Chapter 5

PROLONGED LINEAR AND RADIAL ACCELERATIONS[1]

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Aviation and space medicine specialists have centered their attention for many years on the influence of accelerations on the human body. A vast number of investigations has been made into the nature and degree of manifested reactions by body systems to various acceleration parameters, establishment of tolerance thresholds, determination of basic mechanisms of disorders caused by acceleration, and determination of means and methods to increase body stability to acceleration. A great deal of accumulated material on these problems is found in textbooks [8, 94, 189] and summarized in monographs [73, 94, 175, 195]. The effects of acceleration sometimes reach the limits of physiologic tolerance, and may cause not only significant disruptions in man's work capacity, but also certain pathologic changes.

Increased interest in recent years in the effects of acceleration on the human body resulted from the need to solve pressing problems related to spacecraft flight support. These flights are characterized by specific peculiarities, which are analyzed in this chapter.

The medical problems of acceleration, related to the introduction of new aviation equipment, could form the subject of a special analysis. These problems are touched upon in this chapter only as necessary. Limited space prevents as complete a discussion as might be desirable on all problems concerning the effects of accelerations on the body. Problems relating to long-term (weeks, months, or years) effects of increased gravitation, causing not only physiologic but also pronounced morphologic changes in the body, are not discussed. Results of these important investigations were touched upon in the preceding chapter, *Principles of Gravitational Biology* (Volume II, Part 2, Chapter 4). Finally, data on the effects of alternating accelerations, or combined effects with other flight factors, are not studied. A great deal of material is presented briefly in the form of individual statements without analysis or corresponding references to the literature. However, within the space allotted, as much information as possible is presented, of possible use for specialists involved in space-flight support.

CLASSIFICATION AND TERMINOLOGY

Acceleration occurs when the velocity or direction of motion of a body changes. The magnitude of acceleration, measured in m/s^2 or as a multiple of the ratio of the velocity of a body

[1] Translation of, Vliyaniye na Organizm Dlitel'nykh Lineynykh i Radial'nykh Uskoreniy, Volume 2, Part 2, Chapter 2 of, *Osnovy Kosmicheskiy Biologii i Meditsiny (Foundations of Space Biology and Medicine)*. Moscow, Academy of Sciences USSR, 1973.

Sincere appreciation is expressed to E. M. Roth, E. G. Teichner, and R. L. Craig for their objective review of the work on this chapter.

falling freely in airless space (9.81 m/s²), is determined to a great extent by the force acting upon the body and its mass. This is represented by the letter g (the first letter in the Latin word *gravitas*—i.e., gravity). An acceleration of 35 m/s², for example, can be rounded off and expressed as 3.5 g.

This chapter is concerned only with linear and radial accelerations. Linear accelerations occur when the rate of motion of a body is increased or decreased without a change in its direction. Radial or centripetal accelerations occur when the direction of motion of a body is changed—one of the clearest examples is the acceleration noted in a centrifuge or when an aircraft executes a turn during a dive. Linear and radial accelerations, depending on the time of application, are arbitrarily divided into impact acceleration (up to tenths of a second) and long-term acceleration, which is further classified [72, 73, 178].

The direction of the inertial force is always opposite to the direction of acceleration. The term G-load (inertial force) is frequently used in medicine and biology. The G-loads have no dimensions and are expressed in relative units, which essentially show the number of times the weight of a body has been increased by a given acceleration in comparison to ordinary terrestrial gravitation, i.e., it is the ratio of its dynamic weight to its static weight at rest or during linear motion [95].

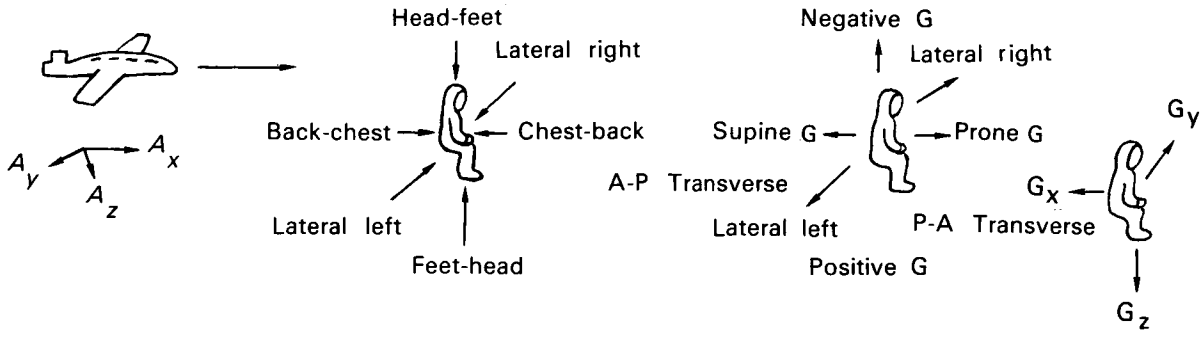
A distinction is made between longitudinal and transverse G-loads, depending on the direction of action of G-loads in relation to the vertical axis of the body. If the G-load vector is directed from head to feet, it is called a positive G-load; if it is from feet to head, it is called a negative G-load. Furthermore, a distinction is made between transverse (back-chest and chest-back) and lateral (side-side) G-loads. The direction of the G-load vector is significant for determining the nature of the body's response reactions. Unfortunately, there is still no unified terminology and classification of accelerations, which frequently leads to erroneous interpretations of identical facts and causes difficulties in understanding certain material in the literature.

The table of terminological equivalents accepted by representatives of a number of countries with membership in the Aerospace Committee on Problems of Acceleration [162] is worthy of attention. Although it does not cover all aspects of this important problem, the table can still be useful in the practical activity of researchers.

Only the relevant portion of a more complete table is presented in Table 1, data which relate directly to the problems under discussion here. Following recommendations of the Aerospace Committee, all terminology is divided into two main groups: columns A and B. Column A, which presents two varieties of terminology on accelerations most frequently used in astronautics, is based on the direction of acceleration of a mass. In column B, the terms are based on the inertia of organs, tissues, and fluids in an undamaged body. In the system of coordinates used, the z-axis passes through the body's center of gravity parallel to the spine. The action of an inertial force along the z-axis in the direction downward, from head to pelvis, is represented by the symbol $+G_z$ (physiologically positive acceleration or positive G-load); the action of these forces in the opposite direction is represented by $-G_z$ (physiologically negative acceleration or negative G-load); the symbol $+G_x$ indicates that the force of gravity is directed transversely from chest to back (chest-back G-load), while the symbol $-G_x$ represents a G-load in the back-chest direction. Lateral transverse accelerations are represented as $+G_y$ (right to left) and $-G_y$ (left to right).

The center of gravity of a flight vehicle and the direction of axes in the system of coordinates do not coincide with the center of gravity and the direction of the corresponding axes of the pilot's body, relative to which vectors of inertial effects on the body are calculated. Thus, in evaluating technical acceleration graphs of a flight vehicle from the standpoint of man's ability to tolerate acceleration, the peculiarities of the physiologic terminology as concerns inertial effects on the body must be kept in mind. However, in this table a number of important factors related to the effects of acceleration on the body are not considered (time of action, rise gradient, and so forth), which require further refinement of terminology and classification [178].

TABLE 1.—*Acceleration Terminology* [77]



Linear motion	A Direction of acceleration		B Inertial resultant g-load on body		
	Aviation symbols (System I)	Descriptive terms of acceleration (System II)	Physiological terms (System III)[1]	Physiological symbols (System IV)	Local descriptive terminology
Forward	$+a_x$	Back-chest acceleration	Transverse A-P, G	$+G_x$	Eyeballs in
Backward	$-a_x$	Chest-back acceleration	Transverse P-A, G	$-G_x$	Eyeballs out
Upward	$-a_z$	Foot-head acceleration	Pronel, back-chest Positive G	$+G_z$	Eyeballs down
Downward	$+a_z$	Head-foot acceleration	Negative G	$-G_z$	Eyeballs up
Right	$+a_y$	Acceleration to right	Lateral left G	$+G_y$	Eyeballs left
Left	$-a_y$	Acceleration to left	Lateral right G	$-G_y$	Eyeballs right

[1]In system III, the letter G is used as a unit to express the inertial resultant acceleration of the entire body, a multiple of the value of acceleration resulting from the force of gravity $g_0 = 980.665$ cm/s². A-P = anterior-posterior; P-A = posterior-anterior.

OVERALL EFFECTS ON THE BODY

Man's reaction to acceleration is determined by a number of important factors, such as the amount of acceleration, time of its action, rise rate and direction of the G-load vector in relationship to the trunk, as well as the body's initial functional state, which depends on many external and internal medium conditions [72, 228].

Body changes may result from barely detectable functional shifts to extremely severe states, and be accompanied by acute disorders in the respiratory, cardiovascular, nervous, and other systems. These disruptions may result not only in loss of consciousness, but sometimes also in pronounced morphologic changes. An individual subjected to accelerations experiences a feeling of heaviness in the entire body, pains in chest or stomach, initial difficulty in moving (and subsequent complete inability to move), particularly the extremities. The soft tissues and a number of internal organs are displaced in the direction of the inertial force's application. Depending on the density (specific gravity) of the internal organs, their location, and elasticity of their connections with the surrounding tissues, the nature of the disruptions may differ. Since the most mobile tissues in the body are blood and tissue fluids, hemodynamic disruptions have a leading position in the genesis of physiologic shifts under G-loads.

Displacement of internal organs and their deformation are also significant, which result not only in functional changes in these organs, but also in unusual afferentation to the central nervous system (CNS), frequently disrupting its regulating and corrective functions.

Under great accelerations, disorders of vision are observed. The nature and degree of manifestation of these disorders are determined not only by the amount of acceleration, but also by the direction of its application in relationship to the body's main vessels. With further increases in the magnitude or time of applying acceleration, loss of consciousness, convulsions, and death may occur; but if acceleration is interrupted, there is gradual normalization of all functions.

MAN'S RESISTANCE TO ACCELERATIONS AND EVALUATION OF CRITERIA

Body resistance to acceleration effects depends on the nature of the tolerance criterion selected. Therefore, the boundaries of survivability and limits of physiologic resistance are distinguished and evaluated on the basis of initial signs of disruptions in activity of various functional systems. Boundaries of survivability are determined only in animal experiments. As applicable to man, subjective and objective symptoms are used to estimate limits of resistance. The upper threshold of subjective resistance is the limit of the subject's ability to withstand the physical and emotional discomfort associated with acceleration effects. Vision disruption, pain, fatigue, and severe difficulty in breathing are most frequently encountered. Investigation of the limit of resistance is quite difficult, however, when the criterion of tolerance used must be subjective sensations.

To determine tolerance to $+G_z$ acceleratory stress, estimations should be based on grayout or blackout, reliable precursors of fainting. Headache and lacrimation are equivalent precursors for $-G_z$ stress. The redout visual disorders observed by certain pilots during flights also serve as precursors with $-G_z$ stress. However, neither Soviet nor US researchers have succeeded in observing redout symptoms during many years of centrifuge studies.

The limiting symptoms for transverse accelerations may be visual disorders, dyspnea, discomfort, and pain, which practically cannot be given a standard evaluation. Even a significant criterion such as grayout is variable both for different people and for the same person on different days. Multiple petechial hemorrhages resulting from increased permeability of various etiologic vessels may interrupt acceleration studies. Cessation of acceleration application would be required by such important indicators as disorders of cardiac activity and respiration, disruption of the subject's ability to control, and presyncopal signs.

Critical states of the body (loss of consciousness, collapse, sudden changes in cardiac activity, hemorrhaging in organs, pulmonary atelectasis, and so forth) may arise in certain situations during acceleration and, in this sense, characterize man's survival ability. Objective criteria of man's acceleration tolerance most frequently used are the indicators related to visual disorders: nonreaction to light signals [39, 90, 91, 125, 126, 211], disrupted oculomotor mobility preceding blackout, loss of ability to follow a given object [28], and others.

Criteria of acceleration tolerance should be differentiated according to basic (or leading), supplementary, and prognostic. For example, under $+G_z$ stress, the basic or leading resistance tolerance criteria are visual disorders in the form of grayout or blackout, and nonreaction to light signals, which indicate almost total loss of work capacity and consciousness. A reduction in systolic pressure in the vessels of the concha auriculae to 40–50 mm Hg and disappearance or sharp reduction in oscillations of the ear pulse precede loss of vision in a great majority. Thus, these indicators can be included among prognostic criteria [210].

For $+G_x$ acceleratory stress, when the gravitoinertial component along the head-pelvis axis is equal to 40% or more of the total stress, visual disorders are also basic criteria for evaluation of resistance. If the $+G_z$ inertial component is slight (10–20%), the basic and most reliable criterion of the tolerance limit of $+G_x$ stress is cardiac activity disorder in the form of relative bradycardia, combined with other

cardiac-rhythm disorders, loss of vision, and dyspnea [113].

The criteria or symptom complex of signs, on the basis of which resistance to accelerations is estimated, may be formally identical for a given position, but at the same time may have different significance for the body. For example, under $+G_x$ stress with the test subject in a position where the angle of inclination of his seat back is 25° and 10° from the horizontal, manifest visual disorders may appear, up to complete loss of vision. In the first case, visual disorders precede loss of consciousness, while in the second case, the subject can tolerate 50% more $+G_x$ stress, while continuing to perform certain operations with information input through the auditory channel [16]. Consequently, the same symptom visual disorders, in the first case indicates danger to life, while in the second case, only work capacity is threatened. Therefore, in studies of man's resistance to transverse acceleration effects, evaluating the vital functions state, rather than work capacity, is important. This criterion can be basic and dominant in some cases, while in others it may be supplementary or prognostic, and vice versa.

Man's resistance to acceleration effects is determined by a number of physical and physiological factors. Among the significant physical factors are the magnitude of acceleration, duration of application, direction of the acceleration's resultant vector in relation to the longitudinal axis of the body, acceleration rise gradient, mode of application ("plateau" or "peak"), use of protective systems and body restraints, position of the body and extremities, and environmental conditions (temperature, ambient pressure, and others). The most important physiologic factor is individual resistance, which is dependent on health, age, training, psychologic preparation, and motivation.

Permissible values and acceleration application time are determined on the basis of physiologic tolerability and the influence of acceleration on man's ability to control his flight vehicle. The physiologic limits of endurance and limits of work capacity may be interrelated, but not necessarily equal. As a rule, work capacity deteriorates before resistance limits are reached. Extensive studies have allowed precise determination of man's resistance limits to acceleration effects in various directions, depending on magnitude of acceleration and duration of application (Fig. 1).

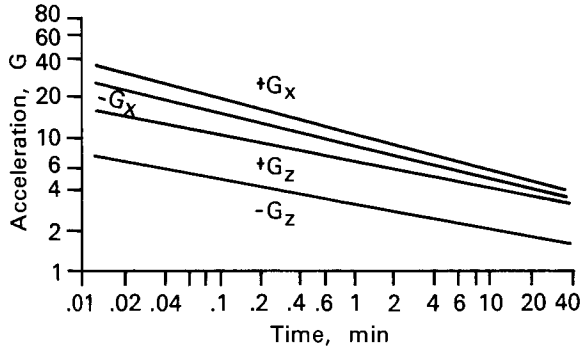


FIGURE 1.—Man's resistance to the effects of acceleration in various directions [45]. Mean data on $+G_z$ (head to pelvis), $-G_z$ (pelvis to head), $+G_x$ (chest to back), $-G_x$ (back to chest) (direction of inertial forces).

Man is least resistant to the effects of $-G_z$ stress where inertial forces are directed along the axis from pelvis to head, and most resistant to $+G_x$ stress where inertial forces are directed from chest to back.

The physiologic tolerance of accelerations is limited primarily by body reactions: redistribution of blood, mechanical hindrance of respiration, and displacement and deformation of internal organs. The more closely the direction of acceleration components coincides with the direction of the body's main blood vessels passing along the spine, the stronger the disruptions of general hemodynamics. Thus, when accelerations are applied along the body's longitudinal axis, the redistribution of blood leads rather quickly to signs of disruption in cerebral circulation, which limits the duration of application.

General hemodynamic changes are significantly less with transverse accelerations. This factor essentially determines the position of the astronaut when subjected to accelerations during spacecraft launch and descent. It has been established from studies that the $+G_x$ direction, when inertial forces act from chest to back, must be considered optimal for acceleration tolerance.

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Endurance Limits

The endurance limit is low for a strictly transverse acceleration direction. Chest pains and dyspnea have prevented reaching high values of acceleration. When $+G_x$ stress is applied while resting flat on the back, the limit of resistance, determined by pain and dyspnea, is at the level of $+8$ G_x [13]. Elevation of the head and thorax yields a positive effect—resistance increases, and pain and dyspnea decrease. However, placing the head and trunk at an angle results in application of the inertial force in the direction of the longitudinal axis of the body and the appearance of related symptoms of loss of vision and consciousness. Consequently, an optimal body position had to be found to provide an intelligent compromise between man's physical capabilities and technically efficient acceleration modes.

In the search for the optimal position for space flight, there was diligent study of man's resistance, in various body positions, to the effects of transverse accelerations [13, 31]. Resistance to $+G_x$ stress proved best with the seat back inclined at an angle of 65°–70° to the acceleration vector (or 20°–25° to the horizontal), and with knees at eye level and legs held in a supporting chair [31]. With the extremities straight, resistance was limited by severe knee pain. However, in this position the primary disruptions in the body's physiologic systems, which limit man's resistance to acceleration, were quite similar to disorders accompanying longitudinal accelerations ($+G_z$ stress), due to the high value of the longitudinal component along the $+G_z$ axis. The limits of endurance in this case generally were not over $+12$ G_x.

In order to decrease the influence of the component along the head-pelvis axis, seats were tested with slight back elevation angles of 2°–12° [17, 48, 55, 117]. Under these conditions, it was possible to reach high values of acceleration with rapid rise rates. Accelerations of 20.0 g could be reached while spinning in a seat with flat supporting surfaces, and 25.0 g accelerations in a form-fitting seat (acceleration rise rate 1.0–1.2 g/s) [48, 50]. Consequently, positioning the body more horizontally in the seat increased resistance limits. This position is now considered optimal by most researchers [17, 91, 92, 117, 228].

When considering the effects of G-loads in the $+G_x$ direction, it is convenient to use the concept of the effective physiologic angle (Fig. 2). This angle is the sum of three angles: the seat angle (SA), angle between the vertical line through the spacecraft and the resulting forces of the accelerated apparatus (ϵ), and aorta-retina angle (ARA). The sum of the first two angles forms the angle of the seat back inclination to the acceleration vector. The retina-aorta angle is determined by lateral roentgenography of the thorax and is enclosed between the lines from the center of the heart to the retina and the longitudinal anatomical axis of the body ($+G_z$ axis).

This angle averages 15°, fluctuating from 13° to 18°. The aorta-retina angle is highly significant, since it changes the direction and magnitude of the acceleration component resulting in visual disorders (the so-called effective retina-aorta component of $+G_z$). For this reason, the value of

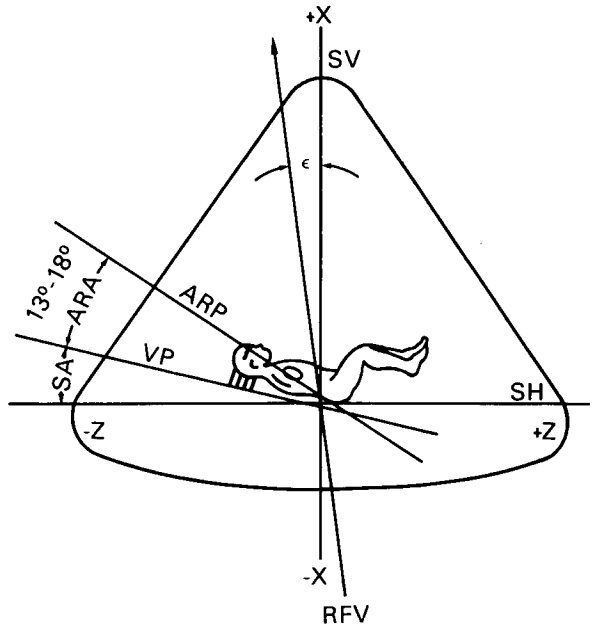

FIGURE 2.—Position of astronaut and terminology suggested by NASA Manned Spaceflight Center [5]. ARA: aortal-retinal-angle; ARP: aortal-retinal plane; ϵ: angle between resulting acceleration vector and vertical line of spacecraft; RFV: resultant vector of acceleration; SH: ship's horizontal; SV: ship's vertical; VP: plane of spinal column; SA: seat back angle; EPA: effective physiologic angle.

the effective physiologic angle allows predicting the resistance limit determined by visual disorders as a function of the inclination angle of the seat back (Fig. 3). The effective retina-aorta component of $+G_z$ varies little during change in a seat back angle of 60°–90° from horizontal. With a 0° angle for the seat back, visual disorders may arise because of an effective $+G_z$ retina-aorta component. Thus, data indicate that the best compromise for tolerance of $+G_x$ stress is a horizontal seat position with angle $SA+\epsilon=8°–12°$, with the astronaut's hips bent so that knees are elevated to eye level. In the Apollo spacecraft, the angle of the seat back ($SA=2°$ and $\epsilon=6.5°$) is 8.5°.

