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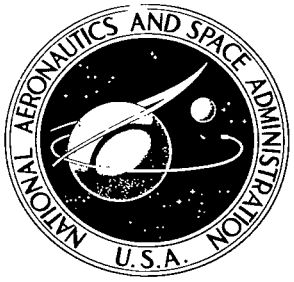

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THE HEART AND SPORTS

Essays on Cardiology in Sports

Edited by V. L. Karpman and G. M. Kukolevskiy

"Meditsina" Press, Moscow, 1968

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION • WASHINGTON, D. C. • FEBRUARY 1971

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Edited by V. L. Karpman and G. M. Kukolevskiy

Translation of "Serdtse i Sport; Ocherki Sportivnoy Kardiologii"
"Meditsina" Press, Moscow, 1968

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ANNOTATION

Sports cardiology is one of the most important sections of sports physiology and sports medicine. The present series of essays covers all of the principal trends in sports cardiology. This book presents analyses of the problems of the physiology of the cardiovascular system, methodological problems and certain principal forms of deviations in the activity of the cardiovascular system of athletes.

The physiological portion of the book presents information on the specifics of the functioning of the circulatory apparatus in trained athletes at rest. A special essay is dedicated to analysis of general physiological regularities of the adaptation of the heart to muscular activity. Finally, original data are presented on the influence of the state of training on the function of the cardiovascular system. In this essay, certain general problems of the diagnosis of the state of training are also discussed.

In the methodological portion of the book, data are presented on a broad range of cardiological methods and methodological approaches which can be used in sports medicine. The essays are divided into chapters in which the methods are combined according to physical and physiological principles. A special chapter analyzes such principal parameters of cardiodynamics and hemodynamics, such as cardiac output and the phases of the cardiac cycle.

This portion of the book discusses the sports medical aspects of electrocardiography, vector cardiography, phonocardiography, ballisto-cardiography, dynamocardiography and sphygmography in detail. Problems of the utilization of X-ray methods in sports, problems of radiotelemetry and modern methods of analysis of cardiological information are discussed.

In the final, clinical portion of the book, essays are presented dedicated to the principal forms of deviations in the state of the cardio-vascular system of athletes. Changes in arterial pressure, the principal forms of disruptions of the cardiac rhythm, as well as acute and chronic heart strain are analyzed in detail. A special chapter is dedicated to certain theoretical problems of the pathology of circulation in sports.

The last portion of the book presents original data, based on broad experience, on the differential diagnosis of systolic murmurs in athletes. In conclusion, certain organizational problems of sports cardiology are analyzed.

The book *The Heart and Sports* is designed for a broad range of doctors, both those working in sports medicine and physiologists, therapeutists, surgeons and other doctors interested in problems of cardiology.

FOREWORD

In recent years, due to the success of sports physiology and sports medicine, the outlines of a new scientific and practical trend -- sports cardiology -- are being drawn. Sports cardiology, a component part of general cardiology, studies an independent range of problems related both with the physiology of the active state of the organism, and with certain clinical problems of medical testing of those who engage in physical culture and sports. This places a definite impression on the treatment of various physiological and pathological changes in the activity of the cardiovascular system of athletes. In this respect, sports cardiology facilitates deeper development of our knowledge in the area of the physiology of healthy humans.

The book herewith presented to the reader is unique in a certain sense -- the world literature has never before seen such fundamental work on sports cardiology. To be perfectly correct, we should indicate the monograph of Reindell et al., *Herz, Kreislaufkrankheiten und Sport* [The Heart, Circulatory Diseases and Sports] (1960), in which only certain particular problems of sports cardiology were analyzed.

This book is not a guide for sports cardiology. It is more properly a presentation of the most recent achievements in this new and interesting area of medical science, written by a large group of leading specialists.

In spite of the fact that the book is a series of essays, as it is read it leaves the impression of a special monograph. This has been achieved by successful separation of the essays into three independent sections, with definite internal connection between the individual essays.

In the physiological portion of the book, rather exhaustive information is presented on the specifics of cardiac activity of sportsmen, the mechanisms of adaptation of the heart to muscular stress and other applied problems.

The methodological portion of the book contains information on a broad range of cardiological methods and methodological approaches used in sports medicine.

And, finally, clinical problems of sports cardiology are discussed in the last, third portion of the book. A number of questions concerning the problem of pathology in sports which are important for medical testing are raised here.

The book *The Heart and Sports* will doubtless aid in the clinical physiological training of a large number of sports doctors. It will also stimulate new investigations on sports cardiology. All of this is particularly important in relationship to the statement of the Central Committee of

the CPSU and the Soviet Government "On Measures for Further Development of Physical Culture and Sports" (August, 1966). I am sure that this book will be read with interest by physiologists and clinicists, who have not studied sports medicine.

The appearance of this publication has resulted to a considerable extent from the initiative of G. M. Kukolevskiy, who organized the team of authors.

Academician V. V. Parin

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PART I

PHYSIOLOGICAL CHARACTERISTICS OF THE CARDIAC ACTIVITY OF ATHLETES

The cardiac activity of athletes is differentiated by a number of characteristic features from the work of the heart of practically healthy people who are not engaged in sports. These differences arise in the process of long-term adaptation of the circulatory apparatus to systematic muscular stress. /5

The specific features of cardiac activity have been noted in athletes both in the quiet state and under conditions of muscular work. In the latter case, the heart of the sportsman works more productively and, which is particularly important, more efficiently than the heart of untrained people.

There is particular interest in analysis of the course of adaptation of the heart in the process of sports training.

The entire range of problems which we have outlined presents a comparatively complete physiological characterization of the muscular activity of trained athletes. It is these problems which are discussed in this, the first portion of this essay book. During the process of presentation, data are presented not only on the cardiodynamics, but also on the blood circulation in the organism and certain other noncardiac parameters. It is assumed that the reader is familiar with the normal physiology of blood circulation.

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

CHAPTER I

THE CARDIAC ACTIVITY OF ATHLETES IN THE QUIET STATE

ABSTRACT. The range of fluctuation of cardiac indicators in healthy persons is quite broad. This article presents general data on the "sports heart" at rest: properties of the cardiac muscle, pulse frequency, dimensions, cardiac output and hemodynamics circulating blood mass, infracardiac and arterial pressure, relationship between expulsion period, systolic index, cardiac rhythm and diastolic pressure in the aorta, relationship between electrical and mechanical activity of the heart, heart blood supply, regulation of cardiac activity in athletes, etc.

Many investigations dedicated to the study of the cardiac activity of healthy persons have allowed us to establish that the range of fluctuation of various physiological indicators is quite broad. It is determined to a considerable extent by the level of daily motor activity. /6

The dynamics of cardiac activity during sports exercises is determined by the significant, frequently maximum tolerable loads performed during the process of training and competition. The qualitative and quantitative specifics observed both at rest and under conditions of muscular work distinguish the "sports heart" from the heart of untrained persons. The changes which develop during systematic training are sometimes so great that certain clinicists consider it impossible to analyze the "sports heart" as a variant of the normal heart which, naturally, is hyperbola. Investigations of the work of the "sports heart" have assured us numerous times that the so-called mean normal indicators, which sometimes have no physiological sense, are quite arbitrary.

Sports activity is exceptionally varied. Therefore, the demands placed on the cardiovascular system during various types of sports activities differ widely. The dynamics of cardiac activity of athletes are determined by the nature of the exercises primarily used in training.

In spite of the considerable interest shown for investigation of the cardiac activity of athletes, some problems of the "sports heart" have been insufficiently studied. For example, the data on the systolic volume and volume per minute are contradictory. Very few data are available concerning

the metabolism of materials in the myocardium, the condition of the coronary circulation, etc. The dependence of the dynamics of cardiac activity on the nature of the training has been little investigated. A study of the structural and functional changes of the circulatory apparatus under the influence of sports training is not only of significance for sports physiology and sports medicine, but also is of general physiological interest. /7

Principal Properties of the Cardiac Muscle

The myocardium has a number of properties which are common to all muscle tissue -- excitability, conductivity, contractability and tonicity. In addition to this, the cardiac muscle has a characteristic specific feature -- the ability for automation, i.e. the ability to contract rhythmically under the influence of pulses arising within the heart itself without the interference of any external factors. We will analyze in this portion of this chapter the first three properties of the myocardium -- automatism, excitability and conductivity. The contractive capacity of the myocardium will be the subject of subsequent portions. The tonicity of the heart has as yet been little studied, so that we will not discuss it separately.

Automatism. In this chapter, we will make exclusive use of the myogenic theory of automaty, according to which the automatic function is performed by the muscle fibers in the conductive system of the heart. Under physiological conditions, the capability for spontaneous rhythmic contraction is highest in the fibers of the sinus node, which acts as the leader of the cardiac rhythm (pacemaker). The sinus, or sinoauricular node, is the first order automatic center. The automatism of the atrioventricular node (the second order center) is more weakly expressed. This means that the frequency with which pulses are formed in the atrioventricular node is lower than in the sinoauricular node. The capability for automatic functioning in the fibers of the conducting system, which is the third order center, is even less.

The frequency of cardiac contractions in man at rest varies over wide limits and depends to some extent on age, sex and body size. The intensity of metabolic processes and the level of daily motor activity, which are determined by the profession or amount of physical exercise, have considerable significance.

The pulse frequency is highest in early childhood. It varies in the new-born between 130-150 beats per minute, and in one-year-olds between 100 and 120 beats per minute. At 12 years of age, the pulse frequency is slowed to 75-90 beats per minute. At puberty the pulse frequency of both boys and girls increases slightly, but usually does not exceed 100 beats per minute. /8 By 20 years of age, the pulse frequency has slowed considerably (65-80 beats per minute). This range (55-85 beats per minute) is retained from then on. A slight increase in pulse frequency is observed in old age. The mean values

decrease somewhat with age. The pulse frequency is somewhat higher for women than for men.

TABLE 1. CHANGES OF PULSE FREQUENCY OF ATHLETES WITH AGE (ACCORDING TO R. Ye. MOTYLYANSKAYA, 1956)

Age in years	15-16	17-18	19-20	21-25	26-30	31-35	36-40	41-45	46-50
Pulse frequency beat/min	70.3	67.9	62.1	58.1	58.6	55.6	58.2	58.5	61.1

The changes in heartbeat frequency with age for athletes were studied by R. Ye. Motylyanskaya (1956). The data which she produced are shown in Table 1, from which we can see that the nature of the dynamics of the cardiac rhythm with age is the same as the changes observed in untrained persons. However, certain quantitative differences are evident.

In athletes, due to their training, powerful cholinergic reactions are developed, one result of which is the negative chronotropic effect and, consequently, the decrease in pulse frequency. There is a definite relationship between the intensity of the negative chronotropic effect and the nature of the training. Athletes training for endurance (skiers, long-distance runners, swimmers, bicyclists, etc.) show particularly great decreases in pulse frequency. Their pulses vary between 30 and 66 beats per minute, while the mean value (according to the data of various authors) rarely exceeds 50 beats per minute. Athletes who have trained for speed do not show such a great decrease in pulse frequency (the range varies between 48 and 78 beats per minute). In some observations, pulse frequency has been found completely unchanged in comparison with the data of untrained persons. However, the mean values are considerably lower (according to the data of various authors, the mean frequency varies around 60 beats per minute). The changes in pulse frequency for athletes whose training includes predominantly static effort

and athletes in which static effort must be combined with dynamic effort show approximately the same types of changes.

The presence of sinus bradycardia has been noted by many investigators /9
(Herxheimer, 1933; Reindell et al., 1949; S. P. Letunov, 1957; A. N. Krestovnikov, 1951; A. I. Smirnov, 1962; L. A. Butchenko, 1958, 1959; V. V. Matov, 1960; V. N. Kalyunov, 1963; etc.). The data of L. A. Butchenko (1963), produced in the examination of a large number of well trained athletes, are to some extent typical. Sinus bradycardia was noted in 67.6% of the cases. In 44.7% of the observations, it was moderately expressed (50-59 beats per minute). In 21.3% of the cases, the pulse frequency varied between 40-49 beats per minute, and only in 1.6% was the pulse as low as 31-39 beats per minute. In the opinion of a number of authors ((White, 1940; Kienle, 1946; Mellerowicz, 1956; etc.) this reaction of the rhythm may be a result of pathological states. However, S. P. Letunov (1954), L. A. Butchenko (1958) and others have shown that sharply expressed sinus bradycardia in athletes is most frequently a characteristic of a well functioning heart, and is a result of high training.

Athletes show a clear relationship between heart dimensions and pulse frequency: as the heart volume increases, the cardiac rhythm becomes slower (Reindell, 1949; Emmrich et al., 1962).

Bradycardia in athletes, as a rule, is accompanied by various degrees of sinus arythmia. According to the data of S. P. Letunov (1957), sinus arythmia in highly trained athletes is observed in 54% of the cases, while according to the data of Wolfe (1957) -- in 47.6%, L. A. Butchenko (1963) -- 60.8%. At the same time, athletes of low qualification and insufficient training show sinus arythmia significantly less frequently. Most investigators look upon sinus arythmia arising in athletes under the influence of systematic training as a manifestation of high functional capacity of the myocardium. However, sharp sinus arythmia may also arise during disruption of the normal cardiac activity. Thus, cases of expressed sinus bradycardia and arythmia should be the subject of careful medical investigation.

There is considerable interest in the investigations of A. G. Falaleyev (1964), V. M. Zatsiorskiy and S. K. Sarsaniya (1965), which defined the indicators of variability of the cardiac rhythm -- the standard deviation (in seconds) and the variation coefficient (in percent). Highly qualified athletes at rest showed a considerable variability of the pulse rhythm. Thus, according to the data of S. K. Sarsaniya (1965), the standard deviation amounted to an average of 0.07 sec, while the variation coefficient was 6.3% with a mean pulse frequency of 52 beats per minute. The author notes an increase in variability with a decrease in pulse frequency (there is a reliable negative dependence between pulse frequency and its variability). A. G. Falaleyev, V. M. Zatsiorskiy and S. K. Sarsaniya assume that the variability of the cardiac rhythm reflects the state of train to a certain extent.

Changes in cardiac rhythm are determined to a considerable extent by the state of train of the athlete. Usually, the lowest pulse frequency is observed with a high state of training, in so-called sports form, while as the state of training decreases, a certain increase in pulse frequency is noted (S. P. Letunov, R. Ye. Motylyanskaya, 1951; N. D. Grayevskaya, 1962; etc.). The dynamics of the cardiac rhythm during the process of training is an important criterion for evaluation of the functional state of the athlete. /10

The pulse frequency of athletes at rest is one indicator of the adaptation of the organism to the effect of the sports training. Training, which leads to an increase of the central tone of the vagus nerve changes the vagosympathetic equilibrium and is reflected in the pulse frequency. It might be assumed that the bradycardia observed in sportsmen indicates an "economy" of the activity of the circulatory apparatus at rest. The decrease in pulse frequency provides an increase in the potential lability of this indicator in athletes; a 5-6 times increase in the pulse rhythm is possible under physical load, while for untrained persons the cardiac rhythm can increase by not more than 3-3 1/2 times.

Excitability. Contemporary conceptions concerning the mechanism of excitation are related to the membrane theory of Bernstein and German developed for nerve conductors in 1902. On the basis of the concepts of membrane theory, successfully developed in the works of Hodgkin (1945, 1951, 1953), Curtis (1942), Huxley (1957, 1959, 1961), Hoffmann and Cranefield (1958, 1962) and others, the development of excitation in the muscle fiber can be represented as follows. The membrane of a muscle fiber is characterized by a certain electrical potential -- the rest potential, resulting from the presence of a concentration gradient for ions of potassium and sodium. There is a high concentration of potassium ions within the cell, and a high concentration of sodium ions in the medium outside the cell. Weidmann (1956, 1959), Lensi, Canniggia (1953) and others have shown that potassium is approximately 30 times more abundant and sodium approximately 20 times less abundant within the cells of the myocardium than in the substance surrounding the cells.

The rest potential in the cardiac muscle is +90 mv. During excitation, the rest potential drops and the membrane is recharged to -30 mv. The change in potential is thus 120 mv. The jump in action potential -- the "spike" -- results from rapid introduction of sodium ions to the cell (Rothenberg, 1949). During this time, the membrane becomes permeable for potassium ions. The departure of this cation from the cell corresponds to the drop in the action potential curve.

As the excitation propagates, the fibers of the myocardium contract. The contracted myocardium does not react to excitation, and the period of nonexcitability (refractory phase) in the heart is considerably longer than in skeletal muscles and nerve fibers. A distinction is made between the absolutely refractory phase (about 0.1 sec), during which even strong excitation of a section of the myocardium cannot result in supplementary /11

contraction, and the relatively refractory phase, which arises approximately 0.1 sec after beginning of the contraction of a portion of the cardiac muscle, during which the excitability of the sector in question is still low. However, extremely strong excitation can still cause contraction. In the sinoauricular node and the ventricular myocardium, the refractory phase is more extensive than in the musculature of the atrium.

One manifestation of the decreased excitability of the myocardium is an extrasystole. Extrasystoles frequently appear with various emotions and may occur under the influence of musculature effort. L. I. Fogelson (1958) assumes that when the excitability of the myocardium is increased, centers of latent pathological excitation (inflammatory, dystrophic and toxic) with relatively low force reach the threshold of excitation and cause preliminary contraction.

Extrasystoles rarely appear in athletes (V. N. Kalyunov, 1964); Z. B. Belotserkovskiy, 1965; etc.). However, at rest they appear relatively more frequently than with untrained persons (see Chapter XII). Their appearance is usually related with an increase in the central tone of the vagus nerve. The extrasystoles at rest, as a rule, disappear during physical exertion. The relatively "benign" nature of this type of extrasystole does not exclude the necessity of careful cardiological testing of athletes with such rhythmic irregularities.

Conductivity. In the normal heart, the excitation wave, beginning in the sinoauricular node, gradually encompasses the musculature of the ventricle, intraventricular septum, then reaching the atrioventricular node, where the so-called atrioventricular pause occurs, the mechanism of excitation of which is not yet clear. After this, the excitation wave extends along the bundle of His, its blades, branches, terminal branches, and reaches the myocardium. Due to the subendocardial location of the terminal branches of the conductive system, the excitation first encompasses the internal layers of the myocardium and then propagates at lower speed to the surrounding layers.

The rate of conduction of excitation differs in different sections of the heart. In the ventricles it varies between 800 and 1000 mm/sec. The highest rate of propagation of the excitation wave is in the conducting system in which it reaches 3000-4000 mm/sec. The lowest rate is in the atrioventricular node, where the so-called atrioventricular delay occurs.

The period required for the excitation to travel through the atriums and the intraventricular conducting system, i.e. the time from the beginning of excitation of the atriums to the beginning of excitation of the ventricles is reflected on the electrocardiogram in the form of the P-Q interval. The length of this interval in healthy, untrained persons varies from 0.12 to 0.18 sec. In athletes, the range of fluctuation is increased to 0.21 sec. This lengthening of the P-Q interval frequently accompanies a retardation in the pulse rhythm and is also related to an increase in the central tone of the vagus nerve. /12

According to the data of L. A. Butchenko (1963), in 2.2% of the observations of athletes, the P-Q interval was increased from 0.22 to 0.40 sec. S. P. Letunov (1957), L. A. Butchenko (1963) and others assume that in cases when the length of the interval does not exceed 0.27 sec and it is shortened under the influence of physical stress and the injection of atropine, the lengthening is a result of parasympathetic influences. Less frequently, the lengthening of the P-Q interval in athletes involves pathological changes of the myocardium. In these cases, corresponding changes in electrocardiograms are noted, and muscular effort, the injection of atropine, etc. either do not change the duration of the P-Q interval, or lead to a slight shortening with subsequent lengthening.

The initial portion of the electrical systole of the ventricles -- the QRS complex -- reflects the process of excitation of both ventricles. Its duration fluctuates from 0.05 to 0.10 sec. In connection with the fact that in various branches, the initial and terminal portion of the complex are sometimes isoelectric, the duration of the QRS complex is defined in the branch in which it is greatest.

In athletes, according to the data of L. A. Butchenko (1961), in 4.1% of the observations a lengthening of the QRS complex is noted. Frequently, this lengthening is combined with other changes of the electrocardiogram, a result of damage to the myocardium. At the same time, there is no convincing proof to indicate the physiological nature of isolated lengthening of the QRS complex, which, in the opinion of Reindell (1949), Mellerowicz (1956) and others, might result from hypertrophy of the myocardium (for more detail on this point see Chapter V, the section on "Electrocardiography").

Dimensions of the Sports Heart

The weight and dimensions of the heart are influenced considerably by muscular activity. Experimental investigations in various laboratories with animals have shown that as a result of training the weight of the heart (in relation to the weight of the body) can increase by 40% (I. A. Arshavskiy et al., 1963, 1965, etc.). The volume of the heart is also considerably increased.

In athletes, the increase in the dimensions of the heart (based on percuteur data) was first noted by Henschen (1898), who evaluated this fact as evidence of the unfavorable influence of sports on cardiac activity. He introduced the term "sports heart" in order to indicate what he believed to be pathological changes in the fundamental state of the heart developing under the influence of physical exercise. Naturally, the views of Henschen on the genesis of the increase in the size of the heart in athletes were not broadly recognized. /13

In many experimental investigations and observations with humans it has been established that high muscular activity causes the development of a

moderate expansion of the ventricular cavities. This type of expansion or tonogenic dilation results in compensatory hypertrophy of the muscular wall of the ventricle ("harmonic growth" according to Linzbach). It is remarkable that the increase in the dimensions of the heart, like the compensatory hypertrophy, observed in athletes is a reversible phenomenon. When systematic training is interrupted, the volume of the heart gradually decreases (N. A. Kurshakov, 1947; A. N. Krestovnikov, 1950; S. P. Letunov and R. Ye. Motylyanskaya, 1951).

In order to define the volume of the heart, biplanar teleroentgenography can be used (see Chapter VII), in which the calculation of the volume of the heart is performed using the Musshoff-Reindell formulas, or Andersson's formula, etc., or the tables of Zdansky, et al.

Roentgenkymography is primarily used in our country for analysis of the dimensions of athletes' hearts (S. P. Letunov and R. Ye. Motylyanskaya, 1951; N. D. Grayevskaya, 1960; T. E. Karu, 1966; N. V. Savina, 1958; etc.).

During the period of growth (i.e., up to 20 years) there is a close interrelationship between heart volume and age (Musshoff, Reindell et al., 1958). Up to 16 years, this dependence is strictly linear. The increase averages 50 cm^3 per year (in the period from 16 to 20 years, the increase diminishes). At 10-11 years of age, the volume of the heart is 411.5 ± ± 9.8 cm^3, and it increases by 19 years to 769.5 ± 15.8 cm^3 (Musshoff et al.).

According to the data of R. Ye. Motylyanskaya (1956), during long-term observations of young athletes, in 30% of the cases an increase in the area of the heart was noted (most frequently during systematic exercises in skiing, light athletics, football or bicycle riding). A moderate increase in heart volume was noted in young basketball players by T. E. Karu (1966). The relatively slight changes in the young people can apparently be explained by the high motor activity normal for this age even when no regular sports exercises are performed.

A quite close relationship is observed between the volume of the heart and body weight (Musshoff et al., 1958; Emmrich et al., 1962; T. E. Karu, 1966; etc.). The mean values of the ratio of heart volume to body weight fluctuate over very narrow limits. Musshoff et al. (1958), who investigated this problem for some time, established that the minimum value of the ratio of heart volume to body weight is observed for women between 22 and 41 years of age (averaging 9.4), while the maximum (averaging 14.19) was noted in athletes of 18-32 years of age. The correlation between heart size and body weight in untrained persons is rather high (r = 0.6-0.8). In athletes, due to the varying hypertrophy of the myocardium, this dependence is considerably weaker (r = 0.44-0.47). Thus, according to the data of Emmrich et al. (1962), 15 female athletes between 16 and 25 years of age (light athletics, skiing, swimming) had a ratio of heart size to body weight of 8.9-14.2. In 104 men (light athletics, bicycling, rowing, skiing, swimming) this ratio

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fluctuated between 9.8 and 18.4. The least range of variation (12.3-15.3) was noted for the skiers. The ratio of heart size to body weight depends little on age. T. E. Karu (1966), on the basis of analysis of literature data and his own data, noted that the proper ratio of heart volume to body weight in the prone position is 11.2 ± 2.89, and is practically independent of sex and age. In the standing position, this ratio decreases and amounts to 9.8 ± 2.89. In athletes (particularly those trained for endurance) this ratio increases.

The relationships between the heart size and height are considerably more variable. According to the data of various authors, the correlation coefficient fluctuates from 0.38 to 0.847, i.e. over quite broad limits. This varied nature of the data is probably principally determined by age. R. Ye. Motylyanskaya (1956) indicates that the relationship between heart size and height, most strongly expressed during the first years of life, gradually decreases. This dependence is most sharply disrupted during puberty.

A closer dependence obtains between heart volume and area of body surface. However, in athletes, due to the wide range of variation in heart size, this correlation is less strongly expressed than in untrained persons.

According to the data of Reindell, Musshoff and Klepzig (1960), heart volume in athletes averages 36.5% greater than in untrained persons (respectively 1010 cm^3 and 740 cm^3). The weight of the heart increases approximately by 125 g, i.e. within approximately the same limits.

The changes in heart size are most strongly expressed in athletes trained for endurance. Thus, Pijade (1954) found the largest hearts in swimmers and middle-distance runners, the smallest hearts in sprinters, volleyball players and boxers (although these athletes also had larger hearts than untrained persons). The maximum absolute heart volume (1100 cm^3) was detected for a tall water polo player (Medved, 1964).

According to the results of the investigations of Emmrich et al. (1962) who examined 119 participants in the winter and summer Olympic Games, the absolute heart volume varied from 505 to 1300 cm^3. The smaller heart volumes were generally found for women and young athletes.

Similar data concerning the dynamics of heart dimensions under the influence of sports training were produced by N. D. Grayevskaya (1962), T. E. Karu (1966), N. V. Savina (1958), in spite of the various methods which they used. /15

In roentgenkymographic investigations of a large number of athletes, S. P. Letunov and R. Ye. Motylyanskaya (1951) discovered changes in the heart configuration in 74% of their observations. Detailed analysis of the data produced allowed the authors to describe three types (or variants) of "sports heart." The first variant is characterized by the absence of any changes in magnitude or configuration of the heart (26% of the observations).

The second variant is characterized by more or less distinctive changes in the left ventricle, influencing the configuration and size of the heart (40.9% of the observations). Finally, in 33.1% of the observations changes were observed not only in the left, but in the right sections of the heart (third variant); changes in the size (area) of the heart are most frequently observed with this third type of "sports heart".

The expediency of typological characterization of "sports heart" is confirmed by many observations. Thus, V. N. Kuz'mina (1954) noted reliable distinctions between the cardiac delivery rate in athletes with the various types of "sports heart." Also, characteristic shifts were noted under the influence of dosed muscular work and other actions. The type of heart determines to some extent the functional state of the myocardium (S. P. Letunov, R. Ye. Motylyanskaya, 1952).

According to the data of V. V. Bulychev, V. A. Khmelevskiy and Yu. V. Rutman (1965), the hearts of athletes are enlarged due to enlargement of both ventricles or primarily enlargement of the right ventricle. The authors did not observe any isolated change in the configuration of the left ventricle.

In the experimental investigations of Van Liere et al. (1964), it was established that under the influence of systematic training (dogs ran on a treadmill for one hour twice a day for two months at 1609 m/hr), moderate hypertrophy of both ventricles developed in the experimental animals, and isolated hypertrophy of both the right and left ventricles was also observed. On the average, the weight of the right ventricle was increased by 10-16%, of the left ventricle -- by 10-13%. The ratio of the weight of the left ventricle to the weight of the right ventricle remained practically unchanged on the average. It is interesting that in the animals of another group which was subjected to hypoxia, sharp hypertrophy of the right ventricle was developed, while the changes in the left ventricle were relatively slight. The difference in weight of right and left ventricles in these animals was almost the same.

An increase in the volume of the heart is accompanied by a significant increase in the relative blood volume (at rest). Thus, Klepzig (1955) established that the ratio of systolic volume of blood to the filling volume, /16
i.e., the entire volume of blood contained in the ventricle at the end of the diastole averages 1:2.2 for athletes (1:1.3 for untrained persons). The increase in the residual blood volume indicates an increase in functional reserve of athletes' hearts.

Cardiac Output and Hemodynamics

The systolic volume and volume per minute of blood circulated are important indicators of the functional state of the cardiovascular system. Investigations of the cardiac output under various physiological and pathological conditions have long attracted the attention of physiologists and

clinicists. There is particular interest in a study of the cardiac output of athletes. Lindhard (1915) is considered to be the first physiologist who studied the dynamics of the volume per minute and per beat for athletes (over one-half century ago he investigated one well trained bicycle rider among a group of healthy persons). Since then, the investigation of these indicators of cardiac activity in athletes both at rest and under conditions of muscular activity has attracted many physiologists and clinicists. However, there is still no final answer to the question of the nature of the changes in the volume per beat and volume per minute pumped by the heart under the influence of systematic training. The reason for this lies in the varying methodological approaches to determination of cardiac output, the very small number of athletes investigated by most authors, as well as the inclusion among those investigated of athletes of low qualification. It should be added to this that frequently all of the attention of the investigator is attracted by athletes trained for endurance. No characterization of cardiac output as a function of sports specialization has ever been done.

Since the most precise methods of measurement of blood volume per minute and per beat are generally traumatic, require complex apparatus, etc., simpler methods which represent no inconvenience for the test subjects but are also less precise are generally used to investigate athletes. They include the Bremser-Ranke and Wezler-Boger methods.

In investigations of the cardiac activity of athletes, the acetylene method of Grollman is frequently used (in our country usually in the Parin-Khrenov modification). This method provides more reliable information on cardiac output than the physical methods. Its relatively insufficient popularity apparently results from the cumbersomeness of investigations using this method. Recently, I. V. Aulik (1965) began to use chromatography, which greatly simplifies the method.

The most precise methods are those for direct determination of cardiac output according to Fick and the methods related to dilution of an indicator. In recent years, Reindell et al. (1960), Mitchell (1963), Astrand et al. (1964), Saltin (1964), Hansome and Tabakin (1965) and others have used these methods to investigate the functional state of the circulatory apparatus of athletes. However, even this seemingly quite reliable information is not a completely objective reflection of the state of the cardiac output. The emotional stress involved in intravenous injection of the indicator, intracardiac catheterization and other manipulations which these methods involved can distort the true indicators of cardiac output (we know that during strong excitation, the volume per minute may triple). /17

It follows from the above that the data of various authors, particularly, when various different methods of investigation are used, can be compared only quite cautiously.

The systolic volume or volume per beat for healthy persons fluctuates quite broadly -- from 60 to 100 mℓ. A certain dependence of the volume per beat on the body surface area has been noted. In connection with this, in

order to compare this quantity for persons of various body types, the systolic or beat index (ratio of volume per beat in mℓ to area of body surface in square meters) has been used. A number of authors have also indicated the dependence of the volume per beat on height, body weight and heart volume. In the standing or sitting position, the volume per beat is lower than in the prone position (Sjostrand, 1956; Rushmer, 1958; Tabakin and Hanson, 1965; etc.). In the prone position, the venous influx of blood to the heart in test subjects increases in connection with the decreasing influence of hydrostatic factors.

The data of various investigators on the absolute value of systolic output of athletes at rest vary considerably. These differences result, as was noted above, from specifics of the methods used, the groups of test subjects used, etc. For example, Mellerowicz (1956), Reindell, Delius (1948), Berger and Olloz (1935), who used physical methods for measurement of the volume per beat, discovered a moderate decrease in this quantity for athletes. Approximately the same results were produced by Barcroft (cited by Mellerowicz, 1956), Krogh (1956), Christensen (1931, 1933), V. N. Kuz'mina (1959), who used gas analytic methods.

At the same time, according to the data of Ye. A. Poruchikov (1963), B. S. Parnov (1958), T. E. Karu (1966) (mechanocardiography), G. I. Markovskaya (1955) (acetylene method), Tabakin and Hanson (1965) (dye dilution method), the mean values of volume per beat in athletes are somewhat higher than in untrained persons. V. S. Farfel' (1960) considers that with clear bradycardia, the volume per beat increases to 100-120 mℓ. Generally, /18
according to the opinion of the overwhelming majority of investigators, the increase in absolute volume per beat in athletes is related to a decrease in the pulse rate. As a rule, the systolic index increases in this case.

However, it might be assumed that with identical cardiac rhythm, in connection to reinforced negative inotropic effects (V. Raab, 1959; Reindell, 1943) the systolic output of athletes at rest is slightly less than equivalent data for healthy persons not involved in sports. This has been indirectly indicated by the increase in residual volume, which is considerably higher for sportsmen than for untrained persons (Klepzig, 1955). Incidentally, these considerations are highly debatable.

Blood circulation volume per minute. In healthy, untrained persons, the blood circulation volume per minute varies between 3-6.5 ℓ/min. This value is closely related to the area of body surface. The cardiac index (ratio of volume per minute to body surface area) evens out differences in blood volume per minute for persons with considerable constitutional differences. Thus, according to the data of most investigators, using the Fric direct method and indicator dilution methods, the normal limits of fluctuation of cardiac index amount to 3-4 $\ell/min/m^2$.

The blood volume per minute changes little when the position of the body is changed. The decrease in volume per beat observed in the orthostatic test is compensated for by an increase in pulse frequency.

There are indications that the volume per minute depends on age, sex, temperature of the surrounding air and certain other factors.

The opinion has been firmly stated in the specialized literature that low values of blood volume per minute are characteristic for athletes at rest. However, even a superficial analysis indicates that this type of data were produced by investigating athletes trained primarily for endurance (the cardiac activity of which shows clearly expressed negative chronotropic and inotropic influences). For example, Mellerowicz (1956) discovered that the blood volume per minute of 107 middle- and long-distance runners averaged 2.74 ℓ/min (for untrained persons he found 4.8 ℓ/min). According to Reindell and Delius (1949), the mean blood volume per minute for 19 stayers was 2.61 ℓ/min. These authors used the Bremser-Ranke method.

V. N. Kuz'mina (1954) who used the Parin-Khrenov modification of the acetylene method noted a dependence of blood volume per minute on heart size. In the first, second and third variants of "sports heart" (according to the classifications of S. P. Letunov and R. Ye. Motylyanskaya, 1951), the volume per minute was approximately 2.76, 3.56 and 4.19 ℓ/min, while the cardiac index was 1.69, 1.92 and 2.22 ℓ/min/m^2 respectively (i.e. considerably lower than that of untrained persons). Most of the athletes investigated by V. N. Kuz'mina competed in light athletics and skiing. /19

The decrease in the volume per minute at rest under the influence of many years intensive training for long-distance runners was noted by Tabakin and Hanson (1965). A decrease in volume per minute under the influence of sports training (primarily for endurance) was also indicated by V. Raab (1959), Mitchell et al. (1963), Astrand et al. (1964), Bevegard (1964), Saltin (1964), Strandell (1964), Collet and Liljestrand (1924), etc.

The decrease in the blood volume per minute at rest under the influence of training for endurance has been indicated by many experimental investigations on animals. A sharp decrease in the frequency of contractions is usually observed (sometimes almost dividing the frequency in half in comparison with the initial rhythm), plus a moderate increase in the volume per beat.

A decrease in the volume per minute at rest in moderate, long and superlong-distance runners, skiers, swimmers and other athletes whose specialties involve the development of endurance, in addition to bradycardia, hypotonia, increased residual blood volume at rest is one of the most important factors providing high functional reserves for the "sports heart." The basis of these changes is the so-called "principle of economy" (Mellerowicz, 1956).

Information on the dynamics of the volume per minute under the influence of training to develop speed, strength, coordination of movements, etc. is relatively lacking. V. V. Skryabin (1950) and A. N. Vorob'yev (1962) found no essential deviations in the value of volume per minute at rest for weight lifters in comparison with the normal values. The same thing was noted by

A. V. Ionina (1939) in gymnasts and by T. E. Karu (1966) for basketball players.

The principal function of the circulatory apparatus is the movement of blood. This makes understandable the tendency to characterize its functional state by defining the time (or rate in the terminology of certain authors) of blood flow. Attempts to determine the functional state of the circulatory apparatus by the time required for blood to flow through the vessels of the systemic circulation have been unsuccessful. The time required for blood to move through any individual loop of the systemic circulation depends on many factors, among which we must note the functional activity of the organ in question, nervous influences, the intensity of metabolism, surrounding temperature, etc. Obviously, the time of blood flow through individual loops of the systemic circulation may differ quite markedly from the mean time of movement of blood from the left ventricle to the right atrium.

The time required for blood to move through the vessels of the respiratory circulation reflects the mean circulation time somewhat more accurately. This allows this quantity to be used to evaluate the functional state of the circulatory apparatus as a whole. /20

The quantity of blood located at any given moment in the respiratory circulation (central blood volume) varies in correspondence to changes in the blood volume per minute. The principal factor determining the quantity of blood moving through the respiratory circulation is the blood flow rate. To a lesser extent, the central blood volume also depends on the total cross section of pulmonary capillaries. In contrast to the several variants of peripheral loops in the systemic circulation, the lengths of individual loops in respiratory circulation differ relatively little from each other. From this it follows that the time of movement of blood through the various vessels of the respiratory circulation fluctuates over relatively small limits.

All of this gives us reason to believe that the determination of time of movement of blood through the respiratory circulation can provide valuable information concerning the functional state of the entire circulatory apparatus.

Most frequently, the blood flow time is determined over the sector lungs-ear, which includes sectors of both respiratory and systemic circulation. Thus, the time required for blood to move over this sector does not reflect the mean time of circulation. However, methodological approaches for measurement of the time of circulation over the lung-ear sector are most convenient.

Analysis of data on the circulation time from the literature indicates that the results of various investigators are frequently contradictory. As A. G. Dembo (1963) showed, the variability of the results depends on sex, age of test subjects and, most frequently, on investigation method used. Even with different variants of the oxyhemometric method (bloodless method,

allowing repeated measurements to be performed) quantities were produced which differed from each other quite essentially. A. G. Dembo (1963) established that the most stable results were still produced by using the oxyhemometric methods in which, after preliminary reduction of the saturation of arterial blood with oxygen, achieved by delaying respiration, the circulatory time is determined from the beginning of the increase in saturation after inhalation.

The blood flow time (over the sector lungs-ear) increases with age, averaging 4.03 sec for children of 10-13 years, and 7.30 sec for adults of 50-60 years (A. M. Tyurin, 1961). For highly trained athletes, the blood flow time over this sector was statistically significantly increased in comparison with the data of untrained persons (6.30 sec as against 5.50 sec). The slowdown in blood flow in athletes apparently results from a decrease in the level of metabolism at rest related to the predominance of sympathetic-inhibitory and cholinergic influences. /21

The fact discovered by Fric (1957) and A. M. Tyurin (1961) of altered blood flow time in correspondence with the state of training is of important significance for sports medicine and sports physiology. It has been found that with increasing training, blood flow becomes slower. This once more confirms the significance of investigation of the blood flow time for an evaluation of the functional state of the circulatory apparatus.

A measurement of the mass of circulating blood is also quite important for determination of the functional state of the circulatory apparatus. The quantity of blood circulating varies over broad limits as a function of physiological and pathological influences. A decrease or increase in the mass of circulating blood is achieved by adding to or subtracting from the blood depot. The functional ability of the circulatory apparatus is reflected not so much by the absolute quantity of blood circulating as by the ability to increase and decrease it under various conditions.

We found no information on the volume of circulating blood in athletes in the available literature. However, analysis of data on the volume per minute and blood flow time allow us to conclude that in athletes at rest the quantity of circulating blood is slightly decreased, while during muscular work it is increased in comparison with that of untrained persons.

According to the well known Vierordt formula, the mass of circulating blood can be determined as follows:

$$Q = \frac{V_m \cdot C}{60}, \qquad (1)$$

where V_m is the blood volume per minute, C is the mean blood flow time. This formula clearly demonstrates the interrelationship of all three hemodynamic

indicators, the importance of each one in an evaluation of the activity of the circulatory apparatus.

Intracardiac and Arterial Pressure

The dynamics of intracardiac pressure during the cardiac cycle is related to the contraction and relaxation of the myocardium. The pressure developed in the atriums is slight, since blood from the atriums moves into the relaxed ventricles. Higher pressure is developed in the right ventricle, which must overcome the resistance of the respiratory circulation. However, the highest pressure is developed in the left ventricle, since the resistance of the systemic circulation is considerably higher than that of the respiratory circulation.

The pressure in the ventricles begins to decrease before the contractive process in the myocardium is completed, during the phase of reduced output. By the end of the diastole the pressure in the ventricles is 3-7 mm Hg. The range of fluctuations of pressure in the arterial lumen is not so great: the diastolic pressure in the arteries is 50-70% of the systolic pressure. Fluctuations in arterial pressure during constant cardiac activity depend on the elasticity of the walls of the arteries and the value of peripheral resistance. /22

Intracardiac pressure in the sportsman has been studied very little. Reindell et al. (1960) found no differences in the pressure in the right sections of the heart from the values observed in untrained persons. Thus, the final diastolic pressure in athletes in the right atruim were 3 mm Hg, in the right ventricle 3 mm Hg, in the pulmonary artery -- 14 mm Hg. Also, no quantitative differences were discovered from normal values of intracardiac pressure by Granath and Strendell (1963), who investigated a group of trained young people in addition to mature people.

Arterial pressure in athletes has been considerably more thoroughly studied. We should note at this time that in the overwhelming majority of works dedicated to analysis of arterial pressure the Korotkov auscultative method was used which, as will be shown in subsequent chapters, has certain limitations in studies of arterial pressure where certain perturbations are present. However, under conditions of physical and mental rest, the Korotkov method is rather precise.

The value of arterial pressure increases with age. However, whereas for people who are not involved in sports the increase in systolic and, to a lesser extent, diastolic pressure is observed throughout life, in athletes this increase is noted only during youth (Table 2). Athletes between 21 and 50 years of age show no essential changes in arterial pressure (R. Ye. Motylyanskaya, 1956; N. D. Grayevskaya, 1960). R. Ye. Motylyanskaya (1956) notes that young athletes with good physical development relatively frequently show high systolic pressure, exceeding the normal values (the

so-called young hypertension). Usually this increase in pressure is eliminated by correcting the training regimen.

A. G. Dembo (1965), N. I. Volnov (1958), M. Ya. Levin (1965) and others have indicated that the arterial pressure of athletes lies within the limits of normal values in approximately 75% of all observations, while increased and decreased pressures are observed with almost identical frequency. However, in highly trained athletes, high arterial pressure is observed significantly less frequently than hypotension.

With increasing state of training, a decrease in arterial pressure is noted in athletes (S. P. Letunov, R. Ye. Motylyanskaya, 1951; A. G. Dembo, 1965; M. Ya. Levin, 1965; Yu. I. Kuznetsov, 1958; etc.). However, this does not mean that all hypotension observed in athletes is physiological in nature. According to the data of A. G. Dembo and M. Ya. Levin (1965), physiological hypotension in athletes was found in only 32.3% of the cases. In the remaining observations, the hypotension was a result of a chronic center of infection, overfatigue, neurocirculatory dystonia, etc. (for more detailed information on secondary changes in pressure in athletes, see Chapter XI). /24

In masters of sports, physiological hypotension was encountered, according to the data of M. Ya. Levin (1965) in 41.3% of observations, whereas second and third rank athletes showed this condition in only 18.5% of cases. Low values of arterial pressure are particularly frequently observed in middle-, long- and superlong-distance runners, skiers, swimmers, bicyclists, i.e., athletes specially trained for endurance.

The decrease in arterial pressure accompanying the development of endurance results from a decrease in the level of metabolism and rest, related to a reinforcement of cholinergic influences and an increase in the tone of the vagus nerve.

The regulation of arterial pressure is a complex and far from completely understood problem of modern physiology. According to the representations of V. M. Khayutin (1965), we can speak of three degrees of regulation of arterial pressure. These include regulation at the organic level, regulation of the constancy of overall systemic resistance with changing regional resistance and regulation by excitation. In the last case, the optimal operating regime of the heart, in which it would pump the volume of blood required by the organs with the least possible load, is established by matching the values of total systemic resistance, degree of emptying of the heart and heartbeat frequency. Mechanoreceptors provide the necessary information, which is converted by changing the operating characteristics of regulation. This is the third, optimizing system of regulation according to V. M. Khayutin.

TABLE 2. ARTERIAL PRESSURE IN ATHLETES OF VARIOUS AGES
(ACCORDING TO R. Ye. MOTYLYANSKAYA, 1956)

Men

Age (year)		15	16	17	18	19	20	21–25	26–30	31–35	36–40	41–50
Systolic pressure	(*M*)	101,9	109,0	112,0	114,5	114,6	122,1	111,6	113,1	110,7	114,9	117,1
	(±σ)	9,5	13,0	11,5	14,5	11,2	15,5					
Diastolic pressure	(*M*)	69,75	70,8	70,0	71,2	72,3	73,6	63,3	70,3	68,4	68,5	77,6
	(±σ)	11,5	11,5	11,5	9,5	10,1	9,5					

Women

Age (year)		15–16	17–18	19–20	21–35	over 35
Systolic pressure	(*M*)	98,0	107,0	108,0	109,0	109,0
Diastolic pressure	(*M*)	58,0	69,0	68,0	68,4	70,7

Note: M indicates arithmetic mean; σ -- mean square deviation.

Note: Commas indicate decimal points.

Dynamics of Cardiac Contraction

In addition to determining the value of mean output and arterial pressure, in order to evaluate the contractive capacity of the myocardium we must study the dynamics of the cardiac contraction. Analysis of the activity of the ventricles -- the principal functional portion of the contracting heart -- allows us to divide the cardiac cycle into individual phases. /25

In correspondence with the ideas of Hurthle (1891) and Wiggers (1921), supplemented by Holldack (1951) and V. L. Karpman (1964), the cardiac cycle consists of 11 phases, of which five are systolic and six are diastolic (a simplified diagram is presented in Chapter IV). The systolic phases include the phases of asynchronous and isometric contraction (their total length corresponds to the length of the period of stress), the protosphygmic interval (the transitional interphase state), the phases of maximum and reduced expulsion (adding up to the expulsion period). Thus, the total systole can be characterized as a portion of the cardiac cycle during which the contractive process occurs in the myocardium. The mechanical systole is the summary length of the phases of isometric contraction and the period of expulsion.

During the mechanical systole, high pressure is maintained within the ventricles (for more details concerning the norms of phase lengths in the systole of the left ventricle and the specifics of the phase structure of the cardiac cycle in athletes, see Chapter IV).

The most widespread method of investigation of the phase structure of the cardiac cycle is polycardiography (synchronous recording of electrocardiogram, phonocardiogram and sphygmogram in the carotid artery) which, like indirect methods, allows only analysis of the dynamics of the left ventricle.

The time characteristics of the contractive activity of the right and left ventricles differ slightly. Whereas the beginning of the contractive process in the two ventricles is practically snychronous, the increase in pressure in the left ventricle begins earlier than in the right ventricle (the mitral valve closes before the tricuspid valve) as a result of which the asynchronous contraction of the right ventricle is longer than that of the left ventricle. The pulmonary valve opens before the aortal valve, which means that the phase of isometric contraction of the right ventricle is shorter than that of the left ventricle. Since under normal conditions the aortal valve closes before the pulmonary valve, while the length of the protodiastolic interval in both ventricles is practically the same, the expulsion of blood from the right ventricle requires more time than from the left. Due to this, the duration of the total systole of the left ventricle is 0.02-0.05 sec shorter than that of the right. In the right ventricle, the duration of the phases of isometric relaxation, rapid and slow filling are short (in comparison to the left ventricle). The systole of the right artium begins before tnat of the left.

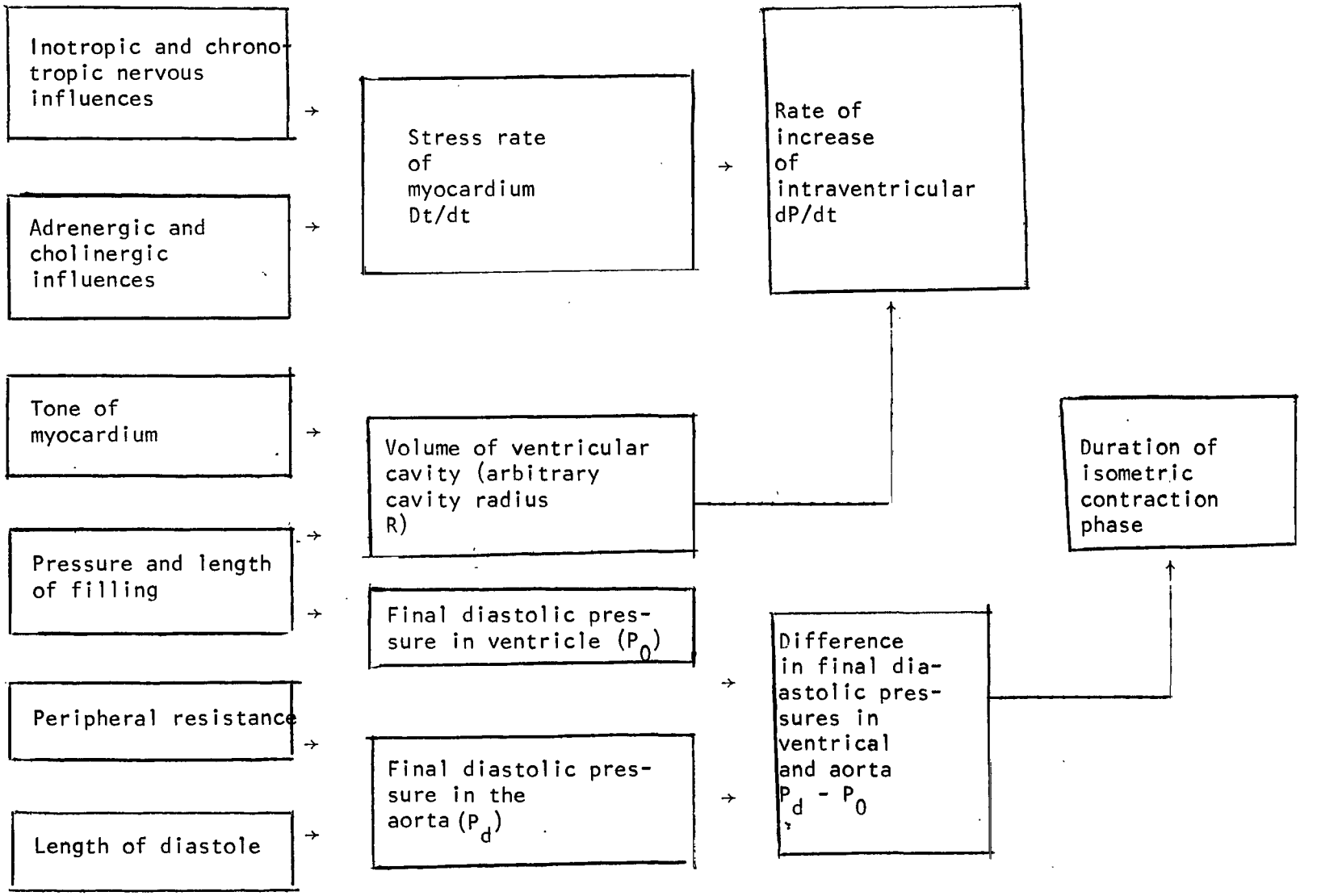
The length of the phases of the cardiac cycle is determined by the interaction of many factors.

Thus, the duration of the phase of asynchronous contraction[1] is determined by the filling pressure, changes in the tone of the heart, resulting from nervous influences, the intensity of metabolic processes in the myocardium, regulating the rate of propagation of depolarization and the value of electromechanical latency in each contracting element and, possibly, by the degree of hypertrophy of the myocardium (Blumberger, 1940; V. L. Karpman, 1964; I. N. Ivanitskaya, 1965; V. N. Brikker, 1965; etc.). The interaction of these factors in athletes determines the moderate lengthening of this phase at rest. The length of the phase of asynchronous contraction, in the opinion of the overwhelming majority of investigators, does not depend on the cardiac rhythm. However, A. D. Butkov (1965) discovered that when the pulse frequency was sharply decreased in athletes (28-36 beats per minute) the length of the phase of asynchronous contraction was statistically reliably lengthened in comparison with values observed with normal heart rhythm. The determinants of phase length of isometric contraction were even more widely varied. Thus, volume loading leads to a shortening of the length of this phase. On the other hand, underloading leads to a lengthening of the isometric contraction. Thus, a change in venous influx of blood to the heart influences the duration of this phase (Wiggers, 1921; V. L. Karpman, I. N. Ivanitskaya, L. A. Ioffe, Yu. K. Shkhvatsabaya, 1965; etc.). The duration of the phase depends essentially on the final diastolic pressure in the principal vessels. An increase in pressure in the pulmonary artery and an increase in pulmonary artery resistance leads to a lengthening of the phase of isometric contraction of the right ventricle (V. L. Karpman, V. S. Savel'yev, 1960), while a lengthening of the isometric contraction of the left ventricle results from corresponding changes in the systemic circulation (Wiggers, 1921; Blumberger, 1940; V. L. Karpman, 1961). There is a weak correlation between the length of this phase and the pulse frequency (as the pulse rhythm increases, the length of the phase is shortened). Predominance of adrenergic influences shortens the phase, while cholinergic influences lengthen it. /26

In his special investigations, Blumberger (1957, 1958) showed that the duration of the phase of isometric contraction was determined by the degree of presystolic stress of the myocardium, its contractability, the pressure gradient in the ventricle-aorta system at the end of the diastole, as well as the inotropic influences on the myocardium. Modern concepts concerning the mechanisms whose interaction determines the length of the phase of isometric contraction are shown on the diagram below.

[1] The period of change in form of ventricles, transformation, deformation, conversion, etc., in the terminology of various authors. Here and in the following we will use the terminology of V. L. Karpman.

DIAGRAM
After V. L. Karpman, 1965



As follows from this diagram, the direct determinants of the length of isometric contraction are the rate of increase in intraventricular pressure ($dP/dt = V_i$) and the pressure gradient in the aorta and ventricle at the end of the diastole ($P_d - P_0$).

The duration of the period of expulsion of the left and right ventricles is closely related to the cardiac rhythm, decreasing as the rhythm increases and increasing as the pulse rate slows (Herkel, 1938; Blumberger, 1940; V. L. Karpman, 1963; etc.). There is also a dependence between the duration of expulsion and the systolic output: the higher the volume of blood per beat, the longer its expulsion requires (Blumberger, 1957; Gobbato and Meda, 1956; etc.). Jones and Foster (1964) also showed that the duration of the period of expulsion was influenced by the diastolic pressure. They described the dependence between the duration of the period of expulsion (E), the /28
systolic index (SI), the cardiac rhythm (R) and the diastolic pressure in the aorta (P_d) as follows:

$$E = 0{,}36646 - 0{,}00108 \cdot R - 0{,}00036 \cdot P_d + 0{,}00076SI. \qquad (2)$$

On the basis of factorial analysis, the authors came to the conclusion that the principal determinant of the duration of the expulsion period is still the cardiac rhythm.

The duration of the diastole is primarily determined by the frequency of cardiac contractions. The duration of the protodiastolic interval varies little under physiological conditions. Its fluctuations are determined by the rate of relaxation of the myocardium and the rate of blood flow from the main vessels. The duration of the phase of isometric relaxation of the ventricles depends on the duration of the protodiastole, the pressure in the atrium and the rate of relaxation of the myocardium.

The duration of rapid filling of the ventricles is primarily related to the resistance to blood flow in the area of the atrioventricular apertures and the volume of the ventricular cavities.

The duration of the phase of slow filling of dthe ventricles (diastasis) is closely correlated to the cardiac rhythm: it decreases in proportion to an increase in pulse frequency. During this phase, filling of the ventricles is slight, which is indicated by the slight change in dimensions of the heart (V. L. Karpman, V. S. Sinyakov, 1965) and increase in pressure in the atrium due to the accumulation of blood in them.

During the systole of the atrium, "forced" filling of the ventricles with blood is performed. The hemodynamic effectiveness of the systole of the atrium decreases as the negative inotropic influences are reinforced and increases under influence of adrenaline.

The specifics of the dynamics of cardiac contractions under various physiological and pathological conditions reflect changes in extracardiac factors, the activity of the ventricles and the state of the contractive function of the myocardium. In spite of the more than 100 years history of study of the phase structure of the cardiac cycle, Blumberger (1942) first demonstrated the significance of phase analysis for evaluation of the contractive activity of the heart only in the 1940's. His work aided in the introduction of phase analysis to clinical practice.

Objective analysis of the physiological mechanisms determining the specifics of cardiac contraction under various conditions was performed by V. L. Karpman (1964). He established that when normal functioning is disrupted and under the influence of various physiological factors, five characteristic combinations of changes in the duration of cardiac cycle phases are observed, which he represented as phase shift syndromes. Thus, for the volume loaded phase syndrome, a shortening of the isometric contraction phase is characteristic, along with lengthening of the expulsion period, /29 an increase in the initial rate of increase of intraventricular pressure, intrasystolic index and mean rate of blood expulsion, and a decrease in the myocardial stress index.

Under physiological conditions, volume loading is a result of an increase in venous return of blood to the heart (clino-orthostatic test, head downward position, cessation of stress, etc.).

Volume loading is accompanied by development of compensatory cardiac hyperfunction, leading to an increase in cardiac output. Reinforcement of the cardiac contraction is achieved by activity of the Frank-Starling mechanism (cardiac heterometric self-regulation), during which the myocardial stress increases resulting from an increase in presystolic extension of its fibers.

Various forms of arterial hypertension are accompanied by development of the phase syndrome of high diastolic pressure. This syndrome is characterized by an increase in the phase of isometric contraction, a tendency to abbreviation of the period of expulsion, a lengthening of the mechanical systole, a decrease in intrasystolic index, an increase in the myocardial stress index and the rate of emptying of the heart. This is the reaction of the heart to loading by an "elastic" resistance.

During the phase syndrome of stenosis of the output channel of the ventricle (aortal or pulmonary stenosis), a shortening of the phase of isometric contraction is observed, along with lengthening of the period of expulsion and mechanical systole, an increase in the initial rate of intraventricular pressure rise, an increase in intrasystolic index and a decrease in the rate of blood expulsion. This is the reaction of the heart to loading by a "hydraulic" resistance.

During resistance loading, compensatory hyperfunction arises, based on the Anrep phenomenon (one of the mechanisms of homeometric self-regulation), according to which the myocardial stress increases in response to an increase in resistance to expulsion.

The dynamics of the cardiac contraction during these syndromes is determined by changes in hydrodynamic operating regime of the heart.

The characteristic changes in the phase structure of the cardiac cycle are observed upon changes in regulatory inotropic influences and disruption of the contractive function of the myocardium. Thus, muscular work, emotional stress and certain pathological conditions (thyrotoxicosis, high temperature, associative and residual hyperfunction, etc.) lead to reinforcement of cardiac contractions. This reinforcement is an essential part of the phase syndrome of myocardial hyperdynamia, for which a shortening of the phase of isometric contraction, the expulsion period and mechanical systole and an increase in intrasystolic index, initial rate of intraventricular pressure rise and rate of blood expulsion are characteristic. The power of /30
the cardiac contraction is increased during this syndrome by positive inotropy of the myocardial nervous stimulation. This same effect is achieved when catecholamines, cardiac glycosides and certain other humoral agents act on the myocardium.

When the power of the cardiac contraction is decreased, the phase syndrome of myocardial hypodynamia arises: lengthening of the phase of isometric contraction, shortening of the period of expulsion and sometimes of the mechanical systole, a reduction in the initial rate of intraventricular pressure rise, a decrease in intrasystolic index and an increase in the myocardial stress index.

This syndrome is observed when the contractive capacity of the myocardium is decreased, while extracardiac conditions remain unchanged.

In case of insufficient filling of the ventricles with blood, functional hypodynamia arises (for example, during the Valsalva experiment, the orthostatic test or in mitral stenosis).

In highly trained athletes at rest, characteristic signs of the phase syndrome of myocardial hypodynamia are observed. Reinforced activity of the sympathetic-inhibitor mechanisms and cholinergic influences, an increase in the central tone of the vagus nerve (V. Raab, 1959, 1963) lead to a reduction in the rate of intraventricular pressure rise and phase shift delays. Thus, the phase syndrome of myocardial hypodynamia developes in athletes as a result of changes in the regulation of cardiac activity related to the effects of systematic training, while in certain patients, similar phase shifts result from disruption of the contractability of the myocardium. This distinction is important: a change in regulatory action such as this provides high functional reserve of the myocardium.

Recently A. D. Butkov (1966) showed that during high training loads the shifts characteristic for the myocardial hypodynamia phase syndrome are regularly supplemented by a lengthening of the period of expulsion and disappearance of the electromechanical difference (duration of the Q-T interval and total systole become practically identical, while the Q-T interval is usually 0.02-0.04 sec longer). These changes are specific for the phase syndrome of acute fatigue (A. D. Butkov, 1966), not included among those described by V. L. Karpman (1964). As the functional state of the myocardium is improved, the characteristics of acute fatigue disappear and the chronocardiogram of athletes once more takes on features of the phase syndrome of myocardial hypodynamia. These facts are of considerable significance for objective control of the training process.

A certain arbitrariness in separating individual phase syndromes results from the fact that the characteristic picture of various shifts may be /31
distorted by compensatory-adaptive reactions of the circulatory apparatus. Atypical phase shifts may develop under conditions of volume loading or resistance loading when the contractive function of the myocardium is disrupted. However, failure of the phase structure of the cardiac cycle to correspond to the expected changes is important for an objective evaluation of the contractive activity of the heart.

Relationship of Electrical and Mechanical Manifestations of Cardiac Activity

The analysis of electromechanical interactions in the heart as a whole is most frequently performed on the basis of a comparison of the electrocardiogram and phonocardiogram. In addition to this, the electrocardiogram is compared to curves of pressure in the cavities of the heart, curves of the apical shock, dynamocardiogram, electrocardiogram, curves of the central pulse, kinetocardiogram and other curves reflecting the mechanical manifestations of the activity of the heart.

The time dissociation between the beginning of electrical and mechanical processes in the heart allows us to distinguish the so-called electromechanical latency. There are two independent variants of this latency (I. N. Ivanitskaya and V. L. Karpman, 1966; etc.). Its first form -- the electrokinematic latent period -- reflects the time from the beginning of the Q wave (beginning of depolarization of the ventricles) to the moment of first notable manifestation of kinematics of the heart, which, however, is still not accompanied by an increase in pressure in the ventricles. The completion of this period corresponds to the beginning of the low-frequency oscillations of tone I, the specific wave on the cardiogram, etc. The duration of the electrokinematic latent period varies from 0.01 to 0.04 sec and does not change essentially under various physiological and pathological conditions. Z. B. Belotserkovskiy (1956) found changes in the duration of this interval in athletes (in comparison to the data of untrained persons).

The second form of electromechanical latency -- the electrodynamic latent period (or phase of asynchronous contraction) -- was characterized by

the time from the Q wave to the beginning of the increase in intraventricular pressure. The factors determining the duration of this interval and its dynamics in athletes are analyzed in Chapters I and IV.

During the analysis of the electromechanical relationships, two more aspects are of interest: 1) the interrelationships between the duration of the electrical and mechanical systoles; 2) the time analysis between the completion of the electrical and mechanical phenomena in the heart.

In comparing the total duration of the electrical and mechanical systoles, it should be kept in mind that the overwhelming majority of the /32
formulas describing the interrelationship between these periods of the cardiac cycle and the cardiac cycle as a whole have been concluded for normal values of cardiac rhythm (fluctuations of the duration of the cardiac cycle between 0.6 and 1.2 sec).

In order to analyze the interrelationships between the duration of the electrical systole and the duration of the cardiac systole in athletes, apparently, the most suitable formulas are those of I. N. Ivanitskaya and V. L. Karpman (1966), A. D. Budkov (1966). It might be assumed that the equations of Butkov, which he produced in the analysis of electrocardiograms of swimmers at a high level of training (participants in the collective team of the USSR) more precisely describe the interrelationship between the electrical systole and the cardiac rhythm in athletes trained for endurance.

The problem is that the absolute value of duration of the Q-T interval, in connection with the delay of the cardiac rhythm in athletes, is frequently increased. Thus, in highly trained athletes the duration of the electrical systole in 29.4% of the observations varied from 0.41 to 0.50 sec (S. P. Letunov, 1957). Lengthening is most frequently noted in marathon racers and skiers, i.e. those athletes specially trained for endurance.

In an investigation of highly qualified swimmers, A. D. Butkov (1966) noted statistically reliable lengthening of the Q-T interval (even with normal duration of the cardiac cycle). In these sportsmen, the interrelationship between the duration of the Q-T interval and the cardiac cycle can be satisfactorily described as follows:

$$S_э = 0{,}16 \cdot C + 0{,}250 \quad (3)$$

$$S_э = 0{,}415 \cdot C^{0{,}415} \quad (3a)$$

where S_e is the duration of the electrical systole (the Q-T interval),

C is the duration of the cardiac cycle (R-R interval).

The formulas of Ye. B. Babskiy, I. N. Ivanitskaya and V. L. Karpman have the following form (symbols same as in formulas (3) and (3a)):

$$S_e = 0{,}124 \cdot C + 0{,}256 \quad (4)$$

$$S_e = \sqrt[3]{0{,}383 \cdot C}. \quad (4a)$$

L. A. Butchenko, G. Bruschke and Kh. Burger (1965) describe the dependence between the duration of the electrical systole and the pulse frequency (between 40 and 175 beats per minute). The authors analyzed data produced both at rest and during muscular work (see Figure 22). The graphical dependence can apparently be satisfactorily described by a logarithmic equation. Still, this approach cannot be acknowledged as completely correct, since the changes in the electrical systole under conditions of work follow laws different from those which regulate the duration of the Q-T interval during muscular activity. Incidentally, this /33 follows from the works of L. A. Butchenko and N. I. Vol'nova (1963), from which we can see that practically none of the existing formulas can satisfactorily describe the interrelationship between the electrical systole and the cardiac rhythm during muscular work.

In comparing the duration of the electrical systole and the duration of the mechanical systole, the judgment concerning the mechanical systole is generally made on the basis of the duration of the Q-II tone interval (i.e., the total length of the systole and the protodiastole).

The duration of the Q-II tone interval, as was established by I. N. Ivanitskaya, L. A. Ioffe, V. L. Karpman and G. V. Sadovskaya (1964) depends on the duration of the cardiac cycle as follows:

$$Q \cdots // \text{ tone} = 0{,}3 \cdot C^{0{,}36} + 0{,}085. \quad (5)$$

In this formula, $0.3 \cdot C^{0.36}$ corresponds to the duration of the mechanical systole, while 0.085 corresponds to the mean summary length of the phase of asynchronous contraction and the protodiastolic interval (V. L. Karpman, 1963).

In athletes, the actual length of the Q-II tone interval frequently exceeds the needed length in connection to the elongation of the phase of asynchronous contraction. In the case of acute myocardial fatigue syndrome, the duration of the Q-II tone interval, generally, is significantly elongated due to lengthening of the phase of isometric contraction and the expulsion period.

A comparison of formulas for the Q-II tone interval (5) and the electrical systole (4) shows that with normal rhythm (i.e. with a duration of the cardiac cycle from 0.65 to 1 sec), the required value of Q-II tone interval slightly exceeds the duration of the Q-T interval, whereas in bradycardia, the inverse relationship is observed, the time of dissociation generally being more expressed. It is possible that the relatively frequently observed fact of II tone preceding the T wave by 0.04 sec observed

in athletes (Hegglin phenomenon) is related to this fact. We note in passing that the Hegglin phenomenon is erroneously called the Hegglin syndrome by some authors. In this syndrome, the precedence of the II tone before the end of the T wave is not less than 0.04 sec, and is determined by lengthening of the electrical systole with simultaneous shortening of the Q-II tone interval in comparison with the normal values calculated from the Hegglin-Holzmann formulas. The Hegglin syndrome is, in the opinion of the author, a manifestation of energetic-dynamic deficiency of the heart, i.e., primary weakening of the myocardium, which may be caused by disruption of the mineral or energetic exchange, toxicosis, etc.

Hegglin (1947) assumed that if the myocardium of the left ventricle is weakened for one reason or another, it is not capable of overcoming the resistance in the aorta over the time period required, contraction is terminated early, which is documented on the phonocardiogram by early formation of the II tone. Lengthening of the Q-T interval, in the opinion of Hegglin, results from energetic deficiency of the myocardium. The separation of energetic-dynamic deficiency as a particular form of cardiac deficiency is disputed by many investogators (Lagrand et al., 1959; Brenner, Zpiv, 1952; V. N. Brikker, 1960). /34

The Hegglin phenomenon (but not syndrome) is relatively frequently observed in athletes at rest (Emmrich et al., 1962; I. N. Ivanitskaya, 1963, 1965; Yu. K. Shkhvatsabaya, 1964, 1965) and, as a rule, is not a reflection of any worsening in the contractability of the myocardium. Nevertheless, cases of this type should be carefully analyzed.

It was indicated above that as the cardiac rhythm is slowed, the duration of the Q-II tone interval is shortened in comparison with the electrical systole. This conclusion is confirmed by the observations of Emmrich, Weissleder and Reindell (1962), according to which the duration of the Q-T interval and the Q-II tone interval practically corresponds with a pulse frequency of 59-62 beats per minute (individual groups of light athletes, bicyclists, rowers and swimmers were studied). With a mean frequency of 52 beats per minute (skiers) the duration of the Q-II tone interval was 0.39 sec, while the Q-T interval was 0.405 sec. Similar data were produced by A. D. Butkov (1966).

Blood Supply of the Heart

One of the most important factors in providing normal cardiac muscle function is the state of coronary blood flow. The blood supplied to the heart is unique in comparison to the other organs. The coronary blood flow amounts to only 8-10% of the cardiac output. However, the weight of the heart is only 0.4-0.5% of the weight of the body, meaning that the blood flow to the heart is 10-15 times more intense than to other tissues of the organism.

Measurement of the coronary blood flow in man became possible after catheterization of the veinous sinus was achieved. In 1949, Bing et al.

first determined the value of coronary blood flow in man using nitrous oxide intake and found that in healthy persons it is 65 mℓ per 100 g heart weight. According to the data of Rowe (1959), who made up a summary table from the results of the investigations of seven authors, the coronary blood flow in healthy persons averages 80.6 mℓ per 100 g heart weight. In women, the coronary blood flow was 26.5% higher than in men (respectively 110 and 72 mℓ per 100 g weight of the heart per minute).

In athletes, the coronary blood flow has only begun to be studied (Keul et al., 1966). At the present time, certain data have been accumulated concerning the factors which determine the state of coronary blood flow, which allow us to represent the changes in coronary blood flow during work and under the influence of sports training. /35

The intensity of changes in cardiac activity are determined by the demands of the organism. For example, during physical loading, the volume of blood per minute may be increased from 4-5 to 25-30 ℓ/min for several minutes. Energy expenditures of the myocardium increase sharply in this case, and its demands for blood supply are also increased. Supporting the activity of the heart at the new, quantitatively and qualitatively different level, is achieved by a corresponding increase in the coronary blood flow. From 200-250 mℓ/min at rest, the coronary blood flow under intensive muscular work conditions may reach 3.0-4.6 ℓ/min.

One distinguishing feature of the blood supply to the heart is the fact that the coronary blood flow is approximately 10-15 times greater than the mean blood flow in other tissues of the organism.

One anatomical specific of the coronary vessels is the fact that, in contrast to the peripheral vessels, the arterial coronary lumen is quite short, while the capillary lumen is quite great (Brose, Schaeffer, 1953). According to the data of Roberts and Wearn (1941), in a normal heart there are over 4000 capillaries per mm^2, while in a hypertrophic heart (pathological) there are about 2500. However, myocardial hypertrophy resulting from sports training is accompanied by the development of a powerful capillary network. According to the data of Petrin, Fjostrand, Sylven (1936), D. S. Sarkisov (1964) and others, the quantity of capillaries in the heart after hypertrophy as a result of training increases not only absolutely, but relatively (per unit area) as well. The development of capillaries provides for sufficient blood supply to the hypertrophic myocardium both at rest and during work and is one of the factors in the increased working ability of the athletic heart. Normal blood supply to the heart is provided by an interaction of many regulatory factors, which are arbitrarily divided into cardiac and extracardiac factors (diagram below).

Among the cardiac factors in regulation of the coronary blood supply we include: 1) the level of metabolic processes in the myocardium (there is a linear dependence between the consumption of oxygen and coronary blood flow); 2) pressure in the aorta (as the aortal pressure is increased, coronary blood flow increases, and vice versa); 3) the cardiac rhythm (increasing the pulse frequency is accompanied by an increase in coronary blood flow, proportional

to the increase in work of the heart and consumption of oxygen by the myocardium); 4) phase influences on the intensity of the coronary blood flow (the coronary blood flow is decreased during the systole, particularly sharply during the phase of isometric contraction, and an increase during the diastole); 5) the tone of the coronary arteries (the tone is decreased as the /37
energy requirements are increased, in case of oxygen shortage, and increase during an overabundance of oxygen, providing correspondence of coronary blood supply to the level of metabolic processes in the myocardium.

The extracardial factors include many reflector actions both directly, and through the cardiac factors influencing the state of blood supply of the heart.

Nervous regulation is a necessary link in the complex mechanism providing adaptation of coronary blood flow to the demands of the myocardium. When the innervation of the heart is disrupted, myocardial hyperfunction, increased level of metabolism and other factors not only do not cause an increase in coronary blood flow, but frequently lead to the reverse reactions.

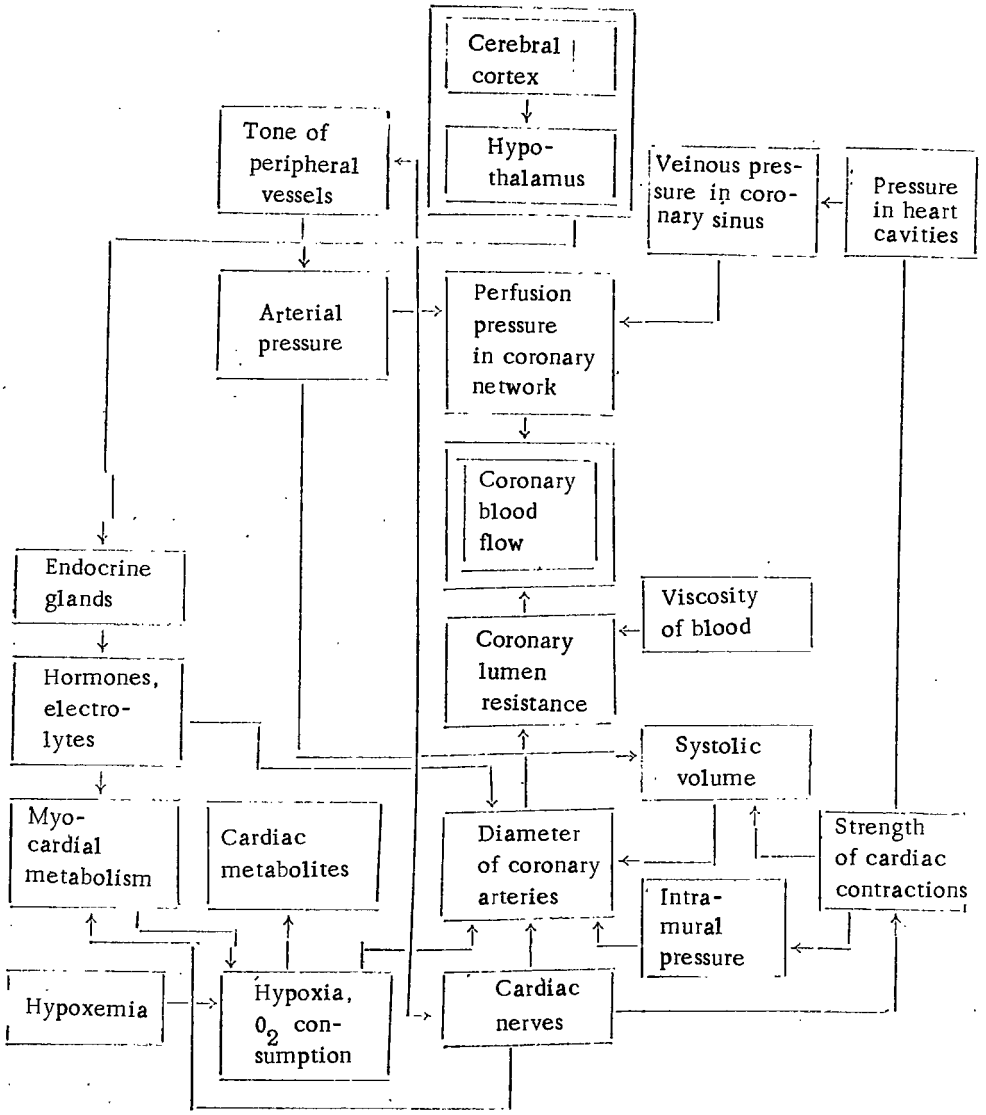
The dependence of the state of coronary blood supply on the influence of the brain, higher vegetative centers and the vascular motor center has been confirmed by many clinical observations. Strong excitement caused by an unexpected situation -- an alarm signal, fear before operations, etc. (Mainzer, 1953; S. I. Teplov, 1962; N. I. Speranskiy and V. I. Ostrovskaya 1955; Sigler, 1961; Adsett et al., 1962) and the so-called daily stressors -- "ordinary" unrest related to professional activity (Ira et al., 1963; G. I. Kositskiy, 1964) are accompanied by changes in coronary blood supply.

An important role in the regulation of coronary blood flow is played by humoral factors. A change in the content of oxygen, carbon dioxide, adrenaline, noradrenaline, acetylcholin, vassopressin and certain other materials leads to changes in the cross section of the coronary vessels, thus influencing the intensity of coronary blood supply.

Certain Specifics in the Regulation of Cardiac Activity in Athletes

Systematic training essentially in any form of sports leads to an increase in the functional capacities of the heart. These changes are most sharply expressed in athletes especially trained for endurance. Runners for middle, long and very long distances, skiers, swimmers, etc. show considerably more frequently than other athletes a tendency to a decrease in atrioventricular conductivity, moderate hypertrophy of the myocardium and an increase in the size of the heart, accompanied by development of a thick network of capillaries, absolute and relative increase in residual blood volume, a decrease in the blood circulation volum e per minute, an increase in the blood flow time, characteristic changes in the phase structure of the /38
cardiac contraction (myocardial hypodynamia syndrome).

DIAGRAM
(According to S. I. Teplov, 1962)



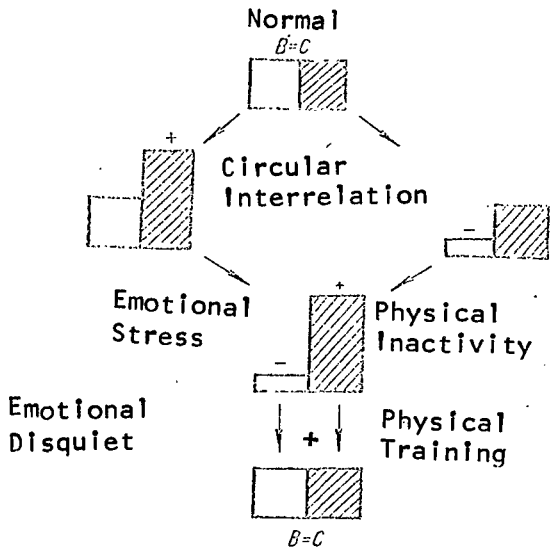


Figure 1. Interrelation of Vagus and Sympathetic-Inhibitory Mechanisms (B) and Sympathetic (C) Mechanisms in Normal Conditions, Under Conditions of Emotional Stress and Decreased Physical Activity, and After Physical Exercises and Emotional Unrest (According to V. Raab, 1963)

All of these changes result from an increase in the activity of the sympathetic-inhibitory mechanisms and an increase in central tone of the vagus nerves. Thus, V. Raab (1959) indicates that in the opinion of Reindell (1943), even the phenomenon of tonogenic dilation is determined by the "relatively tonotropic" influence of the vagus nerve. Changes in the rhythm, atrioventricular conductivity and certain manifestations of excitability are related to reinforcement of negative chrono-, dromo- and bathmotropic effects. Here, a sharp increase in the central tone of the vagus nerve leads to an expressed negative dromotropic influence. Reinforcement of negative inotropic influences of the vagus nerve leads to a decrease in the blood volume per minute, a decrease in the external work of the heart, a slowing of the initial rate of increase of intraventricular pressure and characteristic phase shifts.

Apparently, the condition of the skeletal musculature is of great significance in changes in the regulation developing under the influence of systematic training and the transformation of the activity of the cardiovascular system. According to the investigations of I. A. Arshavskiy (1964), the intensity of energy expenditures and the corresponding level of activity of the respiratory and cardiovascular system during rest are determined primarily by dynamic components of load, carried by the skeletal muscles. The investigations conducted by D. U. Ermatova (1965) with rats and rabbits are of interest. After special training (swimming) causing considerable changes in the state of the skeletal musculature of the experimental animals, a slowdown in the natural respiratory and cardiac rhythms was noted, by a factor of almost 2. The rabbits during the process of training showed respiratory arhythmia, indicating the /39
appearance of tonus in the central nuclei of the vagus nerve (under ordinary conditions, vagus tonus is not present in these animals). The introduction atropine eliminated the respiratory changes in the cardiac rhythm, thus confirming the relationship of their appearance with the formation of tonus of the vagus nerve.

In the opinion of M. R. Mogandovich (1957), the influence of skeletal musculature on the vegetative functions is achieved due to the regulating

influence of the proprioceptive impulsation (motor-visceral reflexes). The interrelationship of motor and vegetative functions is particularly clear during the process of training: improvement of the state of training is accompanied by an improvement in the functional state of the circulatory apparatus.

Many investigations, including a particularly well directed work by V. Raab (1959, 1963), have shown that when movement is insufficient, changes in the regulation of cardiac activity are noted. These regulatory changes are characterized by predominance of the influence of the sympathetic system, resulting both from an absolute increase in the sympathetic tonus, and from a decrease in the activity of cholinergic and sympathetic-inhibitory mechanisms. V. Raab (1963) indicates that the absolute predominance of sympathetic systemic effects results from emotional factors, while relative predominance may result from physical inactivity (Figure 1). A reinforcement of sympathetic influences on the heart may be pathogenic in nature. Cardiovascular diseases are most frequent among those who lead primarily sedentary lives (being particularly frequent if there are accompanying emotional stresses). From the diagram (Figure 1) we see that emotional disquiet and physical training lead to the development of protective, sympathetic-inhibitory and cholinergic mechanisms.

Thus, the relationship between the level of motor activity and the state of regulation of cardiac activity is obvious. The regulatory changes resulting from sports exercises are characterized by reinforced negative chrono-, bathmo-, dromo- and inotropic influences of the vagus nerve and an increase in the activity of the sympathetic-inhibitory and cholinergic mechanisms at rest. Cholinergic features of homeostasis, accumulated as a result of sports training, are the basis of the "economy" of the manifestations of vital activity at rest and result in an increase in the reserve capacities of the organism in connection to increased potential lability of the skeletal-muscular, respiratory and cardiovascular systems in response to various stress factors, including physical loading (I. A. Arshovskiy, 1964-1967).

CHAPTER II

CARDIAC ACTIVITY DURING CONDITIONS OF MUSCULAR WORK

ABSTRACT. The response of cardiodynamics to physical stress is studied. Transient part cardiodynamic proccesses during muscular work are diagrammed and described. Specifically, the alterations in cardiac rhythm, blood pressure, blood volume per beat, permitted a residual blood volume in the heart, as well as relationships between phases of the cardiac cycle as a function of physical stress level are studied. The electromechanical relationships before, during and after physical stress are described, and the recovery period following physical stress is studied.

The contemporary state of development of sports medicine is characterized by ever broader interest in investigations dedicated to a study of the behavior of various systems of the organism and, in particular, the circulatory apparatus directly during muscular work. This type of investigation is becoming possible to a considerable extent due to the technical revolution occurring in circulatory physiology. The recording of various parameters of cardio- and hemodynamics is being performed using highly sensitive parametric and oscillator type transducers. The free behavior of the test subject during muscular work is provided due to the radiotelemetric transmission of physiological information or transmission of this information through special electrical transmission wires. /40

It should be noted that up to quite recent times the problem of investigation of cardiodynamics during muscular stress was solved by comparison of physiological information produced before and at various times after physical loads. However, at the present time there is no doubt that a judgment of the reaction of the cardiovascular system to load based on data of the restorative period is far from complete from the physiological point of view, since the cardiodynamics and hemodynamics under these conditions differ essentially from those observed during muscular activity both quantitatively and qualitatively.

There are not only technical difficulties preventing broad extension of investigations of cardiodynamics during work. The problem is that we still do not have the corresponding methodological apparatus with which we could perform the objective, quantitative analysis of the multifaceted cardiological information produced in experiments with muscular work necessary for sports medical practice. We might find one of the component parts of cybernetics -- the theory of automatic control -- promising and practically /41

useful here. We have in mind primarily borrowing from the theory of automatic control new objective criteria, not yet used in medicine, which might be used to evaluate the reaction of the cardiovascular system to various types of perturbation. Using the concepts from the theory of automatic control, we can produce data, for example, on such new criteria for biology as the "quantity of regulation" of a system.

Investigation of the quantity of regulation in the circulatory apparatus might be performed by analysis of curves of the transient processes constructed according to the data of continuous or partially discrete registration of parameters of cardiodynamics (cardiac output, arterial pressure, phases of the cardiac cycle, cardiac rhythm, etc.) during muscular work.

Transient processes, as we know, characterize the changes of a parameter being studied in regulation systems, arising under the influence of perturbing actions. For the cardiovascular system, which is under the control of a complex biological regulation system, one of the most frequently encountered perturbing factors is muscular work.

Certain General Regularities in the Restructuring of Cardiodynamics During Physical Stress

Under the influence of muscular work, changes in cardiac activity are generally completed in two stages. The first of these corresponds to the working-in period. During this time, each parameter of cardiodynamics gradually changes its value from the value at rest to the value inherent to a given volume of work.

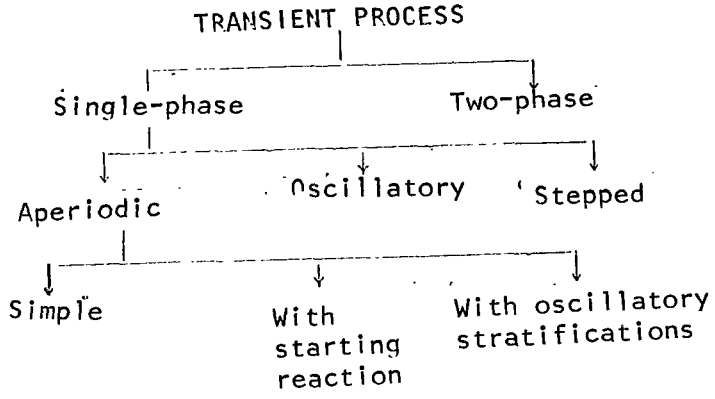
The second stage is characterized by a new stable mode of cardiac activity, which is represented in the literature as the steady state. The steady state of a system or link in a system is characterized by the fact that with constant perturbing influence, the regulated parameter remains constant or changes at constant rate.

Let us discuss some of the regularities of the changes of cardiodynamics during the working-in period and in the steady state.

An investigation of restructuring of cardiodynamics during the working-in period can be most conveniently conducted using the data from the reactions to a stepped perturbation. As concerns muscular work, this type of perturbation is characterized by a sudden beginning and end of the physical load, the performance of which is conducted at constant power. /42

On the basis of analysis of a large number of transient process curves constructed by our colleagues O. N. Delina, Ye. A. Yefimova, V. G. Leoshenko and Yu. K. Shkhvatsabaya for widely varied parameters of cardiodynamics (cardiac rhythm, atrioventricular behavior, asynchronous, isometric and isotonic contractions, mechanical and electrical systoles) as well as data produced by N. V. Zimkin and S. A. Razumov (1965), V. V. Matov and I. D. Surkina (1964), Cerretelli et al. (1964) and others, the possibility

has been created of systematizing the considerable variety of types of dynamics of electromechanical indicators of the activity of the heart during the working-in period. This systematization is presented on the following diagram:



Single-phased stratifications are characterized by the fact that after the beginning of work the parameter of cardiodynamics being studied begins to decrease (or increase) in comparison with its initial value (Figure 2), and the initial inclination of the curve determines its subsequent polarity. With two-phased transient processes, oscillations of the value of the parameter being studied in relation to its initial value accompanied by a change in polarity are characteristic (see Figure 2). For example, the duration of the period of expulsion under conditions of moderate muscular work first increases in comparison with the initial value; then, after half a minute, begins to decrease, becoming shorter than at rest. Single-phase transient processes are predominant, most frequently encountered in the /43 analysis of various parameters of cardiodynamics. Let us note one more important characteristic specific feature of this group of transient processes. It seems that they are observed primarily in those cases when the cardiac activity index being studied is the object of direct regulatory influences. For example, positive inotropic stimulation of the myocardium at the beginning of muscular work leads to an increase in the force of the cardiac contraction, which is directly illustrated by a shortening of the isometric contraction and an increase in the rate of intraventricular pressure rise.

The problem is different in an evaluation of two-phase transient processes. In these cases, the parameter studied is regulated not directly, but indirectly as a result of deterministic changes in other parameters. For

example, the duration of the expulsion period depends both on ino- and chronotropic influences on the myocardium, and on the value of the volume of blood per beat. As a result, during muscular work of slight intensity, the increase in volume per beat leads to an elongation of the expulsion period; then, as the positive ino- and chronotropic influences are increased, its duration gradually shortens.

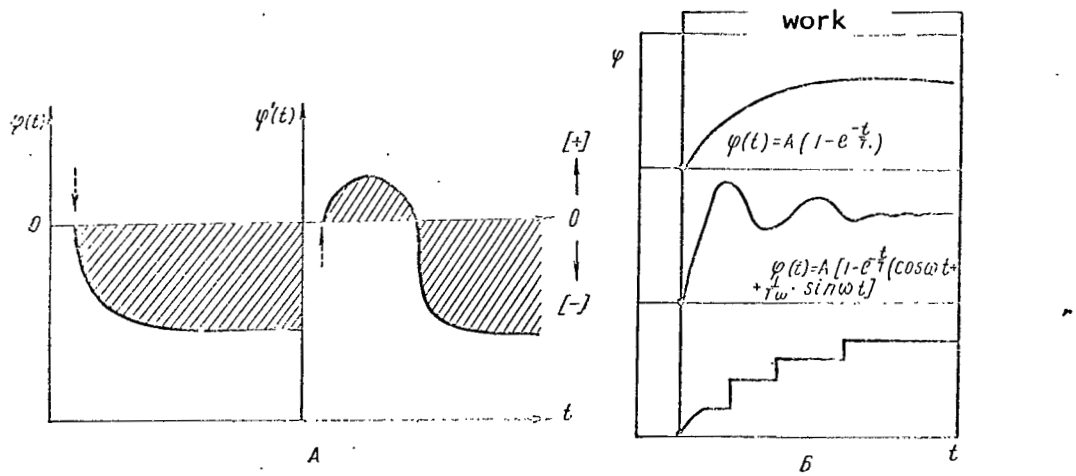


Figure 2. Transient Cardiodynamic Processes During Muscular Work. A, Single-phased and two-phased transient processes; the arrow shows the moment of beginning of work; B, Typical transient processes: aperiodic (top), oscillatory (middle) and stepped (bottom).

The parameters which are regulated indirectly, secondarily, do not always yield a two-phased transient process. Under certain conditions, the transient process may be single-phased. However, in this case it is considerably more highly inertial than the transient processes constructed for the directly regulated parameters. /44

Figure 2, B shows typical transient processes in cardiodynamics observed in trained athletes. A formal analysis of these processes allows us to speak quite obviously of the specifics of regulation of various mechanical manifestations of cardiac activity.

Under the conditions of performance of a laboratory experiment, the transient processes most frequently observed are aperiodic. According to the ideas of Dreshell (1960), aperiodic transient processes with damping of a system near critical indicate in biological objects optimality of the operation of the regulators. It is possible that it is for this reason that the change in cardiodynamics in athletes -- persons with the best "regulated" circulatory system -- are aperiodic.

We distinguish three types of aperiodic transient processes in athletes (V. L. Karpman, 1966). The first type corresponds to curves of transient processes observed in aperiodic lengths of first order automatic control systems. These transient processes are easily approximated by typical exponents (Figure 2, B).

The second type is at first glance reminiscent to an oscillatory transient process. However, a simple graphic reconstruction of this curve shows that it is composed of a "pure" exponential curve and a low-amplitude oscillatory component. This type of transient process is most frequently observed both in our investigations, and in the investigations of other authors (Greshell, 1960; N. V. Zimkin and S. A. Razumov, 1965; etc.).

The third type of aperiodic transient process is characterized by a sharp initial change in the parameter. This change may be observed in the first cardiac cycle after the beginning of muscular work. In order to approximate this type of transient process, we can use a typical exponential curve with the introduction of a constant coefficient at the variable. In this case at moment in time t = 0, i.e. at the beginning of work, the parameter is already changed. Under real conditions, naturally, a certain slight decay of this jump in relation to t = 0.

This type of transient process is distinguished by its high optimality as concerns speed of action. It is observed in highly trained athletes for whom, appearently, optimal stereotype reaction of the circulatory apparatus to physical load has been developed. /45

The oscillatory type of transient process in cardiodynamics (Figure 2, B) is characterized by reregulation, and has been observed in our investigations quite rarely. Apparently, the speed of operation of the system here is achieved inefficiently, and therefore during the process of training the oscillatory element is reduced to a minimum. The stepped transient process (Figure 2,B) on the other hand is observed comparatively frequently. This torpid type of restructuring of cardiodynamics is usually seen in the analysis of the dynamics of secondarily regulated parameters, for example duration of the mechanical systole in the ventricles.

During the working-in period, the activity of the biological regualtion system is similar to the operation of a self-adapting search control system in technology. Its principal task is to find the optimal (for a given intensity of muscular work) level of functioning of the heart.

The duration of the working-in period is relatively short. According to the data of a study of widely varying cardiodynamic parameters, it varies from 20-30 sec to 1 1/2-2 1/2 minutes. The working-in period usually consists of two stages -- the so-called start reaction and the stage of initial stabilization. It must be kept in mind that there is almost no clear boundary between the completion of the working-in period and the steady-state period in actuality. During the initial stabilizaiton, relatively slight fluctuations are observed (±15% in relation to the maximum value) in the cardiodynamic quantities.

Stabilization itself, according to its formal characteristics, should be characterized by complete absence of fluctuations in cardiodynamics. However, a slight level of fluctuation of a parameter should be allowed, not exceeding ±5% in relation to its maximum value, as is done in the theory of automatic regulation.

The degree of expression of shifts in the operation of the circulatory system depends essentially on the mechanical power of the work being performed in the steady state (V. G. Leoshenko, Yu. K. Shkhvatsabaya).

With low work power (up to 500 kg-m/min) the period of initial stabilization is greatly extended. If the work is short, the period of stabilization itself is not observed at all. When the work involves greater power (1700-2000 kg-m/min) the working-in period is replaced by the period of stabilization itself. The stabilization is generally complete, but deviations of a parameter may be considerable.

Up to the present time we have been speaking of changes in cardiac activity under the influence of muscular work performed in the steady state. /46
With unsteady work, the regularities of behavior of the circulatory apparatus naturally will be different.

Complex manifestations of muscular activity which cannot even be approximately described by a stepped function are characterized by an absence of the steady state on the curves of transient processes constructed from the data of various cardiodynamic parameters (Smodlaka, 1963; N. B. Kichaykina, 1965; Shnayder, Gattenlohner, 1965; V. L. Karpman, Ye. D. Yefimova and V. G. Leoshenko, 1967). The transient processes in this case will show the automatic search for the optimal cardiac activity mode for the varying power of the muscular work being performed by the test subjects (similar to the work of an extreme system of automatic control).

When intensive muscular work is performed in a discrete manner, a regularity is generally detected which we call the "Bowditch phenomenon with discrete muscular work." This regularity consists of the fact that with each subsequent cycle of work, the deviation of each parameter increases. This is illustrated on Figure 3 by the increase in oxygen consumption and decrease in duration of isometric contraction. As follows from this figure, the change in the curves is reminiscent of the curves describing the "Bowditch ladder" which allowed us to use the name just mentioned.

Cardiac Rhythm

The change in cardiac rhythm is one of the physiological mechanisms used for adaptation of the organism to muscular work. An increase in blood supply to the working organs and tissues is provided by a significant increase in the volume of circulation per minute. This latter in turn is /47
achieved to a considerable extent by increasing the frequency of contractions.

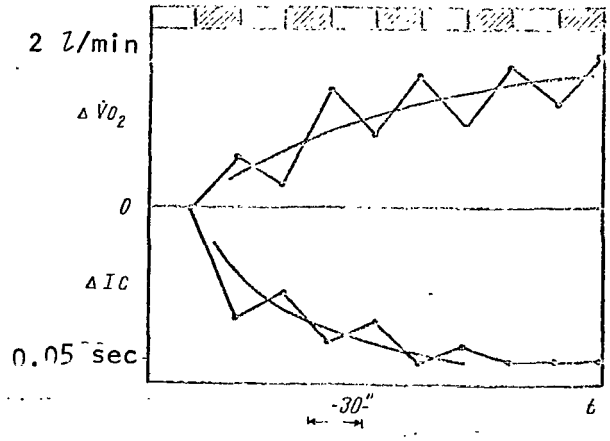


Figure 3. Bowditch Phenomenon with Discrete, High-Power Muscular Work. ΔVO_2, Increase in oxygen consumption; ΔIC, Acceleration of isometric contractions; shaded rectangles correspond to periods of physical load, unshaded rectangles to periods of rest.

Objective graphic recording of changes in cardiac rhythm during the process of muscular work began to be made at the beginning of the twentieth century (Bowen, 1904). Later, Knoll (1932) began to investigate the automatic functioning of the heart during work using an electrocardiograph.

An important contribution to this problem was later made using radiotelemetric transmission of electrocardiographic information (V. V. Rosenblatt, 1962; V. V. Vasil'yeva and V. P. Pravosudov, 1963; V. V. Matov and I. D. Surkina, 1964; etc.). Under conditions of the performance of muscular work, changes arise in the cardiac rhythm, the expression of which is determined by a large number of factors. The most important role belongs to the volume and power of work being performed.

Under conditions of sports activity, a pulse frequency of over 200 beats per minute is frequently observed. However, usually with rather intensive loads the pulse varies in different physically well trained persons between 160 and 200 beats per minute. The "standard working ability" is equated by Sjostrand (1954) to a pulse frequency of 170 beats per minute.

The efficiency of increasing the cardiac rhythm during the performance of physical exercises is analyzed in relation to the so-called critical pulse frequency. It is determined by that minimum length of the cardiac cycle, further shortening of which leads to a decrease in the effectiveness of the contractions (V. L. Karpman, 1964).

A. N. Krestovnikov (1951) assumed that at a pulse frequency of over 210 per minute, the length of the diastole is so reduced that the filling of the heart with blood begins to suffer. It should be noted here that the maximum possible pulse frequency in man according to Hoffmann and Pamerance (1958) is 360 beats per minute (in paroxysmal tachycardia). During sports activity, a frequency of up to 270 per minute has been recorded (V. S. Farfel' and M. V. Raskin, 1947; Christensen, Hogberg, 1958).

Figure 4 illustrates a typical picture of the change in pulse frequency during muscular activity. During the working-in period, the transient processes (according to data on the change in duration of the cardiac cycle)

are primarily aperiodic, but apparently they may be oscillatory in nature as well. The duration of the "search" for an optimal cardiac rhythm for given work may reach one-half minute, it being shorter, the more intensive the work (N. V. Zimkin and S. A. Razumov, 1965).

At the steady state, the Barcroft phenomenon becomes noticeable, which consists, as applicable to the pulse frequency, in that the expression of the physiological arhythmia of the pulse (respiratory and stochastic) during muscular work is considerably decreased. Thus, whereas at rest the variation coefficient of the duration of the cardiac cycle amounts to 5-9%, under work conditions the value of this index of arhythmia decreases to 1.0-1.5% (A. G. Falaleyev, 1964; V. M. Zatsiorskiy and S. K. Sarsaniya, 1964). /48

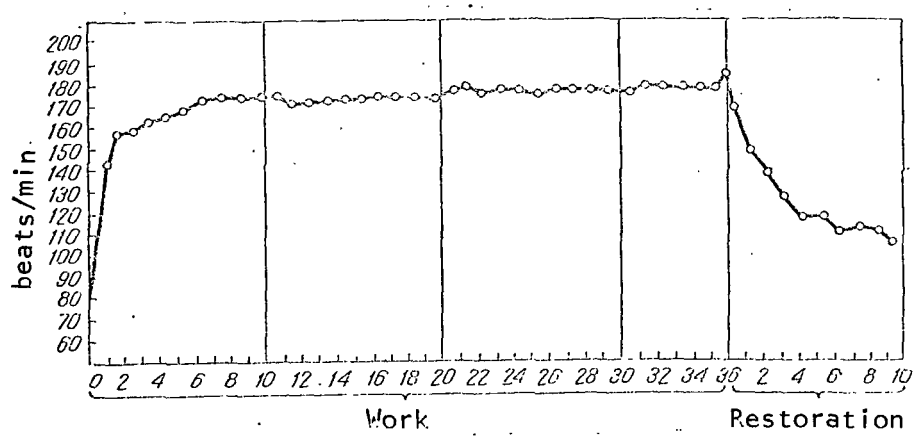


Figure 4. Change in Frequency of Heart Contractions During 10-km Run (radiotelemetric data of V. V. Vasil'yeva and V. P. Pravosudov, 1965)

V. M. Zatsiorskiy and S. K. Sarsaniya (1964-1966) performed a special investigation of the arhythmicity of cardiac activity directly during physical loads. The authors subjected the continual changes in duration of the cardiac cycles in the steady-state condition to autocorrelation analysis. They discovered a slow oscillatory component in the change in duration of the cardiac cycle with a period of an average of 25-30 sec. These slow "waves" of arhythmicity are apparently related to fluctuations in the sympathetic tonus.

With otherwise equivalent conditions, stabilization of the frequency of cardiac contractions depends on the level of training of the test subject. The performance of identical work, according to the data of A. N. Krestovnikov (1951), A. A. Arutsev (1963), V. V. Vasil'yeva and V. P. Pravosudov (1963) and others, is completed for well trained athletes with a lower cardiac rhythm than is the case with insufficiently trained athletes. The optimal zone of pulse frequency during intensive muscular work can be taken as 160-190 beats per minute. As the state of training of a test subject increases, the performance of the same work occurs at relatively lower pulse /49

rate (Brouha, 1960). N. A. Stepochkina (1965) showed that during extended, intensive work by persons trained for endurance, the initial increase in heartbeat is more sharply expressed than in a control group, while by the end of the work the reverse relationship is observed.

The nature and degree of expression of changes in the cardiac rhythm during muscular work are influenced in a certain way by the sex and age of the test subjects (Christensen, Hogberg, 1950; Astrand, 1952; R. Ye. Motylyanskaya, 1956; Brouha, Radford, 1960; etc.). Young people show a higher reaction to load than mature persons. In women the pulse frequency is relatively higher during work.

Under conditions of muscular activity, synchronization on the one hand of the rhythm of cardiac contractions and on the other hand of the respiration rhythm may be observed (A. A. Rutsev, 1963; etc.) and the rhythm of the physical work (V. E. Nagornyy, 1964).

Blood Pressure

Muscular work is accompanied by regular changes in the blood pressure in various sectors of the cardiovascular system. In animal experiments Chapman et al. (1959), Rushmer (1961) showed that during physical loading the systolic pressure in the left ventricle increases markedly. Also, a considerable increase in the rate of the intraventricular pressure rise is noted. All of this indicates an increase in contractability of the myocardium. In the diastole, fluctuations in intraventricular pressure increase comparatively due to a decrease in the initial and increase in the final diastolic pressures. The mean pressure in the left atrium changes little (Rushmer, 1961).

In recent times, a number of investigations have been performed in which changes in the pressure in the right sections of the heart and pulmonary artery were studied during muscular work Musshoff et al., 1957; Granath, Strandell, 1964; Widimsky et al., 1965; etc.). Test subjects were subjected to veinous catheterization of the heart. In these investigations it was established that the systolic pressure in the right ventricle increases regularly during work. However, the mean pressure in the right atrium increases very little. The pressure in the pulmonary artery is also increased. During light physical work, the pressure in this vessel usually does not exceed 20 mm Hg. During intensive work, the pressure may be essentially higher.

