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Physical Features Of Platelet Activity During Low Physical Activity.

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ABSTRACT

In the process of human physiological development, a significant place along with genetics is occupied by the dynamics of the functional activity of the platelet hemostasis system. It becomes clear that the normal development of the functional capabilities of the organism and the morphological development of organs and systems is largely due to optimal tissue perfusion, a strongly strongly determined functional activity of platelets. Low platelet activity causes the rheological properties of blood, its sufficient flow to the tissues, the adequacy of cardiac activity, a high level of resistance to infectious diseases and the necessary metabolic rate. Non-exercising young men revealed a gradual slight increase in the functional activity of platelets. Thus, at the age of 18 to 19 years old, their platelet aggregation was at a low level, then gradually increased with increasing chronological age. This is apparently due to the increase in their sensitivity to exogenous influences. The growth of platelet activity inevitably leads to an increase in circulating aggregates of various sizes, having a negative effect on the microcirculation of tissues in the body of a person who does not exercise physically.

Keywords: age, lack of physical training, platelets, aggregation, intravascular activity.

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INTRODUCTION

At a young age, the body, as a rule, is in an optimal functional state [1-4]. However, adolescence is often the time of the formation of various pathologies [5,6]. Especially with adverse environmental effects [7]. It has been observed that in the process of maintaining homeostasis during the physiological development of a person, the formation of platelet hemostasis activity [10] occupies a significant place along with genetics [8,9]. It becomes clear that the normal development of the functional capabilities of the body and the morphological development of organs and systems is largely determined by the optimal functional activity of platelets [11,12], due to their influence on the rheological properties of blood, its flow to the tissues [13], the adequacy of cardiac activity [14], a high level of resistance to infectious diseases [15] and metabolic rate [16].

At the same time, in healthy people who do not have bad habits and do not exercise regularly, the state of lipid peroxidation (LPO) of platelets and the activity of their antioxidant enzymes, which largely determine the level of functional ability of platelet hemostasis, has not been fully clarified. The features of the aggregation function of platelets under the influence of various inductors and their combinations, which are present in real blood flow conditions, remain unclear in this contingent of individuals. Also, they have not evaluated the degree of morphological activity of platelets in the lumen of blood vessels. Based on this, the goal of the present study was formulated: to determine the activity of platelet functions in healthy young people who do not have bad habits and who do not regularly exercise physically.

MATERIALS AND METHODS

The study was approved by the local ethics committee of the Russian State Social University on September 14, 2016 (protocol №19). The study was conducted on the basis of the Russian State Social University.

The study group included 141 healthy young people who do not exercise physically (29 people 18 years old, 26 people 19 years old, 27 people 20 years old, 28 people 21 years old and 31 people aged 22 years). In the examined, intra thrombocytic LPO was evaluated by the concentration of the basal level of malonic dialdehyde (MDA) in the reduction reaction with thiobarbituric acid and by the level of acyl hydroperoxides (AHP). The number of platelets in capillary blood in the Goryaev chamber was counted. The products of platelet-induced phospholipid-coagulation activator (F_3-platelets) labilization were assessed by the traditional method with calculation of the platelet activity index. Platelet aggregation (AP) was studied by a visual micromethod using as inducers ADP (0.5×10^{-4} M), collagen (dilution 1: 2 of the main suspension), thrombin (0.125 units / ml), ristomycin (0.8 mg/ml), adrenaline (5×10^{-6} M), as well as combinations of ADP and adrenaline, ADP and collagen, adrenaline and collagen to simulate real blood flow conditions. The intravascular activity of platelets was determined visually using a phase contrast microscope. Statistical processing of the results obtained was carried out using Student's t-test.

RESULTS AND DISCUSSION

All young people taken into the study were under constant observation. Before each study, during the entire observation period, they determined the main physiological parameters, carried out morphological and biochemical blood tests. The results of these studies showed that the estimated total functional and biochemical values (temperature, heart rate, respiration rate, general blood and urine tests, blood biochemical tests) in the subjects during all observation periods were within the physiological norm.

The concentration of primary products of POL-AHP in platelets of healthy 18-year-old young people who do not exercise physically was at a level of 2.02±0.13 $D_{233}/10^9$ platelets, not changing significantly by the age of 19, increasing ($p<0.05$) to 22 years up to 2.24±0.07 $D_{233}/10^9$ platelets. At the same time, the level of basal MDA in platelets - the end product of LPO at 18 years of age was 0.52±0.12 nmol/10^9 platelets, having a tendency to increase to 19 years of age and then to increase reliably by 22 years to 0,69±0.19 nmol/10^9 platelets.

The level of catalase and superoxide dismutase activity in the blood plates, which were monitored by healthy young people, did not have reliable dynamics by the age of 19, amounting to this age 9280.0±200.8 IU/10^9 platelets and 1646.0±16.0 IU/10^9 platelets, respectively. In subsequent follow-up periods, a small but

significant decrease in catalase and superoxide dismutase was observed in the examined (at 20 years, 9100.0±126.0 IU/10^9 platelets, 1600.0±17.2 IU/10^9 platelets, 21 years old - 9000.0±130.9 IU/10^9 platelets, 1540.0±11.5 IU/10^9 platelets, 22 years old - 8910.0±166.4 IU/10^9 platelets, 1500.0±16.6 IU/10^9 platelets, respectively).

