

**THE CHRONIC DECREASE OF HEART RATE  
VARIABILITY CAN PRECEDE THE  
DEVELOPMENT OF SOME KINDS OF CANCER**

**Prof. Dr. Svetoslav Danev, D. med. sc.,  
Slav Svetoslavov**

National Center of Hygiene, Medical Ecology and Nutrition,  
blvd Dimitar Nestorov 15, Sofia 1431, Bulgaria

## S U M M A R Y

The purpose of the study is to present experimental evidence about the applicability of computer analysis of heart rate variability (HRV) measurements in early detection of carcinomathosis. By means of created by the author hardware and software facilities, the HRV was followed up every 18 mounts in 2147 practically healthy persons from both sexes (712 man and 1435 woman) aged from 36 to 62 years. It was found, that in the persons having a remarkable drift in sympathetic - parasympathetic balance towards an ergotropic state, the occurrence of different kinds of carcinomathosis is statistically highly significant augmented (  $p < 0.01$ ). This epidemiological study lasted 5-6 years and was prolonged with a comparison between the HRV measurements collected from 247 patients (154 man and 93 woman, mean age 38 years) from Bulgarian Oncology Center and same measurements collected from the ordinary country population (3852 man and 4254 woman, mean age 39.5 years). The collection of the HRV. data from country population was done preliminary for a period of 7 years, as a routine task of the National Center of Hygiene including assessment of the workers functional state. The comparison reveal a clear cut difference between both groups - the vegetative balance in the oncologically ill patients was found to be much more sympathetically oriented ( $p < 0.001$ ).

The paper presents a short description of the HRV method been used, as well as a theoretical concept concerning a possible mechanism underlying cancer development..

It is concluded, that the HRV analysis is an easily applicable method which reflects the sympathovagal imbalance. This method can provide insight into the risk for developing of the cancer.

Key words: Heart rate variability, Cancer, Oncology

## INTRODUCTION

Heart rate variability (HRV) is the amount of the heart beats fluctuations around the mean value. The suppression of HRV (a predominance of sympathetic activity) has been found to occur as a result of mental and physical load (1,2,3), the increase of gestational age (4,5); acute myocardial infarction (6); patients with an increased risk for ventricular fibrillation and sudden cardiac death (7,8,9,10); coronary artery disease (11); essential hypertension (12); after spinocerebellar degeneration, Shy-Drager syndrome (14,15); polyneuropathy due to diabetes mellitus, chronic alcoholism, Guillain-Barre syndrome (16,17); in persons exposed to environmental neurotoxic agents as organic solvents or lead (18,19); workers executing vibratory tool operations (20,21); autonomic neuropathy as a complication of diabetes (22); renal failure (23,24); medication with Atropine (25); Benzodiazepines (26); and in the persons influenced by different kinds of long-term lasting stress factors (27). Contrary, sympathetic activity decrease, respectively parasympathetically mediated fast fluctuations in heart rate increase as a result of beta-adrenergic blockers (25) or calcium channel blockers as Diltiazem (it reduce predominantly low-frequency heart rate (28)).

The relationship between vegetative balance ( or imbalance) and HRV is used to investigate the autonomic nervous system in patients as well in practically healthy persons. Some of the elicited by HRV measurements has a high predictive value towards developing of clinical symptoms (diabetic autonomic neuropathy - 29, congestive heart failure - 30) or for allograft reinervation (31).

Using and developing HRV method more than 30 years, we have observed, that some of our close relatives being in a long-lasting stress state (according to the HRV) become ill from different kinds of cancer (brain, ovarian, leukemia cancer of gl.mamae and So.) with 87% of mortality. This turn our attention to the question: are the persons being in long-term lasting sympathicotonia more exposed to carcinomathosis than "normal" persons, and can HRV be used as a predictor of health risk.