Investigation of the systemic arterial pressure under conditions of muscular work has been conducted for some time. Most of such works were performed by measuring the arterial pressure by the auscultative method. In /50
this case, an increase in the maximum and decrease in the minimum arterial pressures have generally been noted. However, in recent years, data have been produced showing low suitability of the auscultative method to measurement of arterial pressures under conditions of muscular work. The problem is

that the maximum pressure will be artificially elevated due to an increase in the so-called hemodynamic impact, while the minimal pressure will be artificially decreased.

Freser and Chapman (1954), Holmgren (1956), Astrand et al. (1965), Tabakin et al. (1965), Widimsky et al. (1965) measured the arterial pressure during muscular work by introduction of a catheter to the artery, connected to an electromanometer. In these works, an increase both of the maximum and of the mean and minimum pressures were observed. In the steady state, an initial sharp increase in pressure is usually observed, followed by something of a decrease. For example, during performance of steady work (power averaging 1200 kg-m/min) Ekelung and Holmgren (1964) produced the following changes in pressure in the shoulder artery (Table 3).

TABLE 3. CHANGE IN ARTERIAL PRESSURE DURING MUSCULAR WORK (ACCORDING TO EKELUNG AND HOLMGREN)

Pressure in brachial artery	At rest		During 10 min work		After 50 min work	
	Mean	Limits of fluctuation	Mean	Limits of fluctuation	Mean	Limits of fluctuation
	mm Hg					
Systolic	116.7	105-132	167.8	141-188	145.3	117-165
Diastolic	70.8	60-87	79.8	71-102	71.7	60-81
Mean	88.2	77-103	104.7	85-128	94.2	76-104

With otherwise equivalent conditions, the increase in arterial pressure depends linearly on the increase in load. In well trained persons, the increase in arterial pressure is less sharply expressed than in untrained persons performing this same muscular work. Under the influence of systematic training, the reaction of arterial pressure to the same physical load is decreased.

Astrand et al. (1965), V. V. Vasil'yeva (1966) discovered regional specifics in the increase in arterial pressure during the work of various muscle groups. For example, it was found that when the legs work, the pressure in the arteries of the arms increases more intensively. On the other hand, when the arms work, the arterial pressure in the resting lower extremities is increased relatively more. The mechanism of these regional changes is not quite clear. Swedish authors, based on the fact that the peripheral resistance is increased in the nonworking extremities, explain the relative increase in pressure by an additive mechanism: the systolic and pulse pressure is relatively high due to the addition of centrifugal waves and centripetal waves reflected from the periphery. /51

N. A. Stepochkina (1966) explains this fact differently. In the working extremity, due to expansion of the arterioles, blood flow is relatively

accelerated (the linear rate of blood flow is increased). As a result of this, in correspondence to Bernoulli's equation, the pressure on the wall of the vessel is decreased.

In a special experimental investigation Cerretelli and others (1964) noted an increase in the central veinous pressure. With moderate muscular work, this increase is proportional to the increase in oxygen consumption. However, further increase in the oxygen consumption occurs at constant (increased) central veinous pressure.

Blood Volume per Beat, per Minute, and Residual Volume

An increase in the blood volume per beat pumped by the heart during the systole is one of the most important mechanisms of adaptation of the circulation to conditions of muscular activity. The problem is that the volume per beat, together with the heart rhythm, determines the value of the principal hemodynamic parameter -- the blood circulated per minute, an increase in which is necessary for the performance of physical work.

An increase in the volume per beat is achieved by the interaction of various regulatory systems which adapt the work of the circulatory apparatus to the demands placed by the working organs and tissues during physical exercise. The most important fact here is apparently the positive chrono- and inotropic neuro-humoral stimulation of the myocardium. Due to the increased power of contraction of the cardiac muscle in this case, more complete emptying of the heart is achieved using the residual blood volume. It is possible that a definite role in the increased contractability of the myocardium belongs to the mechanism of self-regulation of the heart according to Frank -- a reinforcement of the contraction of the myocardium by increased final diastolic pressure in the ventricular cavities. This increase in pressure in the left ventricle was recorded by Rushmer et al. (1959) in a chronic experiment with dogs performing work on a treadmill.

The degree of increase in systolic blood volume during stress depends on /52
a large number of factors. Thus, the degree of increase in volume per beat in the steady state is determined to a considerable extent by the power of the work being performed. These data were produced by Musshoff et al. (1957), who noted that the increase in cardiac output is proportional to some extent to the increase in work power (up to 150 w). Heavier work (power up to 250 w) can be supported by relatively smaller volumes per beat. The increase in volume per minute is achieved in this case by progressive increase in pulse frequency.

The increase in volume per beat during work depends also on the state of training of the test subject (Asmussen, Nielsen, 1955; Musshoff, Reindell et al., 1957; etc.): with otherwise equivalent conditions in trained persons the cardiac output during stress is higher than in healthy but untrained persons. In untrained persons the volume per beat reaches 100-125 mℓ during work accompanied by oxygen consumptions of only 1 ℓ/min, but remains constant with further intensification of the stress. In women, the increase in volume

per beat during muscular activity is comparatively lower than in men. For example, the maximum increase in cardiac output in women was recorded as 161 mℓ, in men -- 209 mℓ (Christensen, 1951).

In recent years, of course, it has become clear just how the increase in volume per beat is achieved under loading conditions. The key to this was produced in analysis of the changes in systolic dimensions of the heart during muscular work. Kjellberg et al. (1949), Asmussen and Nielsen (1955) and others have shown that the final systolic dimensions of the heart decrease regularly. Consequently, the increase in cardiac output during physical stress occurs primarily by more complete emptying of the ventricles, i.e., by using the residual blood volume.

The physiological significance of the residual volume of the heart is demonstrated during physical stress, when an increase in cardiac output with a sharp increase in pulse is impossible to explain by the increase in final diastolic volume of the ventricles. Under these conditions, the value of residual volume is to some extent a measure of the functional reserve of the heart (for its specific function): the higher the residual volume of a healthy heart at rest, the greater the volume per beat which can be output from the heart during work. This forms the physiological foundation for the increase in heart volume in highly trained athletes, particularly those trained for endurance (S. P. Letunov, 1957; Reindell et al., 1960; Medved, 1965; Yu. A. Borisova, 1967; etc.). The usage of the residual volume during muscular work occurs as follows. At the beginning of work, as Durand et al. /53 showed (1960), the residual volume decreases almost to zero, then increases slightly, but remains lower than at rest.

During the process of performance of muscular work, the volume per beat usually increases at the beginning, then reaches its maximum, determined by the size of the heart and the nature of the work performed. The value of volume per beat approaches the maximum for a given test subject when the cardiac rhythm has reached 110 beats per minute. The consumption of oxygen reaches only 40% of the individual maximum value at this point (Astrand, 1964).

Further performance of muscular work is usually accompanied by a slight decrease (in comparison with the maximum value) of the volume per beat. Ekelung and Holmgren (1964) noted that when work was performed for one hour at 650-1200 kg-m/min, the volume per beat sometimes decreased to the initial values of the rest state or became even somewhat lower.

Asmussen and Nielsen (1955) showed that during work accompanied by the consumption of oxygen at over 2 ℓ/min, the volume per beat of trained persons reaches a constant value on the order of 160-170 mℓ. If the intensity of muscular stress is such that the consumption of oxygen reaches 3.5 ℓ/min, the volume per beat begins to decrease, returning to its initial value (Tabakin et al., 1964). Generally an analogous dynamic picture of the change in volume per beat (increase of this indicator during light work with subsequent decrease right down to the initial value at rest during heavy work) was

observed by Cerretelli and coauthors (1964), Ulmer and Berta (1964), Rossier and Buhlmann (1965), etc. It is illustrated by Figure 5.

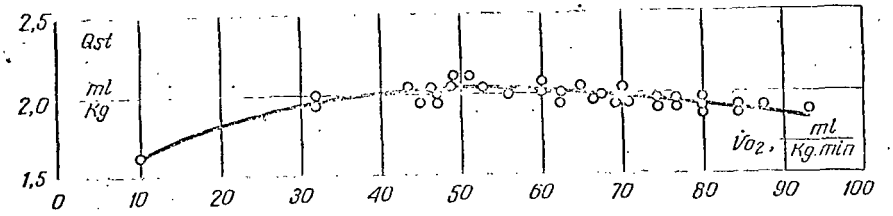


Figure 5. Change in Volume per Beat (in mℓ per kg weight) with an Increase in Work (experimental data of Cerretelli et al., 1965). The amount of work was judged by the consumption of oxygen (in mℓ per minute per kg of animal weight).

The mechanism of the relative decrease in volume per beat during work is not quite clear. In any case, this fact cannot be related to the increase in pulse frequency and as a result of this -- the sharp decrease in the period of filling of the ventricles. Thus, Saltin (1964) during long (3-hour) and heavy (oxygen consumption 75% of the maximum individual value) muscular work observed a gradual decrease in this factor. The pulse frequency fluctuated most frequently between 160 and 180 beats per minute, i.e., was quite far from the extreme value.

It might be thought that in connection with the development of fatigue, the contractability of the myocardium decreases slightly and the utilization of residual blood volume also decreases.

Certain specifics are inherent to the expression of changes in cardiac output as a function of the position of the body of the test subject during the performance of muscular work (Wang et al., 1960). The fact is that at rest in the vertical position the volume per beat is approximately 40% lower /54 than in the prone position. It is for this reason that during muscular work performed in the vertical position the cardiac output is relatively low.

With static stress, the change in volume per beat differs slightly from that described above (V. V. Skryabin, 1950; A. N. Vorob'yev, 1961). The fact is that under conditions of, for example, pressing of weights, in connection with the pressure the intra-alveolar and intraperitoneal pressures increase. As a result of this, the blood flow through the respiratory circulation is sharply decreased and the arrival of blood at the left ventricle is insufficient (as occurs in the Valsalva experiment). With this hemodynamic situation, the volume per beat may be decreased, sometimes quite essentially (an increase in the volume per minute is achieved in this case by sharp tachycardia).

Under the influence of training, a compensatory capability for increased pooling of blood in the capillary-veinous portion of the respiratory circulation is apparently developed. Due to this, as the blood content of the left ventricle is increased, the blood volume per beat even slightly increases (A. N. Vorob'yev, 1961).

The effectiveness of cardiac hyperfunction during muscular activity can be objectively evaluated on the basis of the calculated external mechanical work of the heart (A):

$$A = P \cdot V + \frac{mv^2}{2g}.$$

In this formula, an analysis of which is given below, the value of PV is the static component of the work of the heart, expended to overcome the input resistance in the aorta, $mv^2/2g$ is the kinetic component of the work of the work of the heart expended to accelerate the blood column in the aorta. /55

During muscular activity, the mechanical work per beat is increased both in its static and kinetic components. The increase of the kinetic component of the work of the left ventricle is sharper, due to the considerable linear acceleration of the blood flow.

Increasing the volume of blood circulation per minute is the principal mechanism of adaptation of the organism of the athlete to physical loads. The volume per minute is an integral parameter of circulation, the value of which depends on a number of vegetative functions. This essentially follows from the classical equation of Fick (1870):

$$V_m = V_s R = \dot{V}_{O_2}/AVD, \qquad (6)$$

where V_m and V_s are the volume per minute and volume per beat of blood respectively, V_{O_2} is the oxygen consumption, AVD is the arterial-veinous difference in oxygen.

An experimental analysis of equation (6) has shown that under conditions of muscular work, the blood circulation volume per minute depends linearly on the value of oxygen consumption (Astrand et al., 1964; Asmussen, Nielsen, 1955; Brouha, Redford, 1960; etc.).

Considerably less definite data are available on the relationship of the blood circulation volume per minute on the one hand and the volume per beat and pulse frequency on the other.

The contribution of an increase in volume per beat to the increase in volume per minute in blood circulation has been estimated differently. Thus, many authors (Christensen, 1937; Chapman, Freser, 1954; G. I. Markovskaya, 1955; Musshoff et al., 1957; Chapman, 1960; etc.) have found that an increase in cardiac output has an important role to play in increasing the volume of blood circulation in the circulatory system. On the other hand, in a series of experimental and clinical-physiological investigations (Rushmer, 1959; Holmgren et al., 1960; Wang et al., 1960; V. V. Vasil'yeva, 1964; Widimsky et al., 1965; etc.) only a very slight increase in the blood volume per beat was detected during work, and the increase in volume per minute under these conditions was related primarily to the increase in pulse frequency. This latter point of view apparently is better founded, since a large number of works in recent times performed at a high methodological level have indicated the comparatively slight increase in the volume per beat during intensive physical loads, while at the same time the blood volume per minute and cardiac rhythm show a high degree of correlation under conditions of work.

In conclusion we note that works of recent years dedicated to the change in the value of volume per minute under conditions of muscular activity do /56
not confirm the old data concerning the increase in this indicator of up to 40-45 ℓ/min and higher. The maximum values of volume per minute apparently do not exceed 25-30 ℓ/min.

Phases of the Cardiac Cycle

The investigation of phase shifts directly during muscular activity began to be performed comparatively recently (V. L. Karpman and Yu. K. Shkhvatsabaya, 1962; Yu. K. Shkhvatsabaya, 1964; V. A. Tishler and O. N. Belina, 1964; Strandell, 1964). Earlier, attempts were made to judge changes in the phase structure only by comparing data at rest and data recorded after completion of muscular work (Lehmann, Steinhaus, 1933; Blumberger, 1942; V. L. Karpman, 1956; L. A. Ioffe, 1962; etc.).

Under the influence of intensive muscular work (over 1200 km-m/min) a regular change in the duration of phases is observed, which we call the phase syndrome of hyperdynamia (V. L. Karpman, 1964). A shortening of all systolic phases is characteristic for this syndrome.

The shortening of the isometric (isovolumetric) contraction phase is related primarily to an increase in the rate of increase in intraventricular pressure. The degree of expression of the shortening of this phase is determined by the intensity[1] and duration of the muscular work. During

[1] It should be remembered that the static characteristics for the contractive function of the heart have the form of an S-shaped curve. From this fact it follows that in the area of high powers of mechanical work performed, e.g., on a bicycle ergometer, deviations of the parameters of cardiodynamics studied are not observed. Therefore, we are concerned with the maximum deviation, maximum use of various functions of the myocardium.

intensive work, a situation is frequently observed in which the duration of the isometric contraction becomes practically equal to zero. Contraction of the myocardium most effectively takes on a purely isotonic nature in this case -- the expulsion of blood from the heart occurs throughout the entire mechanical systole. The rate of increase in intraventricular pressure under these conditions increases in comparison with its value at rest by 20-30 times. Based on literature data concerning changes in arterial pressure during work (direct measurements) and our own data on the shortening of the isometric contraction, we calculated that the initial rate of increase of pressure in the left ventricle can climb to 40,000-50,000 mm Hg/sec.

The duration of the expulsion period during intensive muscular work can be decreased by a factor of almost 2 in comparison with its duration at rest. The shortest duration of this phase was recorded as 0.12-0.13 sec. It should /57 be noted here that during the time of expulsion the volume per minute, i.e. the total duration of the period of expulsion in one minute, increases, according to average data, to 24.9 sec in place of 15.4 sec at rest.

The shortening of the time of expulsion is combined with an increase in the rate of expulsion of blood from the heart. Due to this, the volume of blood per beat not only is not decreased, but is even somewhat increased. The duration of the mechanical systole, as follows from the above, is also essentially decreased. The intrasystolic indicator increases regularly by an average of up to 95%. This means that during physical work, most efficient restructuring of the phase structure of the cardiac cycle occurs. In cases when the duration of the isometric contraction becomes practically zero, the intrasystolic indicator reaches 100%.

The processes of shortening of the duration of the principal systolic phases during intensive muscular work or the curves of transient characteristics are homogeneous and aperiodic. In a considerable fraction of the observations there is an initial jump on the corresponding curves -- the start reaction. For this type of transient processes, it has been found possible to select experimental equations satisfactorily approximating the experimental data. Thus, in 1964 we produced two equations (Figure 6) /58 describing the transient processes for the duration of the phase of isometric contraction and the duration of the expulsion phase for conditions of muscular activity averaging 1500 kg-m/min:

$$\Delta IC = 0{,}015 \cdot e^{-0{,}02t} - 0{,}05, \quad (7)$$

$$\Delta E = 0{,}055 \cdot e^{-0{,}4t} - 0{,}085, \quad (8)$$

where ΔIC and ΔE represent the shortening of the phases of isometric contraction and expulsion respectively (in seconds), e is the base of the natural logarithm, t is the time of investigation after the beginning of work (in seconds for ΔIC and in minutes for ΔE). According to the data of Yu. K. Shkhvatsabaya (1965), the deviation of experimental values from calculated values generally does not exceed ±0.03 sec.

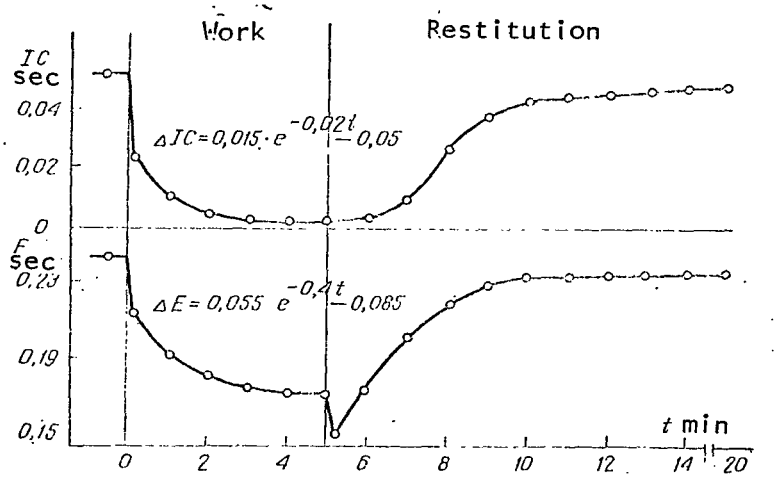


Figure 6. Change in Duration of Isometric Contraction Phase (IC) and Expulsion Phase (E) During Hard Work and Restitution.

The steady state for the duration of the isometric contraction is achieved approximately twice as rapidly as for the duration of the expulsion period (V. L. Karpman and Yu. K. Shkhvatsabaya, 1962). This is explained by the fact that the shortening of the isometric contraction is directly related to the reflector increase in contractability of the myocardium, while the shortening of expulsion depends on stabilization of cardiac output.

The duration of the diastole is shortened during muscular work quite sharply. For example, with a pulse frequency of 200 beats per minute (duration of cardiac cycle 0.3 sec), the duration of the diastole may be shortened to 0.10-0.11 sec. Since this time interval includes the duration of the period of relaxation of the myocardium, filling of the ventricles at this rate requires only 0.05-0.06 sec. However, this time is quite sufficient to fill the chambers of the heart with the necessarya blood volume, since the cross section of the veinous apertures in the heart is rather great (on the order of 5 cm^2), and the filling pressure is probably quite high with this tachycardia.

The expression of phase shifts during physical stress depends to a certain extent on the power and volume of the work performed. Thus, phase shifts have been described observed with work of great intensity. With work of relatively lower intensity (less than 1000 kg m/min), the changes in the dynamics of cardiac contraction are less sharply expressed. The shortening of the isometric contraction is not great, and never reaches its maximal extent. The rate of increase of intraventricular pressure increases by only 1 1/2-4 times.

The duration of the expulsion period during low-intensity work may remain unchanged for some time. Sometimes it even increases (Bowen, 1904; Jezek, 1964). However, most frequently the transient process constructed for the change in duration of this period is two-phased in nature: at first the /59
expulsion is somewhat lengthened, then begins to be shortened in comparison with the duration of this period at rest. The initial lengthening of expulsion with moderate muscular work reflects an increase in cardiac output, while the subsequent shortening indicates an increase in the power of the cardiac contraction with stabilization of cardiac output.

Up to the present, we have spoken of stable operating modes of various intensities. With discrete work, however, the following dynamics of expulsion is observed (Figure 7): during the working cycle, the period of expulsion increases on the average, while during the rest cycle, on the contrary, it decreases (all of this does not hold true for the first working cycle, when the normal reaction of the duration of expulsion period to physical load is observed). The basis for this regularity apparently is the fact discovered by Schneider and Gottenlohner (1965), who noted a successive increase in the blood volume per beat with each cycle of work (in the discrete mode). This value is directly related to the duration of the expulsion period.

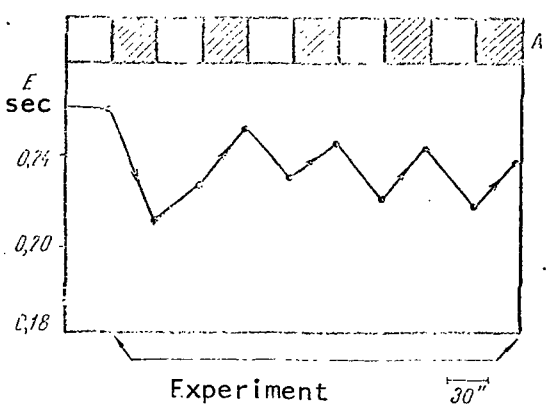


Figure 7. Change in Duration of Period of Expulsion (E) with Discrete Work. Shaded squares represent working period, unshaded squares represent rest.

Electromechanical Relationships

The dynamics of electrical activity of the heart directly during the time of muscular work bwgin to be investigated as long ago as the 1930's (Knoll, 1933). However, more objective works have appeared comparatively recently (Klepzig et al., 1956; V. V. Matov, 1960; V. V. Rozenblat et al., 1962; L. A. Butchenko, 1963-1966; O. N. Belina, 1965; N. A. Stepochkina, 1965; etc.). These works have studied the changes in electrocardiograms during the process of performance of physical work by athletes[1].

Under conditions of muscular /60
activity, due to the sympathetic control, positive chronotropic, dromotropic and inotropic stimulation of the myocardium are developed, and a shortening of

[1] We will not discuss a series of important investigations undertaken by S. P. Letunov (1950, 1957) and his colleagues, as well as a number of other authors, who recorded the electrocardiograms only after the completion of the muscular work.

the duration of most intervals on the electrocardiogram is quite regularly noted[1].

The duration of the "electrical systole" (the Q-T interval on the electrocardiogram) is essentially shortened during muscular work. The degree of shortening depends on the intensity of the work and the physical state of training of the test subjects. The shortening of the Q-T interval most frequently is found to be unproportional to the quantities calculated by the formulas of Bazett (1920), Ashman (1942), etc. This lack of correspondence arises in connection with the inaccuracy of these formulas during tachycardia (Bruschke et al., 1965). Recently V. L. Karpman and O. N. Belina (1967) showed that the following simple formula is suitable for these purposes:

$$Q\text{--}T = 0{,}56 \cdot C + 0{,}035, \qquad (9)$$

where C is the duration of the cardiac cycle in sec.

During physical stress, regular changes in the morphology of the waves and intervals of the electrocardiogram are observed. Thus, an increase in the P wave and a decrease in the QRS complex are noted. Frequently, synchronous displacement of the P-Q and S-T intervals below the isolines is noted (L. A. Butchenko, 1963; N. A. Stepochkina, 1965; O. N. Belina, 1965). It has been suggested (G. L. Lempert, 1963), that this displacement arises in connection with stratification of the negative auricular wave T_a, which increases during work, in the sections of the electrocardiogram under discussion.

The dynamics of the T wave during the process of performance of muscular work is characteristic. At the very beginning of work, this wave is flattened, then during the first few minutes is increased (Klepzig et al., 1956; L. A. Butchenko, 1963; V. V. Matov and I. B. Surkina, 1964; N. A. Stepochkina, 1965; O. N. Belina, 1965).

In our laboratory, the dynamics of the T wave during work was subjected to special analysis by O. N. Belina. In this work, amplitude invariance of the wave was established. During work in the steady state: the amplitude during loading was not related in any way to its initial amplitude. During work, individual variations in the amplitude of the T wave detected at rest were smoothed to a considerable extent (V. L. Karpman and O. N. Belina, 1967).

The mechanism of the initial decrease in the T wave is not quite clear. We assume that the phenomenon is related to insufficient arrival of lactates to /61 the myocardium during the working-in period. The fact is that during muscular activity the human heart consumes blood lactates rapidly. According to the

[1] These questions are discussed in greater detail in Chapter V.

data of Keul et al. (1964), the coronary arterial-veinous difference in lactates increases rapidly during work (it is characteristic that the coronary arterial-veinous difference in oxygen does not change in this case). The accumulation of blood lactates occurs primarily during muscular activity, following an exponential law according to the data of N. I. Volkov (1966), while the concentration of lactates in arterial blood is not great during the working-in period.

The electromechanical latency of the heart frequently changes during muscular activity. These changes are expressed primarily as a certain shortening of the electrodynamic latent period (I. N. Ivanitskaya, 1965; etc.), i.e. the time interval between the beginning of depolarization of the myocardium and the beginning of the mechanical systole. According to the data of our colleague Z. B. Belotserkovskiy, under conditions of physical stress the electrokinematic latency -- the time interval from the beginning of depolarization to the beginning of the systolic transformation of the ventricles -- is also decreased. This type of change is a reflection of shifts in metabolism, due to which the propagation of the contractive process through the fibers of the cardiac muscle is accelerated.

During the investigation of the time relationships between the beginning of the II tone of the heart and the end of the T wave on the electrocardiogram, arbitrarily taken as the completion of the electrical and mechanical systoles (beginning of the II tone corresponds to the end of the protodiastole, lagging 0.03-0.04 sec behind the actual end of the mechanical systole), during work certain specific features are noted. Under rest conditions, the II tone arises most frequently after completion of the T wave or slightly leads the end of the T wave (by 0.01-0.03 sec). During muscular activity, there is a clear tendency toward displacement of the II tone to the left in relation to the end of the T wave. During heavy work, the lead of the II tone over the end of the T wave may reach 0.07 sec (K. M. Smirnov and N. V. Zaytseva, 1964), i.e., the Hegglin phenomenon arises (Hegglin, 1947). The physiological mechanism of the formation of the Hegglin phenomenon under conditions of muscular work is that during hyperdynamia of the myocardium, the mechanical systole is shortened more intensively than the electrical systole (I. N. Ivanitskaya, L. A. Ioffe, V. L. Karpman, G. V. Sadovskaya, 1964; etc.). Thus, during muscular work the formation of the Hegglin phenomenon in athletes is not a pathological characteristic.

Under conditions of muscular work, electrical alternation of the heart may arise (A. A. Beryukovich and V. M. Korol', 1964; V. V. Matov and I. D. Surkina, 1964). According to the data of O. N. Belina, it is manifested as an essential /62
or moderate deformation of the individual QRS complexes. Alternation may be systematic (1:1; 1:2) or accidental. In the first case, it is related to the respiratory cycle (A. A. Beryukovich and V. M. Korol', 1964).

Electrical alternation of the heart, arising during muscular work, is usually not accompanied by changes in the mechanical activity of the heart. Extremely rarely, a combination of electrical and mechanical alternation (of 1:1 type) may be observed. This type of alternation is different from that observed in clinical practice, and is not accompanied by changes in the phase

structure of the systole. Physiological investigations of this state are being conducted at the present time.

Restitution

Cessation of muscular work is accompanied by a complex of cardio- and hemodynamic changes directed in the final analysis toward restoration of the level of functioning of the circulatory apparatus at its initial level or near it. The restoratory period is conveniently divided into three stages: early restitution, restitution itself and the stabilization stage (V. L. Karpman, 1964).

The stage of early restitution is brief, not occupying over 30-60 sec after the completion of muscular work. This stage characteristically shows changes in cardiodynamics caused by a combination of a decrease in veinous blood return to the heart as a result of the cessation of the operation of the "muscular pump" on the one hand, and on the other hand by a continuation of intensive chrono- and inotropic stimulation of the myocardium. As a result of this, immediately after the completion of muscular work (we have in mind a sharp discontinuance of muscular work), a secondary shortening of the period of expulsion and the systole is observed (Yu. K. Shkhvatsabaya, 1964b), along with a decrease in the time of expulsion of the volume per minute (Sore, 1930), a sharp drop with a subsequent increase in the maximum pressure (Freser, Chapman, 1954), etc.

During early restitution, after static effort, a phenomenon similar to that described by Lindgard for respiration -- secondary hyperfunction of the heart -- is observed. It is caused by the fact that after the stress is relieved, the conditions of blood flow in the large veins and in the respiratory circulation are normalized, the heart begins to be loaded by volume.

During the second stage of the restoratory period, the magnitudes of various parameters of cardiodynamics are returned to values similar to the initial values. The end of this stage is characterized by stabilization of each parameter. The duration of the restitution itself depends to a considerable extent on the intensity and duration of the muscular work performed. The course of the restoration of any given parameter of cardiodynamics is usually /63
exponential. The duration of the stabilization stage depends on a large number of factors: the power and volume of work performed, the position of the body during the restoratory period, the level of physical training of the test subject, etc. It should be noted at this point that the course of restoration of cardiodynamics occurs best in the horizontal position, when no regulation of circulation according to the gravitational factor is encountered (V. L. Karpman and Yu. K. Shkhvatsabaya, 1962).

Regulation

The biological system for automatic regulation, under conditions of relative quiet, supports the principal parameters of cardio- and hemodynamics at a rather constant level (homeostatic regulation).

Under conditions of physiological perturbations, including muscular work, the biological automatic control system regulates according to the perturbing action. The nervous centers regulating the activity of the mechanisms of homeostasis are inhibited in this case (P. K. Anokhin, 1958; Braynes et al., 1962). The inhibition of these centers apparently occurs only during the initial portion of the second stage of restitution.

The regulation of the cardiac activity according to the perturbing action under conditions of muscular work is performed using a number of mechanisms beginning with the mechanisms of self-regulation of the heart and ending with cortical mechanisms.

Among the mechanisms of self-regulation of the heart involved during physical loading at the present time we must include the so-called heterometric and homeometric forms of self-regulation (Mitchell, 1963) performed at the level of the contractive elements of the myocardium.

The basis for heterometric self-regulation of the heart includes the mechanisms of Frank and Starling, relating the reinforcement of cardiac contractions to the final diastolic volume and pressure in the ventricles. These mechanisms apparently have a definite role to play in the adaptation of the heart to muscular work. Homeometric self-regulation of the heart (Sarnoff et al., 1960) during muscular work relates the reinforcement of cardiac contraction to the increase in pulse frequency. It is based on the well known phenomenon of the "Bowditch ladder" (Sarnoff et al., 1960; Rozenbluet et al., 1959). Due to the homeometric self-regulation, cardiac output can be increased during tachycardia by more complete liberation of the cavities of the heart of blood with normal or even slightly decreased diastolic volume. The effectiveness of this mechanism during physical loads has not been completely studied. /64
The problem is that the increase in frequency of heart contractions due to electrical stimulation (purely chronotropic influences on the myocardium) is accompanied by a decrease in blood volume per beat (V. L. Karpman, 1965; Ross et al., 1965). The volume per minute, as a rule, is not essentially changed.

Also, the role of another mechanism of homeotropic self-regulation of the heart -- the Anrep mechanism -- in the adaptation of circulation to muscular work is not quite clear.

It should be noted that changes in cardiodynamics resulting from homeotropic self-regulation of the heart have also been detected during stimulation of the sympathetic nerves (Rushmer et al., 1959). In addition to this, reinforcement of cardiac contractions similar to that observed during muscular work but at normal frequency of the heart beat may occur during stimulation of the stimulating nerve of I. P. Pavlov. This fact was obtained by

V. M. Pokrovskiy (1967) on the basis of phase analysis data. By stimulating this nerve in experiments on animals with unchanged pulse frequency, he noted phase shifts characteristic for the syndrome of myocardial hyperdynamia.

The nervous mechanisms of cardiac regulation during muscular work have been traced at various levels in the nervous system. Thus, it has been shown that changes in cardiodynamics and hemodynamics specific for physical stress are produced not only by stimulation of the sympathetic nerves and nervous centers, but also by stimulation of various sectors of the midbrain and hypothalamus (Rushmer et al., 1959).

The regulating role of the central nervous system when the cerebral cortex is the controlling element in changes in cardio- and hemodynamics has been shown in a number of domestic investigations using the method of conditioned reflexes. Thus, the possibility of conditioned reflex increase in the blood circulation volume per minute (K. M. Smirnov, 1941), conditioned reflex changes in electrocardiograms (S. P. Letunov, 1957), roentgenkymograms (N. V. Savina, 1952) have been shown, similar to those observed under conditions of muscular work.

In conclusion, we note that the reaction of the heart to physical stress is still observed with complete denervation (Donald et al., 1964). In this case, purely humoral regulation of cardiodynamics is observed. Adrenaline and noradrenaline have been assumed to be among this type of regulator (changes resulting from humoral regulation occur with considerably more inertia and are considerably less efficient than those changes arising under the influence of normal functioning of the biological automatic regulation system.

CHAPTER III

BLOOD CIRCULATION AND THE STATE OF TRAINING

ABSTRACT. The functioning of the cardiovascular system is one of the most adequate and accurate indicators of the state of physical training of an athlete. The leading diagnostic characteristic of the state of training is not the amplitude of the reaction to a physical load, but rather the type of reaction, the quality and relationships between changes in individual functional indicators. Tabular data are presented indicating the types of reaction to be expected from athletes trained for strength, speed and endurance, in various states of training, to various physical loadings.

The definition of the state of training is one of the principal areas for the work of the sports doctor. Testing the state of health and development of the state of training are closely related, since a proper determination of the state of training is not only an important condition for the effectiveness of the training process and achievement of high sports results, but the principal means of preventing disruptions in the state of health related to the application of loads which do not correspond to the functional state or level of preparation of the athlete. /65

The state of training manifests itself in the ability of the human organism to withstand high physical and mental stress, to react rapidly and adequately to demands by the corresponding increases in the functions, to maintain a level of activity of the organism achieved for the time required (while showing high working ability and maximum sports results under these conditions), and to recover rapidly. The level of training results from a combination of closely interrelated factors -- the degree of physical, technical, tactical and mental preparation of the athlete. Therefore, the most correct definition of the state of training is possible only by combined efforts of doctor and trainer, who, on the basis of analysis and comparison of various factors, characterize the preparedness of the athlete. Medical methods of investigation play a very important role in this process, since the functional state of the organism is one of the principal components of the state of training generally changing in close relationship to the general level of training, resulting from the entire totality of influences touching the human organism in the process of physical training and sports training. /66

Systematic muscular activity causes a corresponding physiological restructuring of the activity of the systems in the organism. The functional

capabilities of all organs are increased, the coordination mechanisms interrelating the motor and vegetative systems are improved, compensatory-adaptation reactions are significantly expanded, etc. The corresponding changes in indicators reflecting the state of the various functional systems, including quite importantly the circulatory apparatus, are revealed during medical examination.

During the process of development of the state of training, a close interrelationship exists at all stages between the motor and vegetative functions. The satisfaction of the demands of the organism during increased muscular activity is possible only with a corresponding reinforcement of circulation. In turn, systematic muscular work facilitates an increase in the functional capacities and a constant increase in the limits of the working ability of the cardiovascular system.

The state of the circulatory apparatus limits the working ability to a great extent and in many cases is the earliest signal of a decrease in working ability. For example, highly qualified athletes in many cases can retain good adaptation of the nervous and muscular apparatus to physical stress even after a clear worsening of the functional state of the circulation can be noted. The disruption of the optimal relationship between processes of excitation and inhibition in fatigue and overtraining rapidly influences the activity of the mechanisms regulating circulation, causing discoordination in the activity of various lengths. All of this indicates the particular significance of the functional investigation of the cardiovascular system in connection with an evaluation of the level of training and sports ability. This is particularly important for types of sports in which the achievement of high results involves the necessity of performing large volumes of physical work.

The great significance of the cardiovascular system in the provision of adaptability of the organism to physical stress is the reason for the particular interest in the study of this function during medical examination. This section in the methodology of medical examination of athletes in general and as concerns the diagnosis of the state of training in particular has been developed in great detail, which has been greatly facilitated by many years investigation of the workers of the sector of sports medicine of the All-Union Scientific Research Institute for Physical Culture, under the leadership of S. P. Letunov, and later by the Leningrad Scientific Research Institute for Physical Culture (A. G. Dembo, L. A. Butchenko, R. D. Dibner and others) and quite recently by the State Central Order of Lenin Institute of Physical Culture (V. L. Karpman) as well as a number of other institutions. /67

Although, as was stated above, the functional state of the circulatory apparatus determines to a considerable extent the level of sports ability, in many cases considerable divergences can be encountered. For example, sometimes a high result may be shown by an athlete who has either not achieved the best indicators (for himself) of the functional state of circulation, or, on the other hand, who has definite functional deficiencies resulting from overfatigue or who manifests the initial phenomena of overtraining. This becomes possible due to the high technical and tactical training of the athlete, his great motor experience, the great stress of his will and tremendous

compensatory capabilities of the trained organism. Most frequently this can be seen in technically complex forms of sports and in forms of sports where many years motor experience determines the results of competition. However, long-term dynamic observations of such athletes always show that the high results are not stable. It is difficult to plan on an increase in results if the necessary adjustments are not introduced to the training process in a timely manner.

Frequently athletes do not show high results even though there is a high functional state of circulation. In these cases, the reason must be sought in the nature of the training, which prevents the athlete from realizing his functional capabilities. Consequently, the results of motor activity cannot be judged as the unique and absolute criterion of the state of training. In order to establish the level of training, it is important to know not only what work the athlete is capable of performing and what result he can show, but the price which his organism pays to achieve this result. Therefore, the divergence sometimes observed in practice between the evaluation of the state of training according to data on the functional state of the cardiovascular system and the external manifestations of working ability represents only an apparent contradiction. The revelation of such "inconsistencies" is quite valuable in the combined work of doctor and trainer, allowing various factors determining the level of training of the athlete to be compared, followed by proper analysis of his training, determination of the true deficiencies in his state of training and achievement of further improvement in training.

The diagnosis of the state of training requires a special methodology of medical examination. Based on the general positions of the clinical disciplines and human physiology, the method of medical examination of athletes has a clearly expressed specific nature, resulting from the necessity of /68
determining the capabilities of the human organism under extreme conditions in connection with its motor activity: great (frequently limiting) physical stress.

In this chapter we will not set ourselves the goal of expounding upon the characteristics of the state of training based on data from individual methods of investigation of the circulatory apparatus, since this has been done to some extent in the presentation of each method, but rather will attempt to shed light on certain of the most general factors which should be taken into consideration in setting up examinations and interpreting the data produced.

What are the main principles which should be used as a basis for evaluating the state of training using data from a study of the circulatory apparatus of athletes?

The primary factor is combined study, i.e. comprehensive study of the functional capabilities of the organism. The ability of the organism of an athlete to withstand high physical stresses is provided by a complex, closely interrelated activity of all organs and systems, by precise functioning of the regulatory apparatus.

During the process of systematic training, the human organism adapts itself to a constant increase in the demands which it must meet, responding to them by restructuring of all links in its activity: the functional capabilities of all organs and systems are expanded, the highest coordination is achieved in the activity of the somatic and vegetative systems during physical stresses. This is a result primarily of conditioned reflex connections in the central nervous system with the motor apparatus and internal organs, arising and constantly developing during the process of training.

The necessity of combined study of this unitary activity of the organism, which determines its adaptability to physical stress, results to a considerable extent from the fact that, as dynamic observations have shown, improvement of various functions and various indicators in the process of development of a state of training does not always occur at the same time and in the same direction. This depends on a number of factors, primarily such factors as the age of the test subject, the initial level of training, individual aptitudes, the type of sport involved, the period and primary direction of training, etc.

The evaluations of the functional state of the organism made on the basis of study of different functional systems might differ considerably in many cases. However, the higher the level of training of the athlete, the less these differences and the more sharply expressed is the integration of motor and vegetative functions,, the more heterochronism is smoothed as they improve, which is manifested in the process of development of training. Thus, /69 experiments involving synchronous recording of various functions in the state of muscular rest and under conditions of various physical load levels for the same athletes at various stages of the development of their training (N. D. Grayevskaya, Ye. S. Stepanova, E. V. Kukolevskaya, 1966) showed that whereas with insufficient state of training of the athletes 54% of the test subjects showed different evaluations of their functional state from the data of studies of the circulatory and nervous-muscular apparatus, later when a satisfactory state of training had been achieved this percentage (for the same group of test subjects) decreased to 28, while still later when sports form had been achieved, the percentage dropped to 18.

These data show that we cannot characterize the level of training on the basis of a study of any single function alone, or even more particularly on the basis of individual indicators. This evaluation can only lead to error. It is difficult to properly evaluate the so-called "acute changes" caused by loading on the basis of determination of individual indicators alone, since the nature of the reaction of each indicator may vary, reflecting mutual compensation and more or less improved methods of adaptation depending on the degree of stress and the level of training. Only a combined approach allows us to select methods which supplement each other, most fully revealing the functional capabilities of the organism in relation to the action of physical stresses on the organism.

This is true both of the general method of determination of the state of training during medical examination, and of the direct study of the circulation. The general methodology should include methods characterizing the state

of the principal functional systems determining high sports ability: the nervous system, nervous-muscular apparatus, vegetative organs, and the internal medium of the organism. The data from investigation of the functional state of circulation, although it occupies one of the leading positions in this combination, still allows us to evaluate the general level of training of athletes only in consideration of the state of the remaining functional links in the activity of the organism. As concerns direct investigation of the circulatory apparatus, the methods used should reveal the functional state of its various links and their interrelationships.

Recent years have been characterized by rapid development of methods of investigation of the functional state of the circulatory apparatus and the introduction to the practice of the sports doctor of many new methods of investigation which, in particular, have been reflected in this collection. However, sufficient material has not been accumulated for many of these methods to allow an evaluation of the level of training to be performed.

A second, no less important principle in the evaluation of the state of training is the obligatory investigation of the athlete under conditions such that high demands are placed upon him, i.e. using various physical loads including significant, limiting type loads. It is quite important here that the athlete be investigated not only after the completion of the load and at various stages of the recovery period, but also directly during the physical loading in order to most completely determine the changes occurring at the height of the load, as well as the transient states from rest to work and vice versa, which provides essential supplementary data for the evaluation of the functional state (L. A. Butchenko, 1963; V. V. Rozenblat, 1966; V. V. Matov, 1964; V. L. Karpman, 1965; etc.).

The reaction to load can be considered the most integral method of determining the state of training, if we evaluate its primary qualities: the working-in period, the reactability, adaptability, stability and recoverability.

If the investigation of the circulation under conditions of muscular rest with increasing state of training is characterized by a certain economy of functions (retardation of rhythm of cardiac contractions, decrease in arterial pressure, slowing of respiration and decrease in respiratory volume, decrease in blood volume per minute, increase in the quantity of residual blood in the /71
heart, lengthening of the diastole, a moderate retardation in conductivity, increased arterial-veinous difference, a decrease in peripheral resistance, retardation of blood flow, etc.), the reaction to physical load is expressed as an acceleration of the working-in, with ever greater adequacy of physiological changes to the nature and magnitude of the load, clearer stability of reactions during repetition and complication of loads, significant acceleration of the restoratory processes. When muscular work is dosed at moderate intensity, as the state of training increases a definite tendency toward economy of functions is noted. A more highly trained organism performs the same work with considerably less expenditure of energy than a less trained organism, the demand for oxygen is satisfied with less stress on the functions

of respiration and circulation. Dynamic observations show a decrease in changes of oxygenation, pulse, blood volume per minute, diastolic pressure, degree of distension of vessels and other indicators of hemodynamics (Table 4). This results from a greater degree of automation of a motor skill and correspondingly lower expenditure of effort for the performance of standard work, an improvement of regulation, increased economy of intracellular metabolism with better utilization of oxygen by the tissues.

On the other hand, with large and intensive physical stress, placing very high demands on the organism and involving the commitment of functional reserves, the trained organism is capable of performing the necessary volume of work at considerably higher intensity (resulting in the achievement of a higher result) by more rapid and complete mobilization of the functions. The physiological changes involved in many cases can be quite great, impossible for a less trained organism. The reinforcement of functions achieved may be supported at the required level without negative aftereffects by an organism for a considerably longer period of time than is possible for a less trained man. As the circulation is investigated, this is manifested as a significant reinforcement of cardiac activity, a reinforcement of the contractive ability of the myocardium, a shortening of all principal phases of the systole, an acceleration of blood flow rate, retenstion of sufficient adequacy of the state of the precapillary lumen and a significant increase in the circulation volume per minute. /72

The leading diagnostic characteristic of the state of training must be considered not the amplitude of the reaction, but rather the type of reaction, the quality and interrelationship of the changes of individual functional indicators. Thus, experimental investigations and dynamic observations show that as the state of training increases the reaction generally becomes normotonic. Signs of discoordination of various links of the respiratory-circulatory system in response to load are significantly decreased or disappear entirely, appearing in those athletes (with similar load but lower level of training) as stepped changes in arterial pressure, respiration, vascular tone, as well as uneven or oppositely directed changes in pulse, arterial pressure and other indicators of hemodynamics after load.

The direction of a change in the amplitude of a reaction in various functional indicators of the circulation may differ: as the state of training increases, a decrease in the amplitude of the reactions of some may be observed with an increase in the degree of changes by others (Figure 8), indicating mutual compensation and qualitative improvement of the means of adaptation of the organism to physical stresses. This is reflected in the materials of the experimental investigation of N. V. Zimkin et al. (1965), which showed that the same effect may be achieved by various combinations of functional changes under physical stress, with a higher degree of mobilization of some and lower degree of mobilization of other functions.

TABLE 4. CHANGE IN THE REACTION OF THE CARDIOVASCULAR SYSTEM TO REPEATED LOADS OF MODERATE INTENSITY WITH INCREASING DEGREE OF TRAINING (AVERAGE DATA)

State of training	Load	During Load				
		Time of stable saturation of blood with oxygen, sec.	Percent decrease in saturation	Duration of cardiac cycle (R-R interval)		
				30 sec	After 1 min.	After 3 min.
Highest (sports form)	1	142,2±17,5	1,7	0,52±0,019	0,52±0,013	0,49±0,013
	2	141,2±20,5	1,9	0,49±0,015	0,49±0,010	0,47±0,014
	3	160,0±15,3	2,0	0,51±0,014	0,48±0,012	0,43±0,013
Quite Satisfactory	1	130,0±20,5	3,1	0,52±0,012	0,50±0,013	0,48±0,015
	2	155,0±19,4	2,9	0,50±0,013	0,49±0,016	0,47±0,012
	3	131,4±22,1	2,9	0,49±0,015	0,49±0,015	0,46±0,013
Insufficient	1	77,7±21,8	3,4	0,49±0,011	0,4 ±0,012	0,41±0,013
	2	131,1±21,6	3,1	0,43±0,012	0,44±0,014	0,41±0,010
	3	115,5±26,0	2,9	0,46±0,010	0,43±0,013	0,41±0,010

State of training	After load					
	Pulse frequency beats per min	Respiratory frequency per min	Max Ap, mm Hg	Min AP, mm Hg	Blood flow rate, sec.	Vessel distension indicator, mm Hg
Highest (sports form)	129,5±4,8	26±1,4	166±3,6	56,8±3	5,5±0,1	79±3,1
	131,6±5,1	27,2±1,6	166±2,8	64±2,4	5,5±0,2	80±2,7
	136,0±5,6	26,8±1,1	168±3	60,6±2,1	5,5±0,1	85±3,2
Quite satisfactory	133,5±2,3	27,1±1,3	159±4,1	65±2,1	5,1±0,3	86±4,5
	150,0±3,7	28,2±0,9	160±4,4	53±2,4	4,6±0,2	83±4
	140,0±3,3	29,0±1,1	155±3,5	48±3	5,1±0,1	79±3,9
Insufficient	145,0±6,3	32,5±2,1	167±5	23±7	4,4±0,1	84±3,2
	153,0±4,7	34,0±1,8	163±7	23±5,2	4,4±0,2	86±3,3
	169,0±5,4	36,0±2,2	153±7	31±10	4,4±0,9	85±3,2

Note: Max Ap = maximum arterial pressure; min AP = minimum arterial pressure
Note: Commas indicate decimal points.

The dependence of the amplitude of a reaction on the nature of the load can be demonstrated by Figure 9, showing the mean values of the reaction of the pulse and systolic arterial pressure in the same athletes (leading sprinters in the country) after various loads as a function of increasing state of training. We see that in the sports form, the pulse reaction to rapid running is considerably higher than the reaction to running at a slower rate. With repeated, less than maximal loads under laboratory conditions (three-minute run with 30-sec dash at the end) in the sports form the pulse reaction is less than at a lower state of training. The systolic pressure reaction, on the contrary, is highest with highest training. A similar direction in changes of pulse and pressure can be observed for repeated loads in training (4 × 60 m). In contrast to this, after competition at a higher level of training along with the higher running speed more expressed changes in pulse and systolic pressure are observed. /73

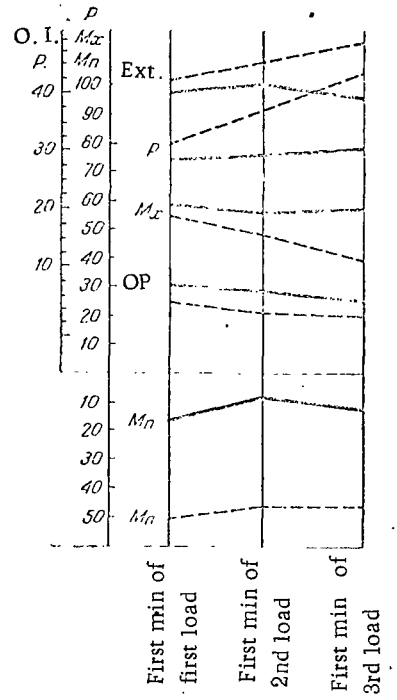


Figure 8. Change in the Reaction of the Cardiovascular System to Loads with Increasing State of Training. Vertical line shows values of indicators studies, horizontal line shows periods of investigation: dotted line, insufficient training; solid line, sports form.

Therefore, the decisive factor in the determination of the state of training according to data from studies of circulation should be not the absolute value of the changes of any given indicator during physical loads, but their correspondence to the nature of the work performed, its intensity and duration.

Great changes in response to work of moderate intensity are not suitable for the organism and always indicate inadequate adaptation of the circulation and, consequently, inadequately high state of training. With higher loads, performed at higher intensity, very great changes may occur in two different cases. In the first case, they are a manifestation of compensatory hyperfunction of the circulatory apparatus and a high level of development of adaptive mechanisms. They are accompanied in this case by no signs of discoordination, by rapid restoration, by retention of good functional state of the cardiovascular system and high working ability during dynamic observations. In other cases, these changes may serve as an early indicator of exhaustion of the compensatory capabilities of the heart, disruption of the electrolytic and energetic balance of the myocardium, with subsequent reduction of the functional capability of circulation. /75

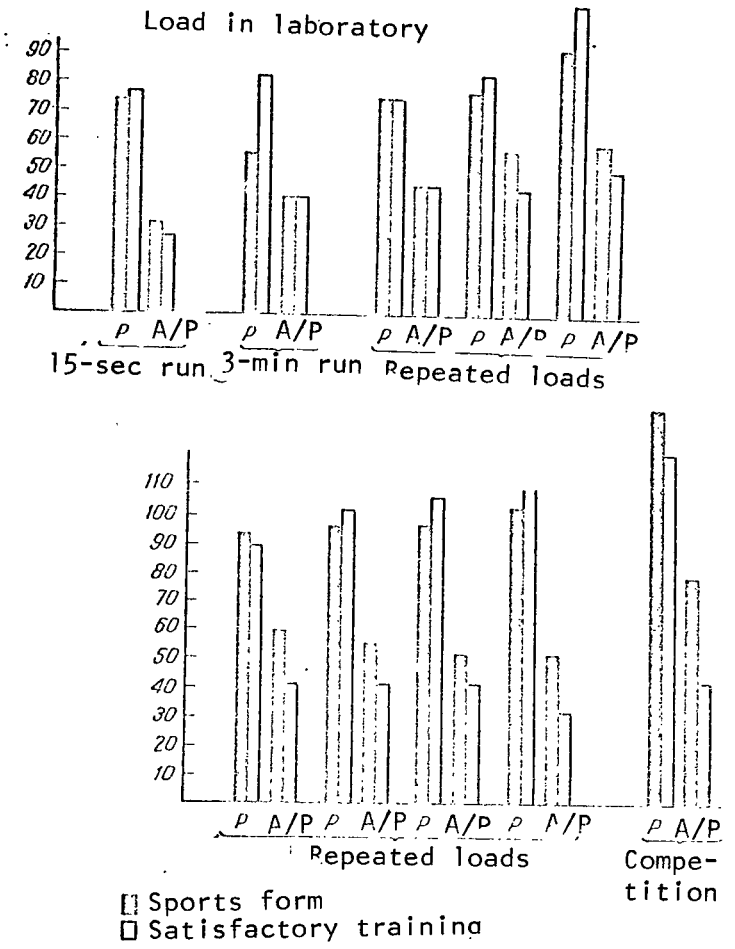


Figure 9. Changes in Pulse and Arterial Pressure After Various Loads in Various States of Training. Dark bars show state in sports form, light bars -- in satisfactory state of training

Consequently, the degree of the changes in hemodynamics in response to load depend not only on the level of training, but on the nature of the load. It is not identical for different functional indicators of circulation, and is influenced essentially by the sports specialty and qualifications of the test subject, individual specifics of his nervous system, etc.

A precise characteristic of the increase in the state of training is the significant acceleration of the working-in, which appears as a more rapid mobilization of the functions, more rapid establishment of a certain rhythm of the activity of the circulatory system optimal for the work to be done. Characteristic also is an easier change in the rhythm with changes in the rate of

work, resulting from the capability of a highly trained organism to adapt its activity rapidly to a change in the conditions of the medium and develop maximum working ability.

An important diagnostic characteristic is a more sharply expressed stability of reactions with repeated loads, in spite of increasing demands on the organism and particularly great acceleration of the processes of restoration. In the state of high training, rapid restructuring of regulation occurs when the load is terminated. This allows the organism to withstand high physical stresses ever better as the state of training increases.

There is considerable interest also in the fact that a change in the response reaction to physical loads appears more sharply in the circulation indicators than in the motor apparatus (the functional state of which is frequently more stable during physical stress than the vegetative component of the reaction). Thus, an experiment with synchronous recording of various functions during physical stresses has shown that in qualified athletes as the state of training increases during the sports season, positive shifts (based on the sum of indicators studied) of the respiratory-circulatory apparatus and nervous-muscular system were seen in 58% of the observations, while shifts of circulation alone were observed in 32% of the cases and in the nervous-muscular apparatus alone -- in 8% of the observations.

Apparently, a change in the functional state of the organism during physical stress resulting from the system of motor-visceral reflexes alone is more rapidly reflected in the less stable link of the motor action -- its vegetative support. It is for this reason that qualified athletes with a high level of general training in many cases retain high adaptation capabilities of the nervous-muscular apparatus even after worsening of the functional indicators of circulation has begun. /76

The selection of loads in the examination of athletes in order to show the degree of their training results from the level of training and qualification of the test subjects considering their age, health, and sports specialization. The loads should be so selected that they reflect both the general and the special state of training, i.e., in addition to the standard functional tests, the reaction of the cardiovascular system to specific loads should be studied.

A good reaction of the circulation to standard loads does not mean that an athlete is capable of showing high results in his own type of sports. For this, in addition to general training, he requires a high level of special training. This latter is achieved not so much by improvement of the general adaptive mechanisms as by improvement of the specific motor actions and adaptability of various functional systems including to a considerable extent the cardiovascular system to the maximum muscular effort (related to repeated performance of a concrete motor act, specific for each type of sports).

The special degree of training is determined using functional tests with repeated loads specific for each form of sports, calling for a comparison of

indicators of working ability and adaptability under various conditions of the external medium (R. Ye. Motylyanskaya and A. V. Martynova, 1958; Ye. F. Likhachevskaya, 1960; Ye. S. Stepanova, 1965; K. F. Slabolyubova and G. A. Goncharova, 1960; V. A. Geselevich, 1966; etc.) as well as a study of the reactions of the organism of athletes to natural maximum training and competitive loads. The selection of cardiological methods in this case is based on the positions listed above but, as a rule, is somewhat simplified in connection with the conditions of the investigation.

As a necessary minimum in a study of the reactions to repeated loads, we should consider investigation of the pulse frequency, arterial pressure, respiration and electrocardiography at the beginning of the first and second minutes after each repetition. It is desirable to record the electrocardiogram during loading. Comparison of data produced with the indicators of working ability during each repetition is obligatory. The selection of methods of investigation under conditions of training and competitive loads must be based on concrete conditions of the investigation, but in any case must be founded on the principle of combined studies. Essential supplementary /77
data in order to characterize the state of training can be produced under these conditions by using the method of additional loading after completion of the principal loading and during various stages of the recovery period.

A third principle in the evaluation of the state of training is the individual treatment of data produced considering the specifics of the athlete, level of his training, type of sport and a number of other factors.

The principal physiological regularities reflecting the development of this state of training are identical for all categories of athletes, but there are certain individual specifics. For example, the higher the general level of training, the less the indicators of general training change during the course of the sports season and the greater, consequently, the attention which should be turned to even slight changes. But even in highly qualified athletes who have relatively high initial level as a result of year-round training, further changes in the functional state of the organism during the sports season, reflecting the dynamics of the state of training, are rather sharply expressed. Although with the large work volume and considerable variety involved in contemporary training in all forms of sports differences in the dynamics of the functional state between representatives of various sports specialties are removed to a certain extent, still the changes in the functional state of circulation, in particular the indicators reflecting the functional state of the heart in connection with the dynamics of training, are more clearly seen in sportsmen training for types of sports involving general and specific endurance. A capability for rapid mobilization of functions and high reactivity of the circulatory apparatus during loads of maximum intensity, more rapid alteration of the rhythm of activity are more clearly seen in athletes training for fast work and in representatives of sports games and other categories.

A considerable role is also played by age. For example, for children and adolescents, as well as for young people even with good training, high

reactivity of the circulatory apparatus is characteristic for any physical stress, as is slower recovery. During adolescence, the functional changes related to increasing state of training are less clearly expressed and are not observed in such regular sequence as in adults. In old age even a well trained organism shows lower reactivity. Thus, older persons show a less sharply expressed pulse reaction, more frequent disruption in cardiac rhythm, and frequently the necessary correlation in the activity of individual links /78
of the cardiovascular system is not present, and changes in arterial-precapillary lumen are particularly noticeable where, apparently, the disruptions in regulation characteristic of age are manifested (Ye. F. Likhachevskaya, 1966).

In evaluating the data produced we should also consider that there are individual athletes who, even in a state of good training, show relatively imperfect individual functions, which is not reflected in their general working ability due to the compensatory-adaptive capabilities of the trained organism.

The clearest increase in the state of training, according to data from studies of the circulatory apparatus, generally occurs during the first stages of training (in general or during the preparatory period for each season after the rest between seasons). As the qualifications of the athletes increase, the values of various functional indicators change over smaller limits. In qualified athletes with today's methods of training, which continue year round, with long competitive seasons and brief preparatory periods, the increase in the state of training occurs quite rapidly. It should be kept in mind that there are essential individual differences in the rate of increase of the state of train in various persons even with identical training methods.

Dynamic observations have shown that during the course of training the changes in principal functional indicators of hemodynamics occur gradually, right up to the achievement by the athlete of a high level of general training. Then, the indicators of the circulatory system defined under conditions of muscular rest and the reaction of the cardiovascular system to functional tests stabilize, while a high level of training is maintained, decreasing during detraining or fatigue. Further increases in working ability in a concrete form of sports are manifested during medical examination primarily as continuing improvement of the adaptive mechanisms to specific loads, reflecting an increase in special training.

In many forms of sports in which competition is performed almost year round, the indicators of special training are also relatively stable, fluctuating slightly during individual stages as a function of the decrease or increase in loads in correspondence with the training plan or as the functional state worsens due to fatigue.

Dynamic observations have also shown that in the state of highest training -- the sports form, each athlete has optimal values of various /79
functional circulation indicators for himself, which are repeated with very slight variations each time the athletes achieve this level of training.

With insufficient training, the same athletes often show significant fluctuations in these quantities (Table 5). In various persons these quantities may differ considerably in the sports form. Thus, each athlete when in his state of highest training, has certain characteristic individual values of most indicators specific for himself, reflecting the functional state of the circulatory apparatus. We observed a lack of this stability only for easily excited athletes and persons who showed the initial signs of fatigue along with sports form of training.

A precise boundary cannot always be established between good training and its highest level (sports form) according to the data from medical examination, since the indicators of adaptability and recoverability are quite similar with these two states of training. However, the sports form, in addition to the highest indicators of working ability and excellent general condition, quite characteristically shows sharply expressed stability of the functional indicators and reactions of the cardiovascular system. /80

As the state of training is decreased by fatigue, the worsening of the indicators of the functional state for various persons may occur primarily due to a change in various indicators. All of this indicates the necessity of strict individual evaluation of the results of investigation of circulation for a given state of training. For this purpose, the quantities measured should be compared not so much with the average indicators, various tables and standards (since the limits of physiological fluctuation of individual characteristics with optimal training for various athletes are rather great) as with the values characteristic for a given athlete in various levels of training.

It is because of the considerable individual differences in the process of development of the state of training that its level can be most properly evaluated by dynamic observations, which also should be looked upon as one of the principal factors to be considered.

It is necessary to consider in each concrete case the conditions of the medium in which the examination is being performed, since the state of the athlete on a given day, the activities immediately preceding the examination (in particular, residual phenomena from fatigue after training exercises, recent consumption of food, insufficient sleep, various disruptions of the bodily regime, general fatigue, etc.), time of examination, etc. may have an essential influence on the results of the examination and cause errors in the evaluation of the state of training of the athlete. This concerns primarily the most labile indicators of circulation, reflecting the function of excitability, level of arterial pressure, etc., which change rapidly as a function of a number of supplementary factors, in contrast to the more stable indicators, for example, those characterizing the function of conductivity and contractability.

TABLE 5. DEGREE OF CORRESPONDENCE OF VALUES OF CERTAIN FUNCTIONAL INDICATORS OF CIRCULATION IN ATHLETES WITH MULTIPLE EXAMINATION WHEN IN SPORTS FORM AND INSUFFICIENT TRAINING (PERCENT OF CASES)

Degree of correspondence and state of training / Characteristics	Identical or very similar quantities				Significant fluctuations in quantities			
	Football		Sprinters		Football		Sprinters	
	Sports form	Insufficient training	Sports form	Insufficient training	Sports form	Insufficient training	Sports form	Insufficient training
Pulse frequency	74.4	12.5	68.5	18.0	13.4	54.3	6.2	53.3
Arterial Pressure:								
Systolic	64.3	30.0	68.7	13.3	15.3	18.7	38.5	46.9
Diastolic	66.3	31.2	56.2	33.5	9.4	12.5	18.0	20.0
Mean	65.8	14.8	70.0	16.0	8.0	23.2	9.8	19.8
Reaction of pulse and arterial pressure to standard loads	92.0	5.0	87.5	13.7	--	58.0	--	56.3

The combined approach, functional direction, dynamic nature of observations and individual evaluation of data produced by investigation of circulation allow us to produce the necessary objective materials for diagnosis of the state of training during medical examination of athletes.

PART II

METHODS OF INVESTIGATING THE CARDIOVASCULAR SYSTEMS OF ATHLETES

Modern cardiology has available a large number of methods for investigating various functions of the cardiovascular system and biophysical manifestations of the functional state of the heart and vessels. Naturally, they are not all equivalent in the information which they yield and, consequently, are not all expedient for utilization in sports medicine. /81

At the present time investigation of the fundamental parameters of cardio- and hemodynamics is taking on ever greater significance in medical testing and sports physiology. In addition to this, attention continues to be turned to the study of the electrical and mechanical manifestations of the cardiac activity of athletes. The arsenal of X-ray methods of investigation of the heart also continues to expand. Finally, radiotelemetric transmission of cardiological information is being ever more widely introduced.

The problem of transmission and collection of information is one of the most important problems. It determines the possibilities for utilization of various methods of investigation of the heart under conditions of the performance of functional tests in athletes or under conditions of natural performance in training and competition. However, the recording of various manifestations of the activity of the cardiovascular system directly during muscular work continues to be difficult. /82

The second portion of our book is dedicated to a broad range of methodological problems which are pressing for sports cardiology. Due to the absence of any single classification of methods of investigation of circulation, the material in this portion is divided rather arbitrarily. Some chapters present data not only on physical and physiological characteristics of the methods of investigation, but also some results produced by the authors using various methods. A certain degree of repetition of data presented in the first portion of the book is unavoidable here (this repetition was reduced to a minimum during the editing process).

CHAPTER IV

INVESTIGATION OF CERTAIN PARAMETERS OF CARDIO- AND HEMODYNAMICS

ABSTRACT. This article is a historical review of the investigatory methods used to define certain parameters of cardio- and hemodynamics. Included are: methods of investigation of cardiac output, methods of phase analysis of the cardiac contraction, particularly as used in athletes, and changes in duration of cardiac cycle phases during the performance of various physical tasks.

Since the time of the classical investigations of Frank (1895), Starling (1914), Straub (1914), and Wiggers (1921), the principal parameters of the dynamics of circulation have been considered the cardiac output (blood volume per beat and per minute), the intracardiac pressure and duration of phases in the cardiac cycle. On the basis of these parameters, we can make up an operating diagram of the heart which characterizes the movement of a representative point in three-dimensional space. The coordinates of this space are pressure, volume and time (P-V-t diagram). /83

In sports medicine, only two parameters are rather broadly studied -- the cardiac output and phases of the cardiac cycle, while the third parameter -- intracardiac pressure -- is analyzed only in special experiments. The problem is that the methodological difficulties and dangers involved in investigation (requirement for veinous or arterial catheterization of heart cavities) prevent the recording of intracardiac pressure during medical examination.

Methods of Investigation of Cardiac Output

The determination of the cardiac output is of great significance for a quantitative evaluation of the contractive function of the heart and the study of the intensity of blood circulation of the organs and tissues under conditions of muscular work. The systolic volume and blood volume per minute are frequently called the principal hemodynamic indicators. The study of these indicators is an obligatory component of each well performed

investigation in sports cardiology. In addition to the important independent significance of information on the systolic volume and blood volume per /84
minute, this information is necessary for calculation of other indicators of cardio- and hemodynamics, for example such indicators as the mechanical work of the ventricles and the hydraulic resistance of the vessels in systemic and respiratory circulation.

Preliminary notes. The quantity of blood in milliliters expelled from the ventricle into the aorta in one contraction is called the systolic blood volume. The blood volume per minute is that quantity which is expelled by the ventricle during one minute. The dimensionality of this indicator is in liters per minute (ℓ/min).

Many investigations have shown that among the factors determining the systolic volume and volume per minute, great significance must be given to the height and weight of the test subject. In order to level individual fluctuations in volume per minute, Grollman (1932) suggested that the blood volume per minute be related to a unit of body surface. The quantity which he produced in this way he called the cardiac index. Its dimensionality is liters per minute per square meter ($\ell/min/m^2$), and has no concrete physical sense. By analogy with the cardiac index, the systolic index or beat index are defined.

The problem of studying cardiac output has almost 100 years history and continues to be developed and improved, as a result of which a large number of various methods are available for investigation. V. L. Karpman (1963) suggested a classification plan facilitating theoretical analysis of the principles upon which the determination of the quantity of cardiac output is based. In correspondence with this plan, methods of determination of systolic volume and volume per minute are distributed into the following groups: 1) methods based on the Fick principle -- the direct method of Fick, the acetylene method, etc.; 2) methods based on the Stewart-Hamilton principle -- methods involving dilution of a dye, the method of thermal dilution, etc.; 3) methods based on the Frank principle -- the methods of Broemser and Ranke (1930), Wezler and Boger (1937, 1939), etc.; 4) methods based on the Newtonian laws of mechanics -- methods using high-frequency and low-frequency ballistocardiography.

This classification covers only the most widely used methods of investigating the blood volume per minute and systolic volume. Certain other methods not included in this classification will also be briefly analyzed. We cannot discuss the methodological details of the determination of cardiac output, so we will present a review only of the principal specifics of the various methods.

Methods based on the Fick principle. In 1870, Fick formulated the first physiologically well founded principle for calculating the volume of blood /85
flow in an integral organism on the basis of the quantity of material entering the blood in one minute (R) and the degree of increase of its concentration in the blood (D). For example, using the arterial-veinous difference it is not

difficult to determine the increase in concentration of oxygen in the blood passing through the vessels of the lungs (D_k). If we know the quantity of oxygen consumed in the lungs in one minute (R_k), then:

$$R_k = D_k \cdot V_m, \tag{10}$$

where V_m is the blood volume per minute, R_k is easily determined from the oxygen consumption in the air being breathed. From this:

$$V_m = \frac{R_k}{D_k}. \tag{11}$$

It is obvious that equation (11) is valid not only for oxygen, but for carbon dioxide and for certain indifferent gases and materials.

In order to calculate the volume per minute according to the Fick principle, an artery is punctured for determination of oxygen in the arterial blood and the right cavities of the heart are catheterized to analyze the content of O_2 in the mixed veinous blood. This is the so-called direct method of Fick, long used in physiological investigations; its usage for the study of the cardiac output of man was begun only in the 1940's, after Cournand (1944) introduced the heart probe method to clinical practice. The direct Fick method is a precision method for determining the blood circulation volume per minute.

The acetylene method developed by Grollman (1928) has been particularly broadly used in physiological and clinical practice. In the Soviet Union, it is generally used with the improvement introduced by V. V. Parin (1935, 1937) and I. I. Khrenov (1940). Acetylene is easily dissolved in blood plasma and has no toxic influence on the organism.

Acetylene and certain other gases indifferent to the organism have a constant solubility coefficient. The volume per minute is determined by dividing the value of the quantity of gas dissolved in the blood during the time of respiration of the gas mixture by the solubility coefficient of the gas.

Calculation is based on the following considerations. If the test subject has inhaled a mixture of air with a known quantity of acetylene and held his breath for t sec, the quantity of acetylene in the mixture exhaled will be less by a certain quantity ΔX. The quantity of acetylene dissolved in the blood in one second can be determined from the ratio $\Delta X/t$, and in one minute -- from the ratio $60 \cdot \Delta X/t$. The quantity of acetylene dissolved in the /86
blood per unit volume will be D_a. The volume of blood passing through the

lungs per minute (V_m) will dissolve $V_m D_a$ acetylene. Thus, we can write:

$$V_m \cdot D_a = 60 \cdot \frac{\Delta X}{t}. \qquad (12)$$

The acetylene method was used by G. I. Markovskaya (1955) in a study of the cardiac output of athletes at rest and under conditions of muscular work.

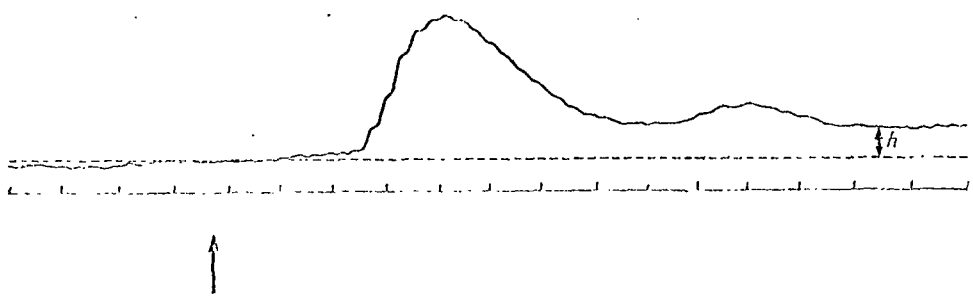


Figure 10. Curve of Dilution of Dye. The arrow represents the moment when the dye (Evans blue) was injected into the vein. The first rise in the curve reflects the change in material concentration during its first passage, the second rise corresponds to the recirculation wave; h is the distance (in mm) between the initial level of the curve and the level achieved after complete mixing of the dye. The vertical lines are time marks (at 4-sec intervals).

The method of determining the volume per minute using repeated respiration of CO_2 is also based on the Fick principle. This method was used by Christensen in the 1930's. In recent years, interest in this method has increased again, particularly in investigations of the volume per minute during muscular work (Defares et al., 1961; Klausen, 1965; etc.).

Methods based on the Stewart-Hamilton principle. At the end of the last century, Stewart (1897) performed quantitative measurement of blood flow by recording the change in electrical conductivity of the flowing blood as common salt was injected into it using a bridge circuit. The Stewart principle was extended a half century later by the works of Hamilton and his colleagues (1928-1933) who suggested a mathematical theory for the method and introduced it to practice.

In determining the cardiac output by the dye dilution method, a solution containing a known quantity of dye (Evans blue, Kumassi blue,

cardiogreen or other coarsely dispersed dye, harmless to the organism) is rapidly injected into the veinous portion of the blood flow, then its concentration is measured in the arterial blood. The change in dye concentration in the blood is recorded using an oximeter (cuvette or ear type), densitometer and special automatic circuits. /87

A qualitatively satisfactory concentration curve (necessary for calculation of cardiac output) can be produced using the Ye. M. Kreps oxyhemograph, manufactured by the "Krasnogvardeyets" plant, in which the chart drive rate can be increased to 2-5 mm/sec.

The increase in the concentration curve (Figure 10) begins after some time has passed following injection of the dye (this interval of time makes it possible to determine the rate of blood flow over the sector: point of injection of dye -- location of oximeter pickup). The curve rapidly reaches its maximum, then begins a smooth decrease, interrupted by a second increase -- the recirculation wave. Kinsman, Moore and Hamilton (1929) in their early investigations established that when the concentration curve was drawn onto semilogarithmic paper, its descending portion (except for recirculation) is linear. Therefore, in order to separate the recirculation wave, linear extrapolation is performed from the beginning of the descending portion of the concentration curve, reproduced on semilogarithmic coordinates (Figure 11).

In calculating the blood volume per minute by the dye dilution method, the following formula is used:

$$V_m = \frac{60 \cdot I}{C \cdot t} \cdot \frac{100}{(100 - Ht)}, \qquad (13)$$

where I is the quantity of dye injected (in mg), C is the mean concentration of the dye (in mg/ℓ) calculated from the area bounded by the dilution curve, t is the length of the wave from the dilution curve in sec (from its origin to the moment when the extrapolated portion crosses the abscissa) or, in other words, the time of passage of the dye, Ht is the hematocrit indicator. The greatest difficulty in determining the blood volume per minute by the Stewart-Hamilton method arises in calculating the area bounded by the concentration curve, since one cannot always be sure that the linear extrapolation has been performed properly. /88

Precise determination of the volume per minute requires calculation of the calibration index which is determined from the ratio of concentration of the dye in the veinous blood after complete mixing (after 10 min) to the quantity h (see Figure 10), determined from the distance between the initial level of the curve and its level established after complete mixing of the dye. The concentration of the dye is determined using a type SF-4 spectrophotometer, a type FEK-M photocalorimeter, by chemical analysis, etc.

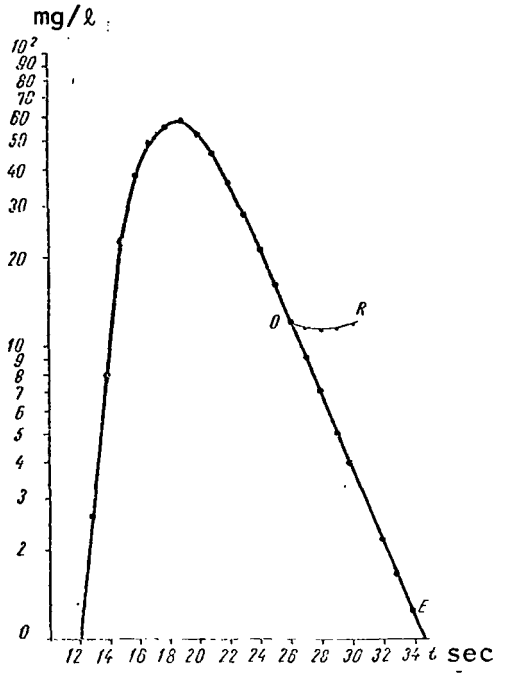


Figure 11. Concentration Curve on Semilogarithmic Coordinates. OR, recirculation wave; OE, extrapolated portion of curve.

The dye dilution method can be used to determine the central blood volume (V_c). For this, the volume of blood per second ($V_m/60$) must be multiplied by the mean time of intrathoracic circulation (T):

$$V_c = \frac{V_m}{60} \cdot T. \qquad (14)$$

The mean time of intrathoracic circulation is calculated as follows:

$$T = \frac{\Sigma C \cdot t}{S}, \qquad (15)$$

where $\Sigma C \cdot t$ is the sum of the products of time (in sec) after the injection and corresponding sectors of dye concentrations, S is the sum of dye concentrations (in mg/ℓ) at each second during the first passage of the dye.

The Stewart-Hamilton principle has begun to be used to investigate dilution of other indicators as well. For example, a logical development of the dye method is the method of injection of materials labeled with radioactive isotopes.

In recent years, the method of determination of the blood volume per minute from curves of so-called thermal dilution has become widespread. In this method, a solution whose temperature is below the temperature of the blood is injected into the cavity of the right atrium or ventricle. A special pickup reacting to changes in temperatures (thermistor) is placed in the femoral artery. The blood volume per minute is determined from the Stewart-Hamilton formula modified for Fegler thermal dilution curves: /89

$$V_m = K \frac{V_I (T_B - T_I)}{\int_0^\infty \Delta T_B \, dt}. \qquad (16)$$

In this formula, $\int_0^\infty \Delta T_B dt$ is the area bounded by the curve of the temperature

change, K is a coefficient reflecting the ratio of specific heat capacity and specific gravity of the solution injected to the specific heat capacity and specific gravity of the blood, V_I is the volume of solution injected, T_I is the temperature of the solution injected, and T_B is the blood temperature.

Analysis of data from the literature shows that the thermal dilution method is quite promising if only due to the fact that it allows repeated measurements of the blood volume per minute in a single investigation. One defect limiting the usage of this method for investigation of athletes is that it requires probing the cavities of the heart. In this aspect, the dye dilution method has an advantage, in that an injection into the peripheral sector of the veinous network yields rather precise information concerning the blood volume per minute and other hemodynamic indicators.

Many comparisons of the results produced using the methods of indicator dilution with results produced by the direct Fick method have shown that there is a high degree of correlation between them (the correlation coefficients according to the data of various authors have fluctuated between +0.85 and +0.99).

Methods based on the Frank principle. The so-called physical methods of determining the systolic volume have been based on the works of Frank (1920, 1926, 1930), dedicated to a study of the wave processes in the arterial system and applications of the theory of elasticity to biological objects. Frank established that the relative increase in cross section of a vessel is directly proportional to the increase in intravascular pressure and inversely proportional to the square of the rate of propagation of the pulse wave.

Based on this, Broemser and Ranke (1930), and later Wezler and Boger (1937) as well as certain other authors suggested a formula for calculation of the systolic blood volume from data produced by measuring arterial pressure and certain sphygmographic indicators. The formula of Broemser and Ranke is as follows:

$$V_s = \frac{Q(P_s - P_d) \cdot z \cdot C \cdot E}{\rho \cdot a \cdot (C - E)}, \qquad (17)$$

where V_s is the systolic blood volume (in mℓ), Q is the cross section of the aorta (in cm^2), P_s is the systolic arterial pressure, P_d is the diastolic arterial pressure (in dyn/cm^2), z is a dimensionless constant equal to 0.6, C is the duration of the cardiac cycle (in sec), E is the duration of the expulsion period (in sec), ρ is the density of the blood, assumed at 1.06, a is the rate of propagation of the pulse wave in the aorta (in cm/sec). /90

The cross section of the aorta Q is determined from Suter or Frucht tables. The arterial pressure is best determined using the oscillographic method. N. N. Savitskiy measures the pulse pressure (P_s - P_d) as the

difference of the lateral systolic and minimal pressure on the tachooscillogram. The pressure values, expressed in millimeters of mercury, are multiplied by 1332 in order to translate them to dyn/cm^2.

The formula of Wezler and Boger is as follows:

$$V_s = \frac{Q \cdot (P_s - P_d) \cdot T_{fem}}{2p \cdot a}. \qquad (18)$$

In this formula the symbols are the same as in formula (17). T_{fem} is the duration of the base oscillation of the femoral pulse in sec. Determination of this quantity is illustrated by Figure 71.

Calculation of the systolic volume using the formula of Broemser and Ranke is easy to perform if there is an N. N. Savitskiy mechanocardiograph or two-channel sphygmographic attachment for the "Orion" five-channel electrocardiograph available. In order to determine V_s using the Wezler-Boger formula, only a qualitative recording of sphygmograms with the carotid and femoral arteries are required, since the arterial pressure is determined after Korotkov. The accuracy of these methods has been tested by comparison with measurements of systolic volume and volume per minute using the Fick method. The differences in systolic volume have been reported by various authors (Hauch, Donnel, 1954; Schmid, Reubi, 1953; Warembourg et al., 1963) as being rather considerable (25-30%). The insufficient accuracy in determining the absolute values of systolic volume using formulas (17) and (18) is compensated for by the possibility of frequent, nontraumatic investigation of this indicator with any test subject. This quality of the physical methods of investigation of cardiac output makes them promising for usage in sports medicine, where dynamic observations of test subjects are of extreme importance.

In recent times, we have begun to use combined determination of V_s using the Broemser-Ranke and Stewart-Hamilton methods (V. L. Karpman and L. A. Ioffe, 1966). When this is done, we can determine for a given test subject not only the precise value of V_s, but also its changes arising under the influence of various actions. The technique of this method consists of the following. While the test subject is at rest, the concentration curve of dye dilution, /91 sphygmogram of the carotid and femoral arteries and a tachooscillogram are recorded. On the basis of these recordings, the blood volume per minute is determined using the Stewart-Hamilton method. The value of V_m is divided by the number of heartbeats and, consequently, the blood volume per beat is determined for the given test subject with good accuracy. Using the value of V_s thus determined, and also on the basis of data on the value of a, (P_s - P_d), C and E, the values of the constant factors in formula (17) are determined for the given test subject. These constants in formula (17) are

Q, z and ρ. Assuming their relationship for the given test subject as $K = Q \cdot z/\rho$, we find it from the following equation:

$$K = \frac{Q \cdot z}{\rho} = \frac{V_s \cdot a \cdot (C - E)}{(P_s - P_d) \cdot C \cdot E}. \quad (19)$$

Knowing the individual value of K, we perform further investigation of the cardiac output of the test subject during work[1] and recovery by using a reduced version of the Broemser-Ranke formula:

$$V_s = K \frac{(P_s - P_d) \cdot C \cdot E}{a \cdot (C - E)}. \quad (20)$$

Similarly, the value of K can be calculated for the Wezler-Boger formula.

Methods based on Newtonian mechanics. Henderson (1905) was apparently one of the first to use the laws of Newton for determination of the systolic volume, using a method which Starr (1939) later called ballistocardiography. Each time the heart contracts, under the influence of the expulsion of blood and other internal reactive forces, the human body receives an acceleration, the value of which is proportional to the operating force, according to Newton's second law of motion:

$$F = m \frac{dV}{dt}. \quad (21)$$

These phenomena are recorded using a ballistocardiograph, and can be used, according to simple theoretical considerations, for determination of the systolic blood volume. Starr and his colleagues (1939), Nickerson (1947) and certain other authors suggested special formulas for calculation of cardiac output using ballistocardiography. However, as it turned out, high- and low-frequency ballistocardiographs do not provide for precise determination of the blood volume per beat. Therefore, the formulas of Starr and Nickerson are not used at the present time. /92

The failures of quantitative ballistocardiography in the first stages of development of the method led to searches for improved instrument designs. As a result, ultralow-frequency and aperiodic elongation ballistocardiographic tables were developed.

[1] During muscular work, measurement of arterial pressure and recording of the tachooscillogram are difficult. The auscultative method is quite unsuitable under these conditions. Therefore, direct measurement of arterial pressure is most desirable under these conditions.

The extensive literature on the investigation of systolic volume under normal and pathological conditions, the simplicity of investigation using elongation ballistocardiography, the positive test comparisons with the Fick method allow us to look upon the Klensch method (1956, 1958, 1959) as a promising method for sports medicine. This is indicated by the investigation of Klensch and Hohnen (1958) in which, using elongation ballistocardiography, changes in systolic volume during muscular work in healthy persons (including athletes) were investigated.

Certain other methods for determining cardiac output. A number of comparatively new methods for determining cardiac output are difficult to relate with any classification plan. It might be assumed that certain of these methods can be used in sports medicine.

The investigation of the artificial systole in corpses allowed Starr (1954) to produce an empirical dependence for determination of systolic volume:

$$V_s = 90{,}97 + 0{,}54\Delta P - 0{,}57P_d - 0{,}61 \cdot B, \tag{22}$$

where ΔP is the pulse pressure (in mm Hg), P_d is the diastolic pressure (in mm Hg), B is the age (in years).

In a special investigation, the values of systolic volume calculated by this formula for 400 patients was compared by Starr with the values calculated using the Fick method, the acetylene method, the dye dilution method.

Satisfactory results were produced. However, in the objective works by Luthy et al. (1957), G. A. Glezer (1964), Shteyngardt (1964) when the quantities produced by the Starr formula were compared with those produced by the Fick and Hamilton method, it was shown that the Starr formula gives satisfactory results only for healthy persons at rest.

A simplified method for determining cardiac output based on the Vierordt formula was suggested by Frick (1963):

$$V_m = K \cdot \frac{V_B}{T}, \tag{23}$$

where K is a coefficient, the value of which was determined by the author on the basis of his own data as 11.7; V_B is the volume of circulating blood; T is the blood flow time over the arm-ear sector. /93

The correlation between the results produced by the Stewart-Hamilton method and the results produced by calculations using formula (23) was found to be quite satisfactory (r = +0.68). The method excludes the extremely

cumbersome portion of the calculations -- the determination of the area bounded by the curve of indicator dilution, which significantly accelerates measurement of the blood volume per minute.

Calculation of certain indicators characterizing the work of the circulatory apparatus based on data on cardiac output. Information on cardiac output is necessary in order to calculate a number of other physiological indicators, in particular for a determination of the mechanical work performed by the ventricle of the heart.

The magnitude of work performed by the left ventricle (A) can be calculated using the following formula:

$$A = P_m \cdot V_s + \frac{m \cdot v^2}{2g}. \tag{24}$$

In this equation: 1) P_m is the mean arterial pressure which in this case can be conveniently written in grams. For this, the value of P_m expressed in millimeters of mercury is multiplied by 1.32. According to the data of Wezler and Boger (1939), the mean pressure can be rather precisely calculated using the following formula:

$$P_m = P_d + 0{,}43(P_s - P_d), \tag{25}$$

where P_s is the systolic pressure, P_d is the diastolic pressure;

2) V_s is the systolic blood volume (in cubic centimeters), m is the weight of the systolic blood volume (in grams). This quantity is produced by multiplying the systolic volume by 1.05;

3) v is the linear speed of blood in the aorta (cm/sec). In order to determine this figure, we must know the transverse cross section of the aorta (Q), the duration of the expulsion phase (E) and the systolic blood volume (V_s). Then:

$$v = \frac{V_s}{Q \cdot E}; \tag{26}$$

4) The acceleration of gravity (g) is 981 cm/sec^2.

The work is expressed either in kilogram meters or in ergs. The work of the heart over any given time interval (for example per second, per minute or

per hour) is frequently calculated or, which amounts to the same thing, the power of the heart contraction is determined.

Under normal conditions, the work of the heart is expended primarily in overcoming the resistance in the aorta ($P_m \cdot V_s$) and to a considerably lesser extent ($m \cdot v^2/2$ g) in accelerating the blood column. However, as the output is increased, the specific gravity of the kinetic factor in the work of the heart increases essentially (up to 30%). This usually occurs during muscular work. /94

The hydraulic resistance of the vessels of the organism to blood flow, an important physiological indicator, is broadly used in theoretical and clinical investigations. The greatest resistance is presented by the arterioles. Consequently, a calculation of the peripheral resistance might be the purpose of a study of arteriolar tonus, its dynamics under various physiological and pathological conditions. This information is necessary for an investigation of the mechanism of regulation of mean arterial pressure.

The value of hydraulic resistance is usually calculated using the Poiseuille formula, which can be written in the following form:

$$\frac{8 \cdot l \cdot \eta}{\pi \cdot R^4} = \frac{P_1 - P_2}{v}, \tag{27}$$

where l is the length, R is the radius of the tube, η is the viscosity of the liquid, $P_1 - P_2$ is the pressure gradient, **v** is the rate of blood flow by volume. The left portion of the equation (27) represents the hydraulic resistance (W):

$$W = \frac{8 \cdot l \cdot \eta}{\pi \cdot R^4}. \tag{28}$$

Obviously, when humans are being investigated equation (28) cannot be used, since we do not know l, R and η, so that equation (27) is reduced to a form similar to Ohm's law for electrical circuits, so that calculation of peripheral resistance is performed using the following formula:

$$W = \frac{P_m}{V} \cdot 1332, \tag{29}$$

where P_m is the mean arterial pressure (in mm Hg); 1332 is the factor for translating millimeters of mercury to dyn/cm^2; V is the volume of blood per second (cm^3/sec), W is the peripheral resistance (in dyn/sec$\cdot$cm^5).

In equation (29), P_m is taken in place of the pressure gradient between the arteries and veins in the systemic circulation, since the pressure in the veins and right atrium is comparatively low and can be ignored.

Under normal conditions, there is a clear dependence between cardiac output and peripheral resistance (Ye. B. Babskiy, V. L. Karpman and M. A. Abrikosova, 1960), which is expressed by the following formula: /95

$$W = 1250 \cdot V_m^{0.75}, \quad (30)$$

where W is the peripheral resistance (in dyn/sec·cm^5), V_m is the volume per minute (in ℓ/min).

It follows from analysis of equation (30) that in the area of normal and high values of peripheral resistance, cardiac output remains practically unchanged, whereas when W decreases cardiac output progressively increases, reaching 30 ℓ/min as W = 175 dyn/sec·cm^5. Equation (30) establishes the normal relationship between cardiac output and peripheral resistance with various values of the resistance.

Investigation of cardiac output in sports medicine. The investigation of cardiac output in athletes was begun more than one-half century ago. Lindhard (1915) first studied the changes in blood volume per minute occurring under the influence of systematic training. Subsequently, investigation of the volume per minute in athletes attracted the attention of Barcroft (1935), Henderson, Haggart and Dolley (1927), Collet and Lilijestrand (1924), Stewart and Watson (1940), Berger and Olloz (1935), Reindell and Delius (1948), Reindell (1943), Schneider and Crampton (1940), Mellerowicz (1956), Israel et al. (1946), etc.

In the Soviet Union, the cardiac output of healthy persons (including athletes) has been studied by S. O. Vul'fovich and A. V. Kukoverov (1935), A. V. Ionina (1939), Yu. I. Kuznetsov (1958), A. P. Polosukhin (1937), V. V. Skryabin (1950), G. I. Markovskaya (1955), V. N. Kuz'mina (1954), A. N. Vorob'yev (1962), V. V. Vasil'yeva (1965), etc. In these investigations various methods were used to determine the volume per minute and systolic volume, including particularly gas analytic and sphygmographic methods.

Judging from the data of most investigators, in athletes at rest the volume per minute is decreased. It is difficult to compare the results produced by various authors, since they used various methods and limited their investigations to small groups of athletes. In an investigation of a large group of athletes (107 persons), a significant decrease in blood volume per minute (2.74 ℓ/min against 4.8 ℓ/min for untrained persons) was noted by Mellerowicz (1956) who used the Broemser-Ranke method. A decrease in volume per minute in athletes was also observed by Reindell and Delius (1948), Berger

and Olloz (1935), Israel et al. (1964) (physical methods), G. I. Markovskaya (1955), V. N. Kuz'mina (1954) (acetylene method) and many others.

A decrease in volume per minute in athletes at rest is the basis of the so-called "economy principle" (Mellerowicz, 1956). The decreased volume per minute, increased residual systolic volume (Klepzig, 1955), decrease in oxygen consumption at rest all go together to provide a high functional reserve of the "sports heart." G. I. Markovskaya (1955) considers that the changes in volume per minute in athletes reflect to some extent the state of training: under conditions of "sports form" the value of volume per minute for a given person at rest is minimal, while during "detraining" it increases. /96

However, there are other data concerning the value of volume per minute in athletes at rest. Thus, Lindhard (1915), Henderson et al. (1927), A. V. Ionina (1939), Yu. I. Kuznetsov (1958) and certain other authors found a moderate increase in blood volume per minute in athletes at rest. Possibly their results were influenced by the small number of test subjects investigated by these authors, while in certain cases (for example, Lindhard, 1915) there were defects in their methodology as well.

Information on the influence of systematic training on the value of systolic volume at rest has been quite varied. Without going into a detailed analysis of this problem (see Chapter I) we note that an increase in systolic volume at rest is noted as a rule with clear bradycardia. Under conditions of normal rhythm the quantity of blood output by the heart in one contraction is generally decreased in athletes.

Under the influence of muscular work (see also Chapter II) the volume of blood per minute in athletes is increased. This increase is proportional to the intensity of the physical load and the increased consumption of oxygen. According to the data of Noder (1964), the interrelationship between the volume of blood per minute (V_m) and the amount of oxygen consumed (V_{O_2}) can be described by the following equation

$$V_m = \sqrt{\frac{V_{O_2}}{K}},$$

where K is an individual constant.

The investigations of Asmussen et al. (1953, 1955), Astrand et al. (1964) (dye dilution method), Reindell (1960) (direct Fick method), Bevegard et al. (1960) (dye dilution method), Saltin et al. (1964) (dye dilution method) have indicated that the systolic volume reaches its maximum value at 40% oxygen consumption as related to its maximum level. Subsequently, systolic volume remains practically unchanged or, in the opinion of Asmussen

et al. and Reindell, as the intensity of the work increases and the pulse frequency goes up, may even decrease.

Qualitatively different changes are observed when static work is performed (Lindhard, 1915; V. V. Skryabin, 1950; A. N. Vorob'yev, 1961, 1962; etc.). When static effort is applied, usually involving increased muscular tension, the volume per minute of untrained persons increases only slightly, or may even decrease. Most frequently, an increase in pulse frequency is observed, along with a decrease in systolic volume resulting from decreased veinous influx. The decrease in systolic volume is frequently so great that it leads to a decrease in the volume per minute. Upon completion of the work, the systolic volume is increased (although it generally does not reach its initial level). Also, the volume per minute increases. This reinforcement of cardiac activity after the completion of static muscular work apparently depends on the increased veinous return of blood after the tension is released. /97

A different picture has been observed in highly qualified weight lifters, in whom during work an increase in pulse frequency, a slight increase in systolic volume and correspondingly more clearly expressed increase in volume per minute were noted in all cases without exception. Upon completion of work, a sharp increase in the volume per minute and systolic volume are observed.

Phase Analysis of the Cardiac Contraction in Athletes

Phase analysis of cardiac activity provides a large volume of useful information concerning the contractive function of the myocardium. On the basis of a calculation of the duration of the phases of the cardiac cycle and combined indicators of cardiodynamics, we can objectively describe the state of the contractive capacity of the myocardium, investigate the quality of the regulation of the circulatory apparatus and analyze the reaction of the heart to various types of loads.

Phase analysis has begun to be used in sports medicine comparatively recently, just since rather precise methods of temporal analysis of the cardiac cycle have been introduced to investigative practice. Thus, in recent times, a number of interesting investigations have been performed using phase analysis in sports medicine (Ya. G. Ablov, 1961; O. N. Belina, 1965; R. D. Dibner, 1963; L. A. Ioffe, 1962; K. M. Smirnov and N. V. Zaytseva, 1964; V. A. Tishler, 1963; Ya. I. Ul'brikh, 1962; N. I. Yachmenev, 1959; Yu. K. Shkhvatsabaya, 1964; V. G. Leoshenko, 1966).

Modern concepts concerning the phase structure of the cardiac cycle are founded on the classification plan of Hurthle (1891) and Wiggers (1921). Hurthle (1891) divided the cardiac cycle into four periods -- the stress period, expulsion, relaxation and filling. Wiggers (1921) differentiated three systolic phases (the phase of isometric contraction, phases of maximum /99

and reduced expulsion) and five diastolic phases (protodiastole, phase of isometric relaxation, phase of rapid and slow filling and auricular systole).

After the work of Holldack (1951), it became obvious that the systolic phases should also be given an electromechanical latent period, beginning practically synchronously with depolarization of the myocardium and ending at the moment of closure of the atrioventricular valves.

The diagram (p. 90) presents the phase structure of the cardiac cycle in somewhat generalized form. A brief physiological characterization of each of these phases will be presented below when we discuss the cardiomanometric method of phase analysis.

Certain methods of investigating the phase structure of the cardiac cycle. At the present time, there are a large number of methodological approaches allowing one to compose an idea of the duration of the phases of the cardiac cycle. Not all of these methods are equal either in their accuracy or in the volume of information which they present.

In this chapter we present information on the two most precise methods of phase analysis -- the cardiomanometric and polycardiographic methods. In Chapter VI we will demonstrate the possibilities of dynamocardiography and kinetocardiography in the study of the duration of the phases of the cardiac contraction, while in Chapter VII we will outline the possibilities of electrokymography.

Cardiomanometry is a precision method of phase analysis. The calculation of the duration of phases using this method is performed by analyzing synchronously recorded (or synchronized) curves of the pressure in the atrium, ventricle and the corresponding main vessel -- the aorta or pulmonary artery and the electrocardiogram (Figure 12). Each of these curves shows physiologically determined points, the intervals between which correspond to the phases of the cardiac cycle.

Let us analyze the phase structure (in the sequence shown on the diagram).

The phase of asynchronous contraction (otherwise called the electromechanical latent period, Umformungszeit, etc.) corresponds to the Q-B interval (Figure 12). It represents the time of propagation of the contractive wave through the myocardium. At the end of this phase (point B), the intraventricular pressure begins to increase. During the phase of asynchronous contraction, a change in the form of the ventricles occurs (Holldack, 1951; V. L. Karpman and V. S. Sinyakov, 1965).

Cardiac cycle
Diastole
Period of relaxation
Protodiastolic interval
Phase of isometric relaxation
Phase of rapid filling
Period of filling
Phase of slow filling
Auricular systole
Systole
Period of stress
Phase of isometric contraction
Phase of synchronous contraction
Mechanical systole
Period of expulsion
Phase of maximum expulsion
Phase of reduced expulsion

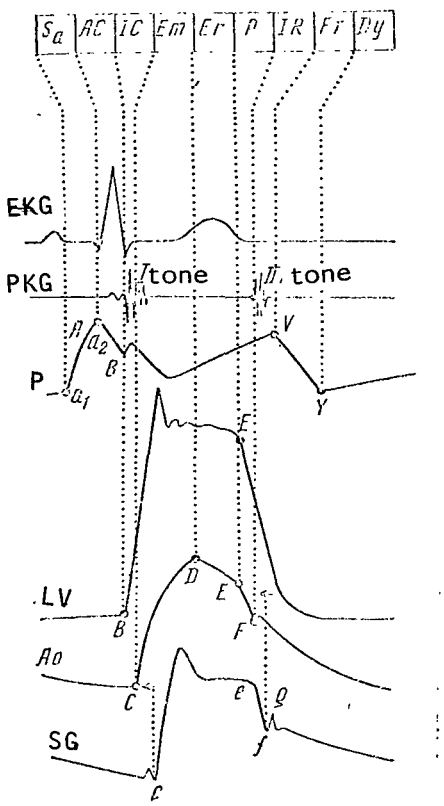


Figure 12. Criterion for Separation of Cardiac Contraction Cycle into Phases. EKG, electrocardiogram; PKG, phonocardiogram; P, curve of pressure in atrium; LV, Curve of pressure in left ventricle; Ao, curve of pressure in aorta; SG, sphygmogram of carotid artery. Abbreviated representations of phases of cardiac cycle: Sa, auricular systole; AC, asynchronous contraction; IC, isometric contraction; Em, maximum expulsion phase; Er, reduced expulsion phase; P, protodiastolic interval; IR, isometric relaxation phase; Fr, phase of rapid filling, Dy, diastasis

The phase of isometric contraction /100
(otherwise called the closed valve period, phase of isovolumetric contraction) corresponds to the B-C interval (Figure 12). It is characterized by a sharp increase in the intraventricular pressure to the level of the final diastolic pressure in the main vessels. Rushmer (1956), and following him others (Hawthorne, 1961; S. F. Oleynik and P. M. Balabayeva, 1963; V. L. Karpman and V. S. Sinyakov, 1965) showed that during the period of closed valves, the ventricles are contracted in a nonisometric regime. The longitudinal diameter of the ventricle decreases, while its transverse diameter increases.

The total length of the phases of asynchronous and isometric contraction correspond to the duration of the period of stress in the concepts of Hurthle (1891) and Blumberger (1942).

During the period of expulsion (C-E interval on Figure 12) the emptying of the heart, as Wiggers showed (1921), is accomplished unevenly. Thus, a considerable emptying occurs at the very beginning of this period. At the end of the period, a negative pressure gradient may even arise in the system ventricle-aorta (Spenser and Greiss, 1962). It follows from this that separation of the expulsion period into individual phases -- the phase of maximum expulsion (the interval C-D on Figure 12) and the phase of reduced expulsion (the interval D-E on Figure 12), as was done by Wiggers (1921), has concrete physiological significance. However, essentially, no criterion for this separation exists. The usage of pressure curves in the principal vessels for this purpose yields only /101
approximate data. Other methods -- sphygmography, electrokymography -- produce even less accurate data.

Our attention is drawn to the physiological uncertainty of the concept "systole." Apparently, it is expedient to speak of two forms of ventricular systoles -- the mechanical and the general systole (V. L. Karpman, 1965). The mechanical systole (interval B-E) corresponds to the time during which high pressure is maintained in the ventricles. The general systole (Q-E interval) is the time during which the contractive process occurs in the myocardium.

The initial portion of the first diastolic period -- the period of relaxation -- is represented as the protodiastolic interval (E-F on Figure 12). The beginning of this interval corresponds to the beginning of relaxation of the myocardium, the end -- to the moment of closure of the aortic valves (point F). This is followed by the phase of isometric relaxation (F-V interval) which the earlier authors referred to as the second period of closed valves. During this phase, relaxation of the myocardium occurs under nonisometric conditions. This resulted in the suggestion that this phase be called the isovolumetric relaxation phase (Hawthorne, 1961; S. F. Oleynik, P. M. Balabayeva, 1963).

After the pressure in the ventricle decreases to the level of the pressure in the corresponding atrium (point V), the cuspid valves open and the period of filling of the ventricles with blood (interval V-Q) begins. Luisada (1958) divides this period into the phases of rapid passive, slow passive and rapid active filling, which in general corresponds to the division of this period into the phase of rapid filling (V-Y interval on Figure 12), diastasis (interval Y-a_1 on Figure 12) and auricular systole (interval a_1-a_2 on Figure 12).

At the beginning of the phase of rapid filling, the fibers of the myocardium continue to relax. During this phase the cavities of the ventricles are practically completely filled with blood. During diastasis, filling of the ventricles is either very slight or completely absent. During the auricular systole, forced filling of the ventricles occurs. During this phase of the cardiac cycle, a certain presystolic stress of the myocardium and its initial presystolic volume (fiber length) are formed.

Thus, the cardiomanometric method of phase analysis allows complete differentiation of the cardiac cycle into periods and phases to be performed. However, the usage of this method in the practice of sports medicine is quite difficult, since it requires catheterization of the heart.

The polycardiographic method of Blumberger (1942) has become widely used /103
in medical testing and in clinical practice. This method is physiologically well founded, precise, completely safe and causes no discomfort to the test subject. Recording of phases according to Blumberger requires synchronous recording of the electrocardiogram, phonocardiogram and central pulse curve (Figure 13). Probably the only defect of the method is the fact that the diastolic phases cannot be differentiated by this method.

