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Influence of physical activity on the values of hemorheological parameters

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Abstract
Physical activity is a major factor contributing to maintaining overall health and properly functioning circulatory system. One of the underlying mechanisms is modifying blood flow by influencing hemorheological parameters, the most important of which are: hematocrit, plasma viscosity, whole blood viscosity, the ability of red blood cells to aggregate, deform and orient. We conducted a literature review to investigate how different types, intensities and phases of physical activity affect rheological properties of blood. The results show that short-term effects of exercise differ from the long-term consequences and rheology of blood varies depending on strength/endurance type of the training as good as on its regularity and intensity.

Findings from this research may be applicable in improving sport's performance and health maintenance in athletes.

Keywords: exercise, physical activity, blood viscosity, hemorheology

Introduction

Rheological properties of blood regulate the passage of blood through circulatory system, contributing to tissue oxygenation, nutrients supply for the cells and preventing blood stagnation, which according to Virchow's triad leads to higher risk of thrombosis and cardiovascular events (1, 2).

The blood flow in a living organism is influenced by factors related to the general condition of the organism. Alternations in the rheological properties of blood are related to the pathophysiology of numerous diseases: cardiovascular, hematological, metabolic, inflammatory and autoimmune (3). Elevated blood viscosity is positively correlated with many cardiac risk factors, including hyperlipidemia, atherosclerosis, hypertension and chronic kidney disease (2). Changes in the values of hemorheological parameters may also be related to the lifestyle, physical activity, type of diet or use of stimulants (4, 5, 6).

Exercise increases muscle oxygen demand making the effective blood flow and tissue perfusion essential. Changes in blood stream during physical activity, like increased heart rate, generate shear stresses which further influence erythrocyte membrane and change its properties. Erythrocyte deformation ability and tendency to aggregation modulate passage of red blood cells through very small capillaries and impact exercise performance by optimizing muscle perfusion and oxygenation (7). However, the impact of different types, intensities and phases of physical activity on hemorheology is still poorly researched.

In this study we decided to take advantage of data available in literature and analyze quantitatively the influence of physical activity on the values of chosen hemorheological parameters. We also consider the importance of blood viscosity regarding general body composition, oxidative stress, obesity and type 2 diabetes.

Rheological properties of blood

The blood flow in a living organism is determined both by the properties of the peripheral circulatory system and the physicochemical properties of whole blood (3). From the latter ones it is blood viscosity that mostly determines the blood flow at a given heart performance. We distinguish multiple factors influencing blood viscosity, including hematocrit, plasma viscosity, whole blood viscosity, the ability of red blood cells (red blood cells, RBC) to aggregate, deform and orient (3, 8). RBC deformability depends on the cell's volume, surface-to-volume ratio and its membrane properties, whereas erythrocyte aggregation is influenced by cellular features, plasma proteins e.g. fibrinogen and shearing forces (9). These two properties of RBC regulate their passage through vessels, enabling them to pass microcapillaries even smaller than their diameter. One of the factors influencing RBC flexibility is oxidative stress. State of inflammation and oxidative stress may lead to erythrocyte senescence and damage. Markers of RBC ageing include cell shrinkage, phosphatidylserine exposure and releasing microparticles connected in order to remove damaged proteins (10).

The plasma viscosity is a Newtonian fluid, which means that its viscosity is constant, not affected by shear rate. Fluidity of blood plasma is determined by the presence of high molecular weight proteins, mainly fibrinogen and globulins, as well as by the level of lipids (3, 11).