Man's resistance to $+G_x$ stress as a function of acceleration value and position is presented in Figure 4. Tolerance to peak accelerations, as applicable to actual flight conditions, has been fully studied. The profiles of maximum tolerable $+G_x$ stresses of various modes are presented in Figures 5 and 6. The dashed line in Figure 5 encloses the set of possible acceleration profiles related to space flight, all of which, according to experimental studies, are tolerable [28, 46, 50, 51, 55, 133]. The acceleration rise rate is highly significant. With regard to the effects of longitudinal $+G_z$ stresses, it has been established that body tolerance for accelerations generally decreases with increasing rise rate [25]. A nomogram, based on experimental material of Stoll [206], shows the expected time grayout appears for a given $+G_z$ accelerator stress plateau and rise rate (Fig. 7).

Under transverse $+G_x$ stresses, on the other hand, slow rise rates decrease the value and time of body resistance [31]. Opposite effects of acceleration rise rate in these two directions ($+G_z$ and $+G_x$) can probably be explained by the significance of changes in general hemodynamics due to head-pelvis inertial force. In the first case ($+G_z$), it is primary, and in the latter case ($+G_x$), it is secondary. A slow increase in accelerations allows the body to develop and utilize its compensatory reactions more completely. Thus, when $+G_z$ stresses are applied, this slow increase helps to prevent or postpone critical situations, primarily related to disruption of cerebral circulation. When $+G_x$ stresses are applied, the threat that such situations might develop is significantly less, while the increase in rise rate of accelerations allows a gain in value and application time without danger of loss of consciousness.

The effectiveness of using different $+G_x$ rise rates with the optimal position in a contoured seat is shown in Figure 8: an increase in the rise rate of accelerations allows attainment of higher values—at 0.1 g/s, +12.0 G_x; at 0.2 g/s, +14.0 G_x; and at 1.0 g/s, +26 G_x. The total duration of

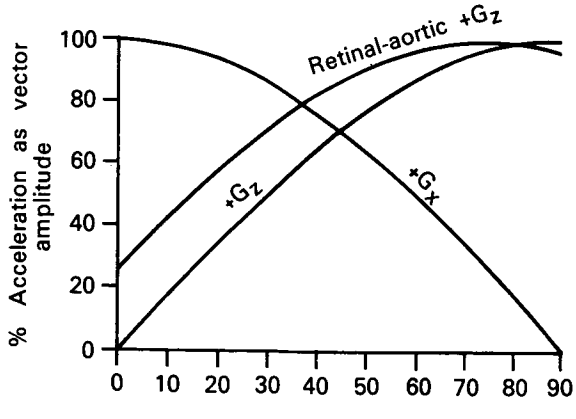

FIGURE 3.—Values of $+G_z$ and $+G_x$ acceleration vectors in percent for any value of seat back angle [91]. Abscissa shows angle of seat back in degrees from horizontal.

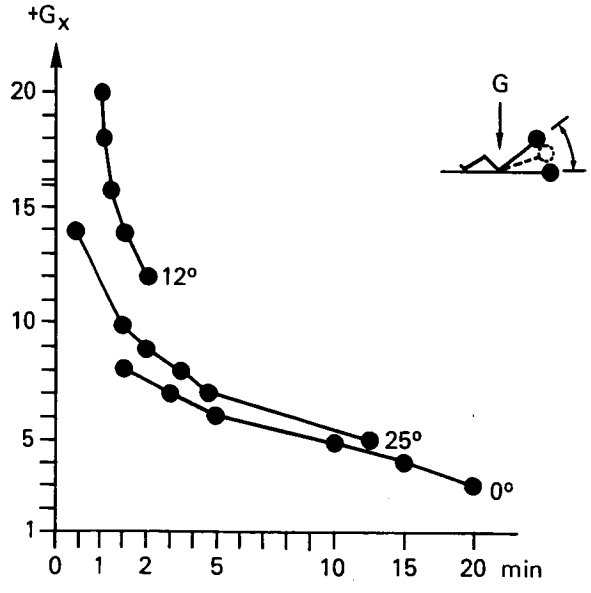

FIGURE 4.—Tolerance time of man to transverse $+G_x$ acceleration as a function of seat back angle [17, 113, 117].

application of transverse accelerations is not less important than their value. The maximum tolerable value of accelerations increase with increasing acceleration rise rate from 0.1 to 1.0. g/s because of reduction in application time of high-value accelerations and absolute decrease in application time of accelerations in general.

Man's resistance to the effects of $-G_x$ stresses, where inertial forces are directed from back to chest, has been studied less; published data on this problem are limited [31, 49, 50, 73]. The most difficult problem when accelerations are applied in this direction is providing body restraint and optimal conditions for interaction with support. The body position and restraint system determine primarily man's resistance to these accelerations (Fig. 9).

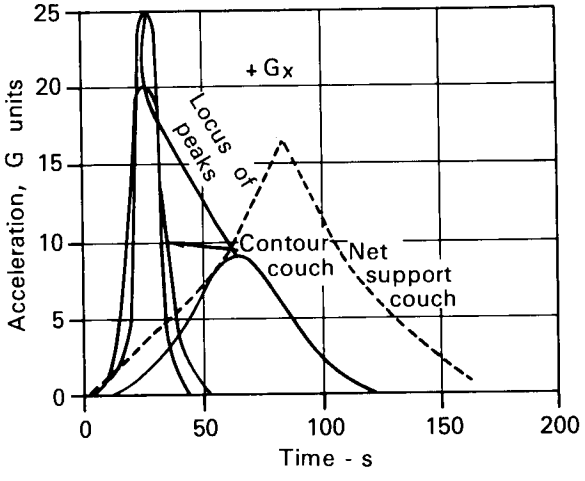

FIGURE 5.—Maximum tolerable acceleration profiles [46].

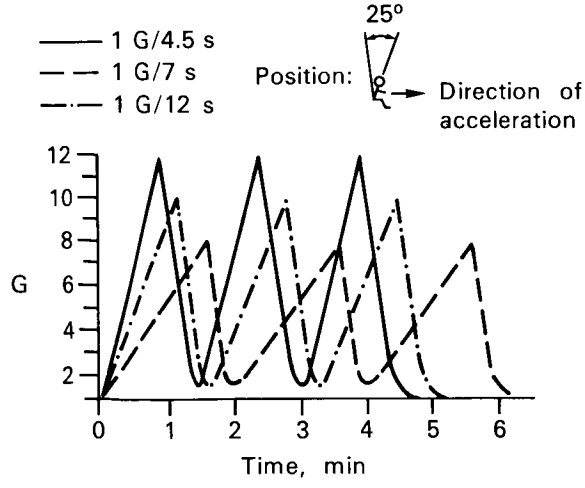

FIGURE 6.—Profiles of accelerations tolerable by man for a three-stage rocket, sufficient to achieve orbital velocity [168].

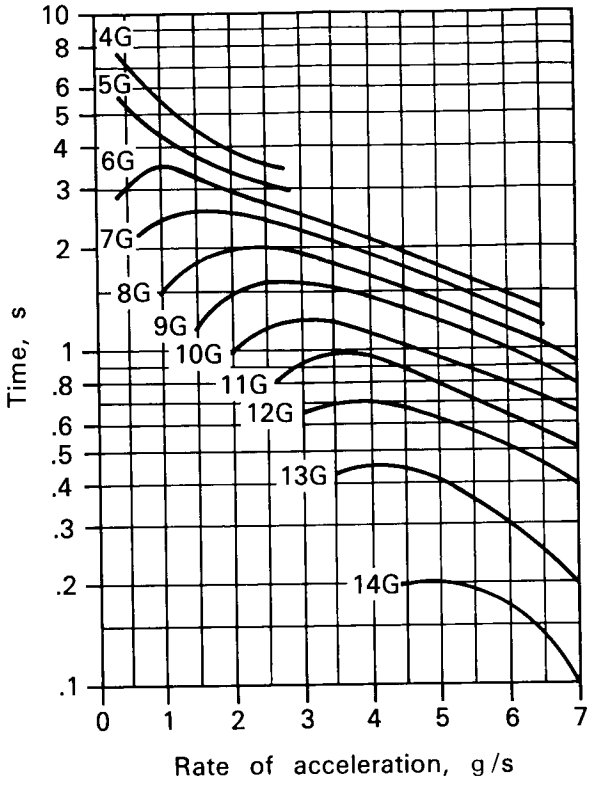

FIGURE 7.—Nomogram of dependence between acceleration rise rate and time of grayout appearance [206].

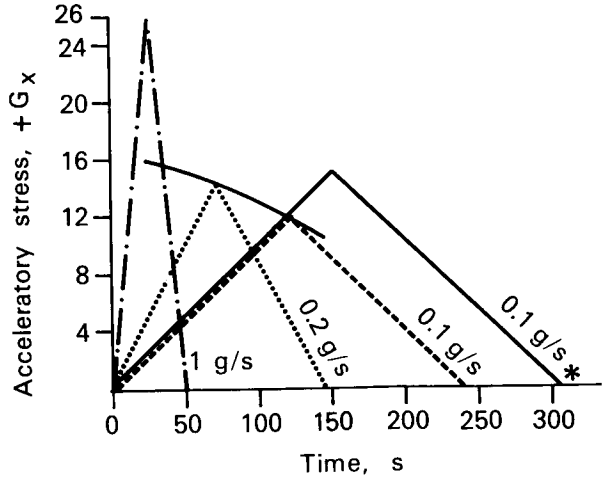

FIGURE 8.—Man's resistance to effects of transverse $+G_x$ peak-type acceleratory stress with various rise rates [17, 113]. Transverse line corresponds to values of acceleration at which visual disorder arises; *—after centrifuge training.

Acceleration Tolerances

Respiratory difficulties encountered with $-G_x$ acceleratory stresses are less than with $+G_x$. However, since pressure is directed forward onto the restraint system, pain and discomfort arise with accelerations producing -8 G_x. The position of the head has bearing on $-G_x$ stresses; if the head is tilted forward, the hydrostatic effect influences brain activity, causing tolerance to $-G_x$ to be still less. Another significant peculiarity of these accelerations is vision disruption (fogging, loss of visual acuity), probably resulting from abundant lacrimation. Extensive data in the literature indicate distinct individual variations in the level of man's resistance to acceleration.

The individual resistance to acceleration is determined by the initial functional state, age, health, degree and nature of the muscular system's development, and state of training for G-loads. When $+G_z$ stresses are applied, the lowest resistance but highest lability of physiologic reactions is observed in test subjects 20–24 years of age (5.6 ± 0.3 G_z), while tolerance to $+G_z$ stress increases 0.5 G by age 30–39 (6.1 ± 0.1 G_z), then decreases again (by 0.4 G) between 40 and 49 years of age (5.7 ± 0.1 G_z). Acceleration tolerance becomes more stable with increasing age, with a smaller range of variation. Apparently, lower acceleration resistance at 20–24 years of age compared with other age groups can be explained by the well-known lability of the nervous-humoral mechanisms of regulation observed at this age. On the other hand, reduction in acceleration resistance at 40–49 years of age (in Suvorov's opinion) results to some extent from the body's aging and accompanying variations inherent at this age. For the effects of $+G_x$ acceleratory stress, dependence on age of toleration to $+G_x$ has not been clarified.

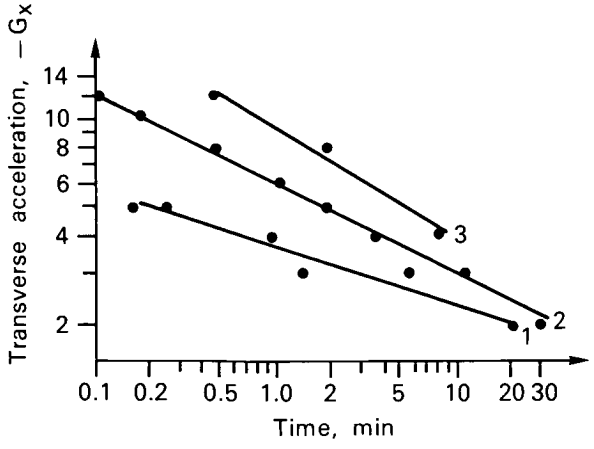

FIGURE 9.—Man's resistance to transverse $-G_x$ acceleratory stresses [73]. 1: "curled" position with fixation of body and head; 2: "curled" position in suit with body, head, and extremities restrained; 3: "supine" position on net support.

The effectiveness of development of the body's adaptive reactions to acceleration effects is also determined by the degree of training. Acceleration tolerance for $+G_z$ averages 0.5 G higher for pilots than for other professionals. Variations in the general state of health are also significant. The least tolerance to $+G_z$ stress was detected where there were indications of vascular-autonomic dystonia [210, 212]. The primary reason for such reduced tolerance is disruption of vascular tonus regulation. In contrast, with first-stage hypertension, there is higher resistance to $+G_z$ stress shown than with vascular-autonomic instability. This results from the corresponding increase in vascular tonus, which provides favorable conditions for maintaining arterial pressure at a high level.

The muscular system is relevant to development of the body's compensatory reactions [11, 189]. When muscular tension is utilized, the threshold of blackout in pilots under longitudinal acceleration is increased by 1.0–2.0 G_z [237]. Tension of abdominal muscles, lower extremities, and shoulder girdle increases body resistance to accelerations by an average of 1.5–1.8 G_z. This effect is apparently related not only to the more favorable type of circulating blood redistribution during the acceleration period, but also reflects influences on cardiovascular system activity [34, 35, 165, 186, 189, 210].

Composition of the ambient gas medium can change man's acceleration resistance. Hypoxia and an elevated oxygen content influence man's endurance under longitudinal acceleration [39, 72, 73, 85, 144]. Degrees of hypoxia which can be compensated by the organism (up to 3–4 km altitude) have no significant influence on the tolerance level for accelerations [63, 73]. Greater

hypoxia reduces acceleration resistance [25, 123]. A deterioration in tolerance to $+G_x$ stress was shown in a study of the summary effects of hypoxia and acceleration [18].

CO_2 content in the gas medium, in the breathing air inhaled, is also significant. An increase in tolerance to longitudinal accelerations of 0.5 G_z was shown when air breathed contained 4–6% CO_2 [147, 177]. Increased resistance to $+G_x$ stress was noted following 10 min respiration of air with 3% CO_2, while prolonged 4-h breathing of this mixture caused deterioration in acceleration tolerance [223]. The sparse data available on the gas medium's influence on man's tolerance to acceleration requires further development, which is important, since gas medium composition in sealed spacecraft cabins might change during emergencies.

The temperature factor has only been studied relative to effects of $+G_z$ stress. Exposure of man to high ambient temperatures (+55°–70° C) reduced tolerance to $+G_z$ by an average of 0.5–1.0 G [40, 87, 146]. Simultaneous exposure to higher temperatures (40° C) and acceleratory stress (+3 G_z for 1 min) led to deterioration in human psychomotor activity [173], which was evaluated on the basis of reaction time to light signals, and numbers of errors. The severe water loss and blood redistribution resulting from surface vessel expansion obviously may influence the circulating blood volume and thus deteriorate the state of the body's cardiovascular system [171].

A special study was made of the influence of dehydration (up to 3.6% body weight loss) on man's tolerance to $+G_z$ stress [87]. It was established that hypohydration led to a 15–20% reduction in the tolerance time to $+G_z$. However, no correlation was noted between the percent loss of body weight, total blood volume, plasma volume, and time of acceleration tolerance.

The influence of reduced temperature on human acceleration tolerance has been studied [146]. With blood temperature of 25° C in the area of the foot, a slight, unreliable increase in resistance to acceleration of +0.4 G_z was noted in comparison to comfortable conditions.

Under spaceflight conditions, accelerations may act on the human body in combination with other flight factors – changes in gas environment, temperature, radiation, and so forth. The influence of these factors may be brief or constant throughout the flight, the end result depending on time and force relationships.

Weightlessness occupies a special position among spaceflight factors. The astronauts' tolerance for accelerations, upon spacecraft reentry into the Earth's atmosphere following long periods of weightlessness, is particularly important in planning long space flights. On the one hand, body asthenia is possible by the end of the flight, while on the other hand, accelerations which might be great are generated upon reentry into the atmosphere's dense layers (Fig. 10). The success of the entire expedition depends on the astronaut's tolerance for accelerations during the flight's final stage.

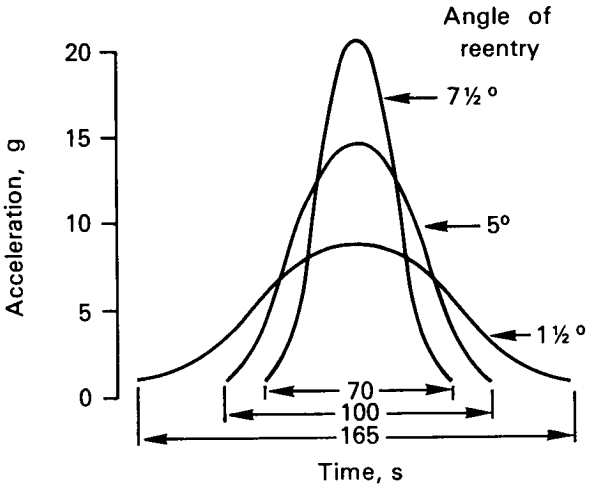

FIGURE 10. – Values of acceleration as a function of spacecraft reentry angle into dense layers of the atmosphere during descent [48].

A reduction in gravitational influence (extended time spent in the horizontal position) and mobility limitations reduce man's resistance to orthostatic effects, up to the development of precollaptoid and collaptoid states. Studies concerned with acceleration effects after weightlessness are limited. Deterioration in man's resistance to effects of $+G_z$ stress was noted after many hours in water [23, 28, 83, 84]; reduction in resistance was 0.50–0.62 $+G_z$. In studies of $+G_x$ accelera-

tory stresses of peak type with a maximum of 8.0 G, reduction in acceleration tolerance was also noted (unreliable), which was evaluated by a compensatory tracking test [23]. Acceleration tolerance in the position used in the Gemini spacecraft was investigated [23]: following 28 d strict bed rest, 22 test subjects were subjected to $+G_x$ stresses reaching a peak of 10.6 G_x, according to the planned reentry schedule of Gemini spacecraft. No reliable difference in the level of individual resistance was found, which was evaluated on the basis of disruptions in central vision. However, an increase in stress on the physiologic systems was observed, resulting from acceleration as compared to initial data (pulse rate increased by 35 ± 20 beats/min).

Acceleration tolerance was practically unchanged following 3 d of hypokinesia (simulating weightlessness) [113, 116]. Increasing the hypokinesia time caused reduction in resistance to $+G_x$ stress. Visual disruptions occurred at lower values of acceleration than usual in these tests (at $+11.6 \pm 0.45$ G_x and 13.6 ± 0.35 G_x respectively) and became primary [113].

Aftereffects

Subjectively, acceleration effects following weightlessness simulation were directly more severe in all test subjects. Muscular weakness with static stress was noted, also more difficult respiration, and earlier development of visual disorders, including total loss of vision. Following hypokinesia, attaining equivalent acceleration values was accompanied by higher functional stress on the physiologic body systems than in initial tests. Cardiovascular system reactions were: an increase in tachycardia and subsequently, earlier development of bradycardia. Progressive reduction of cardiac activity began, following hypokinesia, at 2.8 G_x lower than during usual living conditions (at $+11.2 \pm 1.05$ G_x, respectively).