## METHOD

There exists many different mathematical algorithms for computing the HRV (32,33,34,35,36). All of them are used in order to obtain the best approach to the phenomenon being observed. The main periodic fluctuation of cardiac activity are connected with breathing (respiratory sinus arrhythmic - 0.2-0.4 Hz). There exists also some additional fluctuation connected with the activity of the thermoregulation (0.02-0.06 Hz). Many others internal or external factors can influence HRV in dependence of their force and frequency. The amount of HRV in a close time window (time domain analysis) can be assessed by calculation of standard deviation (SD) of all R-R intervals, amplitude of Mo (%), total spectral power, SD of beat-to beat R-R interval differences and many others mathematical expressions including them in different way. The frequency-specific oscillation of HRV is obtained by frequency domain analysis (power density spectrum), introduced first by Sayers (32) and Penaz et all (37). Spectral analysis involves Furrier transform algorithm and as a result the power spectrum in different spectral areas is displayed (the amplitude of variability as a function of frequency - 36).

Both (time and frequence domain) measurements are influenced by the increased sympathetic activity which cause a decrease of the variability and an increase of the spectral power in the spectral area between 0.01-0.06 Hz.

The preferable measuring time we had used in this study was 10 in (between 9 and 12 O.K.) in reclining position without remarkable movements. Talks were restricted. The assessment of vegetative equilibrium was made by means of a mathematical algorithm including classification of the measured persons with linear discrimination function (f). This method includes a preliminary procedure of selecting 1124 persons into two classes: first with well expressed long-term lasting sympathicotonia, and second, with well expressed vagotonia, according to the result obtained by some autonomic function tests (38,39).

Furthermore these classes were used for adjustment of a numerical scale with values: -100 arb.units for the first and +200 arb.units for the second group. The two classes form the

"teaching" multitude. The method obtaining the linear discrimination function consists of choosing all time as well as frequency domain HRV measures being used from us or from other authors, which are linear independent and describe adequately the sympathetic-vagal difference. The choosed measures are recognition parameters or main components of the examined difference. Their selection was made by means of singular value decomposition in the space of Karunen-Loev. After giving them the real coefficient of weight they were used for construction of a mathematical expression, giving the value of the discrimination function (f) of every new investigated person. This value was named stress coefficient (SC) - fig 1.

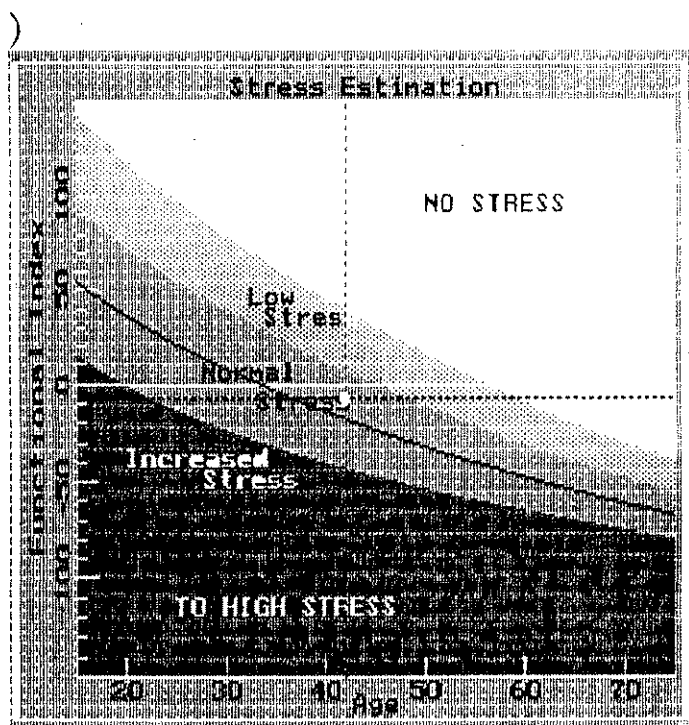


Fig.1. Graphical presentation of Stress Coefficient. It's age-related bioconstant values are shown by dark line.

