	4.52 ± 0.37	4.51 ± 0.09
Plasma Viscosity (mPa .s)	1.55 ± 0.03	1.24 ± 0.01	1.54 ± 0.05	1.54 ± 0.08
Fibrinogen (mg/100 mL)	366.0 ± 61.1	334.6 ± 70.1	372.4 ± 63.6	376.4 ± 64.9

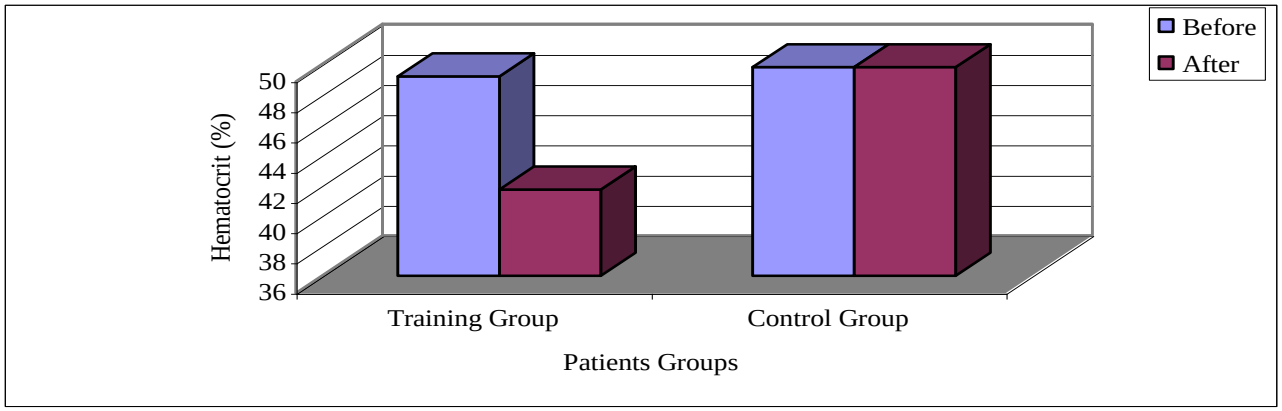

Fig. (1): Mean levels of hematocrit (%) before and after 16 weeks in both training and control groups.

Whole Blood Viscosity:

There was no significant difference in baseline whole blood viscosity between both training and control groups, while, there is significant difference in whole blood viscosity between both training and control groups after sixteen weeks ($P < 0.05$). Whole blood viscosity decreased significantly ($P < 0.05$) after exercise training in the training group, compared with the pre-exercise level. [Table (1), Figure (2)]

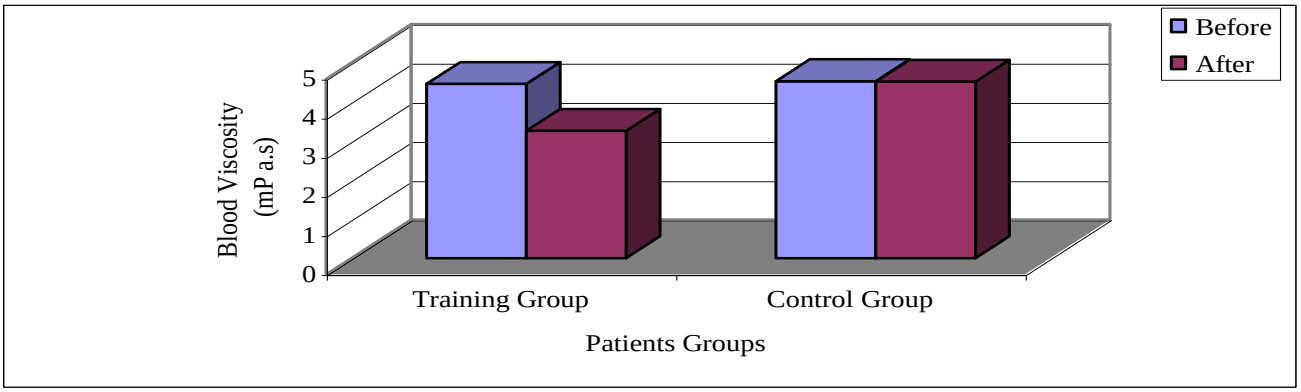

Fig. (2): Mean levels of whole blood viscosity (mPa.s) before and after 16 weeks in both training and control groups.