An extended, multiphase process of arterial pressure normalization was the predominant characteristic during aftereffect: there were periods of reduced pulse pressure, primarily due to increased diastolic pressure. Some test subjects experienced pain around the heart during aftereffect and required medical aid [113] and spasmolytic preparations. Disruption of vascular tonus regulation under such conditions could be decisive. The practical significance should be emphasized of data obtained from prediction of astronauts' states after landing, and determination of the amount of medical aid necessary.

Prolongation of space flights makes it extremely important to determine the dependence between changes in man's resistance to acceleration and the duration of weightlessness. No definite correlation between degree of resistance reduction to $+G_x$ stress and duration of simulated weightlessness has been observed [113, 114]. Following 7–20 d of hypokinesia, tolerance to $+G_x$ was reduced by an average of 2.2 G_x. An increase in the time of simulated weightlessness to 100 d caused no further reduction in resistance limit to acceleration (Fig. 11, Table 2) which was also confirmed by an analysis of human physiologic reactions. During bed rest, adaptation to hypokinetic conditions and the degree of body asthenia, as evaluated by tolerance to $+G_x$ stress, does not progress with prolonged bed rest. Two phases of change in the body's reactivity to accelerations must be distinguished. In the first stage, there is a reduction in resistance to accelerations. The second phase is that of relative stabilization, when tolerance to accelerations, although low compared to its initial level, does not progress as the time of hypokinesia increases, but remains at the level corresponding to 7–20 d of hypokinesia.

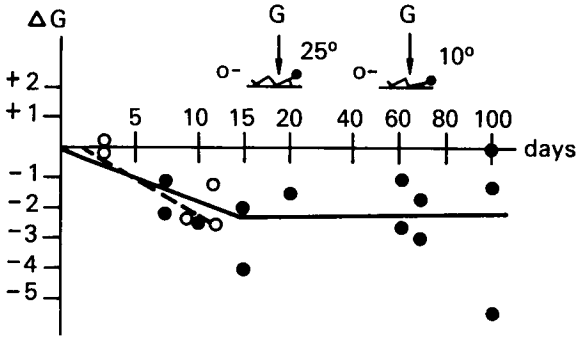

FIGURE 11.—Man's tolerance to transverse $+G_x$ acceleration following simulated weightlessness of various durations [113]. Solid line: seat back angle 10°; dashed line: 25° from horizontal.

Adverse aftereffects under simulated weightlessness can be reduced by physical training and use of pharmacologic agents, which increase subsequent tolerance to $+G_x$ stress. A combination of these protective measures proved the most effective, rather than using one measure alone (Table 3).

Man's tolerance limit to accelerations, following simulated weightlessness, using these protective measures remains the same as in control studies. However, this tolerance for G-loads was achieved at the cost of great stress on the body's physiologic systems. The greatest changes did not involve the cardiovascular system, indicating that preventive measures did not completely eliminate deconditioning.

As changes develop in man's tolerance to transverse $+G_x$ forces following weightlessness, the hypodynamic and hydrostatic factors are of primary importance. Decrease in muscle mass, strength, and endurance; cardiovascular system deconditioning; adjustment of neurohumoral regulation of the physiologic functions; and other changes must, in the final analysis, be considered causes of distinct reduction in resistance to transverse acceleration effects.

In actual space flight, vestibular-autonomic disorders, diseases, fatigue, and other unforeseen situations and factors may arise, which may also influence man's resistance to accelerations during the final stage of return to Earth.

TABLE 2.—*Change in Man's Resistance to Effects of Transverse $+G_x$ Accelerations Following Simulated Weightlessness of Various Durations* [113]

Time of weightlessness, d	Maximum value of $+G_x$ acceleration (M + m)		Mean difference ΔG	Extreme value of $+G_x$ following weightlessness	Overall estimate of change in resistance
	Before	After			
7–20	13.8 + 0.40	11.6 + 0.45	−2.2 ± 0.4[2]	9.8 − 12.9	Reduction
60–70	12.6 ± 0.45	10.5 ± 0.65[1]	−2.1 ± 0.4[2]	9.3 − 14.6	Reduction
100	15.2 ± 0.60	13.0 ± 1.1	−2.2 ± 1.6	10.9 − 14.2	Reduction
Average	13.8 ± 0.40	11.6 ± 0.45[2]	−2.2 ± 0.4[2]	—	Reduction

Peak acceleration with rise gradient, 0.2 g/s. Position: angle of seat back, 10° from horizontal.
[1] $P < 0.05$ [2] $P < 0.01$

TABLE 3.—*Change in Resistance of Humans to Effects of Transverse $\pm G_x$ Acceleration Following Simulated Weightlessness of 60–100 Days, Using Various Preventive Measures* [113]

Time of weightlessness, d	Preventive methods	Maximum acceleration, G_x, M ± m		Mean difference, ΔG	Overall estimate of change in resistance
		Before	After		
60–70	Physical training	12.6 ± 0.55	12.5 ± 0.55	−0.1 ± 0.6	Reduction
70	Pharmaceuticals	11.5 ± 0.8	11.8 ± 0.4	+0.3 ± 0.4	Reduction
100	Physical training plus pharmaceuticals	13.4 ± 1.3	13.1 ± 1.45	−0.3 ± 0.2	Reduction
60–100	Average	12.6 ± 0.5	12.5 ± 0.45	−0.1 ± 0.3	Reduction

Shifts in acceleration tolerance before and after weightlessness and between groups are unreliable. Pharmaceuticals: securinine, caffeine, Phenamine.

Data from actual US and Soviet flights have not revealed any serious disruptions in work capacity or tolerance to accelerations such as those observed in model experiments. However, the possibility cannot be excluded that on longer flights, the deconditioning influence of weightlessness will appear still more sharply, if no preventive measures are found.

Shifts in the physiologic indicators of nervous and emotional origin are apparent during weightlessness, even before spacecraft descent. Toward the end of the flight, astronauts show an increase in pulse and respiration rates, which, apparently, result from anticipation of the descent—the most important, conclusive stage of the flight. Physiologic reactions during descent, as the spacecraft decelerates in the atmosphere's dense layers, are generally more clearly expressed than during ascent into orbit. According to most astronauts, accelerations during descent were subjectively much more severe than similar ones experienced in the centrifuge [115, 225]. Pulse and respiration rates were higher than with the same acceleration in the centrifuge. Some Soviet and US astronauts had pulse rates of 168–190 beats/min. During first flight descent, many astronauts experienced brief vision disruptions, which did not appear under the same accelerations in the centrifuge. Grayout in astronauts during descent might have resulted from orbital flight deconditioning on the cardiovascular system. These data should stimulate broader studies of man's resistance to acceleration effects after prior exposure to weightlessness.

PHYSIOLOGIC SYSTEMS OF THE BODY

Cardiovascular System

Circulatory system disruptions during exposure to acceleration are more significant than other body changes, and have a leading position in physiologic reactions. These disruptions result from redistribution of the blood's circulating mass capable of displacement. The degree of blood redistribution and the resultant general shifts in hemodynamics are determined primarily by the direction of the acceleration action. The greatest changes in general hemodynamics are under longitudinal acceleration ($\pm G_z$ stress), and the least, with transverse acceleration ($\pm G_x$ stress). These changes are explained by the position of the main blood vessels along the longitudinal axis of the body. When inertial forces act from head to feet ($+G_z$), the blood mass is displaced from vessels in the body's upper portion into vessels in the abdominal cavity and lower extremities. Redistribution of the blood changes blood pressure: in vessels located below the heart level, it increases; while in those above, it decreases. Under these conditions, blood flow through veins to the heart will be hindered, and the quantity of blood pumped by the heart is reduced, causing anemia of brain and sense organs, accompanied by vision disorders and possible loss of consciousness. When inertial forces act from the feet to the head ($-G_z$), blood displacement is in the opposite direction, causing blood to accumulate in the upper portion of the trunk with blood pressure above heart level increasing sharply.

Changes in general hemodynamics under transverse accelerations are significantly less than with longitudinal acceleration. However, a strictly transverse position in relation to the acceleration vector is rarely used in practice; in the majority of cases, the subject is in a position with the seat back inclined somewhat, which creates a longitudinal component. Therefore, when transverse accelerations are applied, the longitudinal component determines the value of general hemodynamic changes. Many of the body's organs and tissues have a highly developed network of vessels with approximately even distribution in all directions. Therefore, there will be blood displacement within limits of a given organ regardless of the direction of the inertial forces, which may cause regional circulatory disorders. The transverse component ($+G_x$) causes blood redistribution in pulmonary vessels from front to back.

By spinning animals in a centrifuge at accelerations of +2.5 g, pressure in the carotid artery decreased to 25% of its initial level, whereas it doubled in the femoral artery [100]. The role of the sinocarotid and aortal reflex zones in circulatory compensatory reactions during acceleration was determined in this experiment.

Dependence between the acceleration value and the degree of pressure reduction in the carotid artery has also been determined [9, 170]. These data were later confirmed [139], and it was established that with accelerations increasing at a rate of 1.0–2.0 g/s to a value causing loss of vision, there was an immediate drop in arterial pressure at head level with increased heart rate, reduction in blood content of ear vessels, and decrease in arterial pulse amplitude [129]. The arterial pressure at heart level, however, remained nearly at normal level or increased in connection with the compensatory spasm of the vessels [129].

Under the influence of $+G_z$ stress, animals showed signs of brain anemia. Color motion pictures showed that at an acceleration of $+8$ G_z, the cerebral cortex was pale, capillaries of the pia mater were quite empty, and blood in the large vessels took on a distinctly dark shade [98]. In man, during exposure to $+G_z$, distinct paleness of face and significant expansion of leg veins can be clearly seen. After acceleration has stopped, face paleness is replaced by hyperemia. Insufficient blood flow to the head and increased blood flow in the veins cause rapid emptying of the vascular bed of the head and neck. Simultaneously, reinforced influx of arterial blood to the abdomen and lower extremities is observed, and difficulty in venous drainage from these areas causes pooling of blood. An increase was noted in venous blood [38] and a leg plethysmograph used, to determine an increase in leg volume up to 350 cm^3. Deterioration in venous blood flow may result in a decrease of blood volume per beat and per minute [90, 244].

Blood Pressure

There is a direct dependence between the $+G_z$ forces and arterial pressure decrease. Comparison of curves of blood pressure changes in the carotid artery and increase in acceleration shows a certain lag in the organism's reactions (Fig. 12), and blood pressure changes begin to develop only after a certain time, not immediately after the acceleration application. As a result, at one stage, blood pressure may continue to drop while acceleration remains constant. As acceleration decreases, blood pressure does not return immediately to its initial value, but only after several seconds. These interrelationships indicate why visual disruptions and loss of consciousness arise suddenly sometimes during decreasing acceleration. This lag in blood pressure changes is explained by delay in cardiovascular compensatory reactions to acceleration, which, in turn, results from blood inertia, vessel tonus, and other causes.

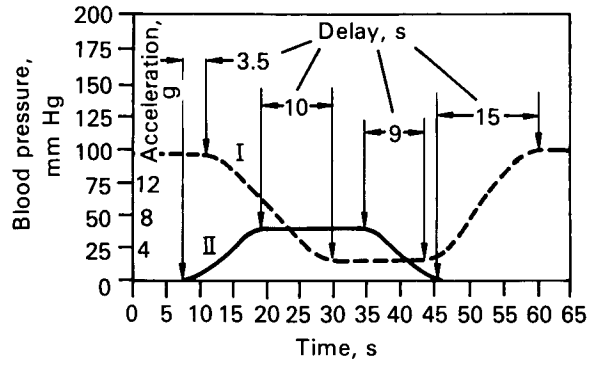

FIGURE 12.—Time relationships of dynamics of change in blood pressure and value of $+G_z$ acceleration [8].

X-ray studies have shown that heart and large vessel images become gradually paler, indicating blood decrease in heart cavities. After rotation stops, there is acute, brief expansion of the heart caused by sharply increased blood influx. Simultaneously, $+G_z$ forces lightened upper portions of the lungs and increased the darkening of lower portions. $+G_x$ forces caused lightening of anterior lung portions with darkening of posterior parts [47, 92, 138, 151].

Direct determinations were made of venous pressure in the jugular vein and arterial pressure at head level during exposure to $+G_z$ [89]. It was shown that with a significant decrease in arterial pressure in the upper half of the trunk, brain circulation was maintained by a pressure drop in the jugular vein of 30–50 mm Hg below 0. The difference between arterial and venous pressure maintained blood circulation even when arterial pressure dropped in the area of the head to 0, caused by the so-called siphon effect. Therefore, it is indicated that the develop-

ment of compensatory reactions retain the vital functions of the body under critical situations.

Investigations of the ear pulse volume showed that under $+G_z$ stress there is a decrease in blood and a reduction in pulse oscillation amplitude. When longitudinal accelerations were applied, a clear correlation was established between the arterial pressure level in the ear's vessels, the state of visual perception, and subsequent syncope. Reduction in blood pressure in vessels located above the heart acts through the sinocarotid zone and other angioreceptors to engage the mechanisms for compensation of hemodynamic disorders. An increase in heart rate and blood vessel constriction in a number of areas results. Under transverse accelerations, hemodynamic changes are primarily regional; therefore, there is no correlation. When consciousness is retained, full vision loss may still occur, while blood pressure at head level may be maintained at a rather high level. Vision disruptions in these cases result from disorders in regional circulation of the eye's vessels.

Complex restructurings in the hemodynamic system involving compensation mechanisms lead to an increase in pulse frequency. There is a rather clear dependence between acceleration and heart rate (Fig. 13). In man, depending on acceleration applied, the heart rate reaches 130–180 beats/min, rarely 190–200 beats/min or more. At transverse accelerations and optimal position, change in cardiac activity usually follows a definite sequence: (1) increase in heart rate; (2) stabilization; (3) decrease in frequency; and (4) restoration of cardiac activity or aftereffect.

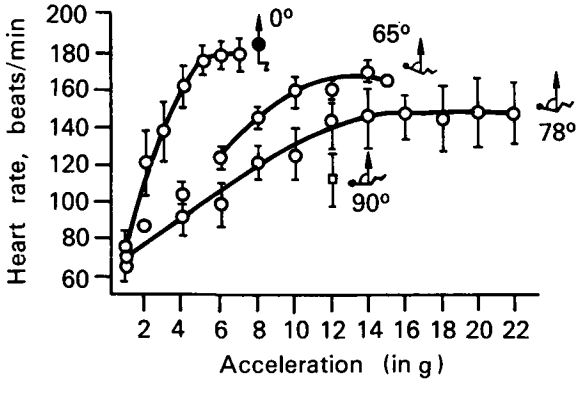

FIGURE 13.—Change in heart rate with accelerations in various directions [214].

No correlation has been found between increase in cardiac rhythm and tolerance to $+G_x$ forces. Disruption of compensation—progressive bradycardia—may occur at various heart rates. Therefore, absolute heart rate level cannot be a reliable criterion for prediction of bradycardia, and, consequently, a criterion for acceleration tolerance.

Special studies on the genesis of bradycardia in man, with preliminary injection of atropine, showed that temporary blockage of nervus vagus eliminated bradycardia not only at accelerations at which it is ordinarily recorded, but even at higher G of 14–16 G_x [113]. Therefore, vagus nerve tonus is important in development of bradycardia under the influence of $+G_x$ forces. Bradycardia developed in apes in spite of atropine injection, although significantly later than in controls [138]. During acceleration, as with many other effects, regulation of cardiac activity and manifestation of its adaptive reactions do not involve only extracardial nervous mechanisms. The heart has a wide range of autonomous adaptive mechanisms, adjusting its function to needs of the organism. Extracardial nervous regulatory influences duplicate largely effects of intracardial regulatory mechanisms [110]. Under transverse $+G_x$ stress, bradycardia may develop by restructure of extracardial influences (predominance of vagus nerve tonus) and by intracardial regulation.

Cardiac Disorders

The pathogenesis of compensatory mechanism failure to regulate cardiac activity is doubtless complex and includes many different changes in the total chain of body reactions to acceleration.

Important factors in overstressing the organism's compensatory capabilities with subsequent cardiac disorders are: functional change of the autonomic centers regulating the cardiovascular system; reflex influences resulting from general and regional circulatory disruptions; displacement of organ positions; myocardial hypoxia; and exhaustion of heart energy resources. The specific significance of each of these links in the patho-

genesis of compensation failure may differ, depending on the acceleration's mode and nature and the body's functional state.

Another disruption in cardiac rhythm under the influence of transverse $+G_x$ stress, encountered no less frequently, consists of various types of extrasystoles (Fig. 14). Ventricle and atrioventricle extrasystoles are encountered most frequently, the auricular type less frequently. Atrioventricular extrasystoles are more unfavorable than ventricular extrasystoles clinically, since auricle and ventricle contractions are simultaneous. Thus, blood from the auricle is not pumped into the ventricle, but back into the veins, hindering emptying of the veins and decreasing systolic volume and volume per minute. The total of extrasystoles arising with a reduction in heart rate is almost 4.5 times greater than those before bradycardia develops. Extrasystoles developing during $+G_x$ stress might be caused by various respiratory maneuvers, hyperventilation, blood oxygenation reduction, possible pulmonary atelectases, as well as fear and alarm, or simply back position, which sometimes facilitates arrhythmia [216]. Most researchers consider extracardial influences dominant, which act on the heart through vagus and sympathetic nerves. Extrasystoles are more frequent with $+G_x$ than with longitudinal $+G_z$ forces.

Attacks of paroxysmal tachycardia have been recorded very rarely [50, 113, 210]. Clinicians believe extrasystoles might be precursors of paroxysmal tachycardia. One instance of paroxysmal tachycardia under $+G_x$ forces was recorded, which was preceded by extrasystoles and relative bradycardia (Fig. 15).

Electrocardiographic studies have shown instances of sinus tachycardia, shortening of the P–Q, Q–T, and R–R intervals, increase in the P-wave, thickening of the T-wave, increase in the systolic index, and various disruptions of cardiac rhythm. Changes in the heart's bioelectric activity might be due to a combination of factors: heart position displacement, changes in tonus of autonomic nervous system and humoral elements, change in blood content of heart cavities, and myocardial hypoxia.

Under $+G_x$ stress, there are significant changes in the hemodynamics of pulmonary circulation – blood redistribution in the pulmonary artery system [104]. Pulmonary circulation disruptions lead primarily to disorders in oxygen delivery from alveolar air to blood. Changes in the normal hemodynamics of pulmonary circulation and reduction of blood oxygen indicate that the heart may undergo hypoxia. $+G_x$ stress might cause myocardial ischemia [199], which in turn might lead to angina pectoris [151]. Changes in electrocardiograms (ECG) have been related to myocardial hypoxia in dogs during $+G_x$ [121]. O_2 in arterial blood and in coronary sinus blood has been shown to be dependent on the value and duration of application of transverse $\pm G_x$ [232], where the demand for oxygen by the heart is indicated by O_2 reduction in coronary sinus blood with simultaneous increase in blood flow from the sinus. Maintenance of a sufficiently high level of coronary blood flow is obviously a primary factor compensating for increasing oxygen demands of the myocardium. Slight ECG changes disappear rapidly after rotation when an increased flow rate from the coronary sinus is observed. In experiments where decreased blood flow was observed at this point, ECG changes were sharper and lasted longer.

Blood Volume

Systolic blood volume, under forces up to $+5$ G_x for brief periods, either remains unchanged or increases slightly. An increase in acceleration duration to 10 min causes a drop in systolic volume [140]. Blood volume per minute, under forces up to $+5G_z$, also remains unchanged or increases. Consequently, reduction in volume per minute depends primarily on pulse frequency. Increases in the blood pressure of aorta and right auricle have been clearly observed [140, 244].

