N O T I C E

THIS DOCUMENT HAS BEEN REPRODUCED FROM MICROFICHE. ALTHOUGH IT IS RECOGNIZED THAT CERTAIN PORTIONS ARE ILLEGIBLE, IT IS BEING RELEASED IN THE INTEREST OF MAKING AVAILABLE AS MUCH INFORMATION AS POSSIBLE

(NASA-TM-76525) THE EFFECT OF SHORT-TERM ANTIORTHOSTATIC HYPOKINESIA ON CENTRAL AND INTRACARDIAC HEMODYNAMICS AND METABOLISM OF A HEALTHY PERSON (National Aeronautics and Space Administration) 17 p HC A02/MF A01 CSCL 05/52

N81-22689

Unclas
42210

NASA TECHNICAL MEMORANDUM NASA TM-76525

THE EFFECT OF SHORT-TERM ANTIORTHOSTATIC HYPOKINESIA ON CENTRAL AND INTRACARDIAC HEMODYNAMICS AND METABOLISM OF A HEALTHY PERSON

V. Ye. Katkov, V. V. Chesturkhin, O. Kh. Zybin, S. S. Sukhotskiy, S. V. Abrosimov, V. N. Utkin

Translation of "Vliyaniye kratkovrennoy antiortostaticheskoy gipokeniezii na tsentral'nuyu i vnutriserdechnuyu gemodinamiku i metabolizm u zdorovogo cheloveka," Kardiologiya, 1978, Number 12, pages 69-75

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON, D.C. 20546 FEBRUARY 1981

STANDARD TITLE PAGE

1. Report No. NASA TM-76525	2. Government Accession No.	3. Recipient's Catalog No.
4. Title and Subtitle THE EFFECT OF SHORT-TERM ANTIORTHOSTATIC HYPOKINESIA ON CENTRAL AND INTRACARDIAC HEMODYNAMICS AND METABOLISM OF A HEALTHY PERSON		5. Report Date February 1981 6. Performing Organization Code
7. Author(s) V. Ye. Katkov, V.V. Chesturkhin, O. Kh. Zybin, S.S. Sukhotskiy, S. V. Abrosimov, V. N. Utkin		8. Performing Organization Report No. 10. Work Unit No.
9. Performing Organization Name and Address Leo Kanner Associates Redwood City, California 94063		11. Contract or Grant No. NASw -3199 13. Type of Report and Period Covered Translation
12. Sponsoring Agency Name and Address National Aeronautics and Space Administration Washington, D.C. 20546		14. Sponsoring Agency Code

15. Supplementary Notes
Translation of "Vliyaniye kratkovrennoy antiortostaticheskoy gipokinezii na tsentral'nuyu i vnutriserdechnuyu gemodinamiku i metabolizm u zdorovogo cheloveka," Kardiologiya, 1978, Number 12, pages 69-75

16. Abstract

The right parts of the heart and the radial artery were catheterized in healthy male volunteers before and 5 days after strict bedrest in antiorthostatic position of the body (-4.5°). After immobilization most values of central circulation showed no essential changes; the only exception were indices characterizing the inotropic myocardial condition. A shift in the direction of acidosis of a mixed character was noted in mixed venous blood, the β-lipoprotein content increased. A decrease in the arteriovenous difference in oxygen was encountered in blood draining from the heart (from the coronary sinus).

17. Key Words (Selected by Author(s))	18. Distribution Statement
	THIS COPYRIGHTED SOVIET WORK IS REPRODUCED AND SOLD BY NTIS UNDER LICENSE FROM VAAP, THE SOVIET COPYRIGHT AGENCY. NO FURTHER COPYING IS PERMITTED WITHOUT PERMISSION FROM VAAP.