The index of platelet activity was 18 years in the surveyed 20.9±0.14%, remaining at this level by 19 years. This indicated the stability during this age period in the blood plates of healthy young people who do not exercise physically, the level of labilization products of platelet phospholipids - blood clotting activators. An assessment of the platelet activity index of young people who are not experiencing physical exertion, of an older age showed an unexpressed, but significant increase in it - 20 years 22.2±0.09%, 21 years old - 23.2±0.08%, reaching 22 years old level of 23.7±0.11%.

In young people at 18 years of age, the time of development of antibodies under the influence of collagen was 33.9 ± 0.27 s, being at the same level at the age of 19 years. Similar AP activity in healthy 18 year old untrained young people was observed under the influence of ADP (44.6 ± 0.06 s) and ristomycin (48.9 ± 0.20 s). Later, thrombin and adrenaline AP developed, being at age of 56.7 ± 0.14 s and 106.3 ± 0.10 s, respectively, not changing significantly during the next year of life. The absence of AT dynamics in non-physically trained young people by the age of 19 with the combined use of inductors was also established: for ADP + adrenaline - 36.7 ± 0.14 s, for ADP + collagen - 27.8 ± 0.15 s, for adrenalin + collagen - 29.5 ± 0.12 s. At the same time, since the age of 20, there is a significant acceleration of AP with isolated inducers and their combinations, which are most pronounced in the group of patients surveyed from 22 years of age (Table 1).

Table 1. Platelet aggregation activity in healthy young people, not exercising physically

Stimulated platelet aggregation	Young people who do not exercise physically, n=141 M±m				
	18 years, n=29	19 years, n=26	20 years, n=27	21 years, n=28	22 years, n=31
in response to ADP, s	44.6±0.06	44.7±0.15	44.2±0.12 p<0.05	43.6±0.19 p<0.05	43.0±0.05 p<0.05
in response to collagen, s	33.9±0.27	33.8±0.26	33.3±0.12 p<0.05	32.7±0.17 p<0.05	32.0±0.04 p<0.05
in response to thrombin, s	56.7±0.14	55.4±0.19	54.8±0.16 p<0.05	54.1±0.11 p<0.05	53.6±0.16 p<0.05
in response to ristomycin, s	48.9±0.20	48.7±0.06	48.0±0.13 p<0.05	47.5±0.06 p<0.05	47.1±0.15 p<0.05
in response to H_2O_2, s	49.4±0.17	49.6±0.09	49.0±0.19 p<0.05	48.5±0.09 p<0.05	48.0±0.18 p<0.05
in response to adrenaline, s	106.3±0.10	105.6±0.22	98.3±0.02 p<0.05	97.6±0.11 p<0.05	96.8±0.11 p<0.05
in response to ADP + adrenaline, s	36.8±0.15	36.7±0.14	35.7±0.06 p<0.05	35.1±0.13 p<0.05	34.6±0.17 p<0.05
in response to ADP + collagen, s	27.9±0.17	27.8±0.15	27.2±0.05 p<0.05	26.7±0.14 p<0.05	26.2±0.12 p<0.05
in response to adrenaline + collagen, s	29.6±0.20	29.5±0.12	28.9±0.09 p<0.05	28.2±0.06 p<0.05	27.7±0.05 p<0.05

Legend: p - reliability of differences in the estimated indicators in different age periods.

The level of blood cells in healthy untrained young people at 18 and 19 years of age was 85.6±0.15%, and 85.4±0.10%, respectively. The number of disco-echinocytes, spherocytes, sphero-echinocytes and bipolar forms of platelets, also remained stable in their bloodstream from 18 to 19 years. As a result, the sum of active forms of platelets also did not undergo significant changes, amounting to 19 years to 14.6±0.12%. In the blood of the examined young people who did not exercise physically, the levels of free-circulating small and large platelet aggregates did not have reliable dynamics, reaching by the age of 19 3.0±0.11 and 0.07±0.009 per 100 free-lying platelets, reaching 22 by the age of 3.6±0.04 and 0.10±0.007 per 100 free-lying platelets

(p<0.05), respectively. The number of platelets involved in the process of aggregation also did not change between 18 and 19 years in the examined, making up to 19 years of age 6.1±0.12%. However, by the age of 20 in healthy untrained young people, a small significant increase in intravascular platelet activity was observed, gradually increasing by 22 years of age (the sum of active forms 20.0 ± 0.09%) (Table 2).