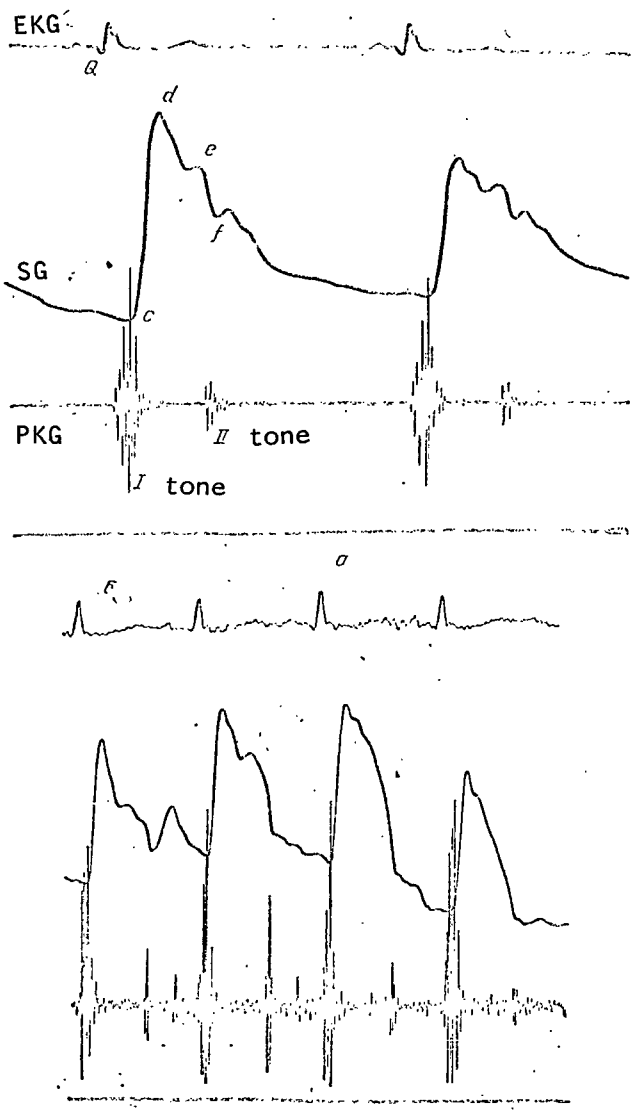


Figure 13. Synchronous Recording of Electrocardiogram, Phonocardiogram and Carotid Artery Sphygmogram Used to Calculate Length of Cardiac Cycle Phases. a, Normal polycardiogram of healthy person (athlete); b, Altered polycardiogram during muscular work. Chart drive speed on Figures a and b identical. Explanation in text.

When an electrocardiogram is recorded, an effort must be made to produce a clear Q wave.

Under conditions of athletic activity, it is expedient to record electrocardiograms using the leads described by Neb (1938) or according to the method of L. A. Butchenko (1963).

The recording of phonocardiograms should be performed using the middle frequency characteristics of Maass and Weber. In a special investigation, Holldack (1951) showed that for qualitative phase analysis, recording of the phonocardiogram should be performed using amplifiers with a "dip" in the frequency characteristic below 150 Hz.

In sports medicine practice, the central sphygmogram should be recorded from the level of the carotid artery.

Yu. K. Shkhvatsabaya (1963) developed a technique for remote recording of polycardiograms. Figure 14 shows the location of pickups and the fastening system allowing the electrocardiogram, phonocardiogram and sphygmogram to be recorded during muscular work.

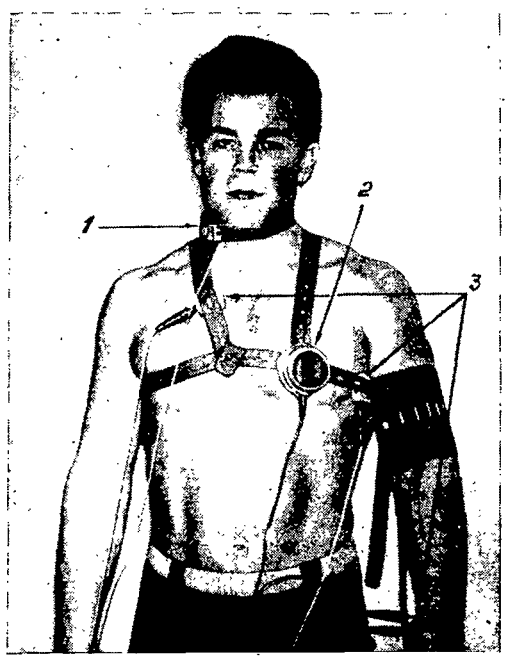


Figure 14. Fastening of Pickups for Recording of Polycardiogram During Muscular Work. 1, Pulse pickup; 2, Microphone; 3, Electrocardiographic electrodes

In order to perform phase analysis, the following characteristic points are distinguished: on the electrocardiogram -- the beginning of the Q wave; on the phonocardiogram -- the beginning of the high-frequency oscillations of tone I and the beginning of tone II; on the sphygmogram -- the beginning of the anacrotic elevation (c), the beginning of the incisure (e) and its lowest point (f) (see Figure 13). Calculation of the phases is performed as follows:

Asynchronous contraction (AC) = Q -- I tone;

Isometric contraction (IC) = (I tone -- II tone) -- (c -- f);

Stress period (T) = AC + IC; period of expulsion (E) = c -- e;

Mechanical systole (S_m) = IC + E;

General systole (S_0) = T + E;

Protodiastole (P) = e -- f.

It must be kept in mind that the division of the period of expulsion into phases of maximum and reduced expulsion using the sphygmographic technique is impossible; therefore, sphygmography allows us to judge only concerning the total duration of the period of expulsion. When it is difficult to find point e (see Figure 13) the end of the period of expulsion is determined after N. N. Savitskiy (1956) by drawing a tangent to the descending portion of the incisure. Certain authors (Blumberger, 1942; etc.) calculate the duration of the period of expulsion from the c -- f interval. This point of view is doubtless improper, since relaxation of the myocardium (its diastolic state) begins before point f, namely at point e.

Phase analysis calls not only for calculation of the duration of the phases of the cardiac cycle, but also for determination of certain special indicators characterizing the dynamics of cardiac contractions. These include primarily the two indicators of intrasystolic phase relationships. These are the intrasystolic indicator and the myocardial stress index (V. L. Karpman, 1965).

The intrasystolic index (ISI) is the percent ratio of the duration of the period of expulsion of blood from the heart to the length of the mechanical systole. It characterizes the effectiveness of the time of the mechanical systole in the ventricles.

The myocardial stress index (MSI) is the ratio of the time of stress to the duration of the general systole (in percent). This index gives us an idea of the time of the cardiac contraction expended in preparation for expulsion of blood.

Muller (1941), L. A. Leshchenskiy (1957), I. N. Bronovets (1963) and certain other authors have suggested several other indicators for the relationships between phases.

An objective evaluation of the state of the contractive capacity of the myocardium is possible also using indicators characterizing the relationship of the principal parameters of the cardiac contraction: the volume, pressure and duration of the phases in the cardiac cycle. We can include the rate of /105
increase of intraventricular pressure (V_i) and the rate of expulsion of blood from the heart (V_e).

Investigation of the first derivative of pressure within the heart and within the vessels with respect to time, i.e. calculation of the rate of the change in pressure during various phases of the cardiac cycle has recently become widespread. This problem is solved either using special differentiating devices or analytically. Since the most valuable information concerning

the contractability of the myocardium can be produced by calculating V_i during the phase of isometric contraction, V. L. Karpman (1961) suggested that this quantity be calculated from the following relationship:

$$V_i = \frac{P_d - 5}{IC}, \tag{31}$$

where P_d is the diastolic arterial pressure, IC is the duration of the phase of isometric contraction, 5 is an arbitrary value for the final diastolic pressure in the left ventricle.

M. N. Syvorotkin (1964) recently once more demonstrated the importance of investigating the mean rate of expulsion of blood from the heart, calculated from the following relationship:

$$V_e = \frac{VB}{E}, \tag{32}$$

where VB is the blood volume per beat, E is the length of the expulsion period.

Useful physiological information can also be produced using the time of expulsion of one minute's blood volume. This index is calculated by multiplying the duration of the period of expulsion by the pulse frequency in beats per minute.

Normal standards of duration of phases. Table 6 shows data on the mean duration of the principal phases of the cycle of contraction of the left ventricle in athletes and in practically healthy persons not involved in sports.

The duration of the phases of the cardiac cycle observed under normal conditions is determined primarily by the contractability of the myocardium. In addition to this, it depends on a number of factors, including the cardiac rhythm, cardiac output and arterial pressure.

The cardiac rhythm is one of the most important determining factors influencing the duration of a number of phases of the cardiac cycle. As the frequency of heart contractions changes, the duration of the expulsion period, the mechanical systole, diastasis and the filling period change. The remaining phases of the cardiac cycle depend little or not at all on the pulse frequency.

Consideration of the interrelationships between phase duration and cardiac rhythm has important practical significance, allowing individual /106

specifics of the phase structure arising in changes of the pulse frequency to be evened out.

TABLE 6. MEAN DURATION OF PHASES OF CARDIAC CONTRACTION (IN SEC) AND CERTAIN INDICATORS OF CARDIODYNAMICS IN HEALTHY PERSONS NOT INVOLVED IN SPORTS AND IN ATHLETES

Names of phases and indicators	Athletes	Non-athletes
Cardiac cycle	1,00	0,864
Phase of asynchronous contraction	0,065±0,001	0,051±0,001
Isometric contraction phase	0,049±0,002	0,031±0,002
Stress phase	0,114±0,002	0,082±0,002
Expulsion phase	0,261 (0,268)	0,265±0,005
Mechanical systole	0,309 (0,300)	0,297±0,005
General systole	0,374	0,348
ISI	84,5	89,2
MSI	30,4	23,5
Rate of increase of intraventricular pressure (mm Hg/sec)		
(expulsion time of 1 min volume (sec)	1220 15,6	2005 18,3

Note. Parentheses enclose values calculated for given rhythm from formulas (33) and (44).

The dependence of the time of blood expulsion from the ventricles (E), the mechanical (S_m) and general (S_0) systole on the duration of the cardiac cycle (C) can be described by the following equations (V. L. Karpman, 1963):

$$E = 0{,}268 \cdot C^{0{,}36} = 0{,}109 \cdot C + 0{,}159, \tag{33}$$

$$S_m = 0{,}3 \cdot C^{0{,}36} = 0{,}114 \cdot C + 0{,}185, \tag{34}$$

$$S_o = 0{,}12 \cdot C + 0{,}235. \tag{35}$$

We note that all of these formulas are suitable for analysis of cardiac activity at rest.

Gobbato and Meda (1956) were among the first to show the dependence of the period of expulsion on the value of cardiac output. This fact has been confirmed many times since. Weissler et al. (1961) concluded an equation describing the interrelationship between the period of expulsion, the value of cardiac output and cardiac rhythm. /107

Under physiological rest conditions, the value of arterial pressure in healthy persons has no essential influence on the duration of phases of the systole. However, under physiological excitation, when the arterial pressure is sharply changed, the phase structure takes on new features. Thus, as the diastolic arterial pressure is decreased, the duration of the phase of isometric contraction is reduced, while as the pressure is increased, on the other hand, it is lengthened.

Special investigations of phase duration in the cardiac cycle for athletes were first performed by Reindell and Klepzig (1949). Subsequently, in investigations of highly trained athletes, Emmrich et al. (1956) turned attention to the elongation of the stress period. Mellerowicz (1956) found that in athletes the mean duration of the stress period was 0.115 sec, with individual variations from 0.09 to 0.16 sec. We performed special investigations in which cardiodynamics data from 246 athletes were compared with data from 107 practically healthy persons not involved in sports. These data are presented in Table 6.

In the athletes, the duration of asynchronous contraction has practically the same range of fluctuations as in the untrained persons. However, in a significant number of the observations the duration of this phase in the athletes is at the upper range of the norm. It is in connection with this that the relative elongation of asynchronous contraction is observed on the basis of average data presented in Table 6. The relative elongation of asynchronous contraction in athletes was also observed by Emmrich et al. (1956).

The duration of isometric contraction in athletes fluctuated from 0.02 to 0.11 sec. Here, in approximately 30% of the highly trained athletes the duration of this phase exceeded the upper boundary of the norm. V_i in most athletes was found to be relatively low.

The duration of the stress period in most athletes was extended, by elongation both of the asynchronous and of the isometric contraction. The duration of the period of expulsion in athletes was either normal or relatively decreased. In contrast to the data presented by Mellerowicz (1956), we observed no relative elong-ation of the period of expulsion in athletes.

In highly trained athletes in a certain number of the observations, relative elongation of the mechanical systole is observed in relation to the expected values. The same thing can be said concerning the duration of the general systole.

Figure 15. Performance of Cardiodynamics Investigations for Athletes During Work on the Bicycle Ergometer (Laboratory of Sports Cardiology of State Central Order of Lenin Institute of Physical Culture)

The intrasystolic index (ISI) of athletes is most frequently decreased. Thus, according to average data, it amounts to 84.5%, whereas in a control group it was 89.1%.

The myocardial stress index in athletes, on the other hand, is most frequently higher. Thus, according to average data, in athletes this indicator was equal to 30.4%, while for untrained persons it was 23.5%.

Summing up all of the above, we can conclude that the characteristics of the "sports heart" can be supplemented by data of phase analysis: in athletes we most frequently see predomination of high values of duration of the stress period and its component phases, a decrease in intrasystolic index, V_i and an increase in the myocardial stress index. This combination of phase shifts is characteristic for the phase syndrome of myocardial hypodynamia (V. L. Karpman, 1965).

Phase analysis of cardiac activity in athletes during muscular work and recovery period. At the present time, the problem of recording the curves required for phase analysis under laboratory conditions has largely been solved. Figure 15 shows the results of investigation of cardiodynamics in athletes during work on the bicycle ergometer. /109

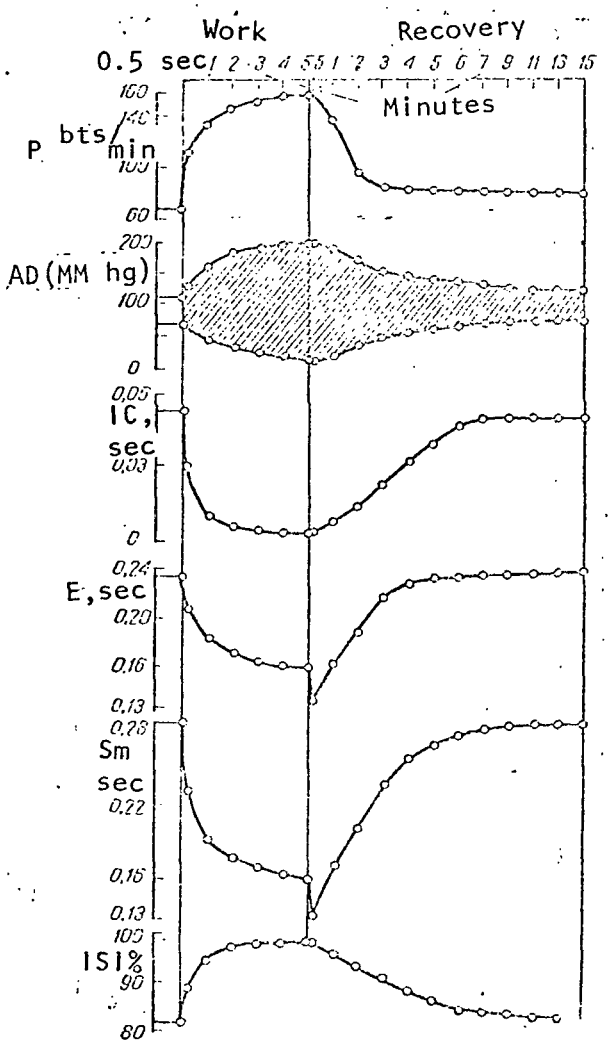


Figure 16. Changes in Principal Parameters of Cardiodynamics During Work and Recovery. P. pulse frequency; AP, arterial pressure; IC, duration of isometric contraction phase; E, duration of espulsion period; Sm, duration of mechanical systole; ISI, intrasystolic

Figure 16 shows data on changes in the duration of cardiac cycle phases in a group of athletes performing work on bicycle ergometer equal to 7500 kg-m (at a constant rate of 245 w). As we can see from this figure, the cardiac rhythm of the athletes investigated increased to 157 beats per minute on the average. Regular changes were noted also in an investigation of the principal phases of the cardiac cycle. Thus, the duration of isometric contraction was sharply decreased, becoming in many cases equal to zero. The duration of the period of expulsion, the mechanical and general systoles also decreased. Asynchronous contraction remained unchanged in a number of observations, and in some cases was shortened. The intrasystolic index increased sharply during muscular work, reaching in some observations 100% (in those cases when the phase of isometric contraction was equal to zero).

The rate of intraventricular pressure increase increases with work.

The time for expulsion of one minute's volume during the process of physical loading increases by approximately 1-1/2 times, while the rate of emptying /110 of the ventricles increases by 3-4 times, reaching 1000-1125 mℓ/sec.

These phase shifts, characteristic for the syndrome of myocardial hyperdynamia (V. L. Karpman, 1965), arise under the influence of the positive inotropic and chronotropic effects.

Analysis of changes in cardiodynamics during performance of muscular work can be used to evaluate the quality of regulation of the circulatory apparatus in athletes.

If the cardiovascular system were regulated "ideally," changes in the duration of the isometric and isotonic contractions of the heart would have a

stepped nature during stepped loading. However, actually, as we can see from Figure 16, the transient processes for these parameters for work at a rate of 245 w are exponential in form, which indicates some inertia in the operation of the system controlling the circulatory apparatus. These exponents, as was shown above, can be described mathematically (see equations (7) and (8)). Both of these equations allow us to predict the extent to which the phase of isometric contraction and the period of expulsion of blood from the heart will be decreased at any moment in time t after the beginning of muscular work.

The changes in phase duration of the cardiac cycle during the recovery period after physical loading have been discussed in the literature. In these works, it has been shown that during recovery a regular lengthening of all phases of the cardiac cycle is observed until they reach their initial values.

The recovery of the length of cardiac cycle phases after cessation of muscular work is nonlinear. A study of the corresponding curves allows the entire recovery period to be divided arbitrarily into three stages: early restitution, restitution itself and late restitution.

Early restitution is observed on the average for 30 sec after muscular work is stopped. The duration of isometric contraction remains unchanged during this period. The duration of the period of expulsion, on the other hand, is sharply decreased (see Figure 16). The mechanism of this secondary "paradoxical" shortening of the period of expulsion is as follows: when the pedaling on the bicycle ergometer is suddenly stopped, the "muscular pump," which greatly aids veinous return of blood to the heart ceases to operate. As a result, the filling of the cavities of the heart with blood is relatively decreased. However, since the power of the cardiac contraction during early restitution continues at the same level as at the end of work (which is indicated by the constancy of the isometric contraction), emptying of the heart cavities is achieved in a shorter time. The presence of this very /111
mechanism of secondary shortening of the expulsion period was detected in a special investigation (Yu. K. Shkhvatsabaya, 1964), in which early restitution was studied with the test subject in a horizontal position, when the stoppage of the work of the "muscular pump" has less influence on veinous blood return to the heart. As we can see on Figure 17, when the test subject is in the horizontal position, the secondary shortening of the expulsion period is not noted.

Formation of the Hegglin phenomenon is characteristic in a large number of cases during the first stage of recovery. This time represents a condition in which the II tone on the phonocardiogram precedes the completion of the T wave on the electrocardiogram by 0.04 sec or more. Hegglin (1947) considers that essential distortions of the interrelationship between the electrical and "mechanical" systoles indicate weakness of the cardiac contraction. However, observations of the formation of this electromechanical phenomenon under conditions of muscular work do not confirm the concepts of Hegglin (1947). As follows from our data, as well as from the data of K. M. Smirnov and

N. V. Zaytseva (1964), the Hegglin phenomenon under early recovery conditions indicates myocardial hyperdynamia rather than weakness of the myocardium.

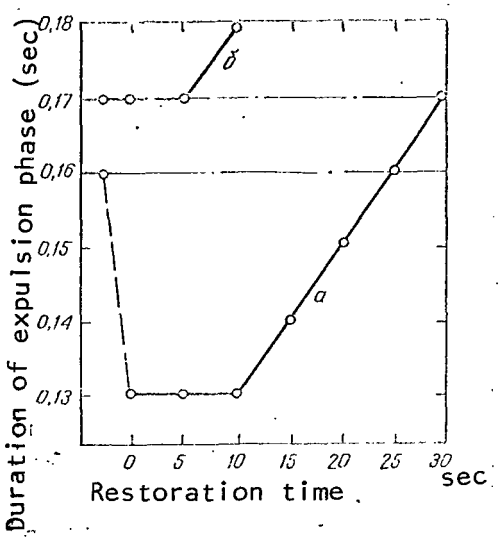


Figure 17. Physiological Analysis of Secondary Shortening of Duration of Expulsion Period After Muscular Work. a, Change in duration of expulsion period with body in vertical position; b, In horizontal position. Analysis in text.

The second stage of the recovery period is characterized by progressive lengthening of all phases of the cardiac cycle. As we can see on Figure 16, the lengthening of the isometric contraction is achieved more slowly than the lengthening of the expulsion period.

The third, last stage in recovery is characterized by stabilization of the structure of the cardiac contraction (see Figure 16). This stabilization begins for the phase of isometric contraction 8-10 minutes, and for the expulsion period 5-6 minutes after completion of intensive muscular work. By 15-20 minutes, complete recovery of the lengths of phases to their initial balues at rest is generally achieved.

Phases of the cardiac contraction /112
during certain functional experiments. A change in the position of the human body relative to the plane of rest is an element of many sports exercises. This type of perturbation has long been used to evaluate the functional state of the cardiovascular system (orthostatic test).

A change in the position of the human body causes redistribution of the mass of the blood in the organism so that a certain portion of blood is briefly pooled either in the lower portion of the trunk (during standing) or in its upper portion (during transition from the horizontal position to the head-downward position). As a result of this, the volume of veinous return to the heart changes: in the first case, it decreases, while in the second case, on the other hand, it increases. Thus, orthostatic disturbances, as applicable to the work of the heart, consist of an increase or decrease in blood filling of heart cavities.

In order to study cardiodynamics under conditions of orthostatic disturbance, it is most expedient to use the tilt table, with which passive changes of the position of the test subject's body can be made. Figure 18 shows the changes in the duration of ventricular systolic phases during rotations of the body from the horizontal position to the head-up or head-down position. As the body is rotated to the head-up position, in athletes a lengthening of the /113
isometric contraction and a shortening of the expulsion period are regularly

observed, i.e. the phase syndrome of myocardial hypodynamia is briefly noted. This hypodynamia is functional in nature, since it results from the decrease in veinous return of blood to the heart.

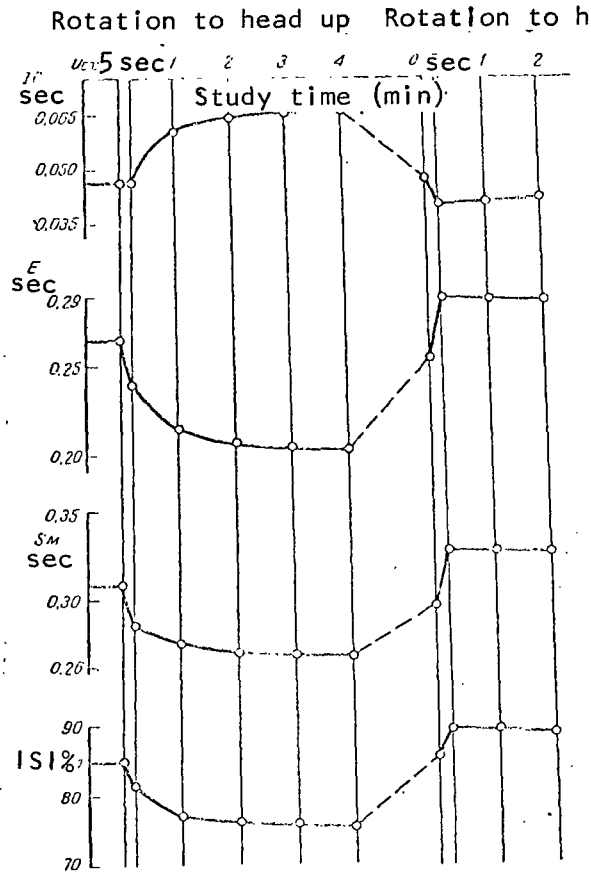


Figure 18. Cardiodynamics Under Conditions of Orthostatic Excitation. Symbols same as on Figure 16.

When the body is rotated to head down, the dynamics of the heart are opposite to those just described (see Figure 18). Analysis of the phase shifts arising in this case shows that the phase syndrome of volume loading arises (V. L. Karpman, 1965).

When nervous regulation of the circulatory apparatus is disrupted and in certain pathological states, changes in the phase duration of the cardiac cycle under the influence of orthostatic disturbances are no longer aperiodic as seen on Figure 18, but rather oscillatory in nature. Consequently, orthostatic tests can be used to evaluate the quality of regulation of the cardiovascular system.

The Burger modification of the Valsalva experiment was earlier broadly used to study the changes in cardiac rhythm and arterial pressure. A. I. Gefter et al. (1964), V. L. Karpman et al. (1965) and others changes in the duration of cardiac cycle phases under the influence of the Valsalva experiment. Due to straining which occurs in the Valsalva-Bruger experiment, the intrathoracic and intraalveolar pressure increase sharply (up to 40 mm Hg). As a result of this, blood flow through the pulmonary capillaries decreases and a deficit in blood filling of the left ventricle arises: the heart begins to work under conditions of volumetric deficiency. The Valsalva-Bruger experiment causes the phase syndrome of myocardial hypodynamia (Figure 19).

The combined three-moment functional test is broadly used in sports medical practice. As we know, as this test is performed the test of the reaction of the cardiovascular system to loading is performed on the basis of data concerning the change in the nature of restoration of the pulse /114

frequency and arterial pressure after standard loading. It seems promising to use phase analysis when performing the S. P. Letunov experiment. Figure 20 shows the changes in duration of principal systolic phases arising under the influence of each component of the experiment. It should be noted that the first experiment using this type of investigation showed that the nature of recovery, according to pulse and arterial pressure data on the one hand and cardiac cycle phase length on the other hand, in many cases was found to be different. This type of divergence apparently allows us to deepen our information produced in this experiment.

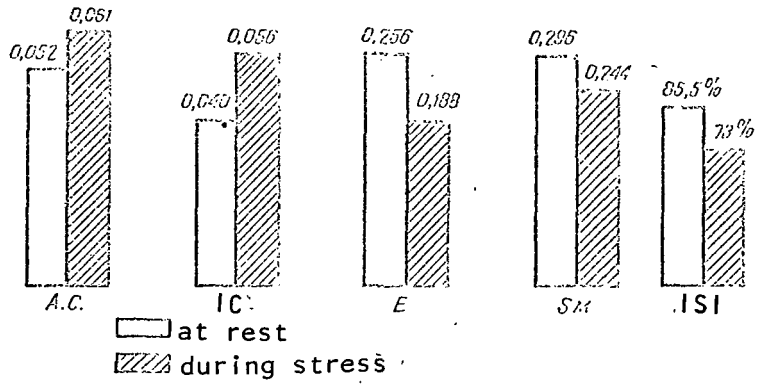


Figure 19. Changes in Duration of Phases of Cardiac Cycle During Valsalva-Burger Test. AC, duration of asynchronous contraction phase. Remaining symbols same as on Figure 16.

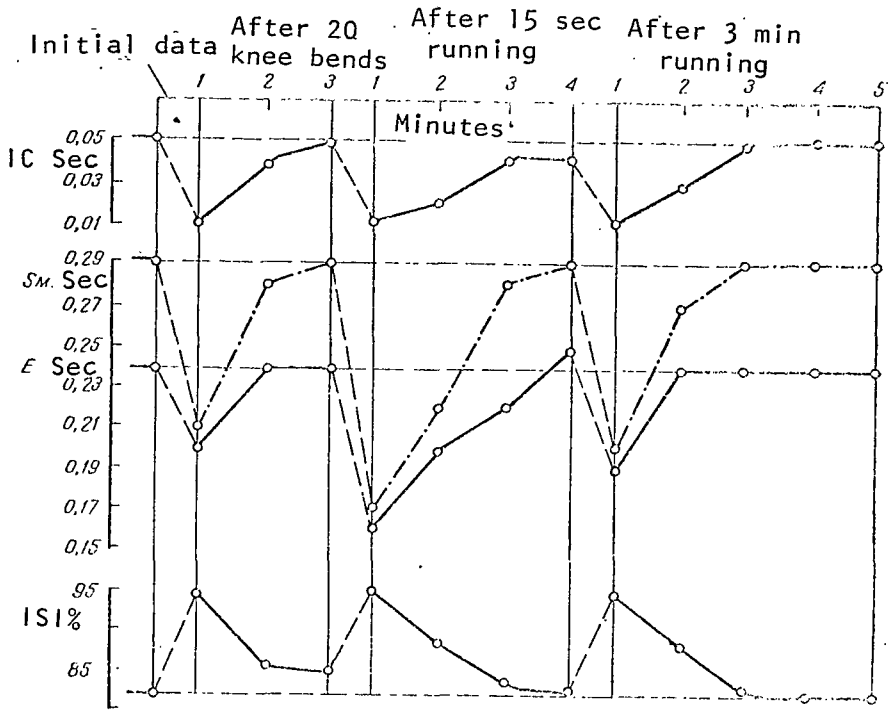


Figure 20. Cardiodynamics During Performance of S. P. Letunov Test. Symbols same as on Figure 16.

CHAPTER V

ELECTRICAL MANIFESTATIONS OF CARDIAC ACTIVITY

ABSTRACT. The methods of recording and analysis of the electrical activity of the heart are analyzed. Included are classical methods of electrocardiography, as well as recent modifications and improvements, ranging from the application of new leads to improved methods of analysis, both statistical and graphical, of raw electrical data. Case histories are presented to illustrate characteristics and appearance of electrocardiograms, vector cardiograms, etc. in various types of athletes with and without pathological heart problems.

The investigation of the electrical activity of the heart continues to /115
remain one of the principal and most widespread methods of objective evaluation of the functional state of the myocardium in sports medicine. It should be noted that the electrocardiograms of athletes differ in certain ways from curves recorded for healthy persons not occupied with sports. In connection with this, ordinary clinical guides for electrocardiography are insufficient for the sports doctor. The purpose of this chapter is to fill this gap. It presents data on scalar and vector electrocardiography of athletes, based on the broad personal experience of the authors.

Electrocardiography

An evaluation of the functional state of the heart of an athlete and determination of changes arising during the process of sports exercises cannot be done without using the electrocardiographic method of investigation. Electrocardiography also has great significance for early determination of pathological and prepathological changes in the "sports heart."

Methods of electrocardiographic investigation of athletes. Modern sports training places high demands on the organism, including the cardiovascular system of athletes. In connection with this, the volume and methodology of electrocardiographic investigation of athletes should be sufficient for early /116
discovery both of physiological and of pathological changes in the heart. This demand is satisfied, as our investigations have shown, by obligatory recording of the electrocardiogram at rest and after muscular work, and in many cases directly during muscular activity (L. A. Butchenko, 1963). At

rest, standard leads I, II and III should be recorded, standard lead III should be recorded with deep inhalation, as well as leads aVL, aVR, aVF, reinforced monopolar leads from the extremities and monopolar thoracic leads from V_1 to V_6. For a deeper study of changes on the electrocardiogram of athletes, monopolar precordial leads V_{3R}, V_7 and bipolar precordial leads D, A and J as suggested by Nehb (1938) should be recorded. The recording of all these leads in athletes does not differ from their recording in normal clinical practice.

The muscular activity used during electrocardiographic investigation of athletes includes measured loads under laboratory conditions (50 deep knee bends, work on the ergometer, 2-3 minutes running in place, lifting of weights, swimming for 25-50 m, etc.) as well as nondosed loads (training and competition).

Electrocardiographic investigation after physical loading should consider the specifics of the recovery period. The problem is that recovery after loads usually occurs irregularly (Hill, 1927; Ya. A. Egulinskiy, 1938). Thus, after 2-3 minutes running in place, restoration of the pulse frequency, the duration of the P-Q interval, the position of the S-T and P-Q segments, the direction of the electrical axis of the P and T waves and the QRS complex occurs considerably more rapidly during the first two minutes and more slowly in the subsequent 2-3 minutes of rest (L. A. Butchenko, 1958).

Training and particularly competition, due to its considerable intensity and in many cases its great duration, cause considerably greater functional changes in the organism than measured loads. This means that the recovery period after these activities is greatly lengthened. In connection with this, for a more complete evaluation of the effects on the heart of physical loads, the electrocardiogram should be recorded for five minutes after 2-3 minutes running in place, while after training and competition it should be recorded for at least 15 minutes of recovery. The electrocardiogram should be recorded more frequently in the first few minutes after loading and less frequently in subsequent minutes. Using a single-channel electrocardiograph, it is impossible to record a large number of leads immediately after loading. Therefore, we might limit ourselves to recording of only two (V_2 and V_5) monopolar precordial leads.

The V_2 and V_5 leads characterize the electrical activity of the "right" /117
and "left" heart to a great extent, since the difference electrode in the first case is turned toward the epicardial surface of the right ventricle, and in the second case -- toward the epicardial surface of the left ventricle. The usage of leads V_3 and V_4 is less suitable, since they reflect potential of the transition zone between the epicardial surface of the right and left ventricles. In those rare cases when leads V_2 and V_5 also reflect the

potentials of the transition zone between the epicardial surface of the right and left ventricles, leads V_1 and V_6 should be recorded after physical loading.

Of course, when multichannel electrocardiographs are used, the volume of electrocardiographic investigation after physical loads should be expanded both by increasing the number of monopolar precordial leads, and by connecting reinforced monopolar leads from the extremities.

Electrocardiographic investigation at rest and during the recovery period after various loads can yield only approximate information concerning the functional changes and bioenergetic processes in the myocardium which occur during work. In many cases, it is also insufficient for early determination of prepathological and pathological changes in the heart. Therefore, electrocardiographic investigation during muscular activity is of essential significance.

An electrocardiogram not distorted by muscular currents can be recorded during muscular activity using leads H_1, H_2 and H_3 (L. A. Butchenko, 1955, 1963, 1966). Lead H_1 records the difference in potentials between points V_4 and V'_1. Lead H_2 records the potential difference between points V_2 and V'_1, while lead H_3 records the difference between points V_4 and V_{4R}. Leads H_1 and H_3 produce electrocardiograms identical in form but differing in amplitude. Leads H_1 and H_3 have greater diagnostic value than lead H_2. This is explained by the fact that leads H_1 and H_3 record the potential difference between the left (V_4) and right (V_1 and V_{4R}) ventricles of the heart, whereas lead H_2 records the potential difference between two sectors (V_2 and V'_1) of the right ventricle.

Therefore, when single-channel electrocardiography is performed, leads H_1 and H_3 should be recorded, not H_2. It should be considered that lead H_1 gives good curves during running, knee bends and bicycle ergometer work. Lead H_3 also gives good curves during work with the arms and trunk (hand ergometer, lifting of weights).

When an electrocardiogram is recorded during muscular work, it is also necessary to use special chest electrodes and electrode paste[1]. /118

[1] The methodology of electrocardiographic investigation during muscular work has been presented in detail in the book of L. A. Butchenko *Elektrokardiografiya v Sportivnoy Meditsine* [Electrocardiography in Sports Medicine], Medgiz. Press, 1963.

If the loading is brief (20-30 sec), the recording of the electrocardiogram should be continuous during performance of the work. When loads 2-5 minutes in length are performed, recording of the electrocardiogram should be performed each 30 sec. If the load continues 30 min and more, the electrocardiogram should be recorded each 3-5 min.

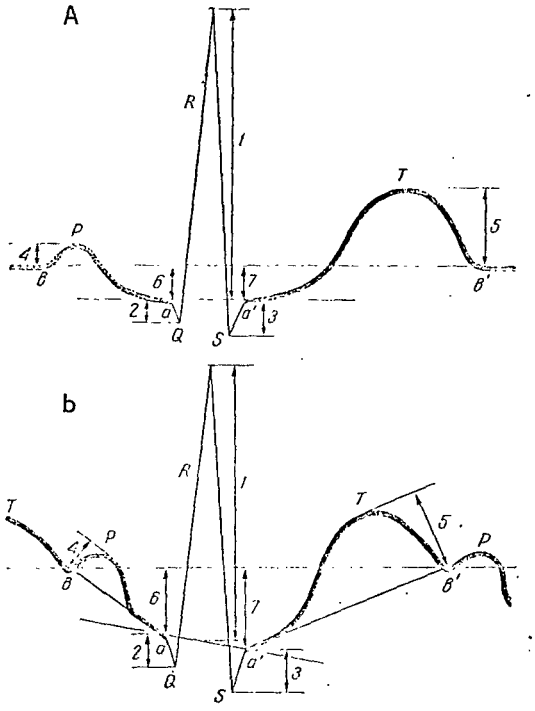


Figure 21. Method of Measuring Elements of Electrocardiogram. A, when T-P segment or isoline is present; b, when T-P segment is absent and descending branch of P wave and ascending branch of T wave are decreased; BB', isoline; aa', line connecting beginning of Q wave and end of S wave; 1, Amplitude of R wave; 2, Amplitude of Q wave; 3, Amplitude of S wave; 4, Amplitude of P wave; 5, Amplitude of T wave; 6, Decrease in segment P-Q relative to isoline; 7, Decrease in S-T segment relative to isoline

The decoding and analysis of electrocardiograms taken at rest after and during the process of performance of various loads is performed according to the general methodology. However, with great tachycardia resulting from muscular activity, due to /119 the merging of the P and T waves the T-P segment is frequently absent, as is the isoelectric line, and we frequently observe a lowering of the P-Q or S-T segments, the QRS complex, the descending branch of the P wave and the ascending branch of the T wave (Figure 21). These changes during clear tachycardia result from the degree of displacement of the P-T_a interval, i.e. the interval from the beginning of the P wave to the end of the auricular T wave. In connection with this, the measurement of the amplitude of the P, T, Q, R and S waves by the ordinary method, i.e. from the isoelectric line, cannot be performed. Figure 21 shows a method of measuring elements of the electrocardiogram considering the influence of the P-T_a interval on them (Bruschke, Butchenko, 1965).

An estimate of the proper length of the electrical systole or the Q-T interval in athletes has its specific problems. The thing is that the empirical formulas suggested by Bazett (1920), Fridericia (1920), Schlomka and Raab (1936), Hegglin and Holzmann (1937), Aschman (1942) and others for the estimation of the proper duration

of the electrical systole of the heart in persons not occupied in sports cannot be used for athletes. This is explained by the fact that they do not consider the elongation of the electrical systole of the heart at rest which is characteristic for athletes (L. A. Butchenko and N. I. Volnov, 1963). With expressed tachycardia resulting from muscular activity, the empirical formulas suggested for estimation of the length of the electrical systole at normal pulse frequency at rest cannot be used not only for athletes, but also for those not occupied in sports. In connection with this, we, together with G. Bruschke and H. Burger (1965), developed norms for proper electrical systole in athletes, not using empirical formulas, but using actual data. For this, we studied electrocardiograms taken at rest and after muscular activity of varying intensities for 2402 (1769 men and 633 women) well trained athletes of high caliber: 1754 masters of sports and first rank sportsmen, 496 second rank sportsmen and 152 third rank sportsmen. Our data were presented in graphs, separately for men and women. Fluctuations from the mean by $\pm 2\sigma$ were considered variants of the norm (Figure 22).

Electrocardiogram of the athlete. The variants of normal electrocardiograms for athletes have been studied insufficiently to date. This frequently leads to the fact that the specific features natural for the electrocardiogram of the athlete are treated as pathological changes, whereas pathological changes themselves are no less frequently attributed to physiological changes occurring in the heart during the process of training.

Variants of electrocardiograms in athletes were investigated for 3180 intensively trained masters of sports, first rank sportsmen and second rank sportsmen (4.7%). Among those investigated were 232 with diseases and deviations from the norm, primarily in the cardiovascular system. The electrocardiograms of 2,652 persons were recorded using the EKP-4 apparatus with linear frequency channel from 3 to 60 Hz, while 528 electrocardiograms were recorded on a "Siemens" apparatus with a linear frequency range in the amplifier channel of from 0.2 to 150 Hz. /121

In athletes, sinus bradycardia is frequently observed, resulting from the increased tonus of the vagus nerve (Herxheimer, 1933; A. N. Krestovnikov, 1951; etc.). Thus, in well trained athletes, according to our data, the pulse frequency averages 56.5 ± 9.2 beats per minute. More sharply expressed sinus bradycardia is observed in those athletes who turn a great deal of attention in their training to the development of endurance. However, even in these athletes the pulse frequency is generally not lower than 40 per minute. Only in 1.6% were pulse frequencies as low as 39-31 per minute observed. White (1942), Kienle (1946), Mellerowicz (1956) believed that this acute sinus bradycardia may be a pathological sign. However, in this case sinus bradycardia, as our data showed, is combined with an expansion of the QRS complex, sharp sinus arhythmia, low voltage and extrasystolia. When these changes are absent, even clearly expressed bradycardia is an indicator of good functional condition of the heart.

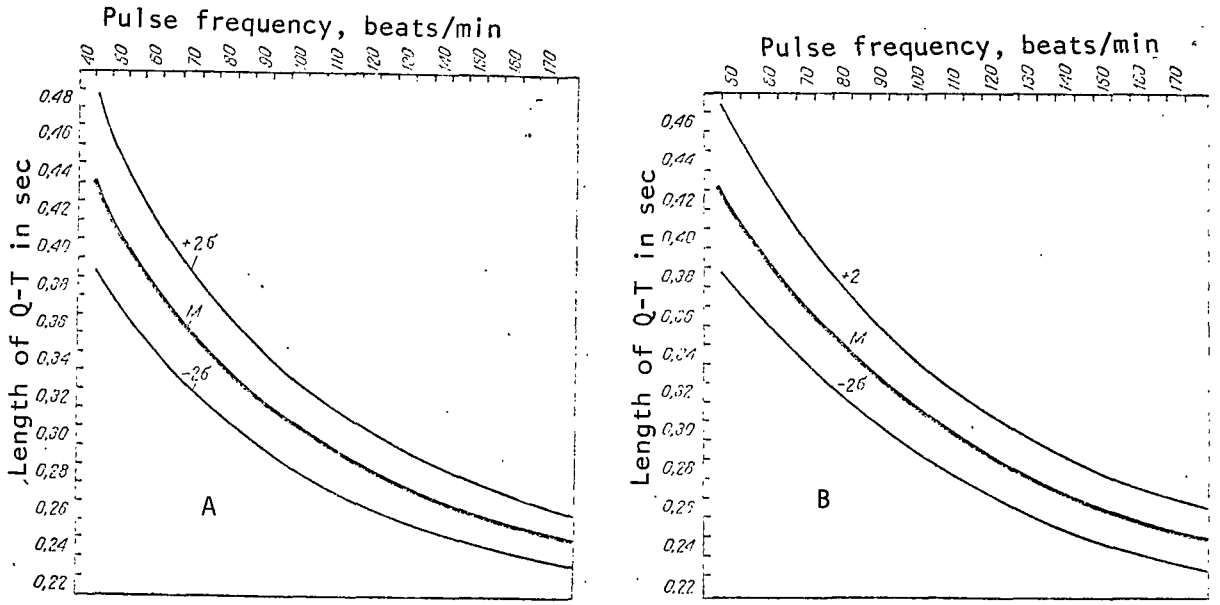


Figure 22. Graphs for Estimation of Duration of Electrical Systole in Athletes at Rest and After Muscular Activity (According to Data of G. Bruschke, L. A. Butchenko and H. Burger, 1965). A, men; B, women.

The question of the clinical significance of sharply expressed sinus bradycardia should be answered individually for each athlete by complex medical examination.

Sinus arhythmia is frequently observed in athletes at rest. According to our data, it is encountered in 60.8% of the athletes. Most frequently moderate sinus arhythmia is observed, the difference in the duration of cardiac cycles varying from 0.10 to 0.15 sec. Less frequently, sharply expressed sinus arhythmia is observed, in which the difference between the cardiac cycles varies from 0.16 to 0.30 sec. Acute sinus arhythmia, with which the difference between cardiac cycles varies from 0.31 to 0.60 sec, was encountered in only 3.6%.

The frequent presence of sinus arhythmia in athletes is explained primarily by the fact that young people predominate in this group, in which this arhythmia results from their age (Schlomka and Reindell, 1936; Jordan, 1955; M. I. Petrenko and R. Ya. Pis'menyy, 1959; R. E. Mazo, 1961). However, this is not the only reason. For example, our data indicate that moderate and expressed sinus arhythmia may arise or be reinforced during the process of sports training in parallel with an increase in the state of training. As concerns acute sinus arhythmia, it is a result of a very high increase in the tonus of the vagus nerve and, as a rule, is not observed in well trained athletes (S. P. Letunov, 1950). Its presence may indicate a disruption of the normal activity of the heart (V. I. Stovbun, 1959). In particular, this may be confirmed by the fact that it is frequently combined with extrasystolia, a lengthening of the P-Q interval, sharp sinus bradycardia and other changes in the electrocardiogram of pathological significance. It is frequently observed during overtraining and soon after diseases (influenza, angina, infections of the upper respiratory tract). However, in some cases sharp sinus arhythmia may be encountered in well trained athletes. /122

Thus, acute sinus arhythmmia in athletes has various clinical interpretations. It should be interpreted unfavorably when it is combined with changes in the electrocardiogram indicating a sharp increase in the tonus of the vagus nerve (lengthening of the P-Q interval, extrasystolia and acute sinus bradycardia).

In those cases where sharp sinus arhythmia is not combined with changes in the electrocardiogram indicating a great increase in the tonus of the vagus nerve, it may be a variant of the norm. Analysis of clinical and medical-pedagogic data in these cases allows the nature of the problem to be determined.

The direction of the electrical axis of the heart in athletes is related to a certain extent to their sports specialty. Thus, deviation of the electrical axis of the heart to the right is more frequently observed (38-45%) in football players, skiers and swimmers, to the left (23.8-35.2%) in boxers, weight lifters, and long- and middle-distance runners (L. A. Butchenko, 1963). Regardless of the type of sports, the normal position of the electrical axis of the heart predominates in athletes. Thus, the α angle of the QRS complex

in 50.4% of the cases is located between +30 and +70°. In 31.6% of the cases, moderate (to +90°) or clearly expressed (up to +120°) deviation of the electrical axis of the heart to the right is observed. In 18% of the cases, the electrical axis of the heart is deflected to the left. This is indicated by the α angle of the QRS complex, which is generally located between +29 and zero degrees, less frequently between zero and -30 degrees.

The direction of the electrical axis of the heart is determined by hypertrophy and dilation of the ventricles, disruption of the intraventricular conductivity and by the position of the heart in the rib cage.

The position of the heart in the rib cage depends to a great extent on the type of physique and age. It has been found that young persons and persons with asthenic build characteristically show the electrical axis of the heart deflected to the right, while older persons and persons with hypersthenic build characteristically show deflection to the left. /123

One-time determination of the direction of the electrical axis of the heart in athletes is not of great significance for an evaluation of the effects of sports on the heart. For this purpose, the position of the electrical axis of the heart in athletes must be determined during the course of sports training.

The duration of the auricular-ventricular conductivity (P-Q interval) in athletes generally varies between 0.12 and 0.21 sec. The upper boundary of the norm of duration of the P-Q interval is most frequently encountered during bradycardia and under these conditions is an expression of the increase in the tonus of the vagus nerve (T. A. Davydova, 1954; Lepeschkin, 1957; Zuckermann, 1957). Elongation of the P-Q interval to more than 0.21 sec is encountered in athletes in 2.2% of cases (S. P. Letunov, 1950; L. A. Butchenko, 1963). It also results most frequently from an increase in the tonus of the vagus nerve and is a variant of the norm (Herzum, 1940; Schoppe, 1940; O. V. Kacharovskaya, 1948; Reindell, 1949; S. P. Letunov, 1950, 1957; N. B. Tambian, 1956; Mellerowicz, 1956; etc.). Our data indicate that elongation of the P-Q interval after physical loading or the injection of atropine may be leveled and its duration shortened to the norm. In some cases the lengthening of the P-Q interval may indicate changes in the myocardium (Doliopoulos, Bangou, 1953; Becner, Winsor, 1954).

In these cases after physical loading the injection of atropine generally does not cause the length of the P-Q interval to shorten, a slight shortening being observed in a few cases with subsequent lengthening during the recovery period to more than the initial length.

The duration of the QRS complex (intraventricular conductivity) in athletes, as in healthy persons not occupied with sports, generally varies between 0.05 and 0.10 sec. In 4.05% of cases, an increase in the duration of the QRS complex to over 0.10 sec has been observed (L. A. Butchenko, 1963). It is frequently combined with other changes in the electrocardiogram: with

low voltage, negative T_3 wave with deflection of the electrical axis of the heart to the right, with acute sinus bradycardia, extrasystolia and partial or complete block of the right branch of the bundle of His.

Our data show that in athletes the expansion of the QRS complex to over 0.11 sec, combined with block of the right branch of the bundle of His (partial or complete) or other pathological changes in the electrocardiogram should be evaluated as an indication of damage to the myocardium. Clinical investigation in most of these cases generally allows the reason for damage to the myocardium, usually not related with sports, to be determined.

An isolated increase in the duration of the QRS complex in athletes may indicate physiological hypertrophy of the ventricles (Delius, Reindell, 1938; Reindell, 1949). However, in healthy athletes with an increase in the size of one (frequently the left) or both ventricles of the heart, we have never observed an increase in the length of the QRS complex by more than 0.1 sec (L. A. Butchenko, Ya. G. Ablov, G. M. Kukolevskiy). /124

The amplitude and form of the waves are determined to a great extent by the technical capabilities of the apparatus used to perform the investigation. The broader the frequency range of the electrocardiographic amplifier, the more correctly will the amplitude and form of the electrocardiographic waves be recorded. In connection with this, amplitude norms are presented on the basis of investigations performed with the "Siemens" apparatus, which records frequencies from 0.2 to 150 Hz without distortion. In all, 528 athletes were investigated using the "Siemens" apparatus. They did not differ in sex, age or sports qualifications from the entire group of athletes investigated in any essential way.

Using the standard leads, the athletes show low amplitude of the P wave and high amplitude of the R and T waves, frequent (75-55%) displacement of the S-T segment up to 1 mm above the isoline in leads I and II (Figure 23).

Low P waves are characteristic for the electrocardiograms of athletes, apparently resulting from an increase in the tonus of the vagus nerve (Herxheimer, 1933; Klemola et al., 1960; Mellerowicz, 1960).

The genesis of the T wave is closely related to the metabolic processes in the myocardium (M. Ya. Ar'yev et al., 1941, 1947; I. N. Bukhalovskiy, 1951; V. G. Popov, 1953; K. S. Logunova, 1954; etc.). The investigations of Ye. S. Troshanova (1951), N. N. Yakovleva (1955) have shown a considerable increase in myoglobin, inorganic phosphorus, glycogen and activation of the enzyme systems in the cardiac muscle in the process of training. Apparently, it is here that we should seek the reason for the increased amplitude of the T waves in athletes.

Using the III lead in athletes, we frequently encounter negative, two-phase and isoelectric P and T waves. Here, the electrical axis of the heart is located normally or deflected to the left. During deep inhalation, these

waves generally become positive. This indicates that their origin to a considerable extent results from the position of the heart in the rib cage (L. I. Fogel'son, 1957). However, further investigations are required for a final answer to this problem.

In 4.75% of the cases in athletes, the negative, two-phase and isoelectric T_3 waves are encountered with deflection of the electrical axis of the heart to the right. These electrocardiograms in 74.3% of the cases are variants of the norm (L. A. Butchenko, 1963). They result from deflection of the electrical axis of the heart to the right and displacement of the top of the heart forward (Hoskin, Ionescu, 1940) or from specifics of the excitation of the myocardium of the ventricles, manifested in the frequent (55%) presence of partial block of the right branch of the bundle of His in athletes (L. A. Butchenko, 1959). /125

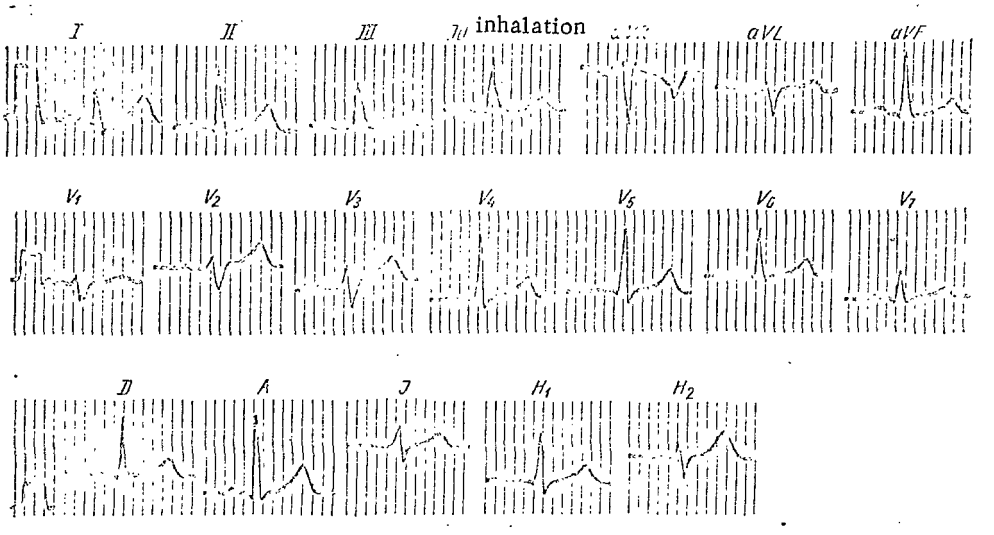


Figure 23. Electrocardiogram of Athlete K, 19 Years Old (Class I Boxer). Monopolar leads from thorax, Nehb lead, H_1 and H_2 leads on electrocardiogram 1, as on others, taken with control millivolt of 5 mm. In order to reduce to standard values, amplitudes in these leads were doubled.

In 25.7% of the observations, electrocardiograms with altered T_3 waves and deflection of the electrical axis of the heart to the right are the result of pathological hypertrophy of the myocardium of the ventricles or inflammatory, ischemic, and sclerotic damage to the myocardium. In these cases, the electrocardiogram also shows characteristics of pathological hypertrophy of the left ventricle, extrasystoles and other rhythmic disruptions,

characteristics of ischemia of the myocardium or focal myocarditis, and an elongated P-Q interval (Figure 24).

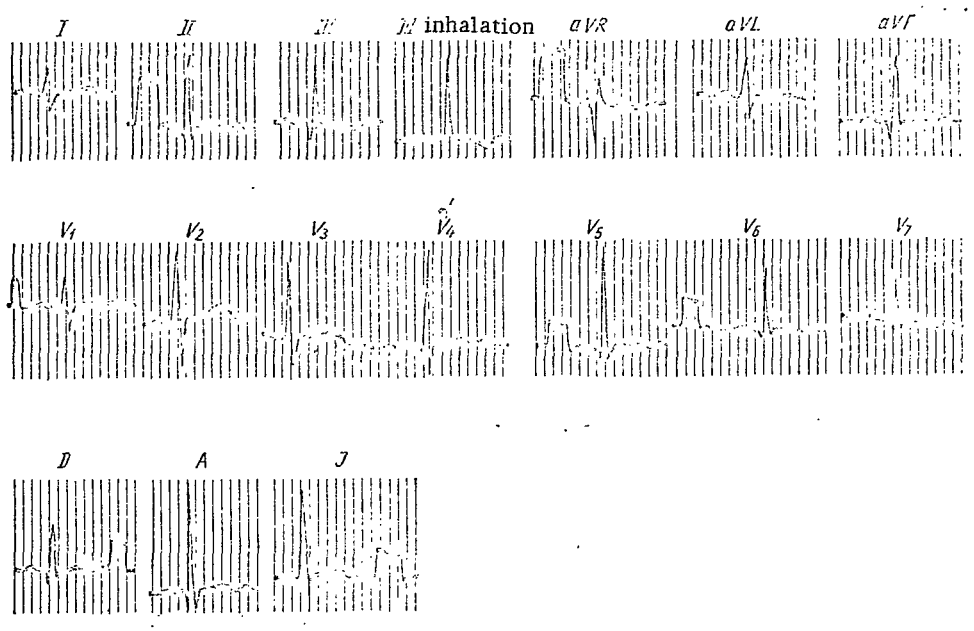


Figure 24. Electrocardiogram of Athlete S, 20 Years Old (Class 1 Fencer). Explanation in text.

The high voltage of the R waves in athletes apparently results from physiological hypertrophy of the myocardium of the ventricles (L. A. Butchenko, 1963).

Athletes may also show low voltage electrocardiograms. As our investiga- /126
tions showed, they result primarily from extracardiac factors and specifics of the position of the heart within the thorax. The extracardiac factors include massive development of the skeletal musculature. A good conductor, this musculature increases the electrical conductivity of the body, causing "shunting" of the action currents of the heart and leading to a decrease in the voltage on the electrocardiogram. The specifics of the position of the heart within the rib cage resulting in a decrease in voltage in the electrocardiogram include deflection of the integral vector in the sagittal plane caused by a displacement of the top of the heart to the rear, as a result of which its projection in the frontal plane is sharply decreased.

In 33% of the cases, the low voltage resulted from the influence of cardiac factors and indicated pathological changes in the heart (myocardiodystrophy, cardiosclerosis). The electrocardiograms in these athletes, in addition to the low voltage, also showed extrasystolic arhythmia, elongation

of the P-Q interval, not disappearing after the injection of atropine or physical exercise, and other pathological changes.

As concerns the displacement of the S-T segment over the isoline, it /127
apparently reflects good blood supply to the myocardium (G. F. Lang, 1936). It is also possible that the lifting of the S-T segment indicates reinforcement of oxidative processes in the myocardium (Wendt, 1946). However, a more probable reason for the displacement of the S-T segment above the isoline is an increase in the tonus of the vagus nerve, characteristic for well trained athletes (Reindell, 1949; Mellerowicz, 1956; Klemola et al., 1960).

The normal electrocardiogram of athletes in monopolar chest leads has its specific features. The P wave in the monopolar chest lead has low amplitude, generally not exceeding 1 mm. In the V_1, V_2 and V_3 leads, it may be two-phased and negative. According to the data of Meyers et al. (1947), Lepeschkin (1957), etc., these changes in the P wave are a variant of the norm.

The waves of the QRS complex and the T wave in monopolar chest leads, as with standard leads, are considerably larger in athletes than in healthy persons not occupied with sports (Table 7).

In the V_1 and V_2 leads, negative and two-phased T waves are frequently encountered, accompanied by a QRS complex of the rS type, which, according to the data of Goldberger (1953), M. B. Tartakovskiy (1958), etc., is a variant of the norm. The S-T segment in monopolar chest leads, as in standard leads, is displaced above the isoline in most athletes (from 43.5 to 96.8%), sometimes up to 3.5 mm. In monopolar chest leads we frequently see sharp (by 8 mm and more) increases in the amplitude of the R wave in the V_4 lead in comparison with the V_3 lead, as well as moderate rotation of the heart in a counterclockwise direction about its longitudinal axis (L. A. Butchenko, 1963). Frequently, athletes recorded with monopolar chest leads show a relationship of the amplitude of the R and S waves exceeding that observed in untrained persons. Thus, the R/S index in the V_6 lead in 14% of the observations exceeds the upper limit of the norm, which is 22. The $S_{v1} + R_{v5}$ index in 31.4% of all investigated athletes exceeds the upper limit of the norm, 35 mm, while the $R_{v1} + S_{v5}$ index exceeds the upper limit of the norm, 10.5 mm, in 20% of all those investigated. These indices were suggested by Sokolow and Lyon (1949) for the diagnosis of pathological hypertrophy of the heart. They have been well studied and are used in clinical practice (Kalliomake et al., 1960; Ionash, 1963; etc.). Komadel and Feriencik (1957), V. V. Bulyychev (1962) also used them to diagnose hypertrophy of the heart in athletes. However, hypertrophy of the heart in athletes differs in principle from pathological hypertrophy. With the physiological hypertrophy of the heart observed in athletes, the increase in the contractive and reserve forces is /129
not accompanied by any disruption in metabolism or blood supply to the myocardium. Metabolic disorders and a worsening of blood supply of the

myocardium are characteristic for pathological hypertrophy. In connection with this, it is hardly proper to use indices suggested for the diagnosis of pathological hypertrophy for the diagnosis of physiological hypertrophy. Also, the investigations of L. A. Butchenko, Ya. G. Ablov and G. M. Kukolevskiy (1966) have shown that the increase in the Sokolow and Lyon indices are encountered in athletes identically frequently both with enlarged heart and with normal size hearts. Therefore, these indices cannot be used for athletes for diagnosis of hypertrophy of the heart. Since the electrocardiographic method as yet has no reliable characteristics for "diagnosis" of physiological hypertrophy of the heart, the increase in the Sokolow and Lyon indices, as well as a sharp increase in the R wave in the V_4 lead in comparison with the V_3 lead and a moderate rotation of the heart in a counterclockwise direction about its longitudinal axis in athletes should be considered variants of the "sports" norm. These changes in the electrocardiogram arise in the process of extended sports training and indicate good functional condition of the heart.

In 10.5% of the observations of athletes, partial block of the right branch of the bundle of His was observed. The duration of the QRS complex did not exceed the upper boundary of the norm in any case, fluctuating between 0.08 and 0.10 sec. This partial block of the right branch of the bundle of His is evaluated by most investigators at the present time as a variant of the norm. However, the possibility cannot be excluded that in some athletes it is a result of hypertrophy of the right ventricle. This is particularly indicated by investigations performed by Zh. A. Teslenko (1964).

The characteristic features of the electrocardiogram with amplified monopolar leads from the extremities in athletes include low-amplitude P wave, high-amplitude R and T waves, and partial presence of vertical and semivertical electrical position of the heart (see Figure 23). Thus, according to our data, vertical and semivertical electrical division of the heart is encountered in 71.2% of the observations, intermediate position -- in 19.3%, horizontal and semihorizontal position in 9.5%. The presence of the vertical and semivertical electrical position of the heart in the overwhelming majority of athletes is apparently explained that in these persons, as in young persons, the heart is located more vertically than in older persons. It is also possible that this is a result of the sports activities, since during extensive training, a change in position of the heart in the rib cage arising as a result of training in connection with the descent /130 of the diaphragm as respiration deepens may become permanent.

The possibility is also not excluded that physiological hypertrophy of the ventricles of the heart and constitutional specifics of the organism are significant in the formation of the electrical position of the heart of athletes.

TABLE 7. AMPLITUDE OF Q, R, S AND T WAVES (IN mm) IN MONOPOLAR CHEST LEADS IN ATHLETES AND HEALTHY PERSONS NOT OCCUPIED IN SPORTS WITH AMPLIFICATION 1 mv = 10 mm

Lead	Groups of persons studied	Q			R			S			T		
		Minimum	Average	Maximum	Minimum	Average	Maximum	Minimum	Average	Maximum	Minimum	Average	Maximum
V_1	Athletes	0	0	0	0,5	4,7	14,0	2,0	11,7	28,0	—3,0	+1,8	+9,0
	Healthy non-athletes	0	0	0	0	3,8	11,8	2,5	11,5	26,2	—2,7	+1,3	+7,6
V_2	Athletes	0	0	0	1,5	8,7	20,0	3,0	15,1	40,3	—1,0	+7,1	+17,0
	Healthy non-athletes	0	0	0	0,4	6,3	15,2	2,4	17,3	39,2	—1,6	+5,0	+12,8
V_3	Athletes	0	0,06	2,0	2,5	13,3	37,0	1,5	11,3	34,0	+1,5	+8,0	+17,2
	Healthy non-athletes	0	0,03	0,4	0,7	10,9	24,1	0,5	12,0	27,5	—0,8	+5,7	+16,2
V_4	Athletes	0	0,75	4,0	4,0	21,3	48,0	0	6,7	24,0	+1,5	+7,6	+17,5
	Healthy non-athletes	0	0,10	2,0	3,8	13,2	34,7	0	7,2	28,8	+0,7	+6,0	+14,2
V_5	Athletes	0	1,34	5,0	5,0	20,6	48,0	0	3,5	14,0	+1,5	+6,4	+17,0
	Healthy non-athletes	0	0,40	3,0	3,0	11,3	28,4	0	2,3	16,1	+0,8	+4,1	+8,5
V_6	Athletes	0	1,48	5,0	6,0	16,0	30,0	0	1,6	10,9	+1,3	+4,9	+16,5
	Healthy non-athletes	0	0,50	2,6	2,2	10,7	22,6	0	1,6	14,3	+0,5	+3,3	+6,9

Note. The data from the investigation of athletes (528 persons) presented by L. A. Butchenko (1963), data for investigation of untrained persons (116 persons) by Waquero et al. (1947).
Tr. Note: Commas indicate decimal points.

In amplified monopolar leads from the extremities, the amplitude and direction of the waves and the form of the electrocardiogram are determined by the electrical position of the heart. In the aVR lead, P and T waves are always negative, the r wave is relatively small and frequently the Q wave or S wave is small. The amplitude of the P wave, as a rule, is low, while the T wave is frequently large.

In the aVL lead, the P and T waves are generally positive. If there is a negative T wave and it is combined with a QRS complex of the rR or QS type, this is a variant of the norm (Goldberger, 1953). The P wave in most athletes is low.

In the aVF lead, negative P wave and T wave may also be encountered. It should be emphasized that the negative T wave is observed only in cases of horizontal electrical position of the heart. In this case, the QRS complex has the rS form which, according to the data of Cazes (1950) and M. B. Tartakovskiy (1958), is a variant of the norm. The R and T waves are frequently large, while the P wave is frequently of low amplitude.

In the Nehb leads and in the H_1, H_2 and H_3 leads in athletes the same changes are observed as in the leads analyzed above, namely: low amplitude of P wave, high amplitude of waves in QRS complex and T wave (see Figure 23).

In the H_1 lead, the electrocardiogram has no essential differences from the electrocardiogram in the A lead after Nehb.

The QRS complex, consisting of the q and R waves, is observed in the H_1 lead when the heart is rotated about its longitudinal axis in the counterclockwise direction. When the heart is rotated about its longitudinal axis in the clockwise direction, the QRS complex in the H_1 lead consists of the R and S waves (A. G. Dembo and L. A. Butchenko, 1958).

In athletes in the H_1 lead, the QRS complex, in contrast to healthy persons not involved in sports, is more frequently represented by the q and R waves and less frequently by all three component waves. Thus, the QRS complex, consisting of the q and R waves in athletes is observed in 29% of the observations, while in healthy persons not involved in sports in only 15%. The QRS complex consisting of all three waves is noted in athletes in 62% of the cases, in nonathletes in 78%. The QRS complex consisting of the R and S waves is encountered identically frequently in athletes and in healthy nonathletes -- in 7% of the cases. The differences in form of the QRS complex /131 in the H_1 lead in athletes and healthy persons not involved in sports can be explained by the fact that in athletes rotation of the heart about the longitudinal axis in the counterclockwise direction is more frequently observed.

In the H_1 and H_3 leads the S-T segment in 56% and in the H_2 lead in 90% of the observations is located above the isoline by up to 1.5 mm.

The electrocardiograms in the H_2 leads in most athletes are reminiscent of the electrocardiogram in right precordial leads. In 96% of those observed, the QRS complex consists of the R wave or the r and S waves. All waves in the QRS complex are expressed when there is a sharp rotation of the heart around its longitudinal axis in the counterclockwise direction (in 4%). In monopolar chest leads in this case, beginning with the V_3 lead, the ventricular complex has the form qRS or qRs, characteristic for potentials of the epicardial surface of the left ventricle.

As examples of normal and pathologically changed electrocardiograms of athletes, we present the following two cases.

1. K, 19 years, I rank boxer. Healthy, well trained. X-ray examination reveals increase in size of left ventricle of heart (see Figure 23). Moderate sinus arhythmia with 65 heart contractions per minute. Difference between maximum and minimum R-R interval 0.15 sec, P-Q = 0.15 sec, QRS = 0.10 sec; Q-T = 0.36 sec (norm 0.362 sec).

Electrical position of heart using Wilson and Goldberger method vertical: QRS complex in aVL lead similar to QRS complex in V_1 and V_2 lead and has form rS, while QRS complex in aVF lead similar to QRS complex in V_5 and V_6 lead and has form qRs. Moderate rotation of the heart about the vertical axis in counterclockwise direction noted: beginning with V_4 lead, QRS complex has form qRs, indicating that difference electrode is turned toward epicardial surface of left ventricle. In V_1 and V_2 leads, initial portion of ventricular complex has form rS, indicating that the thoracic electrode is turned toward the epicardial surface of the right ventricle. In V_3 lead, there is a transient form of QRS complex (RS). In D lead, QRS complex consists of small q wave and high R wave. In A and H_1 leads, QRS complex has form qRs. In J and H_2 leads, QRS complex is similar to QRS complex in right monopolar chest leads and has form rS and RS.

Electrocardiogram (see Figure 23) shows changes characteristic for athletes. In addition to moderate rotation of the heart about its longitudinal axis in the counterclockwise direction, these include: decrease in all leads (with the exception of aVF, D and A) of P wave; high amplitude of T waves in I, II, aVR, V_2-V_5, D, A, H_2 leads; displacement of S-T segment above isoline in I, II, aVL, V_1-V_6, D, A, J, H_1 and H_2 leads; sharp (by 13 mm)

increase in the amplitude of the R wave in V_4 lead in comparison with V_3 lead; $Sv_1 + Rv_5$ index = 37 mm (norm up to 35 mm).

2. S, 20 years old, I rank fencer. Suffers from chronic tonsillitis. X-ray examination shows enlarged left ventricle. Electrocardiogram (see Figure 24) shows tendency to deflection of the electrical axis of the heart to the right: identical amplitude of R and S waves in I lead and high R wave in III lead. In III lead, T wave is negative. Electrical position of heart vertical. QRS complex in aVL lead similar to QRS complex in V_1 and V_2 leads, while QRS complex in aVF lead similar to this complex in V_5, V_6 leads. In aVF lead, QRS complex has form qR, indicating deflection of top of heart forward. However, it does not seem possible to explain the presence of negative T_3 wave with deflection of electrical axis of heart to the right by these specifics of the location of the heart in the rib cage. Changes in electrocardiogram in this athlete result from pathological hypertrophy of left ventricle. This is indicated by the displacement below the isoline of the S-T segment in aVF, V_6 and V_7 leads and two-phase T wave in aVF, V_4-V_7, D, A and J leads. Pathological hypertrophy of the left ventricle is also manifested in standard leads with vertical electrical position of the heart in the form of deflection of the electrical axis of the heart to the right, combined with negative T_3 wave.

Change in electrocardiogram during and after muscular activity. During muscular activity of moderate and high intensity in healthy persons, in addition to an increase in the pulse frequency, a shortening of the P-Q and Q-T intervals, displacement of the P-Q and S-T segments below the isoline, an increase in the amplitude of the P wave, we also see at first a decrease in the T, R waves and a deepening of the S and Q waves, after which the T wave increases.

During the recovery period at first the T wave is elevated, after which it declines (Knoll, 1932; Marzehn, 1936; Schlomka et al., 1939; Reindell, 1949; Slapak, 1956; L. A. Butchenko, 1956, 1966; V. V. Matov, 1960; Metzner, 1960).

The changes noted in the electrocardiogram during muscular activity, during the restoratory period by Klepzig et al. (1956) is considered to be a result of vegetatively caused restructuring of metabolism in the myocardium. Prokop (1962), in addition to vegetative influences, relates the dynamics of the T wave during muscular activity with a change in the position of the heart in the rib cage in relation to the increased depth of respiration. Nordenfelt (1940, 1941) attaches essential significance to the decrease in blood supply to the heart as a result of tachicardia and the increase in arterial pressure. Thus, the genesis of changes in the electrocardiogram during and after muscular activity cannot be considered fully known.

In order to study this question, 118 healthy, well trained masters of sports (14 persons), first (64 persons) and second (40 persons) rank sportsmen were studied during three minutes work on the stationary bicycle, at 75 rotations per minute with a standard load and for five minutes recovery period (L. A. Butchenko, 1965). In addition to recording the electrocardiogram using the H_1 lead, the arterial pressure was measured by the Korotkov auditory method and the saturation of the arterial blood with oxygen was determined using an O-36 oxyhemograph.

During three minutes work on the stationary bicycle and the recovery period, all of the changes in electrocardiograms described above were noted. /133
As an example, we present the electrocardiogram of athlete Sh, 18 years, a II rank skier (Figure 25).

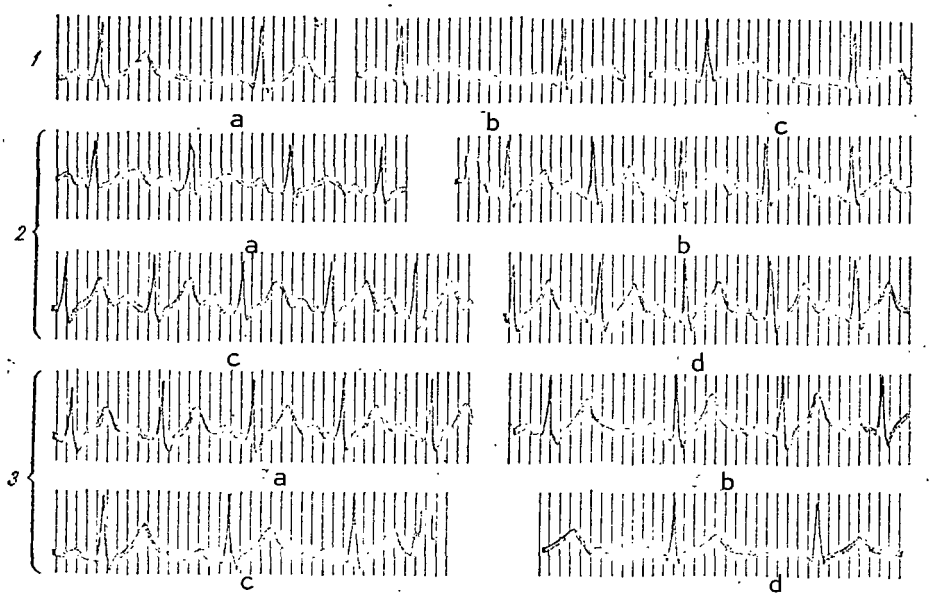


Figure 25. Electrocardiogram of Athlete Sh, 18 years old (II Rank Skier). 1, sitting quietly: a, ordinary respiration; b, Deep inhalation; c, Deep exhalation; 2, Loading: a, 30 sec; b, 50 sec; c, 2 min; d, 3 min; 3, Resting seated: a, 1 sec; b, 30 sec; c, 2 min; d, 5 min

In the electrocardiogram at rest during deep inhalation, we see a slight decrease in the T and R waves. During physical loading, an increase in pulse frequency, increase in the P wave, deepening of the S wave and displacement of the S-T and P-Q segments below the isoline are noted. The T and R waves decrease at 30 seconds loading, and the T wave increases beginning at 50 seconds loading. During the recovery period at the thirtieth second, a

further increase in the T wave is noted, replaced at the five-minute mark by a decrease.

It follows from the investigations performed that the reduction in the T wave and other changes in the electrocardiogram arising at the beginning of loading do not result from a change of the position of the heart due to increased depth of respiration. It might be considered that a role is played by redistribution of blood in the organism occurring during the working-in period. This redistribution of blood in the organism occurs in such a way that the heart receives less blood than is required: a relative decrease in coronary blood flow occurs, which in turn is manifested on the electrocardiogram in the form of a decrease in the T wave[1]. /134

The increase in the T wave arising at the end of the loading apparently results from a change in hemodynamics during the process of performance of the loading and a change in the bioenergetics of the myocardium resulting from hypoxemia. Thus, at the end of performance of the work, we see a clearly expressed increase in pulse frequency, a sharp displacement of the S-T and P-Q segments beneath the isoline, a significant increase in the P wave and a shortening of the P-Q interval, an increase in the maximum and minimum arterial pressures and a decrease in saturation of arterial blood with oxygen by more than 3% in comparison with the data while at rest.

A further elevation of the T wave, occurring at the end of the first minute of the recovery period, apparently results from vegetative changes and changes in metabolism related to the intensive course of the recovery processes. This might be indicated by the decrease in pulse frequency in comparison to the work period, the return to the initial value of the P-Q interval and the decrease in minimal arterial pressure. The decrease in minimal arterial pressure can also be explained by relaxation of the muscles during the recovery period.

The drop in the T wave observed at the beginning of the fifth minute of the recovery period probably results from pooling of blood in the periphery, decreased blood arrival at the heart and coronary vessels (Reindell, 1949; Slapak, 1956; Prokop, 1962).

It should be noted that changes in the electrocardiogram during physical work in athletes, in addition to the intensity and duration of the loading, also depend on the state of training. Well trained athletes do not show an increase in the T wave in response to small loads. In many cases, the recording of the electrocardiogram during loading allows pathological changes in the heart which cannot be detected by other methods of investigation to be revealed in athletes. As an example, we present the following electrocardiogram.

[1] This point of view, naturally, does not exclude other mechanisms for changes in the T wave during muscular activity. Further investigation in this direction is quite promising -- Ed.

III rank weight lifter V, 25 years old, healthy, satisfactorily trained (Figure 26). The electrocardiogram was taken in the H_2 lead. At rest, a sinus rhythm with a pulse frequency of 74 beats per minute was recorded; P-Q = 0.14 sec. During loading (eight deep knee bends carrying a 110-kg weight), in addition to an increase in pulse frequency to 133 beats per minute, a decrease in the T wave, a decrease in the P-Q interval to 0.11 sec, manifestation of frequent auricular extrasystoles with aberrant ventricular complex was noted. At rest and during the recovery period, these features were not observed.

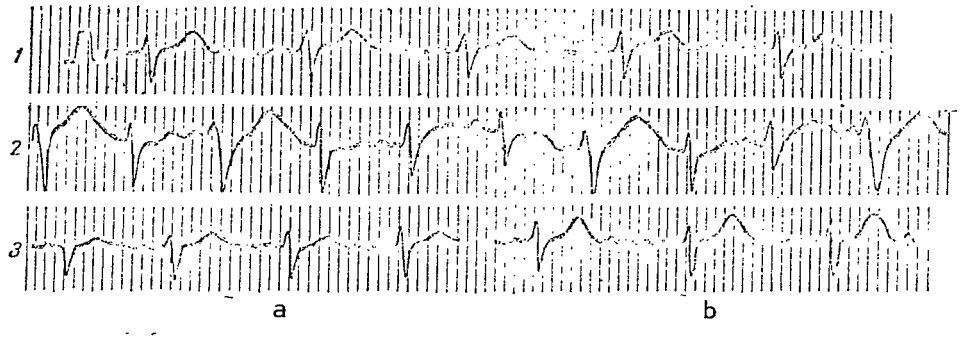


Figure 26. Electrocardiogram of Athlete V, 25 Years Old (III Rank Weight Lifter). 1, at rest seated; 2, under load (8 sec); 3, resting seated: a, 10 sec; b, 2 min

/135

The electrocardiogram of an athlete is most frequently recorded after 2-3 minutes running in place at 180 steps per minute and after various training exercises and competition. Electrocardiographic investigation after 2-3 minutes running in place allows, in addition to an evaluation of the functional state of the myocardium, determination of the general state of training of the organism. Investigation after training and competition allows an evaluation of the special state of training of the organism or its sports capabilities.

After 2-3 minutes running in place a well trained athlete shows a moderate (by 21-30% of the initial value) increase in pulse frequency. Sinus arhythmia becomes more clearly expressed and is encountered more frequently than at rest. The electrical axis of the heart does not change its position markedly, or is shifted slightly to the right.

The P and T waves, as a rule, increase slightly in the II, III and right precardiac leads. The P-Q interval is shortened or remains unchanged.

The electrical systole is also shortened or remains unchanged in comparison with its normal value. The restoration of these changes to their initial values, as a rule, is completed within five minutes of rest.

Athletes with insufficient general state of training and untrained persons show considerable increases in pulse frequency after 2-3 minutes running in place (by 43-54% of the initial value), as well as a decrease in the S-T and P-Q segments below the isoline, a decrease in summary voltage of the R waves ($R_1 + R_2 + R_3$) and increased restoration periods.

The changes in the electrocardiogram after training and competition are much more sharply expressed than after measured laboratory loads. Thus, after training the pulse frequency at three minutes into the recovery period exceeds the initial data by 60-100%. the S-T and P-Q segments of all test subjects are displaced below the isoline. Frequently, a decrease in summary voltage of the R waves is observed. In many athletes, lengthening of the electrical systole in comparison with its normal value by 0.05-0.08 sec and an increase in the P and T waves in the three standard and precordial leads are noted. Sometimes, the increase in the T waves in the precordial leads is expressed considerably more sharply than in the standard leads. /136

The recovery period after training is delayed: at 15 minutes into the rest period, the pulse frequency is 20-60% higher than its initial frequency. The S-T and P-Q segments, as a rule, remain displaced below the isoline. The summary voltage of the R waves is restored to its initial value by that time, while the electrical systole corresponds to its proper value. As concerns sinus arhythmia, immediately after training it disappears. During the recovery period, it rises again when the pulse frequency exceeds the initial value by an average of 50%.

These electrocardiographic changes are relatively more sharply expressed after training, the end purpose of which is the development of speed. They are somewhat less sharply expressed after training for general physical improvement or training designed to develop endurance and, finally, are least sharply expressed after training consisting of work on the technical performance of exercises.

After competition (primarily due to the great finish efforts) the changes in the electrocardiogram are expressed even more intensively than after training. Thus, the pulse frequency at three minutes of rest exceeds the initial value by 80-120%. The increase in the electrical systole in comparison with its proper value and the decrease in the S-T and P-Q segments and the summary voltage of the R waves are more frequent and more intensive than after training. An increase in T and P waves in standard and precordial leads is more frequent and more sharply expressed.

The recovery period after competition is extended: at 15 minutes into the rest period, the pulse frequency exceeds the initial value by 50-70%. In many athletes, restoration of the decreased summary voltage of the R waves does not occur. The electrical systole does not occur in correspondence with its proper value, the displacement of the S-T and P-Q segments below the isoline at 15 minutes into the recovery period has generally not yet been liquidated. All of these changes, apparently, are a result of the maximum utilization of the functional reserves of the heart and the physiological /137

fatigue of the organism after performance of intensive and extended work. This is confirmed by the fact that 12-24 hours rest liquidates all changes in the electrocardiogram completely.

These changes in the electrocardiogram after training and competition are observed in well trained athletes. If the athletes are insufficiently well trained or are overtrained, the lack of correspondence between the training loads and their preparation is manifested in an inadequacy of the reaction of the electrocardiogram to the load. Changes in the electrocardiogram after 2-3 minutes running in place become similar to those observed after training, and after training -- similar to those observed after competition. After competition, the electrocardiographic investigation of these athletes shows characteristics of overstressing of the heart. They are expressed in the appearance of flat positive two-phased or negative T waves, as a rule in the III lead, elongation of the P-Q and Q-T intervals and a deflection of the electrical axis of the heart to the right.

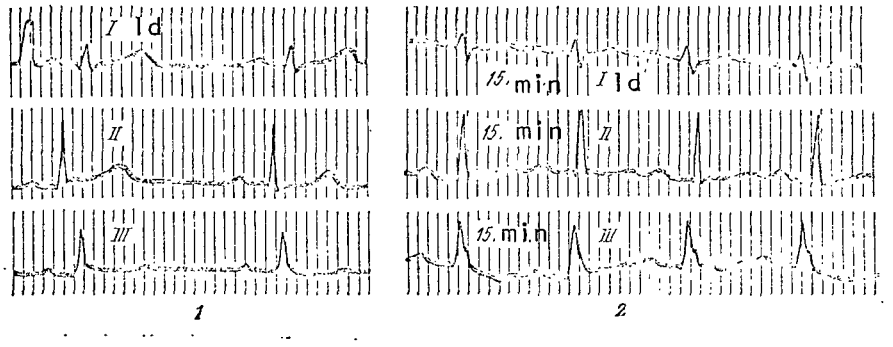


Figure 27. Electrocardiogram of Rower I, 21 Years Old. 1, before competition; 2, after competition.

As an example, we present data produced in an investigation of rower I, 21 years old, participating in competition while in the overtrained state. After the competition he complained of acute fatigue, pounding of the heart, and stabbing pains in the left half of the chest. Objectively, he appeared pale, his lips were bluish, he was breathless and manifested tachycardia.

On the electrocardiogram (Figure 27) at rest before competition we note moderate sinus bradycardia: the pulse frequency was 56 beats per minute. The remaining indicators show no peculiarities: P-Q = 0.19 sec, QRS = 0.08 sec, Q-T = 0.40 sec (norm 0.382 sec).

After competition, in addition to a sharp increase in the pulse frequency (102 beats per minute after 15 minutes rest), we note a negative T_3 wave, low T_1 and T_2 waves and a deflection of the electrical axis of the heart to the right. The auricular-ventricular conductivity is elongated (P-Q interval = 0.23 sec). /138

These changes in the electrocardiogram are characteristic for sharply expressed overstress.

Vector Cardiography[1]

The vector cardiographic method has not become widely used in sports medicine. However, this method could be of great significance for objective evaluation of the functional state of the "sports" heart.

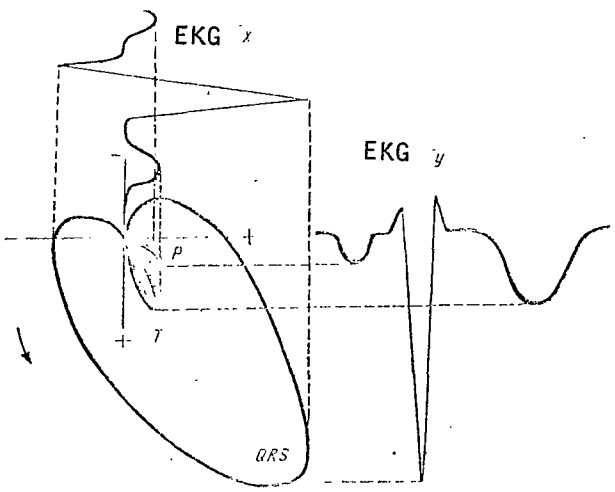


Figure 28. Schematic Drawing of Vector Cardiogram Formed of Two Electrocardiograms: the EKG_x and the EKG_y.

Vector cardiography significantly expands the possibilities of ordinary, scalar electrocardiography, which is a projection of the electromotive force of the heart on the so-called lead line. Thus, for example, in /139 cases when the resulting electromotive force vector is perpendicular to the lead line, its projection is a point, and is therefore not recorded on the electrocardiogram.

Methodological problems of vector cardiography. In vector cardiographic investigation, two pairs of electrodes are used to record the biopotentials arising in connection with the work of the heart as they vary with time. These potentials, after preamplification in the two channels of the vector electrocardiograph, are sent to the opposing pairs of plates in a cathode ray tube. As a result, a complex figure is recorded on the face of the screen -- the vector cardiogram, consisting of three loops: P, T and QRS (Figure 28). These loops correspond to the P, T and QRS complex waves on the electrocardiogram.

[1] This method is more precisely known as "vector electrocardiography." The reason is that at the present time such methods as vector ballistocardiography and vector dynamocardiography are used, so that the term vector electrocardiography should be used to represent the vector principle of analysis of the electrical manifestations of cardiac activity -- Ed.

Attempts to graphically record the dynamics of the resulting electromotive force of the heart during the course of the cardiac cycle in the form of a continuous curve have been made by Mann (1920). In 1926, he first recorded a vector cardiogram on a three-wire galvanometer. Schellong (1936), and later Wilson and Johnston (1938), independently used the cathode ray oscillograph to record the vector cardiogram.

During the first stage of its development, vector cardiography was primarily of theoretical significance. A number of systems were devised for location of the electrodes for the recording of vector cardiography. These include the double cube system, the equilateral tetrahedron system, the cube system and a number of others (Wilson, Johnston, 1938; Duchosal, Sulzer, 1949; Burch, 1950; Grishman and Scherlis, 1951; etc.).

In the Soviet Union, the method of vector cardiography has become widely used due to the invention by I. T. Akulinichev of a domestic model vector cardioscope. In 1953, I. T. Akulinichev suggested a system of precordial vector cardiographic leads, using five points on the rib cage: in the second intercostal space on the right (1), in the left subclavian area (2), in the fifth intercostal space along the anterior midaxillary line (3), on the right at the ensisternum (4), at the level of the lower angle of the scapula (5) at the spine. However, all of these points, except 1-3, which are used in Nehb's A lead, are not used in practical electrocardiography. This makes it more difficult to compare electrocardiographic and vector cardiographic data. Therefore, M. B. Tartakovskiy (1959) suggested that the fifth point of I. T. Akulinichev's system be moved to the fifth intercostal space along the rear midaxillary line (5a), which makes it possible to use the following classic bipolar precordial leads of Nehb in the vector cardiographic system: from points 1-3-A (anterior), from points 1-5a-D (dorsalis) and from points 5a-3-I (inferior), the diagnostic value of which is known in electrocardiography. /140

This system of leads is most acceptable for sports medicine not only since it makes it possible to compare vector cardiograms with electrocardiograms, but also since it is the Nehb lead which is used in taking electrocardiograms of athletes under load.

The method for analysis of the vector cardiogram is determined by the system of leads. Vector cardiograms made according to the systems used by Western authors are analyzed by constructing a three-dimensional vector curve of three projections in orthogonal coordinates or by a three-dimensional study using a stereoscope. These methods are cumbersome, complex and their clinical value is limited.

The methods for analysis of vector cardiograms described in the works of domestic authors vary (I. I. Bykov, 1958; Z. Z. Dorofeyeva and I. F. Ignat'yeva, 1958; M. I. Kechker, 1960; etc.). However, in our opinion, the most complete and objective method is the method of analysis suggested by M. B. Tartakovskiy (1959, 1962, 1964). In comparing the five vector

cardiographic leads BA_1, BA_2, BA_3, BA_4 and BA_5 (B indicates the vector cardiogram, A the Akulinichev system), the regularity introduced to the I. T. Akulinichev system consisting in that the electrocardiographic lead on the Y axis (from points 1-3) is common for the leads BA_1, BA_2 and BA_3, while the leads BA_1, BA_4 and BA_5 combine the electrocardiographic lead on the X axis (from points 2-4) was taken into consideration. It follows from this that the differences in the first three vector cardiograms depend on the recordings with the leads from the X axis (from points 2-4, 4-5a, 2-5a) with their common Y lead (from points 1-3). The same can be said concerning the leads BA_1, BA_4 and BA_5.

Analysis of vector cardiograms should be performed as follows:

1. The form of the QRS loops is studied. In place of the descriptive principle for evaluating forms ("leaf-shaped", "oval", "odd-shaped", etc.) used by most authors, we divide this loop into two types depending on the number of axes which can be drawn in the loop, and whether the axis intersect the loop. If one axis can be drawn in the QRS loop, which does not intersect it, we call it a type I loop. If the loop has two or three axes and they intersect the loop itself, it is called a type II loop.

2. The orientation of the QRS and T loops is determined, established by considering the quadrant and angle β between the axis of the loop and the X axis. Counting of the angle is performed from (+) on the X axis to (+) on the Y axis. The quadrants are represented as follows: quadrant I -- between (+) on the X axis and (+) on the Y axis; quadrant II -- between (+) on the /141
X axis and (-) on the Y axis; quadrant III -- between (-) on the X axis and (-) on the Y axis; quadrant IV -- between (-) on the X axis and (+) on the Y axis. The polarity in each of the five vector cardiographic leads is determined by the polarity of the electrocardiographic leads attached to the X and Y axes. The direction toward which the R wave of the electrocardiogram is turned is considered positive, and the opposite direction is considered negative (Figure 29).

3. The angle between the axes of the QRS and T loops is determined.

4. The direction of recording of the QRS loop is established (from the development of the loop).

5. The amplitude of the axes of the T and QRS loops is measured (in millivolts).

6. Using an increased amplification level, nonclosure of QRS and T loops is taken into consideration.

7. The total area of the QRS loops is determined, i.e., the area of the /142
loops in all five leads. This makes it possible to evaluate the vector cardiogram as a whole, since the value of area and QRS loops in the

individual leads depends on the electrical position of the heart (I. A. Ovsyshcher, 1962; M. B. Tartakovskiy, 1962, 1964).

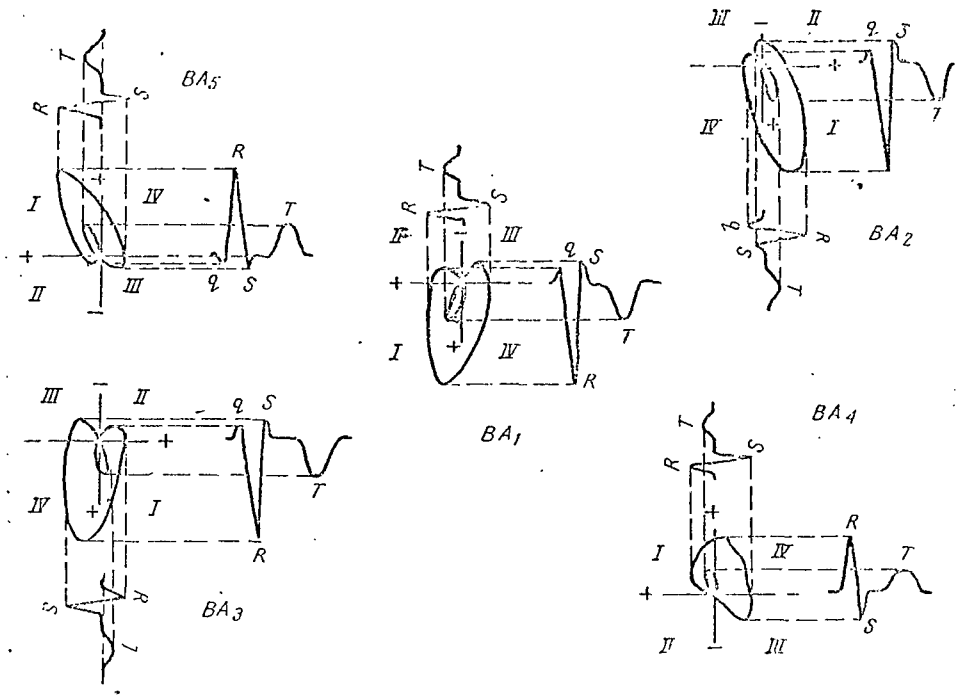


Figure 29. Polarity and Representation of Quadrants in the Five Leads of the System of Bipolar Precordial Leads. Roman numerals represent quadrants.

The vector cardiogram of athletes. The literature contains descriptions of norms for vector cardiograms of healthy persons not occupied in sports (S. L. Mailyan, 1954; M. I. Kechker and M. P. Shurgaya, 1958; V. I. Klemenov, 1961; V. S. Yurasov, V. V. Bulychev and V. Ya. Preyger, 1962; V. S. Gasilin, 1963; B. K. Myakishev, 1963; etc.). However, these norms cannot be completely used in medical testing for athletes, since the occupation with sports causes a number of changes differentiating the heart of athletes from the heart of healthy persons not occupied in sports. The available works dedicated to the study of normal vector cardiograms of athletes have been performed using the cube system (V. Ya. Preyger, 1960; V. V. Bulychev, V. S. Yurasov, and V. Ya. Preyger, 1962).

We studied the vector cardiograms of athletes using the modified I. T. Akulinichev system for 100 healthy, well trained athletes between 17 and 34 years of age -- representatives of the principal forms of sports, with high qualifications (masters of sports, first rank athletes 68%). The vector cardiograms were taken using the VEKS-OI apparatus in five leads

(BA_{1-5}) with amplification to 20 mm per 1 mv, as well as with increased level of application to 60-80 mm per 1 mv (using a circuit designed by Engineer I. D. Pupko, 1962). Increasing the sensitivity of the device allows us to determine such valuable characteristics as nonclosure of the QRS and T loops, and to clarify the direction of the T loops, which cannot always be determined with ordinary amplification and small T loop.

According to our data, the vector cardiograms of athletes are characterized by the following indicators.

1. The QRS loop, as is the case for persons not involved in sports, may be either type I or type II. However, in the BA_I lead in athletes the II type loop predominates; this may be related to the fact that in 25% of the athletes uneven excitation of the myocardium of the right ventricle or a delay in excitation of the right epiventricular crest is observed, which is a variant of the norm.

2. The location of the QRS and T loops shows no important differences in the system of coordinates used.

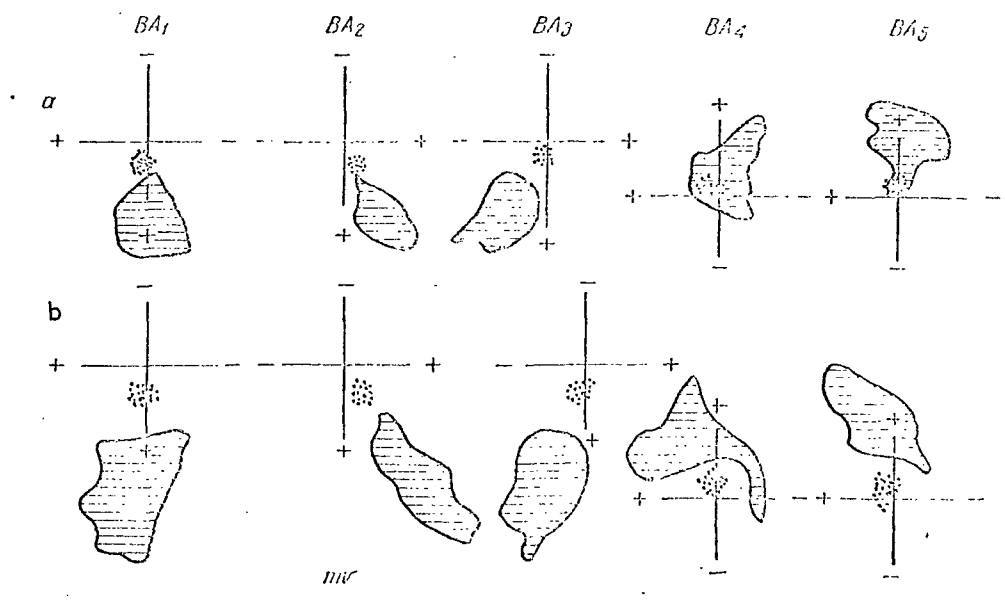


Figure 30. Diagram of Location of QRS and T Loops in Vector Cardiograms. a, healthy persons not involved in sports (according to M. B. Tartakovskiy); b, Athletes

Figure 30 shows the orientation of the QRS and T loops schematically in the system of coordinates; the locations of the QRS loops are shaded, and the T loops are marked with points. At the top (a) we see data on the

orientation of the QRS and T loops in the vector cardiograms of persons not occupied in sports, while at the bottom (b) we see similar data for athletes. As we can see from Figure 30, the location of the QRS and T loops in the vector cardiograms of nonathletes and athletes do not differ essentially. In some cases the T loops in the BA_5 leads in athletes are located in quadrant II. This should be considered a variant of the norm, since the D lead of Nehb is included in the formation of this vector cardiogram in the Y axis, which normally shows a negative T wave. /143

3. The direction of recording of the QRS loops in the vector cardiograms of athletes and untrained persons differs slightly from each other. The direction of recording of the QRS loops is of considerable significance, since a number of pathological processes in the heart result in changes in the movement of the beam recording the QRS loop.

In the vector cardiograms of athletes, the recording of the QRS loops in the BA_2, BA_3 and BA_4 leads is similar to the direction of movement of the beam forming the QRS loops in the vector cardiograms of persons not involved in sports: in leads BA_2 and BA_3 it is always mutually opposite (in BA_2 counterclockwise, in BA_3 -- clockwise), while in BA_4 the recording of the QRS loops occurs equally in both directions. The differences concern BA_1 and BA_5. Thus, in lead BA_1 in athletes we see a QRS loop with the movement of the ray in the clockwise direction three times more frequently than in untrained persons. In lead BA_5 in athletes, clockwise movement predominates, while for those not involved in sports in lead BA_5 recording of the QRS loop occurs only counterclockwise. These differences are a result of uneven excitation of the myocardium of the right ventricle. /144

4. As concerns the magnitude of the axes of the QRS loops, the vector cardiograms of athletes are not homogeneous.

In almost one half of the vector cardiograms of athletes, the axes of the QRS loops are greater than the maximum values of axes of PRS loops in the vector cardiograms of persons not involved in sports. The magnitude of the axis of the QRS and T loops is given considerable significance. Thus, Z. Z. Dorofeyeva and I. F. Ignat'yeva (1958) relate a decrease in the axes of the QRS loops to pathological changes of the myocardium arising during the period of intensification of the rheumatic process, while M. I. Kechker and Sh. I. Shurgaya (1958) relate an increase in the axes of the loops to the development of hypertrophy of the myocardium.

A number of authors note high R waves in the electrocardiograms of athletes (S. P. Letunov, 1957; L. A. Butchenko, 1958; etc.); they explain this by the increased tonus of the vagus nerve or by the development of physiological hypertrophy and an increase in the electromotive force of the heart. A second explanation seems more probable to us, since the increase in

the tonus of the vagus nerve should be accompanied by a decrease in the P and T waves, while the T wave in athletes is characterized by high values. Considering that the vector cardiogram is formed of two electrocardiograms, it might be assumed that an increase in the axes of the QRS loops in athletes depends on hypertrophy of the myocardium developed in the process of training. However, in order to solve the problem of hypertrophy of the ventricles of the heart, we must measure the area of the QRS loop, which depends not only on the maximum amplitude of the waves of the electrocardiogram, but also on the time relationship of the processes of excitation along the X and Y axes, recorded by the vector cardiogram.

The T loop in 15% of the observations in athletes is increased in comparison with the maximum value of the T loop axis in the vector cardiograms of persons not involved in sports. High values of the axes of the T loop, like high T waves in the electrocardiograms of athletes, can be explained by a reinforcement of metabolic processes in the myocardium under the influence of training (Ye. S. Troshanova, 1951; N. N. Yakovlev, 1955).

5. The area of the QRS loops, defined planimetrically, was found to be greater in almost half of the athletes studied than in persons not involved in sports.

We can see from Table 8 that the mean values (M) of the area of QRS loops in vector cardiograms of athletes are also higher than in healthy persons not involved in sports. A high value for the mean square deviation (±δ) indicates significant individual fluctuations in the area of the QRS loop in athletes. The increase in the area of QRS loops apparently results from hypertrophy of the heart (M. I. Kechker, 1958; M. B. Tartakovskiy, 1960; Ye. I. Kuznetsova, 1963; etc.). /145

TABLE 8. COMPARISON OF AREA OF QRS LOOPS (IN cm^2) ON VECTOR CARDIOGRAMS OF NONATHLETES AND ATHLETES (MEAN DATA ACCORDING TO M. B. TARTAKOVSKIY, 1964)

Lead	Healthy non-athletes		Athletes	
	M	$\pm\sigma$	M	$\pm\sigma$
BA_1	4,2	±2,4	5,7	±5,1
BA_2	4,21	±2,6	6,46	±5,25
BA_3	6,4	±3,5	7,7	±4,56
BA_4	1,6	±1,1	3,3	±2,64
BA_5	3,0	±2,0	3,5	±2,69

Tr. Note: Commas indicate decimal points.

6. Nonclosure of QRS and T loops, which was not seen in persons not involved in sports, was determined in 13% of the athletes. This nonclosure was observed in two variants. The first was characterized by the fact that the end point of the QRS loop was located in quadrant I; this corresponds on the electrocardiogram to displacement of the S-T segment above the isoline. This phenomenon is explained by a number of authors by an increase in the tonus of the vagus nerve.

The second variant is characterized by location of the end point of the QRS loop in quadrants II and III, which corresponds on the electric cardiogram to displacement of S-T beneath the isoline and is combined with an increase in the area of the QRS loop and, consequently, indicates hypertrophy of the myocardium.

Thus, in the form of the loops, and their location in the coordinate system, the vector cardiograms of athletes do not differ from the vector cardiograms of persons not involved in sports. The differences concern the direction of recording of the loops in the BA_1 lead, the magnitude of the axes of the QRS and T loops, the area of the QRS loops and the nonclosure of the QRS and T loops. The predominance of the recording of QRS loop in the BA_1 lead in the clockwise direction is explained by the uneven excitation of the myocardium of the right ventricle, which may depend on the age of the test subjects. In athletes, an increase in the axes and areas of QRS loops also occurs, related to the development of myocardial hypertrophy, and the nonclosure of the QRS and T loops is encountered in two variants, one of /146
which apparently depends on an increase in the tonus of the vagus nerve, while the other results from development of hypertrophy of the myocardium.

The location of the T loops in the II quadrant in the BA_5 lead should be considered a variant of the normal vector cardiogram for an athlete. All of the vector cardiogram parameters for athletes described are presented in Table 9.

The vector cardiograms presented below illustrate what we have stated concerning the normal vector cardiogram of an athlete and its specific features.