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified	15	

THE EFFECT OF SHORT-TERM ANTIORTHOSTATIC HYPOKINESIA ON CENTRAL AND INTRACARDIAC HEMODYNAMICS AND METABOLISM OF A HEALTHY PERSON

V Ye. Katkov, V. V. Chesturkhin, O. Kh. Zybin,
S. S. Sukhotskiy, S. V. Abrosimov, and V. N. Utkin

The stay of a person in conditions of decreased motor activity /69* in combination with gravitation redistribution of blood (weightlessness, immersion, strict bedrest, etc.) does not cause major deviations of such integral indices as the frequency of cardiac contractions or arterial pressures; however, the contractile state of the cardiac muscle changes noticeably [3, 4, 7-9, 15, 28, 33, 39]. The question of causes of these changes and primarily the interconnection with central hemodynamics and metabolism both in the organism in general and in the heart in particular remain little studied; moreover, its solution can have an important significance for clarifying the mechanisms of adaptation of the myocardium to the new conditions of functioning.

The purpose of our work was to study the effect of short-term antiorthostatic hypokinesia on the central and intracardiac hemodynamics and metabolism of a healthy person.

Material and Methods

The basis research conditions. Ten male volunteers participated as the test subjects in the research; they underwent catheterization of the heart and main vessels.

After catheterization in the initial period, six persons were transferred to strict bedrest with an antiorthostatic position of the body (the lower end of the bed was raised by 4.5°), repeated probing

---

*Numbers in the margins indicate pagination in the foreign text.

was done after 5 days. One should note that before probing the heart, the test subject underwent selective catheterization of the main blood vessels of different organs (brain, liver, kidneys, etc.) taking blood samples with total volume 250-300 ml. The time for conduct of the research and its sequence before and after immobilization were identical.

The selection and composition of patients. The question of admission to the study was decided by a physician-expert commission of the Institute of Medical and Biological Problems of the Ministry of Public Health of the USSR, after a preliminary examination, including: examination by clinicians (therapist, surgeon, neuropathologist, oculist, otolaryngologist, dermatologist), a cardiologic study (EKG at rest, during ortho-tests and measured physical load -- 600 kgm, 8 min), roentgenoscopy of the organs of the thoracic cage, general clinical and biochemical analysis of the blood, analysis of the coagulating system of the blood, acquaintance with the reports from the ambulatory card and dispensaries in the patient's living area.

Anthropometric data of the patients are presented in Table 1. One should note that most of these (8 persons) in the past had been active in sports (combat, gynmastics, skiing, swimming) and had a high sports qualification (class 1, candidates and masters of sports). /70

TABLE 1. ANTHROPOMETRIC DATA OF THE TEST SUBJECTS

Patient	Age Years	Height, cm	Body Weight kg	Body Surface cm^2
L.V.V.	30	165	78	179
N.A.F.	30	185	72	194
S.N.G.	35	173	71	182
N.Yu.M.	39	173	70	183
K.D.D.	26	185	78	200
K.Ye.Ye.	39	178	83	199
I.N.Yu.	22	172	64	171
K.V.Ye.	28	176	74	185
Ch.V.V.	35	170	70	180
B.A.R.	22	172	64	171
Average	30.6	174.9	72.4	184.4

Probing of the heart and the main blood vessels. The study was made on an empty stomach or after a light breakfast. One hour before the study the test subjects received 10 mg of Seduxen.

The probing was conducted in a supine position using an angiographic unit, the Tridoros-5S from the Siemens firm (FRG) along with two

catheters: a soft arterial (Sherwood Medical Inc., USA) and a venous (Cournand, N 7), which under local anesthesia with an 0.5% solution of novocaine were introduced by puncture into the radial (or brachial) artery and the ulnar vein, respectively. After the probe of the main blood vessels of different organs, a venous catheter was introduced into the pulmonary artery; after this it was returned to the cavity of the right ventricle of the heart where it was left after first taking samples of blood from the coronary sinus.

The probe was conducted under roentgenotelemonitoring control during which an EKG was recorded, as well as the curve of pressure and index of oxygenous blood.