As physically blood is an extremely dense suspension of red cells in plasma, its rheological properties are highly non-newtonian. It means that in physiological range of shear rates the decrease in shear rate is associated with increase of blood viscosity and in contrast higher shear rate results in decrease of blood viscosity. The structure of the circulatory system is perfectly adjusted to these properties providing maximum perfusion at minimum flow resistance. This is accomplished by appropriate delicate balance in the number and length of vessels with given diameters because it is the vessel cross section that determines the shear rate, and hence blood viscosity, while the vessel length strongly influences the flow resistance. The deformability of erythrocytes becomes crucial for providing sufficient perfusion of all tissues. Proper blood flow conditions require a perfect balance between all these different aspects: heart performance, circulatory system geometry, blood non-newtonian viscosity and red cells mechanical properties. Disorders in some of them can be partially compensated by changes in other ones but only to certain extent. There exist self-regulatory mechanisms that try to restore sufficient perfusion in response to certain stimuli. It is interesting to investigate the interdependencies among different rheological and physico-chemical parameters of blood under such conditions (3, 8).

Influence of exercise's intensity on blood hemorheology

Changes in the values of hemorheological parameters differ depending on the type and intensity of physical activity. Elasticity of erythrocytes does not change during exercise until the lactic acid level threshold is greater than 4 mmol/L and then begins to decline. This phenomenon can be considered an adaptation leading to vasodilation in muscles (5). The so-called "crossover concept" is based on the use of lipid oxidation during endurance training, as opposed to strength training, which promotes carbohydrate metabolism, due to the increased activation of the sympathetic nervous system component (12). The above two types of training are distinguished by the level of LIPOXmax (maximum lipid oxidation, LIPOXmax) which takes place below the lactic acid threshold level (4 mmol/L). At low intensities of training preferential oxidation of lipids is observed, in contrast to high intensities promoting carbohydrate oxidation. LIPOXmax values vary depending on the concentration of citrate synthase in the muscles and the ability of the mitochondria to oxidize fatty acids. Training at the LIPOXmax level is effective for people with a sedentary lifestyle to reduce body weight and increase FFM (fat-free mass), increase the ability to burn fat, lower blood cholesterol, lower glycemia and HbA1c (glycated hemoglobin, HbA1c) in type 2 diabetes (13). During low intensity training - below the LIPOXmax, RBC deformability remains unchanged, while high-intensity training promoting carbohydrate expenditure is characterized by lactic acid release, acidosis, high systolic pressure and high shear stress, resulting in reduced RBC deformability (13).

The "healthy primitive lifestyle" hypothesis assumes that the widespread prevalence of diseases such as type II diabetes and obesity is due to genetic and epigenetic factors promoting the so-called "Sparing phenotype". This phenotype is favourable for the hunter-gatherer lifestyle of our ancestors, with a diet high in complex carbohydrates and proteins and low in fat. In such a model, obesity became a way of survival because an individual who under physiological conditions practiced light or moderate physical activity, in conditions of immobility, e.g. due to illness, had to have an energy storage strategy. The so-called the obesity paradox, indicates that due to the past of our species, gaining weight is an adaptive response to a reduced amount of movement (5).

A "healthy primitive lifestyle" is a state between two extremes - athletic and sedentary. This level of physical activity promotes fat burning and carbohydrate saving, while a sedentary lifestyle induces fat storage. In both groups, athletic and sedentary, abdominal obesity measured as WHR (waist-to-hip ratio, WHR) is connected with deterioration of hemorheological parameters. However, in people with a high level of physical activity WHR is positively correlated with hematocrit and plasma viscosity, whereas in sedentary people, obesity itself increases hematocrit and its ventral location increases plasma viscosity regardless of the hematocrit (6).

Impact of various sport disciplines

Endurance training is characterized by medium intensity, aerobic exercise performed in long period of time and includes for example long-distance running, swimming and cycling. Different types of endurance sport vary in their hemorheological effects. A 10 km run in endurance trained athletes induced improvement in RBC deformability, decreased erythrocyte aggregate strength and blood viscosity, whereby hematocrit remained unchanged. This running exercise did not result in elevation of eryptosis markers (14). Running an ultra-marathon may cause exercise associated anemia, which indicators are decrease in RBC count, hemoglobin and hematocrit. The factors that may induce sports anemia include hemodilution, oxidative stress, iron deficiency, gastrointestinal bleeding, foot-strike hemolysis and hematuria (15, 16).