The vector cardiogram of a girl not involved in sports (Figure 31, A) attracts attention by the small area of the QRS loops (total area of QRS loops 9 cm^2), while the QRS loops in all leads except for BA_3 are located in quadrant I, and the T loops in quadrant I in all leads. The angle between the axes of the QRS and T loops does not exceed 17°. The direction of recording in the BA_1, BA_2 and BA_4 leads is counterclockwise, in BA_3 and BA_5 -- clockwise.

TABLE 9. CHARACTERISTICS OF VECTOR CARDIOGRAMS OF ATHLETES

Angle between axes of QRS and T loops			Amplitude of axes (mv)						Total area of QRS loops cm^2
			Of QRS loop			Of T loop			
M	±°	M±°	M	±°	M±°	M	±°	M±°	
13°	±10°	74%	2.8	±0.9	67%	0.75	±0.26	77%	
8°	±5°	68%	2.85	±0.95	66%	0.8	±0.38	77%	
15°	±13°	78%	2.75	±0.95	67%	0.75	±0.75	81%	26.3
--	--	--	1.6	±0.56	73%	0.5	±0.19	85%	
16°	±13°	68%	1.6	±0.5	72%	o.5	±0.15	82%	

Vector cardiographic indicators	Form of QRS loops (in %)		Direction of recording of QRS loop (in %)			LβQRS			LβT		
Lead	I Type	II Type	Clockwise	Counter-Clockwise	Self-intersecting	M	±°	M±°	M	±°	M±°
BA_1	47	53	23	55	20	88°	±14°	70%	78°	±18°	69%
BA_2	84	16	--	100	--	65°	±6°	72%	71°	±7°	74%
BA_3	62	38	96	--	4	101°	±13°	71%	88°	±20°	71%
BA_4	64	36	42	43	15	--	--	--	60°	±16°	76%
BA_5	63	37	62	19	19	66°	±21°	75%	61°	±22°	78%

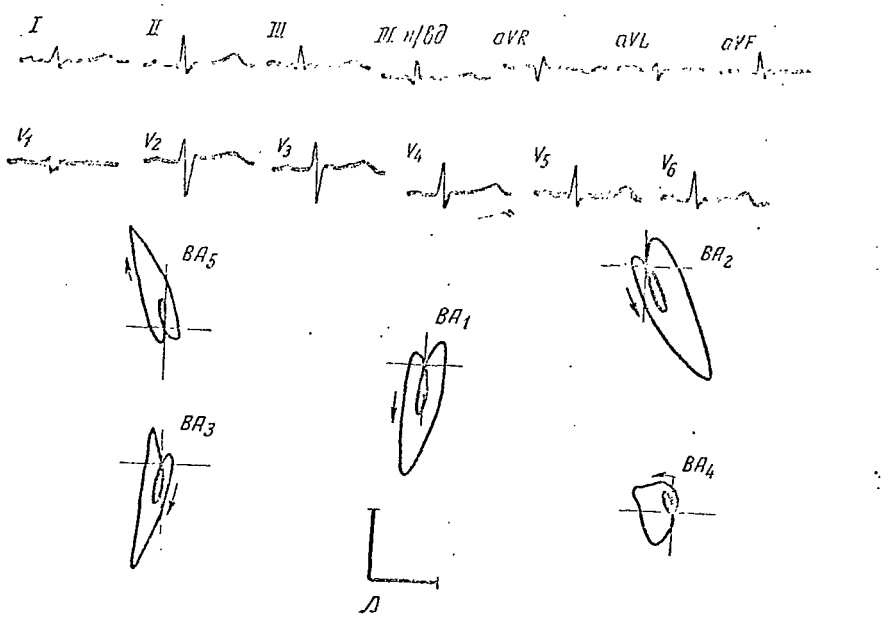


Figure 31 A. Electrocardiogram and Vector Cardiogram of Nonathlete A, 21 Years Old

Female athlete Sh (Figure 31 B) shows an increase in the total area of QRS loops to 51.4 cm^2, which is a characteristic of physiological hypertrophy of the left ventricle. The direction of recording in leads BA_1, BA_2 is counterclockwise, while in leads BA_3, BA_4 and BA_5 recording is clockwise. The QRS loops in leads BA_1 and BA_3 are located in quadrant IV, in the remaining leads -- in quadrant I. The T loop in all leads (except BA_3) is located in quadrant I. The angle between the axes of the loops QRS and T does not exceed 20°.

Changes in vector cardiogram with physical loading. Considering the great significance of determination of the functional state of the heart in sports medicine, we investigated changes in the vectorcardiograms of 60 athletes under the influence of muscular work. The investigations were /148
were performed before and after two minutes running in place at 180 steps per minute. At one minute into the recovery period, the vector cardiogram was recorded in lead BA_1, at two minutes -- in leads BA_2 and BA_3, at three minutes -- in leads BA_4 and BA_5. At four and five minutes, all leads of the vector cardiogram were recorded. Also, vector cardiograms were taken for 15 athletes after training for ten minutes of the recovery period.

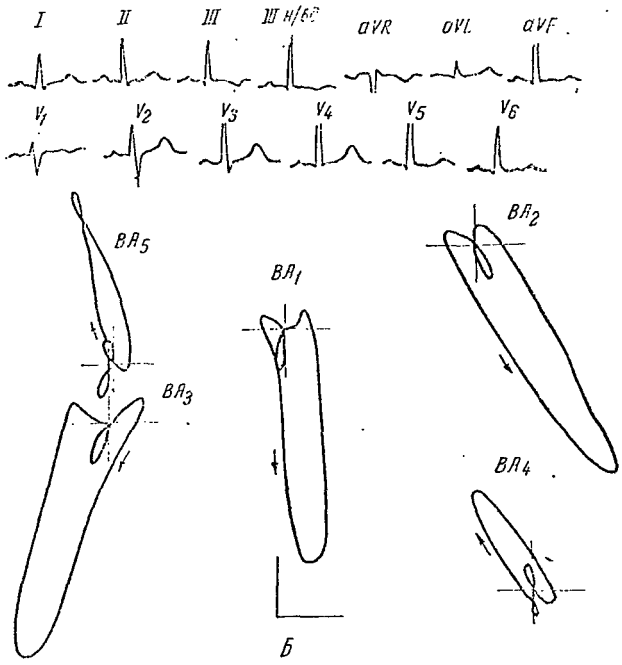


Figure 31 B. Electrocardiogram and Vector Cardiogram of Athlete M, 30 Years Old (Honored Master of Sports in Canoeing)

In order to determine the dependence of the functional state of the heart of athletes on various trends in the training process and the degree of training, we distinguish three groups among the test subjects. The first group consisted of highly trained masters of sports in canoeing and kayak, members of the Olympic team of the USSR, the training process for which consisted predominantly of training for endurance. This group of athletes, which showed high functional /149 capacity of the myocardium, was compared on the one hand with a group of less trained athletes (III rank and novices), also trained for endurance, and on the other hand with a group of athletes with insufficient general physical preparation, the training of which concentrated on high forces (boxing, weight lifting).

In the highly trained athletes with good functional capacity of the heart, the vector cardiograms showed only insignificant changes, consisting of displacement of the axes of the QRS and T loops to the right or left of the Y axis with a change in the angle between them, slight fluctuations in the amplitudes of the T loops (±2-3 mm) after two minutes running. The initial picture was returned in the fourth or fifth minute. This is illustrated by the curve of honored master of sports for canoeing M, 23 years of age (Figure 32).

In athletes of the two other groups, more significant changes in the /150 parameters of the vector cardiograms were observed: variously directed movement of the axes of the QRS and T loops with increasing angle between them up to 34°, considerable (up to ±13 mm) changes in the magnitude of the T loop, changes in the form of the QRS loop; a slight increase in the recovery time for the parameters of the vector cardiograms in comparison with the initial data. These changes in the vector cardiograms are a reflection of the satisfactory functional capacity of the myocardium.

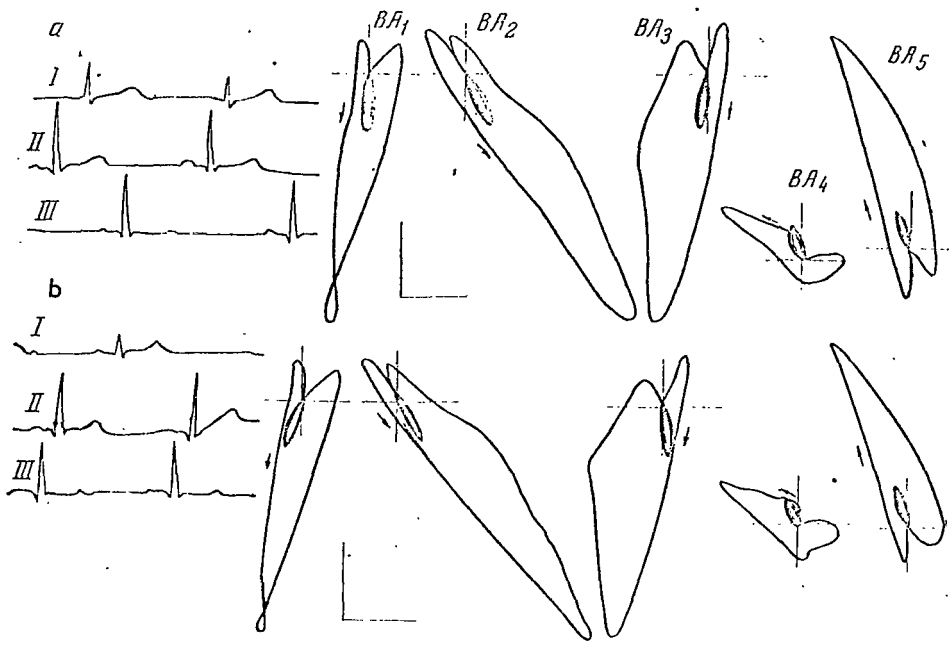


Figure 32. Electrocardiogram and Vector Cardiogram of Honored Master of Sports for Canoeing M, 23 Years Old. a, at rest; b, after 2 min running in place

With unsatisfactory functional capacity of the myocardium, even greater changes are seen on the vector cardiogram after two minutes running. The T and QRS loops are displaced into quadrants where they are not found on a normal vector cardiogram. An example is the electrocardiogram and vector cardiogram of football player K (Figure 33).

The changes in the vector cardiogram observed after training sessions (displacement of T loop to the right, varying direction of axes of QRS loops and T loop and increase in angle between them, significant change in magnitude of T loops, nonclosure of QRS and T loops, delay in recovery) are a result of clear physiological fatigue. /151

Vector cardiographic investigation performed after laboratory or training loads allows an evaluation to be given to the functional capacity of the heart, and in some cases allows pathological changes (insufficiency of coronary blood supply) which cannot be determined by electrocardiograms to be found.

Vector cardiogram and hypotrophy of the myocardium in athletes. Considering the possibility of vector cardiography in the recognition and evaluation of hypertrophy of the myocardium, we studied the vector cardiographic characteristics of hypertrophy of the left ventricle in 80 athletes

and 20 patients suffering from hypertension and aortal heart defects in detail.

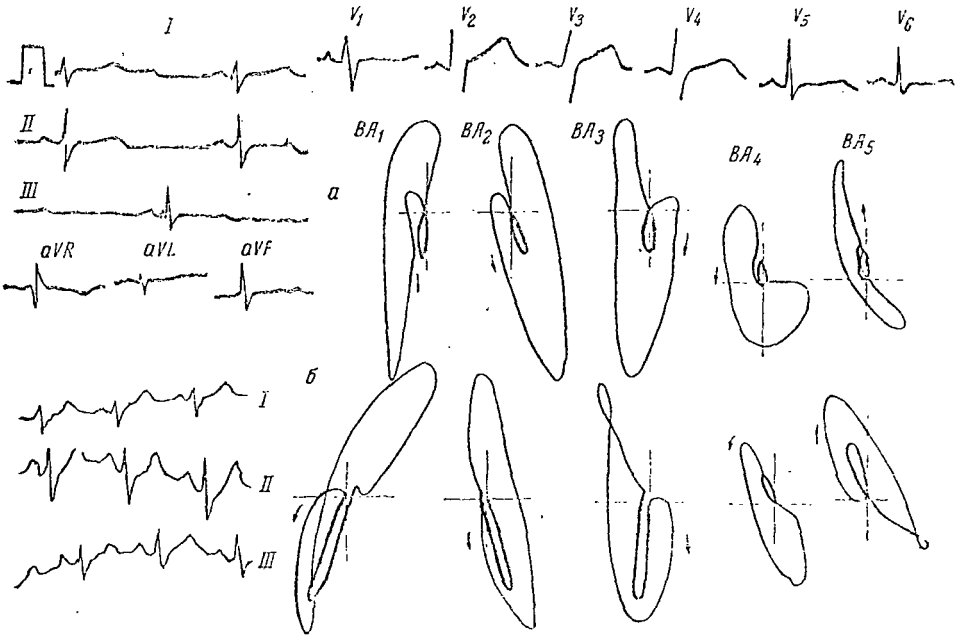


Figure 33. Electrocardiogram and Vector Cardiogram of Athlete K, 18 Years Old, I Class Football Player. a, at rest; b, after 2 min run. The vector cardiogram after the load shows a sharp increase in the T loop (by 20 mm) and the terminal portion of the QRS loop, which causes the appearance of a second axis, which becomes the main axis of the QRS loop and is shifted into quadrant III; the QRS and T loops show nonclosure. Recovery occurred after 8 min.

Myocardial hypertrophy in the athletes was established on the basis of electrocardiographic criteria. In 75 of them, the following characteristics of hypertrophy of the left ventricle were noted: high R wave (> 19 mm in aVF lead in 44%), high R wave (> 26 mm in lead V_5 in 60%), counterclockwise rotation about longitudinal axis (in 40%), increase in time of local electronegativity in left precordial leads (0.05-0.07 sec in 64%), increase in amplitude of R wave (up to 8 mm) in V_4 lead in comparison to V_3 lead (in 92%), index $S_{v1} + RV_5 > 35$ mm (in 93%). According to the literature data, the most convincing characteristics of physiological hypertrophy of the left ventricle are the increase in the amplitude of the R wave in the V_4 lead in in comparison with the V_3 lead in combination with elongation of the time of local electronegativity, as well as the $S_{v1} + RV_5$ index > 35 mm.

In five athletes, characteristics of pathological hypertrophy of the left ventricle were detected: a drop in the T wave in the II lead, uneven negative or decreased T waves in the left precordial leads and displacement of the S-T segment beneath the isoline. In all the patients, the vector cardiogram showed various stages of development of hypertrophy of the left ventricle, according to the criteria described in the literature (M. B. Tartakovskiy, 1960; M. I. Kechker, 1960).

Comparing the vector cardiograms of patients with hypertrophy of the left ventricle and athletes, we found that in both groups there was an increase in the summary area of the QRS loops. Thus, the summary area of the QRS loops in the vector cardiograms of athletes with hypertrophy of the left ventricle was 37.5 cm^2, while the patients showed 37.6 cm^2. Also, the mean values of summary area of QRS loops in healthy persons not involved in sports, as well as athletes without essential hypertrophy of the myocardium differ considerably from the mean values of the summary area of QRS loops in the vector cardiograms of athletes with hypertrophy of the left ventricle. They are considerably less, 19.6 and 26.3 cm^2 respectively.

The summary area of QRS loops was greater in athletes in those types of sports in which endurance loads predominate, and relatively less in those types of sports in which these loads are less. Thus, in a group of skiers /152
the summary area of QRS loops averaged 32.5 cm^2, while rowers showed 27.8 cm^2, middle- and long-distance runners -- 25.6 cm^2 and weight lifters -- 15 cm^2, gymnasts -- 14.7 cm^2 (the difference between these mean values is statistically reliable).

Analyzing the vector cardiograms of athletes with hypertrophy of the left ventricle, we noted that three of the four stages of hypertrophy of the left ventricle distinguished in clinical practice were observed in these persons.

In the athletes, hypertrophy of stage I predominated (82.5%) and hypertrophy of stage IV was completely absent, reflecting the sclerotic and dystrophic processes in the myocardium. In 11.2% of the athletes, hypertrophy of stage II was noted, while 6.3% showed signs of the III stage of development of hypertrophy of the left ventricle.

Comparison of electrocardiographic and vector cardiographic data showed that of 75 athletes with indications of hypertrophy of the left ventricle on the basis of electrocardiographic data, 66 showed stage I on the basis of vector cardiographic data, while nine showed stage II of hypertrophy of the left ventricle. Consequently, in nine athletes (11.2%) the vector cardiogram showed the initial phenomena of disruption of processes of repolarization (nonclosure of QRS and T loops) not shown on the electrocardiogram.

Of the stages of hypertrophy of the left ventricle discovered in the athletes only in stage I (increase in the axes and areas of QRS loops, displacement of QRS loops from quadrant I to quadrant IV) is disruption of of the process of repolarization absent, allowing it to be looked upon as

physiological. Stage III, in which disruption of processes of repolarization are clearly shown (stages I and II include displacement of the T loops in the coordinate system and nonclosure of QRS and T loops), reflects pathological hypertrophy of the left ventricle. Stage II, in which the initial indicators of disruption of repolarization appear (in addition to changes of stage I, nonclosure of QRS and T loops is observed), apparently should be looked upon as a transition stage between physiological and pathological hypertrophy.

Using vector cardiographic criteria for hypertrophy of the myocardium of the left ventricle, we can diagnose hypertrophy even when it cannot be seen on an ordinary electrocardiogram.

As an example of the diagnostic value of vector cardiography in the diagnosis of hypertrophy of the left ventricle, we present an electrocardiogram and vector cardiogram of athlete M, 23 years old, long-distance runner I class. The electrocardiogram shows no characteristics of myocardial hypertrophy, while the vector cardiogram shows first stage hypertrophy of the left ventricle (the summary area of the QRS loops = 43.3 cm^2), the QRS loops in all leads except BA_2 are located in quadrant IV, the T loop is deflected in leads BA_1 to the right of the Y axis (Figure 34).

Analyzing the vector cardiograms of 300 athletes, we found that in 165 athletes (55%), indications of physiological hypertrophy were present. The hypertrophies most frequently developed in those forms of sports in which training for endurance predominates. Thus, in rowers, hypertrophy of the left ventricle was detected in 70% of the test subjects, in middle- and long-distance runners -- in 70%, in boxers -- in 30.7%, in gymnasts -- in 26.6%. /153

Vector cardiography in evaluation of partial block of right branch of bundle of His in athletes. One frequently encountered specific of the electrocardiogram of athletes is the so-called partial block of the right branch of the bundle of His, appearing in the right precordial leads (V_1, V_2) as the following characteristics: additional duration of local electronegativity in R^1 or r^1 wave of 0.05-0.08 sec, duration of intraventricular conductivity not over 0.12 sec. These changes are observed in 12% of the athletes according to the data of Rasch et al. (1958) and in 10.5% according to the data of L. A. Butchenko (1960). /154

The same changes in the electrocardiogram were detected by certain authors in healthy persons not involved in sports (Holzmann, 1937; Freundlich, Lepeschkin, 1939; Sokolow, Friedlander, 1949; etc.).

However, partial block of the right branch of the bundle of His may also be a manifestation of a number of pathological processes: cardiosclerosis, focal myocarditis, myocardial infarct, pulmonary artery embolism, hypertrophy of the right ventricle, etc. There are two different points of view concerning the interpretation of indications of partial block of the right branch of the bundle of His in healthy persons. Some authors acknowledge the

possibility of partial block of the right branch of the bundle of His, i.e. disruption of conductivity through the right branch of the bundle of His in healthy persons, and evaluate this as a variant of the norm (Wilson, Rosenbaum, Johnston, 1947; Plas et al., 1957; Zuckerman, 1937; etc.).

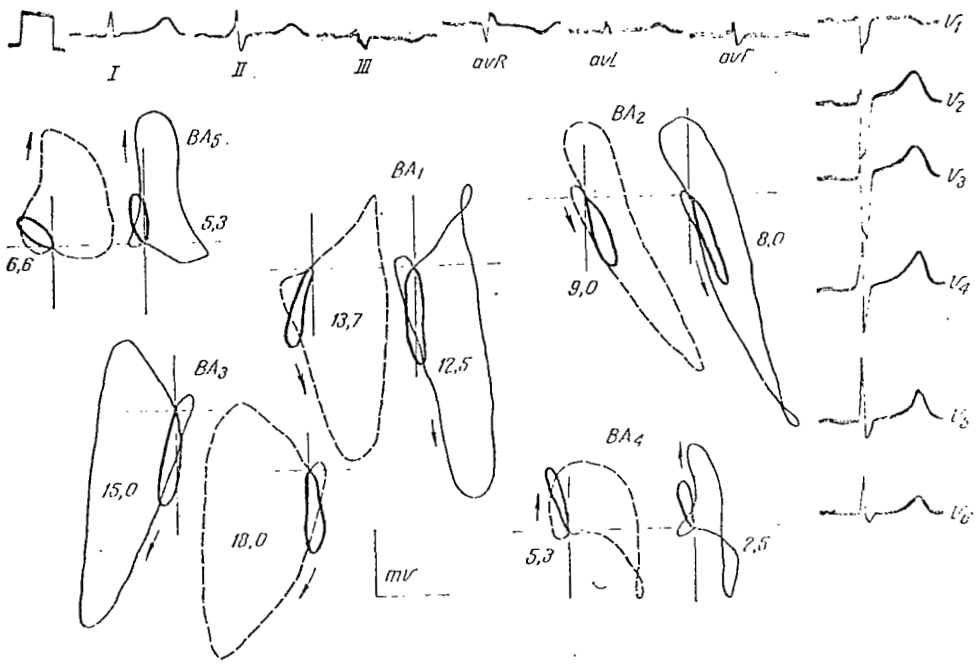


Figure 34. Electrocardiogram and Vector Cardiogram of Athlete M, 23 Years Old (Long-Distance Runner). Dotted lines show vector cardiogram taken upon inhalation.

Other authors do not explain the appearance on the electrocardiogram of the additional R^1 wave as a block of the right branch of the bundle of His, but rather indicate a special type of excitation of the myocardium, caused by the anatomic structure of the right ventricle, later excitation of the outflow paths of the right ventricle (conus arteriosus or conus pulmonalis).

Kossman et al. (1950), Lenegre (1953) and other authors, using direct leads from the epicardial surface of the right ventricle attached during operations, have found that over a small sector in the area of the epiventricular crest, a typical form of the complex rsR' or rsr' for block of the right branch is recorded. The appearance of an additional R' wave is related by these authors to a delay in excitation of the right epiventricular crest, which is observed in children and young persons more frequently than in persons of middle or advanced age.

Differential evaluation of these electrocardiographic changes, which are different in etiology but similar in form by the method of electrocardiography, is not considered convincing by many authors, since it is based on measurement of the time of local electronegativity, which shows great individual fluctuations.

With the development of vector cardiography, due to the possibility of comparing two electrocardiographic leads, the possibility appeared for deeper analysis of disruptions in conductivity. A delay in one electrocardiographic lead relative to another as a result of disruption of conductivity through the right branch of the bundle of His in case of a partial block caused by any pathological process leads to considerable changes in the vector cardiogram. The vector cardiogram takes on a form differing completely from a partial block of the right branch of the bundle of His (Figure 35) in case /155
of a delay in excitation of the right epiventricular crest (M. B. Tartakovskiy, 1959).

Considering the variety of factors causing the electrocardiographic picture of a partial block of the right branch of the bundle of His, and the possibility of vector cardiography to differentiate between these changes, we performed vector cardiographic analysis of the characteristics of partial block of the right branch of the bundle of His revealed on the electrocardiograms of athletes. We found 35 such athletes from 300, i.e. 12%. Clinical examination of these athletes showed no deviations in the state of health. In none of these persons did vector cardiographic investigation show a disruption in conductivity through the right branch of the bundle of His, i.e., true block of the right branch, which shows a clear vector cardiographic picture differing from that of uneven excitation of the myocardium of the right ventricle or delay in excitation of the right epiventricular crest (see Figure 35, b, c).

We found four variants of forms of QRS loops resulting from delayed excitation of the right epiventricular crest (see Figure 35, c).

Vector cardiographic investigation of the 35 athletes whose electrocardiograms showed indications of a partial block of the right branch of the bundle of His allowed us to divide all those investigated into three groups:

1) 18 athletes showed characteristics of delayed excitation of the /156
right epiventricular crest; an example is the vector cardiogram of athlete B, 19 years old, boxer I class (Figure 36, a);

2) in 15 athletes, the delay in right epiventricular crest excitation was combined with indications of hypertrophy of the left ventricle, which is illustrated by the vector cardiogram of athlete Zh, 17 years old, a skier II class (Figure 36, b);

3) for only three athletes were the electrocardiographic indicators of partial block of the right branch of the bundle of His evaluated according to the vector cardiogram as a manifestation of the initial characteristics of

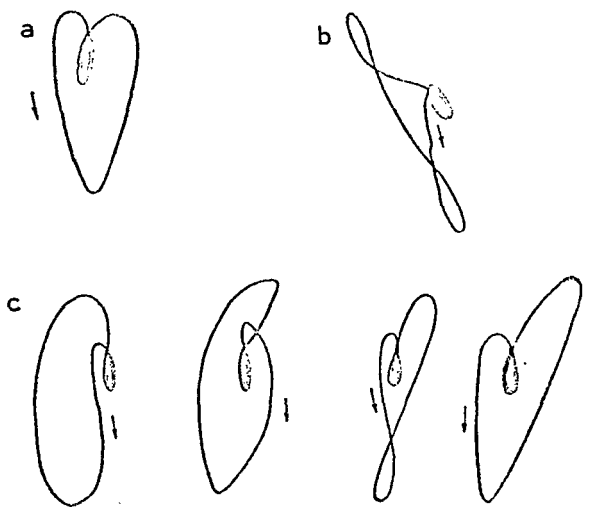


Figure 35. Vector Cardiogram in Lead BA_1. a, with normal course of excitation; b, with partial block of right branch of bundle of His; c, with delayed excitation of right epiventricular crest.

hypertrophy of the right ventricle (displacement of the QRS loop in the BA_3 lead into quadrant I and a change in the direction of recording of the QRS loop counterclockwise, orientation of T loops in the IV quadrant in all leads, except for BA_2 (Figure 36, c).

The usage of phase analysis of vector cardiograms of 300 athletes revealed that 35.7% manifested a delay in excitation of the right epiventricular crest, which is usually interpreted as a partial block of the right branch of the bundle of His in athletes, and is revealed electrocardiographically in 12%. /157

Considering the literature data on the dependence of characteristics of partial block of the right branch of the bundle of His on the type of sports (Slapak, 1957; Wolf, 1957; etc.), as well as our own data on the precise distribution of delayed excitation of the right epiventricular crest according to the type of sport, we assume that further investigations are necessary in the direction of differential diagnosis with the initial stages of hypertrophy of the right ventricle.

The vector cardiogram in the evaluation of changes in the T wave and the S-T interval in athletes. In the last 4-5 years, cases of changes of the final portion of the ventricular complex of the electrocardiogram in athletes have become more frequent. Thus, according to the data of the Leningrad Medical-Physical Culture Dispensary, in 1.5% of the electrocardiograms of athletes, generally as a chance finding, changes in the end portion of the ventricular complex are noted which might be interpreted as disruption of coronary blood supply to some portion of the heart. These changes consist of flattening or inversion of the T wave in the standard and precordial leads, and displacement of the S-T segment beneath the isoline.

These changes in the end portion of the ventricular complex of the electrocardiogram in the left precordial leads are observed in patients with various pathological processes such, for example, as pathological hypertrophy

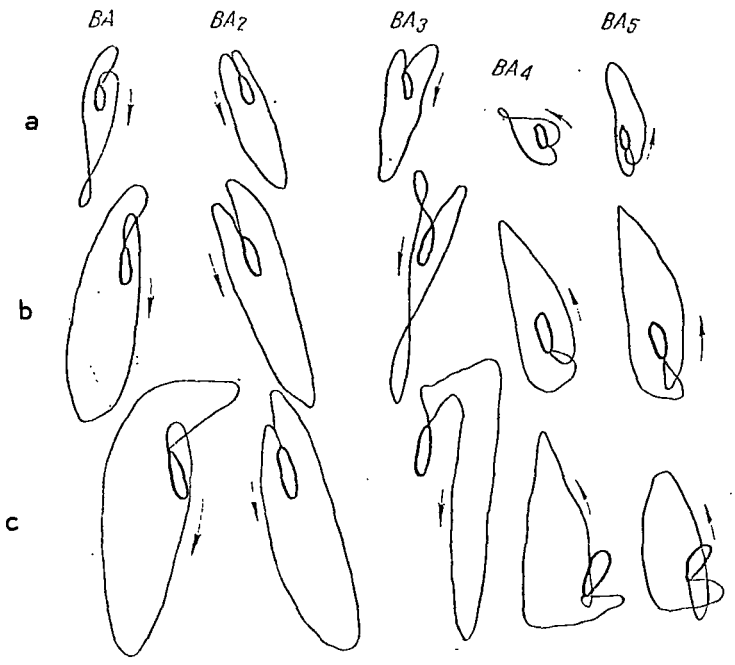


Figure 36. Vector Cardiograms of Three Athletes. a, B, 19 years old, class I boxer; b, Ah, 19 years old, class II skier; c, K, 20 years old, class I, academic rowing.

of the myocardium of the left ventricle, overstress of the myocardium as a result of a sharp increase in arterial pressure in acute nephritis and hypertension (Goldberger, 1951).

Similar changes in the electrocardiogram in the right precordial leads may be caused by overstress of the muscles of the right ventricle, arising upon sharp increase in the pressure in the respiratory circulation (for example in case of pulmonary artery embolism). Sclerotic changes in the myocardium, as well as disruptions in coronary blood flow, may be seen on the electrocardiogram in the form of the changes in the end portion of the ventricular complex described above.

Considering the possibilities of vector cardiography in the diagnosis of the diseases described above, causing similar changes in the electrocardiogram, we performed vector cardiographic investigation of 60 athletes for the purpose of differential evaluation of these changes in the end portion of the ventricular complex. These were active athletes of young age with various sports specialties and ratings.

It was found that in 15 persons showing these changes in the right precordial leads, the vector cardiogram showed no essential deviations from the norm; only a slight reduction in the amplitude of the T loop was noted /158 (within the limits of the norm) in the BA_4 lead. Upon inhalation, the amplitude of the T loop was increased, and the changes in the electrocardiogram normalized. This proves the dependence of these changes in the T waves

in the V_1-V_3 leads on the position of the heart in the rib cage; consequently, they are variants of the norm.

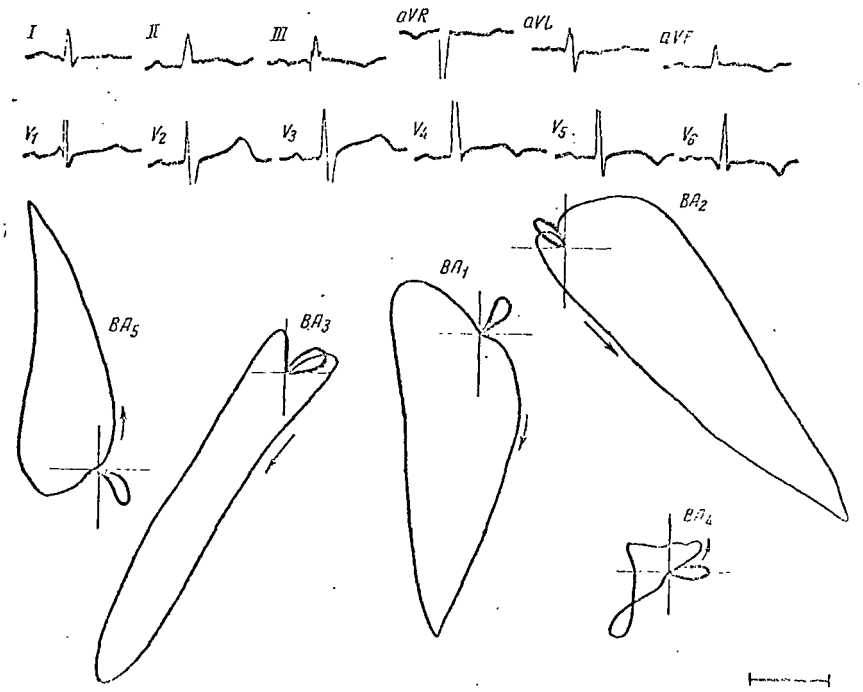


Figure 37. Electro- and Vector Cardiograms of Athlete V, 22 Years Old

In five athletes, the changes in the end portion of the ventricular complex in the left precordial leads resulted from pathological hypertrophy of the left ventricle, since they showed stage III of development of hypertrophy of the left ventricle (increase in summary area of QRS loops, nonclosure of QRS and T loops, with end point of QRS loop located in quadrants II and III with displacement of T loop into the same quadrants). In five athletes, the changes in the electrocardiogram were found to be manifestations of myocarditic cardiosclerosis, principally of rheumatic etiology. The vector cardiograms of these athletes, in addition to displacement of the T loops in the coordinate system (which is a manifestation of disruptions of the processes of repolarization) also showed the intraventricular blockage syndrome, which is a valuable criterion in the diagnosis of cardiosclerosis. /159

Seven athletes showed insufficient coronary blood supply. These athletes complained of stabbing or constrictive pains in the area of the heart resulting from overtraining or intensive loads in combination with

disruption of the rest regime, intensive study, training or participation in competition during or after sickness. The vector cardiograms of four athletes showed characteristics of insufficient coronary blood supply in the area of the apex cordis -- the T loop was displaced into quadrant II or III in the BA_4 lead, one athlete showed only a reduction in the T wave in the aVF lead on the electrocardiogram, while the clinical picture was characteristic for coronary insufficiency. The electrocardiogram of another athlete contained indications of deficient coronary blood supply only in the area of the rear wall, while the vector cardiogram showed deficiency of coronary /160
blood supply on the front left wall of the left ventricle as well. In three athletes in this group, the vector cardiogram showed coronary insufficiency in the form of nonclosure of the QRS and T loops and the location of the end point of the QRS loop in quadrants II and III.

In 25 athletes, these changes in the electrocardiogram were evaluated as a manifestation of dystrophic changes as a result of overstress of the left ventricle, arising as a result of incorrect or inefficient training without any organic changes in the heart muscle and coronary vessels. Timely detection of these changes and cessation of training causes the electrocardiogram and vector cardiogram to normalize. In many cases, normalization does not occur. The suggested reasons for dystrophy of the myocardium resulting from overstress of the left ventricle are either disruption of the electrolytic equilibrium (hypopotassemia), or a disruption of the relationship between neurohormones of the sympathetic and parasympathetic nervous system, predominance of sympathetic catecholamines, or Selier cardiopathy (ESCN) (see Chapter XIII). However, final solution of the problem of the reasons for these changes requires further investigation.

The electro- and vector cardiograms of two athletes presented below illustrate the capabilities of vector cardiographic investigation in differential evaluation of changes of the end portion of the ventricular complex of the electrocardiogram.

Athlete V, 22 years old, is a class I skier (Figure 37). The electrocardiogram indicates changes in the area of the posterior lateral wall of the left ventricle: negative T wave in II, III, aVF and left precordial leads, while form of T waves indicates insufficient coronary blood supply: they are sharp and equilateral. Vector cardiographic investigation indicates pathological hypertrophy of left ventricle, which is indicated by the significant increase in mean value of summary QRS loop area for patients with hypertrophy of the left ventricle and displacement of T waves in coordinate systems in all leads.

For athlete G, master of sports in rowing, 24 years of age, the electrocardiogram (Figure 38) shows similar changes in the left precordial leads, smoothed T waves in lead II, a two-phase T wave in lead aVF. On the vector cardiogram we see normal summary area of QRS loops, plus displacement of the T loops into the II quadrant in leads BA_1, BA_3, BA_4, BA_5, and into

quadrant III in lead BA_2, which indicates disruption of the repolarization processes (Figure 38, a). On the electrocardiogram (Figure 38, b) two months after a considerable reduction in load, we see normalization of the changes, which were a result of overstress of the left ventricle.

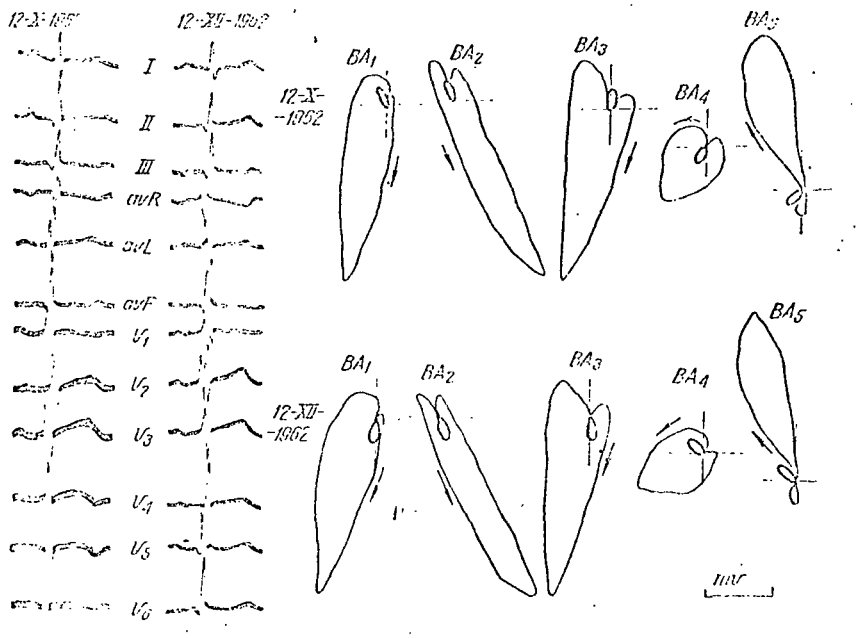


Figure 38. Electrocardiogram and Vector Cardiogram of Athlete G, 34 Years Old, Master of Sports Rowing.

In conclusion we note that the vector cardiographic method of investigation does not replace electrocardiography. Vector cardiography essentially supplements and expands the capabilities of electrocardiography, and should be widely used in sports medicine.

CHAPTER VI

MECHANICAL MANIFESTATIONS OF CARDIAC ACTIVITY AND THE FUNCTIONAL STATE OF THE VESSELS

ABSTRACT. Various methods for analysis of the mechanical manifestations of cardiovascular activity are described and analyzed. Methods include phonocardiography, dynamocardiography, vector dynamocardiography, Ballistocardiography, kinetocardiography, rheocardiography, tachooscillography, and various methods of sphygmography. Each method is described and diagrammed, case histories are presented, including illustrations of the various cardiograms and sphygmograms, and methods of mathematical analysis of the significance of phemenona revealed by the various methods are presented.

The mechanical manifestations of cardiac activity are quite varied. /161
Suffice it to say that the range of cardiogenic vibrations stretches from 0.6 to 1000 Hz. The infrasonic vibrations (0.7-16 Hz) arise primarily as a result of the kinematics of the heart in the thorax, and also as a result of the interaction of the movements of the heart with the human body as a mass. The sound frequency oscillations arise primarily in relation to stresses on the heart valves and the myocardium itself. All of these mechanical processes are recorded by a large number of methods. This chapter presents only those methods which are currently used in sports medicine. In this chapter we also thought it expedient to present information on methods of investigation of the large vessels.

Phonocardiography

At the present time, the place of phonocardiography among the other methods of investigation of the cardiovascular system has become precisely determined. Having a number of doubtless advantages over auscultation (objectivity, production of a documentary record, and possibility of recording sound phenomena which can be but poorly perceived by the ear), phonocardiography has become quite significant principally in the diagnosis of heart defects and to a certain extent in the evaluation of the functional state of the myocardium.

The idea of graphic recording of the sounds accompanying heart contrac- /162
tions appeared at the end of the nineteenth century. At that time, first Hurthle (1893), then Einthoven and Geluk (1894) recorded phonocardiograms. However, the broad usage of phonocardiography in clinical practice became

possible only considerably later, after improved electroacoustical apparatus was perfected.

Techniques of phonocardiography. The principal elements of modern phonocardiography are the microphone, amplifier and recording system.

Using a microphone placed over certain parts of the wall of the thorax, the oscillations arising in the heart are received and transformed into electrical oscillations. Further, the electrical signals are fed to an amplifier, where after amplification and frequency selection they are recorded by the recording device in the form of a curve -- the phonocardiogram.

At the same time as the phonocardiogram, it is necessary to record an electrocardiogram. Special requirements are placed on the phonocardiographic strip chart recorder -- its galvanometers must operate at high frequencies. Among such chart recorders are those which use photographic recording, as well as those with jet galvanometers. Pen drawing strip chart recorders are not suitable for the recording of phonocardiograms, since they have comparatively low natural oscillating frequencies.

These demands are satisfied by the domestically produced two-channel phonoelectrocardiograph (FEKP-2), which is broadly used at the present time.

The recording of phonocardiograms (PKG) is performed with the test subject in a horizontal position with the breath held after full exhalation. The microphone is attached to the surface of the thorax in sequence in the areas of projection of the apical thrust, aorta, pulmonary artery, tricuspid valve and Botkin's point. The quality of the phonocardiogram depends to a great extent on proper application of the microphone -- hermetic sealing and the pressure with which it is pressed against the thorax. The recording of a phonocardiogram at the points indicated is performed using the following frequency characteristics (according to Maass and Weber, 1952): the auscultative -- A, low-frequency -- H (35 Hz), first middle frequency -- C_1 (70 Hz), second middle frequency -- C_2 (140 Hz). Under certain conditions, phonocardiograms may be recorded with high-frequency characteristics -- B_1 (250 Hz).

Depending on the purpose of the investigation, the volume of investigation may be decreased or increased. Valuable supplementary data are produced by recording the phonocardiogram while lying on the side, standing, after physical loading, or while holding the breath at various phases, etc. (R. B. Minkin, 1959; A. I. Kolbents-Mishke, 1960).

Tones of the heart in athletes. The phonocardiogram of a healthy person /163 has been rather fully studied. However, the existing norms for phonocardiograms for healthy persons not involved in sports (R. B. Minkin, 1959; Rappaport, Sprague, 1942; Luisada, Mendoza, Alimurung, 1949; etc.) cannot be

fully transferred to sports medicine. The heart of the athlete differs in a number of physiological specifics, which are reflected in particular in the phonocardiogram.

A special study of the phonocardiograms of athletes was made by Ya. I. Ul'brikh (1960), V. V. Solov'yev and V. V. Bulychev (1962), R. D. Dibner (1960), etc.

Two groups of oscillations are always observed on the phonocardiograms of athletes. These are tones I and II of the heart, corresponding to the beginning of the mechanical systole and the beginning of isometric relaxation.

Up to the present time, there is still no single opinion concerning the origin of tone I of the heart. It is formed by a complex mechanism of muscle and valves. Three[1] principal parts of the first tone are differentiated on the phonocardiogram: the initial, central and terminal portions (Luisada, Mendoza, 1959; etc.).

The initial part of the I tone is a low-frequency tone. It consists of 1-2 waves of low amplitude, beginning at the same time as the Q wave of the electrocardiogram or following it slightly. The central portion of tone I results from stress on the parts of the atrioventricular valves, arising as a result of an increase in intraventricular pressure. It consists of high-frequency and comparatively low-amplitude oscillations. The terminal portion of tone I consists of 1-2 small, high-frequency oscillations which, it is assumed, result from stress on the walls of the large vessels as blood is expelled into them. The frequency range of oscillations of tone I is 30-120 Hz.

The second tone of the heart results from the closure and stress of the semilunar valves of the aorta and pulmonary artery. The beginning of tone II corresponds to the end of the T wave of the electrocardiogram approximately (±0.04 sec); tone II can be divided into two components: the first or aortal and the second or pulmonary. The frequency range of the oscillations of tone II is 70-150 Hz.

One of the principal indicators characterizing the tones of the heart is their duration. Data which we have produced in a study of the duration of heart tones of 120 athletes are presented on Table 10 as statistically processed.

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[1] In his later works, Luisada analyzed a four- and six-component structure of the I tone of the heart, while in 1964 he once more analyzed the structure and genesis of the I tone -- Ed.

TABLE 10. DURATION OF TONES I AND II OF HEART IN ATHLETES ACCORDING TO PHONOCARDIOGRAM

Point of placement of microphone (projection)	Frequency characteristic	Duration of tone I M (sec)			Duration of tone II (sec)		
		Lying	Standing	After physical load	Lying	Standing	After physical load
Apex of the heart	H	0,13±0,02	0,13±0,02	0,13±0,02	0,09±0,02	0,08±0,02	0,09±0,02
	C_1	0,11±0,02	0,11±0,01	0,11±0,01	0,09±0,01	0,08±0,01	0,09±0,01
	C_2	0,08±0,01	0,10±0,01	0,10±0,02	0,08±0,01	0,08±0,01	0,08±0,01
	A	0,10±0,01	0,11±0,02	0,10±0,02	0,07±0,01	0,07±0,01	0,07±0,01
Aorta	A	0,08±0,01			0,07±0,01		
Pulmonary artery	H	0,11±0,02	0,12±0,02	0,11±0,02	0,08±0,02	0,09±0,02	0,09±0,02
	C_1	0,11±0,01	0,10±0,02	0,11±0,02	0,08±0,01	0,08±0,01	0,08±0,02
	C_2	0,08±0,02	0,08±0,01	0,08±0,01	0,07±0,01	0,07±0,01	0,07±0,01
	A	0,11±0,02	0,10±0,01	0,09±0,01	0,07±0,01	0,07±0,01	0,07±0,01
Tricuspid valve	A	0,11±0,02			0,07±0,01		
Botkin's point	A	0,10±0,01			0,07±0,01		

Tr. Note: Commas indicate decimal points.

The duration of the tones of the heart depends on the point of recording and the frequency characteristic. When recording in the low- and first middle (C_1) frequency ranges, the duration of the tones is increased due to the better recording of low-frequency oscillations. /164

In comparing the duration of heart tones in athletes and healthy persons of the same age not occupied with sports (R. B. Minkin, 1959), greater duration of the heart tones in athletes was noted (by 0.01-0.04 sec).

When a transition is made from the horizontal position to the vertical position, the duration of the tones increases significantly. After physical loading (two minutes running in place at 180 steps per minute) the duration of the tones remains unchanged. The absence of any clear changes in the duration of tones I and II after loading in healthy persons was noted by Borter and Muller (1955).

The amplitude of the tones of the heart depends on the thickness of the chest wall, the thickness of the air layer between the heart and the chest wall (which in turn

depends on the phase of respiration), the position of the body of the test subject and a number of other factors. Therefore, there are considerable individual variations in the amplitude of the heart tones[1].

A comparison of the amplitudes of tones I and II showed various relationships at the apex and at the base of the heart. In the area of the apex of the heart, the amplitude of tone I was less than the amplitude of tone II in more than half the athletes. At the same time, in healthy persons who are not athletes the amplitude of tone I generally exceeds that of tone II or is equal to it at the apex of the heart (V. V. Solov'yev, 1957). /165

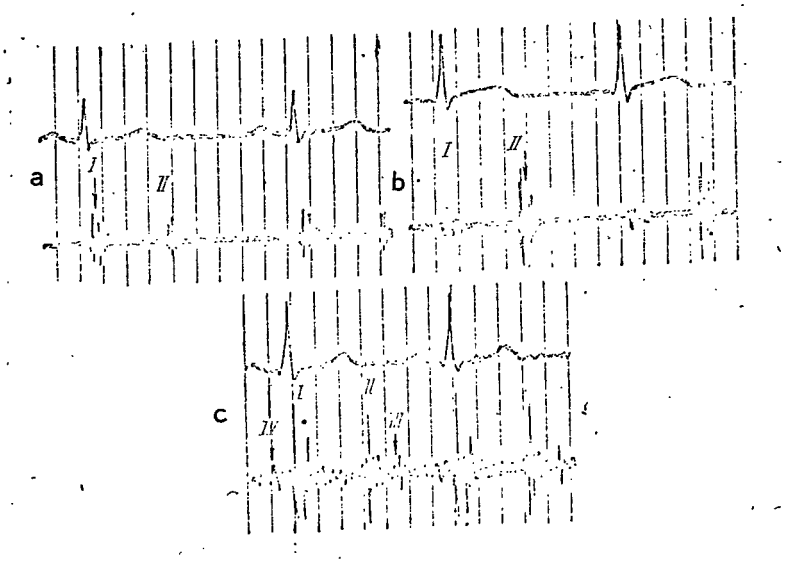

Figure 39. Phonocardiogram of Athlete M, 18 Years Old. When recorded with characteristic C_2 in the prone position in the area of the apical thrust, splitting of Tone I (a) was recorded, while in the area of the pulmonary artery -- splitting of tone II (b) was noted; c, phonocardiogram of athlete I, 17 years old; at Botkin's point, III and IV tones of heart were recorded; recording made with characteristic C_1.

The changes in tone amplitudes after physical loading and changing of the position of the body are irregular in nature: the amplitudes of the tones of the heart may increase, decrease or remain the same.

Splitting of tones I and II in healthy young persons is a frequent auscultative phenomenon, depending on nonsimultaneous closure of the atrioventricular or semilunar valves (Leathem, 1957; Luisada, Aravanis, 1957).

On the phonocardiogram, splitting of tone I is manifested as a decrease in the amplitude between two groups of oscillations in the central portion of tone I. The duration of the interval which arises is 0.01-0.02 sec (Figure 39, a). This brief interval between the two portions of tone I will not be perceived by auscultation. Therefore, Holldack (1952) /166

[1] The principal difficulty in amplitude analysis of phonocardiograms is the absence of any generally accepted method for calibration of the height of oscillations on the curve -- Ed.

emphasizes that the splitting of tone I perceived by ear (in contrast to the splitting of tone II) cannot be looked upon as a physiological phenomenon.

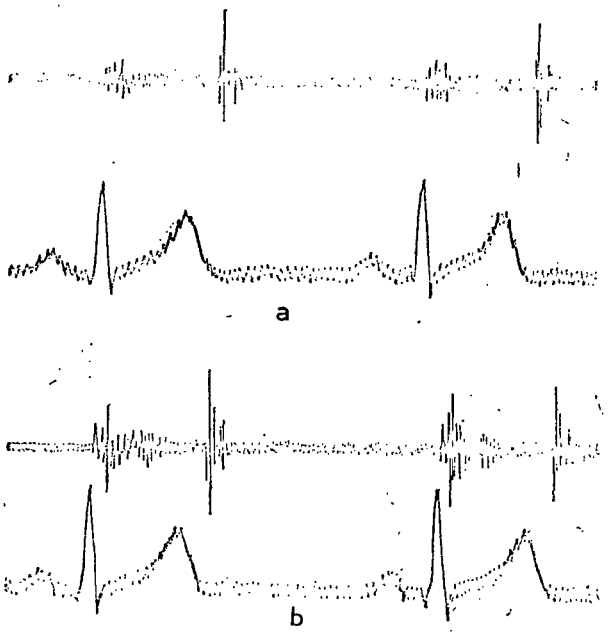

Figure 40. Phonocardiogram of Athlete K, 22 Years Old. In the prone position, splitting of tones I and II was noted (a); after physical loading (b), splitting of tones was not noted. Recorded with auscultative characteristic at Botkin's point.

The splitting of tone II, resulting from essentially nonsimultaneous closure of the valves of the aorta and pulmonary artery, appears graphically as does the splitting of tone I, with an interval between the two groups of oscillations not exceeding 0.03 sec (Figure 39, b). The splitting of tone II is frequently observed upon deep inhalation.

Phonocardiographic examination of athletes in the prone position has shown splitting of tone I in 11.6% and splitting of tone II in 10% of the test subjects. The splitting of tone I is observed primarily during the recording of the phonocardiogram in the area of the apex of the heart and Botkin's point, while the splitting of tone II is noted at the base of the heart, corresponding to data noted in healthy young persons. After transition to the vertical position, the frequency of splitting of tone I increases in comparison to the data in the prone position. Physical loading causes almost complete disappearance of the splitting of both tone I and tone II of the heart noted in the rest state (Figure 40). This fact can apparently be explained by more synchronous contraction of both ventricles and closure of the atrioventricular and semilunar valves.

The third and fourth tones of the heart are observed in athletes comparatively frequently. Thus, tone III of the heart, consisting of 1-2 small oscillations of low frequency, was recorded in 26% of the athletes (Figure 39, c). The interval between tones II and III fluctuates between 0.13 and 0.17 sec. After physical loading, tone III is recorded considerably more frequently than at rest -- in 78% of all athletes (Figure 41).

The reasons for the formation of tone III are interpreted variously. The most popular explanation is a theory, according to which tone III can be explained by the elastic reaction of the wall of the left ventricle resulting from rapid influx of blood from the atrium (Weber, 1956).

The fourth, atrial tone is formed of low-frequency vibrations arising 0.04-0.12 sec after the beginning of the P wave on the electrocardiogram or 0.08 sec before the beginning of tone I (Figure 39, c).

The fourth tone on the phonocardiogram is noted in the form of 1-2 low-frequency oscillations in 20% of athletes; in the standing position, it is less frequently recorded, while after physical loading, on the other hand, it is more frequently recorded.

We note that according to the data of V. V. Solov'yev and V. V. Bulychev (1962), splitting of tones I and II, as well as III and IV, is more frequently noted on the phonocardiograms of athletes than was the case in our observations.

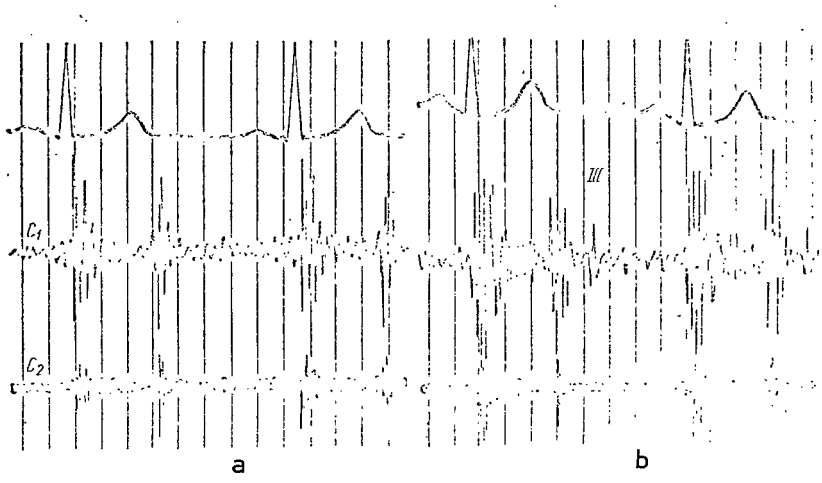

Figure 41. Phonocardiogram of Athlete A, 19 Years of Age. After physical loading (b) with recording in the area of the apical thrust with characteristic C_1, the III tone is clearly seen, which was not seen before loading (a).

Thus, the phonocardiogram of an athlete differs in a number of ways in comparison with the phonocardiogram of a healthy nonathlete. Characteristic features include greater duration of tones I and II, relatively lower amplitude of tone I at the apex of the heart, higher frequency of recording of tones III and IV. /167

In evaluating the functional state of the myocardium in clinical and sports medical practice, considerable significance is attached to the sound

of the tones of the heart. We know that dull tones are rather frequently observed in persons involved in sports, and therefore are looked upon as a specific of the "sports heart." According to the data of S. P. Letunov and L. G. Serkin (1939), dull tones in trained athletes are noted 5.5 times more frequently than in healthy persons not involved in sports.

The appearance of dull heart tones may result from a number of causes. Doubtless, a role is played by extracardiac factors (thickness of the anterior chest wall, emphysema of the lungs, etc.). The condition of the /168
vegetative innervation is of great significance. We know that an increase in the tonus of the vagus nerve leads to weakening of tone I of the heart due to the fact that the increase in intraventricular pressure is delayed.

In addition to these factors, dull heart tones may be a result of decreased contractive capacity of the myocardium due to heart muscle disease, heart defects, etc.

A proper evaluation of dull heart tones in general and in athletes in particular is of great practical significance. However, the capabilities of auscultation are limited. They do not allow us to differentiate mechanisms of the formation of dull heart tones (L. I. Fogel'son, 1932; S. P. Letunov and L. G. Serkin, 1939; R. Ye. Motylyanskaya and S. P. Letunov, 1949). In connection with this, the interpretation of the auscultative concept of "dull tones" frequently represents considerable difficulties.

From this point of view, attempts of a number of authors (R. B. Minkin, 1959; N. Ye. Kavetskiy and A. A. Stupnitskiy, 1961; McGregor, Rappaport, Sprague, Friedlich, 1956) to use phonocardiography to evaluate the sounds of the tones of the heart are quite interesting.

In order to develop differential diagnostic criteria for evaluation of the mechanisms of formation of dull tones of the heart, we performed phonocardiographic and polycardiographic investigation of 30 athletes with dull heart tones and 30 athletes with clear heart tones (control group). Among /169
those studied were representatives of various types of sports with high qualifications (master of sports and first class) varying in age from 18 to 35 years.

Clinical examination showed no deviations in the state of health and functional state of the athletes in the control group. As concerns the group of athletes with dull heart tones, the group was varied in its clinical characteristics: 20 of the men were judged healthy (first subgroup), while 10 (second subgroup) had or might have had irregularities in the cardiovascular system (rheumatism in anamnesis, chronic tonsillitis with frequent aggravation, electrocardiogram irregularities, complaints of dyspnea and pains in the area of the heart).

Recording of phonocardiograms was performed both at rest and after physical exercise (two minutes running in place).

Analysis and statistical processing of phonocardiographic data showed that the auscultative concept of "dull tones" on the phonocardiogram corresponds to a reduction in the amplitude of oscillations primarily of tone I of the heart. The mean amplitude of tone I in athletes with dull heart tones was considerably less than in persons with clear tones when recorded at all frequency characteristics. However, the most indicative in this respect were the auscultative and second middle frequency (C_2) characteristics, recordings at which for persons with dull tones showed amplitudes of tone I 2-2.5 times less than in athletes with clear heart tones, indicating the low frequency of the dull tones.

Essential differences were also found in a study of the relationships of amplitudes of tones I and II, particularly when recorded with characteristic C_2. In athletes with dull tones of the heart, the amplitude of tone I, as a rule, was less than the amplitude of tone II, while in athletes with clear tones, predominance of tone I over tone II or equal amplitudes of these two tones are the rule.

A demonstration of the above is the phonocardiogram of athlete T, 33 years of age, master of sports in sailing, and athlete I, 22 years of age, master of sports in rowing. Auscultation of T indicated dull heart tones. The phonocardiogram showed low amplitude of tone I, particularly with characteristics A and C_2 (Figure 42). In I, the tones of the heart are clear, and the phonocardiogram shows high amplitude of tone I, exceeding that of tone II at all frequency characteristics (Figure 42).

Thus, phonocardiographically, dull heart tones are distinguished from clear heart tones by low absolute values of tone amplitudes, different relationships between tones I and II at various frequency characteristics. These indicators can be used for objective evaluation of the sounds of the heart tones. However, they do not help to solve the problem of the causes of dullness of the tones. /170

In order to explain the role of the tonus of the vagus nerve in the formation of dull heart tones, we can use data from phonocardiographic investigation after physical loading. We know that under the influence of physical loading, the tonus of the sympathetic nervous system is increased with simultaneous weakening of the central tonus of the vagus nerve. All of this, as we know, facilitates an increase in the rate of rise of the intraventricular pressure and therefore a reinforcement of the tones of the heart. The performance of this type of investigation has allowed us to establish that in 70% of athletes with dull heart tones, an increase in the amplitude of tone I is observed after physical loading.

What are the reasons for dull heart tones in those athletes for whom no increase in the amplitudes of the heart tones is observed after physical loading? The answer to this question can be obtained by a comparison of the the data of phonocardiographic and polycardiographic investigations. /171

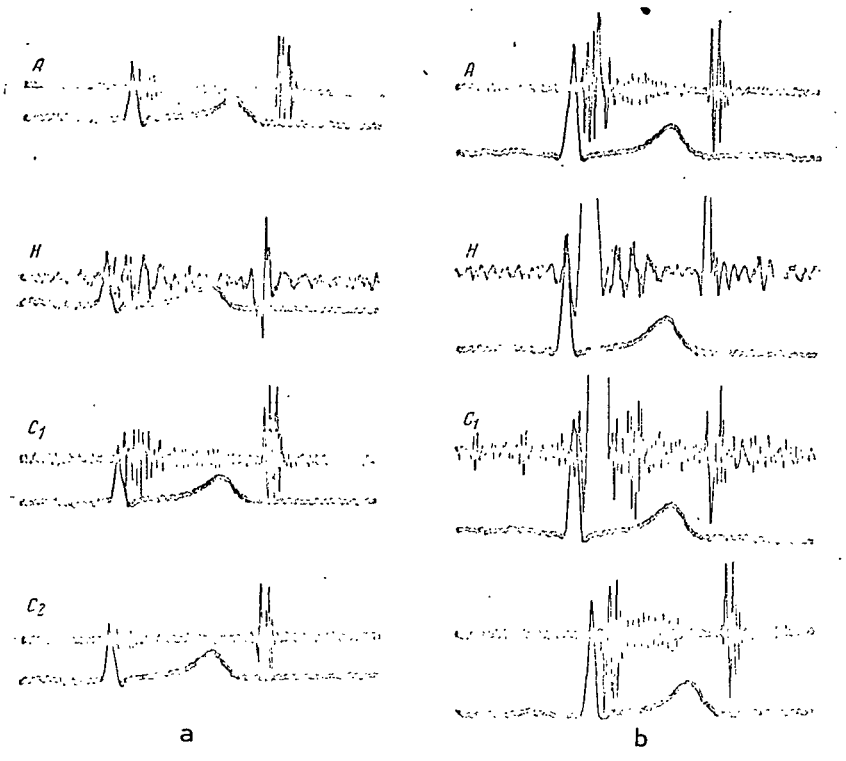

Figure 42. Phonocardiograms of Athletes. a, T, 33 years; b, I, 22 years. Recorded in area of apical thrust. Explanation in text.

In comparing the results of phase analysis in two subgroups of athletes with dull heart tones, it was established that in clinically healthy athletes (first subgroup) the duration of the phases of the cardiac cycle correspond to values in a control group (athletes with clear heart tones). In athletes in the second subgroup, an elongation of the isometric contraction is observed, along with a shortening of the expulsion period.

These changes in the duration of phases of the cardiac cycle, as has been shown by physiological and clinical investigations performed by a number of authors, can be looked upon as a manifestation of disruptions in the contractive function of the myocardium. In these cases there is sufficient reason to explain the dullness of the heart tones as changes in the cardiac muscle and resulting hypodynamia of the myocardium.

Functional noises in athletes. One characteristic of auscultative specifics of the athlete's heart is frequent presence of systolic noise of a functional nature. The particular frequency of systolic noises revealed during auscultation of athletes has been described by D. F. Shabashov (1930)

(40-50%), R. Ye. Motylyanskaya and S. P. Letunov (1949) (up to 39%), Deych and Kauf (1926) (25.9%), etc. The nature of the systolic noise in each individual case requires differential evaluation, since it is quite clear that a person may be involved in sports and still have various valve defects of the heart. This is particularly true of insufficiency of the mitral valve -- a defect with which the physical working ability is retained for some time and in some cases high sports results are achieved. However, in spite of the good compensation of this heart defect, a definite opinion has been gathered at the present time that the presence of mitral valve deficiency is incompatible with systematic sports activity.

In solving the question of the functional nature of systolic noise during auscultation, we usually base ourselves on such characteristics as the gentle nature and low intensity of the noise, as well as its inconstancy and variability in strength, extent and localization as a function of the phase of respiration, position of the body, physical stress, nervous-mental influences, etc. However, it should be emphasized that some of the auscultative characteristics described for functional systolic noise have relative value.

The diagnosis of functional systolic noises using phonocardiographic /172
methods is considerably more effective. A number of foreign works have appeared (Evans, 1947; Luisada et al., 1957; Besterman, 1955; Wittig, 1956; Wells, 1957), dedicated to phonocardiographic investigations of the functional systolic noises.

Systolic noise on the phonocardiogram shows a picture of comparatively high-frequency oscillations (oscillating frequency from 70 to 600 Hz) located between tones I and II of the heart (Figure 43).

The usage of phonocardiography in sports medical practice has revealed a very high frequency of registration of systolic noise in athletes. V. V. Solov'yev and V. V. Bulychev (1962) noted systolic noise on the phonocardiogram in 84% of those athletes whom they examined. According to our data, the frequency of appearance of functional systolic noise in athletes is even greater -- amounting to 92%.

We performed a special study of the phonocardiograms of 112 athletes with functional systolic noise. The phonocardiograms were recorded with the test subject in the prone and standing positions and after standard physical loading with four frequency characteristics from the usual points of auscultation of the heart.

In 60% of the athletes, the clearest expression of functional systolic noise was noted in the area of the apex of the heart and in 40% -- in the area of the pulmonary artery.

The characteristics of functional systolic noise include a damping nature of the noise (decrescendo), clearest recording at moderate frequencies

(C_1) and low amplitude of oscillations, not exceeding one half the amplitude of tone I as a rule (see Figure 42). One distinguishing feature also is the short duration of the noise -- occupying from one fourth to one half of the length of the systole. A late beginning of the functional systolic noise is particularly typical -- 0.03-0.08 sec after tone I. Evans (1947) and Wells (1957) turned particular attention to this phonocardiographic characteristic of functional systolic noise.

The comparison of phonocardiographic data recorded in the prone and standing positions has confirmed that the intensity of functional systolic noise decreases as the body is moved to the vertical position. This has been documented by a decrease in the amplitude of the oscillations of the noise.

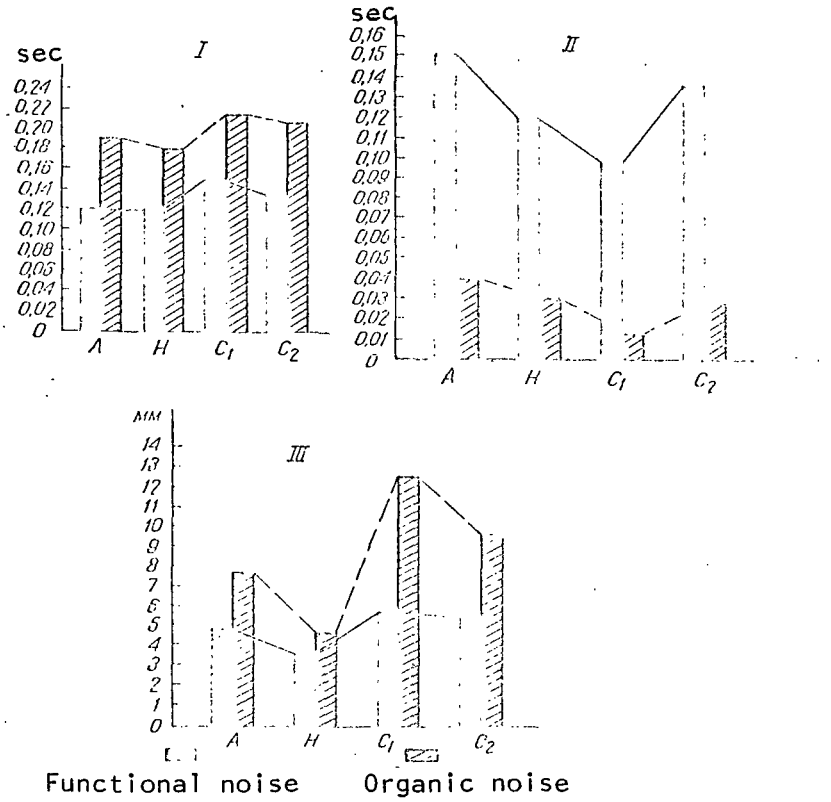

Figure 43. Comparative Characteristics of Functional and Organic Systolic Noises (Average Figures). I, duration of systolic noise; II, duration of interval between noise and tone II; III, amplitude of systolic noises. A, H, C_1, C_2, frequency characteristics.

Since there is no single opinion concerning the influence of physical stress on functional systolic noise, an objective phonocardiographic) evaluation of the nature of systolic noise after physical loading should be of great practical interest. The investigations performed allowed us to state that after physical loading, the amplitude of systolic noise increases significantly in all athletes. This indicates reinforcement of the noise and /173 in turn can be explained by a reinforcement of the contraction of the ventricles (V. V. Antipov, 1956) and an increase in the rate of blood flow. Consequently, the existing assumption that functional systolic noise becomes quieter after physical loading is contradicted by the data of phonocardiographic investigation. Similar observations are presented by V. V. Solov'yev and V. V. Bulychev in their work (1962).

A demonstration of the characteristics of functional systolic noise can /174 be seen from the following observation.

K, 22 years of age, is a class I fencer. He has been involved in the sport for five years. He has no health complaints. Upon auscultation, soft systolic noise is heard at the apex of the heart, reinforced after physical loading. X-ray examination shows normal size and configuration of the heart. The electrocardiogram has no deviations from the norm. On the phonocardiogram (Figure 44, a), a short noise of low amplitude is seen, arising 0.04 sec after tone I and ending in the middle of the systole. After physical stress (Figure 44, b) the amplitude of the systolic noise sharply increases.

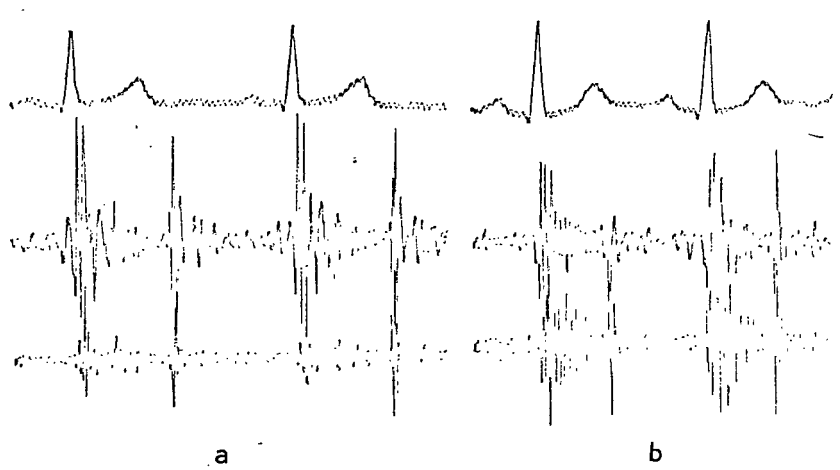

Figure 44. Phonocardiogram of Athlete K, 22 Years Old. Recorded in area of apical thrust in prone position. a, before physical loading; b, after physical stress. Explanation in text.

We must discuss specially the problem of changing intensity of functional systolic noise during the process of training. The greatest difficulties usually arise in evaluating those cases when a reinforcement of

systolic noise is noted with the passage of time. It is believed by many that this type of change of functional systolic noise is an unfavorable sign, indicating the development of fatigue or overtraining. Phonocardiographic study of this problem has shown certain interesting regularities contradicting this point of view.

A group of highly qualified rowers (42 masters of sports) investigated in dynamics during the course of the sports season showed that as the state of training increased and the functional state of most (70%) was improved, the phonocardiogram showed an increase in the amplitude of systolic noise, regardless of its point of localization. The noise amplitude is increased gradually, reaching its maximum in the competitive stage of the main training period. /175

As an example, we present below the phonocardiogram of a master of sports in rowing.

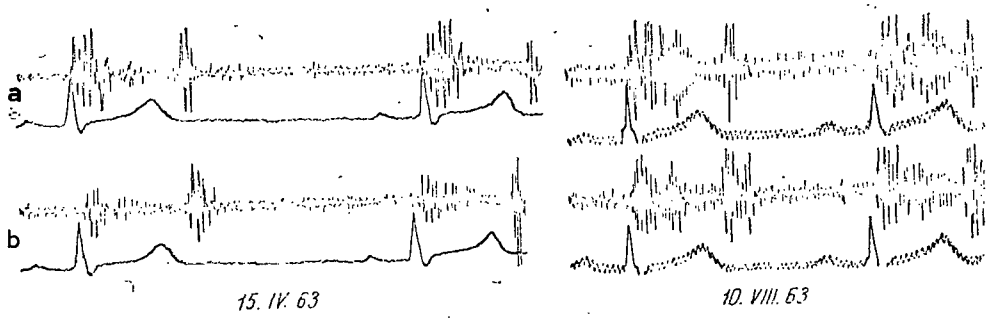

Figure 45. Phonocardiogram of Master of Sports G, 25 Years of Age. Recorded at characteristic C_1: a, In area of apical thrust; b, In area of pulmonary artery. Explanation in text.

G, 25 years old (Figure 45), healthy. Functional state throughout sports season good. On phonocardiogram of 15/4/1963 (Figure 45, a) a short, low-amplitude functional systolic noise is recorded in area of apex of heart and pulmonary artery. On the phonocardiogram of 10/8/1963 (Figure 45), the amplitude of systolic noise is sharply increased. During this period, the athlete took part in important competition successfully.

One characteristic feature is a dependence of these changes on age and sports ranking of the athlete. Young athletes with relatively low sports ranking usually show this dynamics in the change in amplitude of noise during the process of training quite clearly. Experienced athletes 28-30 years of age show an increase in the amplitude of systolic noise comparatively less less frequently, and it is expressed to a lesser extent.

On the basis of experimental data of V. V. Antipov (1963), it might be assumed that the increase in the amplitude of systolic noise in the process

of increasing state of training results from reinforcement of the contraction of the ventricles of the heart. Also, an increase in the tonus of the vagus nerve, most clearly seen in the process of sports training in young athletes, is probably of great significance.

In conclusion to this section, we must discuss once more one problem /176
which has not been discussed in the sports medical literature. We are concerned with the functional diastolic noises in athletes.

Diastolic noises are high-frequency oscillations (frequency range from 120 to 600 Hz according to Schmidt-Voigt, 1955), located between tones II and I of the heart.

Clinical observations (A. I. Arinkin, 1926; M. Ya. Ar'yev, 1948; B. N. Turusevich, 1955; M. Yu. Rappaport, 1957; Z. K. Chkhaidze, 1963; etc.) clearly prove the existence of diastolic noises with certain diseases, not accompanied by organic damage to the valve apparatus of the heart, but leading to a decrease in the tonus of the cardiac muscle.

In addition to that, in the literature in recent years we have seen individual reports of the possibility of appearance and phonocardiographic recording of diastolic noises in practically healthy persons (Paulin, Mannheimer, 1957; Wolf, 1961) and in healthy persons with labile vegetative nervous system L. M. Fitileva, 1962).

In large scale phonocardiographic investigations of athletes at rest (250 persons) we did not record diastolic noise in any case. However, in recording the phonocardiograms after physical loading (two minutes running in place) in eleven athletes we discovered diastolic noise. Clinical examination negated the presence of pathology of the cardiovascular system of these persons. Also, in eight of the eleven persons a short functional systolic noise was noted auscultatively and phonocardiographically, which increased after physical loading. In the other three persons after physical loading, systolic noise was also present. It is characteristic that these were women between 17 and 23 years of age, involved in various types of sports for 3-5 years, who had achieved high sports qualifications during this time.

The appearance of diastolic noise in healthy persons only after physical stress, the absence on the phonocardiogram of a click upon opening of the mitral valve and normal values of Q-tone I interval, i.e. signs characteristic for mitral stenosis in which diastolic noise is regularly heard, give us reason to evaluate this noise as functional. Phonocardiographically, they can be characterized by a number of indicators: diastolic noises are primarily recorded at Botkin's point, in time they arise at the beginning of the diastole, the duration of the noises is from 0.12 to 0.25 sec (corresponding to 1/6-1/3 of the duration of the diastole). The interval between tone II and the beginning of the noise is nil or 0.03-0.08 sec. The amplitude of the noise is slight and always less than the amplitude of /177

tone II by 2-4-5 times. An illustration of this is the following observation.

K, 17 years old, has been involved in sports gymnastics for four years, and has first ranking. She is healthy. On the phonocardiogram at rest (Figure 46, a) there are no noises. The tones are of normal amplitude and duration. Q-I tone = 0.05 sec. After physical stress (Figure 46), the appearance of systolic and diastolic noises is noted. The latter arises directly after tone II, its duration is 0.18 sec, about one third of the duration of the diastole, its amplitude is 4 mm (one-fourth amplitude of tone I).

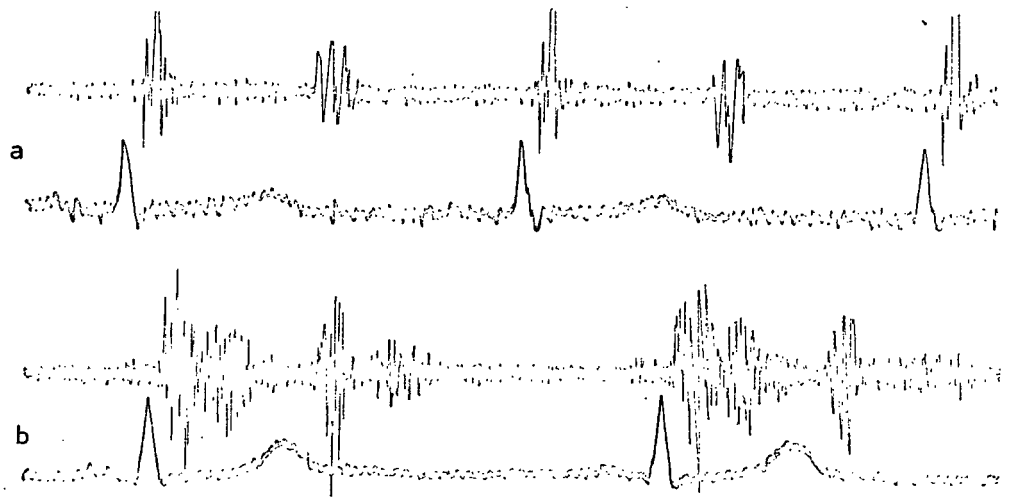

Figure 46. Phonocardiogram of Athlete K, 17 Years Old. Recorded with auscultative characteristic at Botkin's point: a, before physical loading; b, after physical loading. Explanation in text.

The explanation of the mechanism of formation of functional diastolic noise in athletes after physical loading involves considerable difficulties. Apparently, as is the case for the functional systolic noise, acceleration of the blood flow and the change in the tonus of the cardiac muscle, which depends on the tonus of the extracardial nerves, are of considerable significance.

Dynamocardiography

The development of electronic equipment which has occurred in the post-war years has allowed a number of new methods to be created for the investigation of the cardiac activity of man. Among such methods new in

principle is dynamocardiography, developed by Ye. B. Babskiy, T. S. Vinogradova, V. S. Gurfinkel', E. L. Romel' and Ya. S. Yakobson (1952). /178

Using this method, it has been found possible to analyze the mechanical processes accompanying the cardiac contraction. This type of analysis is performed by recording movements of the center of gravity of the thorax and the thrust components of the kinematics of the heart.

Dynamocardiographic techniques and methods of investigation. The dynamocardiograph consists of three independent parts: the receiving device, electronic amplifier and recording system (oscillograph). The main and original portion of its design is the receiving device, which receives forces from the thorax of the test subject and converts them into electrical signals.

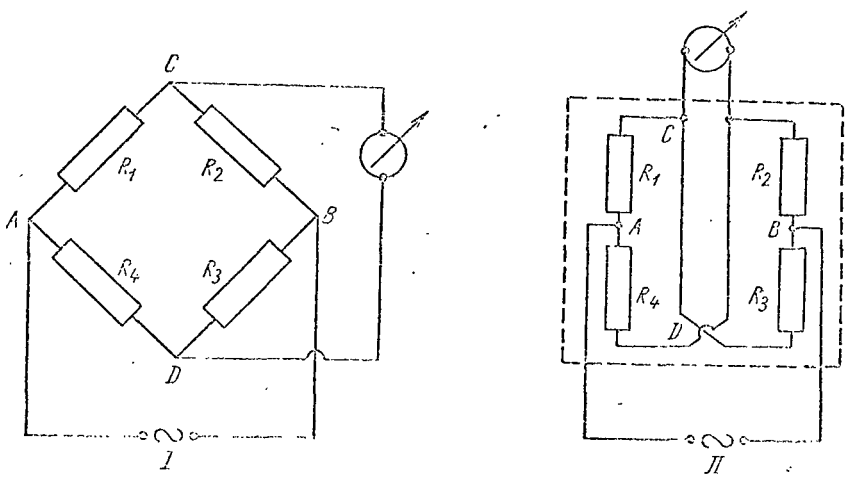

Figure 47. Wheatstone Bridge Circuit (after Ye. B. Babskiy and V. L. Karpman, 1963). I, Diagramof bridge; II, commutation of pickups of dynamocardiograph receiving device (shown by dotted line).

The receiver device has the form of a flat box and consists of two rigid metal or plastic plates 30 × 30 cm in size, between which there are elastic elements (steel rings, cantilever beams or balls) with wire tensometers attached to them (usually constantan).

The receiver device of the dynamocardiograph uses a differential bridge /179 circuit to connect the tensometers (Figure 47). The electric power supply of this bridge is with 4000 Hz ac from an undamped oscillator connected into the supply diagonal of the bridge.

The electrical circuit of the tensometric amplifier used in the dynamocardiograph differs from broadly used electrocardiographic amplifiers. This difference is determined by the fact that the amplifier performs

amplification of ac (carrier frequency) modulated in the receiver device. The amplified signal is then detected using a special phase-sensitive rectifier bridge, as a result of which a useful signal is produced at the output, recorded on the oscillograph in the form of a curve -- the dynamocardiogram.

At the present time, two types of dynamocardiographs are being series produced using models developed by members of the special design bureau "Biofizpribor" A. B. Venediktov and M. I. Tishchenko, together with Ye. B. Babskiy and V. L. Karpman (1960). The first variant is an independent three-channel device allowing synchronous recording of longitudinal and transverse dynamocardiograms and an electrocardiogram. The second variant is a ballistodynamocardiographic attachment to the five-channel electrocardiograph produced by the Orion firm (Hungary). A number of attachments are produced for this device, allowing synchronous recording of electrocardiogram, dynamocardiogram, ballistocardiogram, phonocardiogram, pulse oscillations in various vessels (sphygmogram and phlebogram), as well as the pressure in the cavities of the heart and main vessels.

Under actual conditions, loads which periodically change in magnitude and point of application act on the receiver device of the dynamocardiograph, these loads being made up of the mechanical effect of respiratory movements and mechanical processes accompanying the cardiac contraction.

The recording of processes arising during the work of the heart, during movement of the mass of the heart and the mass of blood make up the physiological essence of dynamocardiography. However, the recording of a dynamocardiogram requires that movements related to respiration be excluded.

The problem is that respiration influences the deflection of the dynamocardiograph recorder approximately 4-5 times more intensively than cardiac activity, which naturally makes analysis of the dynamocardiogram recorded during respiration more difficult. In order to eliminate the effects of respiratory movements, recording of dynamocardiograms is performed while the test subject holds his breath (usually after moderate exhalation).

Recording of a dynamocardiogram requires little time after some skill has been accumulated. The test subject is placed on his back so that the top edge of the receiver device is at shoulder level. Movement of the test subject relative to the receiving device changes the projection of the mechanical processes occurring within the thorax and therefore causes distortion of the dynamocardiogram. The bridge balancing adjuster is used to eliminate the influence of the weight of the thorax. /180

Then, balancing is adjusted with the breath held and the strip chart drive mechanism is started for the recording of the dynamocardiogram. The breath is held for 7-8 sec, allowing recording of 5-10 cardiac cycles. This is quite sufficient for objective analysis of the dynamocardiogram under quite varied conditions.

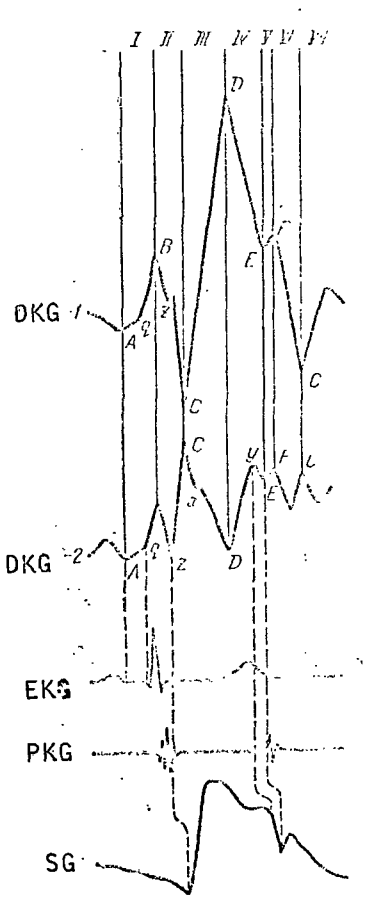

Figure 48. Time Comparison of Longitudinal (DKG-1), Transverse (DKG-2) Dynamocardiograms, Electrocardiogram (EKG), Phonocardiogram (PKG) and Carotid Artery Sphygmogram (SG).

In order to compare the results of investigations using various devices or using a single device recorded in various amplification modes, and also for quantitative evaluation of the indicators of cardiac activity, dynamocardiographs are calibrated. In calibration, a defined weight (usually 100 g) is moved by a given distance alternately along the longitudinal and transverse axes of the receiving device. The value of the calibration index (i) is determined as follows:

$$i = \frac{P \cdot l}{D}, \qquad (36)$$

where P is the weight of the load (in g), l is the distance over which the load is moved (in cm), D is the deflection of the oscillograph beam (in mm). The dimensionality of the calibration index is [g·cm/mm].

The amplitude equivalent of any sector on either the longitudinal or transverse dynamocardiogram is defined by multiplying /181 the height of a given sector (in mm) by the value of the calibration index. Calibration is performed after investigation. For example, when a load weighing 100 g is moved by 10 cm (load initially set at null point in the center of the receiver device at the intersection of the longitudinal and transverse axial lines) the oscillograph beam is deflected by 50 mm. The calibration index in this case is 10·100/50 = = 20 g·cm/mm. With a height of the dynamocardiograph sector being investigated at 40 mm, its amplitude is 800 g·cm, while if the height is 70 mm its amplitude is 1400 g·cm, etc.

Longitudinal dynamocardiogram. The recording of the moment of forces relative to the transverse "zero" line in the receiver device allows us to produce a longitudinal dynamocardiogram. This curve is complex, repeated with each heart contraction (Figure 48, 50).

The principal waves of the dynamocardiogram, corresponding to bend points of the curve, are represented with the Latin A, B, C, D, E, F, G, H, while the intervals between them are represented with Roman numerals. Points q, z, and x are also distinguished on the curve.

The overall picture of the longitudinal dynamocardiograms of healthy persons remains basically identical. The waves of the curve in the intervals I, III, V and at the beginning of interval VII are positive, while the waves in intervals II, IV, VI are negative. The elevation of the curve in interval I is frequently preceded by a short inclination. In intervals I, II and III, there are splits, represented by the letters q, z and x respectively (Figure 48). After elevation G-H (at the beginning of interval VII), a low-amplitude, aperiodic oscillation follows.

The picture of the dynamocardiogram is determined by displacement of the projection of the center of gravity of the thorax and changes in the value of the vertical load. Obviously, the dynamocardiogram reflects the thrust processes (atrial and cardiac thrusts), as well as phenomena related to movement of masses in the thorax (for example, displacement of masses of blood corresponding to systolic volume along the principal vessels, displacement of the mass of the heart during the cardiac cycle, etc.). On this basis, we might assume that certain waves of the dynamocardiogram are related to physiological processes occurring in the circulatory apparatus.

Synchronous recording of the dynamocardiogram with the curve of pressure in the right ventricle, esophagocardiogram, electrocardiogram, phonocardiogram and sphygmogram of the carotid artery have allowed us to determine the physiological essence of various dynamocardiographic waves (Ye. B. Babskiy, V. L. Karpman, 1963; V. L. Karpman and L. A. Ioffe, 1960; etc.). It /182
has been established that wave A corresponds to the beginning of the atrial systole. Wave q reflects the beginning of the contraction of the fibers of the myocardium. The absence of this wave in approximately one third of the observations results from the relatively low mechanical activity of the heart at the beginning of the general ventricular systole. Point B is synchronous in time with the beginning of the increase in intraventricular pressure and the beginning of high-frequency oscillations of tone I on the phonocardiogram. It, therefore, reflects the beginning of isometric contraction. Point z corresponds to the beginning of the anacrote on the curve of carotid pulse (considering the time of propagation of the pulse wave). This allows us to determine the beginning of the period of expulsion of blood using this point.

Investigations performed by Ye. B. Babskiy et al. (1953) give us reason to believe that the formation of wave C is related to the end of the inertial processes accompanying the cardiac thrust and the beginning of expulsion of blood from the cavities of the heart. These authors showed that the form and amplitude of the dynamocardiogram during the intervals I, II and V are determined by the thrust processes, while in the remaining intervals they are determined by displacement of masses in the human thorax.

The genesis of sector C-D depends primarily on the displacement of the mass of blood through the principal vessels. It is assumed that the bend in the curve at point D is related to the arrival of a portion of the systolic volume of blood at the carotid arteries and the decrease in the load on the receiving device resulting from this. It is also possible that wave D is formed in relation to a change in the direction of movement of blood expended by the heart into the aorta: as the blood moves along the ascending aorta, oscillations are recorded directed toward the head end of the device (sector C-D); the displacement of blood through the descending aorta is accompanied by oscillations directed downward (sector D-E). In one way or another, the principal influence on the formation of sector C-D is that of the displacement of masses of blood through the principal vessels. Under conditions of muscular work, as well as in certain forms of cardiac pathology, changes in the amplitude of sector C-D correspond satisfactorily with changes in systolic blood volume (V. L. Karpman, 1956; L. A. Ioffe, 1962).

Point E corresponds in time to the beginning of tone II and the lower point of the incisure in the carotid arteries on our sphygmogram (with correction for propagation time of pulse wave). Consequently, point E of the dynamocardiogram documents the moment of closure of the semilunar valves, or, which amounts to the same thing, the completion of the protodiastolic period. Since point E corresponds to closure of the semilunar valves, it becomes clear that the formation of sector E-F is related to the "impact" of blood against the closed semilunar valves.

The mechanism of formation of the sector of the curve in interval VI is /183 not yet quite clear.

The low-amplitude waves in interval VII apparently reflect the filling of the ventricles and atrium with blood.

Transverse dynamocardiogram. The transverse dynamocardiogram is produced by recording the moments of forces relative to the longitudinal "zero" line of the receiving device. The receiving device is oriented so that displacement of the load from left to right causes the oscillograph beam to deflect upward.

The picture of the transverse dynamocardiogram is more broadly differentiated than that of the longitudinal dynamocardiogram (Figure 48, 50). However, the individual variability of curves is less marked and essentially consists of varying depths of the z wave, height of the C wave, direction and depth of the D wave (the principal waves of the transverse dynamocardiogram are represented in the same way as the waves of the longitudinal dynamocardiogram).

The sector A-q of the transverse dynamocardiogram is usually two-phased, less frequently having the form of a smooth elevation. After the q wave, which is more frequently observed than on the longitudinal dynamocardiogram (85%), the steepness of the rise increases gradually. Point B is sometimes rounded or wavy. Frequently, the q-z sector is reminiscent of the letter M.

In these cases, point B is located in the "saddle." Thus, point B on the transverse dynamocardiogram is frequently less clearly expressed than on the longitudinal dynamocardiogram.

In healthy persons, the sector of curve B-z is usually directed sharply downward (sometimes after a brief elevation). The z wave is much more clearly expressed than on the longitudinal dynamocardiogram, and also corresponds in time to the beginning of the anacrote on the carotid pulse wave, thus reflecting the moment of opening of the semilunar valves and the beginning of blood expulsion.

At point z begins the steep rise in the curve which ends with the C wave. After this wave, the curve drops, the steepness of the descent usually decreasing slightly after point x. This wave corresponds in time to the splitting in the initial portion of the C-D wave of the longitudinal dynamocardiogram. Further descent of the curve ends at point D (frequently not corresponding in time with point D of the longitudinal dynamocardiogram), after which a slow rise begins. The peak of this rise (point y) corresponds to the beginning of the incisure on the carotid pulse curve and, thus, reflects the end of expulsion and the mechanical systole of the ventricles. The E wave corresponds to the beginning of tone II and the lower point of the incisure. After point E, a short rise in the curve is noted (sector E-F). The G wave was missing in a significant portion of the transverse dynamocardiograms. /184

The above allows us to conclude that longitudinal and transverse dynamocardiograms can be used to perform phase analysis of the cardiac cycle. Thus, the duration of the phase of asynchronous contraction can be determined on the basis of the q-B interval, reflecting the process of propagation of the contraction wave through the myocardium; the B-z interval allows us to determine the duration of the phase of isometric contraction; interval z-y corresponds to the period of expulsion, while interval y-E corresponds to the protodiastolic period. Synchronous recording of longitudinal and transverse dynamocardiograms facilitates the calculation of phase durations of the cardiac cycle (on the longitudinal dynamocardiogram the B wave, as a rule, is clearer in its configuration, while the gransverse dynamocardiogram shows the z and y waves more clearly). The latter does not appear in some observations of longitudinal dynamocardiograms.

Synchronous recording of longitudinal and transverse dynamocardiograms with electrocardiograms, phonocardiograms and sphygmograms of the carotid artery (Ye. B. Babskiy and V. L. Karpman, 1957; V. L. Karpman and L. A. Ioffe, 1961) have shown that phase analysis of the cardiac cycle performed using dynamocardiography produces results practically identical to the data produced on the basis of polycardiographic investigation.

Determination of the duration of the intervals on the dynamocardiogram is of considerable significance for evaluation of the contractive capability of the myocardium. V. L. Karpman (1956) showed that in healthy persons there

is a dependence between the duration of the intervals II and III plus IV and the cardiac rhythm. This dependence is described by the following formulas:

$$I_{II} = 0{,}081 \cdot \sqrt{C}, \quad \text{or} \quad I_{II} = 0{,}046 \cdot C + 0{,}035; \tag{37}$$

$$I_{III+IV} = 0{,}241 \cdot \sqrt{C}, \quad \text{or} \quad I_{III+IV} = 0{,}137 \cdot C + 0{,}106, \tag{38}$$

where I_{II} and I_{III+IV} are the duration of the intervals II and III plus IV respectively (in seconds); C is the duration of the cardiac cycle. Essential deviations of the actual duration of the intervals from the normal values indicate disruptions in the contractability of the myocardium and hemodynamics.

Vector dynamocardiogram. The mechanical processes accompanying cardiac activity and displacement of the blood in the vessels of the thorax have spatial orientation. It follows from this that a study of cardiodynamics using only one (longitudinal or transverse) "lead" of dynamocardiograms is insufficient.

Vector analysis of dynamocardiograms gives us a more complete accounting of the mechanical processes accompanying the cardiac contraction.

A curve characterizing the displacement of the equilibrium force applied /185
to the receiver device in dynamocardiography can be constructed by amplitude analysis of the longitudinal and transverse dynamocardiograms for each given moment. Deflections from the "zero" line on the longitudinal dynamocardiogram are laid out on the ordinate, while deflections from the "zero" line on the transverse dynamocardiogram are laid out on the abscissa. The "zero" line is passed through point A (both on the longitudinal and on the transverse dynamocardiograms). It is sufficient in practice to calculate deflections of the principal points. The graphic method of contructing vector curves requires a great deal of time, which prevents broad introduction of this method to daily practice.

The vector dynamocardiogram can also be recorded automatically using a cathode ray oscilloscope.

The vector dynamocardiogram is a complex curve (Figure 49), extended in the direction of the longitudinal body axis. The systolic portion of the curve consists of four loops: qBz, zCx, xDy, and yEF. Loop qBz characterizes the mechanical processes arising during the stress period. /187 Loop zCx corresponds approximately in time to the phase of rapid blood expulsion, while loop xDy characterizes the mechanical processes in the thorax accompanying the displacement of the mass of blood at the end of the expulsion period. Loop yEF is formed at the beginning of the diastole of the

heart in connection with the mechanical processes arising during closure of the semilunar valves.

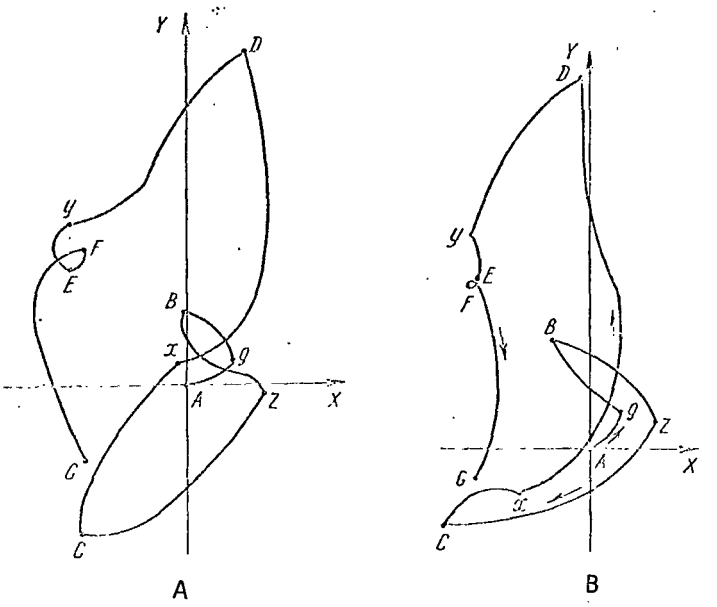

Figure 49. Frontal Vector Dynamocardiogram of Healthy Persons. A, untrained; B, athletes

A quantitative evaluation of the vector dynamocardiogram is performed in polar coordinates, for which the angle of inclination of the radius-vector in relation to the positively directed abscissa is determined, and the distance of the principal points to the center of coordination, i.e. the scalar of the radius vector, is calculated. The angular rate of rotation of the radius vector (ω) is determined, along with the linear rate of movement of the point on the hodograph ($\underline{v}$) and the length of individual sectors of the vector dynamocardiogram ($\bar{R}_2 - \bar{R}_1$).

Vector dynamocardiographic investigations performed under various physiological and pathological conditions have revealed a characteristic, physiologically founded dynamics of the picture and quantitative indicators dynamocardiograms, which makes it possible to characterize the contractability of the myocardium using these figures (L. A. Ioffe, 1962).

Dynamocardiographic investigation of cardiac activity of athletes. The cardiac activity of athletes at rest and under conditions of muscular work has been studied by V. L. Karpman (1956) and L. A. Ioffe (1962). It has been established that under rest conditions, the picture of longitudinal dynamocardiograms has certain specifics in comparison to the curves of untrained persons. The x wave is considerably more frequently encountered and more

clearly expressed (Figure 50), which in some observations made the definition of the C wave difficult.

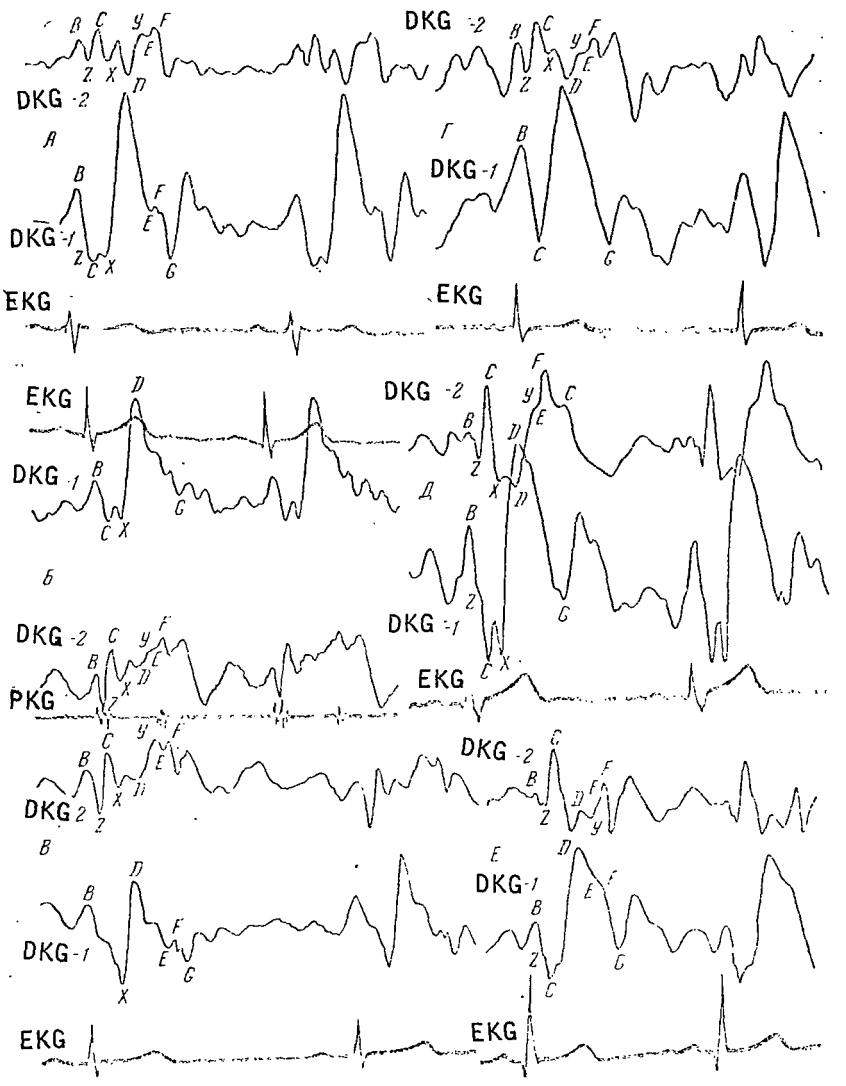

Figure 50. Variants of Longitudinal and Transverse Dynamocardiograms in Athletes.

In a number of observations, the E and F waves were absent or expressed as hardly noticeable steps. In the interval D-G, the curve sector in these cases was a straight line (Figure 50).

The amplitude of the sectors B-C and C-D was moderately lower in comparison with the curves of untrained persons.

The duration of the intervals of athletes' dynamocardiograms, according to our data, generally correspond to the durations expected for a given rhythm. Only a moderate lengthening of interval III (C-D) and interval VII (G-A) were noted (Table 11).

The mean duration of intervals on the dynamocardiogram of healthy but untrained persons and athletes (in seconds) are presented in Table 11.

The appearance of the transverse dynamocardiogram basically corresponded to curves recorded for untrained persons. We could note only the appearance of a high x wave, and in some observations an equally high C wave (Figure 50). Frequently, the D wave was of lower depth than that of untrained persons, and in individual observations this wave was found to be "inverted" (see Figure 50). /188

TABLE 11. MEAN DURATION OF INTERVALS ON DYNAMOCARDIOGRAM OF HEALTHY UNTRAINED PERSONS AND ATHLETES
(in sec)

Category of test subjects	Dynamocardiogram intervals						
	I	II	III	IV	V	VI	VII
Untrained persons	0,085	0,083	0,125	0,131	0,032	0,063	0,499
Athletes	0,090	0,088	0,135	0,130	0,029	0,067	0,621

Tr. Note: Commas indicate decimal points.

The amplitude of individual sectors of the transverse dynamocardiogram of athletes remains practically unchanged, with the exception of the D-y sector, where it is noticeably decreased.

The overall appearance of the vector dynamocardiograms of athletes does not differ from the curves of untrained persons. However, detailed analysis allowed L. A. Ioffe (1962) to note certain changes (less in the general appearance of the curve than in its quantitative indicators) (Table 12).

We can see from Table 12 that the quantitative indicators of the vector dynamocardiograms of athletes during various phases of the cardiac cycle differ from those of untrained persons. Thus, in untrained persons the highest angular velocity is noted in the B-z interval, the highest linear velocity in the C-D interval. in athletes, the maximum angluar end linear /189

velocities are noted in the z-C interval. Comparison of quantitative indicators of vector dynamocardiograms of athletes and untrained persons in various intervals indicates a reduction in the mechanical activity of the heart of athletes practically throughout the entire systole (with the exception of the z-C interval, which characterizes the mechanical processes at the beginning of the period of expulsion). The increase in the angular and linear velocities in this interval reflects the more powerful beginning of emptying of the ventricles than in untrained persons. The general reduction in the mechanical activity of the heart of athletes at rest is indicated by the decrease in summary length of sectors of the systolic complex (3630 g·cm, as against 4030 g·cm for untrained persons) and the decrease in the mean systolic linear rate ($11.2 \cdot 10^3$ g·cm/sec, as against $13.5 \cdot 10^3$ g·cm/sec for untrained persons).

The change in the picture of longitudinal and transverse dynamocardiograms and vector dynamocardiograms, the decrease in the amplitude of the main systolic wave (sector C-D) of the longitudinal dynamocardiogram, the decrease in the number of quantitative indicators of the vector dynamocardiogram result from specifics of the cardiac activity of the athlete at rest.