Recording of hemodynamic indices. Pressure was measured by the Statham P 23 Db (USA) electromanometers; the contracton of the myocardium was evaluated according to the dynamics of the derivative of pulse pressure of the ventricle (max dp/dt, max dp/dt/P, - max dp/dt), which were obtained using electronic differentiation; max dp/dt was calculated as a partial derivative from division of pressure developed at this moment (Dr) by time from the initial isometric contraction (the R deflection of the EKG) to the positive peak of the first derivative (R - max dp/dt), - max dp/dt was calculated in a similar way assuming for the initial isometric weakening II, the tone of the phonocardiogram and for its completion -- the negative peak of the first derivative [16-18, 26, 27, 32, 34, 35]. When recording and processing the results obtained, we took into consideration the peculiarities existing in the curve of pressure in the right ventricle and its derivative [19,29]. The parameters were recorded on the Siemens -- Elema (FRG) apparatus and the recording was made at a paper rate of 50, 100, 250 mm/s.

The minute volume of the heart was determined according to the Fick's direct method, the total peripheral resistance, resistance in the lesser circle of blood circulation, the working index of the right ventricle were all calculated according to the generally used formula.

The volume of circulating blood was determined for 24 hours before catheterization using the albumin of human blood serum, with the I-131 marker.

Recording of biochemical and hematologic indices. The gas composition of the blood was recorded on the Astrup ABC-1 apparatus from the Radiometr Firm (Denmark); the blood samples were calibrated with carbon dioxide in a concentration of 4.8 and 8.2% with further calculation according to the Ziggard-Andersen nomogram at a tempertaure of 37°C. /71

The content of glucose was determined by a glucooxidase method on a glucose analyzer from the Beckman Firm (USA) [22], insulin by a radioimmune method using double antibodies [11], lactic acid -- according to the Barker and Summerson method [12], β-lipoproteides -- by a turbidimetric method [5].

The content of hemoglobin was recorded using the Hemometer apparatus, saturation of the hemoglobin with oxygen -- the Oximeter apparatus, the hematocrit -- by a method of centrifuging blood on a microcentrifuge at 5000 rpm for two minutes (an apparatus from the American Optical Company in the USA).

Mathematical processing and statistical analysis. Mathematical processing of the results was done on a BECM-6 EVM [computer], and the programs were written in the FORTRAN language; the Student t criterion was used for statistical analysis.

Results and Their Discussion

In a state of physiological quiet, antiorthostatic hypokinesia essentially did not affect ($p > 0.05$) the majority of indices of the central blood circulation; an exception was the indices characterizing the inotropic state of the myocardium whose changes were statistically verified ($p < 0.05$; Table 2). Immobilization led to a decrease of

TABLE 2. THE EFFECT OF ANTIORTHOSTATIC HYPOKINESIA ON THE INDICES OF THE CENTRAL BLOOD CIRCULATION AND THE INOTROPIC STATE OF THE CARDIAC MUSCLE (M±m)

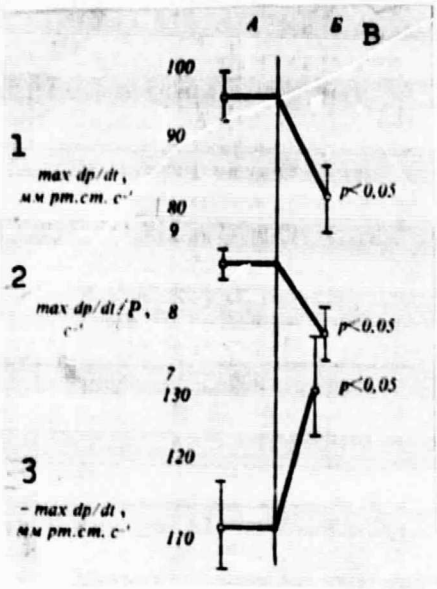
Parameter	Before antiortho-static hypo-kinesia (n=10)	After antiortho-static hypo-kinesia (n=6)	
Pressure in the right ventricle mm Hg:			
systolic	21.0±0.6	19.7±0.6	−1.3
initial	1.5±0.3	2.0±0.8	+0.5
final diastolic	4.9±0.5	5.8±0.8	+0.9
max dp/dt, mm Hg·s^{-1}	97.1±3.6	83.1±4.1*	−14.0
max dp/dt/P, s^{-1}	8.6±0.2	7.7±0.3*	−0.9
- max dp/dt, mm Hg·s^{-1}	110.9±5.6	131.2±7.0*	+20.3
Work of the right cardiac ventricle, kgm/min/m^2	0.38±0.04	0.29±0.04	−0.09
Average pressure in the pulmonary artery, mm Hg	11.8±0.4	10.8±0.6	−1.0
Total pulmonary resistance, dyn·cm·s^{-5}	190.0±11.0	161.5±18.0	−28.5
Average arterial pressure, mm Hg	85.3±2.2	82.3±3.9	−3
Total peripheral resistance, dyn·cm·s^{-5}	1373.0±63.7	1230.0±137.0	−143
Cardiac index, l/min/m^2	4.97±0.16	5.35±0.5	+0.38
Stroke index ml/m^2	73.0±1.8	78.6±7.0	+5.6
Frequency of cardiac contractions per minute	68.0±1.0	68.0±2.0	0
Demand for oxygen, ml/min	295.0±7.8	308.0±17.0	+13
Volume of circulating blood, ml/kg	66.6±7.0	60.1±4.1	−6.5
Volume of circulating plasma, ml/kg	34.6±9.8	32.1±2.0	−2.5
Volume of circulating erythrocytes,- ml/kg	32.0±3.2	28.0±2.7	−4

