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MECHANISM OF SELECTIVE LESION OF THE CARDIOVASCULAR
SYSTEM IN PSYCHO-EMOTIONAL STRESS

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(NASA-TM-76035) MECHANISM OF SELECTIVE LESION OF THE CARDIOVASCULAR SYSTEM IN PSYCHO-EMOTIONAL STRESS (National Aeronautics and Space Administration) 14 p
HC A02/MF A01 CACL 06S G5/52 28018 N80-28045 Unclas

Translation of "Mekhanizm izbiratel'nogo porazheniya serdechno-sosudistoy sistemy pri psikhoemotsional'nom stresse," Vestnik akademii meditsinskoy nauk, SSSR, Vol. 8, 1975, pp. 71-76.



NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON, D.C. APRIL 1979

STANDARD TITLE PAGE

1. Report No. NASA TM-76035	2. Government Accession No.	3. Recipient's Catalog No.
4. Title and Subtitle MECHANISM OF SELECTIVE LESION OF THE CARDIOVASCULAR SYSTEM IN PSYCHO- EMOTIONAL STRESS		5. Report Date April 1980
		6. Performing Organization Code
7. Author(s) Yu. M. Repin and V. G. Startsev, USSR Academy of Medical Sciences Institute of Pathology and Therapy		8. Performing Organization Report No.
		10. Work Unit No.
9. Performing Organization Name and Address Leo Kanner Associates Redwood City, California 94063		11. Contract or Grant No. NASW - 3199
		13. Type of Report and Period Covered Translation
12. Sponsoring Agency Name and Address National Aeronautics and Space Adminis- tration, Washington, D.C. 20546		14. Sponsoring Agency Code
15. Supplementary Notes Translation of "Mekhanizm izbiratel'nogo porazheniya serdechno- sosudistoy sistemy pri psikhoemotsional'nom stresse," Vestnik akademii meditsinskoy Nauk, SSSR, Vol. 8, 1975, pp. 71-76. N 76-12 713		
16. Abstract A species predisposition to hypertensive and ischemic heart disease occurs in mammals only at the level of primates, and is associated with social regulation of biological reactions. The specific physiological mechanism giving rise to psychoneurogenic pathology may be an inhibition of the motor component of the aggressive-defensive response. Repeated combination of pursuit with subsequent immobilization resulted in 4 out of 5 experimental baboons developing serious arterial hypertension and ischemic lesion of the heart which lasted many years.		
17. Key Words (Selected by Author(s))		18. Distribution Statement This copyrighted Soviet work is reproduced and sold by NTIS under license from VAAP, the Soviet copyright agency. No further copying is permitted without permission from VAAP.
19. Security Classif. (of this report) Unclassified	20. Security Classif. (of this page) Unclassified	21. No. of Pages 14
		22.

MECHANISM OF SELECTIVE LESION OF THE CARDIOVASCULAR SYSTEM IN PSYCHO-EMOTIONAL STRESS

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Sufficiently detailed and thorough study of the causes, conditions, and mechanisms of causation and development of psychoneurogenic cardiovascular pathology is possible only in experiments. Analysis of the extensive factual material amassed by experimenters has permitted us to draw methodologically material conclusions about the lack of a specific predisposition in traditional laboratory animals to arterial hypertension or ischemic disease of the heart as neurogenic nosologic units: in the best case it was possible to obtain only more or less transitory responses in dogs as a physiologic expression of defensive excitation (V. A. Gavlichek, 1952, 1962; A. V. Napalkov, 1952; V. N. Chernigovskiy and A. Ya. Yaroshevskiy, 1952) or destructive changes in the myocardium as a result of unnatural injuries to this or that region of the nervous system (S. V. Anichkov et al.).