Plasma Viscosity

There was no significant difference in baseline plasma viscosity between both training and control groups, while, there is significant difference in plasma viscosity between both training and control groups after sixteen weeks ($P < 0.05$). Plasma viscosity decreased significantly ($P < 0.05$) after exercise training in the training group, compared with the pre-exercise level. [Table (1), Figure (3)].

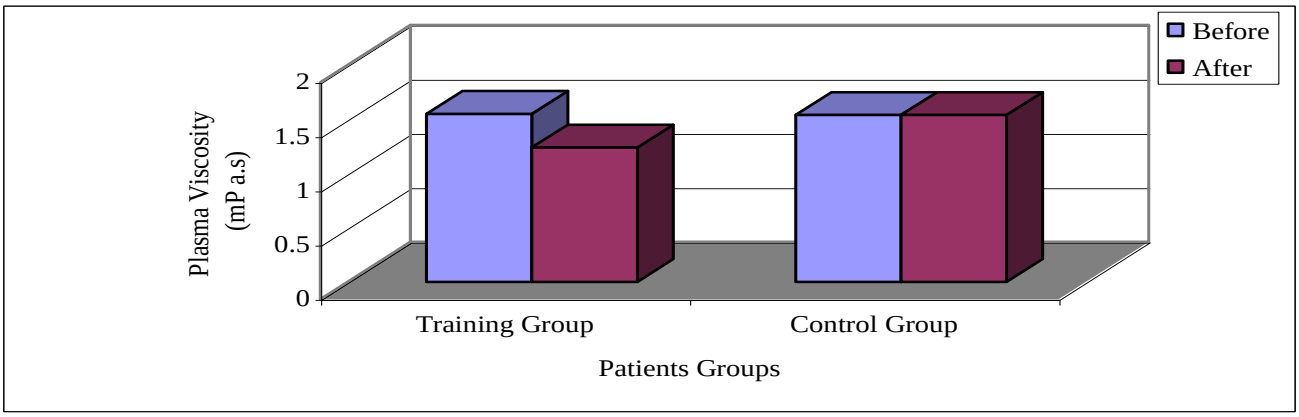

Fig. (3): Mean levels of plasma viscosity (mPa.s) before and after 16 weeks in both training and control groups.

Fibrinogen

There was no significant difference in baseline fibrinogen concentration between both training and control groups, while, there is significant difference in fibrinogen between both training and control groups after sixteen weeks ($P < 0.05$). Fibrinogen concentration decreased significantly ($P < 0.05$) after exercise training in the training group, compared with the pre-exercise level. [Table (1), Figure (4)].

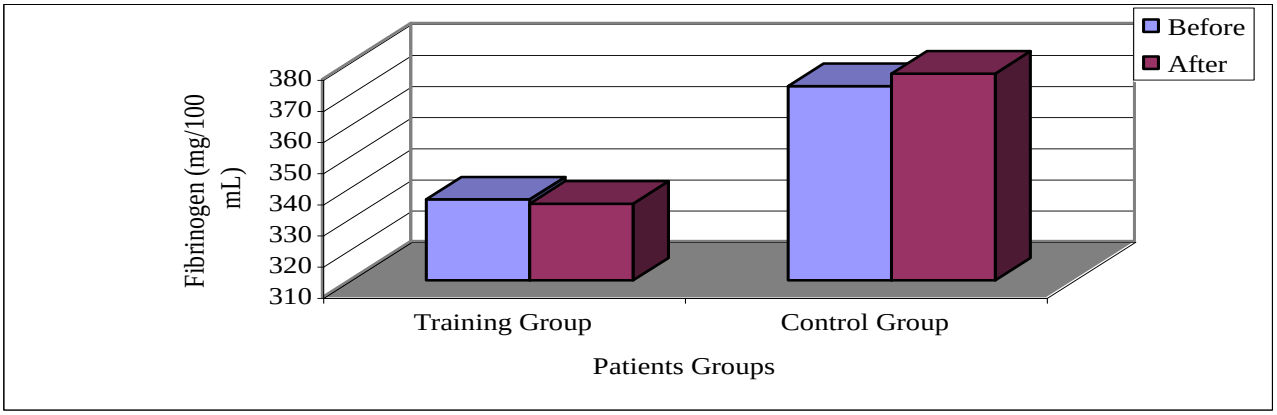

Fig. (4): Mean levels of fibrinogen (mg/100 mL) before and after 16 weeks in both training and control groups.

DISCUSSION

The results of this study showed that an aerobic training program for 16 weeks demonstrated a significant reduction in hemorheologic factors including hematocrit, whole blood viscosity, plasma viscosity, and fibrinogen concentration.

