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ALTERATIONS OF HEMOSTATIC PARAMETERS FOLLOWING ACUTE ENVIRONMENTAL STRESS EXPOSURE

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Acute hypoxia is a significant environmental stressor capable of inducing adaptive and pathophysiological changes in the hemostatic system. The obtained findings demonstrate that acute hypoxic exposure leads to marked disturbances in the hemostatic system, reflected by significant changes in coagulation parameters associated with the development of a hypercoagulable and prothrombotic state. The present study aimed to evaluate alterations in blood coagulation parameters under conditions of acute hypoxia and during the post-hypoxic recovery period.

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***Keywords*:** acute hypoxia, hemostasis, blood coagulation, fibrinogen, hypercoagulability, adaptation, environmental stress factors.

Introduction. The hemostatic system is one of the principal regulatory systems of the body responsible for maintaining blood fluidity and preventing hemorrhage. Various environmental factors are capable of inducing adaptive responses that affect both vascular-platelet and coagulation components of hemostasis. Of particular interest are acute environmental stressors, including hypoxia, abrupt temperature fluctuations, changes in barometric pressure, and intense physical or psycho-emotional stress, as these conditions may provoke rapid alterations in blood coagulation properties [1].

In recent years, considerable attention has been devoted to investigating the effects of acute hypoxia as one of the most common extreme environmental factors. Exposure to hypoxic conditions is accompanied by activation of compensatory and adaptive mechanisms associated with alterations in microcirculation, blood rheology, platelet activity, and plasma coagulation factors. According to current concepts, hypoxia induces the expression of hypoxia-inducible factors, which play a key role in the regulation of vascular tone, erythropoiesis, inflammatory responses, and hemostatic processes [2]. Despite numerous studies addressing hemostatic disturbances under adverse environmental conditions, the available data remain controversial. Some investigators report the development of hypercoagulability and increased thrombotic risk under hypoxic conditions, whereas others demonstrate minimal or no significant alterations in coagulation potential following short-term hypoxic exposure. These inconsistencies indicate the complexity of hemostatic adaptation mechanisms and emphasize the need for further investigation [2].

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Alterations in blood coagulation parameters in response to acute environmental stressors are of considerable theoretical and practical importance. Understanding the mechanisms of hemostatic adaptation may contribute to the assessment of physiological reserves, prediction of thrombotic complications, and development of preventive strategies for disorders associated with impaired microcirculation and vascular dysfunction under extreme conditions.

Materials and Methods. Measurements of Prothrombin time (PT), Activated partial thromboplastin time (APTT), Thrombin time (TT), Fibrinogen concentration and clotting time were assessed under normal (intact) condition, immediately after hypoxic exposure, 3 *h* after hypoxia, and 10 days later.

A hypobaric hypoxia model was employed in the present study. During the experiments, the rates of compression and decompression within the hypobaric chamber were maintained at 15–20 *m*/*s*. Animals were exposed to simulated high-altitude conditions equivalent to 7000 *m* above sea level for a duration of 40 *min*. Prior to hypoxic exposure, the animals were immobilized on the experimental platform by fixation of the upper and lower limbs and allowed to adapt to the restrained condition for 30 *min*, after which cardiopuncture was performed for blood collection. Hemostatic parameters were analyzed using the HumaClot Junior analyzer. Blood samples were collected into plastic vacuum tubes containing 3.2% sodium citrate as an anticoagulant.

Results and Discussion. The data presented in Table demonstrate that acute hypoxic exposure induces significant alterations in the hemostatic system, characterized predominantly by activation of the coagulation cascade and the development of a hypercoagulable state. These changes were observed immediately after hypoxic exposure, reached maximal expression 3 *h* later, and partially regressed by the 10th day following exposure.

Changes in blood coagulation parameters under conditions of acute hypoxia

Test	Prothrombin time (PT), *s*	Activated partial thromboplastin time (APTT), *s*	Thrombin time (TT), *s*	Fibrinogen / Cloting time, (*mg*/*dL*) / *s*
Norm	15.8 ± 1.04	33.0 ± 1.81	35.2 ± 1.58	104.0 ± 19.47 / 22.2
Hypoxia exposure	15.0 ± 1.04	29.0 ± 0.81*	32.1 ± 1.02 *	156.0 ± 11.71 / 29.2 *
3 *h* after hypoxia	13.9 ± 1.48 *	27.1 ± 1.58 *	31.7 ± 1.75 *	158.0 ± 11.63 / 30.1 *
10 day later	14.9 ± 1.58*	31.9 ± 1.08	34.8 ± 0.47	126.0 ± 11.53 / 26.3 *

Note: * Indicates the level of statistical significance of the changes at $p < 0.05$.

Immediately after hypoxia, a statistically significant shortening of APTT was observed, decreasing from 33.0 ± 1.81 *s* in the control group to 29.0 ± 0.81 *s* ($p < 0.05$). TT also decreased significantly from 35.2 ± 1.58 *s* to 32.1 ± 1.02 *s* ($p < 0.05$). These findings indicate an acceleration of intrinsic coagulation pathway activity and enhanced conversion of fibrinogen to fibrin, reflecting increased coagulation potential.

The most pronounced alterations were detected 3 *h* after hypoxic exposure. PT decreased significantly to 13.9 ± 1.48 *s* ($p < 0.05$), while APTT and TT further shortened to 27.1 ± 1.58 *s* and 31.7 ± 1.75 *s*, respectively ($p < 0.05$). The simultaneous reduction in PT, APTT, and TT suggests activation of both the extrinsic and

intrinsic coagulation pathways, indicating the development of a marked hypercoagulable state. These changes may be attributed to hypoxia-induced endothelial dysfunction, increased expression of tissue factor, platelet activation, and enhanced synthesis or activation of coagulation factors.