An additional study proved that the correlation coefficients between SC and sympathetic tone is 0.91 ( $p < 0.01$ ) and with vagotone - 0.87 ( $p < 0.01$ ).

The assessment of HRV was made on the basis of SC. Some additional measures were also collected: mean cardiointerval ( $\bar{X}$ ), short-time variability ( $\sigma$ ), long time variability (SD),  $M_0$ ,  $AM_0$ ,  $Ex$ ,  $As$ ,  $As$  of Pearson, some spectral coefficients (index of

centralization, total power, spectral index, the power in different spectral areas and So.), frequency distribution histogram of all R-R intervals, scatterogram etc. All artifacts were automatically filtered. The hardware was a modulus (pocket format). It converts ECG signal to rectangular beats, processes them and gives on display SC, pulse, number of extrasystoles and estimated health risk. The connection between the modulus and IBM compatible computer allows to transfer all R-R intervals and mentioned indexes to PC for further more complete processing.

## SUBJECTS

In the first part of the study took part 2147 practically healthy persons from both sexes (712 man and 1435 woman) aged from 36 to 62 years. All of them were workers (operators, mechanics, maintaining personnel, administration and So.) from biggest Bulgarian steel or cooper producing plants, chemical industry and cotton or wool processing industry. The assessment of vegetative status was made every 18 months as a part of a paid by the Ministry of health prophylactic program. From all population 250 persons with SC showing a long-term lasting sympatheticotonia, and 250 persons, with SC showing a prevalence of the vagotone were selected and investigated for developing of different kinds cancer for a period of 5-6 years (according to the messages from the plants polyclinics).

In the second part of the study took part 247 patients from Bulgarian Oncologic Center (154 man and 95 woman, mean age 38 years) with different kinds of cancer (carcinoma pulmonum, carcinoma ventriculy carcinoma colli uteri, carcinoma corporis uteri, carcinoma ovarii, carcinoma tubae, carcinoma basocellulare, melanoma malignum, mycosis fungoides, meduloblastom, sarcoma maxillariss, leucosis, leucosis blastica, myeloma multiplex, lymphoma malignum, carcinoma gl. prostatae, seminoma testis, carcinoma papil. v. urinariae etc. The biggest part of the patients had not intoxication.

Their individual values of SC was compared with the corresponding age-related bioconstant value of SC, obtained as a result of a long-term lasting investigation program including 8106 practically healthy persons (3852 man and 4254 woman) aged from 20 to 70 years.

## PROCEDURE

In order to exclude circadian biorhythm the HRV measures were obtained (between 9-12 a.m.) The measuring time was 10 min. in reclining position without movements or talks. This procedure was repeated after 30 min. and the mean SC was calculated.

For excluding ill persons (except the oncological patients) every subject was submitted to a computerized test for psychosomatic complaints. The measuring of blood pressure, cardiac auscultation, hepar percussion and screening of the gl. tiroidea were also provided, as well as a short anamnesis. All persons with diabetes, thyreotoxicosis, alcohol dependence or using tranquilisators or beta-blockers were also excluded.

## RESULTS

### a) First part

The value of SC obtained in both groups (cohorts) followed up for a period of 5-6 years are presented on tabl.1. The cohort study, also known as longitudinal or prospective, which was employed compare two subsets (group with highlevel of long-term lasting overstress showing more sympaticotone resp.high values of SC and group with low values of SC, showing more parasympathicotone. First group was named SChigh and second SClow. The estimation of relative risk was done by comparing the sune of the persons in groups SChigh and SClow developing cancer later. This procedure reveal the relationship between the value of SC (risk factor) and the examined disease (carcinomatosis).