It is well documented that submaximal aerobic training program increases blood volume due to an expansion of plasma volume and an increase in red blood cell mass[20]. The relative percentage increase in plasma volume is greater than that for red blood cell mass, and this explains why haematocrit values in athletes are lower than those in non-athletes[21].

Although the balance of the evidence available suggests that endurance training lowers whole blood viscosity, the exact physiological mechanisms responsible for this phenomenon remain, to a large extent, speculative. It is possible that these rheological adaptations to training are due, at least in part, to the blood volume of trained individuals being at least 20 % higher than that of sedentary individuals [3,12,21].

It is known that low haematocrit is associated with low whole blood viscosity and this is usually accompanied by an increase in cardiac output. In addition, animal studies indicated that whole blood volume is inversely related to cardiac output and the pressure drop across the capillaries is directly related to plasma viscosity. Therefore, it is reasonable to suggest that trained athletes with low whole blood viscosity and plasma viscosity would possess a higher blood flow rate and smaller pressure drop in the capillaries[12].

While endurance training can increase RBC mass, the rise in plasma volume is greater. This would lower blood viscosity. Endurance training may increase the rate of RBC production concomitantly with accelerated turnover, resulting in the presence of a steady-state population of younger RBCs. Athletic performance may be enhanced because younger RBCs are more deformable and deliver oxygen to working muscle more efficiently than older cells[1,7,12,21].

It is suggested that an improvement in blood fluidity can be induced by regular physical exercise regardless of whether the blood rheology was normal or abnormal at baseline. The relevance of these findings could be three-fold. Firstly, the "better than normal" blood rheology in athletes may contribute to enhanced blood flow in the working musculature and thus increase work output. Secondly, the data supports the thesis of a link between blood rheology and atherogenesis. Thirdly, regular exercise might be a way of therapeutically increasing blood flow in ischemic vascular diseases[4].

Reticulocyte counts have been shown to be elevated by 100 % 2 days after a marathon; and the plasma haptoglobin concentration decreased significantly. It has been also reported that the rate of RBC destruction increased 2-fold during a 3-week period of moderate endurance training by previously untrained males. Increased reticulocytosis and RBC creatine content are indicative of the presence of an increased proportion of younger RBCs which have an increased rate of deformability leading to a decreased blood viscosity[1,7,36].

Atrial natriuretic peptide, which regulates fluid homeostasis, increases the filterability of RBCs by elevating their membrane mobility. Because the plasma concentration of atrial natriuretic peptide increases significantly during moderate exercise, this hormone may contribute to the regulation of oxygen delivery to working muscle by maintaining or increasing RBC deformability[7].

Because exercise training stabilizes sympathetic nervous activity, it is thought to decrease red blood cell release from the spleen and other organs. This effect of exercise might also be the explanation for the decrease or lack of any increase in the plasma haematocrit during exercise[21].

It has been suggested that the improvement in fibrinolytic activity and reduction in platelet aggregation and blood viscosity following moderate-intensity aerobic training are a result of action on haemostatic balance[14]. Low-density lipoprotein lipid peroxides play a role in modulating and attenuating platelet aggregation during strenuous exercise[17].

Exercise-induced enhancement of fibrinolysis has been repeatedly demonstrated using a wide range of exercise protocols. The increase in fibrinolysis is due to a rise in tissue-type plasminogen activator (which is released by the vascular endothelium) and decrease in plasminogen activator inhibitor[10,29].

The end products of fibrinolysis, fibrin degradation products, have been shown to increase after endurance exercise of different types, D-dimers, which are products of the breakdown of activated factor XIII (fibrin stabilizing factor) and fibrin, are often used as a marker of fibrin degradation, and have also been shown to increase[13,16].

Muscle tissue is directly responsible for altering both local and systemic blood flow and that exercise of muscle could, in and of itself, prove modulatory to some of the major morbidity factors. The mechanical rigors of repeated exercise stimulate capillary beds within the muscle, resulting in the repeated activation of fibrinolytic cascades to maintain the flow of blood to the active muscle[15].

High levels of low-density lipoprotein (LDL) increased platelet aggregability, secretion, and thromboxane A_2 release from activated platelets. It had been reported that LDL level was decreased by exercise training [11]. Exercise training could increase high-density lipoprotein and decrease lipoprotein lipase activity and lipogenesis. Therefore, the changes of platelet function induced by exercise training might be partially explained by the alteration of lipoproteins after training[5,29].