One of the most significant findings was the increase in fibrinogen concentration. Fibrinogen is known to be an acute-phase protein that plays an important role in increasing blood viscosity, promoting cellular aggregation, and facilitating thrombus formation. A substantial increase in fibrinogen concentration was also observed following hypoxic exposure. Fibrinogen levels increased from 104.0 ± 19.47 *mg*/*dL* in the control group to 156.0 ± 11.71 *mg*/*dL* immediately after hypoxia and remained elevated at 158.0 ± 11.63 *mg*/*dL* 3 *h* later ($p < 0.05$). Elevated fibrinogen is recognized as an important marker of both acute-phase inflammatory responses and prothrombotic conditions, contributing to increased blood viscosity and enhanced thrombus formation.

The detected hypercoagulable state is consistent with contemporary studies demonstrating that hypoxia activates tissue factor expression, endothelial cells, and hypoxia-inducible factors, all of which contribute to enhanced coagulation processes. Previous studies have also reported an increased risk of thrombosis during exposure to high-altitude and experimental hypoxia [2].

Interestingly, clotting time increased from 22.2 *s* under normal conditions to 29.2 *s* immediately after hypoxia and to 30.1 *s* 3 *h* later. This apparent discrepancy may reflect the complex regulatory nature of hemostasis, whereby activation of coagulation pathways is accompanied by compensatory activation of anticoagulant and fibrinolytic mechanisms aimed at limiting excessive thrombus formation and maintaining vascular patency.

Partial normalization of coagulation parameters after 10 days indicates activation of adaptive mechanisms and gradual restoration of hemostatic balance.

By the 10th day after hypoxic exposure, partial normalization of coagulation parameters was observed. APTT (31.9 ± 1.08 *s*) and TT (34.8 ± 0.47 *s*) approached baseline values, whereas PT (14.9 ± 1.58 *s*) and fibrinogen concentration (126.0 ± 11.53 *mg*/*dL*) remained significantly different from control values ($p < 0.05$).

The observed hemostatic changes are likely associated with activation of complex compensatory-adaptive mechanisms aimed at maintaining tissue oxygenation under hypoxic conditions. In response to oxygen deprivation, the cardiovascular and respiratory systems undergo functional adaptation intended to optimize oxygen delivery to tissues. Simultaneously, profound biochemical and metabolic alterations occur at the cellular level, particularly in organs and tissues experiencing insufficient oxygen supply. These adaptive responses involve modulation of oxidative metabolism, vascular tone, endothelial function, and activation of coagulation pathways [3, 4]. Under prolonged or severe hypoxic conditions, compensatory mechanisms may become unstable, resulting in persistent deviations from physiological homeostasis and increased thrombotic potential [5, 6].

These findings are of considerable physiological and clinical importance, as they contribute to a better understanding of adaptive mechanisms under extreme environmental conditions and may help predict the risk of thrombotic complications associated with hypoxic states.

Conclusion. Overall, the results demonstrate that acute hypoxic exposure induces substantial disturbances in hemostatic balance, characterized by activation of coagulation processes and the development of a transient hypercoagulable state and may increase the risk of thrombotic complications through activation of multiple coagulation mechanisms The peak severity of these alterations occurs approximately 3 hours after hypoxic exposure, suggesting that this period represents a critical phase in the hemostatic response to acute oxygen deprivation.

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ՀԵՄՈՍՏԱԶԱՅԻՆ ՑՈՒՑԱՆԻՇՆԵՐԻ ՓՈՓՈԽՈՒԹՅՈՒՆՆԵՐԸ ՍՈՒՐ ՇՐՋԱԿԱ ՄԻՋԱՎԱՅՐԱՅԻՆ ՍԹՐԵՍԻ ԱԶԴԵՑՈՒԹՅՈՒՆԻՑ ՀԵՏՈ

Սուր հիպօքսիան հանդիսանում է շրջակա միջավայրի կարևոր սթրեսային գործոն, որը կարող է առաջացնել հեմոստազի համակարգի հարմարվողական և պաթոֆիզիոլոգիական փոփոխություններ: Ստացված արդյունքները ցույց են տալիս, որ սուր հիպօքսիկ ազդեցությունը հանգեցնում է հեմոստազի համակարգի արտահայտված խանգարումների, որոնք դրսևորվում են մակարդման ցուցանիշների էական փոփոխություններով և կապված են հիպերկոագուլյացիոն ու պրոթրոմբոտիկ վիճակի զարգացման հետ: Սույն հետազոտության նպատակն է գնահատել արյան մակարդման ցուցանիշների փոփոխությունները սուր հիպօքսիայի պայմաններում և հետհիպօքսիկ վերականգնման ժամանակահատվածում:

С. Г. СААКЯН

СДВИГИ ГЕМОСТАТИЧЕСКИХ ПОКАЗАТЕЛЕЙ ПОСЛЕ ВОЗДЕЙСТВИЯ ОСТРОГО ЭКОЛОГИЧЕСКОГО СТРЕССА

Острая гипоксия является значимым фактором окружающей среды, способным вызывать адаптационные и патофизиологические изменения в системе гемостаза. Полученные результаты свидетельствуют о выраженных нарушениях системы гемостаза, что проявляется значительными изменениями показателей свертывания крови и связано с развитием гиперкоагуляционного и протромботического состояния в условиях острого гипоксического воздействия. Настоящее исследование направлено на оценку изменений показателей свертывания крови в условиях острой гипоксии и в период постгипоксического восстановления.