In study comparing two long-distance running competitions: a 40 km race and a 171 km race, some differences were observed. Blood viscosity and RBC aggregation increased after classic trail race, while it remained unchanged after ultra-marathon. The better hemorheological profile after longer distance was mainly caused by the decreased hematocrit. Another observation was that 171 km race promoted releasing microparticles from RBC, resulting in their lower deformability and was connected with IL-6 (interleukin 6, IL-6) increase. The shorter race did not show any signs of membrane vesicle release but induced RBC senescence, presented by higher phosphatidylserine exposure on erythrocyte membrane. Both distances were not associated with rise in markers of hemolysis and oxidative stress (9). The probable cause of this phenomenon might be that endurance training promotes erythrocyte ageing also leads to increased RBC turnover (17).

Acute cycling exercise results in increase of blood viscosity and erythrocyte aggregation with no impact on RBC deformability, whereas running causes increased RBC deformability and no change in blood viscosity (18). A study performed on well-trained athletes showed decreased deformability and increased aggregation of RBC after acute cycling exercise.

This type of endurance training also was not associated with stronger eryptosis nor increase in its markers (19).

Soccer players, whose training consists in longer, medium-intensity physical activity, present lower RBC aggregation and higher lactate generation than ice hockey players. In contrast, ice hockey players who train mainly at higher intensities in short periods of time (strength exercise), showed better RBC deformability and more efficient lactate elimination. Probable cause is better endurance of soccer players and higher VO2 max (oxygen ceiling, VO2 max - a measure of the body's oxygen capacity, defined as the number of liters of oxygen taken from the environment per minute) obtained during exercise, compared with ice hockey players (8).

A study conducted on professional female rowers showed lower erythrocyte level, lower plasma viscosity, lower RBC deformability and higher aggregation after the 10-month rowing training period (20).

In contrast to endurance exercise, yoga is characterized by lower intensity and oxygen demand and higher heart rate. Regular practitioners of yoga demonstrated significantly lower whole blood viscosity than sedentary participants with no differences in hematocrit. Regular exercise was defined as Hatha yoga practice for at least one hour, three times per week for three years. Another observation was tendency to lower systolic blood pressure, which may be a result of more favourable rheological status (21).

Effects of sport's regularity and overtraining

Irregular, too intensive physical activity leads to impairment of hemorheological properties. In contrast, adequate and regular sports training induces state of "hemorheological fitness" which markers are decreased plasma and blood viscosity, hematocrit and higher erythrocyte deformability with lower tendency to aggregate (22). This phenomenon discloses the difference between short-term and long-term effects on blood rheology induced by sport, which will be discussed further in this article.

Compared with trained athletes, after high-intensity exercise untrained people present higher blood viscosity and RBC aggregation with lower RBC deformability. The same parameters were significantly more favourable in trained, professional sportsmen (8).

However, sedentary or non-athlete patients can also benefit from regular physical exercise, even if the impact is less evident than in sportsmen who train regularly. 6-week endurance training program involving submaximal running resulted in higher proportion of young RBC, increased deformability and improved exercise performance in untrained participants (17). Dance movement in elderly, sedentary women affected blood properties by improving erythrocyte deformability and reduced aggregation (23).

Overtraining is one of the negative consequences of exercise, defined as a process of reducing the efficiency of training, despite increasing its intensity and may manifest itself in general weakness, pain in muscles and joints, loss of appetite and depression. Excessive exercise may also have a negative impact on hemorheological parameters - in overtrained people, increased blood viscosity is observed due to an increase in plasma viscosity and hematocrit.

However, no changes in deformability and aggregation of erythrocytes were found. These changes reflect the reversal of the process of "autohemodilution" present in well-trained athletes, consisting in increasing blood volume and reducing its viscosity (23, 24, 25, 26).