TABLE 12. CHARACTERISTICS OF INTERVALS OF SYSTOLIC COMPLEX OF VECTOR DYNAMOCARDIOGRAMS OF ATHLETES

Intervals	$\varphi_2 - \varphi_1$ Radians	$\overline{R}_2 - \overline{R}_1$, g·cm	t, sec	$v \cdot 10^3$, g·cm/sec	ω Radian/sec
$B - z$	1,466	441	0,040	11,0	36,6
	(1,711)	(400)	(0,032)	(12,5)	(53,4)
$z - C$	3,019	752	0,048	16,0	62,9
	(2,042)	(820)	(0,057)	(14,3)	(35,8)
$C - D$	2,077	1830	0,135	13,6	15,4
	(2,704)	(1900)	(0,125)	(15,2)	(21,6)
$D - y$	0,454	610	0,100	6,1	4,5
	(0,768)	(910)	(0,077)	(11,8)	(9,9)

Notes: 1. ϕ_2 and ϕ_1 are angle of inclination of radius vector to positively directed abscissa; $\overline{R}_2$ and $\overline{R}_1$ show length of sector of vector dynamocardiogram; t shows duration of vector dynamocardiogram interval; v shows linear rate of rotation of radius vector; ω shows angular rate of rotation of radius vector. 2. Parentheses include indicators of vector dynamocardiograms of healthy but untrained persons.

Tr. Note: Commas indicate decimal points

V. L. Karpman (1956) investigated the influence of forty deep knee bends per minute on cardiac activity. The most important of the amplitude and time changes in the longitudinal dynamocardiogram which he observed are shown in Table 13.

Judging from average data, the cardiac rhythm increased by approximately 50%, indicating a clear reaction of the cardiovascular system to the muscular work. Restoration of the initial rhythm was observed after about 3.5 min following completion of work.

The duration of the II interval was shortened by 0.01-0.05 sec (by an average of 0.027 sec). Shortening of the duration of intervals III + IV in many cases reached 0.10-0.15 sec, and averaged 0.055 sec. The intervals II + III + IV (mechanical systole of the ventricles together with the protodiastole) was shortened by 0.02-0.17 sec.

Restoration of initial dynamocardiographic indicators occurs most intensively during the first 1 1/2 min.

The change in the appearance of the curves is primarily determined by amplitude shifts. The most important are the increase in the amplitude of the dynamocardiogram in interval III. At the same time, the relative amplitude shifts of sector C-D correlate satisfactorily with the data of other authors on changes in systolic volume under the influence of measured muscular work. /190

TABLE 13. MEAN VALUES OF CERTAIN DYNAMOCARDIOGRAPHIC INDICATORS BEFORE AND AFTER PHYSICAL WORK (40 DEEP KNEE BENDS) IN ATHLETES

Indicators	At rest	Investigation time after loading (min)		
		½	1½	3½
Pulse frequency per min	63,8±2,4	96,2±4,6	77,2±3,0	69,0±2,4
Duration of interval II, msec	78±2 (78)	51±2 (63)	59±2 (72)	68±2 (76)
Duration of interval III + IV, msec	241±6	186±4	220±6	223±3
Duration of interval II + III + IV, msec	319±7 (315)	237±5 (252)	278±6 (286)	289±4 (302)
Intrasystolic indicator of dynamocardiogram, %	75,5±0,6	78,4±0,8	79,1±0,7	76,4±0,5
Increase in amplitude of sector C-D after load, %	—	71,5±0,7	35,1±0,5	10,9±0,4

Note: Parentheses include values normal for given cardiac rhythm.

Tr. Note: Commas indicate decimal points.

TABLE 14. CHANGES IN DYNAMOCARDIOGRAPHIC INDICATORS AS A FUNCTION OF MUSCULAR WORK OF VARIOUS LEVELS OF DIFFICULTY

Number of deep knee bends	Duration of interval II, sec.		Duration of intervals, III + IV, sec		Increase in amplitude of sector C-D after loading, percent of initial amplitude
	At rest	After loading	At rest	After loading	
20	0,08	0,08	0,25	0,20	18
40	0,08	0,06	0,27	0,20	68
50	0,08	0,05	0,26	0,18	90

Tr. Note: Commas indicate decimal points.

Using dynamocardiography, we can evaluate the degree of the influence of muscular work of various levels of difficulty on cardiac activity. An example is the following observation (V. L. Karpman, 1956).

Master of sports T, European champion rower, 30 years old. Dynamocardiogram recorded on various days before and after 20, 40 and 50 deep knee bends (Table 14).

The changes in the systolic complex of the dynamocardiogram are determined by restructuring of cardiac activity under the influence of extracardiac nerves and humoral inotropic influences. V. L. Karpman (1956) assumes that the influence of the stimulating innervation can be determined by comparing the actual duration of intervals on the dynamocardiogram with the expected values. The expected values show the changes which should occur under the isolated influence of the accelerating nerves. Table 13 clearly demonstrates that the duration of interval II is short in comparison with the expected values, while the duration of intervals III + IV generally corresponds to the expected values. This gives us reason to believe that the changes in duration of intervals III + IV result from an increase in cardiac rhythm under load, while the changes in duration of interval II and the mechanical systole are determined by some additional factors, including most probably the influence of Pavlov's stimulating nerve. The stimulation of cardiac contraction is accompanied by an accelerated increase in intraventricular pressure. Consequently, interval II, which includes the phase of isometric contraction, should be shorter than the expected value. The degree of shortening of interval II depends little on the increase in pulse frequency, and is shortened considerably even with minimal increase in pulse frequency, whereas there is a proportional dependence between the shortening of intervals III + IV and the increase in pulse frequency. /191

These data were confirmed by us (L. A. Ioffe, 1962) in a study of the dynamics of the indicators of the dynamocardiogram under the influence of high-intensity loading. The test subjects pedaled on a bicycle ergometer

for five minutes at a rate of 1,100 kg-m/min. In addition to longitudinal dynamocardiograms, transverse dynamocardiograms and vector dynamocardiograms were recorded.

Due to the high power of the work, amplitude shifts were more clearly expressed. Thus, the amplitude of sector C-D in the longitudinal dynamocardiogram was increased by an average of 88%, and on the transverse dynamocardiogram by 97%. In correspondence with this, the amplitude of sector C-D of the vector dynamocardiogram is doubled (maximum increase 3.5 times).

The amplitude shifts influence changes in the appearance of the longitudinal and transverse dynamocardiograms and particularly of the vector dynamocardiograms (Figure 51). The total length of sectors of the systolic complex were sharply increased after the work, averaging 6,870 g·cm. A comparison of the amplitude changes in the vector dynamocardiogram with the literature data on changes in systolic volume and volume per minute under the influence of loading showed that they were proportional. This indicates the role of cardiac output in the formation of the curve of displacement of /192
the center of gravity of the thorax. The reinforcement of cardiac kinematics during muscular work is reflected as a sharp increase in the linear velocity during the systole (Table 15).

TABLE 15. CHANGES IN LINEAR VELOCITY (v) IN INTERVALS OF SYSTOLIC COMPLEX OF VECTOR DYNAMOCARDIOGRAMS OF ATHLETES UNDER INFLUENCE OF MUSCULAR WORK

Intervals	Linear velocity $v \cdot 10^3$ g·cm/sec	
	At rest	30 sec after muscular work
$B - z$	10,9	22,8
$z - C$	16,7	44,2
$C - D$	13,7	31,2
$D - y$	6,6	23,2
$B - y$	11,5	30,9

Tr. Note: Commas indicate decimal points.

Table 16 shows the changes in duration of phases of the cardiac cycle during the recovery period according to the data of dynamocardiography. These changes are similar to those observed when the polycardiographic method is used.

Initial dynamocardiographic indicators in most test subjects were restored by the fifth minute. The nature and duration of the recovery period /193
allows us to make some judgment concerning the functional state of the myocardium.

TABLE 16. CHANGE IN DURATION OF PHASES OF CARDIAC CYCLE IN ATHLETES DURING RECOVERY PERIOD

Time of investigation	Duration of cardiac cycle, sec.	Phase of asynchronous contraction, sec.	Phase of isometric contraction, sec.	Expulsion period, sec.	Mechanical systole, sec.
Data at rest	1,082	0,057	0,040	0,276	0,316
Time after completion of work					
0,5	0,650	0,054	0,026	0,196	0,222
1,5	0,810	0,056	0,031	0,242	0,273
2,5	0,815	0,058	0,033	0,246	0,279
3,5	0,865	0,058	0,036	0,251	0,287
4,5	0,885	0,059	0,038	0,260	0,298
5,5	0,894	0,059	0,040	0,266	0,306
6,5	0,894	0,060	0,040	0,267	0,307
7,5	0,960	0,060	0,040	0,270	0,310

Tr. Note: Commas indicate decimal points.

Master of sports A-n, 24 years old, 400-m runner. Has no complaints. Volume and intensity of training load during period preceding investigation moderate. Figure 51 shows A-n's vector dynamocardiogram at rest and after 30 sec following five minutes pedaling on bicycle ergometer. Changes in curves show changes in cardiac kinematics and hemodynamics in respiratory circulation. Thus, the change seen in certain points of the vector dynamocardiogram, particularly points B, Z, C and D, in quadrants II and III of the rectangular coordinates, i.e. shifts to the right, indicate reinforcement of mechanical activity of the right ventricle. Reinforcement of the apical thrust is reflected in an increase in the scalar of the radius vector drawn to point C. The increase in systolic volume is documented by an increase in the length of sector C-D of the vector dynamocardiogram (4,190 g·cm; at rest -- 1,696 g·cm). A reflection of the overall reinforcement of mechanical activity of the heart during the systole is the increase in summary length of sectors of the systolic complex (7,180 g·cm; at rest -- 3,900). In connection with the reinforcement of cardiac kinematics, the linear rate of displacement of the point on the hodograph increases sharply: the initial mean systolic velocity, $13.1 \cdot 10^3$ g·cm/sec, is increased to $33.2 \cdot 10^3$ g·cm/sec. A shortening of the phases of asynchronous and isometric contraction in the period of expulsion is noted. At rest, their duration amounts to 0.06, 0.04 and 0.28 sec respectively, while after loading these figures change to 0.05, 0.01 and 0.19 sec. /194

Five minutes after completion of the work, restoration of the investigated indicators to their initial values was noted. The nature of the changes occurring under the influence of muscular work and their rapid recovery indicate high contractive capacity of the myocardium of this test subject.

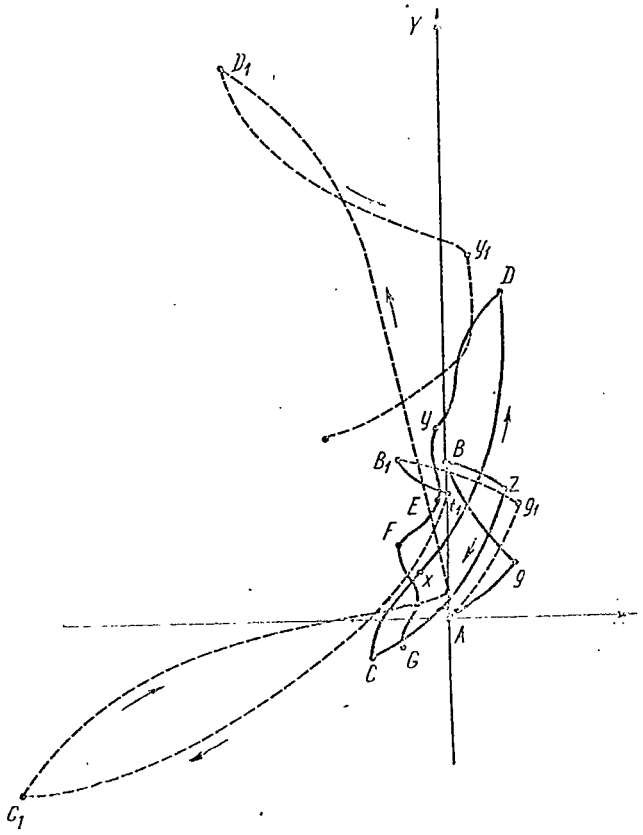

Figure 51. Frontal Vector Dynamocardiogram of Master of Sports A. Solid line, at rest; dotted line, after muscular work.

Thus, dynamocardiography makes it possible to consider fine changes in the mechanical manifestations of cardiac activity. It can be assumed that the method will be useful for a study of the cardiac activity of athletes at rest and under conditions of muscular work. Particularly great significance for objective evaluation of the contractive capacity of the myocardium is that of the usage, together with data on the phases of the cardiac cycle, of new quantitative indicators characterizing the displacement of the center of gravity of the thorax.

Ballistocardiography

Ballistocardiography is one of the latest methods for studying the cardiodynamics of humans.

Modern ballistocardiography was developed by Starr (1939), who designed the high-frequency ballistocardiographic table with natural oscillating frequency of 12-14 Hz. Starr suggested the currently generally used symbolization of the waves on the ballistocardiogram. In 1944, Nickerson and Curtis developed a low-frequency, critically damped ballistocardiograph. Then, Rappaport (1956), Noordergraaf (1958) and others suggested ultralow-frequency ballistocardiographs.

Using the systems just mentioned, the oscillations of a platform resulting from the mechanical activity of the heart of a test subject lying on the platform are recorded. This group of methods has come to be called indirect ballistocardiography.

In 1949, Dock and Taubman suggested a method of direct recording of the oscillations of individual sectors of the body arising during the work of the heart, which has come to be called direct vallistocardiography. Indirect ballistocardiography has not become widely used due to the cumbersomeness and /195 complexity of the apparatus, as well as the difficulty of its utilization.

In contrast to this, direct ballistocardiography, due to the simplicity and ease of the method, has come to be rapidly introduced into medical practice in the area of physiological investigations both abroad and in the USSR. A great role in the popularization and development of the method in our country has been played by V. V. Parin.

When direct ballistocardiography is used, displacements of various body sectors resulting from displacements of the center of gravity of the body as a result of cardiac activity are recorded. The micromovements of the body are recorded by the receiving devices are forced oscillations, arising as a result of the work of the heart and the movement of blood in the principal vessels.

Ballistocardiographic techniques. In the practice of medical testing of athletes, devices are generally used which record direct velocity ballistocardiograms. An example of one such device is the Dock ballistocardiograph, consisting of a plate with an induction coil, a stator carrying a magnet and a support of shaft filled with sand.

In Dock type devices, the ballistocardiogram may be recorded using piezoelectric, photoelectric or optical pickups. In order to record velocity ballistocardiograms, electromagnetic pickups are used, while acceleration ballistocardiograms are recorded using accelerometers. As our investigations have shown, the usage of such a portable ballistocardiograph for the investigation of athletes not only in the laboratory but under field conditions justifies itself fully.

In order to improve the quality of ballistocardiogram curves produced, they should be recorded directly on the floor. The test subject is placed on a matress up to the level of the popliteal fossae, and the sand-filled bars placed beneath the Achilles tendon. Recording is always performed under identical conditions of rest. For recognition of the ballistocardiographic waves, it is desirable to record an electrocardiogram simultaneously, which can be done if a two-channel device is used.

Normal ballistocardiogram. The direct ballistocardiogram differs significantly in its appearance from low-frequency and ultralow-frequency ballistocardiograms. Since these systems have not been used in sports medicine, in the following we will analyze only direct ballistocardiograms. Ballistocardiograms of this type are somewhat reminiscent of a sine wave, periodically repeated. The H, I, J, K waves (Figure 52) arise during the ventricular systole, while the L, M, N, O and G waves arise during the diastole.

The genesis of the H wave is complex. It is related both to the atrial systole (Nickerson, 1944) and to the movement of the atrioventricular septum toward the head during the phase of isometric contraction of the ventricles (Dock, Mandelbaum, 1953). /196

The I wave appears as a result of the recoil reaction occurring during rapid expulsion of blood from the ventricles.

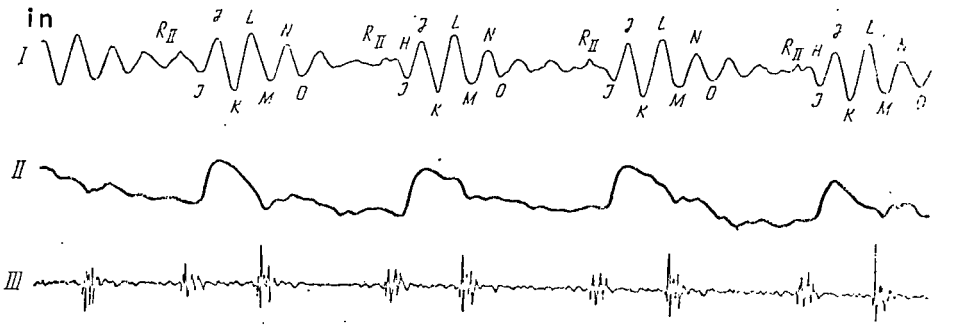

Figure 52. Normal Acceleration Ballistocardiogram of Well Trained Athlete (Class I According to Lapicirella Classification) Shown with R Wave of EKG. II, sphygmogram; III, phonocardiogram (Botkin's point, frequency characteristic C_1)

The ventricular J wave is recorded at the end of the phase of rapid expulsion and results from slowing of the blood flow as it moves through the arc of the aorta.

The K wave is observed at the end of the ventricular systole. It is formed principally by forces acting upon impact with the bifurcation of the ventral aorta.

The L, M, N, O, etc. waves arise during the ventricular diastole.

It should be noted that the genesis of ballistocardiogram waves is not completely clear and has not been completely studied as yet.

The amplitude of the waves of the ballistocardiogram, in particular the magnitude of sector I-J is determined by the energy expended by the heart on the expulsion of blood. Both the rate of expulsion and the quantity of blood expelled are of considerable significnace. Theoretically, the dependence between these values and the quantity of energy expended on expulsion of the blood can be expressed after Starr (1931) by the formula for the vital force:

$$F = \frac{mv^2}{2}, \qquad (39)$$

where F is the vital force (BKG-effect), m is the quantity of blood expended, v is the velocity of expulsion. It follows from this formula that the ballistocardiographic effect varies in proportion to the mass of the blood expended and to the square of the velocity of systolic expulsion. This has been confirmed in experiments on corpses by Starr (1958) who showed that as the expulsion of blood into the vascular lumen is retarded by 50%, the effort of the cardiac contraction is decreased by 75%. The rate of expulsion in turn depends on such indicators as the pressure in the aorta and pulmonary artery and the cross section of these vessels. /197

The amplitude of the waves on the ballistocardiograph and the duration of its intervals change considerably with the phases of respiration.

Detailed studies of respiratory fluctuations were made by Starr and Schroeder (1940), De Lalla and Brown (1959), who showed that during inhalation the ballistocardiographic effect of the right ventricle predominates, while during exhalation the effect of the left ventricle predominates in connection with the variation in blood filling of the cavities of the heart.

The amplitude of the ballistocardiographic waves is also influenced by constitutional factors and age variations in the tissues and organs, with which the frequency of natural oscillations of the body changes and the degree of damping increases (Tannenbaum, Vessel, Schack, 1956). In Starr's opinion, the amplitude of I-J decreases with age.

Thus, in evaluating ballistocardiograms we must consider all of these factors.

The study of the functional state of the cardiovascular system by the ballistocardiographic method assumes qualitative and quantitative evaluation of the curves. In a qualitative characterization, we consider the morphological changes of the complexes, the regularity of their alternation and disruptions in their interrelationships.

In 1940, Starr suggested a classification for ballistocardiograms, which takes into consideration changes in the systolic complex indicating a reduction in contractability of the myocardium. They include: 1) the increase of the H wave or splitting of the J wave, which are represented as "early M"; 2) an increase in the L wave, the amplitude of which becomes equal to or nearly equal to the magnitude of J, represented as "late M."

A change in the appearance when the K wave is sharply deepened with small H and smooth J is called "late negative deflection."

In additon to these forms, intermediate forms were distinguished, including those with split or wavy nature of individual waves.

Later, a number of classifications have been suggested to evaluate changes in ballistocardiograms by degrees. However, the most common classification is that of Brown, De Lalla, Hoffmann and Epstein (1952), in which the respiratory variations of the curve are taken into consideration. Five morphological variants of the curves are differentiated:

zero step -- unchanged ballistocardiogram; /198
I step -- at the output, the amplitude of IJ of the minimum complex is less than 50% of the maximum amplitude of the sector; quantity of decreased complex is less than 50%;
II step -- quantity of decreased complex is less than 50% of those recorded;
III step -- amplitudes of waves at input reduced, during output complexes are deformed;
IV step -- chaotic curve of low amplitude.

As the contractive function of the myocardium is decreased, changes in ballistocardiograms of steps III and IV are noted.

In order to evaluate the ballistocardiograms of athletes, the Lapicirella classification is used (1958):

I step -- absolutely normal ballistocardiogram;
II step -- sum of complexes distorted, amplitudes decreased;
III step -- all complexes distorted, amplitudes decreased;
IV step -- voltage of complexes decreased, complexes distorted beyond recognition.

One defect of the Starr and Lapicirella classifications is the absence of analysis of respiratory fluctuations in the curve, while the Brown classification does not consider morphological changes in individual complexes of the ballistocardiogram. This has led to the development of new classifications and supplements to existing classifications. One such attempt is the method suggested by R. M. Bayevskiy in 1962 of evaluating morphological changes which allows us to judge both concerning the amplitudes of waves and the form of individual complexes.

Quantitative analysis of ballistocardiograms consists of measurement of the amplitude of waves and time intervals with subsequent calculation of relative indicators, which makes it possible to compare curves recorded for individual persons using various transducers (Figure 53).

The amplitude of the waves can be most conveniently measured using the values for the sectors H-I, I-J, J-K, K-L, etc., not from the base line passing through the middle of the diastolic wave to the peak of the corresponding wave as was done by Kazmeier and Schild (1957), etc.

Calculation of mean values of wave amplitudes on ballistocardiograms is performed by various investigators from various numbers of complexes. I. K. Troitskaya (1963), in statistically processing materials, showed that when the I-J and J-K amplitudes were calculated, reliable differences of

averages were not discovered beginning with the calculation of four complexes in the ballistocardiogram at rest, or five complexes in a ballistocardiogram taken after physical loading.

Of the relative indicators, the following are usually calculated: /199

$$\frac{HJ}{IJ};\ \frac{JK}{IJ};\ \frac{KL}{IJ};\ \frac{KL}{JK}.$$

The quantitative changes in respiratory variations are expressed by the respiratory coefficient (the quotient from division of the maximum sector I-J by the minimum) or the ballistocardiographic index (Dock, Mandelbaum, 1953), which is the quotient from the division of the maximum I-J by the maximum I-J or J-K. The normal value of respiratory coefficient is up to 2.0, the ballistocardiographic index -- from 0.4 to 1.0.

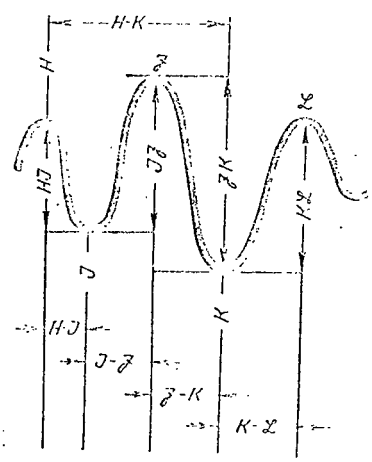

Figure 53. Diagram of Measurement of Amplitude of Waves and Time Intervals of Ballistocardiogram. Explanation in text.

The fundamental investigations of Starr and his colleagues (1939-1958) established that displacement of the body recorded by seismic devices (according to the terminology of V. V. Parin and R. M. Bayevskiy, 1961) are proportional to the force of the cardiac contractions. In experiments on corpses, a retardation of the initial rate of blood expulsion led to disappearance of the I wave, while the diastolic wave increased. Nonsimultaneous expulsion of blood into the pulmonary artery and aorta led to splitting of the peak I-J. Significant morphological disruptions of the complexes arose when the rate of expulsion of blood in various phases of the cardiac cycle was changed. A study of the ballistocardiograms of sick persons showed sharp respiratory variations in cases of pathological cardiac reaction to changes in blood filling of heart cavities.

A. V. Mareyev (1963), in experiments on dogs and studies of ballistocardiograms of patients with coronary deficiency shows that the H-I segment is formed not only by the recoil arising upon expulsion of blood, but also by the forces related to movement of the heart. It was shown that the impulses related to heart movement are added primarily with the recoil. Therefore, when the contractive function of the myocardium is decreased, the amplitude of the I-J waves is decreased (decrease in ballistocardiographic contribution of the ventricles), while the

amplitude of H-I increase is due to compensatory hyperfunction of the heart and an increase in the ballistocardiographic effect resulting from reinforced movement of the heart.

The predominance of various factors of hemodynamics upon a decrease in /200
the contractive capacity of the myocardium leads to the formation of various types of complexes. For example, upon predominance of hypertrophy of the left ventricle with sufficient compensatory capabilities, we observe the formation of the "early M" type complex, as a result of the primary increase in segment H-I, resulting from reinforcement of the movements of the hypertrophic section of the heart. In this case also a decrease in respiratory variations of the curve is noted in relation to the equalization of the mechanical effectiveness of the two ventricles. On the other hand, when compensatory capabilities of the left ventricle are decreased, a shortening of I-J and merging of waves H with J are noted, accompanied by relative deepening of the K wave. The decrease in the ballistocardiographic effect of the weakened ventricle facilitates an increase in respiratory variations of the curve. The decrease in initial rate of blood expulsion which arises in this case leads to a deepening of the K wave (Starr, 1958).

These qualitative changes are reflected in quantitative analysis of ballistocardiograms as well. Thus, with the first type of changes in the systolic complex, the HI/IJ ratio is increased, and JK/IJ and KL/IJ are decreased, while with the second type HI/IJ is decreased and JK/IJ and KL/IJ are increased.

Ballistocardiography in sports medicine. The first ballistocardiographic observations of athletes were performed in 1953 by Doliopoulos and Bangou, who showed that the amplitude of the waves and their duration in trained athletes were higher than in untrained persons. Subsequently, Medved and Horvat (1956) found that in athletes trained for endurance, the amplitude of ballistocardiogram waves was higher than in representatives of sports games and fencers. The study of the ballistocardiograms of athletes was also undertaken by Bober (1954), Bodrogi (1957), Jokl et al. (1958), A. N. Kokosov (1959), V. P. Kushelevskiy, I. A. Manuylov and I. L. Manuylova (1960); L. L. Orlov, V. V. Bulychev and Yu. I. Akimov (1962), Yu. P. Panov (1960); etc.

The most complete and comprehensive ballistocardiographic investigations of athletes were performed by L. I. Stogovaya (1958, 1963), who studied the qualitative and quantitative indicators of the ballistocardiogram with various states of training and compared them with the indicators in healthy persons not involved in sports.

In studying the morphological specifics of ballistocardiograms of athletes, it is expedient, as has been indicated above, to use the Lapicirella classification with supplemental analysis of the structure of the /201
complexes using the method of R. M. Bayevskiy (1962). We used this combined method of evaluation in analysis of the ballistocardiograms of 90 leading

athletes -- masters of sports in academic rowing during assemblies at various stages of training. Of these, 37 persons were observed continually for between one and two and one half years. The recordings of ballistocardiograms were performed as part of combined medical examinations using the S. P. Letunov functional experiment, with the recording of electrocardiograms, etc.

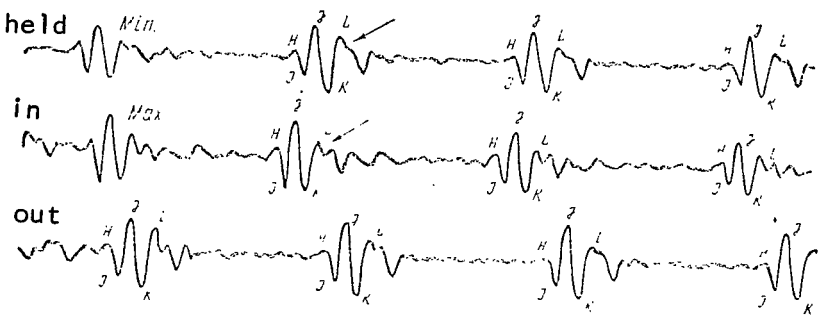

Figure 54. Change in Ballistocardiogram Step II (According to Lapicirella) in Master of Sports P, 22 Years Old. Broadening and splitting of L waves shown by arrows.

In more than 50% of the athletes observed over an extended time period, the ballistocardiographic curves were related to step II of changes according to the Lapicirella classification due to deformation, expansion and splitting of the K-L waves with no decrease in wave amplitude (Figure 54). Step III changes in the ballistocardiograms were observed rarely: only during the main period of training with a decrease in the functional state of the myocardium. As an example, we present the following data.

1. Master of sports B, 22 years old. Total sports ranking eight years. Academic rower, four years. During intensive training, periodically shows high blood pressure up to 140/90 mm Hg during periods of rest. On electrocardiogram, shows characteristics of diffuse changes of myocardium. In addition to an increase in arterial blood pressure, nonrestoration of maximum and minimum pressure is observed at rest after functional test, along with weakness, worsening of rowing technique. Ballistocardiogram taken at rest (Figure 55, a) shows deformation of I-J and decrease in amplitude of this segment primarily during exhalation phase. At fourth minute after 15 seconds run (Figure 55, b) during inhalation phase, splitting of I-J and decrease in amplitude of this segment during exhalation with high amplitude of H-I noted. On the whole, changes in curve indicate decrease in contractive function of left ventricle.

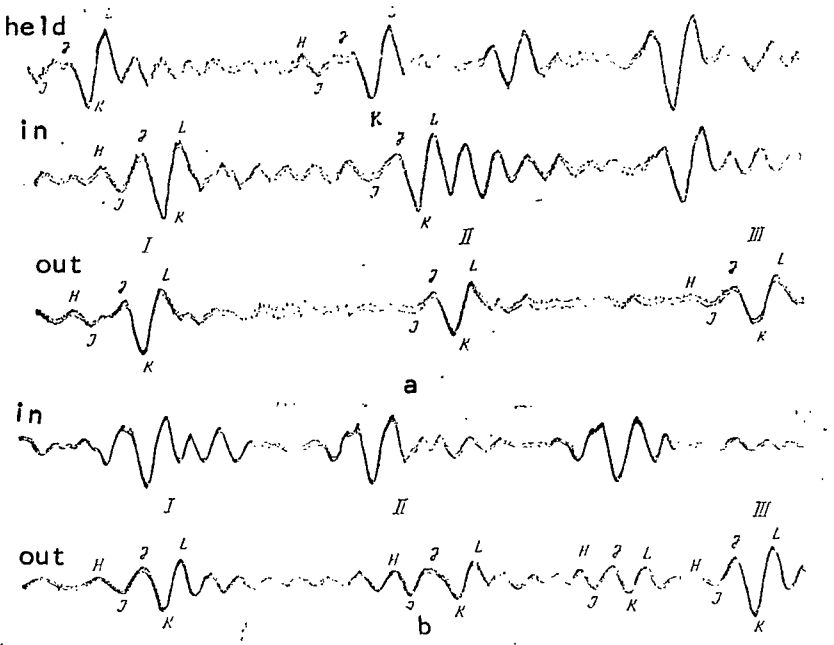

Figure 55. Example of Changes in Ballistocardiogram, Step III (According to Lapicirella) in Master of Sports B, 22 Years Old. a, at rest; b, fourth minute into recovery after 15 sec run. Explanation in text.

2. Master of sports Ch, 29 years old. Sports ranking (academic rowing) 13 years. State of clear fatigue: decrease in sports indicators, gradual maximum pressure reaction to 15 seconds run, incomplete restoration of pulse after 15 sec and three minutes run. Electrocardiogram shows indicators of incomplete block of right branch of bundle of His. On ballistocardiogram taken at rest (Figure 56, a) during inhalation phase shows "fused H-J" with deepening of K wave. Changes indicate decrease in contractive function of right ventricle and worsening of compensatory capacities, noted even when at rest. After 15 sec run during fourth minute of recovery period, same ratio is retained (Figure 56, b).

The analysis of the morphological structure of complexes in the test subject athletes which we investigated allowed us to determine certain specific features of the ballistocardiograms of well trained persons. The data which we produced agree with those described in the literature (Doliopoulos, Bangou, 1953; Lapicirella; L. L. Orlov, 1959; L. I. Stogova, 1963; etc.). These specifics consist of the following:

1) in athletes, relatively high amplitude of the J wave is observed;

2) on the ballistocardiogram, deepening of the JK waves and high amplitude of I wave are generally observed (in 84% of athletes examined); similar observations were made by Lapicirella (1958), B. S. Panov (1960) and L. L. Orlov (1959); /203

3) broadening, splitting or deformation of ballistocardiogram sector in K-L interval observed in 58% of athletes (see Figure 54);

4) in 49% of the athletes during the exhalation phase splitting of the I wave is observed with formation of an additional small wave; this is usually combined with incomplete block of the right branch of the bundle of His and expressed hypertrophy of the left ventricle.

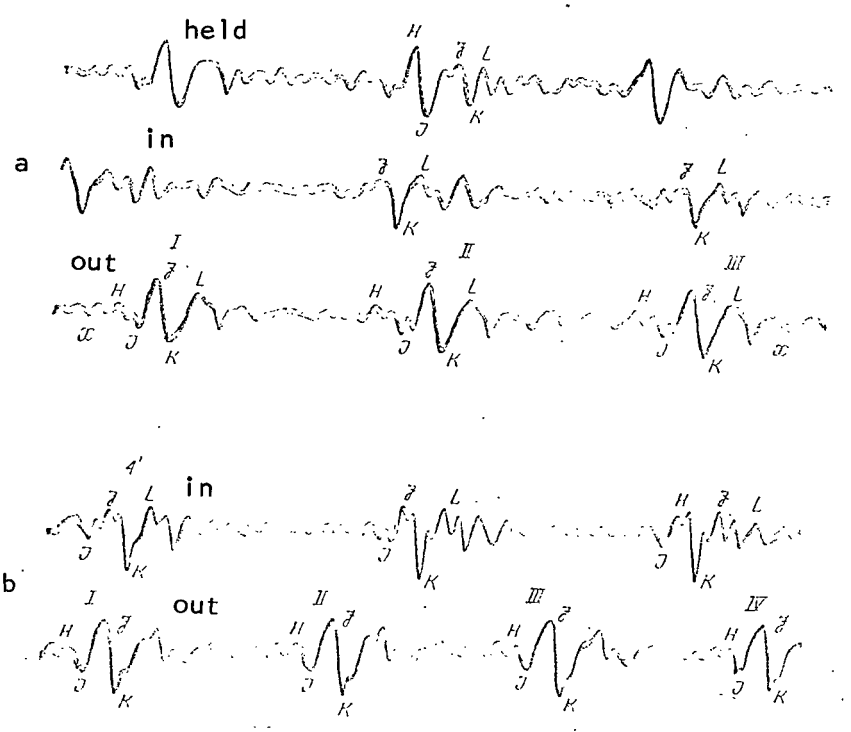

Figure 56. Example of Changes in Ballistocardiogram, Step III (According to Lapicirella) in Master of Sports Ch, 29 Years Old. a, at rest; b, fourth minute into recovery after 15 sec run. Explanation in text.

An increase in the state of train is characterized in a qualitative evaluation of the ballistocardiogram by an increase in the amplitude of the J and JK waves (L. I. Stogova, 1963).

Changes in ballistocardiograms after loads in well trained persons appear as an increase in the amplitude of the sectors I-J and K-L. With insufficient training and overtraining, the sector I-J is more sharply increased, and the sector K-L is decreased. Splitting and waviness of these complexes may decrease or disappear completely or may be deepend immediately after loading. /204

Analysis of the time indicators of the ballistocardiogram (L. I. Stogova, 1963) has shown that in highly trained athletes, the duration of the direct velocity ballistocardiogram intervals is increased (Table 17).

TABLE 17. DURATION (IN SEC) OF TIME INTERVALS IN BALLISTOCARDIOGRAM IN ATHLETES AND NONATHLETES (L. I. STOGOVA)

Indicators	Healthy nonathletes	Athletes
H--*K*	0,267	0,299
H—*I*	0,053	0,066
I—*J*	0,095	0,101
J—*K*	0,119	0,130
R—*H*	0,109	0,099
Duration of cardiac cycle	0,913	1,03

Tr. Note: Commas indicate decimal points.

In the analysis of ballistocardiographic indicators at rest, we determined an increase in KL/IJ and KL/JK. After the standard physical load we, like L. I. Stogova (1962), detected a primary increase in the relative index JK load/JK rest in comparison with the relative indicator IJ load/IJ rest during the main period of training with good physical state (favorable type of reaction on the ballistocardiogram). During the preparatory period, we observed the reverse relationship of these indicators (Figure 57). We distinguish three variants of the change in IJ load/IJ rest from the preparatory period to the main training period during the process of increasing state of training: 1) primary increase in IJ load/IJ rest; 2) IJ load/IJ rest unchanged, while JK load/JK rest increases; 3) JK load/JK rest unchanged, while IJ load/IJ rest decreases.

Worsening of the state of training, overfatigue and overtraining were accompanied by a significant increase in the relative indicator IJ load/IJ rest (Figure 57) and simultaneous decrease in the relative indicator JK load/JK rest. /205

The usage of ballistocardiography in sports medicine has promise, since it can be used to perform evaluations of the contractive function of the myocardium of the ventricles.

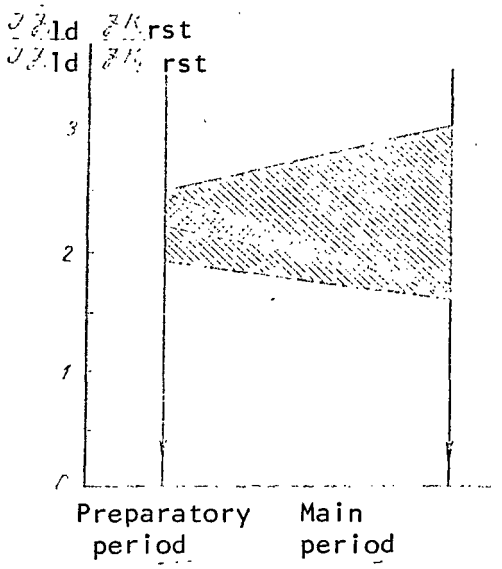

Figure 57. Dynamics of Indicators IJ Load/IJ Rest and JK Load/JK Rest with Changing State of Training. Solid lines show changes in indicator IJ load/IJ rest, dotted lines JK load/JK rest, fine lines -- changes in indicators with increasing state of train, heavy lines -- incase of overtraining.

Kinetocardiography[1]

The first curves of low-frequency precordial vibrations were recorded by Marey (1878), who used pneumatic capsules to record movements of the apex of the heart. This method was called cardiography, but it was not widely used, and was almost abandonded in the 1930's.

At the beginning of the 1950's, investigations in the area of studying precordial vibrations using modern electromechanical transducers were once more begun.

The beginning of these investigations was performed by Miller and White (1941), and subsequently a great deal of attention was turned to this problem by Johnston and Overy (1951). One important development is the method developed by Rosa (1955-1956) of recording precordial vibrations -- stethography. /206

In later years, kinetocardiography became broadly used abroad -- a method suggested by Eddleman et al. In the Soviet Union, this method was first used in 1961 by L. B. Andreyev.

In 1958, independently of Eddleman, we developed a method for acceleration kinetocardiography (I. Ye. Ranskiy).

Techniques of recording in acceleration kinetocardiography. Kinetocardiography is based on the principle of conversion of mechanical movement into an electrical signal. The vibrations of the chest wall in the infrasonic range have great oscillating amplitude, many times greater than the

[1] In this section, we are concerned with a new method of recording precordial vibrations. It differs from the "classical" kinetocardiography of Eddleman (1959) first of all in that we record only the second derivative of displacements of the thorax, secondly in that the receiver device is fastened to the chest of the test subject, not fixed at a point in space using a rod -- Ed.

amplitude of the sonic vibrations; therefore, they can be recorded easily in the form of a curve of displacements, velocities and accelerations of the chest wall.

The receiver devices (transducers) converting the mechanical vibrations into electrical signals in our method are accelerometers, either liquid (A. A. Sukhanov, I. Ye. Ranskiy, 1958; etc.) or piezoelectric type transducers.

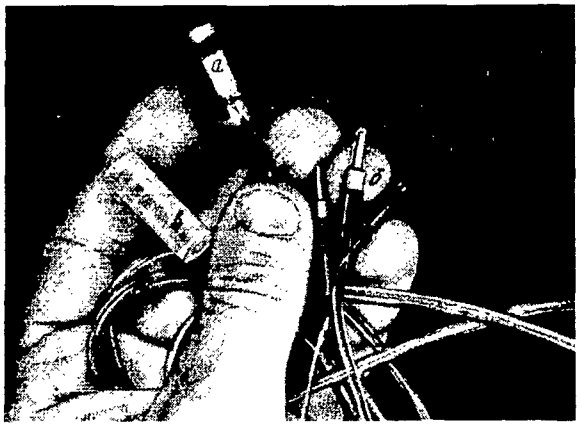

Figure 58. Acceleration Transducer. a, piezocrystal with load; b, Leads; c, overall view

The basis of the piezoelectric transducers is the property of certain crystalline dielectrics of creating an electrical charge under the influence of stress or deformation.

The value of the electrical polarization Π is proportional to the deforming force P_x:

$$\Pi = K \cdot P_x, \qquad (40)$$

where K is a coefficient, called the piezoelectric constant. The sensitivity of the transducers is higher, the higher the value of K. The greatest sensitivity is that of Seignette salt crystals and barium titanate crystals. /207

The acceleration transducer consists of a base with a mounting plate inserted in it, which carries the crystal, and the load, suspended on the free end of the plate (Figure 58). Displacement of the base of the transducer relative to the load deforms the crystal. The amount of charge arising is proportional to the acceleration of the movement of the chest wall.

The natural frequency of the transducer is many times higher than the frequencies recorded, on the order of 800 Hz, a necessary condition for the production of undistorted curves. The transducer records accelerations and is standardized in g units. With an amplification of 1 mv = 2 mm and an acceleration of 1 g (981 cm/sec^2) a curve with an amplitude of 16 mm is recorded.

Kinetocardiographic investigation should be performed in a room removed from working elevators, X-ray and physical therapy machinery.

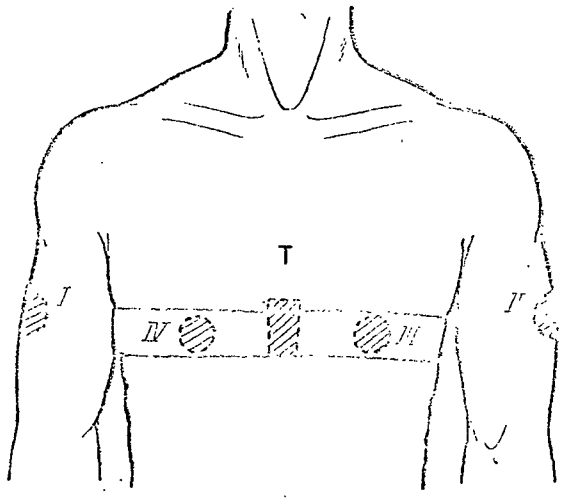

Figure 59. Diagram of Location of Electrodes and Transducer. I, "right arm" electrode; II, "left arm" electrode; III, "left leg" electrode; IV, "right leg" electrode; T, transducer.

The recording of a kinetocardiogram can be performed with the test subject in the lying, sitting or standing position, depending on the purpose of the recording. Most frequently, kinetocardiograms are recorded with the test subject lying on his back. The transducer is fastened to the chest using elastic bandage at the point of attachment of ribs 4-5 (left ventricle) (Figure 59). The correctness of connection of the transducer to the input of the electrocardiograph is tested by tapping the transducer. A tap on top causes initial deflection of the curve downward. Recording should be performed using a two-channel amplified apparatus, simultaneously with an electrocardiogram.

The movement of the heart resulting from contraction and relaxation of its musculature, intracardiac and intravascular displacement of blood, opening and closing of the heart valves, rotation of the heart and its lateral oscillations cause vibrations of the thorax, which are recorded as the kinetocardiogram curve. This curve consists of two sets of waves, represented by the letters of the Latin alphabet from e to z inclusive (Figure 60). /208

Normal kinetocardiogram. To aid in clinical-physiological analysis of the kinetocardiograms, we performed synchronous recording of kinetocardiograms plus phonocardiograms, electrocardiograms, ballistocardiograms, dynamocardiograms and carotid artery sphygmograms.

The comparison of hemodynamic indicators produced by catheterization (V. L. Karpman and V. S. Savel'yev, 1960; Wiggers, 1921; Blumberger, 1940; etc.) with the results of analysis of kinetocardiograms allowed the elements of this curve to be interpreted as follows. /209

The wave complexes from g to s reflect the cardiac systole. The second complex (u wave) corresponds to the beginning of the diastole and corresponds in time to the beginning of formation of tone II. The heart diastole corresponds to the interval v-e.

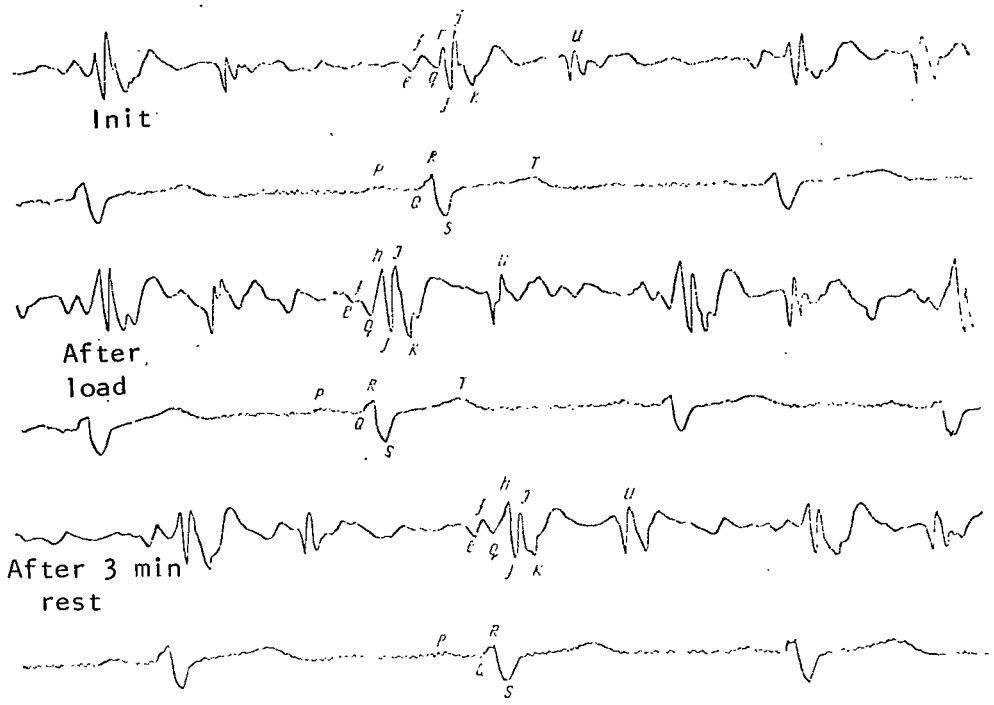

Figure 60. Kinetocardiogram Recorded for Athlete K During Performance of S. P. Letunov Experiment.

The R wave of the electrocardiogram occurs at the beginning of the h wave (point g) and its end corresponds to the high-frequency component of tone I. The u wave corresponds to the end of the T wave of the electrocardiogram, and tone II of the phonocardiogram and the lower point of the incisure on the reproduced carotid artery sphygmogram.

During the first phase of the systole -- the phase of asynchronous contraction -- the interventricular septum contracts, the atrioventricular valves close, the apex of the heart moves upward and inward. After closure of the atrioventricular valves during the phase of isometric contraction, the inclined muscles of the ventricles begin to contract, the heart rotates to the right, about its axis.

Point g of the kinetocardiogram is the beginning of the mechanical systole, when the entire myocardium is excited, gradually leading to an increase in muscular stress with the heart valves closed. The most intensive stress occurs at point h -- the peak of the wave, while the entire wave is a result of the forces acting during the phase of isometric contraction of the ventricles.

Wave i corresponds to the initial portion of the expulsion period -- the phase of rapid expulsion. At this moment, over a comparatively short time period (0.05-0.10 sec), two-thirds of the systolic blood volume is forced from the ventricles into the aorta with maximum acceleration; therefore, the j wave which characterizes this phase rises most rapidly.

The phase of rapid expulsion ends at point k. At this moment, the rate of expulsion of blood drops, and the phase of slow blood expulsion begins.

The decrease in pressure in the ventricle leads to opening of the semilunar valves. The impact of blood on the valves resulting from back flow leads to movement of the entire mass of the heart and appearance of peaked wave u on the kinetocardiogram.

The opening of the mitral and tricuspid valves begins the phase of rapid filling of the ventricles, which is reflected on the kinetocardiogram as the bell-shaped elevation in the u wave.

As the pressure in the atria and ventricles begins to equalize, the blood flow decreases, the velocity slows, the phase of slow blood influx to the ventricles begins. The kinetocardiogram takes on the form of a wavy line at this moment, tending toward the zero position.

The ventricular diastole ends with contraction of the atria. The /210
contractive activity is greatly influenced by the neighboring organs and large vessels. The outfolw and inflow of blood, movement of heart valves and variations in pressure within the thorax are of great significance in the pulsating movements.

The atrial systole is recorded on the kinetocardiogram as wave f. It arises after the P wave of the electrocardiogram.

Thus, the waves of the kinetocardiogram have the following physiological significance. The h wave results from forces operating during the phase of isometric contraction of the ventricles. The j wave results from the flow of blood during the phase of rapid expulsion of blood (see Figure 60). The interval k-s corresponds to the period of slow expulsion of blood from the ventricles. Wave u results from the impact of the column of blood on the semilunar valves. Interval v-e corresponds to the ventricular diastole (see Figure 60).

Analysis of the duration of waves on the kinetocardiograms of 100 healthy persons showed that the duration of cardiac cycle phases calculated from the kinetocardiogram corresponds completely to values produced using other methods.

During the phases of respiration, as the position of the body is changed, after physical loading and after taking various drugs, various hemodynamic changes occur which are reflected on the kinetocardiogram.

The changes in the kinetocardiogram during respiration depend both on the changes in topographic relationships of the organs in the thorax, and on the increase or decrease in intrathoracic pressure, which leads to redistribution of blood and changes in veinous return. Studying the influence of respiration on the ballistocardiogram, Brown, Epstein, de Lalla and Hoffmann (1952) noted an increase in waves of the systolic complex during inhalation, while Louson, Blumfield and Cournand (1953) established that the leading role in this belongs to the right ventricle. They showed that during inhalation the influx of blood to the right atrium is increased, leading to an increase in the volume of the right ventricle and greater mechanical effect of its contraction.

During the phase of deep exhalation, the arterial pressure is decreased, the volume per beat is decreased, and the beating force of the heart drops. True, we have not succeeded in finding such precise interrelationships on the kinetocardiogram, although this interpretation is quite acceptable as an explanation of changes in wave amplitudes of the kinetocardiogram. Furthermore, the amplitude of waves on the kinetocardiogram decreases during the phase of deep inhalation, which results most probably not from hemodynamic, but purely from mechanical factors.

During a very deep inhalation, the volume of the thorax increases, as does the air gap between the chest wall and the heart, and the effect of /211
contraction is thus somewhat lost and damped in the lung tissue. During quiet, shallower breathing, these sharp contrasts are not observed. Therefore, kinetocardiograms can be recorded during free breathing, or during the phase of shallow exhalation, for the production of clearer curves.

As the position of the body is changed, redistribution of the blood and variations in the blood filling of the heart occur, influencing both the phase structure of the cardiac contraction, and the kinetic function of the heart. In the standing position, blood filling of the heart is decreased by decreased veinous influx. This leads to a decrease in the volume per beat and a change in the isometric contraction of the ventricles.

As was shown by Eddleman, Willis, Heyer (1950), etc., in the vertical position we note a shortening of the entire systole and expulsion period. The phase of rapid filing (beginning of the diastole) is lengthened upon standing as a result of retardation of the blood in the lower extremities and of blood flow to the heart.

The kinetocardiogram recorded with the test subject in the standing position frequently shows splitting of the j wave, which disappears in the prone position. In a healthy person after physical loading the amplitude of the j wave increases, characterizing the phase of rapid expulsion.

In the presence of atherosclerotic cardiosclerosis, physical loading leads to a deformation of the curves, particularly the jk complex.

Smoking causes important changes in the kinetocardiogram. Our observations of 100 healthy persons showed that smoking causes various reactions in the kinetic function of the heart. The greatest changes in the appearance of the curve after smoking occur in the k-s and v-e intervals; the j wave also changes. Thus, in 60% of the observations the k-s interval was increased by 0.02-0.03 sec. The h wave does not change after smoking, but its amplitude is noticeably decreased.

Kinetocardiography in sports practice. In the literature available to us, we encountered only a few works on the usage of kinetocardiography in sports practice. However, as our experience has shown, this type of investigation can be useful in evaluating the condition of the myocardium of athletes, particularly in response to physical load. Kinetocardiography primarily allows analysis of the phase structure of the cardiac cycle. Also, the changes in the amplitude of the curve can be used to judge to a certain extent concerning the mechanical activity of the myocardium, the kinetics of the cardiac contraction.

In studying cardiodynamics of athletes, there is great interest in differences in changes in the kinetocardiogram after performance of various types of physical load (dynamic and static), as well as the influence of extrasystolic arhythmia in athletes on the kinetocardiogram. Investigations performed with 50 athletes showed the possibilities of this method in the solution of these problems. /212

A group of team athletes involved in various types of sports was used in an investigation before loading, immediately and five minutes following three minutes running (with acceleration during the last 15 sec).

In analyzing our curves considering the amplitude and time characteristics, it was found that the reaction to dynamic physical loading was expressed as an increase in pulse frequency, a marked decrease in the duration of the cardiac systole in its expulsion period and an even more sharply expressed decrease in the period of filling of the heart.

After loading in many cases we observed asynchronism of the ventricles during the period of rapid expulsion, which appeared as an M-shaped splitting of the j wave. The amplitude of the waves after loading is usually increased, particularly during the period of rapid expulsion and rapid filling of the ventricles, and sometimes also during their active filling during the atrial contraction. This indicates an increase in the rate and force of expulsion during the moment of expulsion and an increase in the volume of blood arriving at the moment of rapid expulsion from the ventricles (see Figure 60).

Another group of athletes (weight lifters in classes I-III) was investigated directly during training under static load conditions. The kinetocardiogram was recorded in the prone position: before beginning of training, immediately and also ten minutes after performance of 1-2 exercises (pressing, snatching or jerking).

The nature of the changes after the exercises with the weights was similar to the changes in phase structure of athletes performing dynamic loads (three minutes running). True, the degree of increase in pulse frequency with the shortening of the systole and diastole was more expressed. Immediately after loading the weight lifters showed statistically reliable increases in pulse frequency, shortening of the systole primarily due to slow expulsion and shortening of the diastole (particularly the period of slow filling). In this group also, after ten minutes following completion of the exercises, the duration of the electromechanical systole and its components was completely restored to the initial level. Changes in the amplitude and configuration of curves were expressed as an increase in the h wave and particularly the j wave (by an average of 25-30%), and also as a splitting of these waves in connection with asynchronism of the contraction of the ventricles at the moment of isometric contraction or rapid filling.

Thus, in both groups the response to dynamic or static physical loading consisted of changes of the same nature (although with varying degrees of expression) with subsequent complete restoration of the duration of the systole even with relative shortening of the cardiac cycle. /213

Among athletes who had earlier shown sporadic extrasystoles, but showed proper rhythm at the moment of investigation, the phase structure of the cardiac cycle differed from the data produced by investigation of athletes of the preceding group, in that we found relatively longer duration of the electromechanical systole.

The test subjects in this group rather frequently showed splitting of the waves of the kinetocardiogram during the phase of isometric contraction and rapid expulsion, indicating asynchronism of the mechanical activity of the ventricles. The amplitude of the waves, reflecting the force of the cardiac contractions, did not differ from the values observed in persons with no disruption in heart rhythm.

For those athletes in whom we succeeded in recording extrasystoles during the investigation, the kinetocardiogram was studied in the main, extrasystolic and postextrasystolic cycles. Using the average values for this group, we did not succeed in discovering any essential differences in the duration of the phases in the extrasystole in comparison with the main cycle. Naturally, in most cases, particularly with ventricular localization of the extrasystole, a shortening of the diastolic phase before the extrasystole and an elongation of the diastole in the subsequent complex after the extrasystole were observed (Figure 61).

It is important to note that the amplitude of the waves on the kinetocardiogram during the extrasystolic and postextrasystolic cycles was frequently noticeably decreased. This indicates a decrease in the kinetic activity of the myocardium, particularly a decrease in the force of blood expulsion from the heart (see Figure 61). This indicator, we believe, can be used as a criterion for hemodynamic effectiveness of the cardiac contraction. A confirmation of this is the decrease in amplitude or even omission of the

pulse wave observed in recordings of the sphygmograms in the carotid, radial, digital and femoral arteries in these cases. Another proof of disruption of the mechanical activity of the myocardium in athletes with extrasystolic arhythmia is more frequent and sharply expressed asynchronism of the ventricles during the period of rapid expulsion, which has been documented by the appearance of M-shaped j waves on the kinetocardiograms, particularly after physical loads. The electrocardiogram sometimes shows a negative T wave in the postextrasystolic complex in these cases.

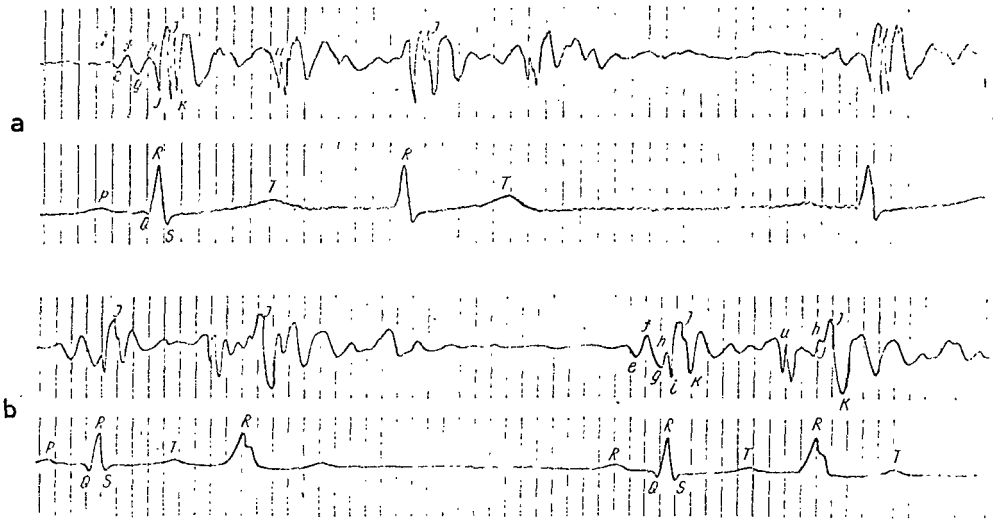

Figure 61. a, Kinetocardiogram of Athlete E-va. Extrasystolic complexes with low amplitude of j wave are noted; b, kinetocardiogram of Athlete M-a. Extrasystolic complexes arise after period of rapid filling of ventricles with blood, their amplitude is normal.

The most unfavorable indicators on kinetocardiograms (time and amplitude) were observed with early extrasysoles, particularly in cases of allorhythmia, grouped or partial individual extrasystoles. It is these extrasystoles which were frequently incomplete in the hemodynamic respect and /214 accompanied by disruption in repolarization of the myocardium with a decrease in heart kinetics according to the data of kinetocardiograms and other indicators.

Thus, kinetocardiographic investigation allows us to evaluate the kinetics of the heart of the athlete at rest, under the influence of physical loading, as well as in cases of disruption of the functions of the myocardium automatism, excitability, etc.).

Rheocardiography

Rheocardiography is a new method of investigating the cardiovascular system. It records the changes in electrical resistance of the body as a high-frequency current is passed through it related to the rhythmic activity of the heart and the movement of blood through the vessels. Mann (1937) was apparently the first to suggest that an alternating current bridge be used to study peripheral blood flow. He established that when a portion of the human body was introduced to one branch of a Wheatstone bridge, disruptions in the equilibrium of the bridge synchronous with the pulse are observed. Soon after the works of Mann, A. A. Kedrov (1941, 1949) recorded fluctuations in the electrical resistance of the body related to the work of the heart. In 1945, the Austrian scientists Holzer, Polzer and Marko published a monograph, in which they presented data on pulse fluctuations of the electrical conductivity of the body which they produced under physiological and clinical conditions. They connected the test subject in series into a circuit with a 16-kHz alternating current oscillator. The electrodes for recording the curve were placed as they are placed during the recording of an electrocardiogram in the second standard lead: "left arm -- right leg." They called their method rheocardiography. After the works of Holzer et al. (1945), the method of rheocardiography became widespread. /215

Rheocardiographic techniques. The circuits of devices for the recording of curves showing the fluctuations in electrical resistance of the body are usually divided into three types: bridge, differential and series or potentiometric circuits. It is assumed that it is most expedient to use series circuits, since they combine good tuning accuracy with comparative simplicity in usage and more standard recording amplitude (A. A. Kedrov, 1948; V. A. Karelin, 1957).

In the Soviet Union, rheograms have been recorded in recent years using the RGI-01 device, developed at the special design bureau "Biofizpribor" (L. G. Terekhova, V. I. Polishchuk, 1960).

Using the RGI-01 as an example, let us discuss the principle of operation of a modern rheograph. The rheograph consists of a high-frequency generator (30 kHz), an amplifier with gain control, a detector, a bridge measurement circuit and a power supply.

The patient is "connected" into one of the arms of the measuring bridge device, so that fluctuations in electrical resistance of the patient related to the work of the heart cause fluctuations in the high-frequency voltage in the measuring diagonal of the bridge. The amplitude modulated voltage is then amplified and detected.

The low-frequency signal separated is fed to the input of an electrocardiograph. The maximum sensitivity of the device is such that as the resistance is changed from 50 to 50.05 ohms (0.1%), an output signal of no less than 1 mv is produced.

In order to produce a calibrated signal, a calibrated resistance (0.01 ohm) is connected in series with the patient using a button contact.

Let us discuss briefly the problem of classification of methods of measuring fluctuations in the electrical resistance of the body, which are combined under the general term "rheography." The problem is that fluctuations in the electrical resistance of the body to high-frequency current may result, on the one hand, from periodic, rapid pulse oscillations, or on the other hand -- from slow and aperiodic fluctuations resulting from a change in the tonus of the vessels. On the basis of the fact that both types of oscillation may lead to changes in the electrical resistance of the body to high-frequency current which are both related to cardiovascular activity, all of these methods are properly classed as rheography. Taking this and also certain technical specifics which arise during the recording of periodic and aperiodic fluctuations in electrical resistance into consideration, we should probably divide rheography into rheosphygmography (recording pulse oscillations) and rheoplethysmography (Yu. Ye. Moskalenko and A. I. Naumenko, 1954) which is essentially identical to mechanical plethysmography. Combining these methods, as is done in certain investigations (A. B. Askent'yev, 1957) apparently should not be done. /216

One variety of rheosphygmography[1], allowing the recording of rapid fluctuations in the electrical resistance of the body, resulting primarily from the activity of the heart, is rheocardiography, which in turn is divided, depending on the type of application of electrodes for recording of the curve, into chest rheocardiography (brustwandrheokardiographie) or simply rheocardiography, and precordial rheocardiography (herznaherheokardiographie, after Heeger, 1957).

Physiological principles of rheocardiography. Let us discuss first of all the physiological analysis and capabilities of investigation of cardiac activity by the method of rheocardiography. The development of this method occurred in 1949-1950, when Polzer and Schuhfried introduced the chest lead rheocardiogram to practice, in which one of the large area lead electrodes is placed over the apex of the heart, the other -- either in the area of the right scapula (Polzer, Schuhfried, 1949) or on the right shoulder (Polzer, Schuhfried, 1950). The curve thus recorded was called the chest rheocardiogram and later simply the rheocardiogram.

The rheocardiographic curve has a clear, two-wave appearance, repeated in each subsequent cycle, remaining constant for any given test subject with similar recording conditions (Figure 62). On the curve, during its descending and ascending waves, we can distinguish seven points and six intervals. Three of the seven points are noted in all curves without exception (points 1, 2, 4), while the remaining points are encountered on curves in various percentages of cases (I. N. Ivanitskaya, 1961). /217

[1] The varieties of rheosphygmography include also rheoangiography and rheovasography.

Depending on certain morphological specifics, the rheocardiograms of healthy persons can be divided into two types: I and II (Garbini, cited by Nattero; Heeger et al., 1958; Polzer, Schuhfried, 1950). The characteristics of curves of type I are as follows: 1) amplitude of the curve in intervals 1-2 is great and exceeds the amplitude in sector 4-6; 2) point 1 is the deepest point of the curve; 3) the amplitudes of the two main waves, descending and ascending, are not equal. The characteristics of curves of type II are: 1) the amplitude of sector 1-2 is low in comparison with the amplitude of sector 4-6; 2) the deepest point on the curve is 4; 3) the amplitude of sectors 2-4 and 4-6 are approximately equal (see Figure 62).

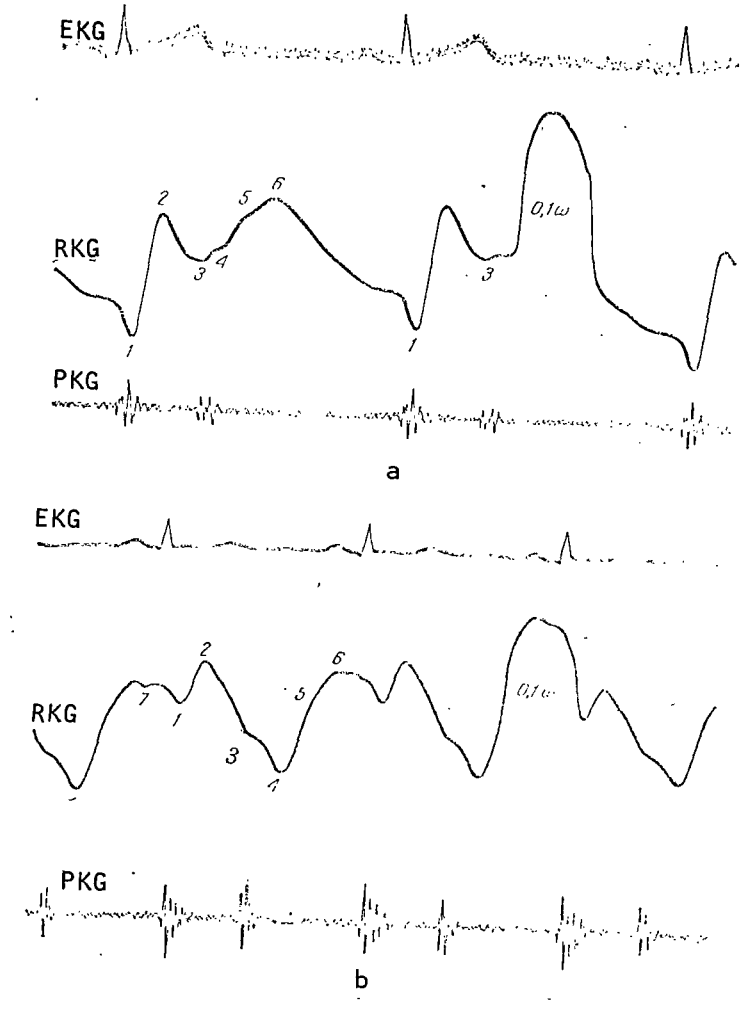

Figure 62. Chest Rheocardiogram of a Healthy Person. a, curve of type I; b, curve of type II

The genesis of rheocardiograms is not completely clear at this time. Polzer and Schuhfried (1949-1950), analyzing the cardiovascular system as a single system of electrical conductors with variously changing transverse cross sections and a definite length, assumed that the principal factor in the genesis of the curve was the changes in blood content of the heart and large vessels in the thorax. Based on these concepts, they interpret the curve as follows. The descending wave (1-4) is looked upon as arterial or systolic, while the ascending wave (4-6) is looked upon as veinous or diastolic. The elevation of the curve at point 1 is related to the beginning of expulsion of blood and results from an increase in conductivity in the aorta, which at this moment predominates over the decrease in electrical conductivity of the heart, since the aorta is the longest conductor in the "heart-vessels" system. The decrease of the curve at point 2 is related to the predominance of blood outflow from the area being measured over inflow to the heart. The elevation of the curve in interval 4-6 is explained in relation to an increase in veinous filling of the ventricles. Further, in correspondence to the new influx of blood to the atrium under conditions of uneven distribution of the blood, a minimum in electrical conductivity follows, and the curve after point 6 continues descending right down to the last elevation of the curve at point 1 (Vetter, 1959; Polzer, Schuhfried, 1950).

In addition to the opinions of Polzer, Schuhfried (1950, Vetter (1949) concerning the genesis of rheocardiograms in relation to changes in blood filling, the literature carries other concepts concerning the reasons for formation of this curve. Thus, the data of Matzdorf (1953, 1961) which he produced during the performance of model experiments, animal experiments and human experiments, indicate that the decisive factor in the genesis of the curve consists not of fluctuations in filling, but of fluctuations in the position of the heart and large vessels in the thorax and their movement during the cardiac contraction. Similar conclusions were reached by the Italian investigators (Furbetta et al., 1956). Our own observations (I. N. Ivanitskaya) performed in animal experiments and human experiments, lead us to conclude that the rheocardiogram reflects changes in the conductivity in the thorax resulting both from fluctuations in blood filling and from movements and displacement of the heart itself. It was found that the influence of heart movement was greater for animals, while the influence of displacement of blood masses was greater for humans. It is possible that this fact can be explained by specific features in the relationship of the heart volume and thorax in dogs and in man. A confirmation of the correctness of these assumptions can be found in the experimental investigations on frogs performed by Stoboy and Nussgen (1957). /219

The reasons for the appearance of two different types of rheocardiograms in healthy persons are not clear. Some investigators relate this fact to the fact that in some cases the vessels have a greater influence on the fluctuations in electrical resistance, while in other cases the activity of the heart itself is most important (Garbini). In addition to this, the opinion has been stated that the principal role in this case belongs to the value of

peripheral resistance; where this resistance is low, curves of type I are recorded, when it is high -- curves of type II are recorded.

There is interest in a comparison of the rheocardiogram with the phases of the cardiac cycle. Data (I. N. Ivanitskaya, 1961) produced in an investigation of 42 practically healthy persons by analysis of synchronously recorded rheocardiograms, electrocardiograms, phonocardiograms, sphygmograms of the carotid artery and longitudinal dynamocardiograms showed the following. Point 1 on the rheocardiogram in most cases corresponds to the beginning of expulsion of blood from the ventricles, which can be determined on the basis of comparison of point 1 with the results of polycardiographic calculation of the phases of the cardiac contraction and by comparing this point with the Z wave of the dynamocardiogram, which reflects the beginning of the phase of expulsion on this curve.

The completion of the true phase length of blood expulsion had no analogs on the rheocardiogram, while the completion of the acoustical systole (tone II) generally corresponded to point 3 on the rheocardiogram, while when this point was absent the beginning of tone II was projected along the lower third of the descending wave of the curve (see Figure 62). The correspondence between other points on the rheocardiogram and the beginning or ending of individual cardiac contraction phases was poor (I. N. Ivanitskaya, 1961). The mean duration of the stress phase, determined from the interval from the Q wave on the electrocardiogram to point I on the rheocardiogram, was 0.085 sec, which correlates satisfactorily with the data on the value of this phase produced using other methods of investigation (I. N. Ivanitskaya, 1961). /220

In an investigation of the hemodynamics of athletes, it might be interesting to evaluate the quantitative analysis of the rheocardiogram. Among the first works of this type is that of Holzer and Polzer (1951) who, comparing the amplitude of the systolic wave of the rheocardiogram with the value of the volume per beat, calculated using the method of Wezler and Boger (1937), concluded that there was a direct correlation between these quantities. The results of subsequent investigations performed in this direction were contradictory. Thus, Spillmann (1952) comparing data on the value of the systolic sector of the rheocardiogram recorded in the Holzer, Polzer and Marko lead (1945), under the influence of various effects, with the literature data on the volume per beat under similar conditions, concluded that there was no relationship between these quantities. At the same time, Vetter and Breu (1951), in clinical investigations, and Stoboy and Nussgen (1957) who recorded rheocardiograms under experimental conditions, established a direct relationship between the amplitude of the curve and the systolic volume.

One defect of these investigations was the absence of direct comparisons of amplitudes of rheocardiograms and simultaneously calculated values of volume per beat in the same test subject. Investigations of this type were made by I. N. Ivanitskaya and G. A. Glezer (1963). The amplitude of the systolic wave of the rheocardiogram calculated in absolute units (ohms) was

compared to the value of the volume per beat calculated by the Stewart-Hamilton method. Both sectors of the systolic wave (1-2 and 2-4) were compared with the volume per beat, as well as the amplitude of the entire systolic wave as a whole (sector 1-4). No dependence was noted between the value of the volume per beat and the amplitude of sector 1-4, although the amplitude of the ascending sector of the systolic wave (1-2), expressed in ohms, correlated satisfactorily with the value of the systolic volume (correlation coefficient + 0.52, $P < 0.01$).

The establishment of this correlation is of interest for clarification of the origin of the rheocardiographic curve, indicating a great role in its formation of the oscillations involved in blood filling. Also, the determination of this dependence makes it possible to approach to a certain extent a qualitative definition of an important hemodynamic indicator, the blood volume per beat.

Indirect confirmation of the possibility of comparative evaluation of /221
changes in the volume per beat using rheocardiography is provided by the data produced by I. N. Ivanitskaya (1962) in a rheocardiographic investigation of a group of athletes who held their breath both in the inhalation and in the exhalation phase. A presumption used in this investigation was the variation in systolic volume during inhalation, amounting to an increase of an average of 25% (De Lalla, Brown, 1950; De Soldatti, 1951).

Comparison of the values of the ascending sector of the rheocardiogram calculated in ohms showed that the mean value of this interval during inhalation exceeds the mean value of this interval calculated when recorded upon exhalation by an average of 23%. The results of the investigation thus showed that changes in the ascending sector of the rheocardiogram were similar to changes in the value of volume per beat. It might be thought that quantitative analysis of the rheocardiogram would be expedient for observations of dynamic changes in cardiac activity of athletes.

Precordial rheocardiography. In recent years in cardiographic investigations, a variety of rheocardiography called precordial rheocardiography, or "herznaherheokardiographie", has become widespread. The first variety of this method was introduced by Heeger (1957), after which the method was developed in detail by Yu. T. Pushkar' (1959, 1961). Precordial rheocardiograms are recorded using small area electrodes (1 × 1.5 cm), one placed in the fourth intercostal space on the left of the sternum, another in the area of the apex of the heart. It is believed that the formation of precordial rheocardiograms (PRKG) is practically determined by changes in the activity of the heart itself, and that the curve recorded in this manner is similar to an electrocardiogram of the area of the left ventricle. In its appearance, the precordial rheocardiogram is reminiscent of a type II chest rheocardiogram (Figure 63) and is characterized by the presence of two waves -- the descending systolic and the ascending diastolic waves.

It is interesting to note that curves similar to type I chest rheocardiograms are generally not encountered during recordings using the precordial lead, and are generally recorded only in a few individual cases. Apparently, precordial rheocardiograms of type I are produced in these cases as a result of improper application of the electrodes and the resulting increase in the role of the vascular factor in the formation of the curve.

The data from the literature indicate considerable capabilities for precordial rheocardiography in the performance of phase analysis of the cardiac contraction. In particular, it has been noted that this curve can be used to determine the length of the stress phase and the expulsion phase (Yu. T. Pushkar', 1961; Heeger, 1957). The results of our own investigations (A. D. Budkov, 1962) lead us to the conclusion that this curve in a high percentage of cases shows only point C, corresponding to the beginning of phase of expulsion of the blood from the ventricles (92%), and point E, corresponding to the beginning of the II tone (95%). The precordial rheocardiogram, when recorded synchronously with electro- and phonocardiograms, can be used to determine the duration of the stress phase (interval from Q wave of the electrocardiogram to point C on precordial rheogram), the duration of the phase of isometric contraction (interval from initial high-frequency oscillations of tone I to point C of precordial rheogram) and the duration of the interval from the beginning of expulsion to the beginning of tone II, including the summary duration of the period of expulsion and the protodiastole (interval C-E). /222

A comparison of the duration of the stress period (T) and the phase of isometric contraction (IC) using the precordial rheocardiogram with the duration of these periods calculated using the polycardiographic method, as well as direct comparison of point C on the precordial rheocardiogram with the Z wave of the dynamocardiogram (see Figure 63), showing the beginning of the phase of expulsion on this curve, indicates that the moment of beginning of this phase is reliably determined on the precordial rheocardiogram. Other points on the precordial rheocardiogram are not as strictly definable as points C and E, and their precise correspondence to individual moments of cardiac activity has not been determined. In the literature, there are indications that in addition to temporal analysis of the precordial rheocardiogram, it is also expedient to perform quantitative analysis of this curve, since, as the results of certain investigations indicate, the value of the systolic sector of the precordial rheocardiogram changes in proportion to changes in the volume per beat (Yu. T. Pushkar', 1961). /223

Usage of the method of precordial rheocardiography in the investigation of hemodynamics in persons involved in sports apparently can be facilitated by the fact that when certain recording conditions are observed (proper location of electrodes, careful lubrication of electrodes with a special paste, reliable fixation of electrodes, achieved by using special light electrodes with plexiglass mounts, precise balancing of the instrument), recording of the precordial rheocardiogram can be performed for human subjects under conditions of muscular work. A. D. Budkov (1962) recorded the precordial rheocardiogram of practically healthy person and athletes of

high qualification (55 test subjects) before and after measured muscular work. He recorded the following simultaneously: rheocardiogram, electro-, phono- and dynamocardiogram and carotid artery sphygmogram (see Figure 63). This investigation made it possible to determine dynamic changes in the duration of the stress period, duration of isometric contraction, as well as changes in the interval from point C to point E, indirectly indicating changes in the duration of the phase of blood expulsion from the ventricles[1].

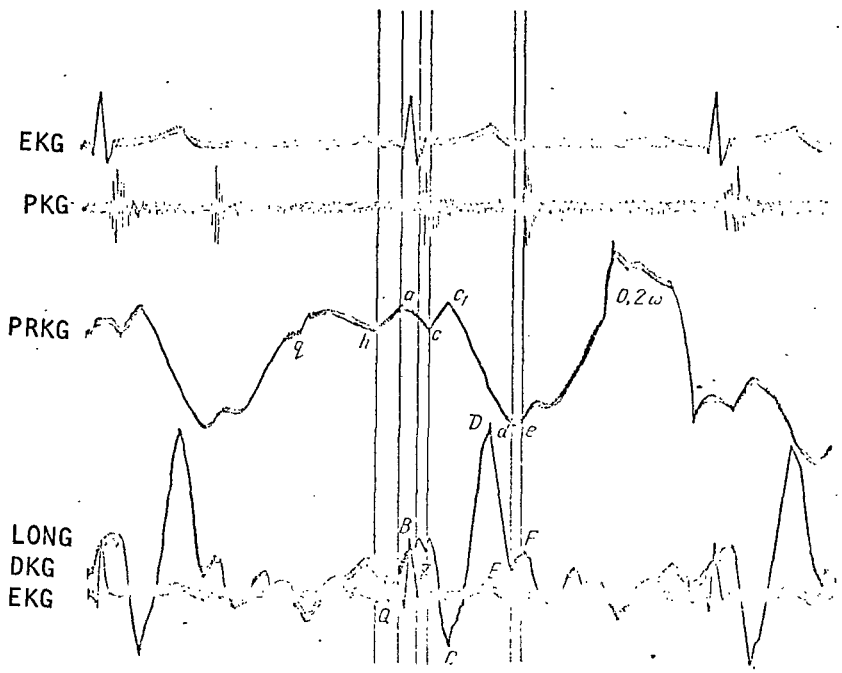

Figure 63. Precordial Rheocardiogram

The results of the investigation performed also demonstrated the expediency of using precordial rheocardiograms in dynamic investigations in order to determine changes in the value of blood volume per beat. It was found that the amplitude of the systolic sector, calculated in ohms, generally increased under the influence of muscular work (Figure 64). The increase in the amplitude fluctuated between 40 and 133%. /224

[1]. Changes in this period are indirect, and therefore not a direct reflection of changes in the duration of the expulsion phase, since it includes the protodiastolic period, the inclusion of which in the duration of expulsion is physiologically unfounded.

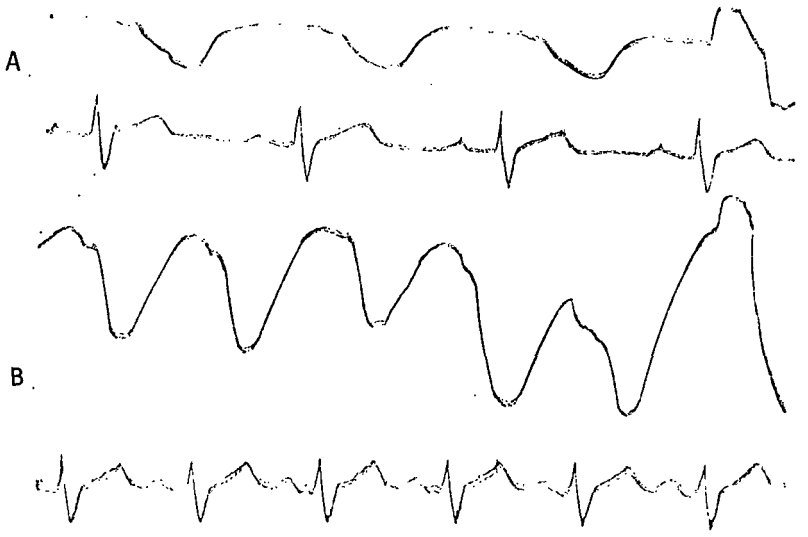

Figure 64. Precordial Rheocardiogram Recorded Before (A) and After (B) Physical Loading. An increase in the amplitude of the descending systolic wave in the curve after loading is noted.

In conclusion, let us discuss briefly the possibilities for using the rheographic method of investigation in studying peripheral blood circulation and the specifics of blood flow in individual organs.

Peripheral rheosphygmograms. The formation of the peripheral rheosphygmogram or simply the extremity rheogram is determined by fluctuations in the filling conditions of the vessels and the related fluctuations in their cross sections (A. A. Kedrov, 1941; I. N. Ivanitskaya, 1962; Nguen Zun, 1964; Bonjer et al., 1952; Kaindle et al., 1959; Matzdorf, 1961). Of principal significance in the formation of this curve are the fluctuations in blood filling of the large arteries. This is particularly indicated by results of investigations involving recording of rheograms in an experiment under conditions of transverse compression of the femoral vein and femoral artery, as well as data produced in man during recording of a rheogram under conditions of compression of the extremities with a collar in which various pressures were created (I. N. Ivanitskaya, 1962). Analysis of the curves thus recorded showed that interruption of arterial blood flow leads to /225
impossibility of recording any curve, while interruption of veinous flow had a considerably lesser influence on the course of the curve. Recording of rheograms under experimental conditions has demonstrated the relationship of the amplitude of the rheogram wave to the electrolytic composition of the blood, which determines its electrical conductivity. This has been determined by investigations establishing an increase in the amplitude of the

rheogram in an experiment immediately after intravenous injection of a 5% salt solution (I. N. Ivanitskaya, 1962) and a decrease after injection of gum arabic into the blood (Bonjer et al., 1952).

An important advantage of the rheographic method of investigation is its great sensitivity, the possibility of standardization and calibration of the curve, and the recording conditions, which do not exhaust the test subject. In an investigation of peripheral blood flow using rheography, two leads of the peripheral rheogram are generally used -- the longitudinal and transverse leads. In order to record a longitudinal rheogram, the electrodes are placed on the extremities, one proximally, the other distally, while the recording of a transverse curve requires that the electrodes be placed at the same level of the sector being investigated, symmetrically opposite one another.

The usage of a transverse rheogram makes it possible to localize vascular damage precisely (Kaindle, 1959; Polzer et al., 1960), while analysis of a longitudinal rheogram can provide valuable information concerning the nature of the damage (functional, organic) (Kaindle et al., 1959) and, furthermore, and which is particularly important in the performance of investigations in practically healthy persons, the longitudinal rheogram can indicate the condition of the vascular tonus and elastic properties of the vessels (V. A. Karelin, 1957; Kaindle, 1959) and to some extent can give information on the condition of the muscular system in the extremities (Fiorio, 1960).

In addition to the qualitative analysis of rheograms (evaluation of amplitude and morphological specifics of the curve), in recent years ever greater significance has been attached to quantitative analysis of the curve. It has been established that the value of the systolic wave on the rheogram, expressed in ohms, reflects the value of the relative pulse volume, ОП, the Jantsch-Schuhfried indicator (Caponetto, 1961, 1962; Matzdorf, 1961). This indicator is calculated as the product

$$ОП = A \cdot 100/R \cdot t, \tag{41}$$

where A is the amplitude of the systolic sector of the rheogram;
R is the resistance according to the indications of the device;
t is the duration of the cardiac cycle (in sec).

Investigations have shown that the value of the relative pulse volume undergoes statistically reliable changes with various changes in the position of the body, functional experiments and various pharmacological influences.

Based on data from the literature, we can see that in addition to the usage of peripheral rheography of the extremities, it is also promising to use rheographic investigation of blood flow to the brain and internal organs in clinical-physiological investigations. /226

The data of A. S. Loginov and Yu. T. Pushkar' (1962) concerning changes in the rheogram of the liver might be of interest for the investigation of athletes, since we know that hard training sometimes causes disruptions of blood supply to the liver.

Tachooscillography

The measurement of the arterial pressure has become firmly entrenched in clinical and sports-medical practice. The most commonly used methods are the bloodless or indirect methods, based on determination of the height of the external pressure necessary to change or completely halt blood flow as an artery is compressed.

At the present time, of all the methods for bloodless determination of arterial pressure suggested, the auditory and oscillographic methods have become practically significant. In the auditory method, suggested by N. S. Korotkov (1905), the indicator used to determine the pressure, as we know, consists of the auditory phenomena arising as the vessels are compressed. Due to its simplicity and accessibility, this method is generally recognized, and is unequaled in practical significance. However, in spite of this, the auditory method has a number of essential defects. Thus, the auditory method for the measurement of arterial pressure is subjective to a significant extent. This is confirmed by a special investigation performed by Wright, Schneider and Ungerleider (1938), who organized collective measurement of blood pressure simultaneously using a single phonendoscope. The variations in the indications of different doctors as concerns diastolic pressure reached 20 mm Hg, systolic pressure -- 16 mm Hg.

Furthermore, the auditory method gives a very approximate idea only concerning the maximum and minimum pressures. Finally, auditory phenomena are nonconstant, and at times their sequence is disrupted, to the point of complete omission of certain phases "auscultative omissions." As the blood flow changes, such anomalies as the "endless tone" (M. V. Yanovskiy, 1911; D. O. Krylov, 1930) are common. The data of the auditory method, as a rule, do not agree with the indications of other indirect and direct (blood) methods (G. F. Lang, S. M. Mansvetov, 1908; A. S. Lebedev, 1911; Bergen et al., 1954).

The oscillographic method of determination of arterial pressure was first suggested by Marey in 1878, due to its objectivity; it seemed that it /227
had broad promise in comparison with the auditory method. The pressure indicator in this method is the change in the magnitude and form of oscillations of the vascular wall under the influence of variations in pressure in the collar. These fluctuations are perceived by a special differential manometer and automatically shown in the form of a curve -- the arterial oscillogram. This method allows not only the maximum and minimum, but mean arterial pressure to be determined.

However, the technical defects of the devices and absence of generally accepted methodology for decoding oscillograms have prevented broad introduction of this method in clinical or sports medicine. Also, we should take into consideration the opinion of G. F. Lang (1959) who indicated that the oscillographic method ... "is technically imperfect and the principles upon which it is based are doubtful."

Due to the defects in ordinary oscillography, the search was continued for different indirect but graphic methods of determining the arterial pressure.

In 1934, N. N. Savitskiy created a new design for a differential manometer, with high sensitivity (80-100 Hz). Using this manometer, it became possible to record a curve of the rate of filling of arteries with blood during the period of the systole and emptying of arteries during the diastole under the influence of increased pressure in a collar. Actually, the N. N. Savitskiy manometer was first used to record the actual differential curve.

Emphasizing the differentiating specifics of this type of oscillogram, N. N. Savitskiy called them velocity curves or tachooscillograms and the method of determination of pressure came to be called tachooscillography.

On the basis of broad experimental investigations and deep theoretical analysis of the form of the tachooscillogram, N. N. Savitskiy developed a principally new method for their interpretation.

The difference in principle of the tachooscillographic method from all other methods is that it determines the arterial pressure regardless of the properties and condition of the vascular walls. The advantages to the method also include its objectivity and good accuracy (error in determining pressure not over 5-6 mm Hg).

The tachooscillogram, in addition to the maximum and minimum pressures, in all cases clearly shows the average pressure. This method first allowed the recording of the lateral or piezometric systolic pressure experienced by arterial walls by an indirect method. Thanks to this, we can calculate the stroke pressure, by which, as we know, the maximum pressure exceeds the actual lateral pressure on the arterial walls (Fabre, 1925; Muller, 1924; G. F. Lang, 1930). /228

Tachooscillographic techniques. Tachooscillograms are recorded using a universal photographic recording device, the "mechanocardiograph" produced by the "Krasnogvardeyets" plant on the basis of the N-106 universal recording device. The manometers of the mechanocardiograph (including the differential manometers) have high natural frequencies (90-100 Hz).

The tachooscillogram is recorded as pressure is increased in the collar which doubtless is a closer reflection of the condition of local circulation than the ordinary method of recording during decompression. The increase in

the pressure in the collar may be at various rates (2-3-5-6.5 mm Hg per sec). Recording of the tachooscillogram is performed at low chart movement speeds -- 5 or 10 mm/sec. At the same time as the differential curve, a curve of the pressure in the collar is recorded, as well as a radial artery sphygmogram and a time mark (Figure 65).

Figure 65. Tachooscillogram (Postdicrotic Type). Top curve, radial artery sphygmogram; center, tachooscillogram; bottom, curve of pressure in collar and horizontal line corresponding to initial pressure of 40 mm Hg. Arrows show descriptive characteristics of pressure (left to right): minimal, mean, lateral systolic and maximal.

The tachooscillogram is read using the character of the changes in diastolic curve sectors (see Figure 65).

The minimum pressure is determined at the moment of a clear drop in the diastolic portion of the differential curve below its zero position. Insignificant and unstable drops in the curve are not considered (see Figure 65). On this tachooscillogram, the differential curve is lower due to deepening of its postdicrotic portion. This postdicrotic type of curve is most frequently formed in older persons. For younger persons, a predicrotic type of curve is more characteristic, in which the diastolic portion is dropped due to a predicrotic wave (Figure 66).

The mean hemodynamic pressure corresponds to the moment of appearance of the thick portion ("hump") on the ascending portion of the diastolic sector of the curve. Since the "hump" appears at the moment of first closure of the vascular lumen beneath the collar, N. N. Savitskiy called it the "closure wave." This closure wave is always clearly formed on velocity curves, usually corresponding with the most positive oscillation, if it is clearly defined.

The determining characteristic of the lateral systolic pressure is the highest negative oscillation after the closure wave. During this time, the optimal possible blood volume penetrates to the vessel squeezed beneath

the collar, the volume which is expended at the maximum possible rate, and the pressure in the collar corresponds to the true lateral or piezometric pressure.

The final systolic or maximum pressure is recorded at the moment of disappearance of pulsation of the radial artery. At this time, the pressure of the collar equalizes not only the lateral pressure, but also the kinetic /229
energy of movement of the blood, and the lumen of the vessel remains closed even during the systole of the heart.

In order to use the tachooscillogram to find the numerical value of pressure, we must drop a vertical line from the descriptive point on the differential curve. The distance from the point of intersection of this vertical line with the pressure curve to the horizontal line is measured in mm. This number is doubled, 40 is added and the height of the arterial pressure in mm of mercury is thus produced.

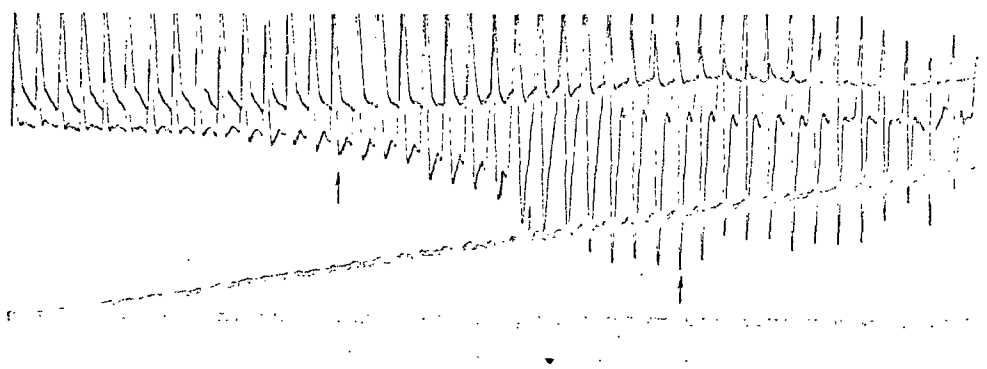

Figure 66. Predicrotic Type of Tachooscillogram. Symbols same as on Figure 65.

On tachooscillograms recorded after physical exercise during increased /230
heart work, respiratory oscillations in the descriptive characteristics of the pressure waves frequently arise. According to the observations of N. N. Savitskiy (1956), these oscillations in pressure are expressed in patients suffering from hypertension at rest. According to our data, after loads the respiration has very little influence on the value of minimal and mean pressure (fluctuations not over 5-6 mm Hg).

Most frequently the lateral systolic pressure fluctuates over considerable limits (up to 10-20 mm Hg). In these cases, according to the recommendations of N. N. Savitskiy, we should find the mean value among the two or three greatest negative oscillations.

Tachooscillography in medical testing. On the basis of generalization of the results of tachooscillographic investigations of various authors, N. N, Savitskiy (1956) believes that in healthy persons the lateral systolic

pressure lies between 90 and 100 mm Hg. The difference between the final and lateral systolic pressure, the so-called "hemodynamic impact" or stroke pressure, averages 15 mm Hg with a limit of fluctuations of ±5 mm Hg. The mean pressure in healthy persons has greatest constancy and its normal range is 80-90 mm Hg. The minimum pressure is also quite constant, near the constancy of mean pressure. The difference between the mean and minimum pressures, according to N. N. Savitskiy (1956), averages 22.8 mm Hg. In healthy persons, it is always greater than the difference between mean and lateral pressure, which averages 15.2 mm Hg.

The tachooscillographic method was the first to allow well founded elimination of possibility of pathology in healthy trained athletes in cases of moderate increases in arterial pressure due only to an increase in the hemodynamic impact of 25-30 mm with normal minimal, mean and lateral systolic pressures (V. I. Kuznetsov, 1947; V. P. Nikitin, 1959; Yu. I. Kuznetsov, 1956).

All authors using the tachooscillographic method for the investigation of athletes at rest have indicated that they did not find any essential differences from the literature data on tachooscillographic investigations of healthy persons not involved in sports (Yu. I. Kuznetsov, 1956; N. V. Savina, 1958; Ye. Ya. Likhachevskaya, 1959; V. V. Vasil'yeva, 1953).

In comparing the data of trained and untrained athletes at rest, a tendency has been observed to some decrease in the minimal, mean and lateral systolic pressures in trained athletes (Yu. I. Kuznetsov, 1956). In comparing the height of the maximum pressure, lower figures were found for long-distance runners, i.e. athletes whose training includes many exercises /231
for the development of endurance. In trained swimmers, rowers, boxers, and weight lifters this tendency was not noted, and the height of the maximum pressure was found the same as in untrained persons.

During functional experiments involving physical loadings, and also during various sports exercises, tachooscillographic data have shown that the lift height of the maximum pressure is influenced by the stroke pressure, which increases in parallel with the intensity of muscular work. The lateral systolic pressure is increased much less than the maximum pressure and returns to its initial level more rapidly.

In trained persons, loads have little influence on the average and minimal pressures. Even after very intensive high-speed loads (running 400 m), in spite of the sharp increase in maximum pressure to 200-220 mm Hg, the average pressure increases only slightly by 5-15 mm Hg. The minimum pressure, according to the data of tachooscillography, also changes little and has clear stability, corresponding to the mean pressure stability.

After loading, the mean and minimal pressure return to their initial values most rapidly, followed by the lateral systolic and finally the maximum pressures (Figure 67).

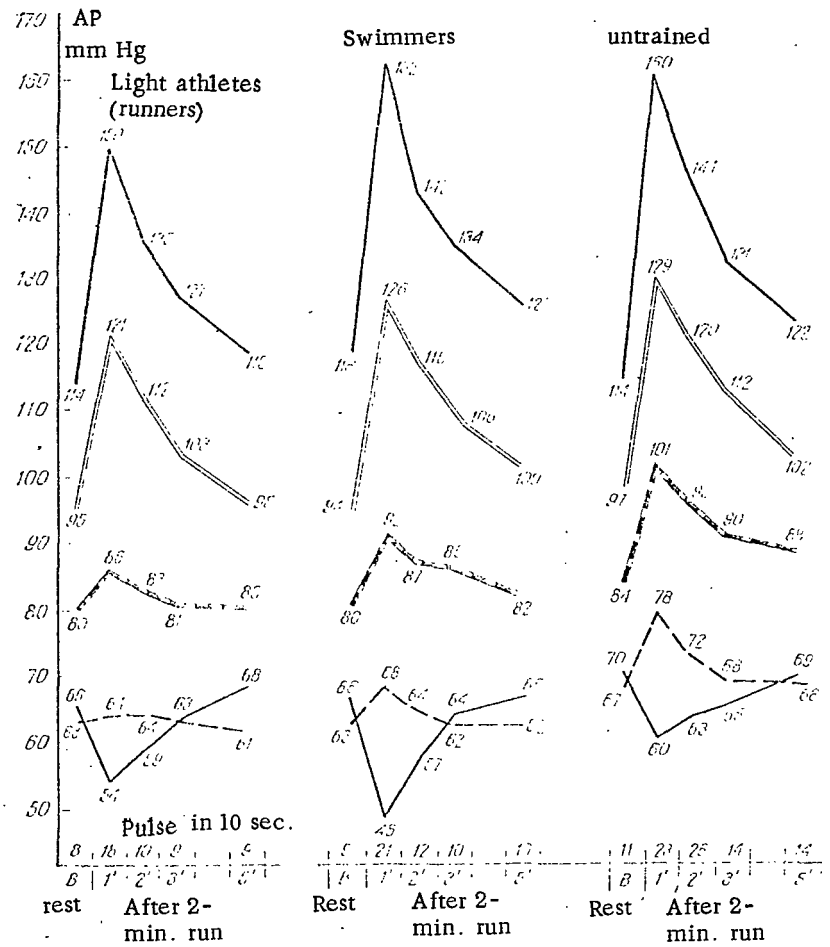

Figure 67. Changes in Pressure During Functional Experiment with 2 min Running in Place According to Tacho-oscillographic Data (Mean Data of Groups of 12 Persons Each). Solid line, maximum pressure; double line, lateral systolic pressure; heavy dotted line, mean pressure; dotted line, minimum pressure; thin solid line, minimum pressure after Korotkov

Some time after the finish, the maximum, as well as lateral, mean and minimum pressure may drop below the initial figures before the run. This is observed after long-distance running.

According to our data, there is no strictly expressed interrelationship between the changes in minimum and mean pressures: with a considerable (after loading) increase in the mean pressure, the minimum pressure increases also. If the changes in minimum pressure are less, the mean pressure also remains essentially unchanged. These data agree with the results of

theoretical calculations, experimental investigations and tachooscillographic observations of N. N. Savitskiy (1963). In contrast to the conceptions properly held, N. N. Savitskiy believes that under physiological conditions the value of true minimum and lateral systolic pressure, as well as mean pressure, are distinguished by their comparative constancy and that there is a definite dependence between them and the mean pressure.

A comparison of the results of investigation of well trained runners, swimmers and untrained athletes allowed two variants of combinations of reactions of the minimum and mean pressures to be distinguished during a functional experiment involving two minutes run[1] (see Figure 67).

Variant I. Minimum pressure remains unchanged or fluctuates slightly /232
within limits of 10 mm Hg. Mean pressure also remains unchanged or increases, but not over 10-12 mm Hg, and absolute value does not exceed 90-95 mm Hg, i.e. does not exceed limits of physiological fluctuations at rest.

Variant II. Minimum pressure generally increases by more than 10 mm Hg. Mean pressure also increases and, as a rule, by over 15 mm Hg, its absolute value exceeding 95-100 mm Hg.

The nature and quantitative expression of the interdependent changes of minimum and mean pressures are related to the general state of training of the athlete and his special training for the type of physical load involved in the functional experiment.

As we can see from comparison of average data (see Figure 67), light athletes with good preparation for running exercises, when subjected to a two-minute run in place, show smaller changes in all values and a more rapid return to the initial values than athletes not trained for running (swimmers) or untrained athletes. Runners, as a rule, show the first variant of the reaction of minimum and mean pressures.

In well trained athletes not trained for running (swimmers) and untrained athletes, the maximum and lateral systolic pressures increase in approximately the same manner. However, the minimum and mean pressures in swimmers change less than in untrained persons, in which primarily the second variant of fluctuations in these pressure values is observed.

The considerable stability of the mean and minimum pressures under loading indicates better agreement of quantitative and qualitative changes on the part of individual portions of the circulatory system (heart, large vessels, precapillary lumen).

[1] The tachooscillograms were recorded after completion of the muscular work -- Ed.

On the basis of our data, the first variant of the combined reaction of minimum and mean pressures can be looked upon as the more favorable variant, indicating good functional adaptability of the cardiovascular system of the athlete to the given load. The second variant of the reaction of minimum and mean pressures is less favorable.

Let us discuss in conclusion the comparison of data concerning arterial pressure determined with the tachooscillogram and by the Korotkov auditory method.

Under rest conditions, rather close correspondence of the figures for minimum and maximum pressures determined by the two methods was found: of 320 investigations which we performed, the divergence did not exceed 18 mm Hg.

The maximum tachooscillographic pressure, as a rule, is higher than the /234
auscultative maximum by an average of 5-6 mm Hg. The tachooscillographic minimum pressure, in a slight majority of the cases, is lower than the auscultative minimum, also by 5-6 mm Hg. At the same time, the true pulse amplitude determined tachooscillographically by the difference between the lateral systolic and minimum pressures is always 15-20 mm Hg less than the figure produced by auditory definition.

In comparing the indications of the two methods after various physical loads in relationship to the maximum pressure, good correspondence is observed with the tachooscillographic data exceeding the auscultative data by 5-6 mm Hg. As concerns the minimum pressure, after loading the indications of the two methods diverge quite sharply at times. This is manifested most notably during the first few minutes of the recovery period after brief and very intensive sports loadings (100-200 m run), when these divergences are retained for 5-10 min. Under these conditions, the auscultative method shows a sharp reduction in minimum pressure (by 40 mm Hg and more), and the so-called zero minimum pressure or "continuous tone" is frequently observed. At the same time, tachooscillographic investigations under these conditions usually show fluctuations in minimum pressure of 10-15 mm Hg, i.e., no important changes are noted (Table 18).

Thus, the data of tachooscillography do not confirm the opinions /235
concerning the nature of fluctuations in the minimum pressure during loads based on indications of the auscultative method and widely accepted in sports medicine. These data indicate the impossibility of using the Korotkov auditory method under conditions of investigation after physical loading to evaluate changes in the minimum arterial pressure.

Sphygmography

Sphygmography is one of the oldest physiological methods used to investigate the cardiovascular system of man. In the last decade, this method has once more become widespread in connection with progress in

medical measurement equipment. Sphygmography is required to make a judgment concerning the functional state of the arterial system in man. In connection with this, its usage in sports medicine is quite promising.

TABLE 18. HEMODYNAMIC CHANGES DURING INTENSIVE HIGH-SPEED WORK ACCORDING TO TACHOOSCILLOGRAPHIC DATA

Method of investigation	Light athlete S-n, 26 years old, I class	At rest	After 400-m run (51.8") 3'	10'	15'
	minimum	63	58	60	60
	average	80	90	85	84
	maximum	94	140	130	100
Savitskiy's tacho-oscillographic method	maximum	115	183	160	130
	pulse	31	82	70	40
	stroke	21	43	30	30
Korotkov's auditory method	maximum	115	180	150	125
	minimum	70	0	0	55
pulse after 10 sec		9	20	19	18

The sphygmographic method is used to record vibrations of the arterial wall resulting from the propagation of the pulse wave through the vessels. Recording of these oscillations is performed using transducers which convert the mechanical oscillations to electrical oscillations. Sphygmography uses quite varied transducers: pizeoelectric, inductive, capacitive, tensometric and other types.