[Commas in tabulated material are equivalent to decimal points]

* $p < 0.05$.

Notation. Max dp/dt- is the maximum rate of increase of systolic pressure in the right ventricle, max dp/dt/P - the ratio of max dp/dt to DP (the Veragut index), -max dp/dt is the maximum rate of drop of the systolic pressure in the right ventricle.

max dp/dt and the Veragut index at 14 and 10%, respectively. One should note that inasmuch as DP is practically unchanged, its decrease was due to an increase in R - max dp/dt for 0.014s ($p < 0.01$). On a background of a decrease in these indices - max dp/dt, characterizing the rate of weakening during the phase of the isovolumetric diastole, was increased by 18% (see drawing).



The effect of antiorthostatic hypokinesia on the indices of the inotropic state of the right cardiac ventricle.

A -- before antiorthostatic hypokinesia, B -- after it.

Key: 1. max dp/dt, mm Hg·s^{-1}
2. max dp/dt/P, s^{-1}
3. -max dp/dt, mm Hg, s^{-1}

After antiorthostatic hypokinesia, a tendency was observed toward a decrease in arterial blood content of hemoglobin, its saturation with oxygen, the content and pressure of oxygen, a shift toward acidosis of a mixed character which was particularly noticeable in the blood flowing from the heart (Table 3).

In the arterial blood also one noticed a tendency toward a decrease in the content of glucose, insulin, lactic acid; the concentration of β-lipoproteides, on the other hand, had increased (Table 4). After antiorthostatic hypokinesia, the coefficient of utilization by the myocardium of the β-lipoproteides had sharply increased and the coefficient of utilization of lactic acid had decreased.

Thus, the antiorthostatic hypokinesia did not cause significant changes in most of the indices studied; this agrees with the results of other authors [3, 7, 15, 28, 39]. Moreover, the myocardium did not remain indifferent to this effect inasmuch as the indices which characterize its inotropic state were noticeably changed.

According to the modern hypothesis, the inotropic state of the myocardium, along with the load volume and resistance, is the most important determining factor in its activity which reflects the fundamental properties of the cardiac muscle [26, 27,32, 34,35]. In our study, the antiorthostatic hypokinesia was accompanied by a decrease

TABLE 3. THE EFFECT OF ANTIORTHOSTATIC HYPOKINESIA ON OXYGENATION AND THE ACID-ALKALI EQUILIBRIUM OF ARTERIAL (A), MIXED VENOUS (B) BLOOD AND BLOOD FROM THE CORONARY SINUS (CS), M±m