During a study conducted on professional athletes, it was observed that people exhibiting one of the typical symptoms of overtraining - the feeling of "heavy legs" - have increased blood viscosity, and the RBC of such people aggregate faster compared to the control group (23, 24, 25, 26). The reasons for the above changes are believed to be the imbalance between the burden and the regeneration of the body, resulting in a mild inflammatory response. Excessive exercise causes injuries to muscles, bones and joints, leading to the activation of circulating monocytes by pro-inflammatory cytokines, such as IL-1β (interleukin-1beta IL-1β), IL-6 (interleukin 6, IL-6), or TNF-α (tumor necrosis factor-α, TNF-α). As increased blood viscosity accompanies many states of inflammation, its presence in over-trained athletes may be caused by an inflammation-dependent mechanism (27, 28).

Short-term, delayed and long-term effects of exercise

Physical activity affects the rheological parameters of blood depending not only on the type of exercise, but also on the phase of the impact (24, 29). Depending on the duration, three main phases are distinguished as short-term, delayed and long-term effects.

The short-term effect is an increase in blood viscosity, which is called "hemoconcentration". It results mainly from the increase in plasma viscosity and the increase in hematocrit and is the result of the action of 5 mechanisms: redistribution of erythrocytes in the vessels, splenic contraction leading to an increase in the number of circulating erythrocytes, increased plasma protein content, water loss with sweat in the process of thermoregulation and water entering into muscle fibers (24, 29). After acute high-intensity interval training a rise in hematocrit, hemoglobin, red cell count, mean cell volume, platelet count, total white cell count was observed. Mentioned values increased immediately after the training, remained elevated until 3 hours after exercise and completely returned to resting levels 6 h after exercise (30).

Another theory explaining the phenomenon of hemoconcentration is that exercise-induced changes in blood viscosity are mainly due to plasma shifts from intravascular space to extravascular spaces rather than plasma volume loss through sweating and respiratory tract (31). In study comparing the effects of exercise in both hydrated and dehydrated state, the rise in hematocrit and hemoglobin was not significantly related to the extent of hydration, but the intensity of training. This confirms the theory of the plasma shift as a cause of hemoconcentration (32).

Another short-term effect of exercise is an increase in RBC stiffness. One of the mechanisms leading to this is lactic acidosis, induced by the organisms' transition to anaerobic metabolism[24]. It has been shown that lactates, as osmotically active compounds, cause swelling of erythrocytes, which leads to an increase in hematocrit, which is the most important factor determining blood viscosity (33, 34). RBC stiffness may also be caused by oxidative stress and the production of free radicals during physical activity - along with an increase in the content of malondialdehyde in the erythrocyte membrane (oxidative stress marker), the stiffness of RBC also increases (24, 29).

Moreover, accumulating oxidative stress leads to shedding damaged components of erythrocytes in a form of microparticels. This mechanism changes the surface-area-to-volume ratio in RBC and alters their deformability (19).

However, as long as the endothelium is vasodilatable, a slight initial increase in blood viscosity, which theoretically should make perfusion less effective, can oppositely optimize blood flow. Increased shear stresses during acute exercise generate nitric oxide synthesis, which is strong vasodilator, and result in decrease in vascular resistance. Furthermore, the rise in hemoglobin during exercise-induced hemoconcentration makes the oxygen supply to muscle cells more effective. Thus, we can interpret the short-term effects of movement as seeking to increase its efficiency rather than impairing it. Today's knowledge enables us to determine which extent of blood viscosity is the most beneficial for exercise's performance, but the changes of hemorheological properties during physical activity are evident and should be further studied (5).