We must differentiate between direct and volumetric sphygmography. In the first case, we record the pulse oscillations using a pelot or funnel with air transmission. In the second method, the volumetric changes in the body sector being studied are measured using a collar. Direct and volumetric sphygmograms are practically synchronous, but may differ slightly in form.

In recording a volumetric sphygmogram or direct sphygmogram with air transmission, we must take into consideration the rate of propagation of the pressure wave in the connecting tubes. It is practically equal to the speed of sound. Air transmission should not exceed 1 m, since wave processes may appear in longer lines, distorting the sphygmographic information. In addition to this, the appearance of the sphygmogram is influenced by bends and angles in rubber tubes acting as transmitting lines.

Electric transmission sphygmographs are doubtless promising for sports medical investigations. In these devices, the transducer is placed in the pelot portion of the device, i.e. the conversion of mechanical quantities to

electrical quantities is performed directly at the point of recording of the pulse wave. The electrical signal formed in the transducer is transmitted by /236 wires to the amplifying and recording portion of the device. No distortions of the signal arise with this type of transmission.

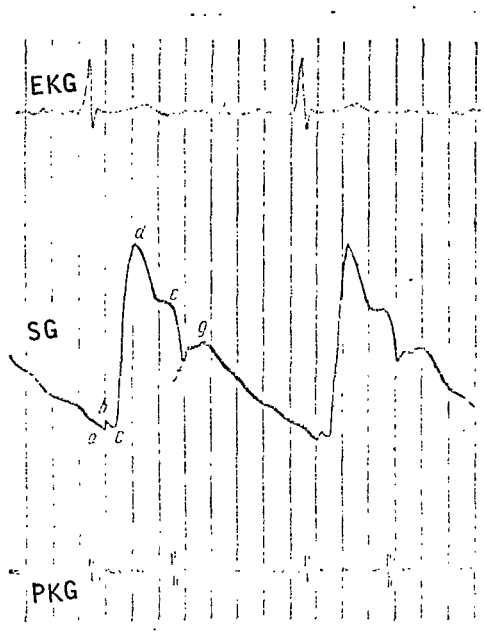

Figure 68. Synchronous Recording of Central Pulse Curve (SG), Electrocardiogram (EKG), and Phonocardiogram (PKG). Analysis in text.

The usage of such transducers in sphygmography has made this method useable in a broad network of treatment and preventative medicine institutions and medical-physical culture dispensaries. Among the domestic devices allowing high-quality sphygmograms to be recorded we can note the N. N. Savistkiy mechanocardiograph, the two-channel sphygmographic device developed at the "Biofizpribor" special design bureau and the sphygmograph manufactured by the "Krasnogvardeyets" plant.

The sphygmograms recorded using piezoelectric transducers, particularly those manufactured on site, are usually low in quality. The capabilities of the sphygmographic method are based on the analysis of the form of the pulse wave and investigation of the rate of propagation of the pulse wave through various vessels. All of these data allow objective evaluation of the elastic properties of the arterial vessels and the changes which they undergo under the influence of various types of physiological actions.

Pulse wave. The oscillations of the arterial wall recorded as a sphygmogram are the final result of the interaction of various physical processes occurring in the vascular system after arrival of the systolic blood volume. Among these processes we must include changes in intravascular pressure, changes in arterial wall tension, wave processes in the arterial system, displacement of the mass of blood within the system, interference phenomena, etc.

Due to the large number of factors which determine the form of the pulse wave and their variable nature, the appearance of the sphygmograms recorded from various arteries may differ significantly. Therefore, it is common to /237 speak of the form of the central pulse and the form of the pulse in the

peripheral arteries. The pulse in the aortal arc, the carotid and subclavian arteries is referred to as the central pulse.

A characteristic sphygmogram of the carotid artery (or the carotid pulse) is shown on Figure 68. This curve, generally, shows a small preliminary oscillation -- the a-b-c wave -- which occurs, according to Frank (1899, 1905), in connection with isometric contraction of the heart. This is followed by a sharp anacrotic elevation (c-d), related to the movement of blood into the aorta. The duration of the anacrotic rise in the carotid pulse under normal conditions characterizes the natural frequency of the aortal compression chamber[1]. The sector of the curve d-e describes the further behavior of the vascular wall during the phase of expulsion of blood from the heart. The sphygmogram during this time depends on the interrelationship between the influx of blood to the aorta and the efflux from it. At moment e, the expulsion phase is essentially completed (as well as the mechanical ventricular systole).

The e-f-g incisure on the curve of the central pulse reflects the mechanical processes related to the closure of the semilunar valve of the aorta: point f of the incisure corresponds to the moment of complete closure of the valve, while point e corresponds to the beginning of closure.

The later diastolic waves are unstable in nature, and their physiological genesis is not understood.

As we can see from Figure 69, the form of the pulse waves recorded from the aortal arc and subclavian arteries differs slightly from the appearance of the carotid pulse curve.

In practice, the central pulse curve is frequently recorded simultaneously with the electrocardiogram and phonocardiogram (see Figure 68). This allows calculation of the duration of the principal phases of the ventricular systole.

The carotid pulse curve itself allows us to determine the duration of isotonic contraction (period of expulsion of blood, of the left ventricle (c-e on Figure 68) and the duration of the protodiastolic period (e-f on Figure 68).

The duration of the period of expulsion of blood from the ventricles calculated from the sphygmogram is compared with its proper length calculated from formula (33), and thus the deviation of the measured quantity from the normal value is determined.

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[1] The term "aortal compression chamber" represents an arbitrary concept -- the windkessel, used in the theory of Weber and Frank.

The concept of "proper duration" is introduced since the time of isotonic contraction in the ventricles depends primarily on the frequency of heart contractions; it is shorter, the higher the pulse frequency, and vice versa. /238

The duration of the protodiastolic period, determined from the central pulse curves, fluctuates over very narrow limits: 0.03-0.04 sec.

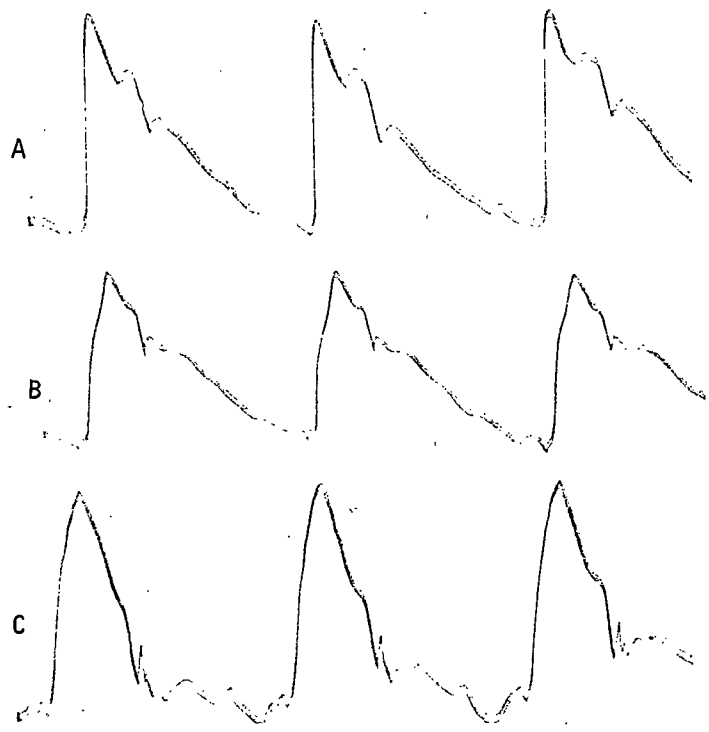

Figure 69. Various Forms of Central Pulse Curves. A, carotid artery sphygmogram; B, subclavian artery sphygmogram; C, aortal arc sphygmogram.

Certain authors have attempted to use the central sphygmogram to separate the period of expulsion into phases of maximum and reduced expulsion. This separation first began to be performed by Wiggers (1921), who used curves of pressure in the principal vessels. However, according to our observations, sphygmography is unsuitable for these purposes. This follows from the data shown in Table 19, from which we see that the duration of the anacrotic and catacrotic portions of the sphygmic wave recorded at the level of the aortal arc, carotid and subclavian arteries differs for one and the same test subject. /239

TABLE 19. DURATION OF SECTORS OF CENTRAL SPHYGMOGRAMS

Point of pulse recording	Duration of expulsion period, sec	Duration of anacrotic rise, sec	Duration of catacrotic descent, sec
Carotid artery	0.235	0.084	0.150
Subclavian artery	0.236	0.124	0.113
Aortal arc	0.232	0.108	0.124

However, it is these components of the sphygmic wave which are suggested for usage to measure the duration of phases of maximum and reduced expulsion.

The appearance of the central pulse curve changes with certain forms of pathology (M. A. Abrikosova, 1964). For example, with aortal stenosis, the anacrotic rise is slow and wavy (Figure 70). With low elastic resistance, the anacrotic rise of the curve is steep, there is a deep incisure, etc.

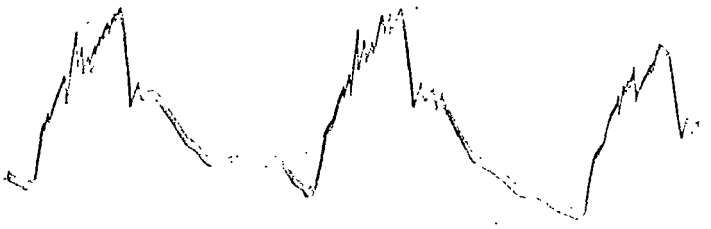

Figure 70. Change in Form of Carotid Artery Sphygmogram with Aortal Stenosis

The reasons for the change in form of the sphygmogram as the pulse wave propagates from the central vessels into the peripheral vessels is considered by Wiggers (1951) to be damping phenomena and the addition of waves in the arterial system. Due to the damping, all high-frequency elements of the central pulse are smoothed, and the addition of centrifugal and centripetal waves causes secondary waves (the dicrotic wave) to be formed. These problems have been studied recently by Remmington and Wood (1956).

The appearance of the curve of the peripheral sphygmogram is comparatively simple (Figure 71). The anacrotic portion of the curve is smoothly replaced by the catacrotic portion. Further, a dicrotic wave is always observed, /240 apparently a result of reflection of the standing pressure wave in the aorta on the sphygmogram.

The form of the peripheral sphygmogram depends on the point where it is recorded. One particular case is the femoral artery sphygmogram (Figure 71, B) which is probably explained by the anatomic proximity of this artery to the aortal compression chamber.

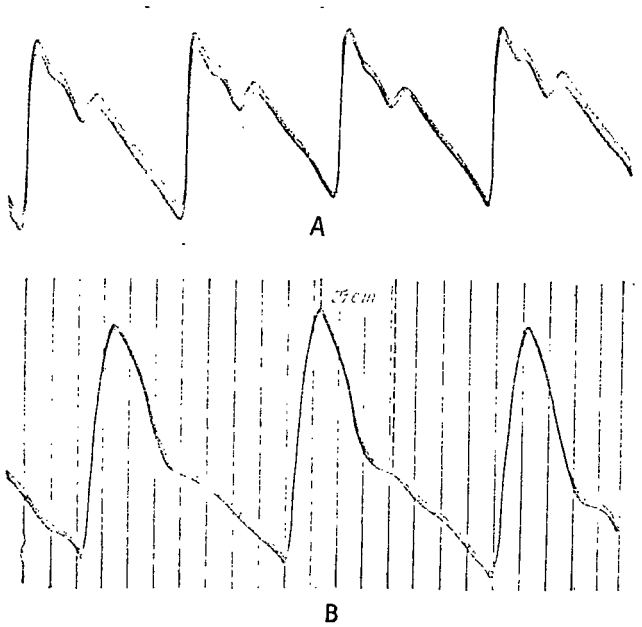

Figure 71. Forms of Peripheral Pulse. A, Brachial artery sphygmogram; B, femoral artery sphygmogram; T_{fem}, principal oscillation in arterial pulse (after Wezler and Boger).

The femoral artery pulse curve frequently shows a low-amplitude dicrotic wave or no dicrotic wave at all. Recording of this curve is most frequently performed at the same time as the recording of the carotid pulse curve for calculation of the rate of propagation of the pulse wave in the aorta.

To record the pulse in the extremities, it is most convenient to use volumetric sphygmography. Usually, the following four levels are selected for the recording of pulse curves: on the legs -- the femur, the upper third of the tibia, the lower third of the tibia, the foot; on the arms -- the shoulder, the upper third of the antebrachium, the lower third of the antebrachium, the hand.

As we can see from Figure 72, the pulse curves recorded at various /241
levels of the lower extremities differ in the height of the anacrote, the pulse amplitude in the upper third of the tibia under normal conditions always being greater than the pulse amplitude at the femur. In cases of

vascular disease in the extremities (for example endarteritis), this relationship is disrupted (M. A. Abrikosova, 1960; L. A. Lakshina and I. S. Klemashev, 1964; etc.).

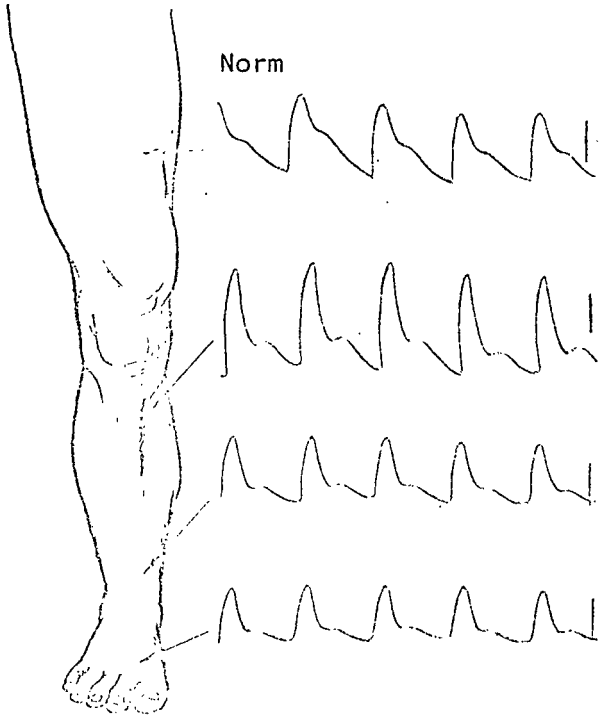

Figure 72. Volumetric Sphygmograms Recorded at Various Levels on the Lower Extremity. Arrows indicate points of recording of sphygmograms. Gain of amplifier identical for all recordings.

M. A. Abrikosova and V. L. Karpman (1959) suggested that quantitative analysis of volumetric sphygmograms of the extremities be performed using the pulse amplitude gradient. The amplitude gradient (Figure 73) is a curve describing the change in amplitude of the pulse at various levels of the extremities. Calculation of the amplitude is performed in percent of the amplitude of the anacrote of the pulse of the femur or shoulder taken as 100%. /242

Investigation of the form of the peripheral pulse may be of important aid in the diagnosis of certain diseases. For example, in patients with open Botallo's duct or aortal valve deficiency, a high-frequency oscillation appears in the anacrote of the femoral artery pulse (Figure 74), the amplitude of which may be relatively high. Similar changes in the curve of the femoral pulse are observed in healthy persons when, under the influence of

various factors, the blood volume per minute increases sharply (see Figure 74). As the cardiac output normalizes in the test subjects, the high-frequency oscillation decreases, then disappears completely. M. A. Abrikosova (1962) established that this change in the form of the pulse wave results from a sharp drop in the elasticity of the vessels which is compensatory, in response to an increase in cardiac output.

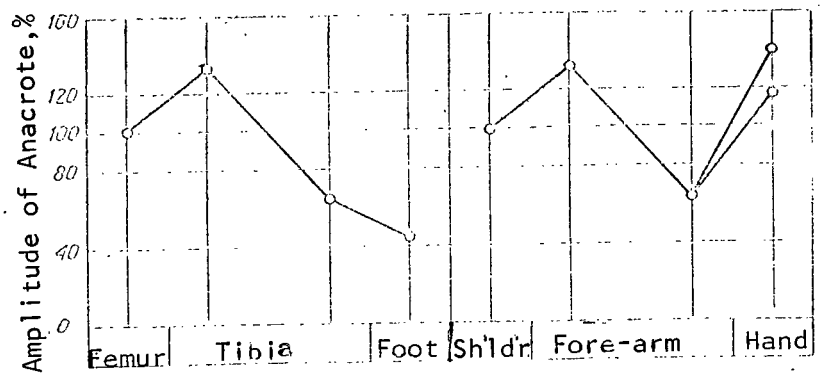

Figure 73. Pulse Amplitude Gradient. Explanation in text.

Under normal conditions, the amplitude of a volumetric sphygmogram recorded on the femur is approximately 1-1/2 times higher than the amplitude recorded at the shoulder.

In certain pathological states, for example constriction of the aortic isthmus, this relationship is sharply disrupted and, as a rule, becomes the reverse, i.e. the amplitude of the pulse recorded at the shoulder is higher than the amplitude at the femur.

In recent times, the recording of the velocity or differential sphygmograms has become widespread. Curves of this type characterize the rate of change in the pulse wave. The second derivative of the pulse wave (acceleration curve) can also be recorded. In order to record velocity or acceleration sphygmograms, simple analog machines are used (Ye. B. Babskiy, V. L. Karpman et al., 1959). D. Dimov and P. Rashkov (1964) developed a special device for this purpose, allowing synchronous recording of the pulse curve, its first and second derivatives (Figure 75). The promise of this new trend in sphygmography is beyond doubt. /243

Some problems concerning the analysis of the form of the central and peripheral pulse which we have not touched upon here are discussed in detail in the reviews of Meiners and Blumberger (1955), V. L. Karpman (1963), the monographs of N. N. Savitskiy (1963), Gadermann and Jungmann (1964). /244

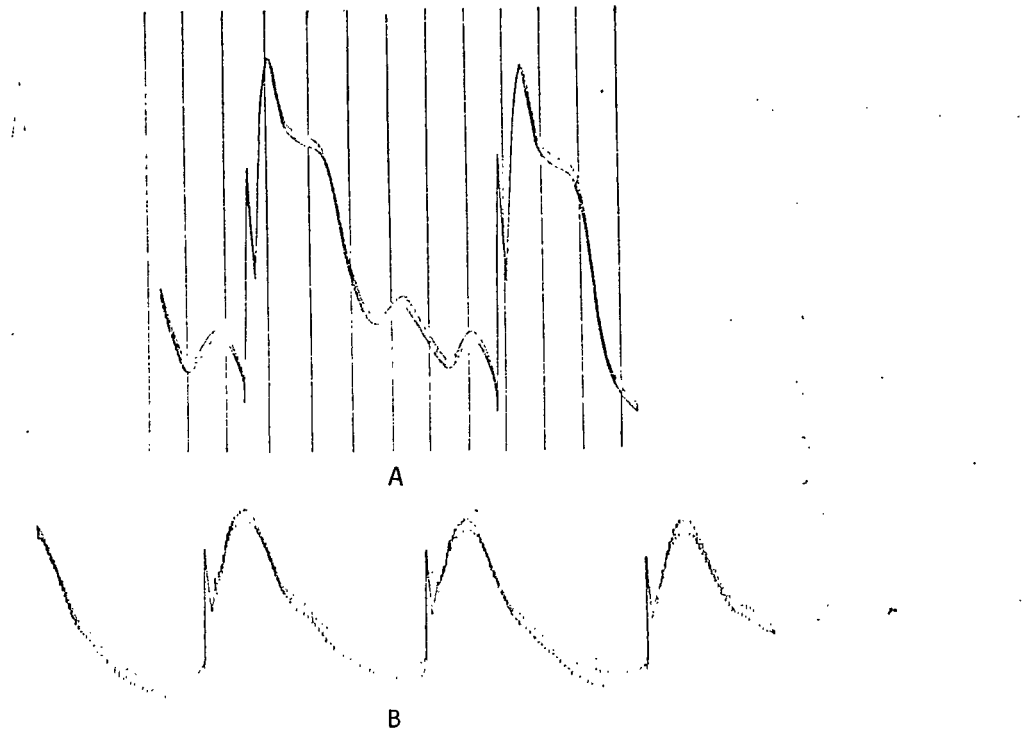

Figure 74. Change in Appearance of Femoral Artery Sphygmogram in Case of Aortal Valve Deficiency (A) and During Sharp, Temporary Increase in Cardiac Output During Alcohol-Antabuse Reaction (B).

Rate of propagation of pulse wave. The possibility of investigating the rate of propagation of the pulse wave through the vessels of both elastic and muscular type is an important advantage of the method of sphygmography. The determination of the rate of propagation of the pulse wave is methodologically quite simple and easy for the test subject. The value of this investigation in physiology and clinical practice is determined by the fact that the rate of propagation of the pulse wave is an indicator of the elastic condition of the arteries.

At the present time, there are two methods for calculating the rate of propagation of a pulse wave in elastic type vessels, more precisely in the aorta. The classical method, based on the Weber principle (Weber, 1850) consists in the following: two pulse receivers are fastened onto the sectors of the carotid and femoral arteries closest to the surface, after which simultaneous recording of the two pulse curves is performed on a single strip of photographic paper (Figure 76). The movement rate in this process should /245 be no less than 50 mm/sec. Calculation of the velocity (V_a) is performed

using the equation:

$$V_a = \frac{L_a}{\Delta t}, \tag{42}$$

where L_a is the length of the vessel, Δt is the time of propagation of the pulse wave through it.

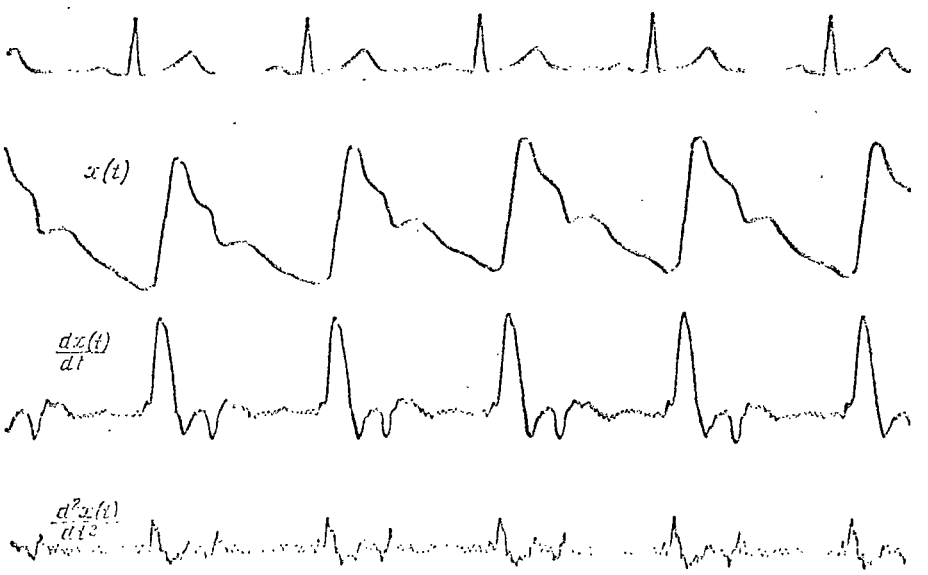

Figure 75. Curve of Central Pulse and Its Derivatives (After Dimov and Rashkov, 1964). Top to bottom: electrocardiogram, carotid artery sphygmogram, curve of rate of change of the sphygmogram and acceleration curve.

In order to calculate the length of the vessel L_a (Figure 77), we must determine the distance from the point of placement of the receiver on the carotid artery to the fossa of the manubrium (l_1), then from the latter point to the branch (l_2) and from the branch to the point of installation of the receiver on the femoral artery (l_3). If we know these distances, the value L_a can be found using the following formula:

$$L_a = (l_2 + l_3 - l_1). \tag{43}$$

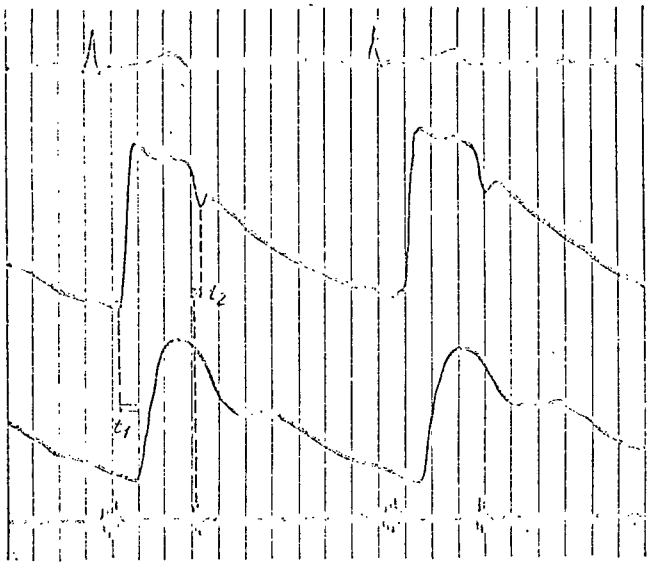

Figure 76. Determination of Propagation Time of Pulse Wave Through Entire Aorta ($t_1 + t_2$). Top to bottom: electrocardiogram, carotid artery sphygmogram, femoral artery sphygmogram and phonocardiogram. Explanation in text.

The value of Δt is determined from the time interval between the beginning of the anacrote of the pulse curve of the carotid and femoral arteries (t_1 on Figure 76). Other, more complex methods of calculating t_1 have been described, which apparently have no better accuracy. This problem is particularly discussed by Meiners (1954). /246

In spite of its general recognition, this method of measuring the central rate of propagation of the pulse wave has one outstanding defect, in that, essentially speaking, V_a is determined only for the descending aorta (see equation (43)). This defect is eliminated if the definition of V_a is performed using a method which consists in synchronous recording of a phonocardiogram and sphygmograms of the carotid and femoral arteries (V. L. Karpman, M. A. Abrikosova, 1962). These data are necessary to calculate the value of Δt.

Using the curves shown on Figure 76, we determine the duration of the intervals t_1 and t_2. The first of these characterizes the delay of the pulse in the femoral artery in relationship to the pulse of the carotid artery.

Interval t_2 characterizes (after Maass, 1949) the delay in the pulse of the carotid artery relationship to the beginning of expulsion of blood from the heart into the aorta. Consequently, the sum of these intervals corresponds to the time of propagation of the pulse wave from the heart to the femoral artery. Then:

$$\Delta t = t_1 + t_2. \qquad (44)$$

The value of L_a required for calculation of V_a is calculated as follows (Figure 77):

$$L_a = l_0 + l_2 + l_3, \tag{45}$$

where l_2, l_3 are the same as in equation (43), l_0 is the distance from the point of projection of the semilunar valves on the thorax (point of attachment of rib III to the sternum) to the fossa on the manubrium.

Thus, this method allows us to estimate the rate of propagation of the pulse wave through the entire aorta.

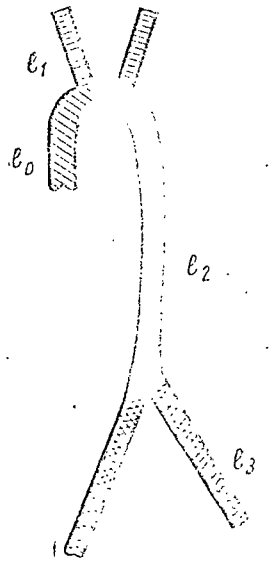

Figure 77. Schematic Drawing of Aorta and Large Arteries Branching from It. Explanation in text.

With a synchronously recorded electrocardiogram, phonocardiogram and pulse curves of carotid and femoral arteries, we can calculate not only the rate of propagation of the pulse wave through the entire aorta, but in its ascending and descending portions. The course of this investigation is illustrated by Figure 76. Here the time of propagation of the pulse wave from the heart to the femoral artery can be found from the sum of $t_1 + t_2$. On the basis of this /247 quantity, we can calculate the rate of propagation of the pulse wave through the entire aorta. The rate of propagation of the pulse wave in the ascending aorta is determined from the formula of V. L. Karpman and M. A. Abrikosova (1962) from the following relationship:

$$V_a = \frac{l_0 + l_1}{t_2}, \tag{46}$$

where t_2 is the time of propagation of the pulse wave from the heart to the carotid artery. The rate of propagation of the pulse wave in the descending portion is calculated by the classical method.

It is most convenient to determine the rate of propagation of the pulse wave in muscular type vessels by recording two volumetric sphygmograms, for which collars on the arms are placed at the shoulder and the lower third of antebrachium, and on the legs -- at the femur and the lower third of the tibia.

When the pelot or other direct sphygmograph is used, the rate of propagation of the pulse wave is usually recorded on the sector between the carotid artery and the radial artery.

The propagation velocities of the pulse wave in the aorta calculated by the classical method were studied most objectively by Hallock (1934), then by Ludwig (1936), Wezler and Boger (1939), N. N. Savitskiy (1963), etc. The juvenile and adolescent contingent was specially investigated by O. Ye. Tsukershteyn (1957) and M. K. Askolkova (1961).

Under physiological conditions, the rate of propagation of the pulse wave in the aorta depends essentially on age: it is higher, the older the test subject. This dependence is apparently related to the changes in elastic properties of the aorta which occur with age. M. A. Abrikosova and V. L. Karpman (1962) suggested a formula allowing us to establish the proper rate of propagation of the pulse wave in the aorta for a given age:

$$V_a = 0{,}1 \cdot B^2 + 1 \cdot B + 380, \qquad (47)$$

where V_a is the proper rate of propagation of the pulse wave in the aorta (in cm/sec), B is the age (in years).

Deflections of not over ±75 cm/sec from the calculated value should be considered normal.

In most healthy persons, the rate of propagation of the pulse wave, determined for the entire aorta using the method which we have suggested, are either lower than the velocity determined by the Weber principle, or the values of pulse wave propagation rate are practically identical. In a special investigation which we performed, it was established that the rate of /248 of propagation of the pulse wave throughout the entire aorta was 5.41 m/sec, in the descending aorta -- 5.79 m/sec.

Under pathological conditions, we frequently note changes in the pulse wave propagation rate in the aorta. An increase in V_a (to 25 m/sec) is noted with hypertension, symptomatic hypertension or aortal atherosclerosis (V. P. Nikitin, 1950, 1959; Ye. A. Morozov, 1953; Ye. A. Moshkin, 1955; L. K. Lakshina, 1957; etc.). A decrease in the propagation velocity of the pulse wave of the aorta can be observed in case of an aortal deficiency, open arterial duct or certain other heart defects (M. A. Abrikosova, 1964). Under pathological conditions, the rate of propagation of the pulse wave through the entire aorta is frequently higher than its rate in the descending portion. This relationship of velocities is encountered more frequently with manifest atherosclerotic changes in the aorta.

The fact of distinction in the values of pulse wave propagation velocity throughout the entire aorta and in a portion of the aorta (the descending

portion) can be explained from the physical point of view by the fact that the pulse wave propagates through the aorta with variable speed, as was shown by Gauer (1936).

In muscular type vessels, the pulse wave always propagates more rapidly than in the aorta. On the basis of the latest works by Noordergraaf and Horeman (1958), this can be explained by the smaller internal radius of these vessels and the relatively thicker vascular wall.

The individual specifics of the propagation velocity of the pulse wave in vessels in the arms and legs under normal conditions depend primarily on the age of the test subject. This dependence, on the basis of data of data of Hallock (1934) concerning the pulse wave propagation velocity in vessels of the upper extremities (553 observations) we have described by the following simple equation:

$$V_p = 8 \cdot B + 425, \tag{48}$$

where V_p is the proper pulse wave propagation velocity in the arteries of the arms (in cm/sec), B is the age of the test subject (in years). Just as in equation (47), equation (48) can be used to determine the proper propagation velocity of the pulse wave in test subjects between 5 and 50 years of age.

Deflections from the expected value not exceeding ±100 cm/sec are considered normal variations of the propagation velocity of the pulse wave in the vessels of the upper extremities.

The rate of propagation of the pulse wave through the vessels of the lower extremities is always higher than in the upper extremities. The normal ratio: /249

$$\frac{\text{pulse wave propagation velocity in vessels of leg } V_H}{\text{pulse wave propagation velocity in vessels of arm } V_p}$$

in healthy, untrained persons averages 1.3. This factor may change essentially both under certain physiological and under pathological conditions, characterizing thereby the specifics of regional blood circulation.

V. P. Nikitin (1950) attached great significance to the ratio of the propagation velocities of the pulse waves in the vessels of the upper extremities and the aorta (V_p/V_a) which under normal conditions is 1.1-1.3. This relationship changes during hypertension and certain other types of pathology.

Elastic properties of the arteries. We know that in an elastic body, an impulse propagates with a finite velocity (C), which is a function of the elasticity modulus of the body (E) and its density (ρ):

$$C = \sqrt{\frac{E}{\rho}}. \tag{49}$$

On this basis, Frank (1920) introduced the concept of the modulus of volumetric elasticity (K), using which we can study the elastic properties of the aortal compression chamber:

$$K = V_a^2 \rho, \tag{50}$$

where V_a is the rate of propagation of the pulse wave in the aorta, ρ is the density of the blood.

According to the data of Wezler and Boger (1939), the normal values of volumetric elasticity modulus fluctuate from $150 \cdot 10^3$ to $1200 \cdot 10^3$ dyne/cm^2.

The value of K changes with age, which must be taken into consideration in comparing values determined with the normal values (see Table 20). It increases in case of aortal atherosclerosis, hypertension (Steinmann, 1942; etc.) and decreases with open arterial duct and certain other heart defects.

A quantitative analysis of elasticity of a vessel taken individually can be performed using the following two equations, suggested by N. N. Savitskiy (1963):

$$E_e = \frac{V^2}{2d}, \tag{51}$$

$$E_m = \frac{V^2}{2d}, \tag{52}$$

where E_e and E_m are the modulus of elasticity for elastic and muscular type vessels respectively. /250

The most important information concerning the elastic properties of arteries can be produced by calculating the coefficient of elasticity of the arterial system (E_0) according to Frank (1920), which is also known as the

elastic resistance:

$$E_0 = \frac{\Delta P}{\Delta V}, \tag{53}$$

This coefficient characterizes the pressure necessary to expand the diastolic volume of the arterial system by 1 cm^3. For example, if an increase in arterial pressure in the systole (ΔP, pulse pressure) is 50 mm Hg, and at the same time the volume of the arterial system ΔV increases by 50 cm^3, E_0 = 1 mm Hg/cm^3, or 1333 dyne/cm^5. The value which we have thus produced lies within the range of normal values of E_0, which vary from 1000 to 3000 dyne/m^5.

According to Wezler and Boger (1939), the highest values of elasticity coefficient (Table 20) are noted in children, the lowest -- in persons 20-30 years of age.

TABLE 20. MEAN VALUES OF MODULUS OF VOLUMETRIC ELASTICITY (K) AND COEFFICIENT OF ELASTICITY (E_0) ACCORDING TO WEZLER AND BOGER (1939)

Age, years	$K \cdot 10^3$, dyne/cm^2	E_0, dyne/cm^5
5	168	4280
10-11	176	2890
16-25	272	1540
26-30	302	1390
31-38	620	2370
46-48	840	2440
64-67	1185	2500

The determination of E_0 in man is performed using a special set of formulas concluded by Broemser and Ranke (1933), then later by Welzer and Boger (1939).

According to Broemser and Ranke (1933), the coefficient of elasticity is determined from the following formula:

$$E_0 = \frac{2 \cdot V_0 \cdot ?}{Q \cdot S}, \tag{54}$$

where V_a is the rate of propagation of the pulse wave in the aorta (in cm/sec), ρ is the density of the blood (= 1.06), Q is the cross section of the aorta [found from the tables of Suter (1897), Thoma (1900) or Frucht (1953)], S is the duration of the expulsion period from the carotid pulse curve. /251

According to Wezler and Boger (1939), the elastic resistance is equal to:

$$E_0 = \frac{4 \cdot V_a \cdot \rho}{Q \cdot T_{fem}}, \quad (55)$$

where T_{fem} is the duration of the principal oscillation of the arterial pulse in sec (see Figure 71, B).

The elastic resistance of the arterial system is frequently studied in relation to the peripheral resistance. The ratio:

$$\frac{\text{elastic resistance}}{\text{peripheral resistance}} = \frac{E_0}{W} \quad (56)$$

is called the damping factor of the arterial system. Normal values of E_0/W vary between 0.7 and 1.3. An increase in E_0/W, in the opinion of N. N. Savitskiy (1963), indicates uneconomical work of the circulatory apparatus.

Sphygmography in sports medicine. In spite of the large volume of useful information concerning the functional state of the arterial system which can be produced by sphygmography, this method has not yet been broadly used in sports medical practice. This is indicated by the comparatively small number of special investigations performed using this method.

The form of the central and peripheral pulse in athletes determined at rest usually has no specific features differentiating it from the pulse curves recorded in healthy nonathletes.

Under the influence of muscular work, certain changes arise in the curves for athletes, primarily resulting from the increase in the pulse frequency. Yu. I. Kuznetsov (1958) turned his attention to the high pre-oscillation wave on the central sphygmogram (we recall that this wave is observed at rest in high-quality recordings of the central pulse). The change in the sphygmic portion of the carotid artery pulse during muscular work consists in the fact that it takes on a triangular, or less frequently

bell-shaped form (see Figure 13, b). These changes in the appearance are related both to shortening of the expulsion period, and to the rapid transition of the catacrotic descent to an incisure.

The rate of propagation of the pulse wave in athletes began to be studied by Reindell (1949), Yu. I. Kuznetsov (1958), Mellerowicz (1956), and then other authors. However, there is still no single opinion concerning the influence of systematic physical exercise on the elastic properties of the vessels. /252

According to the investigations of Yu. I. Kuznetsov (1963), in well trained athletes at rest, the rate of propagation of the pulse wave is slightly decreased in comparison with the normal age standards. Similar decreases in the rate of propagation of the pulse wave in athletes at rest were detected by Ye. F. Likhachevskaya (1959), Ye. A. Poruchikov (1963) and others.

Mellerowicz (1960) discovered that this decrease occurs in athletes older than 30 years of age. In athletes between 20 and 30 years, the rate of propagation of the pulse wave is slightly increased in comparison with its normal values, recorded for healthy nonathletes of the same age.

A relative increase in pulse wave propagation velocity through elastic and muscular type vessels was found by V. V. Vasil'yeva (1965) in an examination of highly trained runners specializing in various distances.

The degree of training also has an influence on the functional state of the arteries. Thus, in an investigation of one group of athletes during the process of training, Yu. I. Kuznetsov (1956), V. V. Vasil'yeva (1957), Ye. F. Likhachevskaya (1958), M. Ya. Levin (1965) and others discovered a gradual increase in the rate of propagation of the pulse wave with increasing state of training. However, R. S. Khachaturov (1965) found a relative decrease in the rate of propagation of the pulse wave through elastic and muscular vessels with increasing sports mastery in gymnasts.

V. N. Artamonov (1966), performing investigations on middle-aged and older persons suffering from cardiovascular diseases, who had not been previously occupied with sports, determined a decrease in the indicators of the elastic-viscous state of the elastic type arteries after two years of systematic physical exercise.

The direct influence of muscular work on the state of vascular tonus has almost not been studied at all. Most authors judge these changes from data produced over various periods of time after work performed, i.e. in the recovery period. Yu. I. Kuznetsov (1963), found an increase in the propagation velocity of the pulse wave through elastic and muscular type vessels after the completion of intensive work (high-speed run) and a moderate decrease in the propagation rate of the pulse wave through the same vessels after longer, but less intensive work (20-km run).

A sharp increase in the rate of propagation of the pulse wave through elastic and muscular type vessels in connection with work was shown by Ye. F. Likhachevskaya (1958), Ye. A. Poruchikov (1963), V. V. Vasil'yeva (1964, 1965), R. S. Khachaturov (1965), V. N. Artamonov (1966), V. Ye. Vasil'yeva and T. D. Vasil'yeva (1967), Gobbato, Turchetto et al. (1964), etc. /253

Quite recently, a series of reports have appeared in the literature dedicated to investigations of the regional vascular tonus (in the arms and legs) of athletes at rest and its changes during the performance of various types of work.

N. A. Stepochkina (1965), in an investigation of marathon runners under conditions of basal metabolism, discovered a relative increase in the pulse wave propagation rate through the vessels of the upper extremities in comparison with the tonic stress of the vessels of the lower extremities. As a result of this, the ratio C_H/C_p (which is normally about 1.3 for nonathletes) became less than 1.0. In the opinion of the author, a decrease in the elastic resistance of the vascular wall facilitates an increase in the flow of blood through the vessels in the lower extremities, which is explained by the specifics of the sports loading of marathon runners.

The clearest changes in regional vascular tonus were discovered in connection with the work of various muscular groups. In individual determinations of the rate of propagation of the pulse wave through the muscular vessels of the upper and lower extremities, V. V. Vasil'yeva and colleagues (1965, 1966) determined the following important fact: the rigidity of the arteries in a nonworking extremity is higher than the rigidity of the arteries in extremities performing work. For example, after a long run, the propagation rate of the pulse wave through the vessels of the arms is relatively more highly increased than the rate of the pulse wave in the vessels of the legs. These regional changes in vascular tonus are observed particularly clearly in swimmers working either with arms alone or with legs alone.

The regional changes in vascular tonus related to muscular work facilitate useful redistribution of blood, providing reinforced blood circulation to the working extremities. It should be noted that the adequacy of regional changes in elasticity of vessels during muscular work, in the opinion of V. V. Vasil'yeva (1965) and R. S. Khachaturov (1966), depends on the degree of adaptation of the athlete to the work which he performs and may be used as an indicator of special training.

The elastic resistance of the arterial system of athletes differs little from the normal values (Reindell, 1949; Mellerowicz, 1956). In addition to this, in an investigation of well trained athletes, Mellerowicz (1956) established a predominance of decreasing values of the damping factor E_0/W. According to his data, the value of this factor varies from 0.4 to 0.6. The author turned his attention to the relationship of the state of training to /254

the degree of decrease in the value of the damping factor. A similar conclusion was reached by N. A. Stepochkina (1965), who found a damping factor of 0.44 in marathon runners at rest.

In recent times, B. M. Stovbun and V. M. Forshtadt (1963) developed a radiotelemetric method for determining the propagation time of the pulse wave in athletes directly during muscular work. This type of investigation consists in measurement of the distance from the beginning of the QRS complex of the electrocardiogram to the appearance of the pulse curve at the terminal phalange of the finger. These authors established that the propagation time of the pulse wave over the sector which they studied during muscular work is decreased by an average of 0.05 sec. Since the propagation time of the pulse wave was studied using the same sector of the vascular system, in the opinion of the authors, it can be concluded that the rate of propagation of the pulse wave increases during physical work.

Radiotelemetric investigations are particularly promising in sports medicine; therefore, the investigations of B. M. Stovbun and V. M. Forshtadt (1963) are of definite interest. Unfortunately, the methodological approach which they used is not without reproach. The problem is that the time interval from the Q wave of the electrocardiogram to the beginning of the anacrote is determined not only by the time of propagation of the pulse wave, but also by the duration of the stress period of the left ventricle. This latter quantity is always decreased by 0.03-0.06 sec under the influence of intensive muscular work. Therefore, we cannot decide what causes the shortening of the interval which they studied during muscular work -- a decrease in the propagation time of the pulse wave or a decrease in the stress period. Similar considerations must be kept in mind in an analysis of the results of the investigations of I. L. Manuylova and I. A. Manuylova (1965), as well as those of D. B. Shmul'yan (1966), who used this same incorrect methodological approach in determining changes in the rate of propagation of the pulse wave during physical stress.

CHAPTER VII

X-RAY METHODS OF INVESTIGATION OF THE CARDIAC ACTIVITY OF ATHLETES

ABSTRACT. Various functional X-ray methods are described, allowing quantitative descriptions of the heart and its operation. Tried and try methods such as roentgenkymography are described, in addition to a new X-ray functional method--electrokymography. Forms of roentgenkymogram rays are diagrammed and significance analyzed. Causes and manifestations of hypertrophy, principally of the left ventrical, are presented. The graphic phase analysis of electrokymograms is described in detail. Case histories are presented. Teleroentgenography, a method of studying the volume of the heart, is described.

In this chapter, we discuss only the functional X-ray methods allowing /255
quantitative descriptions of the heart and its work. In addition to the old and tried methods, such as roentgenkymography, we present data on a new X-ray functional method -- electrokymography.

As has been shown recently, biplanar teleroentgenography is of great significance for sports medicine. We present the modern methodological principles of this type of X-ray investigation.

Roentgenkymography of the Heart

Among the methods used in medicine for evaluation of the state of the cardiovascular system, an important place is held by roentgenkymography. Using this method, it is possible to determine the form, position and dimensions of the heart and large vessels, to delineate the heart chambers and the sections of the aorta, to study the heart in systole and diastole separately, and to evaluate indirectly its contractive capability.

Roentgenkymography fixes the movement of the heart and large vessels and allows an objective determination to be made concerning their changes in relation to the dynamics of training and the influence of physical loads. The roentgenkymogram is a summary curve, determined by a number of factors, the principal of which are: the pulsating movement of the heart, resulting from systolic contraction and diastolic relaxation of the muscular walls; volumetric fluctuations in the heart, arising in relationship to the various /256

types of hemodynamic influences and changes of the intraventricular pressure; the rotational movement of the heart; the movement of the systolic output, etc. The morphological specifics of the waves of the roentgenkymogram allow a functional differentiation of the various sections of the heart.

Certain problems of methodology. The method of roentgenkymography was first used by Sabat in 1911, Goett and Rosental in 1912 in the form of single-slit roentgenkymography, allowing the recording of the contractions of individual sections of the heart. The method became broadly used only after 1928, when Stumpf suggested the multislit roentgenkymograph, allowing simultaneous images of the movement of all points on the outline of the heart and large vessels to be produced simultaneously.

In sports medicine, the method of roentgenkymography has been broadly used to evaluate the effects of physical exercises on the heart by S. V. Shestakov (1933), S. P. Letunov (1949, 1957), R. Ye. Motylyanskaya (1949), A. A. Gorodetskiy (1947), N. V. Potte (1951), N. D. Grayevskaya (1960, 1961), N. V. Savina (1958), Zdansky (1947), Reindell (1940, 1960), etc.

The method of roentgenkymography is based on the fact that X rays pass through the horizontally located narrow slits in a lead lattice placed between the test subject and a screen carrying photographic film. During an exposure, the lattice is moved evenly so that each of the slits (width 1 mm) passes through a distance equal to the width of the lead plates separating one slit from the next. Each slit in the lattice ends its movement at the point over the heart where the movement is begun by the next slit. As a result, an image of the entire outline of the heart and large vessels is produced on the roentgenkymogram.

At the present time, the roentgenkymogram produced by the Institute of Roentgenology and Radiology and the "Rentok" plant is used in the USSR. It consists of a metallic frame containing a lattice, moved by a system of springs. The lattice moves while the film-containing cassette remains stationary. The lattice has 22 strips, each 12 mm in width. The roentgenkymogram is taken in anterior projection, or when a number of problems need to be clarified (for example, the question of hypertrophy of some portion of the heart, changes in vessels, etc.) it may also be taken in the first slanted position (test subject rotated with right shoulder and chest toward cassette at 45° from front projection) or the second slanting position (left shoulder forward at 50-60° angle rotation from front projection).

The recommended operating mode for the apparatus for exposure in the front projection is: current 40-50 ma, voltage 80-90 kv, exposure 2-3 sec, focal distance 70-80 cm.

When the roentgenkymogram is taken in the slanted or lateral positions, /257 the voltage should be increased to 90-110 kv, the focal distance to 80-90 cm.

Since the configuration and dimensions of the heart depend to a considerable extent on the level of the diaphragm, during dynamic observations it is quite expedient to perform exposures with various levels of inhalation, measured at 300-400 cm^3 (depending on the form of the thorax and the lung capacity).

The roentgenkymogram in the state of muscular rest. The normal roentgenkymogram most frequently shows a slanted position of the heart. In young persons and persons with a narrow thorax, the position of the heart is primarily vertical. With a broad rib cage and elevated diaphragm, the position of the heart may approach the transverse.

The degree of roundness of the heart shadow in its individual segments results primarily from the degree of hypertrophy and the tonogenic expansion of the various heart chambers.

In the anterior projection, the outline of the heart is illustrated by 15-18 bands of the roentgenkymogram (Figure 78). The nature of the waves allows us to distinguish the various sections of the heart. The left contour of a normal roentgenkymogram shows the left ventricle, the left atrium, the pulmonary artery and the aorta. The left ventricle usually covers 6-9 bands, the atrium -- not over 1-2 bands, the pulmonary artery -- 2-3 bands, the aorta -- 2-1/2-3-1/2 bands. The section forming the right edge is the right atrium ("ventricular" type waves can sometimes be determined in the lower section). Beginning with the atrial-vascular angle (approximately from the ninth to the thirteenth or fourteenth band) we find the zone of the vessels -- the vena cava superior and the ascending aorta, the waves of which show almost no outline in most cases. /258

In the first slanted position, the anterior contour shows three arcs: the ascending aorta, the pulmonary cone and the left ventricle, while the posterior contour shows the ascending aorta, the left and right atria. In the second slanted position, the anterior contour shows the ascending aorta, the right atrium and the right ventricle, while the posterior contour shows the left atrium, with the pulmonary artery intersecting it in its upper sector, and the left ventricle.

With the hypertrophy of the left ventricle frequently encountered in athletes, its zone is increased to 8-11 bands, and the left atrial zone decreased to 1/2-1 band, or may disappear entirely in cases of considerable hypertrophy.

In case of hypertrophy and tonogenic expansion of the right ventricle in connection with increased function, the number of bands occupied by the "ventricular type" waves on the right contour is increased to 3-4 or more, and in many cases these waves may encompass the entire right contour.

The left ventricular waves are hook-shaped with a longer and slightly convex upper diastolic bend, showing the lateral movement of the contour of the heart during the diastole, a short, straight lower systolic curve and a

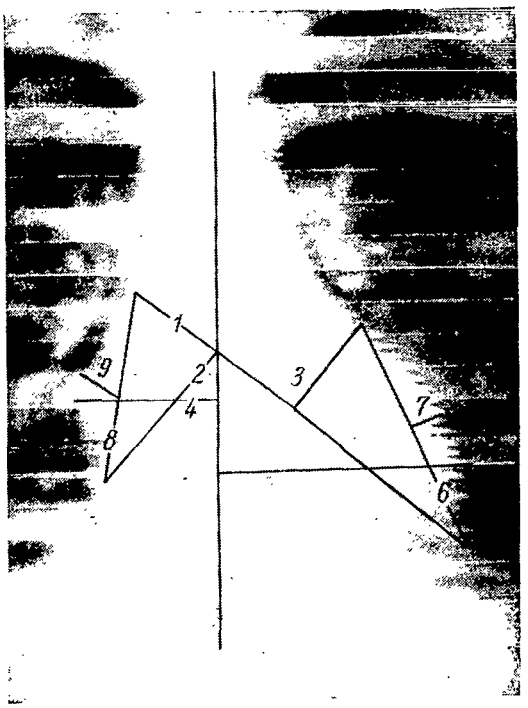

Figure 78. Roentgenkymogram of Athlete S. 1, Longitudinal dimension of heart (line connecting first clearly visible wave over diaphragm to top wave of right contour or right atriobasal angle); 2, 3, Sum of lines (perpendiculars drop to line 1 from left upper and right lower outlines of heart) making up slanted dimension of heart; 4, 5, Sum of lines showing distance between median line and furthest points on waves of right and left outlines of heart, making up transverse size of heart; 6, Height of left ventricle; 7, Thickness of left ventricle; 8, Height of right ventricle; 9, Thickness of right ventricle

sharp or slightly rounded tip. The longer the diastole, the more convex the upper shank appears. Variants of the norm for ventricular waves are [according to V. V. Zodiyev and N. P. Razumov (1953)]: arc-shaped, trapezoidal, double arc-shaped, needle-shaped, hook-shaped with straight shank and with sharp tip (Figure 79). In case of cardiac muscle diseases, deformations may appear in the form of elongations and shortenings of the systolic shank, stepped shapes, waviness, teeth, convexity, splitting of the diastolic shank, etc. These deformations are almost never encountered in athletes.

The form of the waves and the degree to which the tips are sharpened depend on the pulse frequency, respiration phase, cardiac muscle tonus and a number of other factors.

In untrained athletes with manifest bradycardia, we most frequently see hook-shaped waves with clear elongations of the diastolic shank. After physical loading, the waves become sharper.

The vascular waves are mirror /259
reflections of the ventricular waves: an upper short, straight shank corresponds to the ventricular systole, and is followed by an inclined, convex curve reflecting the slow emptying of the vessel during the ventricular diastole. According to the same diagram, the variants of the normal vascular wave (aorta) are waves with extended and elongated peaks. When the elastic wall of the aorta is damaged or has undergone sclerotic changes, deformations appear in the form of the convexity /260
of the systolic shank, or as rounded or sectioned peaks, etc.

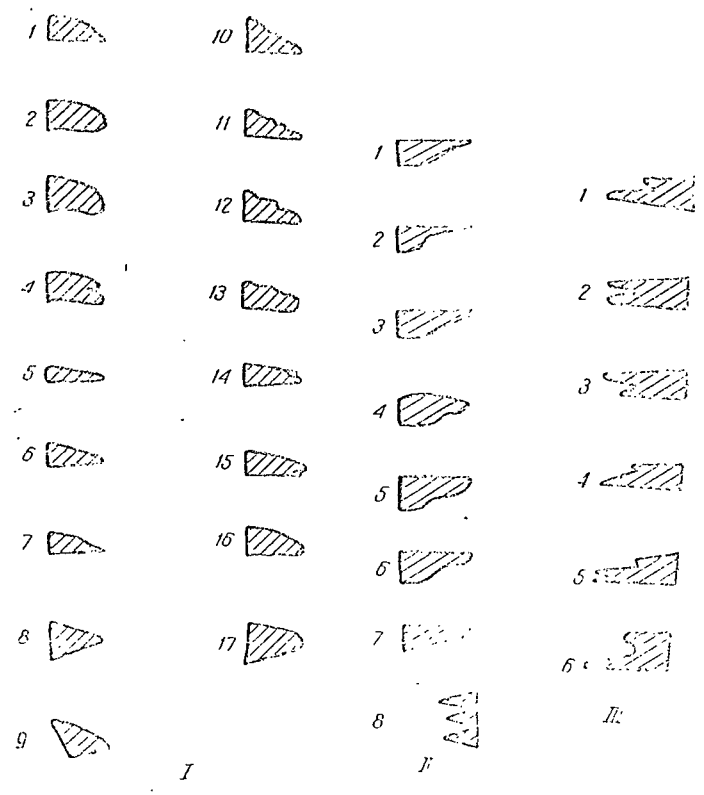

Figure 79. Forms of Roentgenkymogram Waves (According to N. P. Razumov and V. V. Zodiyev). I, left ventricular waves; 1, normal wave form of ordinary hook, 2-7 variants of the norm (2, arc-shaped; 3, trapezoidal; 4, double arc; 5, needle-shaped; 6, hook with straight shank; 7, hook with sharp tip); 8-17, various deformations of waves. II, aortal waves: 1, ordinary spicular; 2, 3, variants of the norm (2, spicular with extended and elongated tip; 3, with split tip; 4-8, deformed waves in case of sclerotic or other aortal damage. III, atrial waves: 1, normal; 2-6, various changes

The atrial waves are polymorphous, but most frequently double, the upper portion of which shows the atrial systole, the lower portion -- the ventricular systole. As the ventricular systole is reinforced, the ventricular component is reinforced and may exclude the atrial component -- ventricular type waves are recorded. Most frequently, the lower branch of the wave is almost twice as long as the upper branch. With disruptions in myocardial function, the tips may be equal, or the upper may be slightly longer than the lower.

The difference between the contour of the heart during the systole and diastole is determined by the amplitude of the waves, one of the most important objective indicators of the contractive activity of the cardiac muscle. The X-ray kymographic curve of pulsator movements of the heart results from systolic contraction and diastolic weakening of the cardiac muscle, rotational movements of the heart and its position.

The amplitude of the waves can best be measured by the length of the systolic shank.

According to the data from the literature, the amplitude of waves of the left ventricle is normally 4-7 mm in the anterior projection and 8-12 mm in the second slanted position. Yu. I. Arkusskiy (1948), N. P. Razumov, V. V. Zodiyev (1953) and others believe that isolated hypertrophy of the cardiac muscle produces small waves (3-4 mm), while high-amplitude waves of varying size are observed in cases of tonogenic expansion of the heart.

On the heart roentgenkymograms of more than 500 qualified athletes which we analyzed, the amplitudes of the left ventricular waves in the overwhelming majority of cases exceeded the norms, established for healthy persons, and were within limits of 5-11 mm in the anterior and 7-15 mm in the second slanted projection. Many years dynamic observations do not allow us to agree with the point of view that high waves are an indicator of decreased tonus of the myocardium.

The worsening of the functional state, the phenomenon of overstress and overtraining are accompanied by a decrease in amplitude, particularly in the caudal section of the left ventricle, right up to appearance of the so-called deaf zones. This corresponds to clinical observations showing that as a result of anatomic specifics, worsening of supply of the myocardium and a decrease in its contractive capacity appear here first.

Evaluating the "deaf zones," we should keep in mind that most frequently in the second slanted position (and in the anterior position after physical loading) clearly expressed pulsation is seen in these sectors (Figure 80). /261
This acts as one of the differential diagnostic indicators for the determination of distinctions between the "sports heart" and the decrease in the contractive capacity of the myocardium resulting from heart muscle disease.

The amplitude of pulsations of the right contour in healthy persons in the anterior projection is 3-3.5 mm, in the second slanted position -- 3-5 mm. In trained athletes, these figures generally lie between 3-6 and 5-10 mm. The ratio of the pulsation amplitude of the left and right ventricles (L/R factor) is generally between 1.5 and 1.6.

The amplitude of the waves in the left atrium did not exceed 2 mm, and was most frequently 0.5-1.5 mm. The amplitude of the pulmonary artery and aorta in trained athletes most frequently varied between 2.5-3.5 and 2.5-4 mm, i.e. was also greater than the quantities presented in the literature for healthy persons (1.5-2.5 mm).

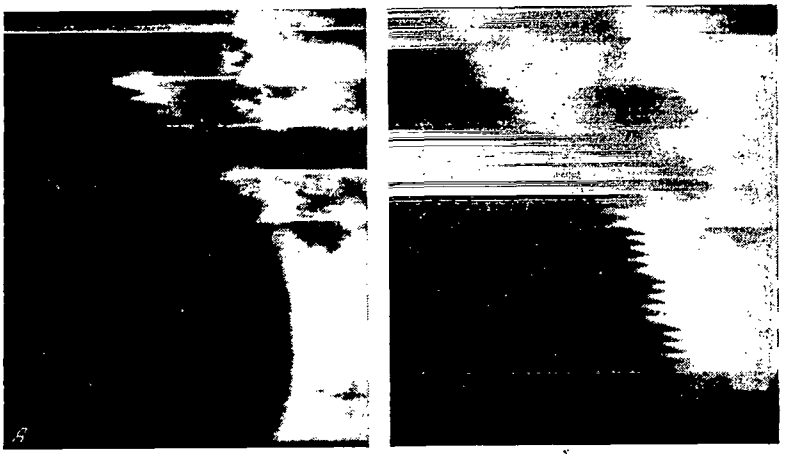

Figure 80. Disappearance of "Deaf" Zone After Physical Loading (Roentgenkymogram of Boxer S in Anterior Projection). A, before loading; B, after loading.

The right side of the vascular contour most frequently shows no waves on the roentgenogram, although small waves are sometimes seen, which depends on the position of the upper vena cava and the type of vascular bundle. In the anterior projection the waves of the aorta are clearly seen only on the left contour. On the right they are seen only if the aorta is bent and for one reason or another is included in the contour. The appearance of a clear vascular pulsation on the right in many cases is seen in athletes of advanced age (without being visible on the roentgenkymograms of the same athletes taken at younger ages). Since the picture of the ascending aorta in the anterior position is blurred, if it is necessary that it be investigated, the roentgenkymogram should be taken in the left slanted position. /262

Three types of pulsation are distinguished: the first -- with maximum amplitude at the apex of the heart, the second -- at the base, the third -- with approximately identical amplitude at the apex and base of the heart. According to the data of V. V. Zodiyev (1957), in forming the type of pulsation, a great role is played by the systolic rotation of the heart. The predominance in healthy persons of the first type of pulsation results to a considerable extent from the fact that the apex of the heart participates more fully in the rotary movement, so that greater pulsation is seen here. Proof of this, in particular, is the fact that upon transition from the vertical to the horizontal position, the first type frequently is replaced by the second.

For a long time it was thought that the second type of pulsation was more characteristic for a functionally less capable heart (Stumpf, 1936; E. M. Gel'shteyn, 1937; V. S. Ginzburg, M. M. Vladysik and B. M. Sosina,

1939; M. A. Ivanitskaya, 1936; etc.). However, later in investigation of physically strong and healthy persons, including athletes, it was discovered that in a significant number of cases the second type of pulsation is encountered with no diseases or functional disruptions (S. V. Shestakov, 1934; S. P. Letunov, 1947; N. D. Grayevskaya, 1961; etc.), and after physical loading the second type generally goes over to the first type in these cases. However, during pathological processes, the second type of pulsation is still frequently seen.

According to our data on completely healthy, highly trained athletes, the second type of pulsation was detected in 20% of the cases, the first type in 70%, the third type in 10%. In comparison with the data of V. V. Zodiyev (1957), which he produced using healthy persons, the first type is encountered more frequently in athletes (according to V. V. Zodiyev, the first type is encountered in 49%, the second type in 39%, the third type in 12% of cases). Dynamic observations of athletes showed, however, that the transition of the first type of pulsation to the second type is most frequently related to a decrease in the functional state of the cardiovascular system according to the data of complex examination or manifest development of age changes.

The most reliable methods for determining the size of the heart are, as we know, ortho- and teleroentgenography. However, in sports medicine, the roentgenkymographic method is rarely used for this. /263

In order to determine the dimensions of the heart on the roentgenkymogram, the corresponding points must be drawn, determining the lengthwise, inclined and transverse dimensions of the heart (see Figure 78). According to the method of Vakez and Borde, the height of the left and right ventricles are also determined (V_S and V_D). For this, straight lines are used to connect the atriovasal and cardiodiaphragmatic angles. The convexity of the arc of the ventricles is measured perpendicular to the height -- to the farthest point on the arc (H_{V_S} and H_{V_D}). V_S reflects to some extent the length of the left ventricle and the state of the paths of efflux, H_{V_S} reflects the expansion of the posterior wall of the left ventricle in the dorsal direction -- the condition of the paths of influx. V_D and H_{V_D} characterize the paths of influx of the right ventricle. Normally, V_S is greater than V_D. Although all these measurements are far from the true anatomical quantities, they still can be used as an approximation for dynamic observations.

The area of the heart is determined on the basis of the longitudinal and inclined dimensions using the proper formulas, nomograms and planimetric methods. The best known is the formula of Hammer, according to which the

area of the heart multiplied by 100 and divided by a derivative from the height, chest circumference and constants (equal to 226 for the planimetric method) varies normally between 95 and 105 cm^2. Values less than 90 or greater than 110 cm^2 are considered to be on the edge of pathology.

After the proper projection correction, the roentgenometric indicators produced are compared with the physical development of the test subject, comparing the actual dimensions of the heart with the proper dimensions for a given age and weight, for which purpose a number of tables and nomograms have been suggested. V. V. Zodiyev (1957) believes that the tables of Yu. I. Arkusskiy (1947) are most accurate, in which the dimensions of the heart are given in relationship to height of the test subject, considering sex and age. In sports medicine, the method of Ungerleider and Gubner (1943) has become widely used, allowing special nomograms to be used to find the actual area of the heart from the values of its longitudinal and inclined diameters, and also indicating the proper values of heart area and transverse diameter for a given height and weight.

In order to determine the volume of the heart from the formulas which have been developed (Rorer, Assman, etc.) additional photographs must be taken in lateral projections. The Bardin formula allows the heart volume to be calculated from photographs made in the anterior projection alone. According to this formula, the volume of the heart is equal to 3/2 its area, multiplied by 0.53. A. V. Grinberg and Yu. I. Vaynshteyn introduced a correction to this formula for the projection distortion. The formula with this correction appears as follows: the volume of the heart is equal to 0.5(0.85 × 3/2 area). According to the data of Yu. I. Vaynshteyn and V. V. Zodiyev, the deviation from the true dimensions using this formula will not exceed 5-7%. /264

There are also a number of methods for determining the size of the heart and aorta in the slanted positions, the best of these being that of Vakez and Borde.

The diameter of the aorta is difficult to determine in the anterior position. In order to determine the diameter of the vascular bundle from the point of crossing of the arc of the right atrium to the aorta, a line is drawn to the extreme point of the arc (height of the aorta), and perpendiculars are dropped to this line from the furthest points of the right and left vascular contours. The total length of the right and left perpendiculars is the diameter of the vascular bundle. It is considered that the diameter of the aorta is one half of the diameter of the vascular bundle. According to the norms of Yu. I. Arkusskiy, the diameter of the ascending aorta in the standing position is: for age 16-20 years, 1.5-2 cm; 20-30 years, 2 cm; 30-40 years, 2-2.5 cm; 40-50 years, 2.5-2.8 cm; 50-60 years, 2.5-3 cm, and older than 60 years, 3 cm.

In a significant number of the athletes, the dimensions of the heart, in comparison to the expected dimensions for a given height and weight, are

greater, which is a result of the higher demands placed on the heart by physical stress.

Most frequently the increase in heart diameters is observed in representatives of types of sports which require training primarily for endurance (skiers, marathon runners, rowers, bicyclists, etc.).

The sports ranking has a certain significance as well; thus, relatively larger heart dimensions were determined usually for athletes with the longest period of training. However, frequently young athletes with low sports ranking show rapid increases in heart dimensions, most frequently a result of forced training, and of little diagnostic significance for determination of signs of overloading.

The greatest heart dimensions were determined during investigations in the horizontal position, since the orthostatic decrease in heart size in the standing position is 15-290 cm^3 (averaging 150 cm^3). The increase in area and cross sectional diameter of the heart determined in the standing position generally does not exceed 10-15% in comparison to the expected values for a given height and weight, but in many cases (particularly in athletes trained for long periods of time with high work volumes) may reach 20-30% or more. According to the data of Reindell (1949), the heart volume of qualified athletes in the prone position is 700-1200 cm^3, reaching in some cases 1250-1400 cm^3, but generally not exceeding 750-785 cm^3 for healthy nonathletes. /265

The increase in the heart size in athletes occurs due to physiological hypertrophy of the cardiac muscle and tonogenic expansion of the cavities. The hypertrophy of the cardiac muscle is a natural result of the hyperfunction of the heart during physical stress.

As the heart in athletes becomes larger, normal proportions are retained between the muscular mass of the heart and the dimensions of the chambers. Reindell and Delius (1948) present data indicating that the size of the cavities shows a greater absolute increase than the muscular mass. The increase in the size of the sports heart is defined by Reindell as "regulative dilation," i.e. an increase occurring due to a significant increase in the residual blood volume (its ratio to the systolic output at muscular rest sometimes reaches 3:1).

The diastole of the hypertrophic sports heart is lengthened. The relative shortening of the systole occurs primarily due to shortening of the expulsion phase, whereas the stress phase is slightly longer. Roentgenkymographic data show reinforcement of the contractive capacity of such a heart: after loading, the healthy heart shrinks noticeably, this shrinkage being observed in the Valsalva experiment as well. The high working ability of the hypertrophic sports heart is indicated by electrocardiographic, ballistocardiographic, electrokymographic data, as well as by the normal values of intrasystolic pressure, which Reindell observed in athletes using direct catheterization of the heart.

Physiological hypertrophy of the cardiac muscle as a reaction to physical stress is developed gradually in the process of systematic training and usually (in contrast to pathological hypertrophy) does not become very great. According to the plan of B. M. Kudish (1932), hypertrophy and tonogenic expansion begin with the paths of efflux, after which various forms of combinations of changes in the paths of efflux and influx are observed, which results in varying degrees of increases in the dimensions of the heart. The weight of the hypertrophic sports heart may reach 350-400 g (in healthy nonathletes, -- 300 g), but even the largest athletes' hearts do not reach 500 g -- the so-called critical weight (Linzbach, 1948), after which further development of coronary anastomoses and capillaries does not occur, so that dystrophic and functional changes begin. Thus, Reindell has presented data indicating that the largest athlete's heart, with a volume of 1400 cm^3, weighed only 400 g. Histological and biochemical investigations have shown a normal picture of hypertrophic athletes' hearts (S. S. Vayl', 1940; P. Z. Gudz', 1965; Reindell, Weyland, 1954). Reindell, Klepzig and Steim /267
(1965) showed that with increasing heart dimensions, the volume per beat increases. The absorption of oxygen per heart beat therefore also increases. A direct indicator of high working ability of the heart is considered by these authors to be the proper relationship between the heart volume, absorption of oxygen and general working ability. The increased working ability of large hearts is explained by these authors in that these hearts are capable of increasing the volume per beat during physical stress to a greater extent, by the expulsion of large quantities of residual blood, which in turn makes possible a greater influx of blood to the heart in the diastole.

The investigations of S. P. Letunov, R. Ye. Motylyanskaya (1948), N. D. Grayevskaya (1952), V. N. Kuz'mina (1955) and others, in contrast to this, show that cardiac hyperfunction due to great increases in size is not the best form of adaptation of the athlete's organism to physical stress. Great enlargement of the heart, particularly its right portion, generally is a symptom of noncorrespondence between the physical load and the working ability of the athlete and may lead to a decrease in reserve strength of the heart and the development of dystrophic changes in the myocardium, i.e. is essentially a step on the way to development of myogenic dilation.

According to the data of Reindell, enlargement of the athlete's heart occurs evenly for all sections.

S. P. Letunov and R. Ye. Motylyanskaya (1948), on the basis of roentgenkymographic investigations, have distinguished three variants (types) of "sports heart" depending on the primary enlargement of one or another section of the heart: 1) without manifest segmentary changes, 2) with enlargement primarily of the left ventricle, 3) with expressed enlargement of both sections of the heart. The degree and nature of the enlargement result to a considerable extent from the type of sports and primarily the volume and intensity of physical loading and the degree of its correspondence to the functional capabilities of the organism of the persons in question.

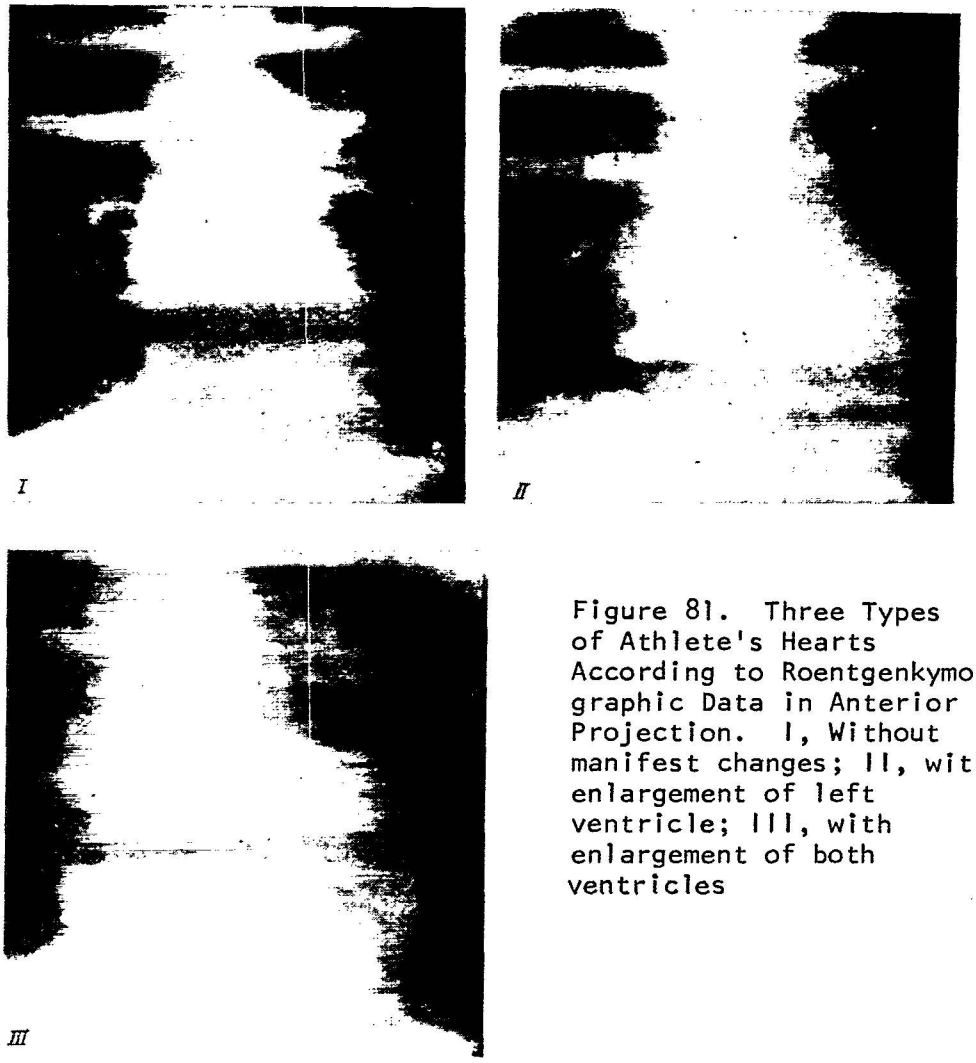

Figure 81. Three Types of Athlete's Hearts According to Roentgenkymographic Data in Anterior Projection. I, Without manifest changes; II, with enlargement of left ventricle; III, with enlargement of both ventricles

For each of these variants, there are certain specifics of the roentgenkymographic picture of the heart (Figure 81).

When the enlargement is primarily in the left ventricle, we see elongation of the heart, enlargement of the arc of the left ventricle and rounding of the apex, a high position of the left atriovascular angle. The waist of the heart is clearly expressed. The zone of left ventricular waves is increased to 9-11 bands with a decrease in the left atrial zone, right up to its complete disappearance. The ratio of the left transverse dimension to the right increases to 1.8-2.4, the chord of the left ventricle is lengthened, its thickness indicator is increased, and pulsation is reinforced. In the second slanted position, a significant swelling of the left ventricle toward the rear is noted as its contour approaches the spine, and with expressed hypertrophy of the heart it enters in this position the shadow of shadow of the spine, completely filling the retrocardial space. /268

For the third type of sports heart, it is characteristic further that the right contour is rounded and stands out, the right atriobasal angle is high, the waist of the heart is smoothed due to an increase in the size of the cone and arc of the pulmonary artery, the chord of the right ventricle is enlarged and its thickness indicator is increased. With manifest hypertrophy, the right ventricle may form the edge, and in these cases ventricular type waves are seen to the right in the lower section, and sometimes throughout the entire contour. The amplitude of pulsation is increased. In the first slanted position, the contour of the pulmonary artery is seen to stand out, and its pulsation is increased.

Morphological changes of the athlete's heart occur to a greater extent due to the paths of efflux, which manifests itself in the left ventricle primarily in its elongation, and in the right ventricle as an enlargement of the pulmonary cone. If changes in the paths of influx of both ventricles also occur, clear enlargement of the transverse diameter and general dimensions of the heart is noted.

The experimental investigations of S. P. Letunov, R. Ye. Motylyanskaya (1949), N. D. Grayevskaya (1952), V. N. Kuz'mina (1954), Ya. G. Ablov (1961) and others have established certain differences in the functional state between the three types of heart. The first and second types in most cases are characteristic for athletes with good functional state of the cardiovascular system (even with a considerable enlargement of the dimensions of the heart, determined frequently with the second type of sports heart).

The third type (manifest enlargement of both ventricles of the heart) is more frequently accompanied by certain indications of decreased functional capacity and may be encountered in representatives of various types of sports when the loading is not matched to the functional capacities of the organism, with overfatigue, or overtraining. Cases when a considerable enlargement of heart (particularly its transverse diameter, which results to a greater extent from changes in the right ventricle) occurs over a relatively short time, which most frequently results from forced training in young and

unprepared athletes, can be of great diagnostic significance. As the functional state improves, the reverse changes frequently occur (Figure 82). This indicates that enlargement of the heart in these athletes occured primarily due to elements of expansion.

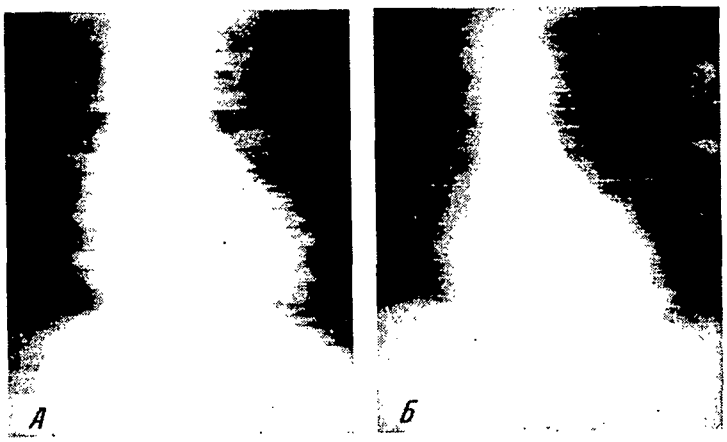

Figure 82. Roentgenkymogram of Heart of Boxer S in State of Overstress (A) and Improved State (B). We can see the decrease in the dimensions of the heart (particularly to the right), manifestation of pulsation in the left supraapical area, while on theright ventricular waves have been replaced by artrial waves.

It should be particularly emphasized that the first variant of the sports heart does not mean that there is no hypertrophy of the cardiac muscle, but rather indicates that /269 the expansion which accompanies this hypertrophy was slight. We know that even considerable hypertrophy without manifest expansion of the cavities may have no essential influence on the size of the heart, particularly as concerns the right ventricle (Zdansky, 1952). Therefore, we might consider that the second variant of the heart does not mean that there is no hypertrophy of the muscular wall of the right ventricle. In athletes, we frequently encounter so-called concentric hypertrophy, causing rounding of the contours without manifest enlargement of the transverse diameter, free retrocardial space, acute cardiodiaphragmatic angle, low area of contact of heart with diaphragm. In the third variant, apparently, elements of significant expansion of both sections of the heart predominate, which, considering the weaker muscular wall of the right ventricle, determines in many cases the more rapid formation of functional disruptions.

The predominance in these cases of expansion elements is confirmed by the comparatively rapid decrease in the dimensions of such a heart (particularly due to the right ventricle) as the functional state of the organism is improved and phenomena of overtraining and overstress are liquidated.

In contrast to the actually generally accepted point of view of the /271 physiological nature of hypertrophy and enlargement of the "sports heart," there is a tendency to see pathology in any hypertrophic athletic heart

(A. G. Dembo, 1966). However, this point of view, which arose as this problem first began to be studied (Selig, 1908; Beck, 1909; etc.), and has been repeated by individual investigators since (L. F. Dmitriyenko, 1926; Landen, 1947), has never been confirmed, experimentally or clinically. However, since hyperfunction always involves maximum mobilization of the resources of the myocardium (F. Z. Meyerson, 1963) athletes with very large hearts should be put under constant and attentive medical observation.

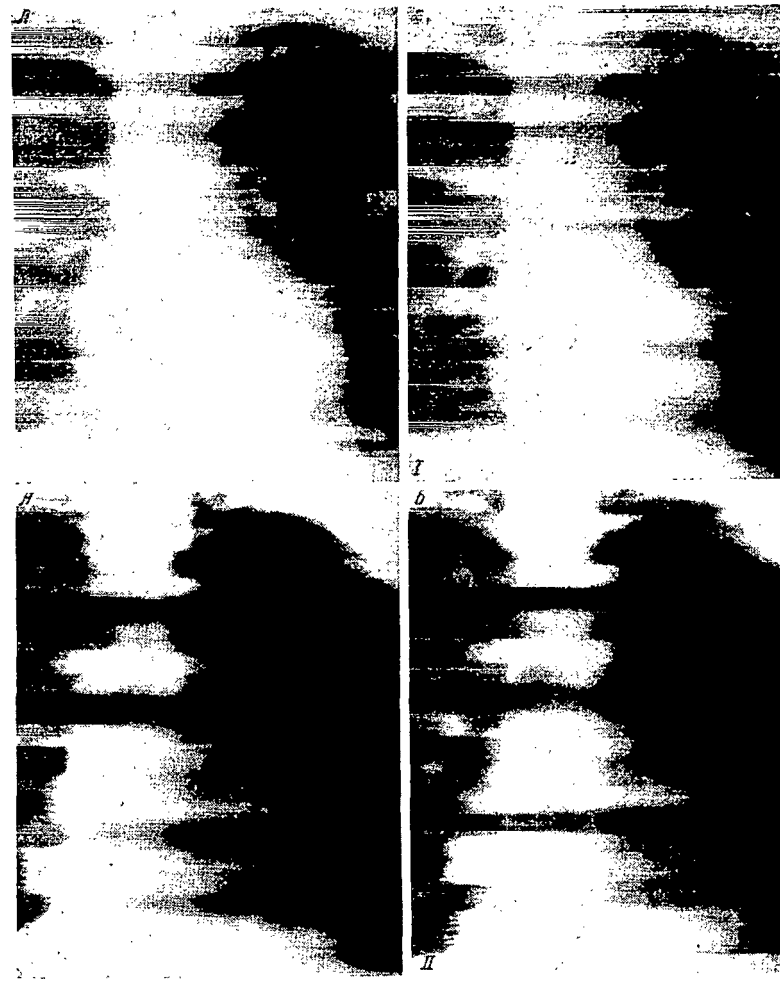

Figure 83. Change in Roentgenkymogram of After Physical Stress. I, roentgenkymogram of heart of athlete N (decrease in dimensions of heart); II, roentgenkymogram of heart of athlete L (increase in dimensions of heart). A, before loading; B, after loading

Changes in the roentgenkymogram under the influence of physical loading. In order to determine the functional state of the athlete's heart, it is expedient to use roentgenkymographic investigation not only in the state of relative muscular quiet, but also after physical loading.

If the physical loading corresponds to the functional capabilities of the organism, the dimensions of the heart during and after loading will be decreased, the amplitude of the pulsations will be increased, and the first type will be retained or the second type will be changed to the first or third type. The area of ventricular waves on the right may be enlarged, and the extent of the remaining zones will decrease little. The waves become sharper, and the systolic shank takes on a steeper direction, the diastole is shortened (Figure 83). The decrease in dimensions of the heart after loading results from more complete emptying of residual blood.

Certain authors (V. V. Zodiyev, 1957) found an increase in the amplitudes of pulsation after loading primarily in insufficiently trained athletes. Special investigations on athletes with various functional states of the cardiovascular system which we performed have shown that an increase in amplitude after loading is discovered in almost all athletes, between 1 and 7 mm. However, well trained persons with normotonic reaction of the cardiovascular system to loading characteristically show moderate increases in amplitude (1-4 mm), while persons with distonic reactions in many cases show greater increases in amplitude. With poor functional state in many cases even a decrease in the pulsation amplitude was noted.

With poor functional state or with extreme loadings, the roentgenkymogram may show an increase in heart dimensions after muscular work with retention of the second or third type of pulsation and a decrease in amplitude (Figure 83, II).

During the recovery period, the dimensions of the heart gradually return /272
to the initial dimensions. Comparative investigations which we performed in various stages of the recovery period after various physical loads showed significant differences in the dimensions of the heart at 10-20 and 60-80 sec following loading. The greatest decrease in heart dimensions was noted in most cases at five minutes following loading. The nature of the load was also significant: the more intensive the loading, the more clearly differences were expressed in the nature of the change in heart dimensions after loading depending on the functional state and type of response reaction of the cardiovascular system to loading, according to the data of other hemodynamic indicators.

Apparently, it is the time differences in measurement of roentgenkymograms and varying nature of loading which explain the variations in data concerning changes in heart dimensions after loading. For example, in contrast to most authors, who found a decrease in the dimensions of the heart after loading (Ya. I. Kaminskiy, N. A. Podkaminskiy, A. N. Krestovnikov, S. V. Shestakov, S. P. Letunov, A. A. Gorodetskiy, V. G. Ginzburg,

N. D. Grayevskaya, N. V. Savina, V. V. Zodiyev, Gotthardt, Moritz, Reindell, Raab, etc.), E. M. Gel'shteyn and A. I. Dombrovskiy observed no decrease in dimensions of the heart, while N. V. Potte, T. Bordosh, G. Komadel, A. Kreze and others even found an increase in heart dimensions after moderate physical loads.

As special experimental investigations have shown, the quantities characterizing heart dimensions and its amplitude may vary in the same athlete over a comparatively short period of time under the influence of various factors, particularly in connection with the nature of the loads being undertaken. The greatest changes in roentgenkymogram indicators of the heart occur in the immediate recovery period at the fifth minute after comparatively slight loading and at the fifth through fifteenth minutes after very heavy ("maximal" type) physical stress. Therefore, during dynamic observations it is important to take the roentgenkymogram at rest under relatively identical conditions with respect to the regime of the preceding day and the time of last loading, and to use identical loadings for each athlete during various stages of observation in order to study the influence of loading on the heart, and to take the roentgenkymogram at the identical time after loading.

Changes in the roentgenkymogram during the course of the training process. The changes in the roentgenkymographic picture of the heart in qualified athletes with increasing state of training during each sports season are comparatively slight: in most cases, we find only a slight increase in the area of the heart, principally resulting from its elongation, /273
and increase in the left ventricular zone and a slight increase in amplitude.

In case of overloading, the enlargement of the heart (increase in transverse diameter and chord of the right ventricle) is one of the most constant symptoms. Also, a decrease in the tonus of the cardiac muscle, and a decrease in amplitude of pulsation are noted.

Dynamic observations of trained athletes who achieve high sports results allow us to distinguish three types of changes of the heart during the process of many years training.

1. A gradual increase in hypertrophy (not reaching any great extent) without expressed elements of expansion. The dimensions of the heart generally correspond to the norms or (rarely) exceed them by not over 5-10% and remain the same over many years of observation (Figure 84). This is /274
usually true of healthy, properly trained athletes involved in those types of sports which do not place unusually great demands on the cardiovascular system.

2. A gradual increase in the dimensions of the heart, occurring principally in the left ventricle. The dimensions of the heart increase primarily during the first years of training, then stabilize, reaching 5-15% greater than the normal values. This heart retains its high functional capacity for many years of training. This type of change is most frequently

observed in athletes trained with large volumes of work, but when this work corresponds to the functional capabilities of the organism.

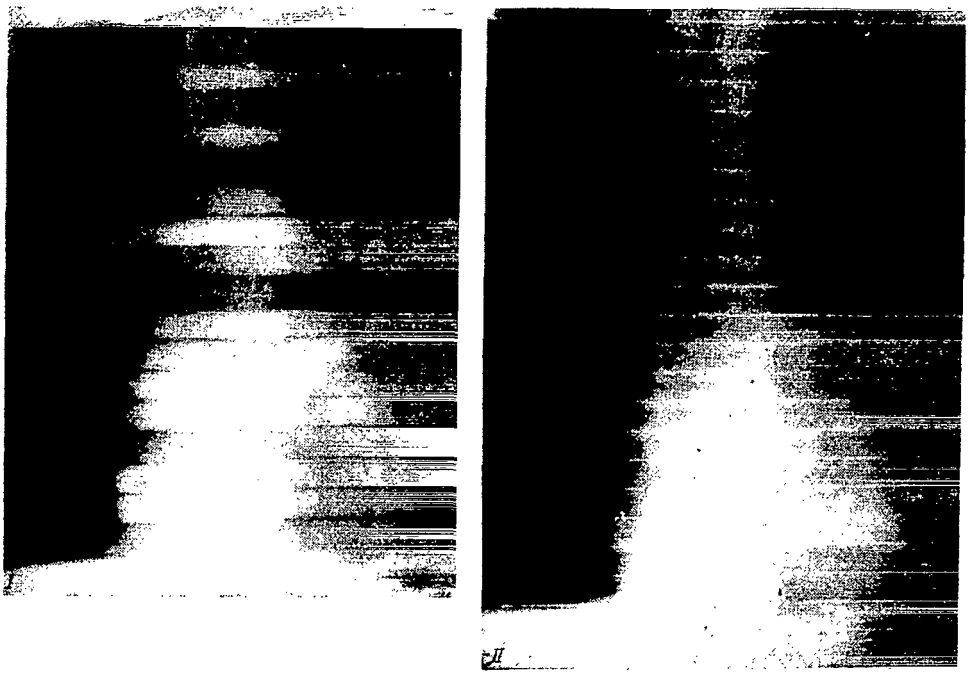

Figure 84. Roentgenkymogram of Football Player I (No Manifest Changes over 10 Years of Training). I, in 1953; II, in 1964

3. A rapid increase in dimensions of the heart, covering both left and right ventricles and the transverse dimension of the heart (Figure 85). The increase occurs most frequently during the first period of training with heavy loads, but in some cases may appear in athletes who have trained for some time as a result of overloading, insufficient recovery, entry into competition while sick, etc. /275

A roentgenkymographic picture characterized by significant enlargement of the heart both right and left may be either stable (in certain cases the characteristics of decreased working ability may not appear) or, if training is changed in time, may show reverse development with corresponding decrease in heart dimensions. However, any degree of enlargement of the right ventricle in most cases still remains -- the third type of "sports heart" is developed or approximated. In athletes with these specifics of the heart, significant fluctuations in heart dimensions are frequently seen over several years of observation (primarily changes in the diameters showing the size of the right portions of the heart). The indicators of the functional state and working ability are generally unstable. Therefore, discovery of these

changes during ordinary roentgenkymographic investigation should always be a signal to the doctor that the athlete needs further comprehensive examination.

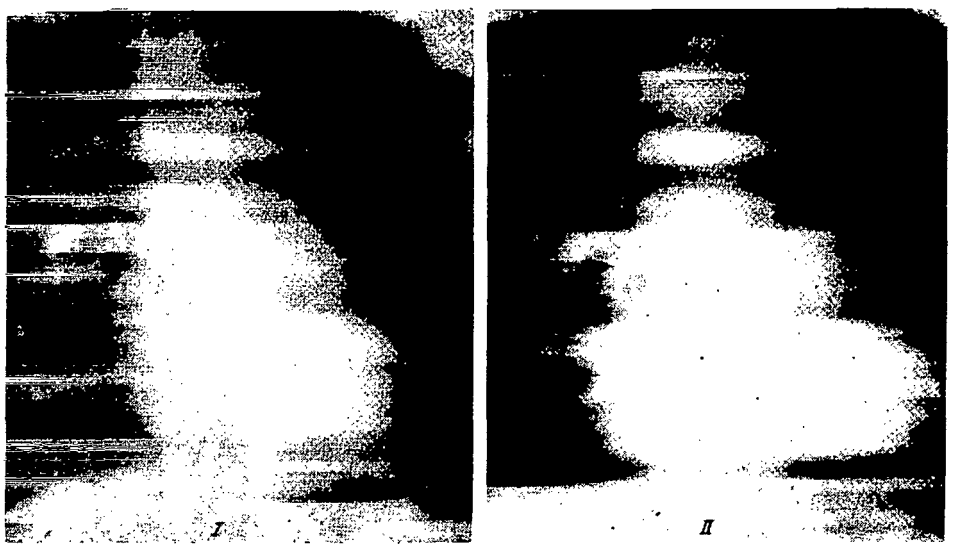

Figure 85. Roentgenkymogram of Football Player M (Rapid Increase in Heart Dimensions During First Stage of Training). I, July 1957 (beginning of training in team of masters); II, November 1957 (after 4 1/2 months)

After long periods following sports training (10-15 years or more after cessation of training directed toward the achievement of high results), two main variants of changes in the roentgenkymographic picture of the heart are observed.

1. Retention of approximately the same picture as during involvement in sports (Figure 86). In this case, we frequently see only a slight decrease in dimensions due to reverse development of the elements of tonogenic expansion. In athletes older than 45-50 years, we may see some elongation of the heart, less roundness of the contours, and lower pulsation amplitude. In many cases, the first type of pulsation goes over into the second or third type. The area of contact of the heart with the diaphragm increases slightly, but the cardiodiaphragmatic angle remains acute or right. This variant is observed in general in persons who continue systematic physical exercise to some extent after cessation of special training and who therefore retain high functional capacity of the cardiovascular system. In old age (after 55-60 years) in many cases we see extended aorta and more clearly expressed pulsation of the right vascular contour.

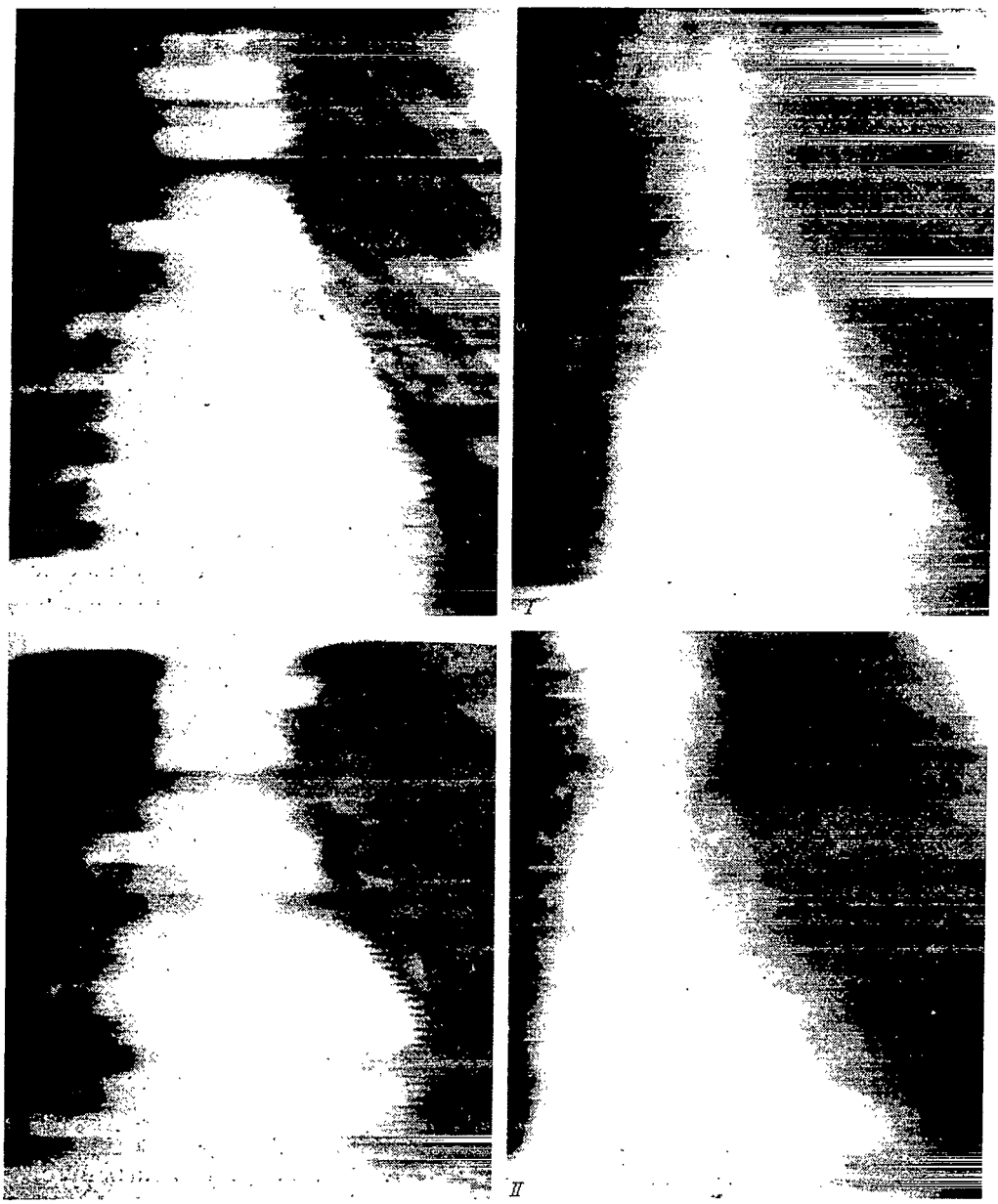

Figure 86. Roentgenkymogram During Period of Sports Training and 10 Years After Cessation of Training. I, with continuation of systematic physical exercise (roentgenkymogram of honored master of sports in football B). A, at 35 years of age (1953); B, at 45 years of age (1963); II, with sudden cessation of physical exercise (roentgenkymogram of honored master of sports in football K): A, at 29 years of age (1952); B, at 39 years of age (1962).

2. With sudden cessation of sports exercise and complete exclusion of physical exercise from the daily regime, manifest changes in the roentgenkymographic picture of the heart develop rapidly. The heart becomes considerably longer, takes on a more transverse position and contacts the diaphragm broadly; the left cardiodiaphragmatic angle becomes obtuse, the /277
amplitude of pulsation decreases notably, and the age expansion of the aorta and pulsation of the right vascular contour are more clearly expressed. The functional state of the cardiovascular system and adaptation of the organism to loads under these conditions generally become considerably worse.

Frequently, these changes appear at comparatively young age (up to 40 years), whereas persons continuing exercises show this picture less frequently (as a rule only after 55-60 years, sometimes considerably later).

Roentgenkymographic investigation of the heart still has considerable significance at the current stage of development of functional cardiology for the determination of morphological and functional features of the athlete's heart. The usage of this method for dynamic observations allows us to establish the effects of physical exercise on the state of the heart, and to study the development of hypertrophy of the cardiac muscle and the dynamics of its contractive capacity, determining characteristics of overloading of the heart in a timely fashion.

Electrokymography

The usage of electrokymography to study the heart in athletes has been the subject of only a few works (Kjellberg et al., 1949; Linko et al., 1958; Ya. G. Ablov, 1961; Yu. I. Akimov et al., 1962; V. V. Bulychev et al., 1965). As the literature data show, this method can be used to study the contractive activity of the heart, which is of particular significance for sports medicine. Electrokymography is a comparatively new method of functional investigation of the heart, large vessels, lungs and other organs. It allows us to record the pulsating movements of individual sectors of the myocardium and main vessels using X rays and a photoelectric cell, and is a continuation and further improvement of the method of roentgenkymography.

The method was suggested in 1936 by the German roentgenologist Heckmann under the name actinocardiography, and somewhat later it was independently developed by Hjalmare (1937) under the name fluorocardiography. Lian, Minot (1939) called this method radioelectrokymography, and Marchal (1946) called it kinedensigraphy.

The decisive role in the development was played by the works of the /278
American authors (Chamberlain, Henny and Boone, 1945), who suggested the now generally used name of electrokymography, and who used a special electron apparatus, due to which it became possible to record curves of the pulsation of the heart -- electrokymograms (EKI) with high-amplitude waves (30-40 mm).

In the USSR, electrokymography was first used by V. V. Zaretskiy in 1956, then by a number of other authors (V. N. Orlov, 1959; Yu. I. Akimov, L. L. Orlov, 1960; V. A. Shanina et al., 1961; Ya. G. Ablov, 1961; I. Kh. Rabkin, E. A. Grigor'yan, 1962; etc.). In 1960, the first domestic electrokymograph, the EKS-60, was developed in our country.

The principle of electrokymography is that fluctuations in the intensity of a stream of X rays arising due to pulsations of the heart are converted by a photocell installed in the path of these rays into electric current oscillations. Changes in the voltage in the circuit of the photocell are recorded by an oscillograph in the form of a curve -- the electrokymogram.

The device consists of three main parts: 1) the receiving fluoroscopic element, contained in a lead case with a rectangular slit measuring 6 × 25mm moved by a special mechanism in the frontal plane between the screen of the X-ray apparatus and the patient; this transducer is connected to a second photocell, installed directly at the X-ray tube and serving to eliminate fluctuations in voltage in the circuit feeding the X-ray apparatus; 2) a filter; 3) a strip chart recorder to produce the electrokymograms on photographic film or paper strips moving at various speeds (from 25 to 100 mm per sec).

The polarity of the device is set so that an increase in the intensity of the X rays causes a drop in the recorded curve, while a decrease in the intensity causes the curve to rise. Consequently, an EKI sector directed downward indicates movement of the edge of the heart inward, while an ascending curve sector corresponds to outward movement of the edge of the heart.

The amplitude of the electrokymogram does not reflect the magnitude of motion quantitatively, since it is determined by a combination of many factors (operating mode of the X-ray apparatus, degree of absorption of rays by the patient, transparency of adjacent sectors of lungs, gain used in the device, etc.). Therefore, although a number of suggestions have been made for standardization of amplitudes (Ring et al., 1949; Lissner, 1957; V. V. Zaretskiy, 1959; etc.), the value of the electrokymographic waves can be compared with confidence only for the same test subject under identical recording conditions.

Proper interpretation of electrokymograms is impossible without /279
synchronous recording of an electrocardiogram, phonocardiogram or sphygmogram. The best variant is simultaneous recording of several curves, the selection of which is determined by the purpose of the investigation and the capabilities of the apparatus used. Also, simultaneous recording of two or three electrokymograms using several photocells can be used (Luisada, Fleischner, 1947; Gambaccini, 1956; V. V. Zaretskiy, 1963; etc.).

Methodology of electrokymography. The recording of electrokymograms is performed under conditions of ordinary roentgenoscopy in the standing,

sitting or lying position. The most convenient position is the sitting position on a special seat with a support for the body and extremities. Since the slightest movements of the patient distort the recording, the standing position is permitted only for physically strong persons, for example athletes.

The photocell transducer is placed in front of the test subject, installed with its slit at the boundary of the cardiovascular shadow and always perpendicular to it at the point investigated, guided by the X-ray screen. Due to this placement of the photocell, linear movement of the edge of the heart is recorded in undistorted form, with the oscillations of the contour not going beyond the limits of the slit. The speed and accuracy of placement of the transducer depends on the experience of the investigator and is checked visually by the quality of the curve on the oscilloscope. The amplification system allows waves up to 30-40 mm high to be produced, which greatly exceeds the heights of the waves on the roentgenkymogram and thereby assures better conditions for their analysis. Recording is performed with the breath held, during which 5-6 cardiac cycles are continuously recorded at a paper movement speed of 50-100 mm per sec. The operating mode of the X-ray apparatus is: voltage 60-70 kv, current 2-4 ma with minimum diaphragm aperture. Electrokymograms are recorded in sequence for the left and right edges of the heart on various sectors of the ventricles, atria and large vessels around the contour of the shadow, using the roentgenological positions (Figure 87). The total investigation usually lasts not over five minutes, during which the irradiation amounts to 20-30 r, 3-5 times less than the permissible one-time dosage.

In order to represent the principal (standard) leads of the electrokymogram, authors writing in the domestic literature have adapted the suggestion of V. V. Zaretskiy (1957). Thus, the first recording of the left ventricle in the area of the apex (influx path) is represented as V_1S, the recording of the middle portion of the left ventricle -- as V_2S, the base of the heart (efflux path) -- as V_3S. The same recordings in the first (right) inclined projection are represented as I-V_1S, I-V_2S, etc., while in the second (left) slanted projection, they are represented as II-V_1S, etc. The electrokymograms of the right ventricle are represented as V_1D, V_2D, etc., of /280 the atria -- as AS and AD, of the arc of the aorta as arc A_0, of the pulmonary artery -- as AP, etc.

At the same time as the electrokymogram, the same paper strip chart is used to record the accompanying curves (one or several), for which the corresponding transducers are placed over the test subject. It must be recalled that during synchronous recording, the electrokymogram is delayed in relationship to the electrocardiogram or phonocardiogram by an average of 0.02 sec as a result of the delay of the electrical signal in the circuitry of the electrokymograph.