As $+G_x$ forces increased, aorta arterial pressure increased and pulse frequency decreased significantly [202] (Table 4). Cardiac output did not change with forces of $+5$ G_x, but decreased progressively at accelerations of $+10$ G_x and $+15$ G_x, due to increase in peripheral resistance, particularly in the vascular lumen of abdomen and muscles. However, resistance of

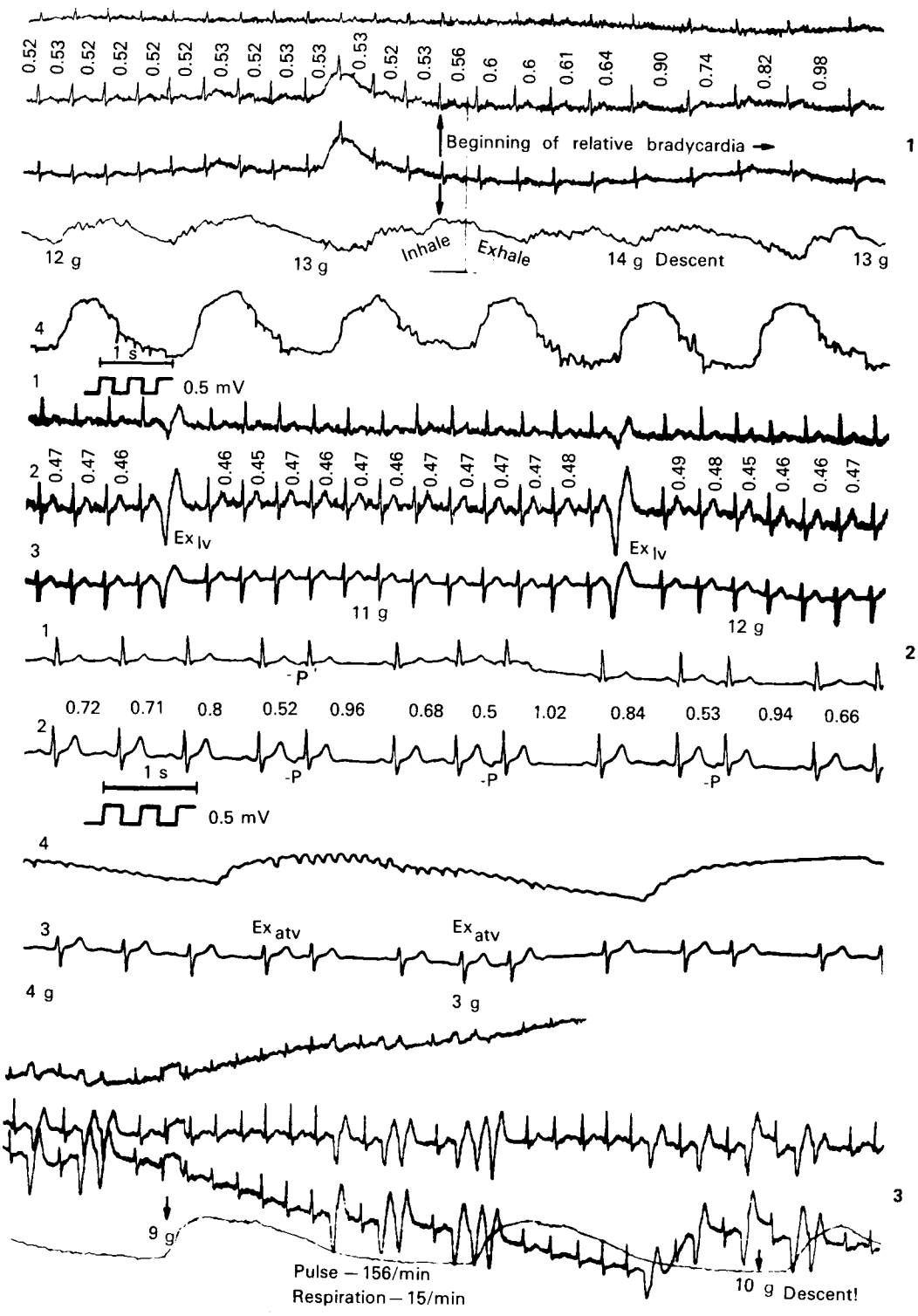

FIGURE 14.—Disruptions in man's cardiac rhythm under transverse $+G_x$ acceleratory stress [113]. 1: Development of bradycardia; 2: individual extrasystoles (Ex_{lv}, left ventricular extrasystoles) (Ex_{atv}, atrioventricular extrasystoles); 3: group extrasystoles.

important vascular formations, such as the left coronary and common carotid arteries, changed only slightly.

Morphologic studies of animal hearts subjected to transverse acceleration indicated oligemia, and in many cases hemorrhaging, edema, and dystrophy of cardiac muscle [162]. There is a definite dependence between cardiac activity

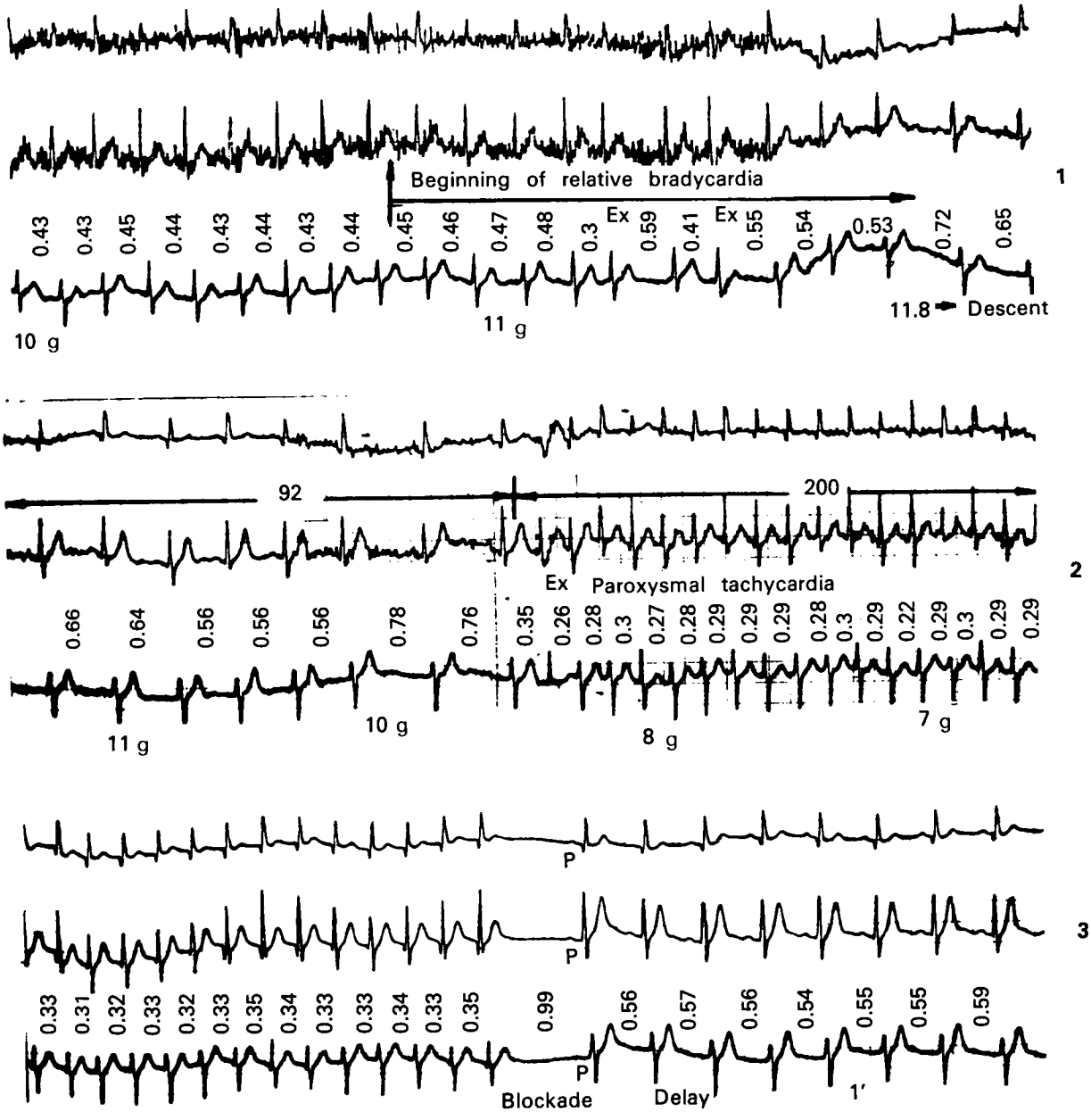

FIGURE 15.—Paroxysmal tachycardia in man under transverse $+G_x$ acceleratory stress [113]. 1: Dynamics of change of cardiac rhythm under + 10.0–11.8 G_x acceleratory stress, againt a background of relative bradycardia (arrow); extrasystoles of atrioventricular type (Ex) were recorded. 2: Relative reduction in cardiac rhythm suddenly replaced by acute paroxysmal tachycardia (200 beats/min), ECG shows no P-wave. 3: Before stopping centrifuge, paroxysmal tachycardia suddenly stopped, and after block, was replaced by sinus rhythm; P-wave reappeared on ECG.

disruption and glycogen content in myocardium and liver [26].

Cardiovascular system regulation is governed significantly by peripheral receptors. A reduction in blood volume per minute to 73–77% of its initial level was observed during exposure of narcotized animals to +9 G_x, whereas in intact dogs, blood volume per minute increased by 52–53% [190]. In all experiments on narcotized dogs, with forces of 5.0–6.0 G_x and more, there were reductions in blood volume per minute and systolic blood volume, with insignificant changes in arterial pressure at heart level. In intact animals and humans, a regular increase in blood volume per minute was observed and an increase in arterial pressure during spaceflight acceleration [76, 118, 228].

Brain blood changes slightly at +7.0–8.0 G_x [150]. At accelerations of +8.0–10 G_x with the seat back inclined at 25° from the horizontal, the pulse blood filling the brain is lower than the initial level, although arterial pressure at heart level increases. The cerebral vessel's blood content during transverse acceleration is determined by the longitudinal component in the head-pelvis direction (+G_z). An increase in blood is observed when this component is 1.6–1.8 G_x, while at +3.0 G_z, it is equal to the initial level, and at +5.0 G_z, begins to decrease.

Thus, acceleration causes changes in frequency and force of cardiac contractions, in cardiac output per beat and per minute, in arterial and venous pressure, and in the general and regional speeds of blood flow. It redistributes circulating blood and causes many physiologic changes.

Respiratory System

The influence of acceleration on external respiration is determined not only by the value and time of application, but also by direction of the acceleration vector in relation to the vertical axis of the body. The principal effects are from changes in respiration biomechanics and increases in hydrostatic lung pressure.

With +G_z forces up to +5.0 G_z, progressive increases in respiration frequency, volume per inhalation and per minute have been observed [10, 63, 73, 210]. There were increases in O_2 consumption, CO_2 liberation, and other respiratory factors [22]. Reduction in lung extensibility was noted at this level, plus a slight increase in functional residual volume [21]. Diaphragm lowering and increased intraperitoneal pressure become significant under these conditions. Increase in pulmonary ventilation with +G_z stress is accompanied by less increase in effective alveolar ventilation [22]. With constant recording of lung volume and pressure gradient, neither elasticity nor resistance to air flow changed up

TABLE 4.—*Change in Hemodynamic Indicators in Dogs Under Transverse +G_x Acceleration* [207]

Indicators	Control	5.0 G_x–2 min	10.0 G_x–2 min	15.0 G_x–1 min
Arterial pressure in aorta, mm Hg	140	164	186	207
Arterial pressure in left ventricle, mm Hg	154/3	171/8	200/24	217/48
Rate of blood flow in ascending aorta, v, cm/s	25	25	20	16
Rate of blood flow in left coronary artery, v, cm/s	43	55	54	81
Rate of blood flow in carotid artery, v, cm/s	31	28	31	37
Pulse rate, beats/min	124	144	70	60

Resistance of vessels was calculated by dividing mean pressure in aorta by various blood flow rates in arteries.

to +3 G_z. However, the total workload of the respiratory apparatus increases due to reduction in elasticity of thorax walls and increase in intraperitoneal pressure.

X-ray studies on men and animals have shown increased transparency of the lung image in the upper portion and increased image density in the lower portions [145, 152]. These changes result from hydrostatic effects of acceleration on pulmonary parenchyma. The alveoli in the upper portions of the lungs expand greatly, and tissue anemia sets in, to the point of total cessation of circulation at forces of 6.0–7.0 G_z, while edema, atelectases, and blood pooling are in the lower portions. Therefore, in spite of increase in respiration volume per minute and alveolar ventilation, uneven air flow distribution in the alveoli and hemodynamic changes resulting from +3 G_z and more cause disruptions in arterial oxygenation [21].

External respiration is most significant for transverse $+G_x$ stress, tolerance for which is frequently limited by respiratory disorders. An increasing external pressure resulting from the transverse acceleration component causes thorax deformation, changes in positions of organs, and difficulty in normal respiration. Respiratory reaction to $+G_x$ stress is characterized by a moderate increase in the volume of respiration per minute (VRM), due primarily to sharp increase in respiration frequency (Fig. 16). The respiratory volume increases up to +6 G_x, then begins to decrease. For this reason, at high accelerations, in spite of the rather high level of VRM, the alveolar ventilation deteriorates. Mechanical compression of the thorax causes a reduction in all pulmonary volume, with some increase in residual air (Fig. 17).

The vital capacity of the lungs decreases, approaching 0 at high accelerations, at which time the tolerance for accelerations is determined by the respiration delay time [47]. The shifts observed originate to a great extent from the increased work of the respiratory apparatus, due to the elastic component and significant reduction in dynamic extensibility of lungs [233]. Thus, the effects of high transverse $+G_x$ forces are ac-

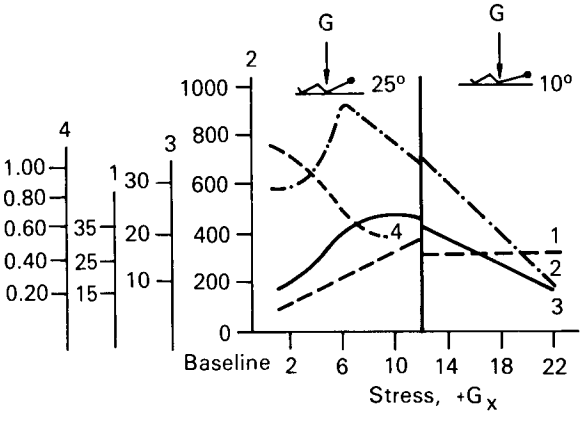

FIGURE 16.—Dynamics of change in man's respiration with $+G_x$ acceleration [17]. 1: Respiratory rate per min; 2: respiratory volume, ml; 3: respiratory volume per min; 4: ratio of vital capacity of lungs during acceleration to value in initial state.

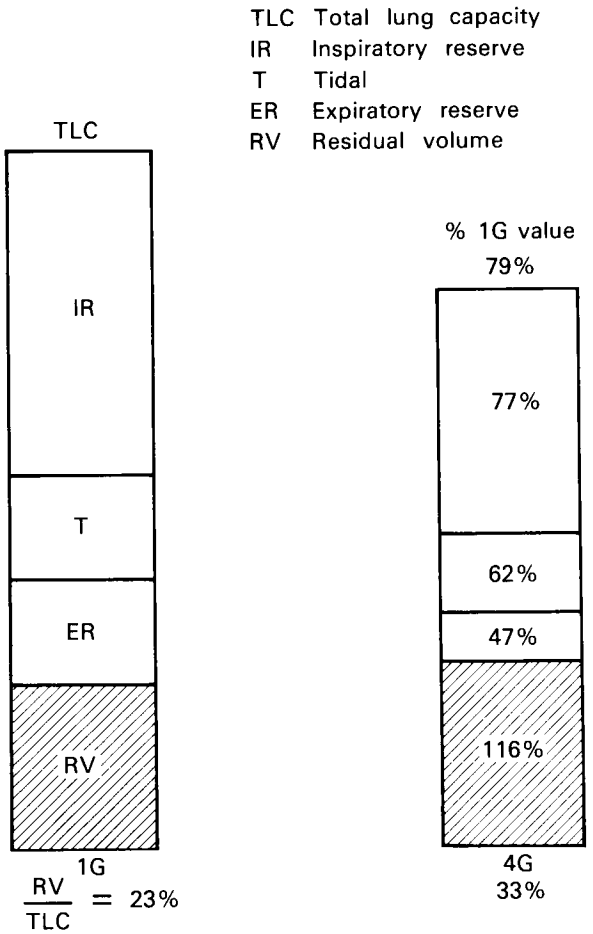

FIGURE 17.—Change in pulmonary volume with $+G_x$ accelerator stress at 4.0 g [47].

companied by significant reduction in pulmonary reserve and deterioration of the effectiveness of ventilation, which is confirmed by experimental data indicating a reduction with increasing acceleration in the oxygen utilization factor, i.e., the quantity of oxygen absorbed in the lungs from each liter of ventilated air (Fig. 18). The hydrostatic effects in the lungs from $+G_x$ are similar in principle to those for $+G_z$ stress. The differences are purely regional, i.e., hypoperfusion and hyperventilation in the anterior portions of lungs, with hypoventilation and hyperperfusion in posterior portions. These effects become obvious after analysis of the relationship between the gradient of alveolar pressure and pressure in lung vessels under acceleration (Figs. 19, 20).

Thus, the hydrostatic effects of acceleration in any direction cause unevenness of ventilation and blood circulation in lungs and, consequently, lead to disruption of arterial oxygenation of blood (Fig. 21). Damage to lung tissue is also possible (congestive hyperemia, perivascular edema, hemorrhage, and atelectases), which has been established in animal experiments [108, 119, 162].

Direct results of disruptions in respiratory system functioning under $+G_x$ stress include shifts in gas metabolism, manifested in reduced O_2 intake and extraction of CO_2 during the actual application of acceleration, with subsequent sharp increases during the aftereffect [17, 193, 200]. These effects are accompanied by increases in the oxygen debt with increasing acceleration (Fig. 22) and apparently are related to the significant deterioration in hemocirculation during acceleration. Deterioration in the diffusion capacity of the lungs might also be significant [247], although this requires further study.

The oxygen debt also reflects an increase in organic metabolic processes during acceleration and is caused by a reduction in anaerobic metabolic products [80].

When transverse forces in the opposite ($-G_x$) direction are applied, respiratory changes are less manifest and approximate those for $+G_z$ forces. With $-G_z$, a slight increase in the respiratory depth and rate is observed up to -3.0 G_z [9]. With accelerations of -6 to -8 G_x, pulmonary ventilation increases due to increased respiratory rate and volume with moderate de-

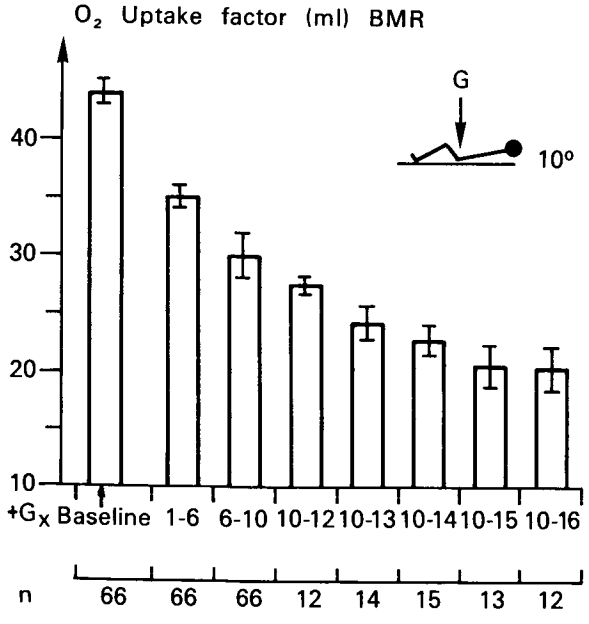

FIGURE 18.—Oxygen uptake factor in lungs under various $+G_x$ acceleratory stresses of peak type [193]. Acceleration rise rate 0.2 g/s; n = number of persons.