Parameter	Before antiorthostatic hypokinesia			After antiorthostatic hypokinesia		
	A ($n=10$)	B ($n=10$)	CS ($n=4$)	A ($n=6$)	B ($n=6$)	CS ($n=2$)
Hb, %	13,8±0,25	13,6±0,3	13,6±0,6	13,1±0,4	12,8±0,6	13,0±0,7
HbO_2, %	98,0±0,4	81,5±0,6	44,0±1,8	96,1±1,0	80,3±1,7	40,0±3,0
C_{O_2}, vl%	18,1±0,9	14,9±0,3	8,0±0,6	16,9±0,5	13,8±0,6	7,8±0,7
$AB\overline{P}_{O_2}$, vl%	—	3,2±0,1	10,1±0,2	—	3,1±0,17	9,1±0,7
Hct %	46,6±1,6	46,4±1,9	—	45,1±1,1	45,6±1,2	—
KY_{O_2}, %	—	17,7	55,8	—	18,3	53,8
pH	7,36±0,01	7,33±0,02	7,31±0,01	7,32±0,03	7,29±0,03	7,26±0,04
PCO_2, mm Hg	38,3±0,8	42,2±1,4	45,3±1,0	41,0±2,0	44,5±2,8	46,5±2,5
SB	21,5±0,7	21,0±0,6	21,3±0,7	20,8±0,7	19,9±0,7	18,5±0,5
BE, meqv/l	−3,5±0,9	−4,04±0,8	−3,4±0,9	−4,3±0,9	−5,7±0,9	−7,3±0,8
BB	44,1±0,9	43,4±0,7	44,3±0,9	43,4±1,0	42,0±1,0	40,5±1,0
PO_2, mm Hg	97,5±5,0	47,6±1,4	25,0±0,7	90,3±4,5	47,5±2,4	25,5±0,5

Notation. Hb -- hemoglobin, HbO_2 -- saturation of Hb with oxygen, C_{O_2} -- the content of oxygen, $AB\overline{P}_{O_2}$ -- arterial venous difference in oxygen, Hct -- hematocrit, KY_{O_2} -- the coefficient of use of oxygen, PCO_2 -- pressure of carbon dioxide, SB -- standard bicarbonate, BE -- excess of base, BB -- buffer bases, PO_2 -- pressure of oxygen

TABLE 4. THE EFFECT OF ANTIORTHOSTATIC HYPOKINESIA ON CERTAIN BIOCHEMICAL INDICES OF ARTERIAL (A), MIXED VENOUS (B) BLOOD AND BLOOD FROM THE CORONARY SINUS (CS), M±m

Index	Before antiorthostatic hypokinesia			After antiorthostatic hypokinesia		
	A ($n=10$)	B ($n=10$)	KC ($n=4$)	A ($n=6$)	B ($n=6$)	KC ($n=2$)
Glucose, mg	93,3±4,3	93,5±4,0	76,0±2,6	83,7±2,6	85,6±2,0	69,5±5,0
Coefficient of the utilization of glucose, %	—	—	18,5	—	—	17,0
Insulin, μunit/ml	11,5±0,8	11,6±0,9	11,0±1,0	10,1±0,4	10,1±0,2	—
β-lipoproteides, unit	0,49±0,05	0,46±0,04	0,49±0,01	0,54±0,1	0,53±0,08	0,44±0,02
Coefficient of utilization of β-lipoproteides	—	—	0	—	—	18,5
Lactic acid, mg%	10,7±1,0	10,6±1,0	7,0±0,7	7,2±1,7	7,3±1,3	5,5±1,5
Coefficient of utilization of lactic acid	—	—	34,6	—	—	23,6

ORIGINAL PAGE IS OF POOR QUALITY

[Commas in tabulated material are equivalent to decimal points]

in max dp/dt of pressure in the right ventricle of the heart by 10%. Other authors, using a method of apex cardiography detected /72
similar changes in the left ventricle of the heart [33]. Thus, short-term hypokinesia in combination with gravitation redistribution of blood has a negative inotropic effect on the cardiac muscle of a healthy person.

The change in contractility of the myocardium during immobilization is a phase process. For instance, while in the 5-day hypokinesia max dp/dt in a rat is decreased by 47%, by the 30th day this index is close to that obtained in the control and on the 60th day exceeds it by 33% [4, 9]. This compensation, as the authors have correctly noted, to a significant degree is due to a decrease in the functional reserve of the heart which later on can result in cardiac insufficiency. With outward similarity in the changes of max dp/dt in a person and a rat, the factors lying at its basis differ: in the first case this occurs due to an increase of R - max dp/dt, then secondly it is primarily the result of a decrease in DP.