We believe that hypertensive disease and ischemic disease of the heart, as psychosomatic nosologic forms, arise only at that stage of mammalian evolutionary development (at the level of lower primates) when, in connection with social regulation of biological behavioral reactions, a qualitatively new type of psychosomatic interaction is created as a structural-physiological substrate of these illnesses. Such an idea finds a basis, on one hand, in the difficulty (or impossibility) of producing chronic psychoneurogenic cardiovascular diseases in dogs, cats, rabbits, or rats, even as the result of "extraordinary" traumatizing influences on the nervous system and where there is a lack of any kind of convincing data about the capability of these animals to develop natural diseases through nosologic forms similar to hypertensive or ischemic cardiac diseases. On the other hand, there are convincing facts about the predisposition of gregarious apes, primarily baboons, to develop spontaneous lesion through myocardial infarct without

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

atherosclerotic changes in the coronary vessels (Groover et al., 1963; Groover and Stout, 1965), coronary insufficiency and arterial hypertension during the prolonged influence of emotional and stress factors (G. O. Magakyan, 1953, 1965; A. A. Fufacheva, 1965) or as a result of experimental neurotization (G. O. Magakyan, 1965; D. I. Miminoshvili et al., 1956; Yu. M. Repin and V. G. Startsev, 1971, 1972, 1973; V. G. Startsev et al., 1972, etc.). Neurogenic neurotic hypertensive disease and ischemic disease of the heart in hamadryad baboons, in a clinical-morphologic regard, are similar in principle to such diseases in humans (B. A. Lapin and L. A. Yakovleva, 1960; G. O. Magakyan, 1965). In our opinion, the simultaneous origination at one stage of evolution of both social forms of intragroup interrelationships of apes in a hierarchially organized society and a specific predisposition to psychosomatic cardiovascular pathology could hardly be fortuitous. A study of the cause and effect relationships between these specific features of lower primates is likely to permit us to approach an understanding of the concrete physiological role of the social moment in the etiology of hypertensive disease and ischemic disease of the heart.

In one of his lectures on the physiology of the blood supply, I. P. Pavlov, recalling the well-known statement of I. M. Sechenov about the link between the diversity of brain activity and muscular contraction, remarked: "One cannot imagine any kind of beast lying about and seething for hours without any kind of muscular manifestation of his anger . . . When, for example, a lion is angry, his emotion immediately takes the form of fighting, while the rabbit's fear immediately translates into muscular activity of another kind - into flight, etc."¹ This statement by the great physiologist wonderfully characterizes the essence of the psychosomatic mutual interactions in animals with pre-primate organization of higher nervous activity - automatic, direct motor expression of emotional reaction. With regard to gregarious apes, it is impossible to imagine even a very hungry baboon taking a delicacy offered to him without the permission of the leader, or a male rabbit attempting to possess a female from the harem of a higher-ranked male, etc. It is even more complicated in humans: ". . . now we can express anger, despair, and various other feelings without producing various kinds of

¹I. P. Pavlov. *Pcl.sobr.soch.*, vol. 5, USSR Academy of Sciences Press, Moscow-Leningrad, 1952, pages 331-332.

movements in the musculature . . ."².

Social regulation of behavioral stereotypes in gregarious apes manifests itself primarily through arbitrary inhibition in an animal of internal, i.e., motor, components of the feeding, sexual or aggressive-defensive reaction, if they do not correspond to "social" subordination. A systematically repeated break in a biological reaction at the stage of its effector completion creates a new kind of psychosomatic interrelationship and is the genesis of a potentially pathogenic situation of the "fixated" (P. K. Anokhin, 1965), "repressed", and "inhibited" (G. F. Lang, 1950; A. L. Myasnikov, 1954) emotional type. In our opinion, herein lies the essence of the vaguely treated "anatomy-physiological proximity to man" (B. A. Lapin and L. A. Yakovleva, 1960) as a methodological prerequisite to the use of apes in medical and biological experimentation. /72

In the 1950s, extensive notariety was achieved by experiments of D. I. Miminoshvili et al. (1956) on the production in apes of cardiopathogenic natural neuroticizing situations based on disturbance of group and sexual relationships. While such conflicts are externally effective and similar to human ones, they turned out to be of little effect for producing cardiovascular pathology, if you consider primary sources: the sole documented result was inversion of the T-wave on the EKG from the standard leads of one ape (Zeus hamadryad baboon). Repeated experiments using D. I. Miminoshvili's method on a large number of apes, with systematic presentation to the animals of more prolonged, varied and clearly emotionogenic conflicting situations over the course of many months (Sh. L. Dzhalagiya, 1960, 1972, 1973, 1974), did not lead to arterial hypertension or ischemic changes in the heart in a single case. The use by these investigators of "errors", i.e., the replacement of practically uncontrolled conflicting situations with disordered stimulation of sexual, or aggressive-defensive, or feeding emotional excitement, did not facilitate, in our opinion, but rather impeded the origination of a psychosomatic pathology, since the psychosomatic effects of neurotization was immediately "dispersed" among several visceral systems. Moreover, a similar setup of experiments made it