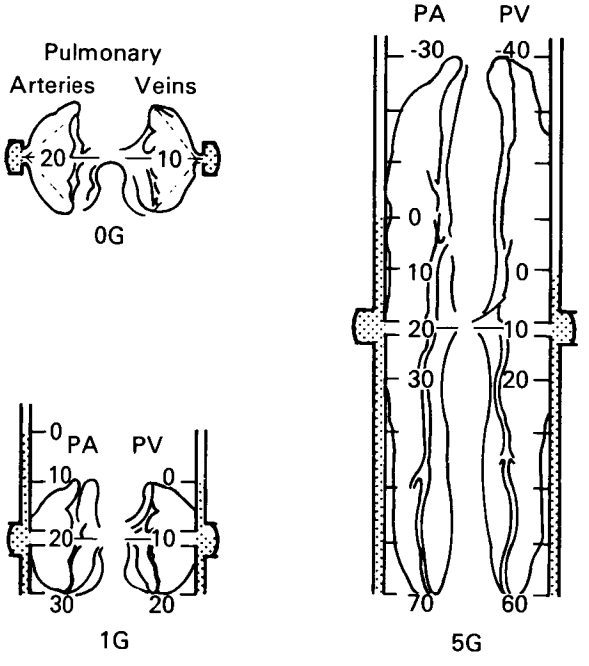

FIGURE 19.—Hydrostatic effects of $+G_x$ acceleratory stress on pulmonary hemodynamics [139]. Numbers show pressure of water column, cm. 0: atmospheric pressure in central portion of thorax; dorsoventral dimension of lungs, 20 cm.

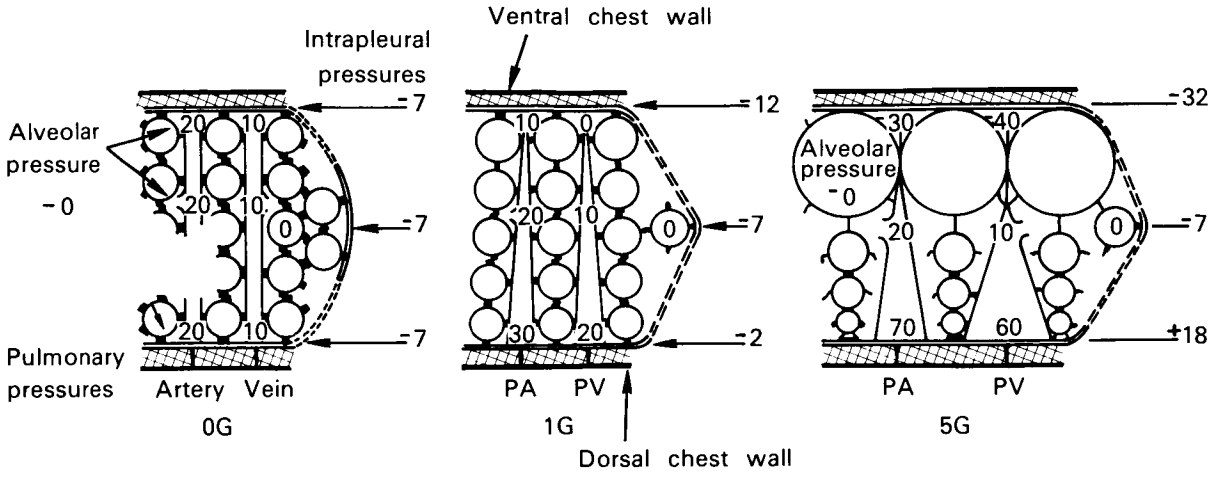

FIGURE 20.—Influence of transverse $+G_x$ acceleratory stress on intraperitoneal pressure [242]. Dorsoventral dimension of lungs, 20 cm; numbers show pressure of water column in cm; 0 shows atmospheric pressure in central portion of thorax in plane of heart.

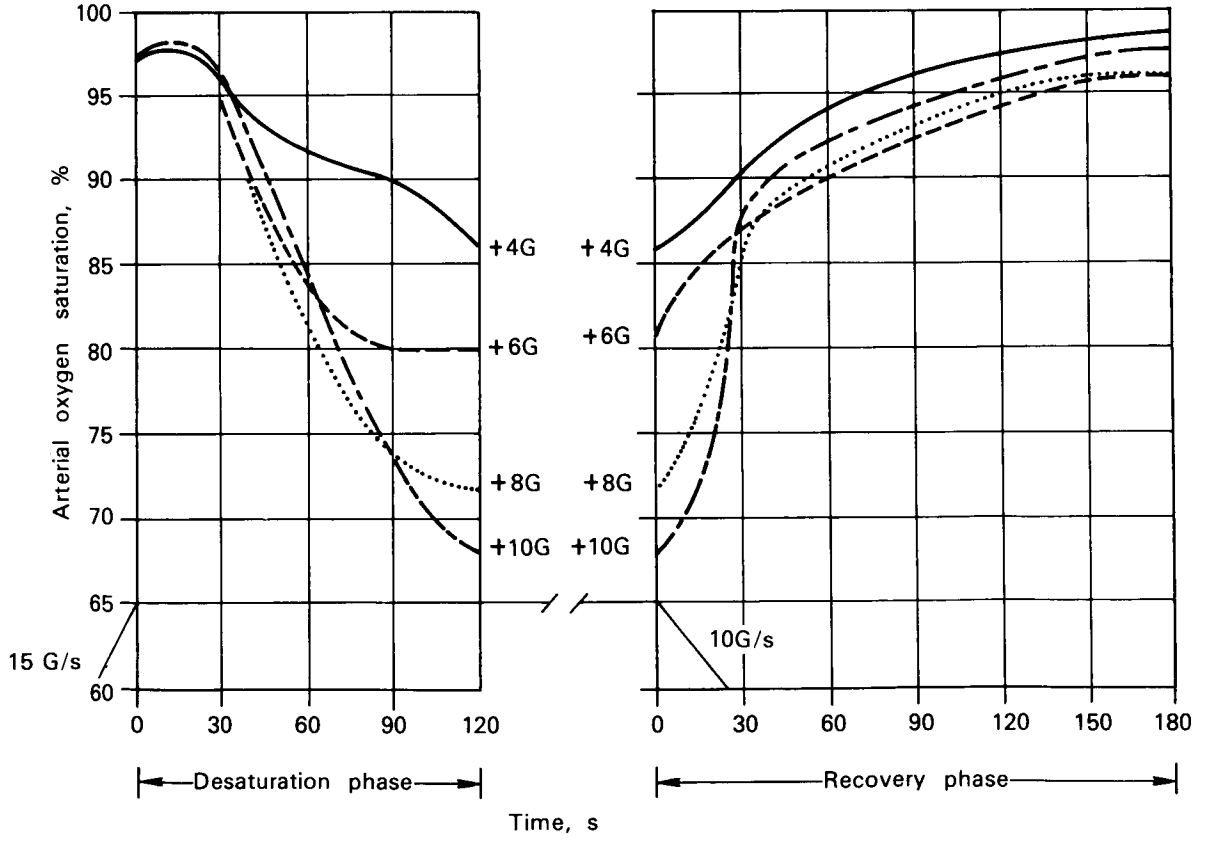

FIGURE 21.—Oxygenation of arterial blood during breathing of air [5]. Ordinate shows arterial oxygenation in %; abscissa shows reduction phase and restoration phase of blood oxygen saturation.

crease in the vital and functional residual capacities of lungs [172, 196].

The arteriovenous shunting and deterioration of gas metabolism in lungs during accelerations unavoidably lead to disruptions in arterial blood oxygenation. This factor, added to the insufficiency of blood supply, is the most important in the mechanism of tissue hypoxia present under long-term exposure to acceleration. Detailed information is available on the $+G_x$ direction of the acceleration vector. There is reduced oxygenation of arterial blood after as little as 30 s exposure to +3.0 G_x or more [4, 104, 201, 232, 242]; such changes are shown in Figure 21. Reduction in oxygenation of blood is never as great as for hypoxic hypoxia, and at high acceleration values is partially compensated by respiration of 100% oxygen [4].

Thus, disorders in the external respiration system are significant in the pathogenesis of disrupted functions of other body systems under the influence of acceleration.

Visual Analyzer

The reliability of astronauts' work depends to a great extent on the functional state of the visual organ. However, vision has proved susceptible to the effects of long-term acceleration in various directions.

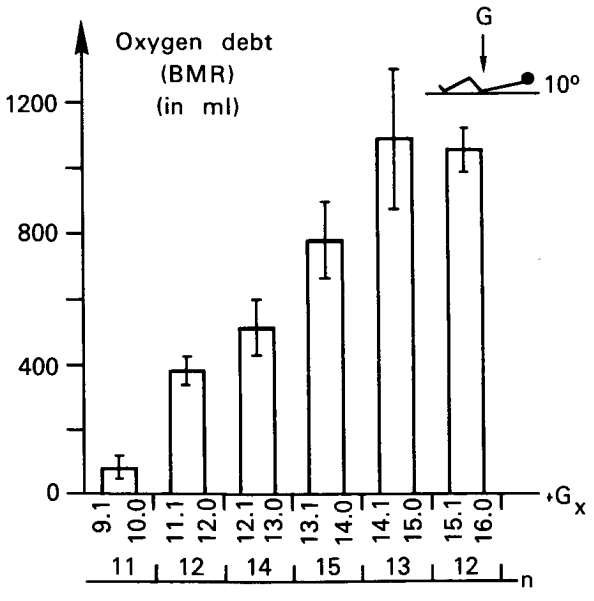

FIGURE 22.—Oxygen debt in man following exposure to $+G_x$ accelerations of various values [193]. Acceleration rise rate: 0.2 g/s; n = number of persons.

In aviation medicine, visual disorders serve as a reliable criterion for the tolerance limit to $+G_z$ forces [11, 33, 38, 73, 114, 189, 210] because disruptions in visual function under $+G_z$ precede loss of consciousness resulting from reduced cerebral blood circulation. Depending on the gravitoinertial value and duration of application, disruptions of vision go through successive phases: constricted field of vision, grayout, then blackout, and finally total loss of vision. The sensations of those who have been subjected to acceleration include: gray veil, fog, whitish fog, looking through rain or fog, and so forth, followed by total blackness but with retention of consciousness and hearing. The threshold of grayout and blackout depends on the subject's position in relation to the inertial force vector, value and duration of its force, and the body's functional state. Visual disorders frequently start at the beginning of acceleration application, then, despite continued acceleration, disappear after 8–12 s, with vision sometimes fully recovered. This is explained by the delayed development of compensatory reactions, primarily those of the cardiovascular system, for which the latent period of engagement is the same (8–12 s).

The thresholds of vision disruption for 1000 test subjects under the influence of $+G_z$ forces [52] are presented in Table 5.

With transverse acceleration, visual disruptions also result. The thresholds of grayout and blackout for $+G_x$ are determined by the subject's position. Calculation data in Figure 3 provide for determination of the relative percentage values of components on the $+G_z$ and $+G_x$ axes from the resulting acceleration vector, as well as the relative retinal-aortal $+G_z$ component (along the axis between the retinas and the arc of the aorta) for various positions of the seated body—from lying-on-the-back to sitting positions. The absolute retinal-aortal component can be used to predict the probability of visual disorders.

The threshold for development of grayout for various effective physiologic angles (Fig. 23) has been established experimentally [22]. With the

TABLE 5.—*Thresholds of Visual Disorders and Loss of Consciousness Under* $+G_x$ *Acceleration* [52]

Symptoms	Mean threshold, G_z	Standard deviation	Extreme values
Loss of peripheral vision	4.1	±0.7	2.2–7.1
Blackout	4.7	±0.8	2.7–7.8
Loss of consciousness	5.4	±0.9	3.0–8.4

Erect "seated" position, muscles relaxed, acceleration rise rate 1.0 g/s.

seat back angle ($SA+\epsilon=20°–25°$) so that inertial forces along the head-pelvis axis are significant, visual disruptions precede loss of consciousness. Under these conditions, similar to $+G_z$ longitudinal forces, the visual function correlates definitely with disruptions in general hemodynamics. With the body in a more horizontal position ($SA+\epsilon=10°–12°$), visual disruptions do not precede loss of consciousness, but indicate disruptions in retinal circulation. Thus, in the optimal position ($SA+\epsilon=10°$), vision is completely switched off at levels of +14.0–16.0 G_x, without loss of consciousness [16, 24, 38, 110, 113]. Consequently, visual disorders caused by the influence of $+G_x$ forces in various body positions have different significance for evaluating the body's condition.

With increasing acceleration, visual acuity decreases, and the width of the vision field narrows with a drop in absolute light contrast sensitivity, and reaction time to light signals increases [15, 70, 73, 97, 196].

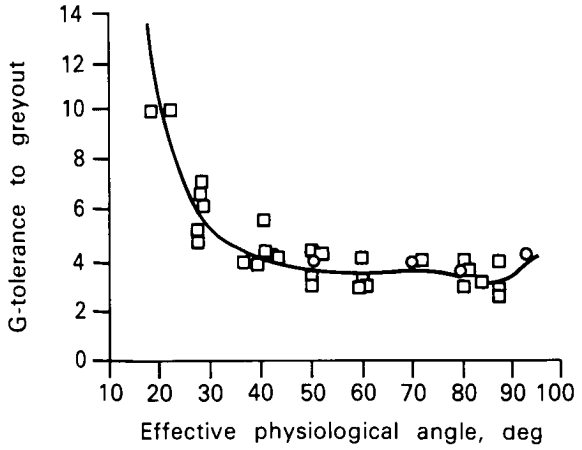

FIGURE 23.—Development of grayout as a function of subject's posture [45].

Visual acuity changes with different directions of acceleration ($+G_z$ and $+G_x$) [222] (Fig. 24). When the limiting $+G_z$ forces are reached (over 6.0 g), visual acuity drops sharply, usually due to visual disorders. Under transverse acceleration, the reduction in visual acuity is less pronounced.

A reliable reduction in visual acuity was observed under transverse forces beginning at +6.0 G_x with the seat back at an angle of 25°, and at +10.0 G_x with the seat back at a 10° angle. In $-G_x$ visual disruptions become distinct at 6.0—8.0 G. It is assumed that fogging and reduction of acuity vision with $-G_x$ might be caused by displacement of the cornea. However, when a special optical device was used [196], there were no changes indicating deformation of the cornea; these visual disorders probably result from abundant lacrimation.

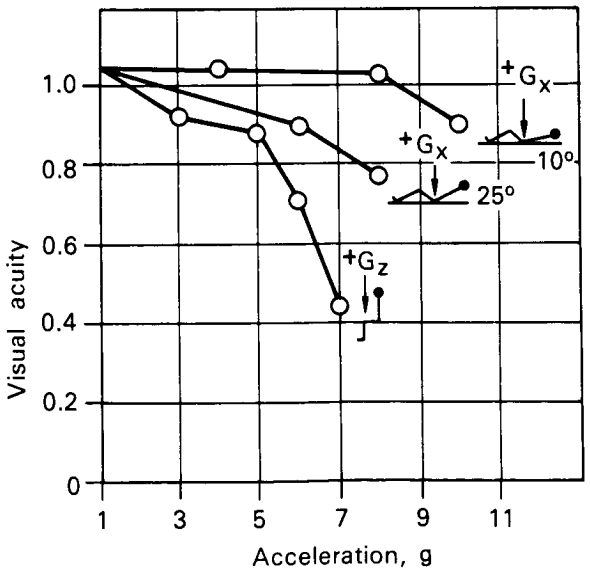

FIGURE 24.—Visual acuity of man during exposure to accelerations in various directions [222].

Visual Disruptions

A contribution to reduction of visual acuity under acceleration might be by the contrast or discrimination sensitivity of the eye, the basis of object vision. High contrast sensitivity is essential for adequate perception of visual information. The thresholds of the eye's contrast sensitivity increase with decreasing background illumination and increase for illumination level with increasing $+G_z$ and $+G_x$ forces. With $+G_z$, the thresholds of contrast sensitivity are higher than with $+G_x$.

Shifts in visual functions were apparent long before hemocirculatory disorders appeared, causing grayout and blackout, according to studies of contrast sensitivity and operators' reaction time [222]. The earliest changes were at $+4\,G_x$ (in the position $SA+\epsilon=10°$). As acceleration increased, reaction time to light signals increased, and finally, a reduction in visual acuity. Another area of interest is the gradual change of absolute visual threshold with acceleration, i.e., the minimum light stimulus which can be perceived. The threshold of central vision at $+3.0\ G_z$ is almost double, and at 4.0 G_z, 3.4 times higher than at 1.0 G_z (with a 50% level of probability) while the threshold of peripheral perception increases 1.5 times at 2.0 G_z, 3 times at 3.0 G_z, and 4 times at $+4G_z$ [236, 237]. Pupil reactions do not change under the influence of acceleration [196]; dilation of the pupil was noted, accompanied by loss of peripheral vision [24].

Thus, long-term acceleration causes constriction in the field of vision, limitation of voluntary motion of eyes to the point of ataxia, deterioration in recognizing low-intensity signals and contrast sensitivity, and in differentiation of fine details. Disruptions in vision are usually explained by disorders in regional hemodynamics and retinal hypoxia [6, 11, 64, 65, 66, 126, 129, 174]. However, such developments are sometimes related to changes in activity of the visual analyzer's cortical portion [87, 165], and finally, a few researchers do not differentiate these mechanisms [239].

Vision is clearly disrupted under the influence of altered hydrostatic pressure, and symptoms of disruption appear at levels preceding loss of consciousness. Under conditions of acceleration, blood supply to the eye can continue normally only if blood pressure in the central retinal artery is greater than the level of pressure in the eye, normally equal to 22–23 mm Hg (with fluctuations from 18–30 mm Hg). This was confirmed by application of a negative pressure of 30–40 mm Hg to the eyeball by means of suction goggles which increased the threshold of blackout [125]. These experiments were later confirmed [101]. In a sealed chamber, when pressure was reduced in front of one eye, this eye regained clear vision, but when pressure was not reduced in front of the other eye, it remained blind.

Direct ophthalmoscopy of the fundus oculi was performed on men and apes during application of $+G_z$ forces at the moment of blackout, and arterial pressure in the radial artery was measured with the arm at eye level [65]. A correlation was established between changes in visual function and morphologic changes in the area of the fundus oculi. Data from this study are presented in Table 6. Photographs were made of the fundus oculi of human subjects during $+G_z$, with and without visual disruption [135, 153]. A definite dynamic sequence of changes of retinal circulation was recorded similar to that obtained by ophthalmoscopy [65]. When vision was clear, practically no ophthalmoscopic changes were observed on the fundus oculi, but were maximally expressed while in blackout, up to cessation of blood flow in the retinal vessels (Fig. 25).

Since the inner layers of the retina are highly sensitive to hypoxia, it can be assumed that hypoxia in these layers starts during retinal ischemia [137]. The critical point of application of hypoxia is at the sympathetic connections of the ganglionic and bipolar cells of the retina. Visual disorders such as grayout appear under the influence of $+G_z$ when the systolic arterial pressure at the head level falls below 50 mm Hg [126, 129, 210]. With transverse $+G_x$ forces and the test subject in the optimal position, when inertial forces along the head-feet vector are slight (not over 18–20% of the total acceleration), visual disorders also indicate disruptions of blood circulation in the retina. These disruptions are apparently related primarily to disruptions of regional circulation in eye vessels, but are not

precursors of a critical reduction in the cerebral circulation level or, consequently, loss of consciousness.

In studies of vascular reactions in the retina following $+G_x$ stress, two phases were differentiated in fundus oculi changes in the aftereffect [123]. During the first phase, directly after termination of acceleration and during the first few hours, reactive hyperemia predominates (hyperemia and blurring of the boundaries of the visual nerve discs, ectasia of the capillaries, expansion of the retinal veins and arteries). During the second phase—several hours to several days—phenomena related to disruption of permeability of the vascular walls in the retina predominate (perivascular edema of the retina, increased dimensions of the physiologic scotoma). The degree of changes manifested in the vascular system of the retina depends on the value and application time of acceleration [111, 245].

Disruptions in regional circulation in the eye's vessels under transverse acceleration may also result from the inertial force component along the forehead-occipital axis.

Visual disorders arising during acceleration are generally thought to result from anoxia of the retina and brain cells due to hemodynamic disruptions, although there are other opinions. Disruption of visual analyzer activity during acceleration could be ascribed to a mixed cortico-retinal mechanism with enforcement of inhibitory processes in neurons of the retina, reduction in neuron excitability of the cortical part of the visual analyzer, and retardation of synaptic transmission in neurons of the visual tract [179].

Central Nervous System

In the last 30 to 40 years, much experimental work has been concerned with the influence of acceleration on various portions and levels of the central nervous system (CNS), using various animal species and man as subjects. A detailed summary of this research is contained in a monograph by Savin [179].