Delayed changes, collectively known as "autohemodilution", are also observed during exercise (24, 29). It is believed that "the more athletic an athlete is, the more fluid his blood is". This phenomenon consists in the reduction of blood viscosity, inter alia, due to a decrease in hematocrit - the initial "hemoconcentration" connected with dehydration, is preceded by the following decrease in plasma volume (4, 35). Autohemodilution is probably an adaptive mechanism of the body, caused by an attempt to adapt to the body's greater oxygen demand during movement. This may be a "reserve" to possibly increase hematocrit for physical activity.

Decrease in hemoglobin concentration and hematocrit lowers the costs of circulation by reduction of blood viscosity with no loss in tissue oxygenation due to increase in circulation rate (36). It has been shown that people with lower hematocrit (< 40 %) are more physically efficient - they have higher aerobic capacity compared to athletes with higher hematocrit. An interesting fact is the so-called "hematocrit paradox" where autotransfusion or doping with the use of erythropoietin increases physical performance, while under normal conditions a negative correlation between erythropoietin levels and physical performance is observed due to the occurrence of "autohemodilution" (37, 38).

Physical activity, apart from short-term effects, also causes changes in the values of hemorheological parameters with a metabolic and hormonal background. These changes are classified as long-term changes. It is promoted by the model of body composition with a low-fat content - this mechanism probably plays a leading role in improving the values of hemorheological parameters induced by regular training (24, 30). A simple, two-compartment description of the body is used here as consisting of FM (fat mass, FM) and FFM (fat-free mass, FFM). Excess FM, represented by adipocytes, causes the development of insulin resistance and metabolic disorders, while FFM, which includes for example muscles, bones, blood and viscera, are the sites of insulin action and promote insulin sensitivity (39). It has been shown that RBC aggregation is proportional to FM and is correlated both with abdominal and appendicular fat mass. Hematocrit level is positively correlated with FFM (including muscle mass and non-muscular mass), abdominal fat and waist-to-hip ratio (40). However, due to the fact that the increase in FFM is a common feature of obesity, it cannot be concluded that its very high level in athletic people is a beneficial phenomenon.

It has been noted that it may contribute to an unfavorable hemorheological profile in some, both sick and healthy populations, possibly due to worsening insulin sensitivity. In the group of postmenopausal women, the increase in FFM was positively correlated with insulin resistance and with high levels of VF (visceral fat, VF) - in addition, this relationship was stronger than between the level of VF itself and insulin resistance. However, it is necessary to distinguish between studies conducted on people with a sedentary lifestyle, genetically predisposed to the phenotype with high FFM content, and those on people with training-induced increase in FFM. Currently, the mechanism of linking the FFM level with the hemorheological profile is completely unknown (39, 41).

Insulin sensitivity is positively correlated with the level of physical activity due to the improvement of body composition and an increase in glucose and lipid metabolism by the muscles, which explains why it is one of the treatments for insulin resistance (24, 29). It has been shown that lifestyle modification, which consists of regular exercise and improving the diet in obese people, results in an increase in fibrinogen levels, counterbalanced by a decrease in erythrocyte aggregation and a decrease in the concentration of LDL and triglycerides in the blood. In this study decrease in insulin concentration and the insulin resistance index was also observed (42).

The direct effect of training (2-3x / week for 45 minutes for 2 months) in people diagnosed with type 2 diabetes is a decrease in erythrocyte aggregation. In addition, the loss of fat tissue caused by movement in such people is associated with a decrease in blood viscosity, and a decrease in the waist circumference causes a decrease in hematocrit, plasma viscosity and aggregation of RBC. In the studied patients, an increase in VO2 max resulted in a decrease in blood viscosity and an increase in RBC deformability (43).

The relationship between the aggregation of blood cells and the amount of adipose tissue is also observed with its normal content in healthy people, which proves that it is not a pathological mechanism, but a physiological relationship. Probably FM is the main factor regulating RBC aggregation (44, 45).