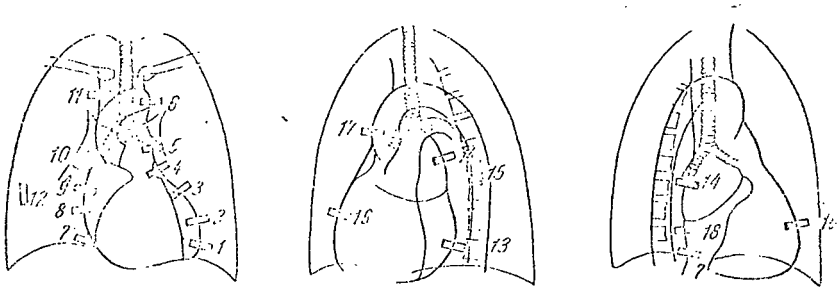

Figure 87. Points of Recording of Electrokymograms of the Heart and Large Vessels in Principal Roentgenological positions (After Luisada). A, anterior-posterior position: 1, apex; 2, left ventricle, middle portion; 3, left ventricle, upper portion; 4, left auricle; 5, pulmonary artery; 6, arc of aorta; 7, inferior vena cava; 8, right atrium, middle portion; 9, right atrium, upper portion; 10, right hilus; 11, vena cava superior; 12, right lung, lower portion. B, left anterior slanted position: 13, left ventricle rear wall; 14, left atrium rear wall; 15, descending aorta; 16, right ventricle front wall; 17, ascending aorta. C, right anterior slanted position: 7, inferior vena cava; 14, left atrium, rear wall; 16, right ventricle; 18, right atrium, rear wall

Form of normal electrokymograms. Normal electrokymograms of the left ventricle (Figure 88, A) have the form of large waves with a long and smoothly ascending shank during the diastole and a shorter and more steeply descending hook during the systole. Directly after tone II or the T wave on the electrocardiogram, before the beginning of the ascending sector of the electrokymogram there is usually a small DEF wave -- the "protodiastolic wave," reflecting the noncontractive activity of the ventricle during the early diastole. Point D in many cases corresponds with the beginning of tone II and corresponds to closure of the semilunar valves of the aorta, i.e. the end of the protodiastolic period and the beginning of isometric relaxation. Opening of the mitral valve most probably corresponds to point E (Luisada et al., 1948; Yu. I. Akimov et al., 1962), after which the course of /281 the curve reflects the nature of filling of the ventricle. In the area of the apex (influx path), filling occurs intensively from the very beginning of the diastole; this process predominates over the positional change of the heart, and the protodiastolic wave is frequently not noted. At the base of the heart (efflux path), on the contrary, this wave is always well expressed.

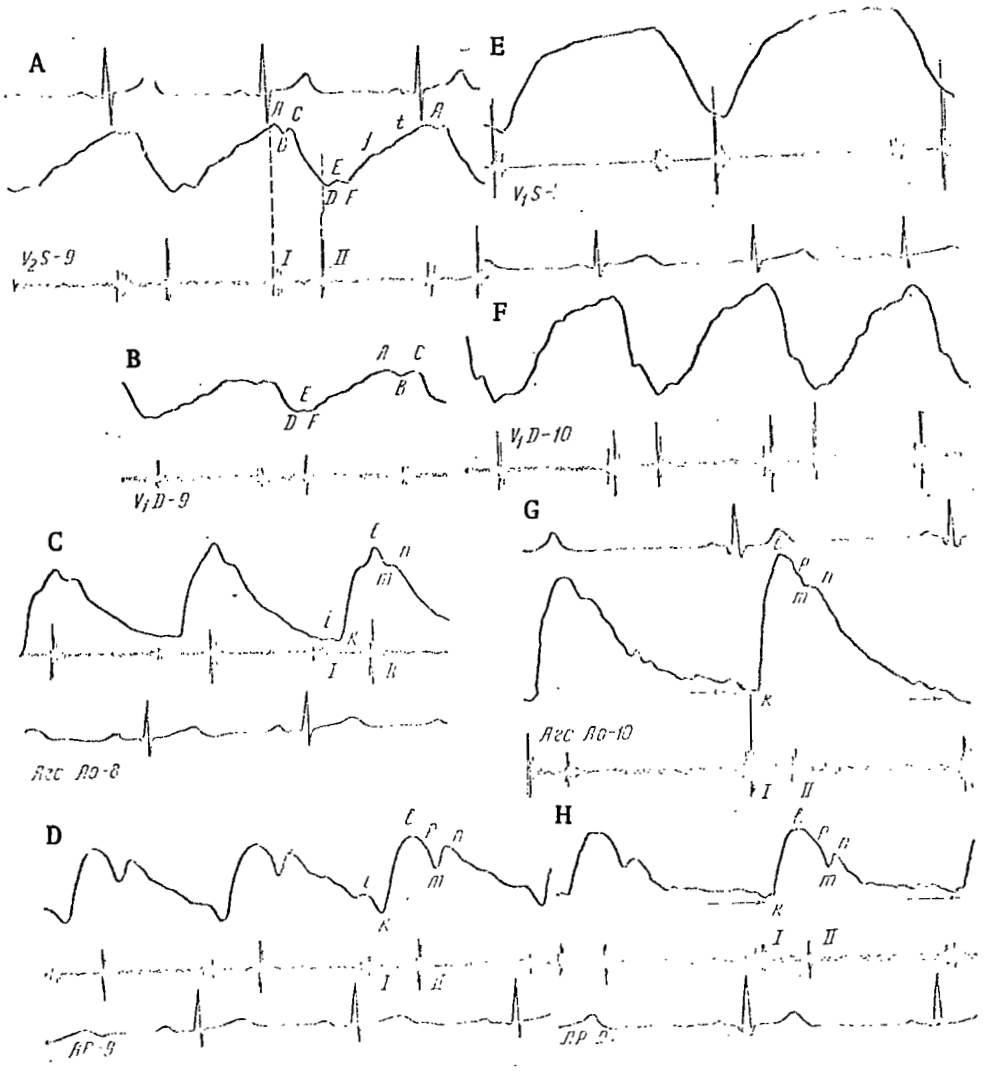

Figure 88. Electrokymograms of Ventricles and Large Vessels in Persons Not Involved in Sports (Left) and Athletes (Right). Explanation in text.

On the ascending shank of the electrokymogram of the ventricle, we distinguish the initial convex, steep sector (phase of rapid filling) and the less steep, final sector (phase of slow filling). However, this differentiation of the slow filling period is usually possible only on electrokymograms recorded near the apex; at the base of the heart, the course of the curves is more gradual, sometimes even concave. This reflects the varying nature of filling of the influx and efflux paths of the ventricle, manifested primarily in the standing position. In the prone position, these differences are

smoothed. At the end of the diastolic sector, we frequently note a small wave, the "terminal wave," which is related to the atrial systole.

The peak of the wave (point A) usually corresponds to the beginning of the high-frequency oscillations of tone I and has been identified with closure of the mitral valve. Between point A and the beginning of the main descent of the curve in the systole (point C) in most cases we see a small deepening AB, the "protosystolic drop," best expressed at the apex. It is related to the change in form of the ventricle during the stress period, contraction of the influx paths and the intraventricular movement of blood from the influx path to the efflux path (Heckmann, 1960). Completion of the stress period and the beginning of the expulsion period is usually disguised on the electrokymogram of the ventricle by the positional shift of the heart -- in most cases a movement to the left and dorsal rotation of the longitudinal axis. This movement results in the formation of the positive C wave, after which the main descent of the electrokymogram begins. The beginning of expulsion can be defined from the aortal electrokymogram (see p. 270); in time, the moment of opening of the semilunar valves corresponds most probably to point B, particularly in the V_2S lead, which has an intermediate position between the blood paths. In the area of the base, the protosystolic drop is less clearly expressed, the C wave is higher, sometimes dominant, and the course of the curve during the stress period and early systole is frequently ascending. This corresponds to deformation of the ventricle and arrival of a volume of blood to the efflux path, which begins to contract later than the influx path. The medial movement through the entire contour of the ventricle reflects its decrease in volume during the phase of blood expulsion.

The time interval between the beginning of tone I and point C is the "latent time" of the electrokymogram (Heckmann, 1952). It frequently exceeds the time of isometric contraction and reflects the duration of oscillating movements of the heart, as well as the intraventricular displacement of blood along the blood flow paths and, as graphic phase analysis of electrokymograms has shown (see below), is of important significance in the determination of the specifics of the mechanical activity of the heart. /283

Electrokymograms of the right ventricle (Figure 88, B) generally are similar in form, but are frequently distorted by the influence of the right atrium. "Pure" curves are usually produced in the middle section of the right contour with the test subject rotated to the right. As a rule, the waves of the right ventricle are lower in amplitude and differ in their time relationships from those of the left ventricle. There are data indicating asynchronous contraction of both ventricles with differences revealed by electrokymograms of the ventricles of up to 0.03 sec. The right ventricle most frequently contracts first.

Electrokymograms of the aorta (Figure 88, C) recorded in the area of the aortal arc, have all the characteristics of the central pulse on a sphygmogram and, when synchronized with the electrocardiogram and phonocardiogram,

allow us to calculate all phases of the left ventricular systole. Also, it must be kept in mind that the nature of movement of the aorta is determined by the relationship of its transverse or eccentric pulsation (expansion and contraction of the walls) and longitudinal pulsation (rigid straightening of the vessel during the systole and increase in durvature during the diastole). Both forms of movement change with age: as the elasticity of the walls decreases, the longitudinal pulsation increases to compensate; the movement of the aorta as a whole thus increases, which causes smoothing of fine details on the electrokymogram (isometric wave, incisure, etc.). These specifics may make it difficult to differentiate individual phases of the systole from the aortal electrokymogram, but they are of diagnostic significance.

The electrokymogram of the ascending aorta is recorded in the second slanted position, and is distinguished by the lower amplitude and considerable deformation resulting from superimposition of the pulsation of the superior vena cava or vessels of the lungs, which makes time calculations based on this curve quite difficult.

The electrokymogram of the pulmonary artery (Figure 88, D) is similar in general form to the curve for the arc of the aorta, but is frequently reminiscent of the peripheral pulse curve. The rise in the electrokymogram during the phase of rapid expulsion is smoother and lower, and an anacrotic drop is more frequent; the peak is more rounded and occurs later in the systole, and the predicrotic incisure is deeper and is placed nearer the base line; the dicrotic wave is larger.

The electrokymograms of the atrium are quite polymorphous since, in addition to movements of the atrial walls, they reflect the transfer pulsation of the adjacent ventricles and vessels. /285

Electrokymograms of the left atrium (Figure 89, A) in the direct position are recorded in the area of the auricle and consist generally of three principal waves: one in the systole and two during the atrial diastole. Also, two- and four-wave curves are encountered. The nonconstant descent A_1-A_2 in the presystole corresponds to contraction of the atrium and ends synchronously with the Q or R wave on the electrocardiogram. During the stress perios in the ventricle, we see the rise A_2-S_2, which sometimes has a wave S_1, related to closure of the mitral valve. This sector results from positional displacement of the heart and filling of the atrium. The peak of the second wave S_2 usually corresponds to the opening of the semilunar valves of the aorta, while the descending sector S_2-S_3 results from movement of the base of the ventricle downward toward the apex during the phase of rapid expulsion. At point S_3, filling of the atrium becomes dominant and its wall moves outward to point S_6, although this movement may show waves

corresponding to the moment of closure of the aortal valves. The peak of the third wave S_6 reflects the beginning of passive emptying of the atrium after opening of the mitral valve; at point D_2, in connection to the low pressure gradient, arrival of blood into the atrium from the pulmonary veins begins to predominate over its efflux into the ventricle, and the walls of the atrium move outward until the beginning of a new atrial systole. The S_2 and S_6 waves are the most constant complex in the atrial electrokymogram (two-wave curve).

The electrokymogram of the right atrium (Figure 89, B) can be well recorded in the direct position and generally repeats the form described for the curve of the left atrium. Since the walls of the right atrium are more movile and thinner than those of the left atrium, the amplitudes of their movements are somewhat greater. At the same time, a less expressed nature of the second wave on the electrokymogram of the right atrium is typical, since the influence of the right ventricle on pulsation of the right atrium is less. There are also certain differences in the time relationships. In particular, data have been presented indicating earlier contraction of the right atrium than the left (by 0.01-0.3 sec).

Graphic phase analysis of electrokymogram according to Heckmann[1]. Individual ventricular electrokymograms give no conception of the nature of the movements of the heart as a whole. Therefore, it is necessary to record electrokymograms at several points on the contour of the ventricles and add 286
them using a special graphic method, which Heckmann (1952) called electrokymogram phase analysis. The technique of this analysis is as follows (Figure 90).

Ventricular electrokymograms recorded with identical amplification at various points on the contour (V_1S, V_2S, etc.) are placed one beneath the other. These curves are synchronized using the phono- or electrocardiograms. In analyzing the systole, the corresponding electrokymogram interval is divided by vertical lines into so-called isophases. The intersection of each electrokymogram with the vertical line corresponds to the position of a given point on the contour at an individual moment in the systole, and the distance from each intersection point to a horizontal line drawn through the intersection of each curve with the first vertical line reflects the amplitude and direction of displacement of the contour in relation to its position at the beginning of the systole, which is shown on a drawing of the heart. On lines drawn perpendicular to the contour of the heart at the point of recording of

[1] We should differentiate phase analysis of cardiac activity (the study of the duration of the phases of the cardiac cycle and a number of indicators of cardiodynamics) and phase analysis in electrokymography. In the latter case, apparently, we might more properly speak of phase contrast or, as the author of this section has used, graphic phase analysis--Ed.

each electrokymogram (V_1S, V_2S, etc.), the amplitude of displacement of the corresponding curve is laid out for the individual isophases of the systole. By connecting the points produced on each perpendicular line at each isophase, a drawing of the total movement of the contour is produced at each point through the entire systole. The same can be done for the diastole. /287

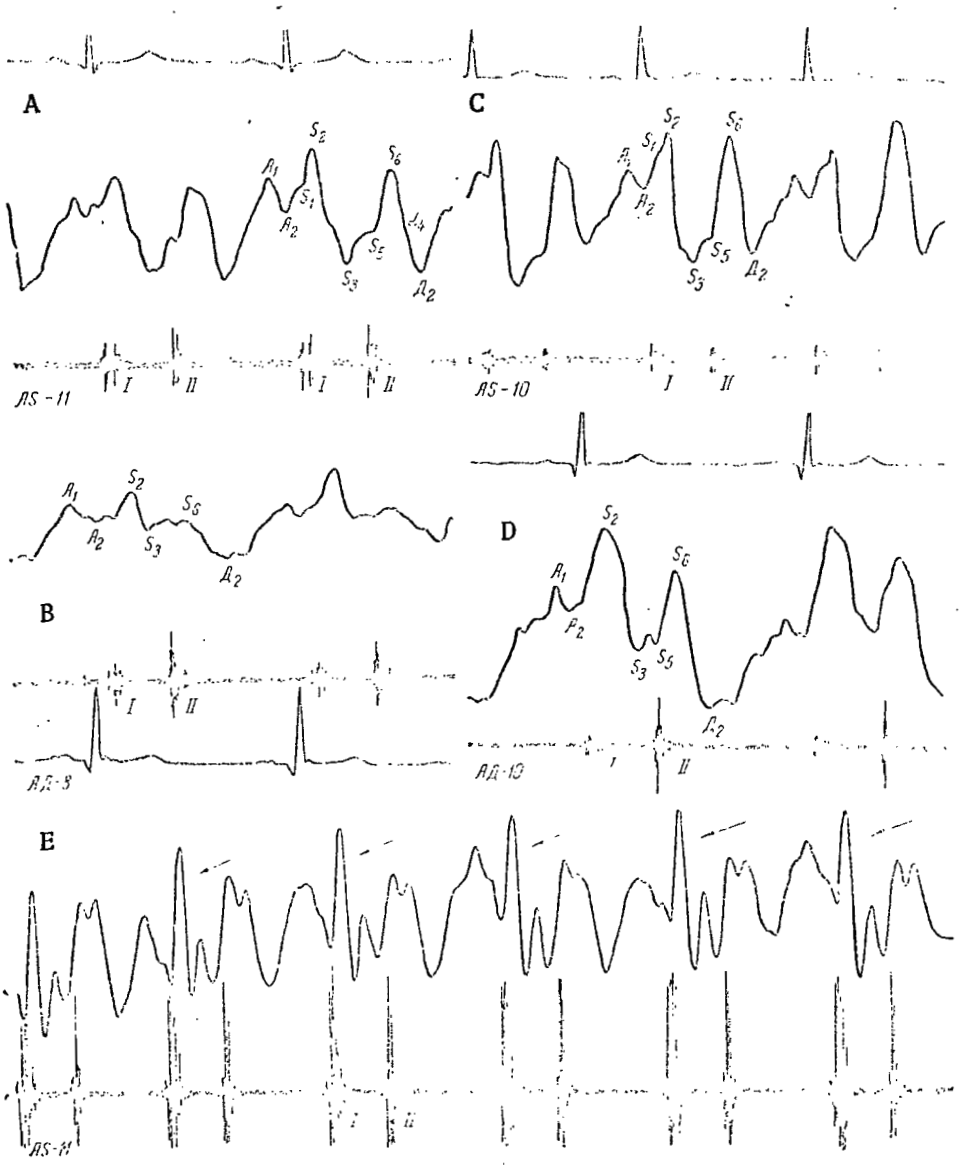

Figure 89. Electrokymogram of Atrium in Persons Not Involved in Sports (Left) and in Athletes (Right). Explanation in text.

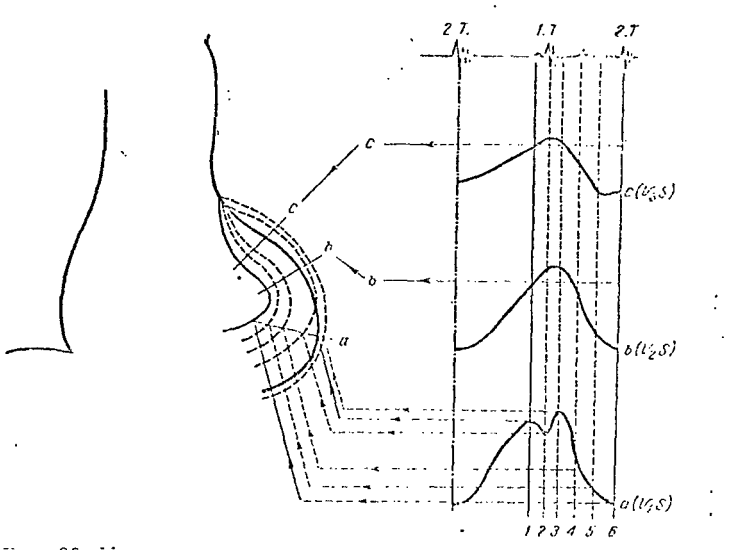

Figure 90. Method of Graphic Phase Analysis of Electrokymograms (According to Heckmann, 1959). Explanation in text.

This type of graphic phase analysis of electrokymograms is called vertical phase analysis, but it may also be performed in the horizontal plane. In order to do this, the ventricular electrokymograms are recorded at one level (for example V_2S) in sequence in the direct and slanted positions, and the graphic analysis is performed through an arbitrary transverse "cross section" of the heart. Without showing the true form of the heart or value of pulsation, phase analysis of electrokymograms does allow us to establish the beginning of movement of the individual sectors of the contour, the direction of the movement and the relative speeds.

Thanks to graphic phase analysis, on each curve we can separate the two main components of the dynamics of the heart: the pulsatory and so-called supplementary or noncontractive movements. These latter movements are best manifested in the isometric phases of the systole and diastole. Generally, they can be divided into movements related to changes in position of the entire heart (displacement of the center of mass), rotation of the heart about its longitudinal axis and the change in position (placement) of the longitudinal axis itself.

In some cases in healthy persons there are no supplementary movements of the heart, and therefore changes in the form of the heart during the stress period are clearly seen: it becomes nearly spherical, in place of the ovoid form at the end of the diastole (Figure 91, A). The movement of the edges of the heart during the systole reflects the normal course of contraction of the ventricular myocardium, the influx paths contracting first, followed by the efflux paths, i.e. the sequence of contraction corresponds to the direction

of the excitation wave from the apex to the base of the heart. As was noted above, filling of the ventricle also occurs earlier and more rapidly in the influx paths.

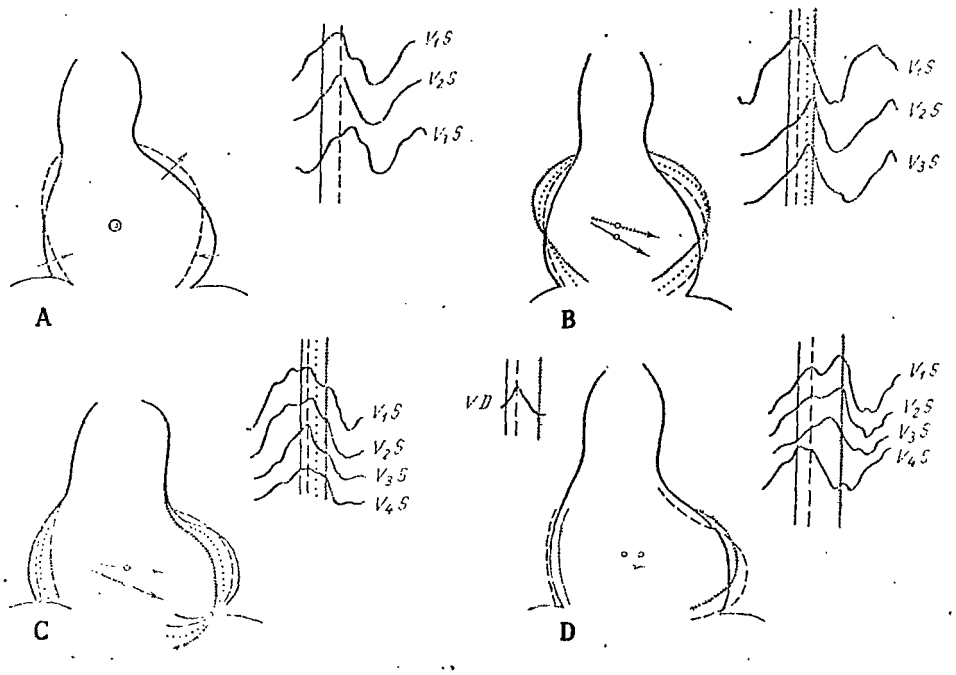

Figure 91. Noncontractive Movement of Heart According to Data of Graphic Phase Analysis of Electrokymograms. Explanation in text.

In the overwhelming majority of healthy persons, there are noncontractive displacements of the heart. They arise as a result of the summary effect on the heart of the surrounding organs (elastic resistance and retraction of the lungs, influence of the diaphragm, etc.), changing in turn as a function of the phase of respiration and position of the body. This effect appears primarily during the diastole, when relaxation of the myocardium is most subject to the influence of external forces. During the systole, the form and position of the heart are determined to a considerable extent by its anatomical structure and contractive ability. The influence of neighboring organs is less expressed and the heart is relatively fixed in its initial position. Phase analysis indicates the following forms of supplementary movement of the heart (Heckmann, 1952; V. N. Orlov, 1960; etc.):

1. Oscillation of the entire heart during the systole upward toward the /288 base (Figure 91, B). The apex lifts away from the diaphragm, while the longitudinal axis rotates upward (latent time of electrokymogram lengthened from apex to base). This type of movement is characteristic for persons with

more vertical position of the heart and low level of diaphragm -- the heart is affected during the systole by the downward directed pull of the idaphragm, and during the diastole drops together with it.

2. Penetration of the apex into the diaphragm during the systole and rotation of longitudinal axis of heart downware (Figure 91, C). During this, a short latent time and even movement of the entire contour inward are observed. This form of movement is particularly manifest with transverse position of the heart and high diaphragm -- contracting, the heart acts against the underlying diaphragm.

3. Movement of the entire heart to the left during the early systole and to the right during the early diastole (Figure 91, D). At the same time as these lateral oscillations (movement of the center of mass), the longi- /289
tudinal axis of the heart rotates dorsally and remains in the most "transverse" position, the latent time of the electrokymogram is lengthened from base to apex, masking the normal course of contraction. During the main portion of the systole, ventral rotation of the longitudinal axis is noted until it is in the steepest position by the beginning of the diastole. The movement to the left is concealed (Heckmann, 1952) and lasts an average of 0.06 sec. It is encountered most frequently with slanted position of the heart and medium diaphragm level in healthy persons.

The noncontractive movements of the heart characterize indirectly the working ability of the myocardium: if the contractability is weakened, regardless of the cause, including in cases of pathological hypertrophy and dilation, they are decreased considerably or disappear completely; at the same time, they are almost always observed in the work of a healthy heart.

Method of calculating phases of cardiac cycle from the electrokymogram. One advantage of electrokymography is the possibility of determining the duration of the systolic and diastolic phases of the cardiac cycle individually for the left and right chambers of the heart. Data on the duration of the phases of the cardiac cycleproduced from the electrokymogram sometimes do not correspond to the results of other methods of investigation due to the varying approach to the delineation of the various phases. The following method for time analysis of the electrokymogram can be recommended.

1. Left ventricular systole:

a) phase of asynchronous contraction -- interval Q-I tone.

b) phase of isometric contraction -- interval from beginning of high-frequency component of tone I to beginning of anacrote on electrokymogram of aorta (minus time correction for propagation of pulse wave, which K. V. Chertinskiy (1963) considers to be equal to the interval between the beginning of tone II and the lower point of the incisure);

c) phase of rapid expulsion -- from beginning of anacrote of electrokymogram of aorta to its peak;

d) phase of slow expulsion -- from peak of electrokymogram of aorta to beginning of incisure (in cases where beginning of incisure is not expressed -- to lower point of incisure minus 0.035 sec, i.e. minus the practically constant duration of the protodiastolic interval).

2. Right ventricular systole:

a) period of stress -- from Q wave to beginning of anacrote on electrokymogram of pulmonary artery minus time correction for propagation of pulse wave (which can be calculated reliably only in cases with splitting of II tone and when, according to K. V. Chertinskiy (1963), it is equal to the interval between the beginning of the pulmonary component of tone II and the lower point of the incisure). The correction can be calculated as a product /290 of the propagation velocity of the pulse wave in the pulmonary artery (I. Kh. Rabkin, E. A. Grigor'yan, 1962) and the distance from the heart to the point of recording of the electrokymogram;

b) expulsion phase -- similar to left ventricular, except using electrokymogram of pulmonary artery.

3. Left ventricular diastole:

a) protodiastole -- descending shank of incisure on electrokymogram of aorta;

b) phase of isometric relaxation -- from beginning of tone II to peak of last wave (point S_6) on electrokymogram of left atrium or to peak of protodiastolic wave (point E) on left ventricular electrokymogram, recorded at middle portion of its contour (V_2S);

c) phase of rapid filling -- from beginning of tone II to point of transition of steep rise of electrokymogram of left ventricle to less steep final sector minus time of isometric relaxation;

d) diastasis (including atrial systole) -- from point of termination of rapid filling phase to Q wave of electrocardiogram.

4. Right ventricular diastole:

a) protodiastole -- descending shank of incisure on electrokymogram of pulmonary artery;

b) phase of isometric relaxation -- from pulmonary component of tone II to point S_6 on electrokymogram of right atrium (or to peak of protodiastolic wave on electrokymogram of right ventricle);

c) filling phase -- similar to left ventricle from electrokymogram of right ventricle.

Specifics of electrokymograms of athletes. The electrokymograms of the ventricles of athletes (Figure 88, E, F), in connection to the frequently observed bradycardia, are more extended in form with manifest smoothing of waves, particularly in the area of the apex of the heart. Since the duration of the diastole is increased, the maximum expansion of the ventricles during filling occurs later, toward the end. On the electrokymogram, this appears in the form of a horizontal course of the curve during the phase of slow filling -- the diastolic plateau.

In contrast to the curves with decreased myocardial tonus, the ascending sector before the diastolic plateau in athletes is smooth. At the same time, the drop in the electrokymogram in the systole is steeper than the rise in the diastole, and is comparatively short, reflecting the active movement of the wall of the heart during the period of contraction. It is characteristic that the protosystolic drop is clearly seen, as well as the protodiastolic wave on the electrokymogram of athletes. This indicates the presence of /291 supplementary movements of the heart.

The latent time of the electrokymograms of athletes' ventricles generally increases from the base to the apex, frequently reaching 0.10-0.12 sec in the area of the apex, which is noticeably greater than the value of this indicator for nonathletes. In addition to elongation of the isometric phase of the systole, which is characteristic of trained athletes, this is explained frequently by the more clearly expressed systolic oscillations of the entire heart to the left. Also, in many cases an increase in the latent time from the apex to the base is observed, i.e. a normal sequence of movement of the contour, not distorted by positional changes of the heart. Comparatively frequently, graphic phase analysis of the movements of the contour in athletes shows intraventricular displacement of a large quantity of residual blood from the influx path to the efflux path.

Along the right contour of the heart in the direct position in athletes, right ventricular electrokymograms are recorded significantly more frequently for athletes than for nonathletes, and are noted over a longer distance from the diaphragm; the electrokymogram of the right atrium frequently is dominated by the ventricular component. The specifics are seen primarily in athletes trained for endurance. In many such cases, the latent time of the electrokymogram for the right contour is increased, corresponding to systolic oscillation of the heart to the right in connection to the predominant enlargement of the right ventricle.

The amplitude of the electrokymographic waves in athletes, as in persons not involved in sports, increases from the base of the heart to the apex. Parallelism between types of pulsation (Stumpf, 1936) on the roentgenkymogram and data from electrokymography is not observed.

Electrokymograms of the vessels of athletes (see Figure 88, G, H) are distinguished by the steep ascent and straight course of the curves during the phase of rapid expulsion, and frequently by increased amplitude. This is

particularly true when the electrokymogram is recorded in the vertical position and indicates the high rate of movement of the walls of the vessels due to the increased contractive power. The "expulsion angle" between the anacrote of the electrokymogram of the vessels and the vertical (Linko et al., 1958), when compared with the same persons, is generally lower on the aorta than on the pulmonary artery. In individual cases, the electrokymograms of athletes show the reverse relationship, which may be an indirect reflection of increased activity of the right ventricle.

With manifest bradycardia, the decrease in the volume of the aorta and pulmonary artery as a result of the efflux of blood to the periphery is frequently terminated long before the end of the diastole. As a result of /292
this, the final sector of the electrokymogram of the vessel corresponds to the isoline (diastolic plateau). On electrokymograms of vessels in athletes, the isometric wave is also frequently well expressed, with a clear depression before the anacrotic uplift, which is related to reinforcement of the displacement of the vessel under the influence of supplementary movements of the hypertrophic ventricle during the stress period.

The electrokymogram of the atrium of athletes (Figure 89, C, D) is distinguished by certain details which reflect the increased influence of the mechanical activity of the ventricles. First of all, this is true of the increase in elevation of curves during isometric ocntraction of the ventricle (sector A_2-S_2), related to the change in form and position of the heart. In many cases, peak S_2 is dominant. The descent of the electrokymogram of the atrium during the expulsion phase (S_2-S_3) is usually increased in amplitude and duration, which indirectly characterizes the force of the contraction of the ventricle. The duration of the atrial systole in athletes is shorter than the norm (averaging 0.05 sec), so that the presystolic drop A_1-A_2 is usually weakly expressed. At the same time, the drop in the electrokymogram of the atrium during the phase of rapid filling of the ventricle (S_6-D_2) is always deep and steep, while the ascent of the curve D_2-A_1 during the phase of diastasis is longer in connection to the slower rhythm.

It should be noted that in individual cases the electrokymogram of the left atrium of athletes shows a regurgitation wave as a result of relative insufficiency of the mitral valve as the ventricular cavity is stretched. The regurgitation wave is narrow and high, since the deficiency is not great, and the force of the contraction of the ventricle is high (see Figure 89, E).

During graphic phase analysis of electrokymograms of athletes, the same types of noncontractive movements are seen as in nonathletes. However, the increase in general and segment dimensions of the heart frequently seen in athletes results in certain specifics of the mechanical activity. It should be noted that even in cases with considerable enlargement of the heart, the supplementary movements in athletes are generally retained. This is

confirmed by the fact that with physiologically "regulative" enlargement of the heart (Reindell et al., 1960), the contractive capability of the myocardium is not decreased.

Most frequently in athletes, as in those healthy persons not involved in sports, systolic oscillation of the heart to the left occurs at the same time as dorsal rotation of its longitudinal axis. In athletes, however, this movement is more sharply expressed and lasts considerably longer; the latent time of the electrokymogram of the left ventricle is correspondingly considerably increased, particularly in the area of the apex, and also the rear wall when recorded in the second slanted position. During roentgenological examination in these cases, we can usually see enlargement of the heart due to preferential hypertrophy and tonogenic dilation of the left ventricle, causing reinforcement of the oscillations to the left in the early systole. In many cases, systolic oscillation of the heart to the right is noted, which is generally not seen in healthy persons (Heckmann, 1952). In athletes in this case, we usually see total enlargement of the heart, principally of the transverse dimension, with particular hypertrophy and dilation of the right ventricle. Among these persons, we more frequently encounter changes in the normal course of the curves and other variations in the state of the heart. /293

Frequently, with significant enlargement of the heart in athletes graphic phase analysis of ventricular electrokymograms shows a characteristic picture of intraventricular displacement of a large quantity of residual blood (Figure 92).

After physical loading (Ya. G. Ablov, 1961) in athletes (Figure 93) in most cases the amplitude of the waves increases significantly. This is particularly seen on the electrokymograms of the ventricles at the apex of the electrokymograms of the large vessels. On the latter, we also see a decrease in the "expulsion angle." These characteristics indicate more powerful contraction of the ventricles. /295

The change in the nature of the activity of the ventricles with adequate reaction to loading is reflected in the form of the electrokymogram in that the waves become sharper, the course of the ascending and descending sectors becomes steeper and straighter, the diastolic plateau disappears, and and certain details are smoothed. Oscillations of the heart retain their nature and direction, although they increase significantly in amplitude, velocity and duration. In a number of cases the electrokymograms of the ventricles show a supplementary wave of small amplitude at the end of the phase of rapid expulsion, the origin of which is similar to tone III of the heart (K. V. Chernetskiy, 1963). Differences are retained in the nature of the curves of the paths of influx and efflux of the ventricles.

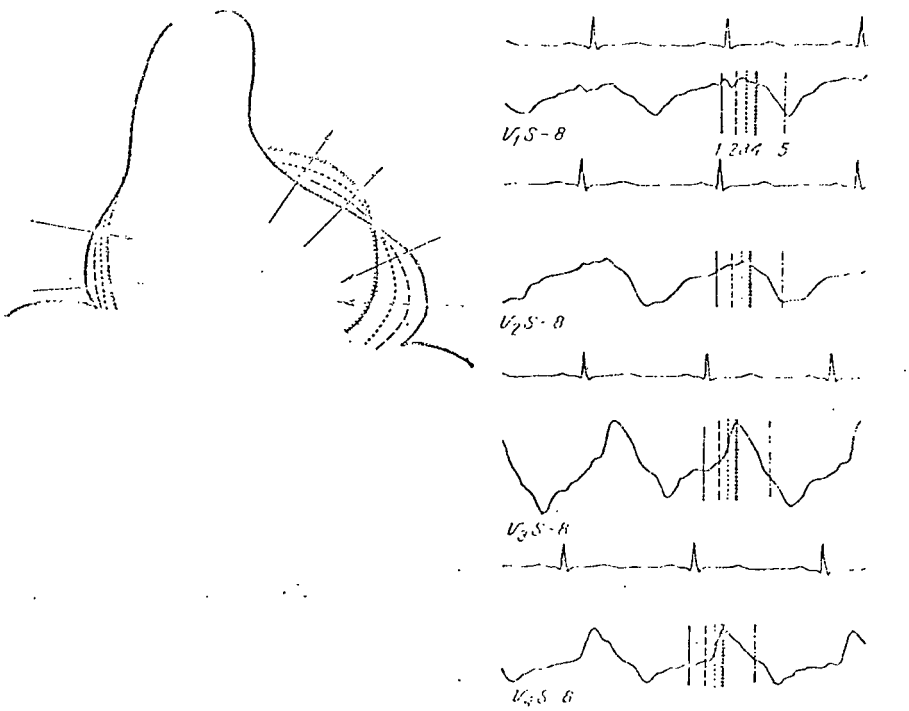

Figure 92. Intraventricular Displacement of Blood During Dilation of Heart (Master of Sports V-v)

The specifics of the electrokymograms of athletes during fatigue, overtraining or overstress have not yet been studied. We have only data (Ya. G. Ablov, 1964) on the changes in curves sometimes encountered in athletes with manifest enlargement of the heart, related to intensive training for endurance (type III "sports heart" of S. P. Letunov and R. Ye. Motylyanskaya, 1948). The deviations of electrokymograms in these cases are not sharply expressed and in general can be equated to changes of stage I, described by V. N. Orlov (1961).

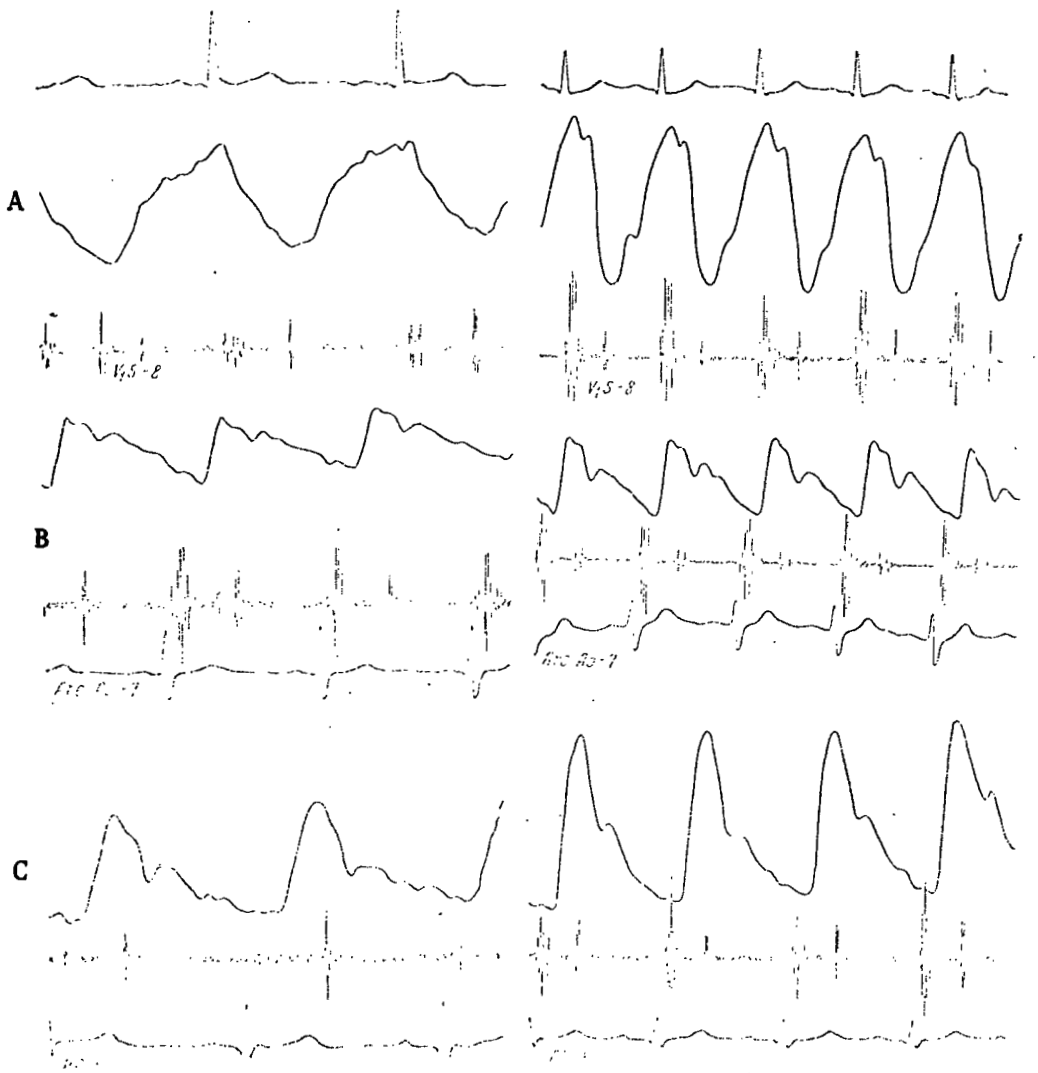

Figure 93. Electrokymograms of Athletes Before and After Physical Loading. A, Apex of heart; B, Aorta; C, pulmonary artery

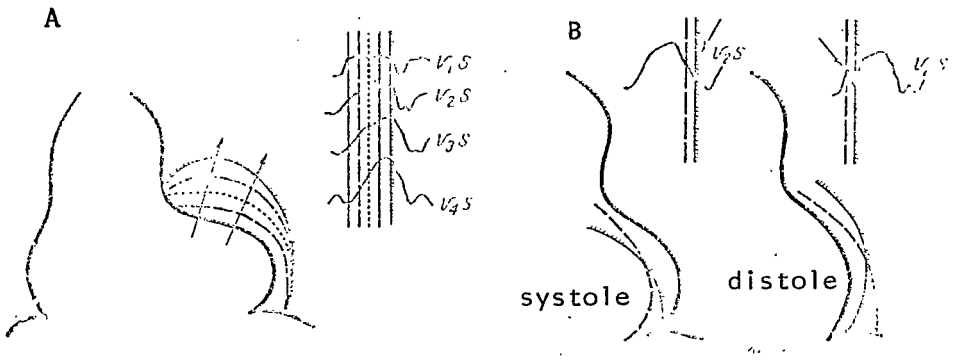

Figure 94. Change in Electrokymograms in Athletes Upon Decrease in Tonus and Dilation (A) and Weakening of Contractability of Myocardium (B)

They include: /296

1) very steep and short diastolic rise of curve with subsequent plateau and decrease in amplitude of electrokymogram at apex in comparison with base of heart. These curves reflect considerable dilation and decrease in tonus of influx path, expansion of residual blood to the extent that the increase in volume in the diastole occurs principally at the base of the heart, i.e. a picture is observed which is the reverse of a healthy heart (graphic phase analysis of this case shown in Figure 94, A);

2) later beginning of medial movement in area of apex (latent time from 0.12 to 0.16 sec). Here, we observe an extended increase in volume in the systole at the apex, which cannot be related to oscillation to the left; the reason for this is also dilation of the ventricle and insufficiency of influx path, which delays transfer of blood to efflux path, since the intraventricular pressure is not immediately overcome; /297

3) reverse movements in the systole and diastole. In graphic phase analysis, these cases (Figure 94, B) show crossing of the intermediate isophases, reflecting noncorrespondence of stress of individual sectors of the myocardium to the changing intraventricular pressure;

4) abnormally high protodiastolic wave as a result of reinforced oscillations of the heart. Figure 95, A shows a case of appearance of such a wave on the electrokymogram after physical stress.

It should be noted that these changes in the form of the electrokymogram in athletes may be observed with no other indicators of a decrease in the functional capacity of the heart according to the data of combined examination.

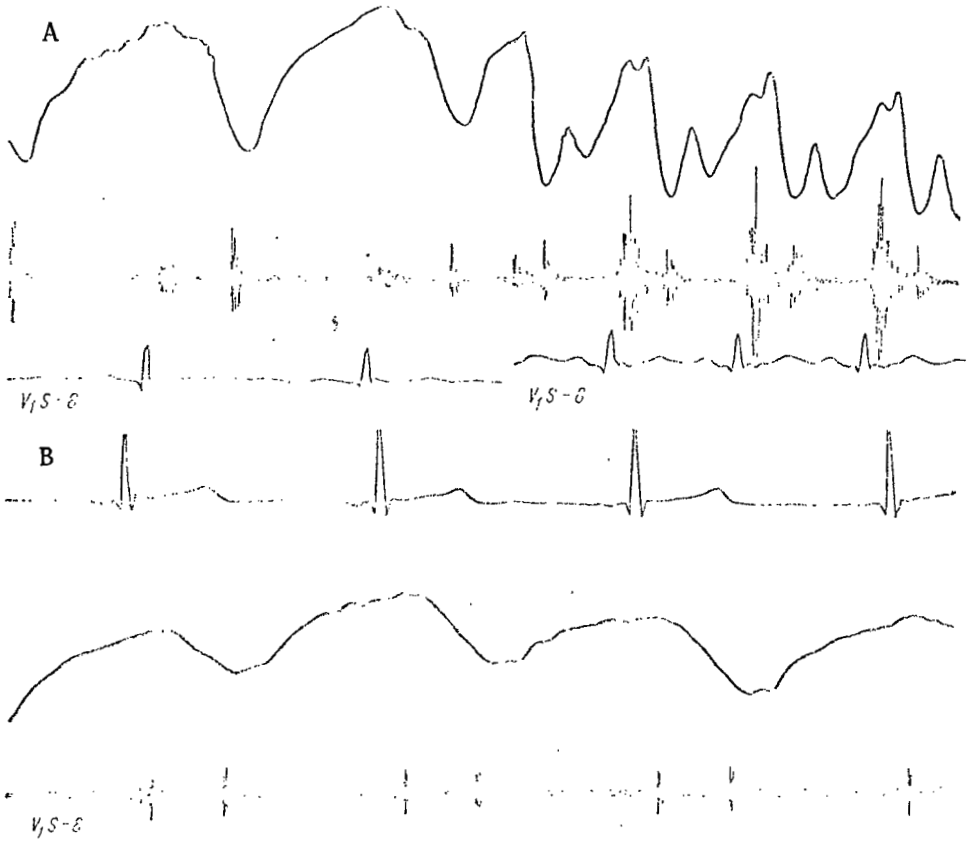

Figure 95. Change in Electrokymograms of Athletes. A, reinforced oscillations of heart; B, overtraining

With manifest fatigur or overtraining in athletes, according to some observations, the electrokymographic curves become the same throughout the entire contour of the ventricles, their form being rounded in the form of a bell, the amplitude decreased, the latent time of the electrokymogram shortened, transitions and small waves being smoothed. All of these characteristics reflect expansion of the ventricles, a decrease in their tonus and contractive capacity (Figure 95, B).

Teleroentgenography

In recent years, Nylin (1957), Musshoff (1955), Reindell (1950), Medved (1963) and others have demonstrated the importance of the investigation of the volume of the human heart. In these works, the dependence of the volume of the heart on a number of parameters has been established, and,

which is particularly important for sports medicine, this dependence on the sports specialty and state of training of the test subjects has been clearly shown.

The first attempts to determine the dimensions of the heart by roentgenological methods were made at the beginning of the twentieth century (Moritz, 1902). However, roentgenography was found promising for the determination of the volume fo the heart only after Keller in 1905 suggested teleroentgenography. This method consists of the production of pictures with a distance from tube to cassette of 2-2.5 m. With this method, it is assumed that the X rays "move in parallel", and the picture shows heart dimensions /298 which are near accurate, i.e. no projection magnification occurs.

Techniques of teleroentgenographic calculation of heart volume. Teleroentgenograms of the heart can be made with the domestic type RUM-5 X-ray apparatus. In order to determine the volume of the heart, we must take the teleroentgenograms in the frontal and sagittal planes. Before beginning to to make the pictures, we must strictly center and reliably fix the X-ray tube. The pictures are made both in the vertical and horizontal position of the test subject. The "lying" position eliminates the orthostatic influence on the filling of the heart. This position makes it possible to compare the dimensions of the heart during dynamic observations of the same person (Musshoff, Reindell, Schmidt, 1960).

After the test subject is placed on the table, the X-ray tube is aimed at the area of the heart. It has been established in practice that the best pictures are produced at the X-ray tube is pointed at the center of the chest (at the level of rib III) with a frontal pictur and at 1in. axillaris media for a sagittal picture. After the tube is thus aimed, it is moved 2 m away from the test subject. Some authors recommend that the tube be moved to 3-4 m. However, in practice, sufficiently accurate data are produced at a distance of 2 m. Moving the tube back to 2 m allows pictures to be made with lower voltage and current than if the tube is moved back to 3-4 m. This decreases the irradiation of the test subject and prevents rapid deterioration of the X-ray tube.

Method of calculation of heart volume. The heart is an irregular geometric figure, so that the determination of its volume cannot be performed easily using formulas for proper geometric bodies. However, if we introduce various correcting constants to these formulas, they do allow us to calculate the volume of the heart with a low percentage of error.

Several formulas are available for calculation of heart volume. They are all based on a definition of the frontal area of the heart using the "Moritz rectangle" on the frontal picture and the transverse "deep diameter" of the heart on the sagittal picture. The products of these parameters allow us to calculate the volume of the heart.

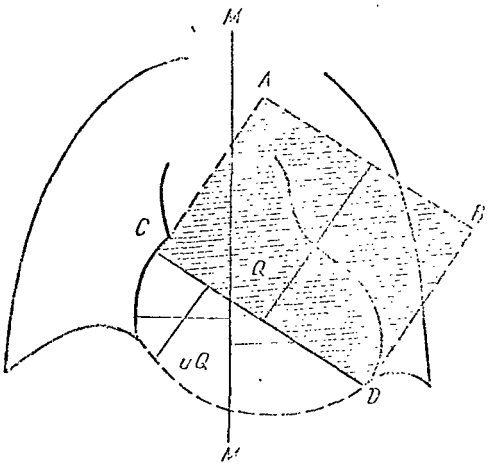

Figure 96. Diagram of Measurement of Area of Heart in Anterior Projection Using "Right Angle" Method (After V. V. Zodiyev). Long line of heart CD serves as base for rectangle CABD. Height of rectangle consists of two sectors: uQ and oQ, i.e. equal to slanted diameter of heart Q.

The "Moritz rectangle" gives us the arbitrary area of the heart. Construction of this rectangle is performed as follows (Figure 96). Using a roentgenogram, a line is drawn through the inclined diameter of the heart. In order to determine this line, the right atriobasal angle is connected by a straight line to the point of contact of the apex of the heart and the diaphragm. The inclined diameter is determined by dropping two perpendiculars from the left atriobasal angle and from the point of contact of the right /299 contour of the heart with the diaphragm to the long line through the heart. The sum of these perpendiculars is the inclined diameter of the heart. Then, two perpendiculars are once more drawn from the long line of the heart to a length equal to the inclined diameter of the heart. The ends of the perpendiculars are connected with a straight line parallel to and equal in length to the long line through the heart. A rectangle is thus produced, whose base is the long line of the heart l, and whose height is the inclined diameter of the heart -- b (on Figure 96, l corresponds to CD, b -- Q).

The area of the rectangle thus produced (S), equal to the product of the base times the height, i.e. $l \cdot b$, is taken approximately as the frontal area of the heart. Since the heart is spherical, a correcting coefficient of 0.75 is introduced, decreasing the error in calculation of the area of the heart. Thus: $S = 0.75 \cdot l \cdot b$.

Then, the "deep diameter" of the heart t is determined on the sagittal picture (see below). Thus, after determining l, b and t, we can determine the volume of the heart using the formula.

Rohrer (1916) at first included t as the absolute front to rear diameter of the heart. This diameter was the sum of two perpendiculars dropped from the furthest points of the anterior and posterior contours of the heart to its diagonal. The diagonal was the distance from the pulmonary artery to the peak of the angle formed by the thorax and the diaphragm.

Rohrer's formula is as follows:

$$V = 0{,}63l \cdot b \cdot t, \tag{57}$$

where 0.63 is a correcting coefficient, the value of which was produced as an average between 0.59 (constant for a paraboloid) and 0.66 (constant for an ellipsoid of rotation). Kahlstorf (1932) made a correction in this formula to the value of t which he represented as t_{max} (the deepest diameter of the heart). For its determination, the two furthest points on the anterior and posterior surfaces of the heart are connected on the lateral picture. The determination of t_{max}, in the opinion of the author, yields less error in the the calculation of the volume of the heart than Rohrer's formula. Subsequently, more practical use has been made of the Rohrer-Kahlstorf formula: /300

$$V = 0{,}63 l \cdot b \cdot t_{max}; \tag{58}$$

The formulas of other authors differ in their varying coefficients.

In the teleroentgenographic method, the Rohrer-Kahlstorf formula has been modified by Musshoff and Reindell (1956), who, considering the distance from the tube to the patient, i.e. 2 m, converted this formula as follows:

$$V = 0{,}4 \cdot l \cdot b \cdot t_{max}. \tag{59}$$

The value of the error in calculations using this formula, in comparison to the investigations by cardiometry, is less than 5% (Reindell et al., 1960).

Zdansky (1949) composed a special table for determination of the heart volume. In order to use this table, it is sufficient to determine the transverse diameter of the heart and the deepest diameter of the heart.

At the present time, only the diastolic volume of the heart has usually been investigated. With the development of phasing devices, it became possible to perform differential systolic-diastolic analysis of heart volumes.

The usage of phasing devices in roentgenology began rather recently (Noble, 1955; A. N. Medelyanovskiy, 1963; etc.). The principle of operation of these devices is that they connect various investigative apparatus at a predetermined phase of the cardiac cycle. This is performed using the R wave of the electrocardiogram as an arbitrary mark indicating the beginning of the cardiac cycle. In correspondence with the program used, the X-ray apparatus

is connected in relation to the position of the R wave on the electrocardiotram after a certain period of time, corresponding to various phases of the cardiac cycle. At the present time, a standard device has been developed in the USSR for these purposes -- the "Fazorentgenokardiograf-61."

In order to study the "sports heart," it is expedient to combine the usage of teleroentgenocardiography and phased roentgenocardiography. The connection of the teleroentgenocardiograph and production of pictures from 2 m distance is performed during the protodiastolic interval and after the atrial systole.

In the first case, the final systolic volume is recorded, while in the second place the final diastolic volume is recorded.

In order to determine the time for connection of the X-ray apparatus in relation to the beginning of the R wave of the electrocardiogram, polycardiograms are recorded for the test subjects (synchronous recording of electrocardiogram, phonocardiogram and sphygmogram). Based on these curves, an analysis is made in each concrete case of the phase structure of the /301
structure of the cardiac cycle and the time between the R wave on the electrocardiogram and the corresponding phases of the cardiac cycle is calculated. On the basis of these recordings, the operation of the phased roentgenocardiograph is programmed so that diplograms of the frontal and sagittal dimensions of the heart are recorded. These diplograms can be used to calculate the long line of the heart during the systole (l_s) and diastole (l_d), the transverse dimension of the heart during the systole (b_s) and diastole (b_d) on the anterior picture and the transverse diameter of the heart during the dydtole (t_s) and diastole (t_d) in the lateral picture. On the basis of these data, we can calculate the final systolic (V_s) and final diastolic (V_d) volumes of the heart using the following formulas:

$$V_s = 0{,}4 l_s \cdot b_s \cdot t_s \qquad (60)$$

$$V_d = 0{,}4 l_d \cdot b_d \cdot t_d \qquad (61)$$

An important advantage of phased roentgenocardiographic investigation is the sharp decrease in irradiation of the test subject, since the time of investigation is reduced to the minimum time for a given X-ray apparatus.

Investigation of the volume of the heart in athletes. Various types of sports place various demands on the organism. This is reflected in the volume of the hearts in athletes of various specialties. Thus, according to Reindell and Frisch (1960), as in our observations, an increase in the volume

volume of the heart occurs in types of sports based on long and intensive physical loads (Table 21).

TABLE 21. DEPENDENCE OF HEART VOLUME ON TYPE OF SPORTS (AUTHOR'S DATA)

Type of sports	Volume of heart	Relative values of heart volume	
		$Q_R\left(\frac{CM^3}{kg}\right)$	$Q_N\left(\frac{CM^3}{M^2}\right)$
Heavy athletics	846	10,2	445
Sprinting	870	12,5	470
Walking, marathon running	970	14,5	535
Bicycling (long distance races)	1030	14,2	550
Untrained men	775	11,4	430

Tr. Note: Commas indicate decimal points.

The volume of the heart depends also on the weight and height of the person. Therefore, in order to level out individual influences of these anthropometric quantities on the volume of the heart, the volume is expressed in relative quantities. The first such relative quantity we shall call the Reindell coefficient (Q_R), produced by dividing the volume of the heart (in cm^3) by the body weight (in kg). The second relative quantity is the Nylin coefficient (Q_N) -- the quotient from the division of the volume of the heart (in cm^3) by the area of the body surface (in m^2).

Musshoff et al. (1959) established the interrelationship between the blood volume per beat during physical work at high rates and the initial volume of the heart. Under these conditions, the greatest volume per beat is observed in persons with greatest initial heart volume. Similar interrelationships were noted between the volume of the heart and the blood volume per minute, recorded during work. The increase in the blood volumes per minute and per beat during work are higher, the greater the initial volume of the heart.

Individual observations of Bilger, Roskam and Reindell (1960) have shown that as a result of intensive training, the heart may be enlarged in two weeks by 150 cm^3. In the work of Medved (1964), data are presented on the enlargement of the heart during three years from 1290 to 1700 cm^3 in athletes in training. Reindell et al. (1960) indicate the reversibility of heart enlargement when training is halted. For example, one light athlete, in one

year after completion of training, showed a decrease in heart volume, while after training was begun again, heart volume increased once more.

Thus, the method of measurement of the heart volume may be an important help in sports medicine.

CHAPTER VIII

FUNCTIONAL TESTS OF THE CARDIOVASCULAR SYSTEM

ABSTRACT. Various types of cardiovascular functional tests are analyzed: 1) dynamic tests using various physical loads; 2) orthostatic tests; and 3) tests with breath held. Attempts to design functional tests specific for the type of muscular activity characteristic to a given sports activity are described. Manifestations of cardiovascular pathology in response to various functional tests are described and tabular data are presented. Problems in the administration of functional tests, including specifically problems related to time lapse between performance of functional load and measurement of cardiodynamic parameter, are discussed.

At the present time, an important or perhaps the most important place in /303
the determination of functional state of the heart in the complex methodology of medical examination of athletes is that of the various functional tests of the cardiovascular system.

Since the functional capabilities of the organism in most cases are determined to a considerable extent by the condition of the cardiovascular system, the broad utilization of functional tests of the cardiovascular system becomes understandable.

Functional tests of the cardiovascular system can be classified as follows: 1) dynamic tests using various physical loads (jumping, deep knee bends, running in place); 2) orthostatic tests (tests based on changing position of the body of the test subject); 3) tests with breath held (inhaled, exhaled).

Most functional tests known at the present time are based on the usage of standard dosed physical loads.

Tests with Physical Loads

The study of the reactions of the cardiovascular system to muscular work makes it possible to judge concerning the adaptation to physical stress and the functional capabilities not only of the circulatory apparatus, but of the organism as a whole.

The usage in the general complex of medical examination of a method with specially introduced, measured physical load also helps reveal the functional /304
interrelationships between various systems of the organism.

The functional test of the cardiovascular system using physical loads in the form of 60 jumps in 30 sec was first suggested by V. V. Gorinevskiy in 1923. This test came to be used during medical examination of physical culture students and athletes at the State Central Institute of Physical Culture. The colleagues of this institute M. D. Chirkin (1924) and D. F. Shabashov (1925) developed a method for performing this functional test and published the first generalized material on the results of its application.

Subsequently, this test was approved by the Scientific Medical Council of the National Commissariat for Public Health of the RSFSR and came to be broadly used in medical examination of physical culture students and athletes, and was called the GTsIFK functional test.

In later years, V. V. Gorinevskiy (1930), D. F. Shabashov (1930), L. G. Serkin and A. V. Ionina (1935), D. N. Korobov (1927), A. P. Yegorov (1927), M. A. Minkevich (1928), N. A. Kevdin (1925), G. K. Birzin (1935), B. P. Kushelevskiy (1933), S. P. Letunov (1937), Ye. F. Likhachevskaya (1953), Flack (1921), A. Martinet (1925), M. Burger and P. Peterson (1929), H. Reindell (1954), P. Chialley-Bert (1956) and many other investigators worked on the improvement of functional tests of the cardiovascular system and suggested a number of various tests. The principal purpose of these works was the attempt to produce the most complete representation of the working ability and state of training of the organism of the athlete.

In the literature, a large number of various functional tests of the cardiovascular system are presented, which can be classified by the nature and volume of the physical loads, their repetition and sequence.

Many authors have stated that the physical loads used in functional tests should be specific for athletes (L. G. Serkin, A. V. Ionina, 1935; Z. P. Firsov, 1938; V. Dimitriu, 1958; I. Shtefan and S. Tinku, 1958; I. Khrastek and L. Samek, 1958). Opinions have been stated that physical loading in a test should be continued until a feeling of fatigue arises (M. Ya. Ar'yev, 1927; A. A. Bakar, 1935; G. A. Smagin, 1929; etc.).

It has been considered expedient to use qualitatively different physical loads in magnitude and duration in functional tests (L. G. Serkin, A. V. Ionina, 1935; S. V. Shestakov, 1936; S. P. Letunov, 1937). Opinions have also been stated concerning the necessity of using the same physical loads, but in different dosages depending on the state of health and state of training (A. Ye. Shafranovskiy, 1948; N. I. Shvarts, 1937; A. A. Vakar, 1935; /305
V. Ya. Kaminskiy, 1928; G. A. Smagin, R. F. Tomashevskiy, 1929).

In spite of the many years experience accumulated in the usage of functional tests and the considerable number of scientific investigations performed in this area, still a number of problems of the theoretical foundation of functional tests, the selection of the most expedient physical loadings and the interpretation of the data produced, cannot be considered completely solved.

In our country, attempts have long been made to unify functional tests and come to a single evaluation and explanation of the hemodynamic changes which occur as they are performed. Analogous attempts have been undertaken on the international scale: in 1956 at the Eleventh International Congress of Sports Medicine in Luxemburg, the problem of functional tests of the cardiovascular system was specially presented for broad discussion. Due to the broad investigations performed in the USSR, which were presented by Soviet scientists in reports at the congress, a single opinion was reached in many problem areas.

The overwhelming majority of functional tests of the cardiovascular system can be divided into tests with small, moderate and large loads. The difference between the individual functional tests concerned primarily the form and duration of the physical exercise and the methods of evaluating the results of the investigation. If we exclude these differences, it is easy to see that most dynamic functional tests of the cardiovascular system include a single load. A smaller number of functional tests include two or three loads, performed in sequence.

Up to the present time, functional tests of the cardiovascular system were divided into three groups: single moment, dual moment and combined.

Single moment functional tests. This type of test includes those in which a onetime physical load is used. Among these, the most popular are the following: the GTsIFK test (60 hops in 30 sec), the Martinet test (20 deep knee bends), the N. A. Kevdin test (40 deep knee bends), the D. F. Deshin and G. I. Kotov test (3 min running in place at 180 steps per minute), the LNIIFK test (2 min running in place at 180 steps per minute), the limiting load test (running in place for 4 min at 180 steps per minute, then at fifth minute at maximum possible speed), the "step-test" (stepping up and down a two-step ladder for 1 1/2-5 min), etc. Among the single moment functional tests, the most widely used in practical work are those with 60 jumps, 20 deep knee bends, three and two minutes run.

The technique of performance of single moment tests is, briefly, as follows: the athlete sits down before the doctor and rests for 3-5 min. During this time, the doctor records the results of the physical examination performed, clarifies certain anamnesis data, and places the collar of the sphygmomanometer or tonometer on the test subject. For rapid measurement of arterial pressure after loading, a long rubber tube with a rapid connection is used, in order to connect the collar to the apparatus without losing time. This part is quite important, since usually the arterial pressure decreases after physical loading during the first one or two minutes. A particularly rapid drop is observed in well trained persons. /306

The pulse frequency is determined at rest with a 10-second interval. This system of pulse measurement is convenient and widely used in sports medicine. After stable pulse figures are produced (for example 10-10-10 or four figures with a constant difference of one beat, for example 10-11-10-11), the nature of the pulse is noted and the data are entered

onto a card. Then the arterial pressure is measured twice, after which the collar is disconnected from the apparatus, without removing it from the upper extremity.

The athlete is then asked to perform 20 deep knee bends in 30 sec or 60 hops, etc. The deep knee bends must be full, with the arms extended forward, the arms being dropped to the sides upon standing. The rate of movement is one deep knee bend each 1-1/2 sec, which is checked using a stop watch. If the test subject does not maintain the tempo, the doctor requests him to speed up or slow down. If possible, the rhythm of deep knee bends (or hops) is fixed with a metronome.

Upon completion of the physical load (deep knee bends or running) the athlete is quickly seated before the doctor. For the first 10 sec, the doctor listens to his heart and counts the number of heartbeats by auscultation. After this, the doctor connects the collar to the apparatus and measures the arterial pressure at intervals between the fifteenth and fortieth second. Then, the pulse is counted at 10-second intervals until the end of the first minute. Subsequently, the same manipulations are repeated during the second and third minutes -- for the first 10 sec the pulse is counted, then the arterial pressure is measured and the pulse is counted once more.

Sometimes, a simpler method is used: after determining the pulse and arterial pressure in the first minute, the pulse is counted at 10-second intervals continuously for the entire second minute and the first 10 sec of the third minute. If the pulse is still elevated, it continues to be counted until it returns to its initial values and only then is the next measurement of arterial pressure made. However, this method cannot be recommended, since it does not allow detailed tracing of the recovery of /307
arterial pressure and in particular makes it impossible to determine the stepped elevation in the second minute (if it occurs). For proper performance of a functional test, one must develop the skill of rapid measurement of arterial pressure. The availability in the apparatus (sphygmomanometer or tonometer) of a double rubber balloon for pumping of air greatly facilitates measurement of the pressure. By pumping the air pressure up to 50 mm Hg, then pressing on the second, soft balloon, one can rapidly increase or decrease the pressure in the apparatus to the required level. The exhaust valve need be opened in this case only in cases with a sharp drop in minimum pressure after loading (the so-called continuous tone). After each measurement, the pressure in the collar must be released by disconnecting it for several seconds from the apparatus.

The results of the functional test are defined from the data of measurement of the pulse and arterial pressure directly after loading and the time required for return of these quantities to their initial levels.

In addition to measurements of the pulse frequency and arterial pressure, the doctor must turn his attention to the external appearance and behavior of the athlete during the performance of the physical load, as well

as after it is completed. Such changes as breathlessness, considerable sweating, sharp changes in coloration of the face, trembling of the hands indicate an unsatisfactory reaction of the organism to the functional test and must influence the conclusion of the doctor.

The functional test should not be performed immediately after eating or physical exercise. On days with considerable physical loadings, the test should not be performed.

The results of the functional test are evaluated according to the following indicators: 1) quality of performance of the physical load (evaluated in arbitrary five-unit system); 2) percentage of increase in pulse frequency; 3) time required for restoration of pulse, as well as beginning of "negative phase" of pulse quieting; 4) change in systolic and diastolic pressure and pulse amplitude immediately after loading; 5) time required for recovery of blood pressure; 6) type of reaction based on curves of pulse and arterial pressure.

The reaction to physical loading in a single moment functional test (test type with 20 deep knee bends, 60 hops, etc.) is usually that the pulse increases in frequency from 10, 11 or 12 beats in 10 sec at rest to 15-20 beats after loading, i.e. increases by 50-70%. The maximum pressure of 100-120 mm Hg at rest is increased after loading to 125-150 mm Hg, while the minimum pressure decreases from 60-80 to 50-70 mm Hg. Recovery of the pulse frequency requires from 1 to 3-4 min, while the arterial pressure requires up to 3-5 min to recover. The pulse pressure rises by 15-25 mm.

Athletes in good training after the functional tests show, in addition /308
to a low increase in pulse frequency, an increase in systolic arterial pressure, a slight decrease in diastolic pressure, or perhaps no change in diastolic pressure. The reaction to the test is expressed to a greater extent in the change in pulse pressure and to a lesser extent in the pulse frequency. In untrained athletes, the reverse phenomenon is noted -- the principal change is the increase in pulse frequency. With good functional condition of the cardiovascular system, the pulse pressure increases primarily by increasing the systolic arterial pressure. This reaction, on the suggestion of B. P. Kuleshovskiy (1933), has been called the normotonic reaction, since it indicates good functional state of the cardiovascular system.

With deficient functional capacity of the heart, the single moment functional test causes a significant increase in pulse frequency, a slight increase in systolic arterial pressure (sometimes it does not change or even decreases), an increase in diastolic arterial pressure and a decrease in pulse pressure. In these cases, recovery occurs slowly, and the reaction to the functional test is called asthenic, which is frequently noted with overstress or various heart diseases.

If the pulse frequency increases significantly and the systolic arterial pressure rises sharply in the functional test (up to 180-200 mm Hg), while

the diastolic pressure remains unchanged or increases slightly, naturally, the pulse pressure increases significantly. This type of reaction to the functional test is called hypertonic. With this reaction, the time for recovery of pulse frequency and systolic arterial pressure is lengthened. If the hypertonic reaction to the functional test is observed in young athletes, the reason for this is frequently forced training with high loads.

When the functional test causes an increase in pulse frequency, a significant increase in the systolic arterial pressure (sometimes up to 200-220 mm Hg) and a decrease in diastolic pressure to zero (the continuous tone phenomenon), this reaction to the test is called dystonic. This reaction is observed most frequently in cases of overtraining, with vegetative neuroses, particularly frequently after infectious diseases. However, Ye. A. Poruchikov (1962-1966) indicated that the phenomenon of the continuous tone appears briefly when the arterial pressure is recorded immediately after completion of the load (for 15-20 sec). It should be noted that due to the brevity of the continuous tone phenomenon, when the usual method is used for performing the functional test, it is rarely observed. The appearance of /309
the continuous tone depends on the extent of the hemodynamic changes during muscular work (pulse frequency, duration of blood expulsion from the heart, power of cardiac contraction, etc.). In recent times, a number of authors have indicated that the "continuous tone" is caused by a cardiogenic mechanism. The tonus of the peripheral vessels apparently is not related to this phenomenon. These observations, as well as the data of M. N. Syvorotkin (1965), T. E. Karu (1966) and others require that previous opinions concerning the nature of the distonic reaction be re-examined.

Unsatisfactory data in the performance of a single moment functional test are observed in cases of disruption of the nervous regulation of the cardiovascular system, heart deficiency or alterations in the state of the peripheral circulatory apparatus.

However, the physical loading in 20 deep knee bends or 60 hops is relatively slight, so that in healthy persons even with insufficient training, the results of the test are frequently quite satisfactory.

Ye. F. Likhachovskaya (1953) investigated several groups of qualified athletes in various states of training (well trained, untrained or overtrained) using a combined method of medical examination using various functional tests and determined that tests with small loadings (single moment or dual moment) are insufficiently sensitive for determination of the state of training or early recognition of functional changes in the circulatory apparatus in qualified athletes.

With diseases of the cardiovascular system (residual phenomenon after myocarditis, rheumatism, after severe infectious diseases, with heart defects with deficiency of circulation, etc.), the small load test will show poor adaptability of the cardiovascular system to physical loads.

A more proper conception of the state of training is given by single moment functional tests with relatively large physical loads. These tests less frequently show a difference in the reaction of the organism to loads as a function of the state of training. In sports medicine in the examination of sportsmen of third rank, or sometimes of second rank, functional tests of the cardiovascular system with physical loading consisting of two or three minutes running in place are frequently used. These functional tests are performed according to the following plan. First, an ordinary examination at rest, then, without removing the collar of the tonometer, the athlete runs in place to the metronome at 180 steps per minute. The test subject raises the knees to hip level, and moves the arms as in ordinary running.

For juveniles, adolescents, girls and women the loading most frequently /310 suggested is two minutes running in place.

Investigations to set a basis for the functional test with two minutes running in place were performed by G. K. Birzin (1958). The data of investigation of adult athletes of 20-25 years of age during the main training period (184 men and 179 women) with normal arterial pressure were studied. G. K. Birzin came to the conclusion that the functional test with a physical load consisting of two minutes running was quite acceptable for examination of adult athletes and young people of either sex. In addition to considering the changes in hemodynamic indicators (occurring under the influence of the functional test), the author recommended that data from complete medical examination, the performance of the running, the nature of the breathing during the load and in the recovery period, the change in coloration of the skin, changes in the auscultative picture of the heart, appearance of feelings of weakness or dizziness be taken into consideration to evaluate the functional state of the circulatory apparatus.

The appearance of breathlessness during performance of the load or in the recovery period is considered to be an unfavorable symptom, indicating compensation of cardiovascular activity by reinforcement of the respiratory function. Cases of breathlessness are more frequent in middle and advanced age. These moments should be taken into consideration in the performance of any functional test.

Functional tests with three minutes running are widely used in sports medicine. These tests are used in the selection of persons for sports youth schools, for admission to competition, in the examination of persons sent to mountain climbing camps and mountain tourist hikers.

The functional test with three minutes running is also used when the doctor, on the basis of objective examination, is doubtful as to the good function of the cardiovascular system, or if the test subject has complaints which indicate disease or disorder of the cardiovascular system, when there is no objective medical data indicating such a problem.

After a test in the form of three minutes running, athletes show a rather clearly expressed reaction to the pulse and arterial pressure.

In athletes in various functional conditions, the degree of change of pulse and arterial pressure differs after the test. Thus, undertrained and overtrained athletes show a greater increase in pulse frequency than athletes in a good state of training, while the restoration of the pulse after the test occurs more slowly, the recovery time generally being longest /311
for overtrained athletes.

The decrease in diastolic arterial pressure in overtrained and undertrained athletes is generally considerably more clearly expressed than in well trained athletes.

The change in systolic arterial pressure in overtrained, undertrained and well trained athletes has no clearly expressed distinctions.

Clear differences are observed in the duration of the recovery period, and particularly the time required for restoration of the diastolic pressure. Thus, in well trained athletes the recovery of all indicators occurs quite rapidly, the diastolic pressure recovering first, followed by the systolic pressure and the pulse.

In undertrained athletes, the recovery occurs relatively slowly. However, the systolic arterial pressure is restored more rapidly than the pulse and diastolic pressure. In athletes in the overtrained state, the systolic arterial pressure also recovers relatively more rapidly than the pulse and diastolic arterial pressure. As concerns types of reaction, regardless of the state of training most (about 80%) show the normotonic reaction. Even in the overtrained state, the normotonic type was observed in 46% of cases.

In trained runners specializing in long distances, skiers, swimmers, football players and others, in most cases the normotonic reaction to the functional test with three minutes running is observed. The pulse after running with initial data of 8-12 beats per 10 sec increased by 6-10 beats (40-100%). The systolic arterial pressure, with an initial level of 90-115 mm Hg was increased by 15-30 mm Hg (15-30%), the diastolic pressure, with an initial level of 45-75 mm Hg was decreased by 5-25 mm Hg (10-35%) or remained unchanged. The recovery period of the pulse averaged 2 1/2 min (from 50 sec to 5 min).

The reaction to the three-minute test in the same athlete changes as a function of the state of training. For example, a long-distance runner after a forced one-month interruption in training showed a clearer reaction to three minutes running. With equal initial values of the pulse, after the test the frequency increased by seven beats per 10 sec (70%), while the maximum pressure was increased by 18 mm. The recovery period of the pulse was lengthened from 50 sec to 2 min and 10 sec.

Persons not involved in physical culture and sports but in good health showed an increase in the pulse to 150-160 beats per minute after the /312
functional test with three minutes running, while the systolic arterial

pressure increased to 140-160 mm Hg, the diastolic pressure dropped by 10-20 mm. These persons showed pulse frequencies still 12-24 beats per minute high after three minutes recovery. The systolic pressure also had not returned to its initial level after three minutes recovery. The pulse and arterial pressure in untrained but healthy persons after running returned to the initial levels after 6-7 min following completion of the functional test.

In cases when the pulse remains high for ten minutes and more, while the systolic arterial pressure drops below the initial level, there is a disruption in the functional state of the cardiovascular system. These same phenomena are noted in athletes in cases of chronic overstress.

It should be noted that when the functional test is performed with three minutes running, and also during other tests involving physical loading (primarily for endurance), it is sometimes noted that the pulse frequency becomes less than the initial level at some point in the recovery period. Thus at the second to third minute after physical loading, the pulse often falls to below the initial level. Then, the pulse frequency increases again and the cardiac rhythm gradually normalizes. The phenomenon of decreased pulse frequency in the middle of the recovery period (by 1-3 beats per 10 sec) is called the "negative pulse phase."

The mechanism of formation of the "negative phase" of the pulse is not yet quite clear. This problem has been the subject of a special investigation (S. Ye. Tseytlovskiy, 1964).

Persons who show the "negative pulse phase" continuing for more than three minutes after the functional test of the cardiovascular system should be subjected to more detailed examination.

The functional test with five minutes running at varying speeds (four minutes at 180 steps per minute, the fifth minute as fast as possible) is much less frequently used, although this test quite clearly shows the reaction of the organism to physical stress depending on its functional state. When this test is performed, considerable changes in the indicators of pulse and arterial pressure are noted. If we compare the data of the functional test for well trained and untrained athletes, the greatest changes are noted in the variation in diastolic pressure, resulting in an increase in the pulse pressure. At the same time, these groups of athletes show changes in pulse and systolic arterial pressure, increased over the initial values, which do not differ too greatly from each other.

The greatest difference in the data of the functional test is observed during the recovery period. Thus, for athletes in a good state of training, /313
the recovery of pulse and arterial pressure does not require more than five minutes, the diastolic pressure recovering first, followed by systolic pressure and pulse. In insufficiently trained athletes, the recovery occurs more slowly. The diastolic and systolic pressures recover almost in parallel with some lagging of the diastolic pressure. The recovery of the pulse occurs after the arterial pressure.

In overtrained athletes, the response to the five-minute load is a significant increase in pulse, increase in changes in diastolic pressure and considerably less increase in systolic pressure. The recovery period may extend to over ten minutes.

Dual moment tests of the cardiovascular system. In these tests, the physical load is performed twice with a time interval between. Examples of dual moment tests are those of D. N. Korobov (1927), L. G. Serkin and A. V. Ionina (1935), Martinet (1925), etc. In the dual moment test of D. N. Korobov, the test subject performs the same load twice -- 60 jumps in 30 sec with a rest period of four minutes. The doctor determines the change in pulse and arterial pressure. He compares the data. Each load in the test is evaluated using the same indicators and reactions, as in the single moment test. Good functional capacity of the cardiovascular system is indicated by a normotonic reaction to each load. If the functional capacity of the circulatory apparatus is disrupted, after the first physical load the normotonic reaction may occur, but after the second load an atypical reaction will be seen. With manifest functional deficiency, the atypical reaction will occur even after the first load.

The dual moment test of L. G. Serkin and A. V. Ionina calls for the usage of differentiated physical loads for strength or speed or for endurance, i.e. the qualities which are characteristic for a given type of sports. For example, a sprinter would be required to run in place twice (with three minutes between) for 15 sec at the maximum possible speed. The pulse and arterial pressure are studied at rest, between loads for three minutes and during the ten-minute recovery period. In addition to testing the pulse and arterial pressure, the external appearance of the athlete during performance of the loading, as well as the quality of performance are considered.

Weight lifters and boxers are asked to perform a strength test, including lifting weights weighing 32 kg from the floor to the height of the chin the number of times equal to the body weight of the athlete divided by four, for example: if the athlete weighs 68 kg, he must lift the weight 17 times at one cycle each 1-1-1/2 sec. The second load, like the first load, is performed after five minutes. The pulse and arterial pressure are /314
determined at rest, in the five-minute interval between loads and for ten minutes recovery period.

Another dual moment test is the Pashon-Martin test, which includes the orthostatic test and 20 deep knee bends in 40 sec.

The investigations of Ye. V. Likhachovskaya (1953) have shown that dual moment tests with small loadings have no essential advantages over single moment tests with the same loading. In athletes in a good state of training subjected to the dual moment tests, the pulse and arterial pressure increase from one load to the next, and the pulse pressure also rises.

In analyzing functional tests, we should consider the type of pulse and arterial pressure reaction to the physical load and the time required for

recovery. In persons with functional deficiency of the circulatory system, as well as insufficiently physically trained persons, fatigued persons, those with vegetative nervous system disorders, the functional tests show a greater increase in pulse frequency, slight increase in systolic arterial pressure, or perhaps even a decrease. The diastolic arterial pressure more frequently increases, the pulse frequency decreases. This reaction of pulse and arterial pressure is referred to as asthenic. These reactions are seen with insufficient adaptation of the circulatory apparatus and, in particular, in case of overstress.

Three-moment functional tests. Medical examination of highly qualified athletes may be performed using three-moment combined functional tests (S. P. Letunov, 1937). The value of this test is that it can be used to study the influence of loads for speed and endurance on the organism. It is these types of physical loads (speed and endurance) which are widely used in the overwhelming majority of types of sports with today's training systems.

The combined three-moment functional test of the cardiovascular system is performed as follows.

The first part of the test -- 20 deep knee bends -- is a warm-up or introductory exercise. It was described above. After this load, the pulse and arterial pressure are measured for three minutes. The second portion of the test consists of a high-speed load, consisting of 15 seconds running in place as fast as possible. After this load, the pulse and arterial pressure are measured for four minutes. The order of investigation is as follows: for the first ten seconds of each minute, the pulse is measured, then the arterial pressure is measured. The last portion of the test consists of a three minutes run in place. This is the endurance load. The run is performed with the knees raised high and the arms are swung energetically. The speed is 180 steps per minute, which is controlled by a metronome. For women and young people, a two-minute run is recommended. After the loading, the pulse and arterial pressure are measured for five minutes. /315

The physical loads in the combined three-moment functional test differ in their effect on the organism and subject the cardiovascular system to varying demands. Thus, the first phase is a preparation for the organism to the main physical loads. The second part -- the 15-sec run -- defines the capability of the organism for sharply increasing blood circulation, which is an indicator of the adaptability of the organism to high-speed loads. The third portion -- the three-minute run -- helps to determine the capacity of the organism for increased circulation over a relatively long period, which determines the capability of the organism to work for endurance.

In analyzing the data of the three-minute functional test, the height of the reaction, degree of matching of the change in pulse and height of arterial pressure and the pulse amplitude, the sequence of changes of all indicators from one physical load to another, the time of restoration of the pulse and arterial pressure to their initial values are all taken into consideration. If the principle of variation is sufficiently adapted to the

training of athletes for various types of sports, good adaptability to all loads in the test will be determined when the cardiovascular system is investigated using this test, although some slight differences will be seen, /318 depending on the specifics of the various types of sports.

TABLE 22. NORMOTONIC TYPE OF REACTION TO THREE-MOMENT COMBINED TEST

At rest	Investigation time, sec.	After 20 deep knee bends			After 15-sec run				After 3-min run				
		1st	2nd	3rd	1st	2nd	3rd	4th	1st	2nd	3rd	4th	5th
		Min			Min				Min				
Pulse 9 9 9	10	16	9	9	22	11	9	9	20	13	10	9	9
	20												
	30												
	40												
	50												
	60	10	9	9	12	10	9	9	14	10	9	9	9
Arterial pressure, mm Hg	110	120	110	110	155	140	130	110	130	140	130	115	110
	70	60	65	70	55	60	65	70	50	60	65	70	70

For well trained athletes, the following test indicators are characteristic (Table 22). The reaction to physical loads in the functional test (see Table 22) have come to be called normotonic. This term was first suggested, as we indicated above, to evaluate single moment tests, but then became more broadly used to evaluate the combined functional test. With the normotonic type of reaction, matching and parallelism are noted in the changes of pulse and arterial pressure, a considerable increase in pulse frequency occurring simultaneously with a rather clearly expressed increase in pulse amplitude resulting from an increase in systolic and a moderate decrease in diastolic arterial pressure. Recovery occurs relatively rapidly. The reaction of the pulse and arterial pressure increased from one load to the next. With proper adaptability of the organism to loads, but insufficient training of the athlete, the normotonic type of reaction is observed, but recovery occurs more slowly.

The normotonic type of reaction to the test is looked upon as favorable, but distinctions are made depending on the age and physical training, which can be seen in the changes of the data on pulse, arterial pressure and recovery time. In Table 23 (after S. P. Letunov) data are presented indicating that the greater the physical load in the functional test, the more the changes in pulse and maximum arterial pressure are seen. In addition to the normotonic reaction, we distinguish dystonic, hypertonic, hypotonic reactions, and also the reaction with stepped increase in systolic pressure. All

of these reactions indicate functional deficiencies of the circulatory apparatus to some degree or another.

The dystonic reaction is distinguished by the high values of the changes in systolic (over 180 mm Hg) and diastolic (down to zero -- the "continuous tone" phenomenon) are to real pressure, and the recovery occurs slowly. The pulse frequency increases considerably and returns slowly (Table 24).

The dystonic type of reaction is most frequently observed in young persons and adolescents, although after 21-22 years of age the dystonic type of reaction is frequently replaced by a normotonic reaction.

The "continuous tone," or appearance of "zero" diastolic pressure after the completion of the physical load is an expression of great lability of vascular tone. The "continuous tone" is frequently heard after extended exercises for endurance, for example after a marathon run it is noted in the majority of cases. There is an opinion that the "continuous tone" is a physiological indication of adaptation of the organism to physical loads and /319
indicates high functional capacity of the cardiovascular system. This opinion primarily concerns physical exercises of relatively high intensity. In cases when the "continuous tone" is heard only directly after loads and is not combined with other changes in the reaction of the organism to the functional test, this phenomenon should not be considered a negative change. However, many authors improperly believe this reaction to be a reflection of good functional state of the circulatory apparatus in all cases.

If the "continuous tone" appears after the physical load in the functional test (not noted before) and is heard for 2-3 min or more, combined with other unfavorable indications in the functional test, all of this as a whole indicates a change in the normal nature of the recovery processes. The fact that the "continuous tone" may appear with functional disorders in the state of good training obligates the doctor to evaluate the data of the tests on a strictly individual basis, particularly in the case of the dystonic reaction.

In the reaction with the stepped increase in systolic arterial pressure immediately after the load, the maximum arterial pressure is lower than at the second and third minutes of the recovery period (Table 25).

This reaction is most quickly seen after the high-speed portion of the /320
test, it being for many people the earliest symptom of a decrease in the functional state of the cardiovascular system. In beginning athletes, this reaction indicates insufficient adaptability of the cardiovascular system to high-speed loads. In certain athletes, this reaction is seen in all examinations, over a long period of observation, which apparently is an individual specific and indicates insufficient adaptability of the organism to high-speed loads or the specifics of the training methods used. As the state of training improves, the stepped reaction is usually replaced by the normotonic reaction. Still, after the high-speed load the stepped increase may in certain cases be retained in well trained athletes.

TABLE 23. CHANGE IN PULSE FREQUENCY AND MAXIMUM PRESSURE WITH

Age, years	Number of observations	Pulse (in 10 sec)							
		Initial data		After 20 deep knee bends in first min		After 15-sec run in first min		After 3-min run in first min	
		M ± m (M)	σ	M ± m (M)	σ	M ± m (M)	σ	M ± m (M)	σ
11	52	13,14 ±0,21	1,70	19,41 ±0,31	2,24	23,45 ±0,37	2,60	24,01 ±0,45	3,50
12	107	12,67 ±0,17	1,80	17,73 ±0,12	1,16	23,05 ±0,26	2,60	23,18 ±0,34	3,5
13	103	12,53 ±0,15	1,50	19,91 ±0,21	2,5	23,60 ±0,[illegible]	2,7	23,95 ±0,31	3,1
14	100	12,0 ±0,14	1,35	19,01 ±0,20	2,05	23,5[illegible] ±0,21	2,36	23,70 ±0,31	3,14
15	100	11,66 ±0,17	1,68	18,9[illegible] ±0,[illegible]	2,03	23,[illegible] ±0,27	2,75	25,[illegible] ±0,45	4,51
16	100	11,18 ±0,14	1,41	18,11 ±0,2[illegible]	2,28	22,57 ±0,31	3,15	24,79 ±0,35	3,50
17	94	10,[illegible] ±0,14	1,38	18,22 ±0,20	2,05	22,17 ±0,27	2,61	22,65 ±0,32	3,08
18	102	11,13 ±0,14	1,45	16,8 ±0,18	1,83	22,08 ±0,27	2,77	23,21 ±0,34	3,50
19	105	10,84 ±0,11	1,45	15,[illegible] ±0,22	2,19	22,52 ±0,22	2,23	23,70 ±0,[illegible]	3,03
20	100	10,43 ±0,17	1,67	16,80 ±0,13	1,83	21,13 ±0,25	2,53	21,95 ±0,35	3,52
21–25	108	10,65 ±0,03	1,41	16,43 ±0,05	2,65	21,19 ±0,07	2,72	21,50 ±0,13	3,44
26–30	101	9,35 ±0,14	1,36	15,[illegible] ±0,20	1,95	20,31 ±0,27	2,76	21,07 ±0,[illegible]	3,24
31–35	121	9,39 ±0,03	1,16	16,[illegible] ±0,05	1,99	20,63 ±0,06	2,06	21,80 ±0,08	3,03
36–40	103	10,42 ±0,12	1,26	16,3[illegible] ±0,21	2,13	20,72 ±0,26	2,62	21,51 ±0,31	3,39
41–45	85	9,67 ±0,15	1,35	16,33 ±0,25	2,27	20,30 ±0,27	2,43	22,10 ±0,[illegible]	2,05
46–50	55	9,97 ±0,23	1,62	16,2[illegible] ±0,23	1,61	20,43 ±0,31	2,20	21,77 ±0,3[illegible]	2,77
50 and more	50	10,9[illegible] ±0,20	1,38	16,98 ±0,34	2,35	20,73 ±0,32	2,34	22,61 ±0,43	3,02

Note. M, arithmetic mean; m, mean square error; σ, mean square deviation
Tr. Note: Commas indicate decimal points

COMBINED TEST FOR SPEED AND ENDURANCE (AFTER S. P. LETUNOV)

Maximum arterial pressure							
Initial data		After 20 deep knee bends in first min		After 15-sec run in first min		After 3-min run in first min	
$M \pm m$ (M)	σ	$M \pm m$ (M)	σ	$M \pm m$ (M)	σ	$M \pm m$ (M)	σ
90,45 ±1,25	9,10	103,40 ±1,71	12,4	116,70 ±1,59	13,70	123,87 ±2,16	15,8
93,20 ±0,87	9,10	115,10 ±1,38	14,2	115,5 ±1,17	12,1	132,05 ±1,58	16,1
91,55 ±0,75	7,8	111,10 ±1,12	11,7	115,45 ±1,20	12,6	126,55 ±1,39	13,8
103,0 ±1,08	10,8	128,25 ±1,31	13,8	138,85 ±1,28	13,75	143,1 ±1,66	16,05
109,0 ±1,07	10,86	125,89 ±1,37	13,60	129,25 ±1,63	16,30	149,69 ±1,73	17,85
111,05 ±1,69	10,9	132,60 ±1,75	13,75	151,80 ±1,75	17,45	173,4 ±1,93	19,32
110,95 ±1,05	10,1	135,70 ±1,75	16,85	141,75 ±1,59	15,90	159,37 ±1,84	16,50
112,9 ±1,62		143,83 ±1,42	14,19	141,56 ±1,70	15,10	165,78 ±1,53	15,85
115,95 ±1,19	11,25	161,25 ±1,57	15,55	159,10 ±1,78	17,80	178,58 ±1,77	17,39
115,75 ±1,0	9,59	156,80 ±1,56	15,19	148,60 ±1,61	16,40	175,8 ±1,55	15,9
115,87 ±0,80	11,9	131,90 ±0,88	14,65	141,75 ±0,48	17,45	177,3 ±2,15	15,25
113,55 ±1,17	10,7	139,99 ±1,62	14,50	140,05 ±2,03	19,25	155,5 ±2,57	14,05
117,45 ±0,91	11,8	153,45 ±0,14	15,30	150,5 ±0,69	21,1	175,71 ±2,5	15,05
117,89 ±1,06	10,11	155,99 ±1,45	15,0	146,19 ±1,69	17,05	162,1 ±1,5	15,35
115,95 ±1,05	9,35	134,50 ±1,69	15,29	141,80 ±1,70	13,45	162,55 ±2,14	14,5
120,1 ±2,38	16,8	136,55 ±2,39	17,80	157,75 ±5,04	20,80	155,75 ±4,3	21,7
132,0 ±3,24	22,9	153,30 ±3,59	25,40	161,40 ±3,90	27,60	182,9 ±3,70	24,7

TABLE 24. DYSTONIC TYPE OF REACTION TO THREE-MINUTE COMBINED TEST

At rest	Time of investigation, sec	After 20 deep knee bends			After 15 sec run				After 3 min run				
		1st	2nd	3rd	1st	2nd	3rd	4th	1st	2nd	3rd	4th	5th
		Min			Min				Min				
Pulse in 10 sec	10	18	12	12	28	16	15	15	30	18	17	15	15
	20												
	30												
	50												
10 9 12	60	13	12	12	18	16	15	15	20	18	16	16	15
Arterial pressure, mm Hg	120	130	130	125	200	190	160	155	210	205	195	150	170
	75	40	55	60	0	0	0	60	0	0	0	60	70

TABLE 25. REACTION TO THREE-MOMENT TEST WITH STEPPED INCREASE IN MAXIMUM ARTERIAL PRESSURE

At rest	Time of investigation, sec	After 20 deep knee bends			After 15 sec run				After 3 min run				
		1st	2nd	3rd	1st	2nd	3rd	4th	1st	2nd	3rd	4th	5th
		Min			Min				Min				
Pulse in 10 sec	10	15	9	9	25	17	14	12	25	18	12	11	10
	30												
9 9 9	50												
	60	10	10	9	18	15	13	12	19	13	11	11	10
Arterial pressure, mm Hg	115	130	120	115	130	155	140	120	150	115	135	120	115
	70	50	65	70	55	60	65	70	60	65	70	70	75

We have observed athletes for whom the stepped increase in systolic pressure was observed for months and years and written off as a manifestation of their individual characteristics. However, observations have shown that even these athletes, when careful individualization of training is used, can replace this reaction with the normotonic reaction. At the same time as the change in the type of reaction, the sports results increase. All of this indicates that the stepped type of reaction cannot be looked upon as a variant of the norm.

The stepped increase in arterial pressure after the 15-second run sometimes observed in trained athletes apparently depends on the fact that the athlete must perform intensive physical loads in the doctor's office without the corresponding warm-up, which should be considered in evaluating the state of the athlete. /321

In athletes in the state of fatigue, overtraining or overstress, the stepped increase in arterial pressure is observed not only after the 15-second run, but also after the three-minute run.

The hypertonic type of reaction is characterized by a sharp increase in maximum arterial pressure (180-220 mm Hg) and a significant increase in pulse frequency. The minimum pressure does not change or increases slightly. The recovery is usually slow (Table 26).

TABLE 26. HYPERTONIC TYPE OF REACTION TO THREE-MOMENT FUNCTIONAL TEST

At rest	Time of investigation, sec	After 20 deep knee bends			After 15 sec run				After 3 min run				
		1st Min	2nd Min	3rd Min	1st Min	2nd Min	3rd Min	4th Min	1st Min	2nd Min	3rd Min	4th Min	5th Min
Pulse in 10 sec 10, 11, 11, 10	10 20 30 50 50 60	20 17	16 13	13 13	19 22	21 20	18 17	16 16	30 23	22 20	19 18	18 18	17 17
Arterial pressure, mm Hg	120 75	150 75	140 75	140 75	205 85	190 95	170 95	160 95	190 90	185 95	175 95	170 100	170 95

The hypertonic type of reaction is most frequently observed in older athletes, in persons suffering from hypertension, atherosclerosis, or in cases of fatigue or overtraining.

If the hypertonic reaction is not accompanied by any other changes, training may be continued, but with limitations on the load and with repeated medical examination.

The hypotonic type of reaction is characterized by a considerable increase in pulse frequency with a slight increase in systolic pressure, which sometimes does not increase, or even decreases. The minimum pressure does not change, or sometimes increases. The recovery period is long (Table 27). The hypotonic type of reaction to the test is observed when the functional capacity of the cardiovascular system is decreased, frequently after infectious diseases. /322

TABLE 27. HYPOTONIC TYPE OF REACTION TO THREE-MOMENT COMBINED TEST

At rest	Time of investigation, sec	After 20 deep knee bends			After 15 sec run				After 3 min run				
		1st Min	2nd Min	3rd Min	1st Min	2nd Min	3rd Min	4th Min	1st Min	2nd Min	3rd Min	4th Min	5th Min
Pulse in 10 sec	10 20 30 40 50	18	11	10	23	13	10	40	24	15	13	12	10
10, 9, 10	60	11	10	10	13	10	10	10	16	13	12	10	10
Arterial pressure, mm Hg	105 60	105 60	100 60	105 60	110 65	105 60	105 65	95 60	115 60	115 60	110 70	105 65	95 60

The three-moment combined functional test reflects the state of training and working ability of the athlete relatively better than the other tests.

The reactions to the physical loads of the functional tests depend to a great extent on the individual specifics of the organism of the athletes. From this, the proper evaluation of the test data with the purpose of determining the state of training is possible only by dynamic observations of the athletes. At the same time, the functional changes in the cardiovascular system, as well as characteristics of fatigue, overtraining and overstress in most athletes will be seen with one-time medical examination using measured loads. The results of a functional test can be used to determine the general

state of training of athletes and in particular the state of training for speed or endurance.

Our data, summarizing the results of medical investigation and pedagogic observations, in most cases (95.3%) showed good adaptability of the cardiovascular system to the physical loads in the functional test and at the same time good working ability of the athlete under conditions of training and competition and high sports-technical results. At the same time in 4.7% of /323
the cases we did not observe complete correspondence between the data of medical examination characterizing the state of training and the sports results. The problem is that the sports-technical results of the athlete, in addition to his physical condition, are determined by the tactical conditions of competition, the technical training of the athlete, his qualities of morale and will, the conditions of performance of the competition and other factors. Therefore, it is not always justified to consider that a good functional state based on the data of the functional test allows an athlete to achieve high competitive results.

N. D. Grayevskaya et al. (1960) showed that the data from investigation of the cardiovascular system using the functional test, like other data of medical examination, are more labile during the initial period of training in each season, while the best indicators of the functional test are produced in the state of good training. For the same athlete in a good state of training but in different years, constancy of the indicators of the functional test will be noted. In the state of insufficient training, considerable fluctuations of these values will be noted for the same athlete.

The data of investigation of the cardiovascular systems of athletes using the functional test, indicating a change in the state of training of the athlete, in most cases agree with other data from medical investigation characterizing improvement of the state of training. These data include functional indicators of physical development, the pulse frequency at rest, a change in the motor reaction time, change in feeling, etc.

In various states of training, the reaction of the cardiovascular system to physical loads, the functional test for speed and endurance differ and depend on the state of training. As the state of training increases, the reaction of the pulse and arterial pressure become more economical, and the recovery period is accelerated.

The changes in pulse and blood pressure after the functional test depend on the specific features of the nervous system of the test subject, the time and conditions of examination, the degree of the preceding load and training, the activity of the athlete during the performance of the physical load of the test itself and many other factors, which should be considered during the performance of examinations and evaluation of the results of the test.

Functional tests are of important diagnostic significance in determination of fatigue, overstress and overtraining. A change in the normal /324
adaptability of the circulatory apparatus to physical loads is one of the

principal criteria for these states. Changes in the reaction to physical loading during overstress and overtraining may be identical, although in the overstressed state we more frequently see the asthenic (or hypotonic) reaction, while in the state of overtraining we generally see the stepped and hypertonic reactions.

As we know, three stages are observed in the development of the state of overtraining, each of which is characterized by a number of symptoms and certain indicators in various methods of investigation (see Chapter XIII). Among the latter, the functional tests of the cardiovascular system have an important position and provide the doctor with rather precise data. Thus, in the first stage of overtraining when the physical loading is applied, worsening of the adaptability of the circulatory apparatus to high-speed loads is noted. In the second stage of overtraining, worsening of the adaptability not only to high-speed loads, but to endurance loads is also noted. In the third stage of overtraining, unsatisfactory "stepped" reactions are noted in all three parts of the functional test.

Frequently, the "stepped" type of reaction is the earliest indication of the onset of overtraining, since, in addition to this, no other deviations are noted. In each case, the "stepped" type of reaction requires careful analysis of its causes. Sometimes this reaction results, as we stated above, not from phenomena of fatigue, but from insufficient adaptability of the organism to high-speed work as a result of individual specifics or insufficient training for fast work.

An evaluation of this phenomenon is much easier with dynamic observations. Thus, if the "stepped" type of reaction appears in the process of intensive training, this is a clear sign of fatigue; a temporary decrease in the load, particularly as concerns high-speed work, is required.

The results of medical examinations indicating a worsening of the functional state frequently correspond to the data obtained from trainers. However, sometimes the doctor sees earlier signs of a worsening of the state which are found only considerably later in a decrease in the working ability and worsening of the sports results. This in most cases relates to fatigue and to the early stages of overtraining. It should be noted that the functional tests of the cardiovascular system are also one of the most important criteria for recovery of the organism of athletes after overstress, overtraining or fatigue.

Specific loads in the functional test. In most functional tests used in /325
sports medicine, the standard load is not related to the type of sports. Some doctors use specific physical loads related to the type of sports involved in performing the functional tests -- so-called specific tests. It is assumed that the nature of the response reaction to the physical load will reflect the specifics of the action on the cardiovascular system of the given type of sports. Up to the present time, there is no single opinion on this question, and assumptions are stated periodically concerning the performance of

differentiated functional tests considering the specifics of a type of sports in the examination of qualified athletes.

A number of investigations have been performed on the question of the adaptive reaction of the organism to specific and nonspecific loads (N. D. Grayevskaya, Ye. V. Kukolevskaya, V. N. Kuz'mina, V. A. Sergeyev, Ye. F. Likhachovskaya). Thus, N. D. Grayevskaya, G. A. Goncharova and V. V. Matov (1960) investigated football players, boxers and bicyclists, who were requested to perform three types of high-speed loads: running in place, work with the high-speed bag and the stationary bicycle. The duration of each type of work was 15 sec with an interval between loadings of of 20 min. It was considered that the specific high-speed loading for the football players was the fast run, while the bag work and bicycling were non-specific. For the boxers, the specific load was the bag, for the bicyclists -- the bicycle, while the remaining physical loads were non-specific. In addition to the combined medical investigation, the pulse was measured, the arterial pressure was measured, the oxyhemogram, pneumogram, electrocardiogram (before and after loading) were all taken. In these investigations, the principal indicators of the process of adaptability of the organism to physical loads were determined -- ability to warm up, reaction and recovery.

As a result of the investigations, it was determined that in most cases (90% of the test subjects), the same types of reactions were detected for all forms of loads, both specific and nonspecific.

An investigation was performed in this same area by Ye. F. Likhachovskaya for runners and boxers using the method of mechanocardiography. The athletes performed rapid running in place at maximum possible rate for 15 sec and worked with the light bag (also at the maximum possible rate for 15 sec). The systolic volume and blood volume per minute, the values of minimum, mean, lateral and final systolic pressure, the peripheral resistance, rate of propagation of the pulse wave through elastic and muscular type arteries were determined. It was found that in 90% of the cases, the same type of reaction to both loads was noted in the same athlete, both specific and nonspecific. /326

R. Ye. Motylyanskaya, I. Ye. Pulkina, L. I. Stogova, I. D. Surkina, L. N. Fatyugova, M. P. Ivanova (1960) studied the influence of specific and nonspecific physical loads in weight lifters. Identical values of decrease in the level of oxygenation of the blood were observed in lifting the weights and riding the stationary bicycle, along with similar pneumograms, similar values of pulmonary ventilation, presence of the Lindhard phenomenon, etc. The functional changes in circulation and respiration both in weight lifting and in work on the stationary bicycle were almost identical. The change in the pulse, arterial pressure, electrocardiographic data, level of blood oxygenation, pneumographic data, value of pulmonary ventilation, presence of the Lindhard phenomenon, etc. after repeated specific and nonspecific physical loads were quite similar, and in some athletes corresponded completely.

K. F. Slavolyubova and G. A. Goncharova (1960) studied the reaction of the cardiovascular system of swimmers to the combined, three-moment functional test. These same athletes swam 25 m (high-speed load) and 400 m (load for endurance), i.e. an investigation was performed for a specific physical load for swimmers. During the process of the investigation it was found that the data of adaptability of the cardiovascular system to running and swimming in swimmers was identical, the changes in pulse and arterial pressure and type of reaction being dependent on the state of training of the athletes. The investigations performed showed that the direction and intensity of the changes in the organism of the athlete during and after the physical load (specific and nonspecific) occurred identically in the overwhelming majority according to the indicators produced by the various methods.

In sports training in order to master various movements, i.e., in the process of improvement of the motor skills- complex and precise interrelationships are established, improved and automated in the cerebral cortex, related to the work of various muscular groups. This determines the specificity of the dynamic stereotype of the motor act.

However, the vegetative component of the dynamic stereotype apparently has no great specificity.

Thus, the investigations described above have shown that the response reaction of the organism results not from the structure and form of the motor act, but rather from the intensity and duration of the muscular work. Consequently, in the doctor's office during examination of athletes from various branches of sports, it is expedient to use a unified functional test, which makes it possible to evaluate the general level of training properly.

The usage of physical exercises specific for each concrete type of sports /327
should be recommended not under office conditions, but during training in the gymnasium, stadium, pool, etc. Investigations of sports training under natural conditions, when the athletes must perform great physical loads, characteristic only for a given type of sports, are directed toward the determination of the so-called special state of training.