Table 2. Intravascular platelet activity in healthy people, not exercising physically

Platelet forms and variants of their aggregates	Young people who do not exercise physically, n=141 M±m				
	18 years, n=29	19 years, n=26	20 years, n=27	21 years, n=28	22 years, n=31
Discocytes, %	85.6±0.15	85.4±0.10	83.0±0.04 p<0.05	82.1±0.03 p<0.05	80.0±0.04 p<0.05
Disco-echinocytes, %	8.5±0.15	8.7±0.11	10.5±0.12 p<0.05	11.3±0.14 p<0.05	12.9±0.17 p<0.01
Spherocytes, %	3.0±0.12	2.9±0.10	3.2±0.10 p<0.05	3.3±0.09 p<0.05	3.5±0.07 p<0.05
Sphero-echinocytes, %	1.9±0.15	1.9±0.07	2.1±0.06 p<0.05	2.2±0.07 p<0.05	2.4±0.03 p<0.05
Bipolar forms, %	1.0±0.09	1.1±0.15	1.2±0.10	1.1±0.07	1.2±0.06
Sum of active forms, %	14.4±0.14	14.6±0.12	17.0±0.15 p<0.05	17.9±0.10 p<0.05	20.0±0.09 p<0.01
The number of platelets in the aggregates, %	6.0±0.10	6.1±0.12	6.3±0.07 p<0.05	6.5±0.08 p<0.05	6.8±0.06 p<0.05
The number of small units of 2-3 platelets per 100 free platelets	2.9±0.14	3.0±0.11	3.2±0.06 p<0.05	3.4±0.07 p<0.05	3.6±0.04 p<0.05
The number of medium and large aggregates, 4 or more platelets, per 100 free-lying platelets	0.07±0.010	0.07±0.009	0.08±0.005 p<0.05	0.09±0.006 p<0.05	0.10±0.007 p<0.05

The structures and functions of the body, ensuring its local and general reactivity, are largely shaped by an adequate supply of nutrients due to the required level of blood rheology, which can change during ontogenesis under the influence of various environmental factors, which undoubtedly include the presence or absence of regular adequate physical training [17-20].

There is no doubt that a large role in the dynamics of the state of microcirculation is played by the level of LPO of platelets and the activity of platelets.

In healthy young people 18 and 19 years old who do not exercise physically, there is a stability of the antioxidant activity of platelets and the level of LPO in them, which causes the constancy of the activity of primary hemostasis. This largely contributes to a stable low activity of the blood platelets in young people who are not experiencing physical exertion at this age.

In the course of research, older, untrained young people showed a gradual increase in the functional activity of platelets. So, already in 20 year old young people, AP turns out to be significantly accelerated, gradually increasing further as the chronological age increases. Obviously, this is due to the increased sensitivity of platelet receptors to exogenous effects on platelets, which include an increase in the concentration of von Willebrand factor in the blood - a cofactor for adhesion of platelets with a simultaneous increase in the number of receptors to it - (GPI) on the surface of blood plates [21]. Receptor rearrangements on the membranes of the blood platelets, caused by the reaction of the hemostatic system to the features of the functional activity of the organism as a whole, are the result of complex adaptive reactions in the body of

the examined and membrane changes of platelets, which ultimately lead to the adaptation of platelet hemostasis to the existing conditions of functioning [22].

Evaluation of AP with a number of inductors and their combinations in young people who do not exercise physically, confirmed the increased aggregative function of the blood platelets. At the same time, the acceleration of AP during binding, incl. Strong aggregation agonists — collagen and thrombin with receptors on the platelet membrane — can be caused largely by the activation of phospholipase C, which stimulates the phosphoinositol pathway through diacylglycerol and protein kinase C, with phosphorylation of the contractile system proteins. The resulting inositol triphosphate contributes to a more active release of Ca^{2+} from intra platelet depots. Strengthening of these mechanisms causes a pronounced intensification of actomyosin contraction. It is possible that the role of acceleration of AP also plays a gradual increase in the activity of the enzyme systems of platelets, including thromboxane formation, causing an earlier response of the platelets to the stimulus.

Similar reactions were noted for weak agonists of platelet aggregation - ADP and adrenaline, interacting with their membrane receptors and causing expression of fibrinogen receptors (GPIIb-IIIa), stimulating phospholipase A_2, regulating the release of arachidonic acid phospholipids with increased formation of thromboxane A_2 [23].

Simultaneous assessment of antibodies with the use of several inductors showed their mutually potentiating action, confirming the patterns found in the study of antibodies with isolated agonists.

A marked increase in intravascular platelet activity in young people who do not exercise physically, after 19 years of age indirectly indicates an increase in blood of the physiological level of aggregation inducers (thrombin, ADP, adrenaline) with an increase in the basal level of platelet sensitivity to them. At the same time, in healthy young people who do not exercise physically, since the age of 20, a significant decrease in the number of intact discoid platelets begins to develop in the bloodstream, indicating an increase in the activity of their receptors. An increase in the level of disco-echinocytes and other active forms of platelets coincides with an increase in the hemostatic activity of platelets, primarily due to the expression on their membrane of fibrinogen receptors (GP IIb - IIIa).

CONCLUSION

As the chronological age increases, people who do not exercise physically increase the activity of platelets. This is manifested by an increase in the content of their active forms in the bloodstream of these people, which inevitably leads to an increase in the number of circulating aggregates of various sizes. As a result, physically untrained people in adulthood increase the activity of hemostasis, leading to the formation of thrombogenic readiness and a high risk of vascular catastrophes.

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