It is well known that physical exercise improves blood pressure and is highly recommended for hypertensive patients. One of the mechanisms underlying this connection is exercises' influence on blood viscosity. According to Poiseuille’s Law, any decrease in blood viscosity should cause a decline in vascular resistance (46). Regular physical activity in long-term results in decrease of blood viscosity and further leads to drop of vascular resistance and systemic blood pressure (47). High intensity interval training can improve blood fluidity markers and also reduce systolic blood pressure in hypertensive patients. It was found that after 8 weeks of high intensity interval training, patients presented lower levels of blood and plasma viscosity, fibrinogen concentration and RBC aggregation and higher erythrocyte deformability (48).

As previously mentioned, the short-term effects of physical exercise also include the formation of free radicals, however, the chronic effects of training make it possible to eliminate this phenomenon, and even to prevent oxidative stress-induced diseases, such as atherosclerosis or cancer, by developing appropriate adaptation processes (49). High intensity training increases oxidative stress and the rise is positively correlated with exercise’s intensity and duration.

The oxidative response is transient and the elevation of its markers is not dangerous. Surprisingly, this outcome might be favorable due to stimulating organism's antioxidant system and improving antioxidant capacity (50). Activation of laminar shear stress leads to the down-regulation of the type II angiotensin receptor, which leads to a reduction in NADPH oxidase activity, resulting in a lower generation of ROS (reactive oxygen species, ROS) and protection of the availability and antiathergenic properties of endothelial nitric oxide (49). Nitric oxide is not only a vasodilating factor, but also leads to improvement of erythrocyte membrane flexibility through direct S-nitrosylation of cytoskeleton proteins, most likely α- and β-spectrins (51). Additionally, an increase in antioxidant enzymes such as superoxide dismutase, glutathione peroxidase and catalase is observed during physical activity (29). All these molecular adaptation processes can lead to increased resistance to systemic oxidative stress (49).

Regular exercise protects against the development of atherosclerotic plaques and narrowing of the coronary vessels. The atheroprotective impact of training arises from abovementioned regulation of oxidative stress as good as from the release of anti-inflammatory cytokines by the contracting muscle. One of them is IL-6, which inhibits the synthesis of TNF-α with a pro-inflammatory effect in adipocytes and macrophages (52). In addition, exercise induces a plaque phenotype that is less sensitive to acute changes such as rupture or thrombus development, protecting against the lethal effects of atherosclerosis (53).

Conclusions

In conclusion, physical exercise seems to have a significant impact on hemorheological parameters of blood. Changes in the values of hemorheological parameters differ depending on the intensity of training. Low-intensity movement below the level of maximum lipid oxidation seems to have positive impact on blood viscosity, however various sport disciplines are associated with different hemorheological effects. It complies with the hypothesis of a "healthy primitive lifestyle", saying that the most beneficial for the metabolic and hemorheological profile of the organism is big amount of regular, moderate, lipid-burning exercise.

Physical activity affects the rheological parameters of blood depending not only on the type of exercise, but also on the phase of the impact. In short term exercise causes "hemoconcentration", characterized by increase in hematocrit and decrease in plasma volume and erythrocyte deformability. In long term sports training causes reduction of blood viscosity, by "autohemodilution", increasing RBC deformability and promoting a positive metabolic and hormonal profile. Furthermore, sport promotes more favourable body composition, characterized by higher level of insulin-sensitive muscle mass and lower level of visceral fat, connected with decrease in aggregation and blood viscosity. Additionally, physical activity induces transient oxidative stress activation, which in long term stimulates antioxidant system and improves antioxidant capacity.

This research confirms that taking up physical activity may have a positive effect on the hemorheological parameters, contributing to more efficient work of the circulatory system, however the relationship depends on many factors. The topic of the sport's influence on blood viscosity should be further investigated and more studies are clearly needed.

Disclosure:

Authors'contribution:

Conceptualization: Anna Maria Szpernalowska, Anna Marcinkowska-Gapińska
Methodology: Anna Maria Szpernalowska
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Writing-Review and Editing: Anna Maria Szpernalowska, Anna Marcinkowska-Gapińska
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