²Ibid.

impossible to separate and analyze the primary and concrete psychosomatic mechanism, which may be a cause-and-effect factor in a vegetosomatic disorder. We are proposing that the search for the cardinal mechanism of visceral system lesion during psychoemotional stress in apes must result in simpler and more strict experiments.

In our previous experiments (V. P. Startsev, 1971, 1972) on the production in apes of neurotical visceral pathology - gastric achylia with precancerous complications, a diabetic condition, sexual disorders, etc. - a large group of facts were obtained which permitted us to formulate our own conception of the principal mechanism of visceral system selective lesion as a result of neuroticizing emotional stress without appealing to the speculative "place of least resistance" or "constitutional factors": the injurious effect of neuroticizing emotional stress is selectively localized in that visceral system whose dominant function is repeatedly cut off by the same stress factor and, consequently, the natural stimuli of which acquire the importance of conditional signals in the stress situation.

Since there is not a single physiological activity which does not contain such a powerful hemodynamic component as aggressive-defensive behavior or emotional forestalling preparedness to it, we might suppose that interruption of the motor component of the dominant aggressive-defensive stimulus may be a concrete psychosomatic mechanism of selective lesion of the blood supply system apparatus during emotional stress. Therefore, our method of producing cardiogenic psychoemotional stress in apes proposes the creation of dominant natural aggressive-defensive stimulation and interruption of the motor component of the flight or defense reaction to a predicted danger. In such a situation, the impossibility of reaching the result of an action and, consequently, the impossibility of forming a sensory correlate for the acceptor of the action must lead to extreme stress on the mechanisms of negative emotion and imbalance in the functional system (P. K. Anokhin, 1968) of aggressive-defensive behavior in the form of a conflict between relative hypokinesia of the sensory-motor apparatus and mobilization of the hemodynamic and neurohumoral system for ensuring maximal motor activity.

We shall cite the results of a five-year experiment begun by us

in early 1969 and continuing to the present time. Five young and healthy hamadryad baboons were compelled for five minutes to save themselves from the danger of being seized by fleeing, and then, at the moment of maximal activation of aggressive-defensive behavior, they were subjected to immobilization on a board for 2-4 hours. Prior to this, it had been established that neither repeated prolonged pursuit (entrapment) nor sessions of immobilization, when used separately, were capable of causing any kind of stable shifts in arterial pressure or EKG in healthy baboons. However, as a result of 5-time combination of pursuit with subsequent immobilization, utilized in sequential experiments once a day, pronounced pathological changes in the cardiovascular system developed in four out of five experimental apes in a later period. Over the course of the first 3-4 months during the aftereffect, a stable (and continuing) increase in main arterial pressure to 100 mm and higher was observed along with an increase in heartbeat frequency during physical calm which was comparable with tachycardia during pursuit. Ischemic shifts in EKG components, recorded initially only from the standard and unipolar leads from the extremities, and then taken from the chest leads as well (Figure 1), developed on a background of a hyperdynamic condition of the blood supply system. Following a second analagous stress influence - pursuit in combination with subsequent immobilization repeated five times, which was undergone 10 months following the first application - serious arterial hypertension (Figure 2) developed in the same animals, and pathological shifts in EKG became more serious and stabilized, approaching the typical picture of a pre-infarct condition. Without any kind of additional special influences, the disease lasted more than five years, never displaying a tendency toward remission and accompanied by a gradually deteriorating overall condition of the animal (Figure 3). The latter is manifested, in particular, as obesity in the ailing apes, a decrease in their motor activity, and dyspnea following minor physical exertion. Hypertrophy of the left regions of the heart and disturbance of myocardial contractile function were determined Roentgeno-kymographically. There was a distorted reaction of systemic arterial pressure to exogenous angiotension-2 (V. G. Startsev et al., 1973). In the fifth year of disease, a 3- to 4-fold decrease, relative to the norm, in 17-oxycorticosteroid content in the peripheral blood

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Fig. 1. The Dynamics of EKG Changes in Hamadryad Baboons. Arrows correspond to periods of stress action. Recording dates below.