Table 1. *The values of SC in group SChigh (n=250, mean age 48 years) and group SClow (n=250, mean age 41.3)*

No of investi- gation	SChigh		SClow		p<
	X	SD	X	SD	
first	-112	18	+46	22	.01
second	-96	21	+58	26	.01
third	-132	24	+39	19	.01
fourth	-124	32	+36	17	.01
total	-126	23.7	+44.7	21	.01

Table 2. Assessment of relative risk (RR) in both groups

No of persons developing cancer within 5-6 years	with cancer	without cancer	total
SChigh	16(a)	234(b)	250
SClow	1(c)	249(d)	250

The risk of carcinomathosis is probability of its occurrence in a period of 5-6 years. It is for "exposed" population (first group) =  $16:(16 + 234) = 0.064$ , and for unexposed population (second group) is =  $1:250 = 0.004$ .

The resultive risk is:  $RR = \{a.(c+d)\} : \{c.(a+b)\} = 16$ . Wich is a very high value.

Thus, the persons from group SChigh are 16 times more likely to develop cancer if their SC value was around -126 in the period of following-up.

The attributable risk (AR) can be computed in two ways:  $AR = (Ip - Ine) : Ip$ , or  $AR = \{(Ie - Ine) : Ine\}$ . In these two ratios - Ip, Ie and Ine are incidence of carcinomathosis in all population, in "exposed" and in not exposed groups. As Ip can't be well defined it is better to use the second ratio. AR in this case is =  $(0.064 - 0.004):0.004 = 0.06:0.004 = 15$ .

The exponential endpoints for 95% confidence intervals for RR are

$$\log\{a.(c+d):c.(a+b)\} + 1.96. 1/a - 1/(a+c) + 1/b - 1/(b+d) =$$

$$= \log 16 + 1.96. 1/16 - 1/17 + 1/234 - 1/483 = \log 16 + 1.96.(0.073).$$

The confidence interval is presenting the probability that SC around -126 is a significant risk for developing carcinomathosis.

These results suggest that the long-term lasting overstress resulting in a tonic sympaticotonia is a risk factor for developing cancer. According to our experience, such a state, which we are named "Disadaptenia" is a very common phenomenon (in about 9% of population). It is caused predominantly by long-term lasting psychoemotional overstress.

## b) Second part

The second study was carried out in Bulgarian Oncological Center. The SI values of investigated patients were compared with their age-related bioconstant values, being representative for country population. The mean group value of SC for patients was -142, +37 arb.un. whereas the bioconstant value -44, +26. This difference is highly significant ( $p < 0.001$ , Student's-test for paired comparison). This indicate more sympathicotone in patients than in healthy population, but it is not sure whether the patients had this state before to become ill, or not. The multiple step-regression analysis as well as discriminant analysis reveal that the main predictor of the illness is SC, followed by depression, lack of the social support, age and neurosis. A lot of patients declare to had a bad sleep before to become ill.

## THEORETICAL BACKGROUND

The psychophysiological substrat underlying the relation "Disadaptenia-cancer" is possible to be a deterioration of the negative feet-backs of the biological system. As a result the system is loosing more ergotropic effort in the moment in which this is not necessary (less production of insulin, more production of catecholamines and corticosteroid hormones in the first 6-18 mounts). The processing of information in hypothalamic brain centers is unfavorably changed. Cancer is a process of uncontrolled cell dividing, and is also unnecessary. A phenomenon of "overshooting" is present in disadaptenia as well as in cancer. A deep inside in this phenomenom is impossible to be extracted only by modern biology, genetics and cellular theory. It is necessary to involve also the bioinformational exchange theory. According to it there exists a holographic image of the whole organism in its parts, and DNA of the chromosomes is not containing the whole genom of the biological system. In the gnom, 95-97% of the chromosomes are silent or "egoistic". It is speculated that they are connected with a quantum-field matrix which can move in the body by water transmission from one cell to another. It is though that chromosomes contain information regulating the topological organization of the body (cells), but this information in not sufficient for learning the biological structure how to live. This means, that chromosomes posses an additional genon which is space - organised holographic matrix (memory) - 40,41,42, named by us "extragenom" or ExGn.