The first studies concentrated primarily on the effects of positive $+G_z$ and, to a lesser extent, negative $-G_z$. It was established that $+G_z$ stress significantly disrupt perception, increase simple and complex motor reaction time, and elongate latent response periods to sound and light signals [38, 231]. These results were later deepened and expanded [56, 105]. Soviet researchers subsequently investigated higher nervous activity (HNA) using conditioned reflexes, and established that even at slight $+G_z$ stress, there is a clearly expressed elongation of latent reaction periods to conditioned stimuli, and that restoration of normal latent conditioned motor reflex usually occurs in waves [180], beginning 20 to 30 s after cessation of acceleration at 3.4–4.0 G_z, and 1.5–2 min after cessation of acceleration at 5.0–7.0 G_z. Experiments with animals are probing the nature and mechanism of higher nervous activity disruptions during acceleration. Experiments with motor-defense reflexes [231] and with food reinforcement [180] indicate that conditioned reflex changes are observed as early as 1.5–3.0 G_z. These acceleration levels primarily damage internal inhibition, cause phase phenomena, and, as accelerations become higher, full inhibition of conditioned reflexes. At the beginning of acceleration, an increase in excitability of the cerebral cortex is observed. These investigation results have been confirmed and expanded [156].

The application of $+G_z$ forces also causes significant changes in brain bioelectric activity [1, 98, 99]. Slight accelerations of $+2.0$–3.5 G_z result in a significant increase in frequency and

TABLE 6. — *Disruptions in Visual Function and Changes in Fundus Oculi with* $+G_z$ *Acceleration* [65]

Stage	Subjective sensations	Objective data
I	Loss of peripheral vision	Pulsation of arterioles, periodic discoloration
II	Blackout	Evacuation and spasm of arterioles
III	Restoration of central and peripheral vision	Restoration of pulsation of arterioles and temporary expansion of veins

amplitude of rapid fluctuations and depression of slow waves, while greater accelerations of +3.0 to 6.0 G_z initially cause δ-waves, which are later replaced by full depression of bioelectric activity.

An analysis of available materials leads to the conclusion that $\pm G_z$ forces cause phasic changes in CNS functions, determined by value and time of accelerations, direction of the inertial force vector, animal species and their initial functional condition.

The effect of transverse $\pm G_x$ forces on CNS functions is characterized by the same changes recorded for $\pm G_z$. Experiments with human subjects established that with an acceleration of +3 G_x, latent conditioned reflex periods are slightly shortened. When G forces increase to +5 G_x, conditioned reflexes pass through two phases. First, there is a significant elongation of latent present reflex periods, while latent periods of trace reflexes are shortened. Subsequently, with greater elongation of latent periods of present reflexes to both light and sound stimuli, there is some elongation of latent periods of trace reflexes with simultaneous sharp increases in numbers of errors. Pronounced inhibition of conditioned reflexes arises. In some experiments, following elongation of latent periods of trace reflexes, visual disorders or even fainting arose [36]. These results were later amplified in experimentation with both humans and animals.

Phasic changes in the functional state of the CNS were found applicable to various conditions in a number of later experiments using electroencephalograms (EEG) [20, 74, 75, 221]. In the first phase, immediately after application of acceleration, there was a significant increase in the number of fast potentials, with simultaneous decrease in amplitude—the reaction of EEG desynchronization [96]. In the second phase, there were high-amplitude slow waves—the reaction of EEG synchronization. The third phase, occurring only with accelerations over 6.0 G_z, showed deepened synchronization of cortical bioelectric activity with decompensation of cardiac activity and respiration.

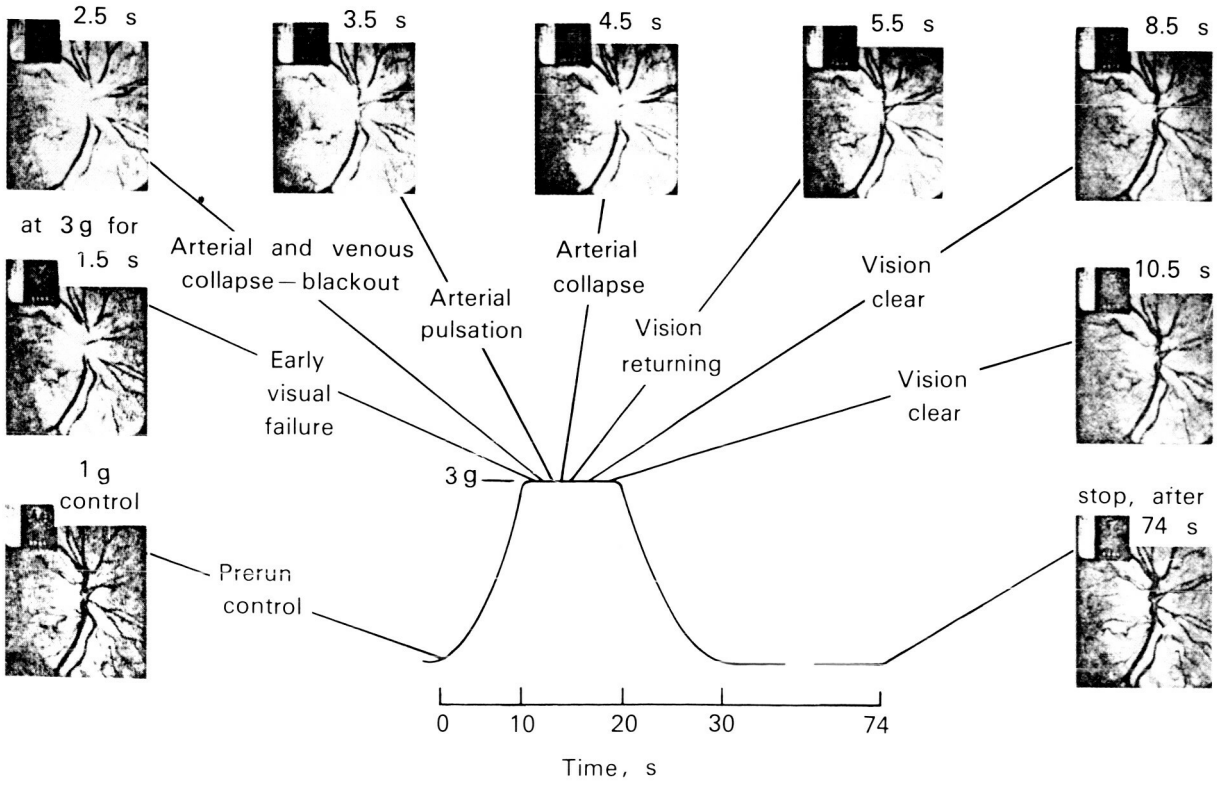

FIGURE 25.—Dynamics of change in circulation in retinal vessels of human subjects during exposure to $+G_z$ acceleratory stress [153].

The desynchronization phase in human subjects was observed with longitudinal forces of 3.0–4.0 G_z, and transverse forces of 5.0–8.0 G_x [75]. The phase of exalted α-rhythm began during longitudinal forces of 5.0–7.0 G_z and transverse forces of 8.0–10.0 G_x. With further increases in acceleration, the slow θ- and δ-waves predominated, usually preceded immediately by visual disorders. Rabbit experiments showed that during the first phase, when there was desynchronization in the cortex, synchronous oscillations of the primary rhythm (5–6/s) increased in the reticular formation and hypothalamus. Under accelerations of + 7.0 G_x, high-amplitude slow waves appeared in the cortex. At this time, slow high-amplitude activity was also noted in the hypothalamus, while reduced amplitude of the primary rhythm, compared to initial data, was recorded in the reticular formation. Then a significant shift in the direction of faster oscillations occurred in the potentials recorded from the hypothalamus. During aftereffect, first the cortex was restored, then the hypothalamus, and finally, the reticular formation.

Functional disorders are often accompanied by histochemical and histological changes [119, 122].

The disruption mechanism of CNS function during acceleration has not been conclusively clarified. Most authors place primary significance on the developing of hypoxia [4, 21, 73, 186], but some believe that the most important aspect of the mechanism is strengthened afferentation from deformed organs and tissues [179]. Thus, accelerations cause functional disorders not only in the cerebral cortex, but also in many other CNS formations [154]. These functional shifts lead to disruptions in regulation and coordination of the CNS, and often reduce man's working capacity.

Endocrine Glands

There are numerous reactions in the endocrine apparatus under the influence of acceleration. The combination of changes is sometimes protective and adaptive, at other times, pathologic. Study of the influence of acceleration on endocrine gland functions must place emphasis on the condition of the sympathetic-adrenal system and the hypophysis which is significant in the body's reactions to various stresses [184].

A one-time application of acceleration causes an increase in the content of adrenalinelike substance in the blood, while repeated exposure to acceleration may cause a significant decrease [14, 81, 102]. No definite correlation between the organism's resistance to acceleration and changes in adrenaline content of urine has been determined [81]. According to observations, however, secretion of noradrenaline is always greater when there is high resistance to acceleration than when resistance is low. However, the secretion of catecholamines increases in both types with increasing acceleration [157].

Decreased resistance to acceleration in adrenalectomized animals has been noted [67, 163, 167] (Fig. 26) and restoration of resistance after parenteral injection of cortisone and hydrocortisone. Data on the effectiveness of the use of deoxycorticosterone acetate (DOCA) are contradictory [34, 167]. Morphological and histofunctional studies [119, 143, 162] indicate cyclical restructuring and stress on adrenal function under acceleration influence. The stressed function often leads to damage of medullary substance cells, with degenerative vacuoles, and indications of other serious damage. Following extended and repeated application of acceleration to white rats and guinea pigs, the weight of the adrenals increased and the relationship between cortical and medullary substances changed, predominantly the latter [59, 136].

The reaction of the hypophysis to acceleration is a complex, multiphasic process. Following 1 to 2 hours' spinning on a centrifuge, the content of corticosteroids in blood plasma usually increased in all animals studied [68]; subsequently, depending on the degree of acceleration, there was either further decrease or increase. In experiments with rats under 4.5 G acceleration, an increase was noted during the first hour in corticosterone concentration by a factor of 4; after 2.5 hours, the content increased by a factor of 7; longer exposure to acceleration resulted in a gradual reduction in its content. Under maximum tolerable negative G (– 25.0 G_z), an increase was noted in synthesis and secretion of hormonal substances by the somatotropic and

adrenocorticotropic cells, with almost total cessation of secretion of thyrotropic and gonadotropic cells.

In studies on rabbits and white rats under $+G_z$ acceleration, there were deep phasic changes in neurosecretory processes in cells of the supraoptic and paraventricular nuclei of the hypothalamic area. In the first phase (increase in functional stress), the antidiuretic activity of the blood plasma increased. Neurosecretions of the anterior portion of the hypophysis decreased [158], which was considered an indication of increasing concentration of antidiuretic hormone in the blood [158]. Acceleration resistance of hypophysectomized animals increased (Fig. 26) and acceleration tolerance decreased upon injection of ACTH before rotation [163].

The reactions and roles of the other endocrine glands during acceleration remain physiologically insufficiently studied. In rat experiments, removal of the thyroid increased survival rate by approximately 38% compared to intact animals, while injection of thyroidin (unpurified extract) decreased survival [167]. This change in resistance to acceleration was considered a result from change in intensity of metabolic processes, in particular, oxygen uptake. Similar data are available on the role of the thyroid gland in animal resistance to $-G_x$ [67].

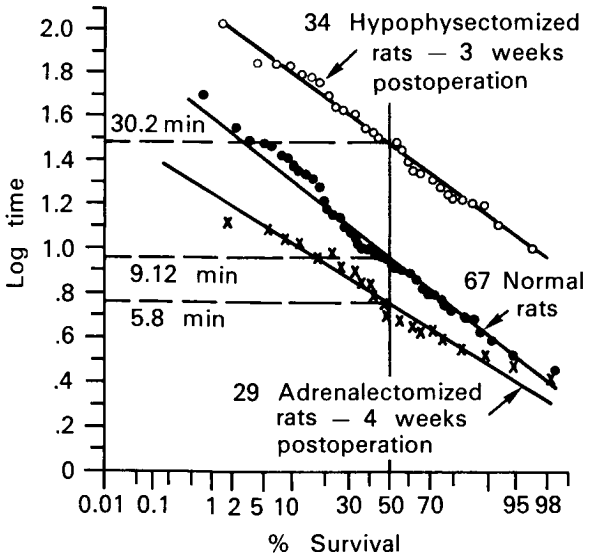

FIGURE 26.—Influence of hypophysectomy and adrenalectomy on survival rate of rats under $+20$ G_z acceleratory stress [163]. Ordinate shows logarithm, of survival time, abscissa shows survival rate, %; horizontal dashed line shows mean survival time, min.

The functional state of the hypothalamus-hypophysis-adrenal system and the other endocrine glands may change significantly under the influence of acceleration; yet, these glands are important in the organism's reaction to acceleration effects of various magnitudes and directions.

Acceleration influences blood distribution in the body, as well as indicators of morphological, chemical, and physical properties of the blood [186]. That acceleration causes hyperglycemia was established some 20 years ago [34]. In later studies with comparatively low values of $+G_z$, changes were noted in the content of acetylcholine [14, 102, 212], adrenalinelike substances [81, 119, 212], histamine [102], serotonin [102], electrolytes [14, 78], transaminase [176, 241], and a number of enzymes, proteins, and other indicators [119] in the blood. The nature and depth of these changes vary primarily with the value and time of acceleration.

There are changes in the composition and values of formed elements in animals and man subjected to $\pm G_x$ forces [14, 69, 186]. Studies on apes [132] under accelerations up to $+10$ G_x showed decreased erythrocytes and hemoglobin, segment-nuclear leukocytosis, and damage to genetic mechanisms of bone marrow cells. Similar data resulted from experiments with mice, rats, and dogs [58, 62]. In addition to changes already noted, these experiments revealed a depression in the mitotic activity of bone marrow cells and increased frequency in disruptions of the nuclear apparatus (adhesion and restructuring of chromosomes).

Different data were obtained in experiments on white mice exposed to -25.0 G_x [213]. Increases were detected in hemoglobin and in the number of erythrocytes in the experimental animals' blood, as well as pronounced leukopenia, reticulocytosis, and an 8–10% increase in polychromatophils. The leukocytic composition showed neutropenia, lymphopenia, and some eosyinopenia. There was an increase in erythrocyte volume with simultaneous decrease in the mean corpuscular concentration of hemoglobin, as well as expansion of osmotic resistance boundaries of red blood cells.

Morphologic studies of bone marrow [246] indicate activation of the erythropoietic and granulopoietic functions following $+G_x$ stress. Erythropoiesis normalizes 1.5–2.5 months later [132, 246], and after the same time, normal values of hemopoiesis in the regional lymph nodes are restored [119]. Under the influence of $-G_x$, anticoagulation properties of the blood increase (activation of fibrinolysis, increase in content of heparin, decrease in quantity of procoagulants) [187]. There is normalization of these indicators 5 days after rotation is stopped. These data are not in agreement with findings of pronounced tendency toward thrombosis in experiments with apes [14]. In spite of intense studies on acceleration influence on the blood system, unsolved problems require further experiments.

Gastrointestinal Tract

The influence of acceleration on the function of the digestive organs has not yet been sufficiently studied. In experiments on cats which were given barium sulfate with food, there was delay in evacuation of stomach contents after exposure to moderate accelerations. A direct dependence was noted between the duration of holding food mass in the stomach and the degree of acceleration [186]. Similar results were obtained in experiments on rats [34, 230] (Fig. 27).

The classical methods of Pavlov were used in experiments on dogs, with clinicophysiologic observations of humans, to study the influence of positive ($+G_z$) and negative ($-G_z$) forces on the evacuatory function of the stomach, as well as the secretory function of the salivary, gastric, and intestinal glands [208, 211]. It was established that the $\pm G_z$ inhibited hunger contractions of dogs' stomachs for 1–1.5 hours. The nature and direction of changes in secretion of digestive tract glands were similar for $+7$ and $+9$ G_z for 20 s and -3 and -5 G_z for 20 s, consisting of initial inhibition of secretion, later replaced by excitation. An increase in intestinal secretion, accompanied by an increase in the dense portion of the juice and change in amylase and phosphatase activity, were observed a few days to a few weeks after exposure to acceleration. These results were confirmed in later studies [164] on the secretory function of the stomach and the small intestine under transverse forces ($+G_x$). Changes in enzyme activity and duration of enzymatic disruption under this type of acceleration was less than with longitudinal acceleration.

Studies on humans with $+G_z$ and $+G_x$ also showed a multiphase secretory reaction – initial inhibition, followed by stimulation. Motor activity of the stomach under ± 5 G_z did not change significantly [208]. With repeated application of accelerations at intervals of 4–5 days, adaptation phenomena developed: inhibition time of salivary secretion was significantly shortened, while the dense residue in the saliva decreased less; changes in amylase content were not observed, nor secretion of acid stomach juice of the empty stomach. The primary genesis of these changes in the secretory-motor function of the gastrointestinal tract is in disruption of interrelations between tonus of the sympathetic and parasympathetic nervous system and nervous-humoral regulation [166].

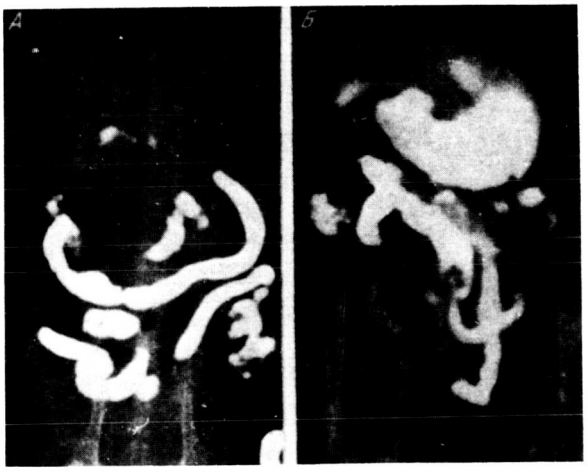

FIGURE 27. – X-ray of gastrointestinal tract in rats following $+G_x$ acceleratory stress [230]. Three hours after injection of barium sulfate: A, control animal; B, following stress.

In a study of the influence of G forces on activity of the digestive glands [37], systematic studies were first performed on the juice and enzymatic functions of the pancreas during and following $+G_x$. In experiments with dogs subjected to $+8$ G_z for 3 min, disruption of the

secretory function of the pancreas was observed: hypersecretion alternated with hyposecretion; the secretory reaction to food stimulus was distorted; and quantitative and enzymatic composition of secretions was disrupted. Restoration of the function required 4 weeks or more. Adaptive reactions of the pancreas following repeated exposure to acceleration appeared to be significant.

In experiments on dogs subjected to positive transverse forces of 4.0–10.0 G_x, there was reduction in bile released and slight increase in concentration of bilirubin and bile acids, retained for 2 weeks. These changes subsequently became uneven, and were restored to the initial level only after 50–60 d [155].

Comparatively slight accelerations cause significant changes in functions of the digestive organs, due to structural and biochemical changes [119]. The literature should probably be referred to in developing regimens and rations for feeding, particularly in the days and weeks following takeoff and landing.

Renal System

Pilots subjected to a stress of 6.0–8.0 G_z frequently manifested erythrocytes in the urine, and in some cases granular cylinders [133]. Similar results were obtained in 10% of experiments with humans [71] and animals [185] rotated on a centrifuge.

Diuresis following +2.0–5.0 G_z was studied in rats, which established that polyuria developed and was maintained for 5–6 h, and concentration of chlorides in the urine was significantly reduced [186].

Contradictory results were obtained in other experiments [198] —stresses of+3 G_z caused the quantity of urine following ingestion of water to decrease from 12.5 ml/min to 7.5 ml/min, while its specific gravity increased from 1.0008 to 1.0086. These data were confirmed with transverse acceleration; however, under these conditions, oliguria developed only after exposure to forces of +7 G_x and more.

Seven dogs were fitted individually with tapped ureters (method of Orbeli) for a study of glomerular filtration, capacity of kidneys for osmotic concentration, and liberation of sodium and potassium before and after exposure to loads of +5.8 and +12.0 G_x. Immediately after acceleration, there was a brief period of oliguria followed by increased diuresis [225]. The degree of these changes is determined largely by the value of the $+G_x$ force. Exposure to twelvefold G-loads causes increases in urine production, excretion of potassium and sodium, and reabsorption of osmotically free water. However, these changes are usually normalized after 1.2–2 h, and all indicators return to their initial levels.