Weakening of the inotropic state of the cardiac muscle which occurs after antiorthostatic hypokinesia can be due, to a lesser degree, to three circumstances: biochemical changes in the cardiac /73
muscle itself, change in the gas and biochemical composition of the arterial blood, adaptation of the heart to a new hemodynamic condition.

On the background of the shift toward acidosis in arterial and mixed venous blood, the tendency toward change of the acid-alkali balance in the blood flowing from the heart was particularly pronounced. This makes it possible to assume that due to the effect of anti-orthostatic hypokinesia in the myocardium, more pronounced metabolic changes take place than in the organism as a whole. It is possible that they occur under the effect of a depressive factor involving motor activity whose existence is postulated by Blomquist and coauthors [15]. However, this indicated hypothesis, based on a few observations, which involve certain difficulties in

probing the coronary sinus, requires further experimental proof.

During immobilization and in weightlessness conditions, in the myocardium, one observes a decrease in the content of catecholamines, ATPase activity of the myosin, the quantity of SH-groups, a breakdown in the ratio between potassium and sodium which can result in weakening of its inotropic state [1,4].

Certain authors have discovered that in rabbits a short-term immobilization can cause not only biochemical but also morphological changes in the myocardium. The latter begins, as a rule, with capillary stasis and then one observes diapedesis hemorrhaging, longitudinal dissociation of the fibers, disappearance of their lateral striations [10]. Obviously, these changes also affect the inotropic state of the cardiac muscle, however, the possibility of their occurrence at such early periods of hypokinesia in a healthy person causes doubt.

A more probable cause for the decrease in the indices of the contractile state of the myocardium is the change in gas and biochemical composition of the arterial blood. It is well known that in conditions of average hypercapnia and a decrease of HCO_3^- the decrease in pH of the blood flowing to the heart causes, as in our study, a decrease of max dp/dt, an arterial venous difference in oxygen and its requirement by the myocardium [24, 32, 37]. It is not excluded that a decrease in the content of lactic acid facilitated this and more truly the coefficient of its utilization inasmuch as lactate being an energy substratum of the cardiac muscle improves its inotropic state and decreases sensitivity to hypoxia [14]. Moreover, the decrease in concentration of insulin which has a direct inotropic effect on the myocardium also could have been responsible for a decrease in the indices of its contractility [31].

Besides these changes, apparently other factors can play a certain role, in particular, the hemodynamic, primarily the decrease of total peripheral and total pulmonary resistance. In this connection, one

should pay attention to the fact that max dp/dt of the right ventricle in a healthy person is considerably lower than in a person with a different cardiovascular pathology. Apparently this is due not only to the method differences in recording and calculating the value of the first derivative but also to nonuniform hemodynamic condition in particular, the value of pressure in the pulmonary artery. For instanc while in the group of our patients max dp/dt was 83-118 mm $Hg \cdot s^{-1}$, in the patients without pathological disturbances in the activity of their right ventricle (a defect of the interauricle membrane and mitral stenosis) -- 223-296 mm $Hg \cdot s^{-1}$, in patients with a chronic distrubance of flow (mitral and aortal regurgitation) -- 309-520 mm $Hg \cdot s^{-1}$, and in patients with a chronic load with pressure (stenosis of the aorta or pulmonary hypertension) -- 382-823 mm Hg X s^{-1}[20]. Moreover, even during a myocardium infarct, the increase in average pressure and resistance in the pulmonary artery leads to an increase of max dp/dt of the right ventricle to 355-380 mm $Hg \cdot s^{-1}$[2]. In other words, /74 load by volume or by resistance, has once more been recently confirmed [19] and leads as a rule to an increase in max dp/dt, whereas load by resistance, obviously, to a large degree affects this index more than load by volume.

Considering from this point of view the results of our study, one can propose that one of the causes for the decrease in contractility of the myocardium could have been a certain decrease in the load on right ventricle by resistance. Actually, after antiorthostatic hypokinesia, one observed a decrease in the total pulmonary resistance by approximately 15%, an even more pronounced decrease in this index, by 24%, occurs in 10 days of bedrest [21].