Acute tissue-type plasminogen activator (t-PA) release from the vascular endothelium declines with age in sedentary adult men. In addition, regular aerobic exercise fully restores the capacity of the endothelium to release t-PA in previously sedentary older men. Regular aerobic exercise may not only prevent, but could also reverse the deleterious effects of sedentary aging on endothelial fibrinolytic function[24,26,29].

Whereas acute strenuous exercise enhances platelet adhesiveness and aggregability, exercise training suppresses these platelet-function parameters. Nitric oxide has potent antiplatelet effects. It stimulates platelet guanylate cyclase, elevates platelet guanosine 3', 5' – cyclic monophosphate (cGMP) levels, reduces agonist-induced rise of platelet intracellular calcium concentration $[Ca^{2+}]_i$, and suppresses agonist-induced platelet aggregation. By similar mechanisms, nitric oxide also suppresses platelet adhesion. Exercise training apparently can increase endogenous nitric oxide release and elevate (cGMP) contents in platelets, which in turn suppress platelet reactivity[37].

There are several possible explanations for the training-induced nitric oxide release. First, exercise training increases norepinephrine– induced nitric oxide release. Therefore, this norepinephrine – stimulated nitric oxide release may in turn diminish the norepinephrine-evoked platelet activation.

Second, increased blood flow during repetitive exercise may increase nitric oxide release. Finally, low-density lipoprotein inactivates or inhibits endothelium-derived relaxing factor and chronic exercise decreases plasma low-density lipoprotein level. Therefore, a third possibility is that exercise training changes the lipid pattern in favor of endothelium-derived relaxing factor release, which inactivates platelets [36,37].

Conclusion

In this study supported by the relevant scientific work conducted in many laboratories, submaximal aerobic training program was associated with reductions in several hemorheologic factors; including hematocrit, whole blood viscosity, plasma viscosity, and fibrinogen. The benefit of submaximal aerobic training on cardiovascular diseases can be strongly supported.

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الملخص العربى

دراسة التغيرات في تيار الدم بعد برنامج للتمرينات الهوائية في المسنين

تم إجراء هذه الدراسة علي ثلاثين فرداً من المسنين. و قد تم تقسيمهم إلي مجموعتين متساويتين، مجموعة قامت بمزاولة برنامج للتمرينات الهوائية ذو الشدة دون القصوي علي الدراجة الثابتة و مجموعة ضابطة لم تقم بمزاولة أية برامج للتمرينات. و كان الهدف من هذه الدراسة هو معرفة تأثير برنامج للتمرينات الهوائية ذو شدة دون القصوي علي اللزوجة الكلية للدم، لزوجة البلازما، النسبة المئوية الحجمية للكريات الحمراء، و كذلك نسبة مكون الفيبرين في الدم عند هؤلاء الأفراد. و قد كان متوسط عمر هؤلاء الأفراد (جميعهم من الرجال) 66.7 ± 3.7 عاماً كما تم أخذ إقرار كتابي بالمعرفة و الموافقة علي الإشتراك في الدراسة من كل منهم.
و قد تم تطبيق برنامج للتمرينات الهوائية ذو الشدة دون القصوي (70 % من أقصي معدل لإستهلاك الأوكسجين) علي الدراجة الثابتة لمدة ثلاثين دقيقة في الجلسة الواحدة و بمعدل ثلاثة جلسات أسبوعياً لمدة ستة عشر أسبوعاً للمجموعة الأولي. و قد تم قياس اللزوجة الكلية للدم، لزوجة البلازما، النسبة المئوية الحجمية للكريات الحمراء، و كذلك نسبة مكون الفيبرين في الدم لكل الأفراد قبل بداية الدراسة و كذلك مرة أخري في نهاية الستة عشر أسبوعاً.
و قد أظهرت النتائج وجود تحسن ذو قيمة معنوية في كل من اللزوجة الكلية للدم، لزوجة البلازما، النسبة المئوية الحجمية للكريات الحمراء، و كذلك نسبة مكون الفيبرين في الدم في الأفراد الذين قاموا بمزاولة برنامج التمرينات الهوائية بينما لم توجد أي تغيرات في المجموعة الضابطة مما يعني أن مزاولة برنامج التمرينات الهوائية ذو الشدة دون القصوي في المسنين يؤدي إلي تحسن ملحوظ في اللزوجة الكلية للدم، لزوجة البلازما، النسبة المئوية الحجمية للكريات الحمراء، و كذلك نسبة مكون الفيبرين في الدم في المسنين.