The water-loading experiments indicate that ability of the kidneys to pass water does not change under repeated acceleration: the function of kidneys for hypotonization of urine, reabsorption of sodium and potassium (and as indicated by other factors) is not disrupted. Protein appears in the urine in slight quantities and disappears after 1.5–3 h. The urine precipitate contains leukocytes, epithelial cells, and fresh, unleached erythrocytes. The data would appear to indicate that the changes resulted primarily from activation of the hypothalamus-hypophysis system.

The importance of neurohormonal mechanisms in changes of kidney function was also noted in experiments with six healthy males subjected to $+4G_x$ [235]. The starting mechanism was considered to be stimulation of volume receptors of vessels in the thorax when fully filled with blood. Under acceleration, similar to the experiments with dogs, brief inhibition of diuresis (20–35 min) was followed by a slight transient increase in glomerular filtration.

Humans subjected to loads up to +10 G_x showed a slight increase in diuresis, clearance, endogenic creatinine, and erythrocytes [109]. Diuresis was attributed to changes in kidney hemodynamics and increasing glomerular filtration. Accelerations also caused various morphologic changes in kidneys of experimental animals [120] which, as a rule, were transient following single exposure to acceleration.

Hematuria following acceleration is believed to result from disruption of blood circulation in the kidneys, increase in the permeability of the capillary walls of the glomeruli, the main membrane, and internal layer of the capsule [107].

WORK CAPACITY UNDER THE INFLUENCE OF ACCELERATION

Studies of work capacity under the influence of acceleration are significant in regard to astronaut activity. During space flight, the astronaut must be in control of the spacecraft, perform diverse maneuvers during various flight stages that require rapid reactions, solve a number of problems, and maintain precise coordination of motions and physical strength. Acceleration effects may cause unfavorable organic and systemic reactions and finally be reflected in the operator's work capacity. Such phenomena relate primarily to disrupted processes of afferent perception, changes in functional state of CNS, and limitations in mobility of operating organs.

Changes in visual perception are vital in an operator's work under acceleration. Depending on the value, duration, and direction of acceleration, a delay in reaction to light may relate to the value of the acceleration, or to the period of acceleration immediately preceding grayout, or to reduced perception of contrast in visual images, particularly with low light levels and diminished ability to distinguish colors [41, 206, 238]. With sufficiently high $+G_z$ and $+G_x$ forces, deterioration in visual perception may lead to total loss of vision.

Processes of auditory perception under the influence of acceleration have received limited study. It has been established that with $+G_z$ and $+G_x$ loads, auditory perception, based on reaction time to sound, is retained significantly longer than the visual function [43, 220].

While the significance of proprioceptive and vestibular stimuli under the influence of acceleration in flight is acknowledged, this problem remains open concerning questions of work capacity. Centrifuge experiments revealed reduced ability to solve arithmetic problems and perform psychological tests, reductions in the critical flicker fusion frequency and short-term memory capacity [93], and deterioration in the results of compensatory tracking [60]. The severest disruption in brain functions, and consequently, work capacity, is under the influence of $+G_z$ forces before loss of consciousness. The condition of motor functions is important in maintaining work capacity under acceleration.

Mechanical forces make the work of the muscle groups more difficult, thereby limiting the motions of working organs. In a centrifuge experiment, large-amplitude motions and generalized locomotor acts such as walking, running, and crawling suffered the most [53]. For example, straightening the trunk becomes impossible at $+4$ G_z; while at $+8$ G_x and higher, it is impossible to raise the body and the extremities. However, motion of arms in the carpal joint was possible up to $+25$ G_x. Under these conditions, professional activities are possible only by systematic repetition of motor skills already well-learned.

Another aspect of work capacity under acceleration is determining the operator's effectiveness and reliability in a "man-machine" system, which was studied in detail [72]. An effort was made to coordinate the characteristics of a man-machine system under conditions of acceleration in order to increase the reliability of the operator's work [19]. In a system simulating manual control of the spacecraft's descent to Earth, it would be expedient to include an auditory signal indicating a mismatch with the desired return trajectory in addition to a visual signal. Such a combined system is necessary due to the possibility of vision loss at forces of $+14.0$ to $+16.0$ G_x, in order to assure satisfactory control quality up to loads of $+18$ G_x (Fig. 28).

Finally, the problem of man's working capacity under the influence of acceleration requires broad experimental studies to establish requirements for control systems to be used when the operator is subjected to long exposure to acceleration.

REACTIVITY OF THE ORGANISM DURING AFTEREFFECT OF ACCELERATION

Reactivity, in the generally accepted meaning, is the body's property of responding to the effects of the external environment. Reactivity, then, expresses the relationship (balance) between the body and the environment [2].

The secretory and motor functions of the gastrointestinal tract show, for an extended period following acceleration, alternately reduced and

increased response reaction to mechanical, chemical, and food stimuli [37, 166, 211].

The organism's reactivity also changes sharply relative to pharmacologic agents [160, 181, 224]. After extended exposure to transverse accelerations, animals exhibited increased sensitivity to cardiac glycosides (Fig. 29), narcotics, and other preparations, and decreased sensitivity to analeptics and analgesics. There are changes in the body's reactions to ingestion of radiation protectors (Fig. 30) and autonomic nervous system mediators. For example, the increase in motor activity of white mice following injection of Phenamine (Benzedrine, amphetamine) following application of acceleration is less clearly expressed (Fig. 31) than in control animals, indicating a decrease in the specific effect of this preparation. The analgesic effect of 1, 2, 5-trimethyl-4-phenyl-4-propionyloxypiperidine hydrochloride (Promedol) and certain other pain-relieving substances is reduced. Acceleration significantly changes the reaction of an animal's body to ionizating radiation [181, 248].

Preliminary application of stress (−10 G_x for 15–30 min) increases by 15–23% the survival rate of mice irradiated at a dose of 700–750 R in air [181]. Animals which were subjected to various values of acceleration and subsequent irradiation with x-rays or γ-rays, as well as high-energy protons, provided the conclusion that sequential exposure to acceleration and radiation, with intervals of 4 to 24 h between, results in an increase in $LD_{50/30}$ by 100 R in comparison to radiation alone. This is explained by an increase in activity of the hypophysis-adrenal-cortical link of the neuroendocrine regulation system under the influence of acceleration, manifested by increased catacholamines and serotonin in the blood, with increased ceruloplasmin activity and other biochemical indicators.

During the aftereffect following acceleration, there are changes in endurance of physical loads, increase in resistance to acute hypoxia and electric trauma, and a decrease in resistance to overheating and overcooling [192]. In centrifuged mice, there is definite correlation between changes in content of ceruloplasmin, biological activity of the blood, and physical endurance [7] (Fig. 32).

Immunobiological reactivity is disrupted in

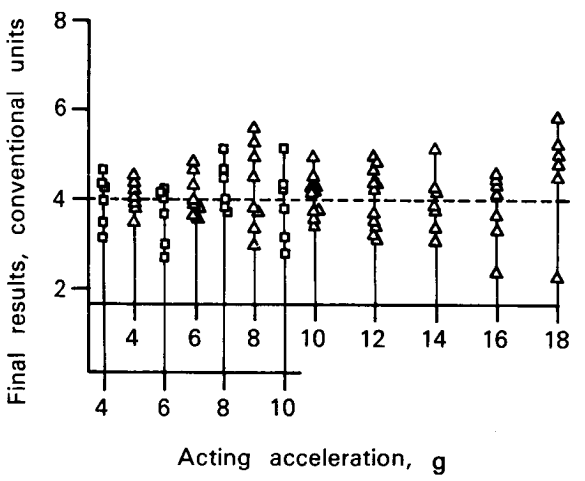

FIGURE 28.—Deviation of final regulation parameter from calculated value with $+G_x$ acceleratory stress [19]. Dashed line (----): calculated value of final result; □: value of final result using light-sound indication; Δ: value of final result using only sound indication.

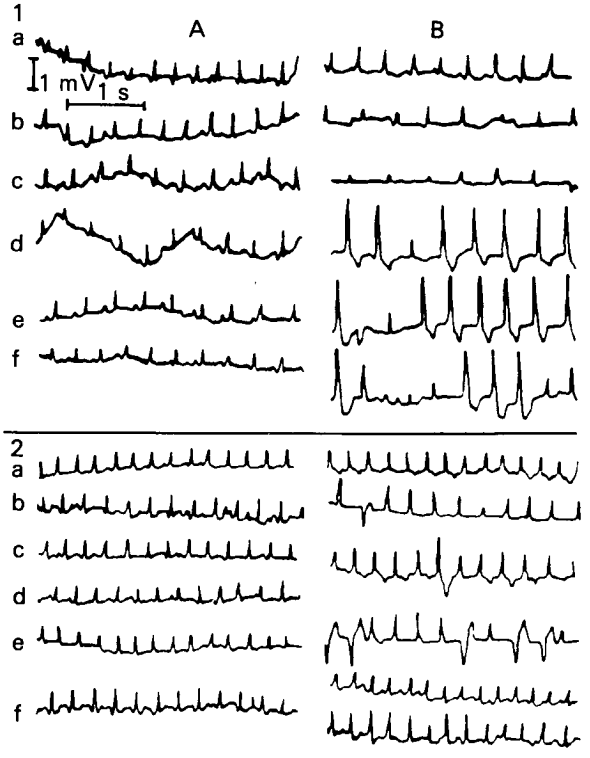

FIGURE 29.—Change in ECG of apes [2, 3] following injection of strophanthin after exposure to +12 G_x acceleratory stress [224]. A: before stress; B: 20 min following stress; a: before injection of strophanthin, dose 0.05 mg/kg body weight; b, c, d, e, f: 1, 3, 5, 7, and 15 min following injection of strophanthin, respectively.

animals subjected to acceleration [3, 188]. A clear dependence on the value and time of acceleration is noted by the nature and degree of changes in the phagocytic function of neutrophils, and bactericidal activity of the blood and saliva. With slight or brief applications of acceleration, immunobiological activity is stimulated, while greater accelerations cause its depression. For example, with forces of -9 G_x and particularly, -12.0 G_x, the absorptive function of the neutrophils and bactericidal properties of skin and saliva were reduced, thus reducing the body's resistance to pathogenic microbes. In experiments with artificially induced acute *Staphylococcus* infection, the death of mice subjected to -30 G_x acceleration was 38% greater than in the controls.

Thus, during aftereffect following acceleration, reactivity of the organism is altered, despite almost complete restoration of basic somatic and autonomic functions recorded at rest (cardiovascular system, respiration, and CNS) to their initial level. Reactivity is increased relative to some environmental stimuli, decreased relative to others, and distorted relative to still others. Knowledge of the nature and depth of possible changes in reactivity during various stages of aftereffect following acceleration, and of the incompletely understood physiologic mechanisms, is necessary for proper selection of medical tactics (recommendations and requirements, preventive and therapeutic measures).

ADAPTATION AND CUMULATION EFFECTS OF ACCELERATION

The influence of adaptive and cumulative effects of acceleration on humans and animals is of scientific and practical significance in preparing man for space flight. The literature indicates that resistance of the organism may increase following repeated exposure to acceleration [34, 71, 72, 164], which, on the other hand, may cause cumulative negative effects [78, 191].

Trained pilots can better tolerate the effects of acceleration in flying than can untrained pilots. Repeated exposure to longitudinal $+G_z$ during several days helps to increase resistance of the organism, but frequent repetition during a single day may have the opposite effect [9]. However, the literature contains as many or more reports indicating the possibility of negative effects and damage to the body following repeated exposure to acceleration.

Daily exposure of apes to acceleration for 3 weeks led to pronounced pathologic changes in kidneys [191], disruptions at the cellular membrane level [78], and dystrophic changes in walls of the cerebral vessels [102]. Extended and repeated exposure to acceleration produced damage to the right ventricle myocardium in rats and apes, which appeared in both ECG and autopsy (myocardial hemorrhage) [86, 98]. The degree and nature of disruptions described by various authors were not the same, and resulted from differing experimental conditions. Repeated application of acceleration caused increased resistance in some animals and significant decrease in others [179]. Frequent repetition of acceleration led to profound morphologic changes in the organs, but long intervals between experiments helped to decrease the pathologic changes [103].

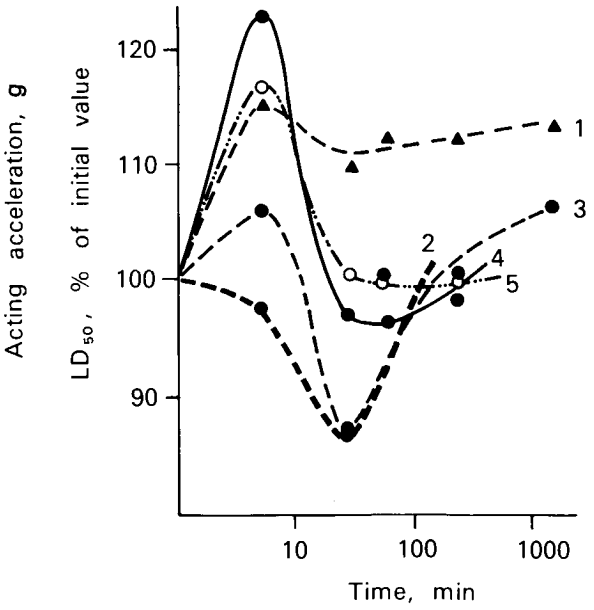

FIGURE 30.—Influence of transverse acceleration on mortality of white mice injected with toxic doses of radiation protectors [7]. 1: 5-methoxytryptamine (-10 G_x, 15 min); 2: s-β-aminoethylisothiuronium bromide hydrobromide (-10 G_x, 15 min); 3: cystamine (-10 G_x, 15 min); 4: cystamine (-30 G_x, 15 min); 5: cystogen (-10 G_x, 15 min).

The duration of acceleration is considered highly significant. For example, it was shown that forces of +8.0 to +12.0 G, lasting up to 15 s, caused no cumulative effects in dogs, even though repeated daily for 26 d [239]. However, when these accelerations were applied for a longer time, significant deterioration in the animals' condition resulted. A combined study of body processes in dogs under the influence of $+G_x$ forces (3.0 to 12.0) applied repeatedly revealed indications of adaptation reactions [112]. Upon repeated application of acceleration, changes in the dogs' respiration, energy expenditures, and hematological and biochemical indicators showed development of adaptive reactions. However, pathomorphological studies indicated structural disruptions in various organs and tissues [106, 108, 119, 120]. Consequently, with repeated exposure to $+G_x$, various effects are adaptive, but on the other hand, clearly show cumulative damage [112].

Thus, incomplete data in the literature do not indicate any conclusive agreement on aftereffects of repeated acceleration. While data indicate the possibility of adaptational changes, possible cumulative negative effects must also be acknowledged. Findings indicate manifestation not only of functional changes, but also of significant structural damage to tissues. However, various body changes doubtlessly result from the modes used in acceleration: value, direction of vector, duration, gradient of increase, frequency of repetition, and intervals between application. It is important to clarify the various aspects of this problem for the development of systems to train man, prior to space flight, to tolerate acceleration.

In designing a system of training on the centrifuge, it is not feasible to draw a complete analogy with sports training. Under acceleration, an abnormal factor acts on the body, while in sports training the organism experiences elevated loads—for which it is adequately adapted through evolution. The solution of this problem requires an approach that must take into consideration the establishment of optimally tolerable acceleration modes, repetition frequencies, individual resistance, and conservation of training effects.

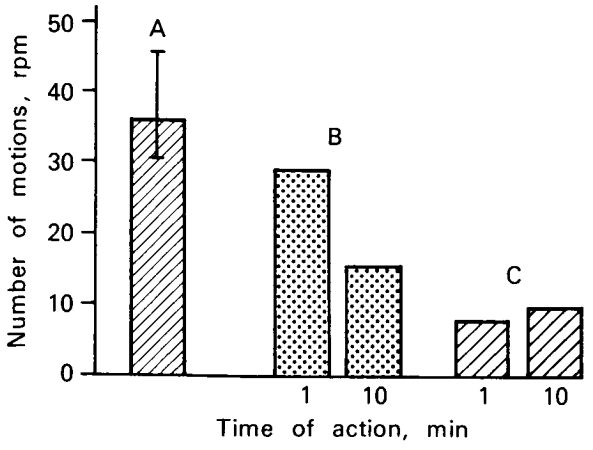

FIGURE 31.—Motor activity of white mice following injection of Phenamine, dose: 10 mg/kg [224]. A: Control animals; B: following exposure to -10 G_x acceleratory stress for 1, and 10 min; C: following exposure to -40 G_x acceleratory stress for 1, and 10 min.

MECHANISMS OF THE INFLUENCE OF ACCELERATION

Acceleration causes functional and morphologic changes in various systems in the body.

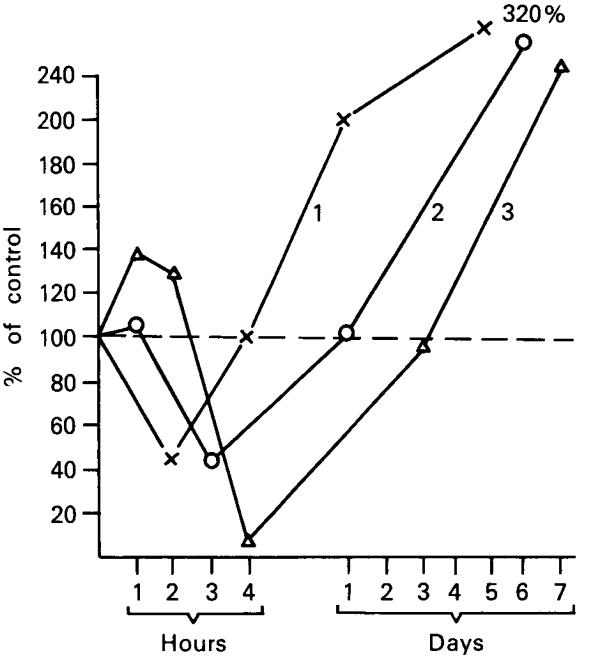

FIGURE 32.—Comparative data on changes in activity of ceruloplasmin. 1: Biological activity of blood; 2: mean effective floating time; 3: following application of acceleratory stress [7].

The most important direct effects of acceleration are blood redistribution in the vascular system, lymph flow impairment, organ displacement, tissue deformation, disruption in respiration, and stress reactions. The specific significance of these primary acceleration mechanisms will change depending on the direction of the acceleration vector. Thus, during longitudinal acceleration, circulatory hypoxia of the brain is the most significant development. Application of acceleration produces visual disorders and when continued or intensified, loss of consciousness and convulsions. At the same time, a decrease is noted in the blood flow to the heart, causing it to overwork.

During transverse acceleration, respiration difficulties are important in the mechanism of disruption. Hypoxemia also develops as a result of strong pressure on the thorax, as well as disruption of blood oxygenation in the lungs from hemodynamic disorders. When the heart cavity and blood vessels of dogs were probed [104, 232, 243] after forces of +2.0 to −9.0 G_x, it was found that venous blood is first pooled, then shunted to pulmonary veins, and oxygenation disrupted, causing reduced volumetric content of oxygen in arterial blood. Consequently, respiratory disruption and hemodynamic disorders lead to hypoxemia and hypoxia of such important organs and tissues as the brain and heart [121, 197]. The unusual flow of afferent impulses facilitates development of central regulatory disorders, which in turn reduces the body's compensatory-adaptive capabilities. Changes in the endocrine glands resulting from disruptions in CNS regulatory activity and hypoxia cause still greater disorders in the body's various systems.

The primary components of the mechanism by which acceleration influences the body's functional systems are schematically presented in Figure 33. Accelerations sometimes become extremely irritating, severely disrupting normal vital processes with a traumatizing or even destructive effect on biologic objects.

Two phases of reaction to G forces have been distinguished [175]: (1) compensatory changes in the body in which primarily the vital functions are reinforced, while some are adaptively suppressed temporarily and others somewhat disrupted; (2) decompensation, in which there is depression and suppression of vital bodily systems.