The aminoacids in chromosomes are transferring linear-chapped information, whereas the holographic matrix is the modulator of the spatial-chapped ExGn. It is possible to accept, that the extragenom interacts with another space-organised holographic matrix, which is objective ( existing permanently in the nature). The last can be named also "cosmic". The process of seems to be living an oscillating interaction between the two holographic matrixes. This interaction needs the state of deep relaxation as it is in the state of vagotonia, which can be produced artificially by autogenic training or naturally by global cortex sensomotor as well as associative suppression (normal sleep). It is possible to regards the cancer as demonstration of unfavorable bioinformational exchange between the objective and subjective parts of the holographic matrixes. This process can be deteriorate by a state of long-term lasting overstress (sympathicotonia). As a consequence the cancer appears to be a bioinformation - exchange insufficiency, occurring in the state of overstress. This affects the DNA transcription on the water molecules (water can take the matrix from DNA and RNA and to transport it through the body as a kind of soliton - holographic information code (43). It is very likely that the living quality presuppose not only oxygen and foodsupply, but also an specific for every biological system additional bioinformation uptake. The bioinformational exchange needs an oscillating day-night modulated trophotropic orientation of the biosystem as a whole, being higher than some threshold value. This process seems to be permanently interrupted by the long-term lasting sympathicotonia (overstress, respectively, disadaptenia).

This theory, is in accordance with the newest phenomena discovered by the physic of living objects (the phenomenon of a cuted leaf for example). It is interesting to note also, that the sleep disturbance among the persons from group SChigh were better expressed than among the persons from group SClow ( $p < 0,05$ ), according to the questionnaire for psychosomatic complaints.

This theoretical background is not postulating the existence of a direct relation between overstress and chronic somatic or psychic overfatigue. Contrary, the last (in dependence of personal vulnerability and the length of uninterrupted exposition) can be a diminishing the overstress (tonic sympathicotone) factor. This is due to the specificity of the regulating systems - more sympathicotone results in more vagotone, because of oscillatory character of the living processes. Overstress is a lack of possibility to get over the stress influence,

so, it has an accumulative effect. This can be observed in the persons not being exposed to the severe stress factors as well as in the persons under specific risk - containing factors: toxicological, radiation, social, etc.

## CONCLUSION

It is possible to pose the question: is disadaptenia preceding the cancer, or vice versa? The answer can be formulated as follows: we found first disadaptenia, so it has certain predictive value towards the carcinomatosis. From another side RR is multiplicative, f.e. if another factor, like smoking, acts independently on disease process, the joint RR is product of two RRs. For excluding such an influence, both groups were equilibrated for such types of differences as well as it was possible.

If the contemporary physic is coming to the conclusions according to which the matter is more wave than corpuscular organised, and the waves are the substrates of all interactions in the known world, it is not very difficult to accept the biological interaction not only as a process caused by know agents (temperature, noise, light) but also as a global subordination of the living beings to the influences, not well described till now, but very well observed in the human history. One of them is the hypothetical bioinformational exchange, partly proved recently by the experiments of Prof. Kaznatcheev (44).