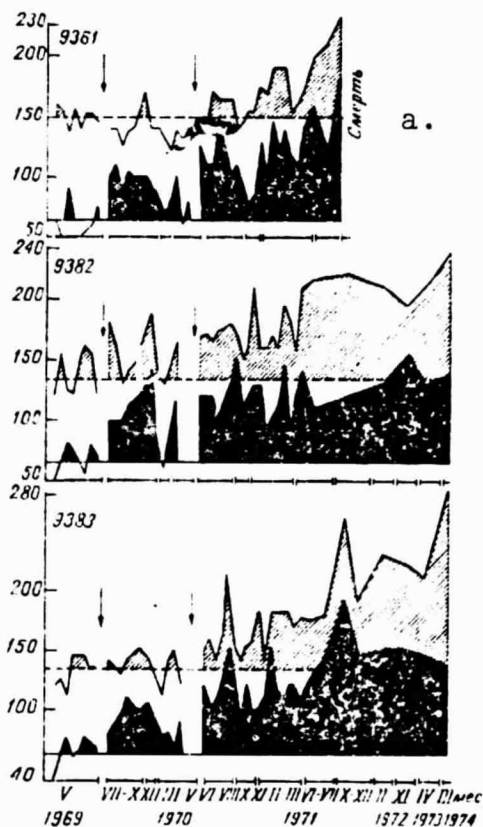


Fig. 2. Systemic arterial pressure dynamics (in the radial artery, Korotov's method) in 3 experimental apes. Solid and hatched areas correspond to an increase in initial levels. Arrows - periods of stress action.
a. death

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was detected, with simultaneous excretion of one of the polar metabolites of hydrocortisone, which is lacking in healthy baboons and the chemical structure of which is under study at the present time. While in the initial period of the disease, judging by data from variational pulsometry, the sympathico-adrenal system is in a state of stable hyperfunction; during the period of stabilization of the pathological process, symptoms of catecholamine insufficiency were detected: epinephrine injection and the influence of emotional stress (pursuit or immobilization) led to short-term normalization of shifts in EKG indices. At the present time, serum creatine kinase and transaminase is acutely

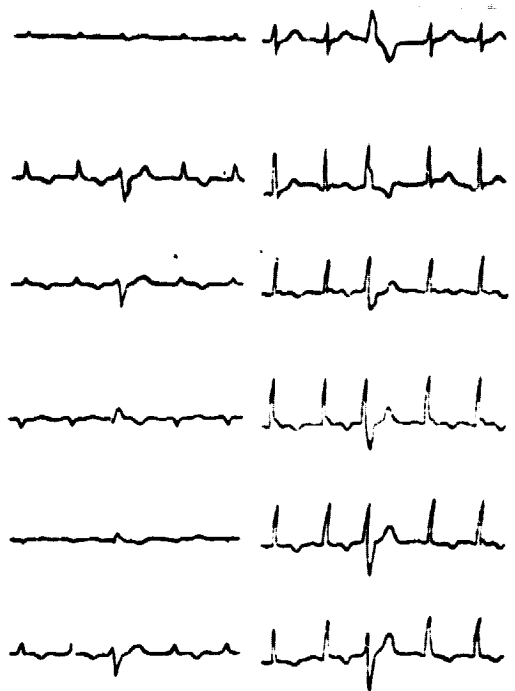


Fig. 3. Typical EKG of An Ailing Ape 5 Years Following the Beginning of the Experiment. Arterial Pressure 270/140 mm.

elevated in the ailing animals, which most likely reflects the destructive process in the myocardium. Macroscopically determined necrotic foci were found in the wall of the left heart ventricle in one ape which died in the third year of the disease.

Hence, following (and, obviously, as a result of) the psychoemotional stress which we applied, one and the same pathological process, representing the closest experimental analog of human hypertensive and ischemic cardiac disease, developed and steadily progressed in four out of five apes.

It was established in control experiments on other healthy apes that depression of the psychoemotional component of the reaction to stress following pursuit and prior to immobilization, haloperidol (0.5 mg/kg), prevented the development of pathological changes in the cardiovascular system.