During the first phase, there are increases in heart rate, blood pressure, volume of blood pumped per minute, regional blood flow, pulmonary ventilation, oxygen uptake, and even an increase of oxygen tension in brain tissues. At the same time, endocrine gland activity is

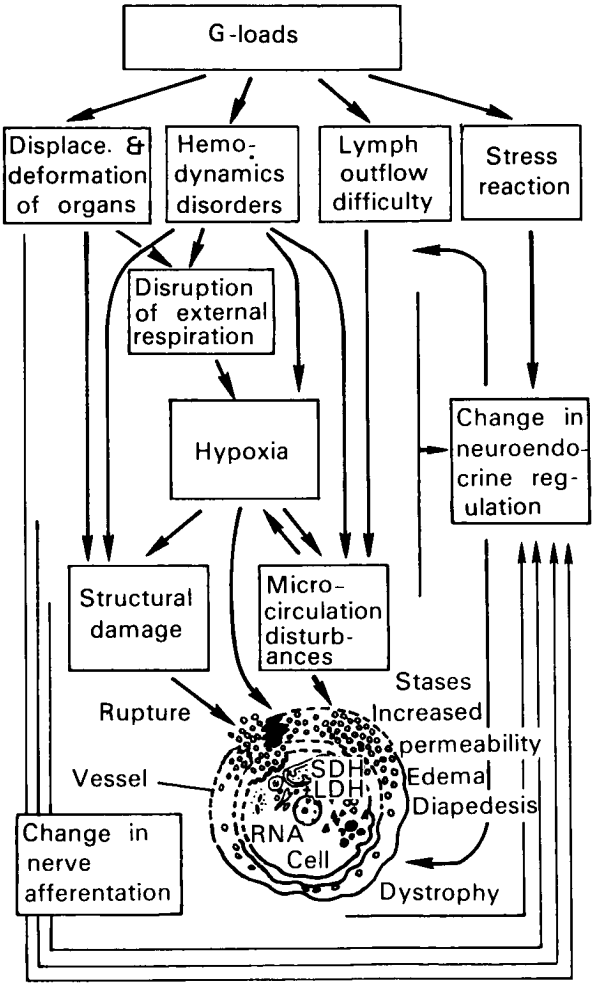

FIGURE 33.—Basic mechanisms of acceleration action on the organism [120]. Changes in the cell: increased activity of lactate dehydrogenase (+LDH); reduction in activity of succinate dehydrogenase (−SDH); reduction in quantity of ribonucleic acid (−RNA) in cytoplasm.

strengthened as judged from an unmistakable change in peripheral blood composition, increase in cholesterol content of blood, and other indications [119, 246]. Changes in the volume of histamine, adrenaline, acetylcholine, potassium, and sodium as reactions to slight and moderate accelerations are also believed to be indications of adaptive reactions [14].

The second phase, interruption of compensation, is manifested by relative or absolute bradycardia, disruption of heart rhythm and conductivity, drop in blood pressure, disruption in redox processes of vital organs [197], regulatory disorders of nervous and endocrine systems, and so forth. These lead to extreme functional and morphologic changes not only at the body, organ, and tissue levels, but also at cellular and subcellular levels [119, 120]. Long-term exposure to high G forces may cause death, usually as a result of cardiovascular or respiratory insufficiency. Another phase of nervous and emotional stress has been suggested [210]: increases in frequency of pulse and respiration, cardiac minute volume, EEG desynchronization, and so forth, immediately before acceleration application, and an aftereffect phase of restoration and normalization of functions. Other models have been described in the literature, establishing relationships, sequence, regularities, and periodicity of the organism's reactions to acceleration effects.

METHODS OF INCREASING RESISTANCE TO ACCELERATION

The search for methods to increase the body's resistance to long-term acceleration has become particularly important since maneuverability of aircraft is now limiting the body's physiologic tolerance.

The rapid development of rocket technology and space flight requires further investigation of anti-G-load measures. The development of effective methods would affect the mode of spacecraft injection into orbit by more fully utilizing the power of rocket engines, increasing the working capacity of crewmembers during the injection into orbit and descent to Earth, and providing more reliable protection in emergencies. These are particularly important in the return of spacecraft to Earth and landings on other planets following extended periods of weightlessness, which may cause pronounced disorders in blood circulation, general asthenization of the body, and other problems.

Increased resistance to accelerations is being sought a number of ways:

1. Mechanical methods
   - anti-G compensating suits
   - special seats for maintenance of optimal position to the acceleration vector
   - individual contoured supports
   - immersion systems
   - breathing under elevated pressure
2. Physiologic methods
   - physical training (specific, nonspecific), general toughening of the body, centrifuge training
   - adaptation to hypoxia
   - changing body reactivity through pharmacological agents and cooling (hibernation)
3. Combined methods

There are advantages and drawbacks to each of these methods.

Anti-G suits (*G-suits*). The primary pathogenic link during $+G_z$ acceleration is displacement of blood from the head and upper sections of the trunk to vessels in the abdominal cavity and lower extremities. The first G-suit models were designed in the early forties to hinder such redistribution of blood under the influence of inertia. G-suits in use at present operate by creation of counterpressure on portions of the lower half of the body. As accelerations increase, pressure in the rubber chambers surrounding the stomach and legs is automatically increased: the greater the G forces, the higher the pressure in the suit.

A detailed physiologic explanation of methods for increasing resistance to accelerations is not necessary since there are many such studies [72, 73, 95, 186, 189, 219, 240]. The use of G-suits has been effective in maintaining many of the body's functions. Arterial pressure in the carotid and brachial arteries is maintained at a higher level, the influx of blood to the brain and heart is improved, and fewer changes are observed in

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vision, bioelectric activity of the myocardium, conditioned-reflex activity and energy expenditure, and so forth. Results from testing in centrifuges, as well as during aircraft flights using various criteria of evaluation have shown that man's resistance increases by 0.8–1.3 G_z when such suits are used [127, 128].

Modern G-suits and improved methods for their use can increase resistance by 2.0–2.5 G_z [42]. In spite of many design studies, further improvement in the effectiveness of G-suits is not likely; at present, they are used primarily for $+G_z$ acceleration. However, the protective influence of G-suits, including decrease in visual disorders, is effective for transverse accelerations of up to $+15.0$ G_x [48]. Other methods of increasing acceleration tolerance are being investigated.

Seat back angle inclination and subject position. The greatest tolerance for acceleration is observed when it is applied transversely to the main blood vessels in the human body (noted previously). Special studies to find the optimal position of a subject under acceleration [31] have established that strict observance is required to the relationship between inclination of the seat back and head support and acceleration vector, as well as between hips, shins, and trunk. The most significant variable is in the trunk and head position. Physiologic studies have established that the optimal inclination of the seat back is 78–80° with regard to the acceleration vector [72, 117, 234].

A tolerance of $+12.0$ G_x with the trunk position inclined to the horizontal plane at 80° was 2–3 times greater in application time than with 65° inclination position [17]. Cardiovascular and respiratory reaction was less intensive in the former case, allowing a higher body resistance as a whole.

Contoured supports. Individualized contoured supports (Fig. 34), which provide a large area of counterpressure, can increase tolerance to 25.0 G_x when the body is in the optimal position in regard to the acceleration vector [48].

Breathing pure oxygen. Respiration and circulation disorders leading to hypoxemia and hypoxia are leading pathogenic factors resulting from transverse acceleration. Experiments on animals and humans have tested the effectiveness of breathing pure O_2 at normal and elevated pressures. If test subjects breathe pure O_2 or a gas mixture under excess pressure, there is a significant increase in acceleration tolerance [12], which is a result of improved gas metabolism in lungs and, consequently, oxygen deficiency is prevented.

Optimal excess pressure for the gas mixture [234] is 2–3 mm Hg/G unit. This helps to prevent changes in respiration indicators and increases tolerance time by 67%, although chest pains do not decrease significantly. On the other hand, some authors are skeptical that breathing under increased pressure will heighten resistance to acceleration. Breathing pure O_2 while undergoing acceleration may cause adverse aftereffects, similar to oxygen poisoning [131].

Immersion systems. There is substantial interest in immersion systems developed by Tsiolkovskiy [217] as a means of increasing acceleration tolerance. Experiments with white mice have produced exceptionally favorable results [149]. When immersed in water, they tolerated forces up to 1300 G for 60 s. Similar data were produced by other researchers [24]. Experiments have also shown that immersion of human subjects in a water tank can significantly increase resistance to acceleration [241]. Effectiveness is determined to a great extent by depth of immersion. Human subjects immersed to the third rib reached accelerations of $+16$ G_z, whereas under ordinary conditions loss of peripheral vision began at 3.25 G_z [48, 82]. When totally immersed in water in a special container

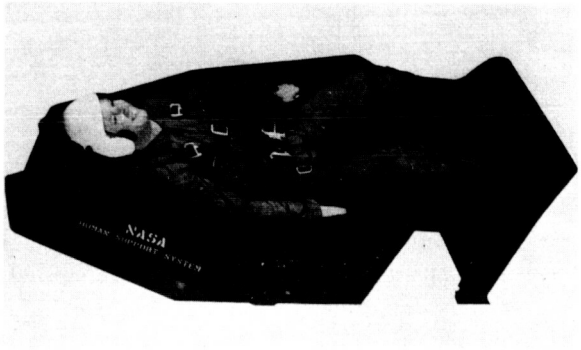

FIGURE 34. – Position of human subject in individual contoured support [48].

(Fig. 35) using special breathing apparatus, resistance to gravitoinertial forces was increased to +26.0–31.0 G, although stomach pains and slight damage to the frontal sinuses were noted. A significant increase in resistance to acceleration from immersion has been corroborated in other experiments [30].

In spite of the great effectiveness of this method, its practical use in modern flight is impossible due to the great complexity and cumbersomeness of immersion systems. Furthermore, placing the pilot in a container filled with liquid sharply reduces his capability to observe and control the spacecraft. Thus, although protection from acceleration by hydraulic systems is valid, other methods to solve this complex problem must be sought. Although some effective methods of protecting the body from acceleration effects have been developed, all physical methods of increasing resistance developed so far depend upon complex devices. Consequently, physical methods using the body's reserves have been studied by means of training, stimulation, and medications.

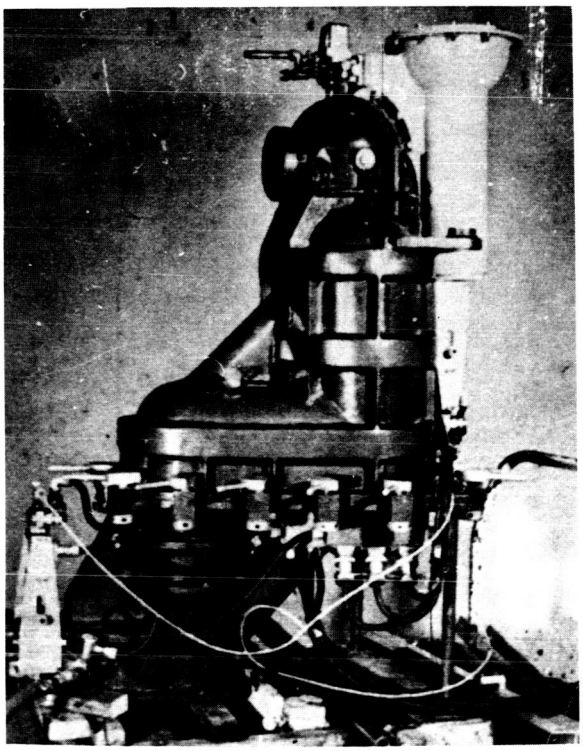

FIGURE 35.—Container used for immersion protection of man from acceleration [48].

General toughening, physical training, and adaptation to accelerations. From observations of many flight physicians, general toughening of the body by special physical exercises to improve the regulatory mechanisms of circulation and respiration, strengthening of abdominal and leg muscles, development of skills in shifting from abdominal respiration to chest type and vice versa, and extended tonic tension in groups of muscles can increase pilots' acceleration tolerance [12]. In studies of sportsmen, it was found that gymnasts, boxers, weight lifters, and sprinters were most resistant to $+G_x$ accelerations [203]. These sports require, by submaximal and maximal work intensity, strength and speed-strength exercises, and a broad range of complex coordinated motions. Data [57] indicating that physical training to develop resistance to acceleration has no positive effect are correct only for specific conditions reproduced in centrifuge testing (total muscular relaxation of subjects). However, it is believed that muscular tension can increase resistance to acceleration, and therefore, regular physical training is probably an important part of the training of both pilots and astronauts.

An increase in the body's acceleration resistance can also be achieved by centrifuge training [11, 34, 71, 142]. Systematic training on the centrifuge can increase man's tolerance by approximately 1.5–2.0 G [32]. In experiments on dogs [202] using physiologic, morphologic, and histofunctional investigation methods, it has been established that with the use of training regimens based on repetition and gradually increasing loads, plus warmups and maximal and submaximal loads, tolerance for transverse acceleration can be significantly increased. In studies with human subjects, centrifuge training using the optimal plan helps increase tolerance by 1.6–5.8 G_x. The effect of training was retained for at least 6 months. Histomorphologic indications of the degree of training for acceleration were also studied [161, 169]. The literature indicates that in addition to training mechanisms for adaptation, exposure to great acceleration for long periods may cause pathologic changes in the body [119, 162].

Adaptation to hypoxia. One of the primary pathogenic mechanisms in $\pm G_x$ forces is oxygen deficiency and hypoxia in the circulatory mechanism. [21, 104, 113, 159, 232]. Therefore, adaptation to hypoxia has been successfully used as one of the nonspecific methods for increasing body resistance to acceleration [114, 194, 227]. Adaptation to hypoxia has been performed both in barochambers and under high mountain conditions.

Animal experiments have convincingly proven that the survival rate of adapted white mice, rats, and guinea pigs under high acceleration was 1.5–2 times greater than the survival rate of a control group. Active adaptation of the animals to hypoxia by systematic physical training was found to be more effective than passive exposure at high altitude. Disruption in the cardiac activity of experimental animals, shown by electrocardiography, began later and was less pronounced [218].

Studies of humans have established that after a stay in the mountains, resistance to $+G_x$ stress of individuals previously unexposed to hypoxia was increased by 2.4 ± 0.2 G_x [114, 227]. The increased resistance of those thus trained, was retained, for the first time, up to 3 months, and significantly longer in mountain climbers who spent vacations in the mountains each year. This method of increasing acceleration resistance might also prove promising for extended space flight [124, 134, 226, 229].

Pharmacologic agents. Pharmacologic agents for increasing acceleration endurance are currently being studied. Although the first experiments started more than 20 years ago [34, 35, 130], such usage was not developed until recent years. In aircraft flights it is not always possible to predict the precise time acceleration begins, which is important for the use of pharmaceuticals, and the specific effects of a number of preparations prevent their use in flight medicine (soporifics, narcotics, phenothiazine series preparations, and so forth). A number of these limitations is eliminated in space flights since spacecraft can be controlled automatically without astronaut participation.

Medicines studied for action on the resistance to acceleration are in these pharmacologic groups: vasoconstrictors [34, 88, 130, 160, 182, 224], vasodilators [129, 183, 224], ganglioplegics [54, 134, 224], narcotics [160, 224], hormones [167, 185, 204, 224], CNS stimulators [34, 183, 224], and other substances [72, 224]. Positive effects resulted from various medicines.

The most favorable results have been obtained with Lucidril (meclofenoxate) and strychnine, sympathomimetic amines, and certain combinations of these substances. Pharmaceuticals (strychnine + caffeine + phenamine) given to test subjects before revolving on the centrifuge after long-term (70–100 d) hypokinesis restored their lowered resistance to acceleration to the initial level or even above it [113, 227]. Thus, pharmaceuticals may be not only effective, but also in certain cases necessary, particularly during descent after extended flights and weightlessness. A complete summary of pharmaceutical influence on human and animal resistance to acceleration (as well as to other spaceflight factors) is in the monograph of Vasil'yev et al [224].

Hypothermia

Deep cooling, in order to save the body under spaceflight conditions, particularly in emergency situations when various factors including acceleration may reach extreme values, is a subject that has received increasing attention recently [27, 29, 44, 226]. A pronounced protective effect was observed in rats cooled to 5–20° only for accelerations over 30.0 g; for accelerations of 20.0 g and less, cooling caused reduced resistance [205]. This difference in the effects of hypothermia is explained by the different mechanisms of animal deaths during different values of acceleration. The positive effect of artificial hibernation in rats, by reducing body temperature to 24–22°, was observed [215]. In these experiments, the survival rate in the experimental group during accelerations of -31 G_x was twice that of the control group. Similar results have been obtained in other studies [61].

Combined methods. The combined application of anti-G forces is the most effective method. In aeronautics, this principle has been used by selecting the proper seat position for the pilot, use of voluntary muscular tension, and the anti-

G suit. The combined application of protective methods against acceleration can be further developed, and effectiveness greatly increased. For example, in experiments on white mice and hamsters, water immersion combined with narcosis and deep cooling of the body increased acceleration tolerance to as much as 1800–2300 g [29]. Water immersion combined with optimal position achieved human acceleration tolerances up to 32.5 g [48]. A comparatively simple means, such as selecting the optimal inclination of the seat back, contoured supports, and respiration under increased pressure, achieved an acceleration tolerance of $+26.5\ G_x$ with a rise rate of 1g/s [17].

These data prompt anticipation that further development of anti-G forces methods will lead to future achievement of more effective protection from acceleration.

PROBLEMS AND METHODS OF FURTHER STUDY

Acceleration, an integral part of space flight, may cause adverse effects in various systems of the body under certain situations. A pathogenetic mechanism primarily producing these disorders must be considered the hypoxic and circulatory form of oxygen starvation.

Man's resistance and his work capacity are of great concern under long-term acceleration. Resistance and work capacity depend on the interaction of a combination of factors, which are not inherently comparable and consequently, can be evaluated only with considerable caution [72].

Sufficient information is not available on permissible limits for a healthy man to maintain work capacity under the influence of various modes of acceleration. Such information is lacking completely for individuals with variations in health.

Further studies are needed for objective criteria to evaluate resistance and for standardization. Objective criteria for longitudinal $+G_z$ forces should probably be the state of regional circulation at head level (in the ear) and the EEG; for $+G_x$ stress, the level of oxygenation of blood and development of bradycardia should be the criteria. Fatigue in connection with acceleration deserves attention, which is particularly important in flight landing, when various environmental factors may cause functional changes.

The effects of lateral $+G_y$ forces have not been sufficiently studied. With proper support, tolerance for $\pm G_y$ or $-G_x$ might be no worse, and respiratory disruption might be less, than for $+G_x$ force vectors ordinarily used.

Studies are needed on a centrifuge with variable radii. The significance of short-radius centrifuges for creating artificial gravity has not been established. Technically, this type of centrifuge is most suitable for solving the problem of acceleration effects in space flight. Theoretically, evaluation must be made of the pressure gradient which arises along the body during rotation in a centrifuge with short radius.

The effects of slight accelerations during long periods must also be studied. Physiologic effects, and resistance and working capacity should be determined under loads of 2.0 G during many days and perhaps months.

Studies should be continued on the effects of various acceleration rise rates. There are probably different optimal rise rates for long-term acceleration, for peak-type acceleration modes, and plateau-type modes. There are apparently optimal combinations of rise rate and relief rate, peak and plateau values, which can assure higher resistance of astronauts to accelerations expected under actual flight conditions. These combinations have not yet been determined. Studies on reactions of cardiovascular and respiratory systems should be continued. Broad investigations are needed on the influence on acceleration resistance of varied composition of the gas media used (or which has been suggested for use) in spacecraft cabins.

The positive effect of breathing oxygen with increased content, or under excess pressure, requires explanation of practical problems such as determination of the optimal time for applying this protective measure, and the values of positive pressure.

Information should be obtained on man's resistance to the effects of $+G_x$ loads in various modes (value, rise rate), as well as combinations of various types of accelerations. This is important when considering the possibility of accidental

rotation of a spacecraft. There should be further studies to develop methods of protection, mechanisms of physiologic reactions at both cellular and subcellular levels, and treatment methods for pathologic disorders. Study of the combined influence of acceleration with other flight factors is a pressing task for physiologists, biochemists, morphologists, and other specialists. Further studies should, hopefully, supplement insufficient information on acceleration influence on the human body, data that will prove highly beneficial for space flight.

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