The inotropic state of the myocardium is determined by changes which occur not only in the systole of the ventricles but in their diastole, more precisely by the ratio of these processes, whose biochemical basis has been studied particularly intensely in recent times. However, the point of view of different authors on the question

of their interrelationship is fairly contradictory: in the opinion of one of the researchers, an increase in max dp/dt must lead to an increase in max dt/dt [6,38], at the same time that other authors have indicated that these same mechanical and chemical stimuli can variously, although in opposite ways, affect the rate of contraction and weakening of the ventricles [13, 23,25, 30]. In our research, the antiorthostatic hypokinesia caused precise reciprocal changes in these indices: a decrease in max dp/dt and an increase in -max dp/dt.

The duration of the diastole pause, as is well known, equals the difference between total duration of the diastole and the time consumed in relaxation[6]. The increase in relaxation rate in the phase of the isometric diastole with unchanged rate of cardiac contractions is evidence of the increase in the diastole pause. When a tendency toward acidosis occurs in the cardiac muscle, decreasing the coefficients of utilization of oxygen and lactic acid, the lengthening of the diastole pause provides, apparently, a more complete restoration of its energetic resources expended in the subsequent contractions.

In conclusion one should note that the change in volume of circulating blood detected occurred due to a more pronounced decrease in the volume of erythrocytes and of plasma; this is reflected in the decrease in the hematocrit. These results differ from data of other authors who, in this period of immobilization detected a decrease in the volume of circulatory blood primarily due to a decrease in the volume of plasma; as a result of this, the hematocrit increased considerably, [21,36]. One of the causes for this difference, undoubtedly, was the condition of our research (see method) inasmuch as among the indices defined before and after antiorthostatic hypokinesia, the volume of circulatory blood decreased during selective probing (the introduction of catheters, taking of blood samples, etc.);

then, if the decrease in volume of plasma was filled, the volume of erythrocytes remained decreased; as a result of this, the index of the hematocrit naturally decreased. Moreover, knowing the value of decrease of volume of circulating blood during operative interference and the study (let us take the maximum -- 300 ml) and the hematocrit, it is possible to approximately calculate that a decrease in the volume of erythrocytes then amounted to about 147 ml; its deficit after antiorthostatic hypokinesia was 288 ml, that is, immobilization per se nevertheless led to a decrease in the volume of erythrocytes by approximately 6%.

Thus, after antiorthostatic hypokinesia on the background of a relative constancy of most recorded indices, the indices of the inotropic state of the myocardium changed noticeably; this could be due to changes in metabolism of the cardiac muscle itself, the gas and biochemical composition of the arterial blood and the hemodynamic conditions.

REFERENCES

1. Gayevskaya, M.S., Veresotskaya, N.A., Kolganova, N.S., et al., Kosmicheskaya biol., 5, 37-40 (1976).

2. Gvatua, N.A., Avdyushko, L.A., Aleksandrova, L.A., et al., Kardiologiya, 12, 53-61 (1976).

3. Kakurin, L.I., Vliyaniye organichennoy myshechnoy deyatel'nosti na organizm cheloveka [The effect of limited muscle activity on the organism of man], Doctoral dissertation, Moscow, 1968.

4. Kuznetsov, V.I., Sokratitel'naya funktsiya i nekotoryye voprosy metabolizma miokarda pri gipokinezii [The contractile function and certain questions of metabolism of the myocardium during hypokinesia], Author's Abstract of a Candidate's Dissertation, Minsk, 1974.

5. Ledvina, M., Labor. delo, 3, 13-17 (1960).

6. Meyerson, F.Z. and V.I. Kapel'ko, Fiziol. zh. SSSR, 58,887-893 (1972).

7. Mikhaylov, V.M., Vliyaniye dlitel'noy gipokinezii na ustoychivost' cheloveka k gravitatsionnoy nagruzke [The effect of prolonged hypokinesia on the resistance of man to gravitational load], Candidate's Dissertation, Moscow, 1969.