A leading causal factor in the origination of the disease was the compulsory motionlessness of the animal while in a dominant-aggressive-defensive state of excitement, i.e., a stress situation, physiologically similar in principle to the willful suppression by a human of the ethically or legally impermissible expression of the biological reactions of fury or fear. Here there arises an excess emotional preparadness in the hemodynamic system and the somato-visceral system associated with it for maximal motor activity in order to implement the vitally important need for defense against a foreseen danger. It is manifested by intensification of cardiac activity and by the output of additional volumes of blood from blood depots into circulation. A greater quantity of catecholamines and steroid hormones simultaneously enters the

bloodstream. Since neither specially mobilized volumes of circulating blood nor high concentrations of humoral agents may be utilized by the relatively inactive skeletal musculature, diastolic hypertension develops, and the myocardium and vasomotor apparatus experience a destructive effect on their tissue metabolism from cardiotropic amines and steroids. The coronary arteries' spasm possible in such a situation may be logically viewed as a means of defending the cardiac muscle from an excess wave of catecholamines. The combination of a biologically imperative demand and "a deficiency of pragmatic information" (P. V. Simonov, 1970, 1972) - the impossibility of attaining the result of an action - reduces the emotional state and accompanying involuntary shifts to extreme tension. Repetition of the combination of natural defensive arousal and the consequent influence of a stress factor in a stereotyped situation adds the conditioning signals of the stress situation to the complex of environmental stimuli which constantly surround the ape, which ensures the future reproduction of the systematic conditioned reflex and consolidation of the psychosomatic effects of the stress situation. An important signaling significance probably accrues also to afferentation from the skeletal musculature and blood supply apparatus during their physiological hyperfunction. As a result, the forestalling emotional preparedness of the body for defensive behavior is converted from a current and adequate reaction into a pathologically stable dominant condition which is fixed through the development of auxiliary mechanisms of adaptation to a new activity regime of the cardiovascular system (myocardial hypertrophy, renin-angiotensin system activation, etc.). Systematic overstimulation of sympathico-adrenal and hypophyseal-adrenocortical system function leads to their exhaustion and a deficit of catecholamine and steroid hormones. Restructuring and activation of renin-angiotension-aldosterone system under these conditions is apparently intended as a substitute for the vasotonic function of catechol-catecholamines. /75

The genesis and development of ischemic cardiac disease and hypertensive disease in apes during our experiments, without preceding atherosclerosis (as in the experiments of G. O. Magakyan, D. I. Miminoshvili, and the observations of Groover et al.) make it possible to propose that genesis of these diseases in humans, at least in young and middleaged

people, may not be caused by an atherosclerotic process. Our experiments also showed evidence of a single etiopathogenesis for ischemic cardiac disease and hypertensive disease.

The results we obtained demonstrate, moreover, the obvious usefulness and prospects for experimental study of psychosomatic pathology using monkeys, based on a new methodological approach. The latter permits us to overcome the well-known limitations of the cortico-visceral approach, which ignores socio-psychological and emotional factors involved in the genesis of the disease, and to overcome the methodological and factual shortcomings of the neo-Freudian psychosomatic concepts of "the disease of the century."

The results of our many years of experiments, taken as a whole, and particularly: 1) the production in apes and detailed study of the etiopathogenesis of psychoneurotic and neurotic diseases, gastric achylia with precancerous polyposis, diabetes mellitus, sexual disturbances and, finally, hypertensive disease and ischemic disease of the heart, 2) the development of pertinent theoretical concepts about the concrete physiological mechanisms of the pathogenic contradiction between the social and the biological, on a factual basis, and 3) clarification of the principal mechanism of selective lesion of the somato-visceral system during psychoemotional stress, seem to permit us to raise the question of whether or not a new trend in theoretical medicine - experimental psychosomatic pathology - does indeed exist and is necessary in the final analysis. This trend, which is natural and applicable to contemporary problems, revives the concept of Pavlovian neurology, and has as its goal the study in experiments on lower primates of sc iato-pathogenic and psychoemotional stresses of a social nature, i.e., the etiology and pathogenesis of the most significant diseases of contemporary man.

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