1. Kleiger RE, Bigger JT, Bosner MS, Chung MK, Cook JR, Rolnitzky LM, et al. Stability over time of variables measuring heart rate variability in normal subjects. *Am J Cardiol.* 1991;68:626-30.
2. Grossman P, Karemaker J, Wieling W. Prediction of tonic parasympathetic cardiac control using respiratory sinus arrhythmia: the need for respiratory control. *Psychophysiology.* 1991;28:201-16.
3. Piha SJ. Cardiovascular autonomic reflex tests: normal responses and age-related reference values. *Clin Physiol.* 1991;11:277-90.
4. Ribbert LS, Fidler V, Visser GH. Computer-assisted analysis of normal second trimester fetal heart rate patterns. *J Perinat Med.* 1991;19:53-9.
5. Van Ravenswaaij CM, Hopman JC, Killee LA, Van Amen JP, Stoeltinga GB, Van Geijn HP. Influences on heart rate variability in spontaneously breathing preterm infants. *Early Hum Dev.* 1991;27:187-205.
6. Rothschild M, Rothschild A, Pfeifer M. Temporary decrease in cardiac parasympathetic tone after acute myocardial infarction. *Am J Cardiol.* 1988;18:637-9.
7. Odemuyiwa O, Malik M, Farrell T, Bashir Y, Poloniecki J, Camm J. Comparison of the predictive characteristics of heart rate variability index and left ventricular ejection fraction for all-cause mortality, arrhythmic events and sudden death after acute myocardial infarction. *Am J Cardiol.* 1991;18:434-9.
8. Farrell TG, Bashir Y, Cripps T, Weiss JS, Schaad JW, Kehoe R, et al. Risk stratification for arrhythmic events in postinfarction patients based on heart rate variability, ambulatory electrocardiographic variables and the signal-averaged electrocardiogram. *J Am Coll Cardiol.* 1991;18:687-97.
9. Kleiger RE, Miller JP, Bigger JT Jr, Moss AJ. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. Multicenter Post-Infarction Research Group. *Am J Cardiol.* 1987;59:256-62.
10. Casolo GC, Stroder P, Signorini C, Calzolari F, Zucchini M, Balli E, et al. Heart rate variability during the acute phase of myocardial infarction. *Circulation.* 1992;85:2073-9.

11. Airaksinen KE, Ikaheimo MJ, Linnaluoto MK, Niemela M, Takunen JT. Impaired vagal Heart rate control in coronary artery disease. *Br Heart J.* 1987;58:592-7.
12. Goldstein DS. Plasma catecholamines and essential hypertension. An analytical review. *Hypertension.* 1983;5:86-99.
13. Sands KE, Appel ML, Lilly LS, Schoen FJ, Mudge GH JR, Cohen RJ. Power spectrum analysis of heart rate variability in human cardiac transplant recipients. *Circulation.* 1989;79:76-82.
14. Kuroiwa Y, Shimada Y, Toyokura Y. Postural hypotension and low R-R interval variability in parkinsonism, spino-cerebellar degeneration, and Shy-Drager syndrome. *Neurology.* 1983;33:463-7.
15. Kuroiwa Y, Wada T, Tohgi H. Measurement of blood pressure and heart-rate variation while resting supine and standing for the evaluation of autonomic dysfunction. *J Neurol.* 1987;235:65-8.
16. Malpas SC, Whiteside EA, Maling TJ. Heart rate variability and cardiac autonomic function in men with chronic alcohol dependence. *Br Heart J.* 1991;65:84-8.
17. Persson A, Solders G. R-R variations in Guillain-Barre syndrome: a test of autonomic dysfunction. *Acta Neurol Scand.* 1983;67:294-300.
18. Murata K, Araki S, Yokoyama K, Maeda K. Autonomic and peripheral nervous system dysfunction in workers exposed to mixed organic solvents. *Int Arch Occup Environ Health.* 1991;63:335-40.
19. Murata A, Araki S. Autonomic nervous system dysfunction in workers exposed to lead, zinc, and copper in relation to peripheral nerve conduction: a study of R-R interval variability. *Am J Ind Med.* 1991;20:663-71.
20. Harada N, Kondo H, Kimura K. Assessment of autonomic nervous function in patients with vibration syndrome using heart rate variation and plasma cyclic nucleotides. *Br J Ind Med.* 1990;47:263-8.
21. Murata K, Araki S, Maeda K. Autonomic and peripheral nervous system dysfunction in workers exposed to hand-arm vibration: study of RR-interval variability and distribution of nerve conduction velocities. *Int Arch Occup Environ Health.* 1991;63:205-11.