8. Parin, V.V., Bayevskiy, R.M., Volkov, Yu.N., et al.,Kosmicheskaya kardiologiya [Space cardiology], Leningrad, 1967.

9. Pruss, G.M. and V.I. Kuznetsov, Kosmicheskaya biol., 6, 45-49 (1974).

10. Tyavokin, V.V., Gipodinamiya i serdechno-sosudistaya patalogiya [Hypodynamia and cardiovascular pathology], Saransk, 1975.

11. Fedotov, V.P. and Ya.A. Sokolov, Sovremennyye voprosy endokrinologii [Modern questions of endocrinology], Moscow, Edition 4, 1974, pp. 186-202.

12. Barker, A. and W. Summerson, J.Biol. Chem. 138, 535-554 (1941).

13. Bass, B.G., Am. J. Physiol. 228, 1708-1716 (1975).

14. Bing, R., Physiol. Rev. 45, 171-213 (1965).

15. Blomquist, C., Mitchell, J. and B. Saltin, Symposium on Hypogravic Hypodynamic Environments. Prodeedings, Indiana, 1970, pp. 197-212.

16. Bos, G.C. van den, Elzinga, G., Westerkof, N. et al., Cardiovasc. Res. 7, 834-848 (1973).

17. Bussmann, W.D., Jooss, B. and M. Kaltenbach, Electromedia (Siemens), 42/5, 148-154 (1974).

18. Cohn, P., Liedtke, J., Serur, J. et al., Cardiovasc. Res. 6, 263-267 (1972).

19. Curtiss, E.,Reddy, P., O'Toole, J. et al., Circulation 53, 997-1004 (1976).

20. Gleason, W. and E. Braunwald, J. clin. Invest. 41, 80-91 (1962).

21. Hyatt, K.H., Kamenetsky, L.G.and W.M. Smith, Aerospace Med. 40, 644-650 (1969).

22. Kadish, A., Litle, R. and J. Sternberg, Clin. Chem. 14, 116-131 (1968).

23. Karliner, J., Le Winter, M., Mahler, F. et al., Circulation 52/3 Suppl. 2, 86 (1975).

24. Ledingham, J.M., McBuge, T., Parratt, J. et al., J. Physiol. (Lond.) 200, 47-48 (1969).

25. Lee, S.J., McClelland, A.R. and G. J. Zarogoza, Acta cardiol. (Brux.) 25, 313-326 (1970).

26. Mason, D.T., Am. J. Cardiol. 23, 516-527 (1969).

27. Mason, D., Braunwald, E., Covell, J. et al., Circulation 24, 47-58 (1961).

28. Melada, G., Goldman, R., Lentscher, J. et al., Aviat. Space Environment Med. 46, 1049-1055 (1975).

29. Oboler, A.A., Keefe, J.F., Gaasch, W. et al., Cardiology 58, 32-44 (1973).

30. Parmley, W. and E. Sonnenblick, Am. J. Physiol. 216, 1084-1091 (1969).

31. Rieker, R., Lee, J. and E.Downing, Yale J. Biol. Med. 48, 353-360 (1975).

32. Sonnenblick, E., Ross, J. and E. Braunwald, Am. J. Cardiol. 22, 328-336 (1968).

33. Spears, W.R., Hyatt, K., Vetter, W. et al., Aerospace med. Ass. Ann. sci. Meet,. 1973, pp. 102-103.

34. Siegel, J.H., Sonnenblick, E.H., Judge, R.D. et al., Cardiologia (Basel) 45, 189-221 (1964).

35. Veragut, U.P. and H.P. Krayenbühl, Ibid 47, 96-112 (1965).

36. Vogt, F.B. and P. C. Johnson, Aerospace Med. 38, 21-25 (1967).

37. Wang, H.H. and R Katz, Circulat. Res. 17, 114-122 (1965).

38. Wiggers, C.J., J. Pharmacol. exp. Ther. 30, 233-243((1927).

39. Zager, P.G., Goldman, R.H., Luetscher, J.A. et al., Aerospace med. Ass. Ann. sci. Meet. (preprint) 1975, pp. 183-184.