22. Hosking DJ, Bennett T, Hampton JR. Diabetic autonomic neuropathy. *Diabetes*. 1978;27:1043-55.
23. Ewing DJ, Winney R. Autonomic function in patients with chronic renal failure on intermittent haemodialysis. *Nephron*. 1975;15:424-9.
24. Zoccali C, Ciccarelli M, Maggiore Q. Defective reflex control of heart rate in dialysis patients: Evidence for an afferent autonomic lesion. *Clin Sci*. 1982;63:285-92.
25. Danev SG, Datzov E. Pharmacological induced sympathetic or parasympathetic influence upon heart rate variability in man. *Proc. of XXI-th Semaine Medicale Balkanique, Varna, Bulgaria, 1990*.
26. Halliwill JR, Billman GE. Effect of general anesthesia on cardiac vagal tone. *Am J Physiol*. 1992;262:H1719-24.
27. Danev S. Informativeness of heart rate variability in work-physiological aspect. *Doct. Diss., Sofia, NIHPZ-MA, 1989 (summary in Engl)*.
28. Bekheit S, Tangella M, el-Sakr A, Rasheed Q, Craelius W, el-Sherif N. Use of heart rate spectral analysis to study the effects of calcium channel blockers on sympathetic activity after myocardial infarction. *Am Heart J*. 1990;119:79-85.
29. Pfeifer MA, Cook D, Brodsky J, Tice D, Reenan A, Swedine S, et al. Quantitative evaluation of cardiac parasympathetic activity in normal and diabetic man. *Diabetes*. 1982;31:339-45.
30. Kienzle MG, Ferguson DW, Birkett CL, Mayers GA, Berg WJ, Mariano DJ. Clinical, hemodynamic and sympathetic neural correlates of heart rate variability in congestive heart failure. *Am J Cardiol*. 1992;69:761-7.
31. Fallen EL, Kamath MV, Ghista DN, Fitchett D. Spectral analysis of heart rate variability following human heart transplantation: evidence for functional reinnervation. *J Auton Nerv Syst*. 1988;23:199-206.
32. Sayers B. Analysis of heart rate variability. *Ergonomics*. 1973;16:17-23.
33. Hyndman B, Gregory J. Spectral analysis of sinus arrhythmia during mental loading. *Ergonomics*. 1975;18:255-270.
34. Bajevskij R. Estimation of the functional state on the basis of mathematical analysis of heart rate. *Wladiwostok*. 1987 (in russian).

35. Danev SG et al. study (1970-1971). In G. Mulder: The heart of mental effort. Thesis. Groningen, 1980.
36. Mulder L.J.M. Assesment of cardiovascular reactivity by means of spectral analysis. Thesis. Groningen, 1988.
37. Penaz J, Roukens J, VanDeWaal HJ. Spectral analysis of some spontaneous rhythms in the circulation. In: Drischel H, Tiedt N; eds. Biokybernetik. Leipzig: Karl Marx Universitat; 1968:233-6.
38. Stalberg EV, Nogues MA. Automatic analysis of heart rate variation: I. Method and references values in healthy controls. Muscle Nerve. 1989;12:993-1000.
39. Ewing DJ, Borsev DQ, Bellavere F, Clarke BF. Cardiac autonomic neuropathy in diabetes: comparison of measures of R-R interval variation. Diabetologia. 1981;21:18-24.
40. Gurvitch AG. Theoria biologiticheskovo polia. M.: Gosizdat, 1944, S. 142. (in russian).
41. Kaznatcheev VP, Gariaev PP, Vasilev AA, Berezin AA. Soliton-holographical genom with collectiv simetrical genetical cod. Accademy of med.sc., Nowosibirsk, 1990 (in russian).
42. Cope FW. Enhancement by high electric fields of superconductivity in organic and biological solids at room temperature and a role in nerve conduction. Physiological Chemistry and Physics. N.Y., 1974, Vol.6, N 5, S.405-410.
43. Bulenkov NA. Kristallografia. 1988;2:424-444.
44. Kaznatcheev VP, Mihailova LP. Bioinformacionnaia funkciya estestvennih elektromagnitnih polei. Novosibirsk, Nauka, 1985, S. 200. (in russian).