Investigations with repeated physical loads. It can be seen from the material outlined above that the usage of functional tests of the cardiovascular system facilitates the determination of the functional capabilities of the organism of the athlete and thus determines his general level of training. Also, the practice of work has placed an important problem before sports medicine, that of determining the special state of training, which is being successfully developed at the present time. In this problem, among the main criteria are the indicators of hemodynamic changes and the response to a specific repeated load.

The process of training exercises and competition provides the best possibilities for determination of the capability of athletes to maximum muscular effort. The development of physical qualities, occurring together with improvement of the motor skills in the form of sports selected,

manifests itself in an improvement of the adaptability of the organism to loads specific for the concrete activity of the athlete in question. Special training of the athlete determines the level of development of the functional capabilities of the organism for the performance of a given type of movement, determined by the form of sports.

In order to determine the special state of training, the doctor must determine not only the general functional capabilities, but also the degree of adaptability of the organism to the maximum physical stresses in the concrete form of motor activity, i.e. in the form of sports selected, The special state of training is selected by studying the response reaction of the cardiovascular system to repeated physical loads of maximum intensity, specific for a given type of sports.

It should be considered that there is a close relationship between general and special state of training, since only on the basis of good general training can special training develop and improve. However, frequently even in the main training period, with a high level of general training we find insufficient special training, which limits the athlete in the achievement of high and stable results. These facts are of great significance not only for sports medicine, but for the practice of sports. The determination of a special state of training is performed in the process of medical-pedagogic observations and performed under natural conditions of sports training. /328

In investigations performed in recent years, the changes occurring in the organism of the athlete during the process of repeated loads and the intervals between them have been studied. The indicators determining the special state of training during investigations with repeated loads are the functional changes in the short intervals between loads and the dynamics of the change in indicators of the working ability. The changes occurring in the process of repeated loads in the work of the circulatory apparatus also reflect the speed of the process of development of quality, which is closely related to the special state of training of the athlete. Rapid warm-up is characteristic for well trained athletes, while slow warm-up or a worsening of the ability to warm up with repeated physical load is characteristic of the poorly trained athlete. An investigation of functional changes in the intervals between repeated loads allows us to determine the dynamics of recovery, which is one of the indicators of the state of training.

The selection of physical loads is performed as applicable to the form of sports, for example in an investigation of swimmers, a swimming load is given, for runners -- a running load, for heavy athletes -- exercises involving weight lifting, for skiers -- skiing, etc. The magnitude of the repeated loads is determined jointly with the trainer, considering the level of training of the athlete, the period of training, etc. It is necessary that the physical load which the athlete must perform approximate in its intensity or magnitude the maximum possible for the given athlete. The selection of the loads depends on the physical qualities to be investigated to the greatest extent -- speed, high-speed endurance, general endurance, or strength.

The repeated physical loads used in the investigations are usually not identical for representatives of various types of sports, but are always identical for the same athlete. The speed qualities of the athlete are determined by his response reaction to repeated exercises performed over short distances with maximum speed of movement, whereas endurance or high-speed endurance is characterized by the response reaction to repeated exercises of considerable volume but lower intensity. The intervals between repetitions are strictly standard and vary between two and ten minutes. In sports medicine, tables have been developed which determine the content of the tests with repeated physical loadings for athletes of various types of sports.

Investigations of the special state of training by the method of repeated loadings is used in those types of sports which are characterized by varying movement structures: a) in cyclical types of sports (running, swimming, skating, skiing); b) in a number of acyclic types, where each exercise is a complete action (lifting of weights, throwing, gymnastic exercises) and c) in mixed types (long jump, high jump, etc.). /329

In types of sports characterized by indefinite forms of movement (football, basketball, volleyball, etc.), the definition of the special state of training is performed on the basis of determination of the adaptability of the organism to repeatedly performed exercises as test investigations.

Before beginning the performance of investigations involving repeated loads, the pulse frequency and respiration are determined in the athlete in the sitting position at rest, the arterial pressure is measured, and the athlete's feelings are recorded. After this, the athlete performs his usual warm-up. After the warm-up, the test exercises are performed.

During the first minute of each interval between the repeated loadings, the pulse is measured (first ten seconds), along with the arterial pressure (during the next 20-30 sec), then the respiration is counted (for 10 or 15 sec) and an electrocardiogram is recorded. All indicators are recorded during the second and subsequent minutes between repeated loads in the same sequence. In order to assure objective examination, any external characteristics indicating fatigue are noted, and the answers to questions concerning the athlete's feelings are recorded.

Upon completion of the investigation, the athlete once more performs his physical loading. An important addition during the performance of a test is objective instrumental recording of the work performed. For example, speedographic recording of sectors of distances run, etc. Usually, the time required to cover each distance, weight of weights lifted, etc. are recorded.

The advantage of the method of repeated loads over standard functional tests is the possibility of comparing the working ability on the basis of the time required to cover distances, weights lifted, etc. and the adaptability of the organism from the response reaction to the repeated exercises. Frequently the data characterizing various aspects of the state of training vary in the same direction, indicating good, satisfactory or unsatisfactory state of

training. However, other variants are noted, when an exercise performed well is combined with poor reaction of the organism. This indicates that the high result was achieved by extraordinary expenditure of efforts. This variant indicates insufficiently stable functional preparedness of the athlete. It is in these cases that development of overstress is most probable. Frequently also good adaptability is observed with moderate or poor working ability, which indicates the possibility of increasing the volume or intensity of the test exercises. Thus, variants of combinations between the quality of performance of the test exercises and the adaptation to the exercises of the circulatory apparatus are quite important for a more complete evaluation of the state of training and proper construction of the training regime. /330

As the state of training increases in athletes, a marked change in the response reaction is observed in the investigations with repeated physical loads, manifesting itself in a more favorable relationship of the pulse and arterial pressure change after loading, as well as in more rapid recovery of these indicators. A certain parallelism is noted in the changes in the reaction of the organism to the standard loads both in the functional test and under training conditions. As the state of training increases, the athlete shows improvement in his adaptability to natural training loads and recovery occurs considerably more rapidly. The specifics of the state of training are usually determined not only in an investigation of the effects of the training exercise as a whole, but also in an investigation of physiological changes in the organism of the athlete during various parts of the training exercise.

Cases are frequently observed when during an extended period of investigation of athletes using the functional test, the cardiovascular system reliably indicates good state of training, while the data from investigations using repeated physical loads continue to improve. This phenomenon is explained by the following: the physiological mechanisms of expansion of the functional capacities are essentially identical both for the general state of training and the special state of training. However, the level of special training, in contrast to the general state of training, results from an increase in finer differential coordination connections, improvement of specific motor and vegetative reflexes for the given type of sports, and formation of a better and more differentiated dynamic stereotype. All of this is achieved only on the basis of a rather high level of general training and physical training of the athlete. Only a high level of training can be characterized by optimal interrelationships of the activity of all organs and physiological systems in the organism of the athlete.

Recent investigations by Yu. K. Shkhvatsabaya (1964), V. M. Solov'yev (1965) and others have demonstrated the promise of using the data from phase analysis of cardiac activity for an evaluation of the reaction of the circulatory apparatus to the loadings in functional tests. The authors considered changes in the phase structure of the cardiac cycle during work and during the recovery period. This expansion of the cardiological information has allowed early forms of overtraining and fatigue to be discovered. The investigations of A. D. Budkov (1965, 1966), who defined the duration of the /331

phases of the cardiac cycle directly under training conditions, are of great value as well. He succeeded in showing that the changes in the chrono-cardiogram are determined by the volume and intensity of the training loads. The objective criteria -- degree of phase shifts, rate of recovery of initial phase durations -- were important supplementary factors in controlling the training regime.

Orthostatic Tests

Tests involving changes in the body position are used to study the regulation of peripheral circulation. Upon transition from the horizontal position to the vertical position, due to the change in the hydrostatic conditions, the return of veinous blood to the heart is decreased. The redistribution of blood leads to a decrease in pressure in the carotid sinus. The systolic output is decreased. The blood volume per minute is maintained by increasing the cardiac rhythm. The systolic pressure decreases slightly or remains unchanged. The diastolic pressure increases upon transition to the vertical position due to the contraction of collaterals in the arterial system.

One of the factors helping to maintain a sufficient veinous return is the contraction of the leg muscles. In general during active changes of the body position, the changes in cardiac activity depend to a certain extent on muscular force. Apparently, the difference in the reaction to the orthostatic test of athletes and healthy persons not involved in sports is related to this (in addition to the nature of regulation). In order to study the influence of redistribution of blood determined by hydrostatic factors, the most suitable mechanism is the passive mechanism of changes in body position. However, this requires that a special tilt table be available.

In clinical practice, the method most used is that of Schellong (1937), in which the arterial pressure is measured in a test subject in the lying position repeatedly using the Korotkov method, at intervals of one minute, along with a measurement of the pulse frequency (until stable results are produced). Then the test subject quietly arises and stands for ten minutes in a free, unstressed position. Immediately after the transition to the vertical position, then each minute thereafter, the pulse frequency and arterial pressure are measured. In the opinion of S. P. Letunov and R. Ye. Motylyan-skaya (1951), the changes in pulse frequency and arterial pressure during the first 15 sec can be used to determine the excitability of the sympathetic segment of the vegetative nervous system. Later data reflect the /332
nature of the recovery of the tonus of the vegetative nervous system which changed when the position was changed.

Upon completion of ten minutes, the test subject once more lies down (clino-orthostatic test) and the arterial pressure and pulse frequency are measured immediately, then each minute for 3-5 min. The collar of the blood pressure measuring device is left on the test subject's arm throughout the

entire investigation; it is recommended that the air be exhausted completely after each measurement.

The study of the dynamics of arterial pressure and pulse frequency in athletes under the influence of the orthostatic test has been covered by an extremely broad literature. Based on many investigations, it is believed that the optimal reaction to the orthostatic test (frequently observed in athletes) is a slight increase in pulse frequency and minimal changes in arterial pressure. However, the physiological limits of the change in cardiac rhythm are rather great -- 10-40 beats per minute. The systolic pressure either remains unchanged, or decreases at the beginning of the test by 5-15 mm Hg, then gradually returns. The diastolic pressure sometimes remains unchanged, but more frequently increases by 5-10 mm Hg. The most sharply expressed changes in cardiac activity in healthy persons upon changing body position occur in youth, which results to a considerable extent from the lability of the vegetative nervous system.

In sports medicine and sports physiology, the opinion has long been current that orthostatic stability increases as a result of special exercises used by gymnasts, acrobats, divers, boxers, etc. This opinion was confirmed in the works of M. R. Mogendovich and his colleagues (1965). These authors showed that this training improves the mechanisms (interoceptive and proprioceptive) which counteract the influence of gravity.

There has been great interest in recent years in studies of orthostatic stability in space physiology. The changes in the reactions of the organism to complex orthostatic influences (long standing test) after many days limitation of motor activity have been investigated. A. L. Myasnikov et al. (1963), L. I. Kakurin et al. (1966), P. V. Buyanov et al. (1965) and many others have shown that under the influence of hypokinesia, orthostatic stability is sharply worsened. Suffice it to say that various authors in 40-80% of their observations noted development of orthostatic collapse, most frequently occurring on the ninth to fifteenth minute of standing. In the light of these data, it is interesting to note that in athletes changes to the reaction in the orthostatic test after ten days limitation of motor activity /333 were considerably less sharply expressed (L. A. Ioffe et al., 1966). Thus, orthostatic collapse occurred in only one of ten athletes investigated. Changes in the cardiac rhythm and arterial pressure in the remainder were relatively slight. These facts confirm to a certain extent the data of M. R. Mogendovich. Further study on the influence of sports training on the retention of orthostatic stability seems promising and important not only for sports medicine and space physiology, but also for clinicists.

The investigations of Rushmer (1959), V. L. Karpman et al. (1965), R. A. Svanishvili (1965) and other authors have shown that the reaction of cardiac activity to changes in the position of the body can be greatly influenced by the Starling mechanism. Here, in the lying and head-downward positions, the "volume loading of the ventricles" leads to a more powerful heart contraction, whereas the "volume underloading" observed in the standing position is accompanied by a weakening in the systolic activity of the

ventricles. It has been established that changes in the phase structure of the cardiac cycle under the influence of the orthostatic test in athletes and untrained persons move in the same direction. With various functional and organic disruptions in the activity of the circulatory apparatus, the orthostatic test is accompanied by distortions in the dynamics of the cardiac contraction.

We note in conclusion that orthostatic tests in sports medical practice are most important for evaluation of the degree of changes in regulation of the cardiac activity resulting from overstress, overtraining, after various diseases, etc., and should be used for dynamic observations of athletes.

Tests with the Breath Held

In clinical and sports medical practice, the functional state of the respiratory and cardiovascular system has been broadly investigated using tests with the breath held voluntarily. A test with the breath held while inhaled was suggested by V. A. Shtange in 1913, and a test with the breath held while exhaled was suggested by Gonczy in 1924.

According to the method of V. A. Shtange, the patient, sitting in a free position on a chair, inhales deeply and holds his breath. The doctor determines the time the breath is held by stop watch (nostrils of test subject held). According to the data of V. A. Shtange, the minimum breath holding time in healthy persons is 30 sec, while patients with circulatory deficiencies cannot hold their breath for more than 10-20 sec.

In the Gonczy test, the test subject in the lying position inhales deeply, then exhales deeply and holds his breath. After a measured walking exercise (44 m in 30 sec), the test is repeated. The breath holding time in healthy persons is no less than 25-35 sec, which is decreased after the measured exercise to 17-22 sec. With circulatory deficiency, the time the breath can be held is decreased (usually quite noticeably after walking -- to 5-15 sec). /334

When the breath holding test is administered to children, it must be recalled that the time the breath can be held at young ages is less. With increasing age, the time the breath can be held increases due to the decreased intensity of oxidative processes and increased resistance to hypoxia and hypercapnia (Z. D. Kovalenko and L. I. Gridina, 1937; N. A. Troitskiy, 1949; etc.).

The results of breath holding tests are determined to a great extent by the qualities of will and discipline of the test subject. Another defect of the test is the absence of any standard method for its performance. Adjustments introduced by various authors relate to the position of the test subject, depth of inhalation, nature of breathing before the test, dosage of loading, etc. All of this makes comparison of data of various authors difficult.

Attempts to objectivize the test of Shtange and Gonczy have involved methods of measuring the duration of holding the breath. Thus, various methods of recording the contraction of the muscles of the thorax and abdomen, and particularly of the diaphragm have been suggested (V. G. Kunevich, 1938; N. I. Tavastsherna, 1954; Skladal and Kochi, 1956; etc.). Movements of these muscles during the time the breath is held occur regardless of the will of the test subject, as the saturation of the blood with oxygen and carbon dioxide changes. In the opinion of S. B. Tikhvinskiy (1958), a common defect of all of these investigations is the fact that the dependence of movements of the diaphragm on the hypoxemia and hypercapnia has never been revealed (only N. I. Tavastsherna determined the content of carbon dioxide in alveolar air and judged the excitability of the respiratory center from the dynamics of this figure).

N. A. Troitskiy (1949), N. P. Likhachova and L. I. Sokolova (1925), S. B. Tikhvinskiy (1958) and others showed that the time the breath could be held depends on the depth of inhalation. According to the data of S. B. Tikhvinskiy (1958), the optimal inhalation depth is 75% of the lung capacity. At the same time, A. B. Gandel'sman et al. (1958) believe that the depth of inhalation for the breath holding test should be 90-95% of the true capacity of the lungs.

One common defect of tests involving holding the breath is the fact that, in contrast to the opinion of V. A. Shtange, even those suffering from diseases of the heart and lungs (and sometimes in the initial phenomena of decompensation), the length of time the breath can be held may be within the range of normal values. Changes in the breath holding time in athletes resulting from changes of the functional state of the organism (overstress, overtraining, etc.) are relatively weakly expressed. This defect of the test has been largely eliminated by observing changes in the saturation of the blood with oxygen during breath holding. /335

Oxyhemometric investigations (S. B. Tikhvinskiy, 1958; A. B. Gandel'sman et al., 1958; S. N. Popov, 1960; etc.) performed in the test with maximum holding of the breath, have allowed the nature of changes in saturation of the blood with oxygen to be determined. Thus, at the beginning of breath holding, a stable level of arterial blood oxygen saturation is observed for a time (according to A. B. Gandel'sman, this is the phase of stable oxygenation). The end of this phase is defined as the time when the oxygen saturation of the blood decreases by one percent in comparison to the initial level. During the second stage of breath holding, a decrease in the level of saturation of the blood with oxygen is observed. The rate of this decrease is influenced by the reserve of oxygen in the lungs and the intensity of its consumption by the organism, in connection with which hypoxemia develops more rapidly when the breath is held upon exhalation than upon inhalation, when the quantity of oxygen in the lungs is greater (A. D. Dembo, 1963). After breath holding is interrupted, the decrease in the level of oxygen saturation continues until the blood enriched in oxygen reaches the sector being studied. After this, recovery of the initial level occurs, first step, then more gradual (according to oxyhemometric data). It is interesting that the time required

for recovery of the saturation by one percent, the so-called recovery quality indicator (Khuan Me-Guan, 1959), which amounted to 6-8 for healthy, untrained persons, but was 3-4 for male athletes, and 5-6 for female athletes (S. N. Popov, 1960). This indicator is calculated by dividing the recovery time by the percentage of the decrease in oxygen saturation. S. N. Popov showed that worsening of the functional state of the athletes was documented by shortening of the phase of stable oxygenation, an increase in the rate of the decrease in oxygen saturation and a delay in recovery time to the initial blood oxygen saturation level. Performance of a test before and after a training exercise is particularly valuable to reveal fatigue or overtraining. Dynamic observations have shown that these data are considerably more sensitive to the state of training than tests with the breath held performed before and after measured loads.

We note in conclusion that in spite of the tremendous significance of functional tests, their data can be properly evaluated only when combined with ordinary medical investigation of an athlete.

CHAPTER IX

SOME METHODS OF TRANSMISSION AND ANALYSIS OF CARDIOLOGICAL INFORMATION IN SPORTS

ABSTRACT. A number of new and applied problems have been developed in cardiology in recent years, including the problem of transmission of cardiological information over long distances without wires. The radio-telemetry systems developed to perform this task are being continually improved in terms of stability, information transmission capacity, etc. This chapter analyzes these problems, as well as the areas of automatic analysis of cardiological information and the mathematical apparatus applied to this information.

The technical re-equipping of sports medicine observed in recent decades /336
and, in particular, the re-equipping of cardiology has resulted in the development of a number of new applied problems. The most pressing problem of today is no doubt the problem of transmission of cardiological information over considerable distances without wires. Considerable successes have already been achieved in this area by the development of various radio-telemetry systems.

These systems are being continually improved -- their interference stability is increased, the volume of useful information transmitted through one or more communications channels has been increased, etc.

In recent times, ever greater significance has been attached to improvement of methods of analysis of special information. The problem is that dynamic observations are widely used in sports medicine for all parameters or combinations of quantities. In these cases, the computational work is quite cumbersome if performed manually, while analysis of a large quantity of numerical material is difficult. Here, sports medicine may be helped by systems with automatic construction of transient process curves.

An important role also belongs to mathematical analysis of cardiological information, in particular auto- and cross-correlational analysis in combination with modern machine computing equipment.

This is the range of problems which is the subject of this chapter.

Radiotelemetry

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Modern sports medicine has reached a level at which ever greater significance is given to observations of the state of various functions directly during the performance of physical loads. It is necessary to observe the functions of the organism under natural conditions, during the process of activity, when the functions being studied are subjected to the combined influence of a mass of factors. The development of such investigations is greatly facilitated by radiotelemetry of physiological data, i.e. recording of these data at a distance by radio communications.

One of the first works in which radio transmission of physiological information was performed relates to the beginning of the 1930's, when the Moscow investigators A. A. Yushchenko and R. A. Chernavkin (1932) developed a radiotelemetric system for the recording of signals from a contact transducer. The contact transducer could be a step meter (which closed the contact with each step), contact device for recording sports movements, etc. However, works on radiotelemetry were primarily developed (both in the USSR and abroad) after the Second World War.

Up to now, three principal trends have been formed in biotelemetry (V. V. Rozenblat, 1965). The first trend, which has achieved marked successes in aviation and space medicine, is characterized by the fact that the test subject and the transducer are on board some object (aircraft, rocket, spacecraft, etc.), moving at tremendous speed and at tremendous distance from the investigator and receiver device. The test subject and transmitter are immobile as concerns the object carrying them. This trend has had a tremendous role to play in the development of space physiology (V. V. Parin, V. I. Yazdovskiy, 1961; Yu. M. Volinkin et al., 1962; N. M. Sisakyan, V. I. Yazdovskiy, 1962; etc.).

The second trend, which has been developed primarily in the physiology of sports and labor, is characterized by the investigation of a freely moving man, who carries with himself all the transmitting device and performs ordinary activities.

The third of the principal trends in biotelemetry, which is primarily of interest for clinical and experimental medicine, is characterized by the introduction of miniature radio capsules to the cavities of the body, principally into the gastrointestinal tract, from which information is transmitted on the temperature, acidity, motor function, etc.

In this section of this chapter, we analyze radio telerecording of cardiological data as the test subject moves freely during the process of his ordinary activity, in particular sports activity. This trend in radio- /338
telemetry has its specific features and difficulties, including the development of stability against interference caused by the energetic movements of the test subject (A. L. Vodolazskiy, 1959; V. V. Rozenblat and A. T. Vorob'yev, 1961), as well as the creation of miniature transmitting devices which, while being carried on the test subject and without interfering with his

activity, must provide the necessary range of transmission of information (several tens or hundreds of meters, or in some cases, for example in investigations of skiers or parachute jumpers, up to several kilometers). These specifics exclude the utilization of ordinary industrial models of apparatus and require the creation of special devices.

In 1948-1965, several dozens of laboratories and design groups in the USSR and abroad (USA, England, Bulgaria, Czechoslovakia, France, FRG, Holland, Hungary, etc.) published information on the first miniature apparatus for radiotelemetry of physiological information from freely moving men or animals.

Among the Soviet authors in this area, we should note first of all the works of B. V. Panin (1956, 1958, etc.) and the works of the designers of the All-Union Scientific Research Institute for Medical Instrumentation and Equipment (T. Ye. Timofeyeva, V. A. Anselevich, 1960; T. Ye. Timofeyeva, 1961), who created the tele-electrocardiograph, which has been series produced by the Moscow Plant for Electromedical Apparatus since 1962. Since 1955, work on dynamic biotelemetry as applicable to problems of sports medicine and labor physiology has been performed by us and our colleagues.

Among the foreign authors, we should note in particular the many years work of a group of American investigators led by Holter (Holter, Gengerelli, 1949; Holter, Glasscock, 1952; etc.), and the Bulgarian investigator L. Basan (1955, 1958). A particularly large number of works has been written concerning the recording of electrocardiograms (Parker et al., 1953; Holter et al., 1957; Beenken, Dunn, 1958; Webb et al., 1958; T. Ye. Timofeyeva et al., 1961; Winsor et al., 1961; Dendal, 1961, 1962; V. V. Rozenblat, A. T. Vorob'yev, R. V. Unzhin, 1962; etc.) and pulse frequency. This latter indicator is recorded by various methods: from the tones of the heart (Seliger, 1950, 1955), by photoelectric pickup on the ear (L. P. Shuvatov, 1959, etc.) or, more frequently, from the R wave in the biocurrents of the heart (V. V. Rozenblat, L. S. Dombrovskiy, 1957, 1959, etc.).

Techniques of radiotelemetry. A radiotelemetric system includes two main parts -- the "patient device" (miniature transmitter located with the test subject) and the "investigator's device" (receiving and recording device).

The "patient device" is the most important part of the system. It /339
includes: 1) the transducers and other devices which pick up the physiological pulse; 2) the amplifier-converter or coder (converting the physiological information into a signal convenient for transmission by radio); 3) the transmitter (usually in the uhf range); 4) the power supply.

The "investigator's device" includes, in addition to the receiver, a decoder (converting the information back to its initial form) and the recording device.

From the point of view of weight and size, the designs for "patient devices" created up to the present time are quite varied -- from electron tube

devices with ranges of 300-1500 m, weighing 600-1500 g (Metzner, 1958; Webb et al., 1958; T. Ye. Timofeyeva, V. A. Anselevich, 1960; etc.) to miniature semiconductor devices with an operating range of 30-100 m weighing around 100 g (R. V. Unzhin, V. V. Rozenblat, 1959, 1962; Winsor et al., 1961; etc.).

From the point of view of volume of information transmitted, the systems described to the present time are primarily designed for single channel transmission of physiological data. Multichannel devices have been described by only a few authors (Beenken, Dunn, 1958; L. P. Shuvatov, 1959, 1960). In addition to the single channel and multichannel systems, the combined structure type is also distinguished, in which a single channel (or each channel in a multichannel system) can be used to transmit several types of information depending on the transducer used (R. V. Unzhin, V. V. Rozenblat, 1962; R. V. Unzhin et al., 1962).

The Sverdlovsk biotelemetric group has designed and used a number of types of radio apparatus (V. V. Rozenblat, 1963) -- from the first radiopulsophone and miniature radiopneumograph, which allows the frequency of respiratory movements of the athlete to be recorded, to a miniature, but more complex radiopulsophone-electrocardiograph (REK) and a number of intermediate devices. One of these is the type KRP combined radiotelemetric device (models KRP-1m, KRP-2, KRP-2m, KRP-3, etc.) (V. V. Rozenblat, R. V. Unzhin et al., 1963), providing for recording of the pulse frequency (indicated by the R wave of the biocurrents of the heart), as well as respiration and movements.

Being a single-channel device, the instrument transmits these indicators at various times depending on the pickups connected. Synchronous transmission of two parameters as a single curve is also possible: for example, the R waves of the electrocardiogram may be superimposed over the rectangular signals of a contact pickup (respiratory mass with switch-valve; step meter in /341 shoes, etc.) (R. V. Unzhin, V. V. Rozenblat, 1963). The RST-1 radiosphygmotachograph has been developed on the basis of the KRP device.

An important step was the development of the first model of the radiopulsophone-electrocardiograph -- the REK-1 (V. V. Rozenblat, A. T. Vorob'yev, R. V. Unzhin, 1962), which allowed the pulse to be counted by sound, and the electrocardiogram to be recorded at moments of interest to the investigator.

With maximum amplification of the recorder connected to the receiver (electrocardiograph), cerebral biocurrents can also be transmitted by radio. The frequency characteristic of the amplifier in the REK-1 transmitting device (linearity between 0.5 and 5000 Hz with a gain factor of about 300) allows a number of physiological indicators to be transmitted by using the proper transducers.

Data on these devices has been published repeatedly including circuits, descriptions and the basis for individual technical decisions (R. V. Unzhin, 1963, a, b; E. I. Rimskikh, 1963, a, b, etc.).

Below, we describe two models of the "patient device" for a system of dynamic biotelemetry developed by R. V. Unzhin in 1965-1966 and used quite broadly by us in recent times.

The first of these is the KRP-7 combined radiotelemetry device, which is used for remote recording of the pulse frequency (using the R wave of the electrocardiogram), respiration frequency (by placement of a thermistor pickup in the path of exhaled air or by a change in the resistance of a rheostat pickup on a belt around the thorax of the test subject), actogram (by contact switch or by the change in resistance of transducers). In addition to these physiological parameters, any other parameters may be recorded radiotelemetrically if they can be first converted using the proper transducers either to electrical signals with amplitudes between 0.5 and 3 mv and frequencies of 1-50 Hz, or to a change in a 5-10 kilohm electrical resistance with an amplitude of change of 500-1000 ohms.

Being a single-channel device, the KRP-7 allows simultaneous recording of two physiological parameters to be performed, as long as the frequencies of the processes do not differ by a factor of more than 5 (pulse frequency-respiration frequency, pulse frequency-number of steps, etc.).

The KRP-7 device provides for graphic recording of physiological parameters using a type EKPSCh 10 recording electrocardiograph (model 60) and an FM radio receiver with a sensitivity of 1-1.5 μv at distances up to 50 m, and audible receiving up to at least 100 m considering propagation of radio waves in the 45-46-megaherz range. A schematic diagram of the KRP-7 device is presented on Figure 97.

The amplifier in the device is a three-stage amplifier, consisting of /342
transistors T1-T3 in a common emitter circuit. The time constant of the amplifier coupling circuits is so selected that the frequency components of the R wave of the electrocardiogram are primarily amplified. Temperature stabilization of the stages is provided by parallel negative voltage feedback (resistors R11-R13) and series negative current feedback (resistors R16-R18). The amplified signal is fed to the charge-discharge circuits of the carrier frequency generator, a symmetrical multivibrator using transistors T4-T5. The marker voltage for the charge-discharge circuit is provided by the voltage divider consisting of resistors R4 and R20. The signal from the amplifier, added to and subtracted from the marker voltage, causes a change in the pulse repetition frequency of the multivibrator, i.e. causes frequency modulation of the subcarrier oscillations. The frequency of the subcarrier oscillations is selected in the audiofrequency range (1000-3000 Hz), toward which the human ear is most sensitive. This allows not only a check of the operation of the channel, but also audible recording, and when necessary allows recording to be made on any series produced tape recorder for subsequent processing.

In addition to the amplifier, the marker voltage may be controlled using contact or rheostat pickups connected to divider R4, R20 through resistors R19, R21.

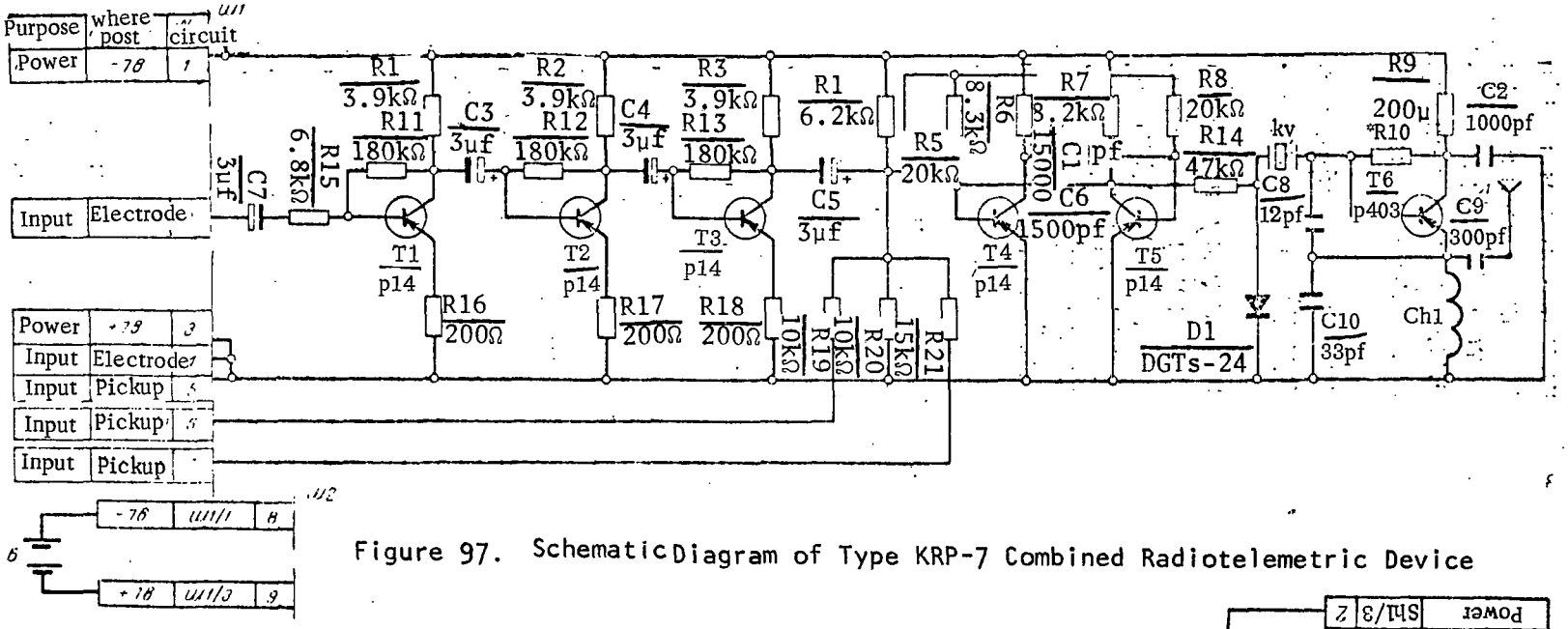
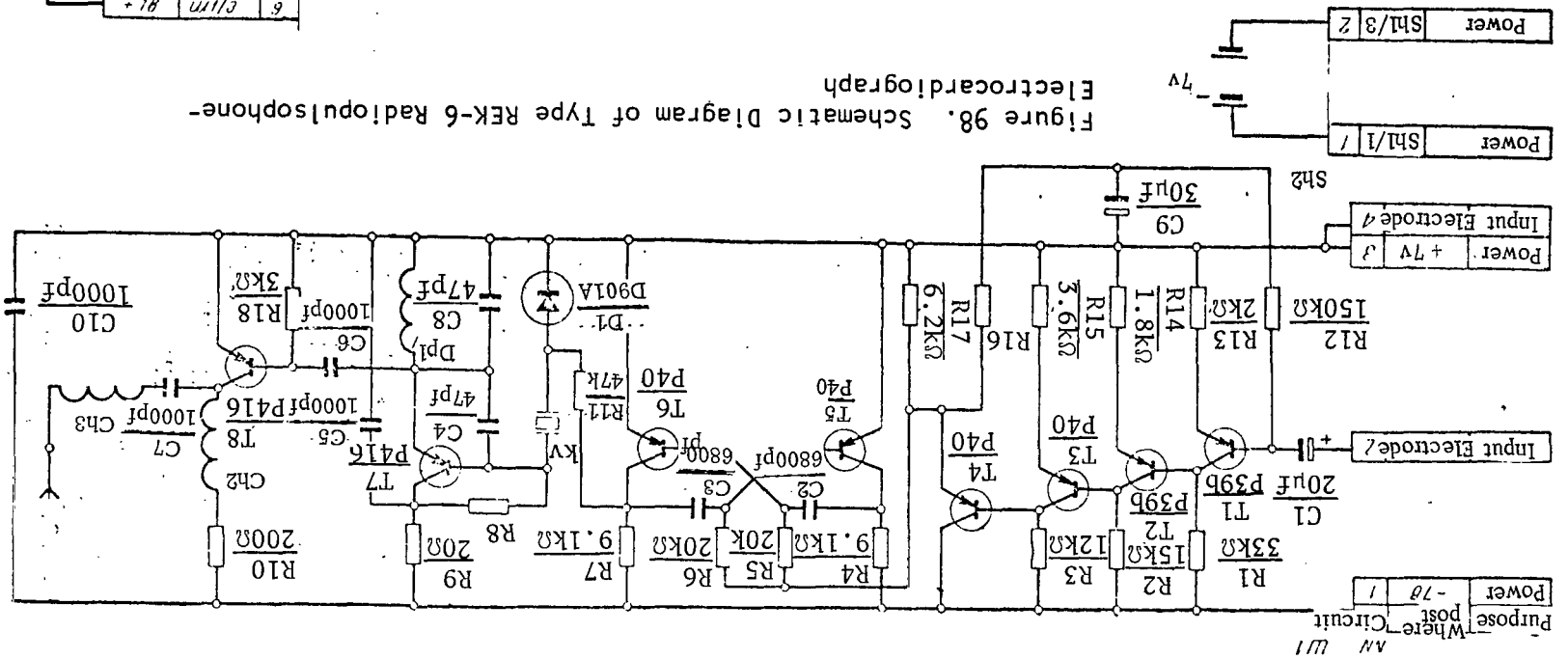


Figure 97. Schematic Diagram of Type KRP-7 Combined Radiotelemetric Device

Figure 98. Schematic Diagram of Type REK-6 Radiopulsophone-Electrocardiograph



The frequency modulated oscillations of the subcarrier are fed to the radio transmitter. It consists of an FM oscillator with a quartz stabilizer. Frequency modulation is performed by changing the frequency of parallel resonance of the quartz resonator using the capacitance of the P-П junction of the planar diode. The frequency modulated high-frequency oscillations from the emitter of transistor T6 are fed to the antenna.

Power to the device is supplied from a battery composed of 7 D006 elements. The operating life of the KRP-7 device before battery exhaustion is about 3 hours.

The KRP-7 "patient device" is assembled in a rectangular body, closed by a plexiglass cover. The supply pack is separate, attached to the floor of the device, and connected through a plug. The device, including power supply pack, is placed on the head of the test subject in a special braided cap. The weight of the patient's device if printed circuitry is used is not over 120-150 g.

Tuning of the device consists of selection of the electrical operating modes of the amplifier and transmitter. If transistors T1-T3 satisfy the tech-/343 nical conditions, the voltage on the collectors should be 4 ± 1 v, while if the transistors are out of specification this voltage can be established by adjusting resistors R11-R13. Resistors R10 is used to set the collector current of transistor T6 at 5 ± 0.5 ma. This current can be established from the voltage drop across resistor R1, which should be about 1 v.

The second model is the REK-6 radiopulsophone-electrocardiograph. Here the "patient device" is used for remote recording by radio of the pulse frequency from the R wave of the electrocardiogram or the complete electrocardiogram in one lead. In addition to the physiological parameters noted, radiotelemetric recording of any other parameters can be performed, providing they can be preliminarily converted to an electrical signal with an amplitude of 0.5-3 mv and a frequency of 0.3-100 Hz.

The REK-6 device provides graphic recording of data using the EKPSCh (model 60) pen recording electrocardiograph and an FM receiver with a sensitivity of 1-1.5 μv with a signal/noise ratio of 5:1 at ranges up to 100 m, or recording of pulse frequency by ear up to 150 m, using transmission in the 45-46-megahertz radio-frequency range.

A schematic diagram of the REK-6 device is shown in Figure 98. The amplifier in the device is a four-stage amplifier, consisting of transistors T1-T4. Three of the stages are directly coupled with a common emitter circuit. Each stage has negative current feedback, which results in high input impedance of the stage. The last stage in the amplifier uses a common collector circuit, which provides low input impedance of the amplifier. The setting of the working points of all transistors in the amplifier is achieved by selecting resistor R16 to provide a voltage of 3 v at the emitter of transistor T4. The amplified signal is sent to the charge-discharge circuit of the subcarrier oscillator. This oscillator uses a circuit similar to the oscillator in the KRP-7 device. The frequency modulated signal of the subcarrier is sent to the radio transmitter, which contains two stages. The master oscillator is quartz stabilized. Frequency modulation is performed in a circuit similar to that in the KRP-7 device The power amplifier consist of transistor T8 in a common emitter circuit. The

output circuit of the amplifier is simple, i. e. the loading circuit is the antenna. The antenna used is a flexible rod 20-30 cm long, tuned to the working frequency by elongating coil Ch3. The design and method of fastening the REK-6 device to the patient are similar to those of the KRP-7 device.

REK-6 devices can be used both for single-channel and for multichannel investigations with channel separation on the carrier frequency, i.e. by using /344 several independent transmitting and receiving devices feeding a common multichannel recording device.

Naturally, the data presented here do not exhaust the entire variety of available radiotelemetric equipment. Radiotelemetry is being ever more widely used. Since 1957, investigations of the pulse frequency, and since 1962 electrocardiograms of athletes during exercises have been performed in Sverdlovsk (V. V. Rozenblat, L. S. Dombrovskiy, 1959; V. V. Rozenblat, 1962; V. V. Rozenblat, A. T. Vorob'yev, L. M. Sanachev, 1961; M. B. Kazakov, N. M. Khodakov, A. T. Vorob'yev, 1963; etc.). Investigations in radiotelemetry have been performed in Leningrad (V. P. Pravosudov, 1959; V. V. Vasil'yeva et al., 1960, 1961). In 1962, a publication appeared by Estonian investigators (E. A. Viru et al., 1962; Kh. Yu. Sil'dmyae, 1962) and the colleagues of the Central Scientific Research Institute for Physical Culture (I. I. Knyazev, V. V. Matov, 1962). Also, material appeared from investigations performed in the USA and Japan (Bellet et al., 1963; Furuja et al., 1963; Jamakawa et al., 1964; etc.).

In the following, we will discuss certain results of the usage of two aspects of radiotelemetry in sports medicine.

Radiopulsometry. The materials which we have produced allow us to judge /345 the dynamics of the pulse frequency with various forms of sports activity (V. V. Rozenblat, 1962, etc.). Figure 99 shows a curve of the pulse frequency recorded directly during a training exercise. On the basis of analysis of such curves, the data from highly qualified skaters has been summarized (master of sports, first rank sportsmen). During winter in training exercises, the pulse frequency is as follows: during the warm-up, 126-144 beats per minute; with various types of technical exercise, 132-162 beats per minute; during short races, the pulse frequency increases to 180 beats per minute, and during acceleration in so-called variable work (alternation of moderate and intensive speed) -- to 186-192 beats per minute.

During the process of summer training, the changes in the pulse during individual exercises were similar to those above: when running at moderate speeds (during the warm-up or during the course of training), 138-150 beats /346 per minute; during exercises using roller skates, 150-174 beats per minute; during short races, the pulse also increases to 180 beats per minute, while during accelerations in variable work -- to 180-186 beats per minute. In some cases in this exercise pulse frequencies of up to 198 or even 204 beats per minute were noted. However, it must be emphasized that the pulse frequency exceeds 200 beats per minute during training exercises only as an exception,

usually reaching only 180-192 beats per minute in moments of most intensive loading.

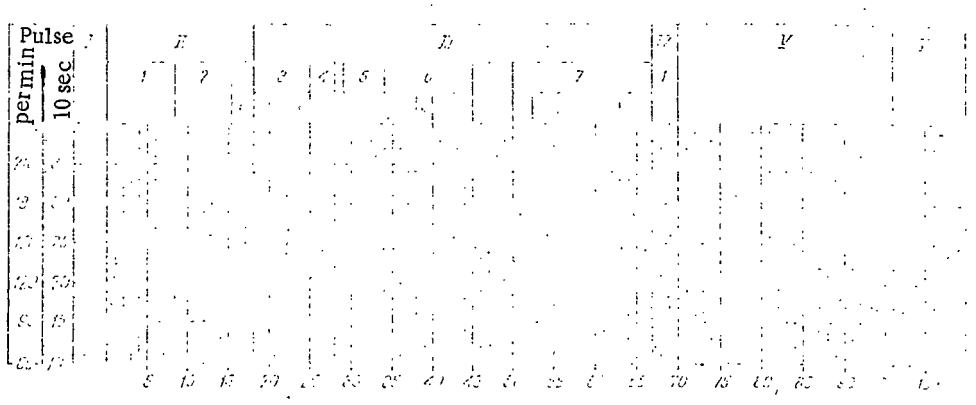


Figure 99. Radiopulsogram During Training. Sh-v, 17 years, class II skater, 10/10/1960. Horizontal line shows time (in min) from beginning of exercise. Exercises: 1, Running; 2, gymnastics; 3, hurdles, 70-80 m, three passes; 4, triple jump from standing start, three times; 5, 70-80-m run, three times; 6, technical exercise, two times; 7, run up and down ladder, five times. I, initial data; II, preparatory portion of exercise; III, main portion of exercise; IV, final portion of exercise; V, recovery; VI, duration of exercise (in min) and mean pulse frequency during exercise; circles, rest.

The typical changes in pulse noted with various exercises for highly qualified skaters have allowed approximate norms of pulse changes to be established for most principal exercises in winter and summer training sessions (Figure 100). The changes in pulse frequency in skiers during training sessions correspond with those of skaters; during the most intensive exercise -- rapid ascent with energetic work of the arms -- the pulse frequency was 168-192 beats per minute. Observations of the training sessions of bicycle racers performed using a radiopulsophone as modified by V. S. Smus' gave basically similar results (V. P. Pravosudov, 1959; V. V. Vasil'yeva et al., 1960, 1961).

In the winter season of 1959-1960, investigations performed for skiers training for ski jumps were performed (Figure 101) (in all, 26 jumps were made /347 with the athletes carrying the radiopulsophone in their caps), showing that the pulse frequency reached 168-180 beats per minute, and sometimes as high as 192 beats per minute during the climb up the ski jump framework on a very steep and clumsy ladder; the jump was performed by many athletes at a pulse rate of 150-170 beats per minute.

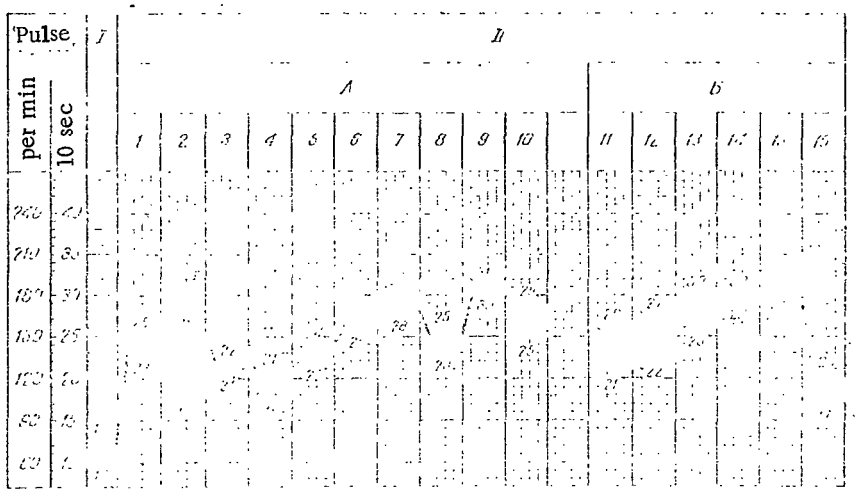


Figure 100. Typical Values of Pulse Frequency (According to Radiotelemetry) in Highly Qualified Skaters During Principal Exercises of Summer (A) and Winter (B) Training Periods. 1, running (during warm-up); 2, gymnastic exercises; 3, walking; 4, cooling-off exercises; 5, imitation of running on skates (technical limitation); 6, leaping imitation; 7, starts, with short sprints; 8, long-distance race; 9, acceleration plus running at variable rate ("variable running"); 10, exercise on roller skates; 11, skating during warm-up; 12, skating for development of technique; 13, starts with short sprint segments; 14, intensive running ("repeated work"); 15, acceleration in variable running; 16, quiet skating. Figures on diagram indicate limits of fluctuation of typical values of pulse frequency (counted in 10-sec segments). Remaining symbols as on Figure 99.

Interesting data were produced in observations of the nature of the pulse frequency of heavy athletes (M. B. Kazakov et al., 1963; A. T. Vorob'yev, 1963). Let us discuss them in somewhat greater detail.

It must be considered that adaptation of the organism to sports stresses depends not only on the general functional capabilities, but on the specific mechanisms of adaptation developed in the process of specialized training. Consequently, establishment of optimal data for increasing the frequency of heart contractions in each type of sports is particularly necessary. The literature data on the frequency of heart contractions after various heavy athletic exercises is somewhat contradictory. Apparently, this is explained by differences in the persons examined -- their qualifications, state of training and degree of loading. Also, the method of investigation of the pulse, particularly the interval between completion of the loading and

beginning of pulse counting, can be of considerable significance. The usage of radiotelemetric recording of the pulse continuously throughout the entire training exercise allows these methodological errors to be avoided.

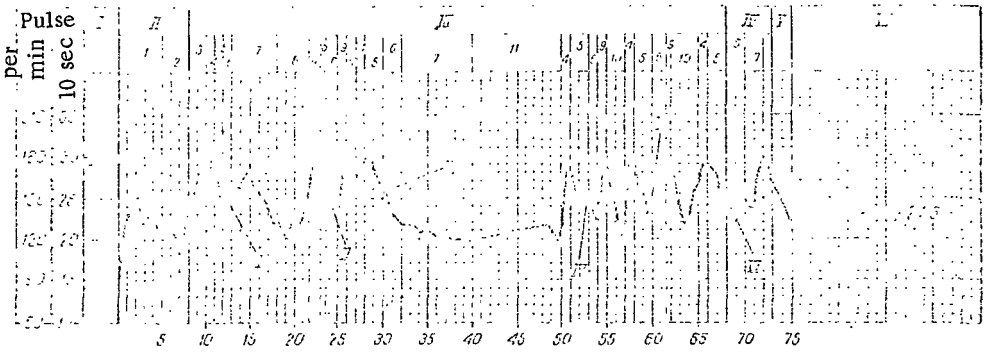


Figure 101. Radiopulsogram During Training. G-n, 19 years, class II skier, 3/12/1959. 1 and 9, ascent to judges' box; 2, gymnastic exercises; 3, climb to framework ladder; 4, climb on framework; 5, waiting before jump; 6, run after landing; 7, climb to doctor's office; 8, move from doctor's office to framework; 10, rest in doctor's office. Roman numerals in circles denote ski jumps. Remaining symbols as on Figure 99.

Two groups of heavy athletes were investigated: masters of sports and first-class sportsmen (first group) and beginning weight lifters (second group). The pulse reaction was determined during each of five phases of movements in the classical set of lifts: 1) before approach (background level); 2) approach to bar; 3) bending over to bar (start); 4) work (press, snatch or jerk).

In the first investigations, a dependence of the prestart pulse reaction on the weight on the bar was noted. Therefore, in studying the nature of the pulse reaction to individual movements in the classical lift, only the indicators of the reaction of the pulse during approaches to bars carrying 70 and 80% and in some cases 90% of the maximum weight for the athlete for a single lift was considered. The data produced in the investigations allow us to determine typical changes in pulse frequency during the process of performance of various movements in heavy athletic training, as well as throughout the entire course of training as a whole.

During investigations in the gymnasium before the training exercises the athletes in the first group showed an increase in pulse frequency of 9-18% in comparison to the data from the morning examination. This increase was looked upon as a positive prestart reaction to the load to be lifted. Warm-up exercises lead to an increase in pulse frequency to 120-136 beats per minute.

The extent to which the pulse changes are expressed during the course of /348
training depends to a considerable extent on the degree of training and the nature of the exercises performed. Table 28 shows data from statistical processing of observations during the training exercises. During the training exercises, the background pulse indicators in both groups fluctuated between 93 and 108 beats per minute.

As they approach the bar, the pulse frequency increases in athletes of both groups. In the first group, the pulse frequency averages 115.8, in the second group -- 122.4 beats per minute. However, the degree of increase depends on the weight of the bar: as the weight of the bar increases, the pulse frequency increases as well. This dependence is particularly clear in the first group, indicating the conditioned reflex nature of the increase in frequency as the so-called "sense of weight of the bar" is developed.

During competition, the following observations were performed for two athletes. The athletes approached the bar to perform the "two-handed press," but were then requested to perform this exercise without the bar ("mimic /349
press"). In the highly qualified athlete, the curve of changes in the cardiac rhythm (decrease in frequency upon inclination, main increase upon completion of movement, etc.) remained the same as in a true press; however, the increase was less and recovery was more rapid. In a beginning athlete, the "mimic press" did not produce these characteristic changes. In this case, the conditioned reflex mechanism had not yet been formed. Bending over the bar led to a decrease in pulse frequency in both test subjects when all the movements of the classical lift sequence were performed. In the first group, the pulse frequency decreased during the press by 14.4, during the snatch by 15.6 and during the jerk by 13.2 (when calculated against the rhythm in one minute); in the second group, the corresponding figures were 9.6, 15.6 and 10.8. The pulse increases once again from the moment work is begun.

Upon completion of the work, during the first few seconds the pulse reaches its maximum frequency, then gradually decreases. In athletes of the first group, the curve of recovery is characterized by a very steep drop, while the recovery for the athletes of the second group is more gradual. The "postwork" increase in vegetative functions upon completion of physical activity containing static components has been described by many physiologists (Lindhard, N. K. Vereshchagin, M. P. Mogendovich, etc.). However, these authors wrote of more or less extended static efforts. In our case, when the work itself lasts only 6-8 sec, the "postwork" increase in cardiac rhythm results simply from inertia of this indicator. The indicator, after receiving a powerful impulse toward increasing frequency due to the motor-visceral reflex from the proprioceptors of the muscles (M. R. Mogendovich, 1957), is not able to reach a high level in the brief time of performance of the work, and therefore continues to increase for a certain amount of time after the effort is stopped.

TABLE 28. MEAN DATA ON PULSE FREQUENCY PER MINUTE IN HEAVY ATHLETES DURING TRAINING EXERCISES

Exercise	Group	Before approach	App-roach	Bending over bar	Work	Recovery: 5-th second	10-th second	15-th second	20-th second	45-th second	75-th second
Press	First	93,6 ±7,2	114,0 ±5,4	99,6 ±7,2	121,2 ±7,1	134,4 ±9,0	134,4 ±4,8	124,8 ±5,3	124,8 ±5,3	101,[illegible] ±8,[illegible]	[illegible] ±[illegible]
	Second	98,4 ±6,7	121,2 ±3,8	111,[illegible] ±7,2	135,6 ±[illegible]	147,6 ±5,0	142,8 ±7,4	134,4 ±11,3	124,8 ±9,2	101,[illegible] ±11,[illegible]	[illegible] ±11,[illegible]
Snatch	First	98,4 ±7,2	110,4 ±13,2	94,8 ±10,7	114,0 ±11,6	132,0 ±12,4	128,4 ±9,6	126,0 ±8,8	118,6 ±10,7	103,3 ±10,2	96,0 ±7,0
	Second	94,8 ±4,0	114,0 ±5,4	[illegible] ±[illegible]	128,[illegible] ±7,[illegible]	144,0 ±[illegible]	141,6 ±3,2	133,2 ±8,0	124,8 ±9,5	114,0 ±9,[illegible]	[illegible] ±5,[illegible]
Jerk	First	98,4 ±8,4	112,8 ±7,8	99,6 ±[illegible]	124,8 7,[illegible]	150,0 ±7,8	150,0 ±9,5	142,3 ±10,3	134,4 ±8,0	110,4 ±10,7	99,4 ±13,6
	Second	108,0 4,2	123,6 5,8	112,8 8,8	135,6 10,4	156,6 7,0	151,3 7,0	147,6 5,2	[illegible] 4,7	123,4 3,5	[illegible] 4,2

Tr. Note: Commas indicate decimal points.

The fact of decreased pulse frequency as the subject bends over the bar and during the first moment of stress is interesting. This phenomenon was observed in both groups, although a greater decrease in pulse frequency was observed in the masters of sports than in the beginners. The more sharply expressed decrease in pulse frequency in qualified athletes indicates that the conditioned reflex mechanisms deepen the later (motor-visceral) reflex.

TABLE 29. MEAN DATA ON PULSE FREQUENCY (PER MINUTE) IN HEAVY ATHLETES IN FIRST GROUP DURING COMPETITION AND TRAINING

Exercise	Before approach	Approach	Bending over bar	Work	Recovery			
					5-th second	10-th second	20-th second	60-th second
Press								
Competition	142,8	158,4	149,1	169,8	181,8	175,8	162,0	147,1
Training	93,6	114,0	93,6	121,2	134,4	131,4	122,4	90,0
Difference	49,2	44,4	49,8	48,6	47,4	44,4	39,6	56,4
Snatch								
Competition	136,8	156,8	142,2	166,2	179,4	167,4	---	143,1
Training	98,4	110,4	94,8	114,8	132,0	126,4	- -	93,6
Difference	38,4	46,4	47,4	51,4	47,4	39,0	- -	49,5
Jerk								
Competition	138,0	155,4	139,2	156,6	162,0	165,0	157,8	139,2
Training	98,4	112,8	99,6	124,8	150,0	150,0	118,8	106,8
Difference	39,6	42,6	39,6	31,8	12,0	15,0	39,0	82,4

Tr. Note: Commas indicate decimal points.

Table 29 shows comparative data concerning the change in pulse frequency during training exercises and competition for athletes in the first group.

We can see from Table 29 that the direction of changes in pulse frequency during performance of movements in the classical lift sequence and during the recovery period during competition correspond to the changes during training. However, the pulse frequency under competition conditions is considerably higher (averaging 30-40 beats per minute higher). In contrast to training /350
exercises, when the maximum pulse frequency was observed in all phases of movement in the jerk, during competition it was higher in the press. This may be related to the greater emotional excitation at the beginning of competition, at which time the press is performed. It was noted that during the phase of bending over the bar, the pulse frequency during competition dropped more sharply than in training. The pulse frequency upon bending is not dependent on its initial frequency. The recovery period during competition is more extended than during training, but after the jerk, in spite of the considerable fatigue at the end of the competition, it is shorter than during the press and snatch, which is also a result of the emotional factor.

Going over to the problem of the individual differences in pulse reactions in various sports specialties, we should emphasize that changes during work are not clearly related to the initial values produced at rest. On the one hand, as can be seen from Figure 102, which shows curves of the pulse recorded at one time for two athletes, the degree of physical preparedness causes a difference in pulse reactions to loads (this difference is much greater than the initial difference). On the other hand, and which is particularly important, as the most intensive loads are approached, a decrease in differences in individual differences is noted. This can also be seen on /351
Figure 102: in spite of the sharp difference in the level of the two curves, during the most intensive loadings their peaks almost correspond, i.e. the curve with initial bradycardia shows a much sharper rise. In athletes, this phenomenon is noted beginning with loads of moderate intensity.

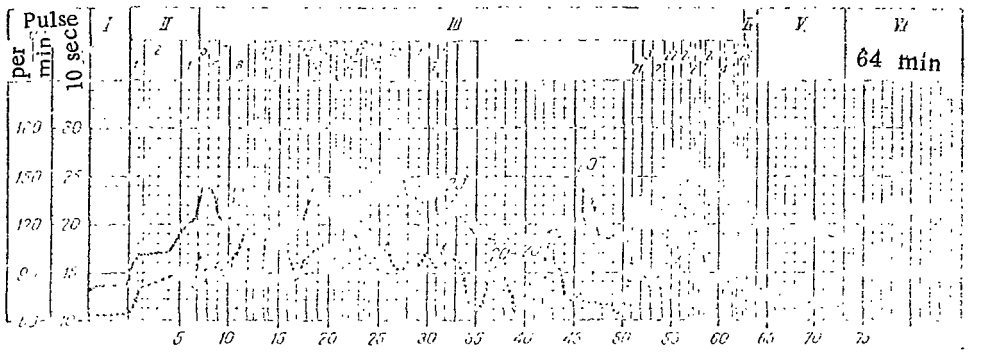


Figure 102. Radiopulsogram During Exercise in "Health Group" 11/5/1960 for T-va, 32 Years Old (Solid Line), and K-va, 48 Years Old (Dotted Line). Exercises: 1, 2, 4, as on Figure 101; 3, walking in place; 5-15, series of exercises with exercise bars; 16, sitting on floor, legs at angled position -- change position of legs; 17, breathing exercises sitting on floor; 18, sit-ups; 19, dance steps in motion; 20, walking on bar, jump; 21, carrying equipment; 22, "polka in the park"; 23, sit-ups, hands behind head; 24, push-up from wall bar, arms at chest level; 25, leg raise, supported on wall bar. Circles mark instructions. Other symbols as on Figure 99.

In analyzing pulse reactions to measured laboratory loads (S. P. Letunov test) it has been shown that the increase in pulse frequency is greater both absolutely and relatively for persons with lower pulse rates. As a result, individual differences in the initial values are smoothed by the influence of loads (I. G. Perlina, 1957; etc.). On the basis of the above, it would be erroneous to evaluate the magnitude of pulse reactions by their relationship to the initial quantities.

It is more proper to evaluate the absolute values of pulse frequency with various loads in practice, comparing these quantities with typical data.

In addition to the study of the reactions of the cardiac rhythm to individual forms of work, continuous radiotelemetric recording of the pulse frequency has allowed a new indicator of the intensity of a training load on an athlete to be introduced -- the mean pulse frequency during the training exercise.

In practice, this indicator is quite useful. In runners and skaters, it /352
averages from 130 to 165 (usually 140-150) beats per minute; in older persons performing gymnastic exercises, the figure is from 90 to 130 (usually 110-120) beats per minute.

In composing approximate norms of pulse reactions and their practical utilization, it is important to consider, as the investigations of skaters and heavy athletes have shown, that the "working pulse" of highly qualified athletes is regularly lower than in less qualified athletes. The question arises: what is the reason for this fact -- the degree of training or the age (the group of highly qualified athletes is always older, and as we know, the amplitude of the pulse reaction decreases with increasing age). An answer to this question was produced in the observations of R. N. Karelina (1963). Summarizing radiopulsograms during training exercises in gymnastics in two subgroups of highly qualified athletes of different ages (averaging 17.2 and 23.1 years), no differences were found in the amplitudes of the pulse reactions.

Thus, within this age range, the decisive influence is the level of general and special training of the athlete.

Factors causing an increase in pulse frequency during muscular activity, and also factors influencing the nature of the pulse reaction, are quite varied. First of all, an extremely important role is played by the energy expenditure of the organism, which determines the oxygen reserve and, through the neurohumoral regulatory system, causes a corresponding increase in the work of the cardiovascular and other systems. Secondly, the degree of muscular stress is very important, i.e. the intensity of the proprioceptive impulses, which cause a number of changes in the mechanism of the motor-visceral reflex (M. R. Mogendovich, 1957). Thirdly, the position of the body has an influence. When exercises are performed lying down or with the head downward, the pulse is increased to a lesser extent. Fourth, the emotional factor is tremendously important.

In female athletes whom we observed playing basketball, during a training game the pulse remained at 180 beats per minute and higher for 15-20 minutes (up to 198 beats per minute), although the intensity of the physical load hardly required this. During competition (running, skating) the pulse frequency according to our data averages 120-132 beats per minute at the start (up to 144 beats per minute in some athletes), averaging 180-216 beats per

per minute as the course is run and 204-230 beats per minute and higher at the finish line.

Whereas during training exercises only in individual cases are pulse frequencies higher than 200 beats per minute recorded, at the finish line in competition, values below 200 beats per minute are less common. The highest value which we recorded was 234 beats per minute. However, the possibility cannot be excluded of even more significant changes, which have been reported in the literature (M. V. Raskin, V. S. Farfel', 1947; A. P. Tambiyeva, Ye. A. Shirokova, 1958). /353

In addition to the various factors related to the nature of the load performed and the emotional background, the pulse frequency during work is influenced by the surrounding situation (microclimate, etc.), as well as a number of factors related to the condition of the organism. We must note particularly the presence of fatigue. This factor can be noted in radiopulsograms primarily by comparing the dynamics of the work performed and the changes in the pulse. Thus, at the finish line in competition, the increase in pulse frequency in some cases is accompanied by an acceleration of the rate in the last sector of the race, while in other cases the running speed even decreases, i.e. the physiological stress is ineffective (V. V. Rozenblat, 1961). Fatigue can also be evaluated by data on the dynamics of recovery during brief pauses, as well as upon transition to cooling-off exercises. For example, in a group of highly qualified skaters after exercises in which the pulse frequency was 180 beats per minute, 30 sec later the pulse usually amounted to: at the beginning of training, 138-144, and at the end of training, 150-156 beats per minute. If no great lengthening of recovery occurs upon multiple repetition of an intensive exercise, this indicates that no great fatigue is present.

Radioelectrocardiography. The data available on radioelectrocardiography in athletes are slight. However, certain facts are known which are of definite methodological, practical and scientific interest.

Until recently, there has been no sufficiently portable apparatus which could record an undistorted electrocardiogram remotely for a freely moving athlete.

The first approximation of such an apparatus in the USSR was the type TEK-1 tele-electrocardiograph (T. Ye. Timofeyeva, V. A. Antselevich, 1960), which was approved by the members of the Central Scientific Research Institute for Physical Culture (I. I. Knyazev, V. V. Matov, 1962; S. P. Letunov, V. V. Matov, 1963).

In February of 1962, we completed the development of the REK-1 radiotelemetric system, which allows remote recording of an undistorted electrocardiogram of an athlete even during very intensive muscular activity. The electrocardiograms were first taken in one chest bipolar lead (similar to a certain extent with standard lead II and particularly to leads V_4 and

V_3). Both electrodes are fastened along the median line of the clavicle: /354
one to the right at the level of the second rib, the other on the left at the level of the seventh rib (A. T. Vorob'yev, V. M. Stolbun, 1963).

The biocurrents of the heart are taken off by fluid electrodes held by suction after treatment of the skin (V. V. Rozenblat, A. T. Vorob'yev, 1961).

The first investigation performed using the REK-1 was recording of radioelectrocardiograms of seven skaters during competition at the First Winter Games of the Peoples of the USSR (V. V. Rozenblat, A. T. Vorob'yev, R. V. Unzhin, 1962). Those investigated included champions and record holders of the world and of the country. The radioelectrocardiograms were taken during competition over distances of 1500, 3000 and 10,000 m during the warm-up, during the race and during the recovery period (quiet skating). Primary attention was concentrated on the dynamics of the electrocardiogram during the transition from moderate loading (warm-up) to the highest stress (competition).

The interference from the biocurrents in the muscles is not too great. The greatest interference results from movement of the electrodes and changes in the resistance between electrodes and skin. Usage of methodology to eliminate this interference (V. V. Rozenblat, A. T. Vorob'yev, 1961) allows recording of biocurrents even during very intensive work.

As the electrocardiogram is being recorded, additional information can be produced. This includes the pulse frequency and respiration frequency.

The time indicators on the electrocardiogram agree completely with the long-established idea that during tachycardia, particularly that caused by physical stress, the cardiac cycle is shortened primarily by shortening the diastole. The P-Q interval and the QRST complex were slightly shortened, but as the rhythm increases to distances up to 180-200 diastole sectors, T-P actually disappears completely, the P wave is superimposed on the T wave and precise determination of P-Q and QRST becomes impossible.

The approximate value of the systolic indicator increases here to 70-80% and even more. The electrical diastole of the ventricles occupies a brief sector in the P-Q interval. The analysis of the changes in configuration of the electrocardiographic curve is limited in that we had data only from one lead. Changes were quite moderate. As a rule, a decrease in the S-T sector was noted in all athletes, which, according to data in the literature, is typical for tachycardia upon physical loading. A tendency was noted to a decrease in the amplitude of the R wave and an increase in the S wave. The T wave during the process of loading changes only slightly and noncharacter- /355
istically[1]. During the first minutes after completion of the loading, a

[1] Data on the dynamics of the T wave differ from the results of most investigators who studied changes in the electrocardiogram during muscular work -- Ed.

tendency is noted to an increase in the T wave. In some athletes, the typical loading changes described above (lowering of S-T interval, decrease in R wave, increase in S wave) were expressed upon transition from warm-up to the stressed competitive state quite clearly, while in others the change was less significant.

We also performed radioelectrocardiographic investigation of heavy athletes during training exercises and competition. In all, 28 athletes were investigated (masters of sports, first, second and third class athletes). Twelve observations were made during training exercises and sixteen in competition.

As a result of the great static-dynamic efforts required in weight lifting, the biocurrents of the muscles are recorded considerably more intensively than in skaters, although this type of "noise" appears only at the moment of the principal movement, which lasts for a few seconds. It practically represents no difficulty in analysis of the form of the electrocardiogram (Figure 103). Noise from muscular biocurrents results from the work of the muscles directly beneath the electrode (locally), while remote groups have no noticeable influence on the electrocardiogram even during intensive work. The proof of this is the "press" exercise (where the chest muscles beneath the electrode work intensively and the biocurrents are recorded heavily) and the "squat with bar on shoulders" exercise carrying a heavy weight, where the biocurrents of the muscles are almost not seen at all on the electrocardiogram, although the stress of the muscles of the back, thorax and lower extremities in this case is greater.

The time indicators of the radioelectrocardiogram were characterized as the pulse frequency increased by the same changes as for the skaters. The configuration of the electrocardiogram upon transition from relative quiet to intensive work was characterized by changes within the limits of the physiological influence of the great physical loads.

In almost all the athletes studied, the amplitude of the R wave decreased slightly during the exercise, and the S wave increased slightly. The amplitude of the T wave with rapid pulse (over 180 beats per minute) was increased. Possibly, its increase in this case was a result of the superimposition of the P wave on the T wave. As the diastole was lengthened and separate P and T waves appeared, the amplitude of the T wave was decreased to its initial values.

In the overwhelming majority of curves in the process of observation no /357
pathological changes were noted. These were discovered only in some individual cases. Thus, when a heavy weight was lifted, two athletes with initial indicators of overtraining (subjective) showed a clear drop in the T wave on the radioelectrocardiogram, which was depressed immediately after lifting the bar and remained low for 30-50 sec, then began gradual recovery of its amplitude to the initial value over 2-3 min (Figure 104). This sharp drop /358
in the T wave indicates disruption of the recovery processes in the myocardium, and although the athlete does not have any physical complaint, in

combination with other data this may be one of the objective indicators of incipient overtraining. In one athlete in competition, several times upon approaching the bar and immediately after the exercise, partial block of the right branch of the bundle of His was noted. In the literature, individual cases of this pathology have been described (V. Tishler, 1959). In another case through the entire competitive cycle immediately after performance of exercises, individual ventricular extrasystoles were noted, which disappeared 0.5-2 min after beginning of the recovery period.

In summarizing the materials produced, we can state that competitive loads, according to electrographic data, are quite adequate to the capacities of the trained athletic heart and cause no changes similar to pathological changes, even at the moment of greatest stress. Consequently, in clinical evaluation of a radioelectrocardiogram during the process of competitive loading, the normal criteria must be used.

Since 1964, we have gone over to two-channel radiotelemetric investigations, concentrating our attention on the clinical and physiological study of athletes with extrasystolic arhythmia.

In conclusion, we note that successful development of dynamic radioelectrocardiography and introduction of this process into sports medical practice will require that a number of problems be solved.

Thus, we must unify the methodology of leads and clarify the specifics of the curves recorded using these leads in comparison to the leads generally used in clinical practice (standard, monopolar from extremities, monopolar precordial). At the present time, quite varied bipolar precordial leads are used (A. Ya. Pleshchitser, 1953; Carbery et al., 1960; Nehb, 1938; Holmgren et al., 1961; L. A. Butchenko, 1963). We must also create reliable multichannel devices for radiotelemetry.

System for Automatic Construction of Curves of Transient Processes in Cardiodynamics

The task of investigation of cardiodynamics during intensive and varied muscular activity is one of the most pressing in sports medicine. One method which yields very complete information concerning the contractive activity of the heart is phase analysis of the cardiac contraction, performed on the basis of polycardiographic techniques. Therefore, the usage of phase analysis for the study of the reactions of the heart to physical loads has theoretical and /359
and practical significance. The curves of transient processes thus produced, reflecting changes in the parameters investigated with time, allow us to determine the mechanisms and quality of regulation of the circulatory system (V. L. Karpman, 1965). This in turn can be used to evaluate the general functional state and state of training of the athlete.

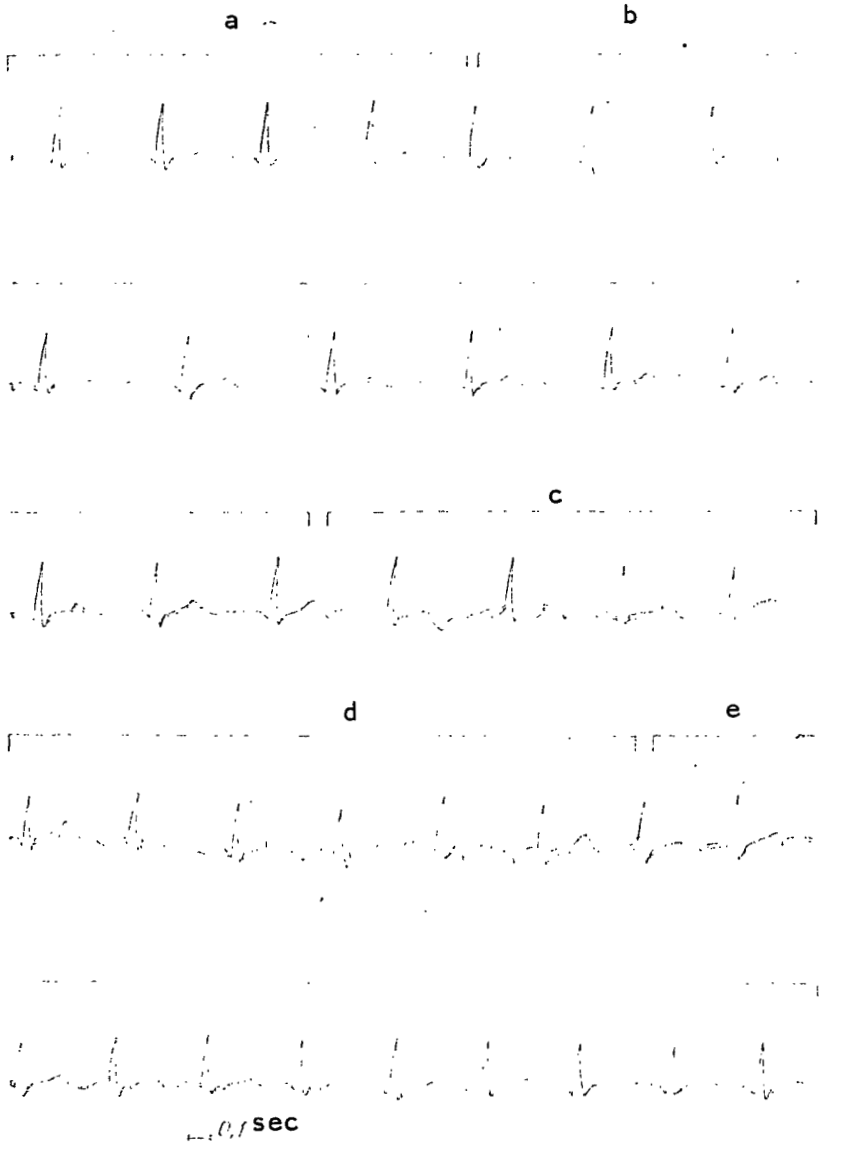


Figure 103. Radioelectrocardiogram of Ch, 24 Years Old, Heavy Athlete Second Class, During Training. Exercise -- pressing 90 kg with two arms: a, Approach to bar (pulse frequency 136 beats per min); b, Bending over bar (pulse 109-100 beats per min); c, lifting bar to chest (pulse 120-135 beats per min); d, pressing bar (pulse 135-142 beats per min); e, completion of exercise (pulse 150 beats per min).

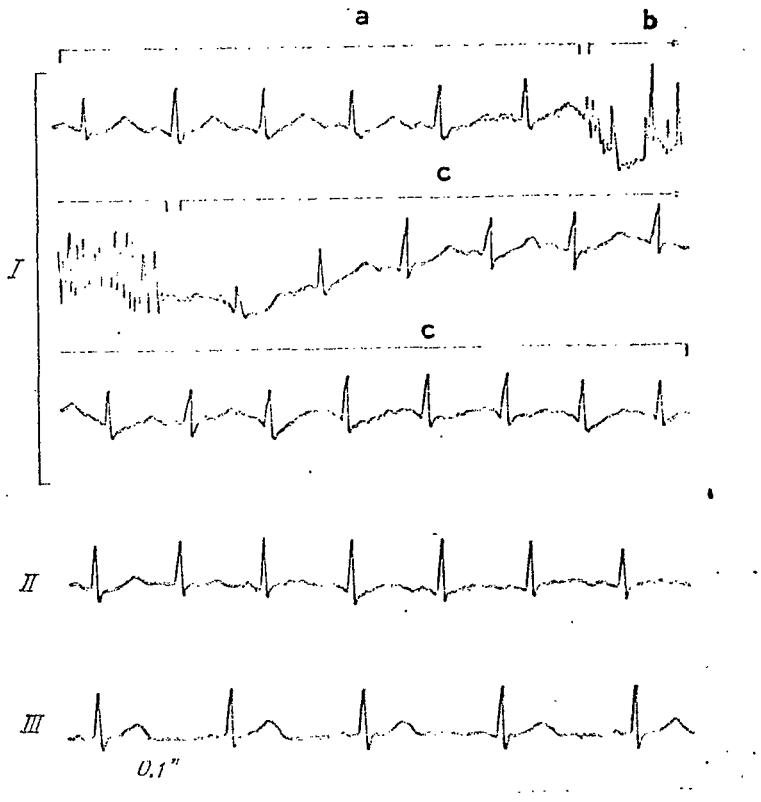


Figure 104. Radioelectrocardiogram of Heavy Athlete, Master of Sports P, 22 Years Old, During Training. Exercise -- snatch 102 kg: I, during process of exercise: a, bending over bar; b, snatch of bar (interference from muscular biocurrents seen); c, upon completion of exercise (note sharp depression of T wave); II, 30 sec after exercise (T wave still depressed); III, 1 min, 20 sec after exercise (complete recovery of T wave to initial value.

The beginning of the active study of the phase structure of the heart contraction during muscular activity was the work of V. L. Karpman and Yu. K. Shkhvatsabaya (1962), who performed synchronous recording of the electrocardiogram, carotid artery sphygmogram and phonocardiogram of an athlete exercising on the bicycle ergometer. However, the processing of these recordings, consisting of cyclical measurement of the duration of the phases of the cardiac contraction and construction of curves of the transient processes was performed manually, which is a cumbersome and difficult task and limits the accuracy of the results.

In connection with this, we, on a suggestion by V. L. Karpman, began working on the development of a combined computer and radiotelemetric apparatus for automatic phase analysis of the cardiac activity of a freely moving athlete. The apparatus which we have constructed allows recordings of the transient processes (immediate information) to be produced directly during an experiment and, furthermore, allows the processes to be recorded on an ordinary tape recorder, in order for more detailed processing to be performed later.

The process of collection and processing of the necessary information is completely automated and includes the following principal operations: 1) transmission of the electrocardiogram, phonocardiogram and central pulse sphygmogram of the athlete through a radiotelemetric channel; 2) location

of characteristic points on these curves delineating the phases to be measured; 3) representation of phase durations in a form convenient for recording.

In correspondence with this, the apparatus developed includes: a four-channel radiotelemetric system, an interference-stable unit for detection of boundaries of time intervals measured and a multichannel intervalometer which converts the durations of these measured phases to analog electrical voltages. In the following, we will not discuss the general principles of radiotelemetry, which have been discussed particularly by V. V. Rozenblat and his colleagues earlier in this collection.

Telemetry system. The multichannel radiotelemetry system forms a communications line between the athlete being investigated and the apparatus for processing and recording of data. As we go over to a description, we will /360 briefly note the electrical and spectral characteristics of the elements of the initial curves which are used for detection of boundaries of the phases measured, considering the specifics of the transducers used.

As we know, an idea concerning the phase structure of the cardiac cycle can be produced by combined analysis of three curves: the electrocardiogram, phonocardiogram and carotid artery sphygmogram.

The beginning of depolarization of the ventricles corresponds to the Q wave, which is immediately followed by the R wave. This sector of the electrocardiogram contains frequencies from 15 to 60 Hz. In order to record the electrocardiogram, the horizontal DS lead suggested by N. A. Agadzhanyan, I. T. Akulinichev et al. (1962) was used, this lead being distinguished by high interference stability and the production of a high-amplitude R wave. The electrodes were fastened to the test subject by a tight rubber collar. With this placement of the electrodes and ether treatment of the skin, the amplitude of the R wave at the input to the electrocardiographic amplifier reaches 2 mv.

Closure of the mitral and aortal valves of the heart is accompanied by appearance of tones I and II on the phonocardiogram, the frequency range of which (excluding the low-frequency range of tone I) is 100-200 Hz. The beginning of high-frequency oscillations of tone I corresponds to the beginning of the mechanical systole, while the beginning of tone II corresponds to the end of the protodiastole. In order to record the phonocardiogram, we used a piezoelectric microphone developing a voltage up to 100 mv into an impedance of 50 kilohms.

In order to determine the moment of transition of the phase of isometric contraction to the phase of expulsion, we use the central pulse sphygmogram. The characteristic point on this curve is the beginning of the anacrotic rise. The correction required for the propagation time of the pulse wave from the heart to the transducer on the neck is introduced to the system in the arithmetic device (Figure 105).

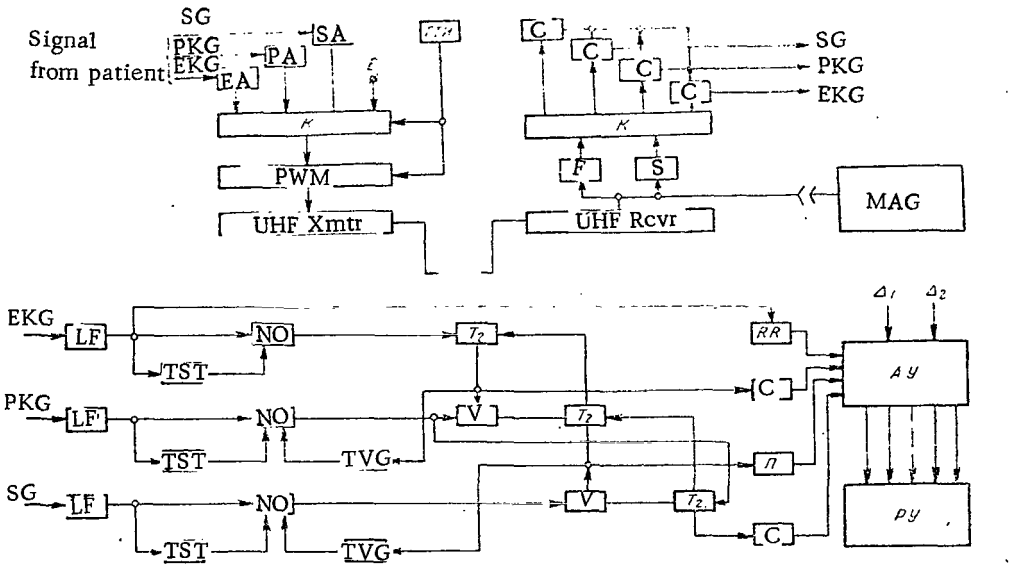


Figure 105. Functional Diagram of Complex of Apparatus for Automatic Phase Analysis of Cardiac Activity of Athlete. Elements of radiotelemetry system: EA, electrocardiographic amplifier; SA, amplifier for first derivative of sphygmogram; PA, phonocardiogram amplifier; E, constant voltage at input of synchronization channel; K, electronic commutator; PWM, pulse width modulator; UHF Xmtr, UHF transmitter; UHF Rcvr, UHF receiver; F, rectangular pulse former; S, synchronization pulse separation circuit; C, PWM-analog voltage converter. Elements of detection unit: LF, linear filter; NO, null organ, threshold decision device; TST, threshold setting tracer circuit; TVG, threshold voltage generator. Elements of multichannel intervalometer: TG, trigger; V, potential-pulse valve; PP, automatic cardiac cycle measuring circuit; AD, arithmetic device; RD, multichannel recording device; MAG, magnetic tape recorder

A special investigation (V. L. Karpman, Z. B. Belotserkovskiy and V. L. Utkin, 1966) showed that for phase analysis, the pulse curve can be successfully replaced by its first derivative. This has allowed the usage of a miniature electromagnetic type rate transducer, with low internal impedance, providing a voltage at the input of the amplifier of up to 2 mv.

The electrical signals from the transducers are sent to the inputs of the channel amplifiers (Figure 105), the frequency characteristics of which are matched to the spectral composition of the characteristic sectors of the initial curves.

After amplification, the electrocardiogram, envelope of the phonocardiogram and sphygmogram are sent to the electronic commutator of the transmitting device of the telemetry system (K on Figure 105). The commutator connects the /361
outputs of the channel amplifiers to the input of the pulse width modulator (PWM on Figure 105) in sequence. The level of the voltage at the input of the modulator is converted to a proportional width of the pulse at its output. As a result, pulses belonging to all channels being transmitted alternate in the overall pulse sequence modulating the carrier frequency of the radio transmitter.

At the receiving end of the communications line, the rectangular form of the channel pulses is restored, then the pulses are distributed into the corresponding channels. The same electronic commutator is used as in the transmitting device.

In order to match the operation of the commutators, one of the channels /362
of the system carries a synchronizing pulse. This same channel is used for compensation of interference in the radio channel. For this purpose, a constant value is transmitted through the synchronization channel, which is distorted in the radio line along with the signals of the electrocardiogram, phonocardiogram and sphygmogram. As a result, the output of the synchronization channel in the receiver device carries a signal which is then subtracted from the information signals distorted by noise.

Thus, this multichannel radiotelemetry system realizes the principle of time division multiplexing of a communications line with pulse width modulation of the subcarrier.

We know that there are two main methods of multiplexing -- time and frequency division. In the first case, the signals transmitted enter the communications channel in sequence, while in the second case they are located in nonoverlapping frequency ranges. Both methods are equally resistant to interference and equally effective in communications channel utilization.

In multichannel biotelemetric systems, time division multiplexing is generally not used, due to the complexity of commutation and synchronization systems (R. M. Bayevskiy, 1965; L. P. Shuvatov, 1959). However, the rapid development of digital computer equipment in recent years has lead to the creation of sets of unified elements for the conversion and commutation of pulses. This allows us in principle to solve the problem of microminiaturization of apparatus for time division multiplexing of biotelemetry communications channels easily, creating the necessary circuits from standard micromodules. Engineers attempting to develop systems using frequency division of channels generally do not have this possibility.

The radio channel of this telemetry system is formed of two standard miniaturized UHG tranceivers with crystal frequency stabilization, providing search-free communications without tuning at ranges up to 2 km (UHF on Figure 105). The weight of the entire apparatus carried on the athlete

during the experiment is not over 1 kg. With tne exception of the power supply switch, the transmitter contains no control or adjustment elements.

The multichannel radiotelemetry system can be used not only in combination with apparatus for phase analysis of cardiac activity, but also independently. This requires no changes in circuitry except for the channel amplifiers in the transmitter, which should be adapted to the particular transducers used. When desired, the number of simultaneously transmitted messages can be easily increased to 8 or 16. If the radio channel is replaced by a magnetic tape recorder (MAG on Figure 105), the radiotelemetric system is converted to a multichannel system for recording of very low-frequency physiological signals on the magnetic signal carrier. /363

Interference stability. The problem of interference-stable detection of characteristic points on the initial curves is the most difficult task in the creation of apparatus for automatic phase analysis. The literature contains detailed descriptions of methods of suppression of interference at the commercial frequency (A. M. Rybakov, 1965, etc.). These materials were carefully studied and used in this development, since the athlete's body and apparatus which he carries are always subjected to the interference induced from power and light wires in the stadium or laboratory where the investigation is being performed. For cases of high level of induction, filters of an original design are provided, with high discrimination factor against counterphase noise of 50 and 100 Hz with minimal distortion of the form of the useful signal.

Unfortunately, the interference with which we must deal in automatic testing of the condition of a human or animal in the process of his normal vital activity amounts to more than the ac background noise. For example, in automatic phase analysis of the work of an athlete's heart, each of the three curves recorded contains, in addition to the useful components, elements of random noise. In electrocardiography, noise arises also as a result of the activity of the skeletal muscles, accompanied by electrical phenomena. Respiratory noises are added to the phonocardiogram during the performance of physical exercises. Movements of the test subject distort the form of the pulse curve particularly strongly.

These noises are random in nature, and the difficulty of eliminating them is increased by the fact that their origin, and consequently the principal spectral and electrical characteristics correspond to a great extent to the characteristics of the useful signal. Proper selection of the design of transducers and their location on the body of the test subject can reduce the level of this noise somewhat, but the principal efforts in combatting noise must be made by the computer and logic devices.

To the interfering influences just mentioned we must add the noise and error introduced in the communications channel, consisting of a multichannel radiotelemetry system and in the case of repeated processing of experimental results, the magnetic recording system as well.

The complexity of the problem has required that the entire arsenal of technical means and methods accumulated by the theory of interference-stable reception be used for its solution. However, the specifics of the problem of automatic phase analysis, consisting in the fact that the detection of characteristic points on the initial curves and measurement of intervals between them must be performed practically instantaneously, adds its own limitations to the selection of a method of noise elimination. In /364
particular, the very effective method of interference stability augmentation called accumulation cannot be used at all. Therefore, we must base ourselves on methods of rapid detection or detection upon onetime reception.

The basis of the method of detection used in the device currently under discussion is the theory of statistical solutions (A. A. Kharkevich, 1963), and the principal circuit elements performing the detection function are the threshold decision device (NO on Figure 105) -- common for the channels of the electrocardiogram, phonocardiogram and sphygmogram. The threshold device, when interference acts on its input, solves the problem of selecting one of two possible decisions concerning the presence or absence of a signal.

The noise in the biotelemetry system for automatic collection of data is a random process and is described by statistical and spectral characteristics. Knowledge of these characteristics is required in order to create an optimal system with respect to interference stability.

The variety of noise sources outlined above allows us to consider the resulting noise acting on the input of the threshold device as a stochastic process with normal, or Gaussian, distribution. This follows from the statement formulated by V. N. Lyapunov that the sum of a sufficiently large number of independent random quantities has normal distribution, regardless of the distribution of the components.

In order to provide high interference stability for the system for automatic measurement of cardiac cycle phases, the radiotelemetry channel carries not the initial curves themselves, but rather short pulses corresponding to the moments of appearance of characteristic points on these curves. For this, the channel amplifiers of the radiotelemetry system are intentionally set in a nonlinear operating mode and therefore perform the operation of preliminary detection themselves. The threshold for preliminary detection is so selected as to minimize the probability of erroneous transmission of a signal.

The presence of information concerning an expected signal form in the form of a short rectangular pulse and the assumption of the Gaussian nature of the distribution of noise acting on the system allows the method of optimal filtration, broadly used in radar for the detection of weak signals, to be used in the interference-stable detection section. The elements of the quasi-optimal filter in the detection section are a linear filter, matched to the frequency characteristic of the expected signal, and a threshold device, connected with circuits for automatic threshold setting.

The decision concerning the presence or absence of the useful signal in the mixture of signal and noise at the input of the threshold device is made on the basis of a comparison of the input and a standard (threshold) voltage. The level of the threshold in the detection system should be set slightly higher than the noise level, but lower than the level of useful pulses. /365

Since the noise amplitude varies over wide limits during the course of an experiment, the threshold level should also change, following the noise. In order to achieve this, the standard input of the threshold device receives a voltage formed by the circuit which tracks the threshold setting, and is equal to the instantaneous value of the noise envelope.

An additional increase in the reliability of detection is produced by using differences in the statistical properties of signal and noise. Random noise may exceed the established threshold with equal probability at any moment in time, while the probability of formation of the next useful pulse in a sequence in each channel changes through the duration of the cardiac cycle. In correspondence with this, the threshold levels of the detection system are continually adjusted using the threshold voltage generator, the form of the output of which is determined by the statistics of the intervals being measured and the instantaneous values of cardiac rhythm (TVG on Figure 105).

The input of the electrocardiogram, phonocardiogram and sphygmogram channel pulse detection unit receives pulses from each channel in strict sequence. This obvious situation was created in order to increase the interference stability of processing of the structure of the intervalometer unit, where each preceding pulse prepares the possibility of arrival of each subsequent signal from the detection section. This helps to suppress false pulses in all channels except for the channel in which the appearance of a pulse is expected.

Short pulses, formed by the threshold circuits in the detector, are sent to the automatic time interval measuring device.

Intervalometer and arithmetic device. The multichannel intervalometer and arithmetic device connected to the recorder make up the terminal unit of the system for automatic construction of transient process curves. The method of phase measurement of the cardiac cycle used in the intervalometer circuit can be called the method of pulse conversion. It consists in that the measured time intervals are represented in the form of rectangular pulses of equal amplitude. Subsequently, these width-modulated pulses are converted to an analog voltage, which is recorded on a strip chart in the form of transient process curves.

It should be emphasized that the pulse width modulation used in the intervalometer circuit has nothing in common to the pulse width modulation used in the multichannel radiotelemetry system. The change in the duration of the pulses in the intervalometer is a result of natural changes in the /366

duration of the phases of the cardiac cycle. At the same time, the repetition frequency of the pulses changes, following the rhythm of the heart.

According to the terminology used in the literature on phase analysis of cardiac activity (V. L. Karpman, 1965), the intervalometer measures: the duration of the cardiac cycle (RR), the duration of the phase of asynchronous contraction (ab^1), as well as the quantities bc^1 and ce^1, which, considering the corrections, are equal to the duration of the phases of isometric contraction and the expulsion period. In most cardiological investigations in sports, it is these principal phases which are investigated, since they carry the most important information concerning the contractive function of the heart.

More detailed investigation of the structure of the cardiac contraction is performed using the analog arithmetic device (AD on Figure 105), consisting of operational or decision amplifiers. The arithmetic device allows automatic calculation of phases of the cardiac cycle to be performed according to the following program:

1) phase of asynchronous contraction	$(AC) = ab$
2) phase of isometric contraction	$(IC) = bc + \Delta_1$
3) period of expulsion	$(E) = ce + \Delta_1 - \Delta_2$
4) stress period	$(T) = ab + bc - \Delta_1$
5) mechanical systole	$(S_m) = IC + E$
6) total systole	$(S_o) = S_m + ab$
7) ventricular diastole	$(D) = C - S_o$

In this algorithm, Δ_2 is the duration of the protodiastole, Δ_1 is the time required for propagation of the pulse wave from the heart to the pulse transducer. The values of Δ_1 and Δ_2 are arbitrarily assumed constant and determined from statistical data.

When necessary, we can also calculate the intrasystolic index $ISI = E/S_m$ and the myocardial stress index $MSI = T/S_o$. For this, circuits are included in the computer device which perform the arithmetic operation of division of two variables.

The low output impedance of the amplifiers allows various multichannel recorders to be used for graphic recording of the transient process curves. Convenient and easily readable graphics are produced on broad (up to 30 cm) diaphragm paper by the type EPP-09 automatic electronic potentiometer. /367

[1] ab -- interval between Q and tone I; bc -- interval between tone I and beginning of anacrote of central pulse; ce -- duration of sphygmic wave.

Quantitative evaluation of the results produced requires that all channels of the intervalometer be calibrated using a special pulse generator. The scale of the recording device is graduated in milliseconds.

The transient process curves produced using this system are used to evaluate the "quality" of regulation of the heart contraction directly during muscular work with the test subject moving freely.

Autocorrelation Analysis in Sports Medicine

In recent years, autocorrelation analysis has become ever more broadly used for the investigation of latent periodicity in general and periodicity in the cardiac rhythm in particular (R. M. Bayevskiy and K. I. Zhukov, 1964; V. M. Zatsiorskiy and S. K. Sarsaniya, 1964; D. Zhemaytite, 1965; P. V. Vasil'yeva, 1965; etc.). Although there are many methods in modern mathematics for the determination of latent periodicity, the most preferable method for the study of the cardiac rhythm is autocorrelation analysis. This method was first used for this purpose by Kusachi (1954), Kato (1956), Tominaga (1956), Matsumoto (1962, 1963), who combined it with other mathematical methods. For example, by eliminating the periodic respiratory component from cardiotachograms by the slipping mean method, they showed that slow periodic components (slow waves) are continually present in the cardiac rhythm of rabbits and dogs. In the overwhelming majority of cases these slow waves are masked by respiratory arhythmia and nonsystematic fluctuations ("noise"), so that they are not directly observed on recordings and can be revealed only by mathematical analysis.

The essence of the autocorrelation method is that values of correlation coefficients (in this case they are called series coefficients) between series of numerical values produced experimentally in the same series shifted by 1,2,..., n terms are calculated. For example, if the durations of cardiac cycles in a sequence are (in tenths of a second):

10, 11, 12, 13, 14, 15, 14, 13, 12, 11, 10,

the first series correlation coefficient can be produced by comparing this series with a series shifted by one term. We produce the following series:

10, 11, 12, 13, 14, 15, 14, 13, 12, 11, 10,
10, 11, 12, 13, 14, 15, 14, 13, 12, 11, 10,

As a result, pairs of values of the numbers located one beneath the other are produced: /368

(11, 10), (12, 11), (13, 12), etc. up to pair (10, 11)[1].

These pairs of values are used to calculate the first series correlation coefficient.

The second series coefficient is produced by shifting the series by two terms relative to the first series, i.e. by forming the pairs (12, 10), (13, 11), etc. up to pair (10, 12). The series correlation coefficient essentially indicates the degree of the dependence of the duration of subsequent cardiac cycles on preceding cycles, i.e. makes it possible to answer the following question: if we know that a given cardiac cycle has a certain duration, with what degree of certainty can we judge the length of subsequent cycles -- the first, second, tenth, etc. If all series correlation coefficients are equal to zero, the process is absolutely chaotic and knowledge of the length of a preceding cycle gives us no basis for saying anything concerning the duration of subsequent cycles.

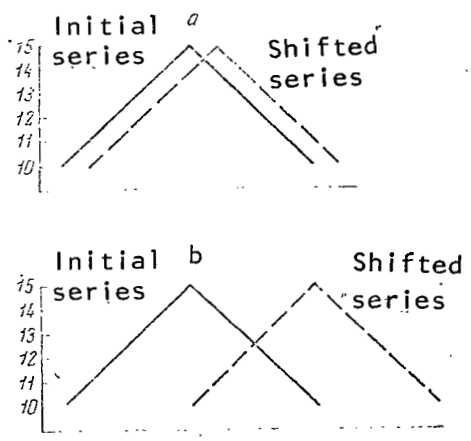


Figure 106. Plan for Determination of Series Correlation Coefficients. a, Shift by one cycle; b, shift by five cycles. Explanation in text.

If there is a periodic component in the process, the following will occur. At first, with small shifts of the series being investigated (Figure 106, a), the high values of preceding cycles will correspond to high values of subsequent cycles, and high positive correlation will obtain. However, as the shift is increased, a moment will occur when the higher values of the initial series correspond to the lower values of subsequent cycles, and the relationship will become negative (Figure 106, b).

Thus, if there is a latent periodic process, values of the series coefficients will decrease to negative quantities, then once more increase.

The graph of values of series correlation coefficients has come to be called an autocorrelogram, and the /369

[1] This example is merely illustrative. Calculation of series correlation coefficients requires considerably larger numbers of observations.

mathematical expression describing the nature of changes of series correlation coefficients is called the autocorrelation function.

Experience in the usage of autocorrelation analysis shows that it is an effective means of revealing latent periodicity even in those cases when the magnitude of the aperiodic perturbations superimposed is quite great.

Some results of autocorrelation analysis for athletes. When the duration of cardiac cycles is recorded using sufficiently precise chronographs, usually considerable scattering is noted. These fluctuations are generally rather chaotic, although in some cases the durations of cardiac cycles depend on the phase of respiration.

It has been established that at least two periodic components are present in the variations in duration of individual cardiac cycles: a) the well known respiratory fluctuation of the cardiac rhythm, synchronous with the respiratory excursions of the thorax (respiratory arhythmia), and b) the so-called slow waves (pulslangwellen, slow rhythms), discovered first by Fleisch and Beckmann (1932). The duration of the slow waves is much greater than the duration of the respiratory cycle.

Whereas respiratory arhythmia is observed relatively frequently, the question is more difficult as concerns the slow waves. Although the fact of existence of low waves has been confirmed repeatedly ((Cerletti, 1946; Matthes, 1951; Penaz, 1957; Wegenmann et al., 1959; E. G. Kyaer-Kingisepp and M. A. Epler, 1960 and 1963; etc.), they are observed, as Penaz noted correctly, only "under particularly favorable conditions."

Attempts to find some regularity in the superficially chaotic fluctuations in cardiac cycle lengths and, in particular, concealed periodicity, have made it possible to produce new data on the mechanisms of regulation of the cardiac rhythm.

For sports medicine, latent periodicity of the cardiac rhythm is interesting as a particular case of the physiological rhythms, which are being given ever greater attention.

The data of autocorrelation analysis of the cardiac rhythm in athletes at rest present an extremely varied picture. With a certain degree of arbitrariness, we have distinguished seven types of autocorrelograms (Figure 107).

Type I autocorrelograms (Figure 107, a) indicate independence of the duration of subsequent cardiac cycles on preceding cycles and absence of aperiodic component. In these situations, the first values of correlation coefficients reach no statistically significant magnitudes (with one percent level of significance). /370

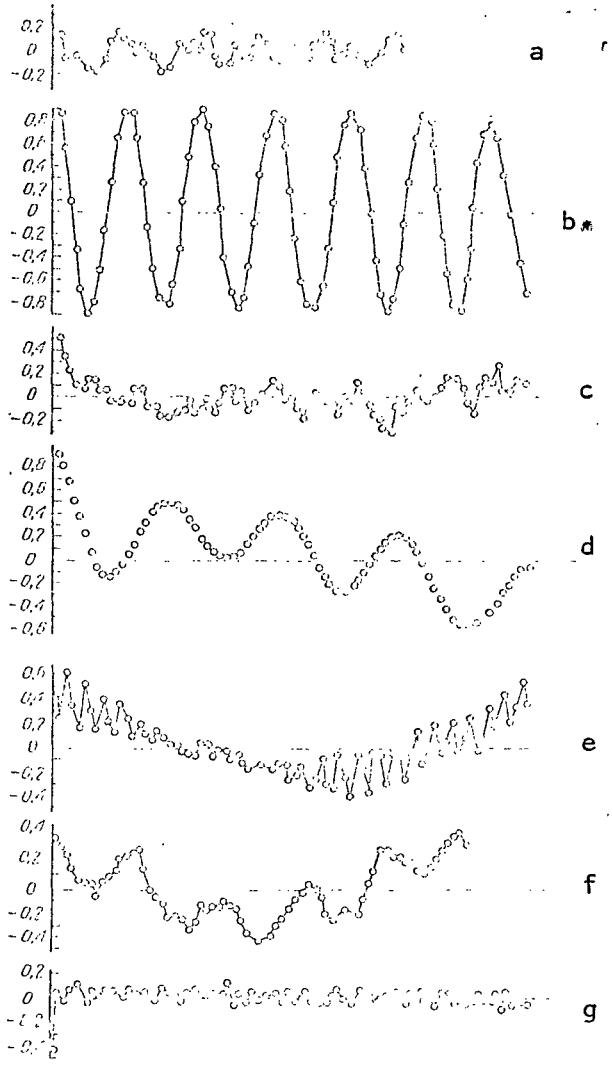


Figure 107. Principal Types of Cardiac Rhythm Autocorrelograms. Explanation in text.

Autocorrelograms of type II (Figure 107, b) are characteristic for respiratory arhythmia: the oscillations are synchronous to respiratory excursions of the thorax.

Autocorrelograms of type III (Figure 107, c) are called "rapidly attenuating." Their values decrease to zero by the second-sixth beat (in 2-6 sec), then do not return to statistically significant magnitudes. This type of correlogram corresponds to weakly expressed periodic and strong aperiodic perturbations ("noise").

Autocorrelograms of type IV (Figure 107, d) correspond to slow periodic components, longer than the respiratory cycle.

Autocorrelograms of type V (Figure 107, e) show the superposition of two "waves." The respiratory frequency waves are superimposed on the slow wave.

Autocorrelograms of type VI (Figure 107, f) show the superposition of two slow waves.

Autocorrelograms of /371
type VIII (Figure 107, g) show the first negative value. This case corresponds to negative dependence between duration of two successive cardiac cycles.

There is particular interest in autocorrelation analysis of the cardiac rhythm during muscular work. Thus, when working on the bicycle ergometer at 400-1500 kg-m/min, correlograms with periodic respiratory frequency disappeared, and the frequency of appearance of slow waves increased.

As a result of a series of special investigations which we performed, it was established that if the test subject breathes at different frequencies, the nature of the autocorrelogram of the cardiac rhythm changes sharply. Thus, at "low" respiratory frequencies (up to approximately 30 per minute), where one respiratory cycle corresponds to more than two cardiac cycles, respiratory arhythmia is clearly seen on the autocorrelogram. The duration of the cardiac cycles changes on a curve which is very nearly sinusoidal.

In cases when one respiratory cycle corresponds to exactly two heartbeats, the duration of the cardiac cycle changes sharply, strictly following the respiratory phases. In these cases, a correlogram with negative value of the first series correlation coefficient arises.

The most interesting results are produced by autocorrelation analysis with "high," arbitrarily fixed respiration frequencies (80-120 times per minute). Here, the respiration frequency exceeds the frequency of heart contractions. In these cases, the oscillograms show a rather chaotic picture and only autocorrelation analysis makes it possible to determine the latent slow waves.

Latent periodicity of the cardiac rhythm changes essentially upon subcutaneous injection of atropine (2 mℓ of 0.1% solution of atropine sulfate). Atropine, as we know, is a cholinergic inhibitor and as such blocks the effect of the vagus nerve on the sinus node.

At the peak of the atropine effect, the amplitude of respiratory variations of the cardiac rhythm is sharply decreased; the nature of the autocorrelogram also changes. After atropinization, slow waves appear more frequently, and may occur even with comparatively slow respiratory rhythms.

Our data indicating that slow waves appear more frequently in cases of muscular work and after the injection of atropine, in combination with reports that autocorrelation analysis can show predominance of slow waves in cases of hypoxia (Sagawa, 1960), emotional stress (in cosmonauts before launching of a rocket -- A. D. Voskresenskiy and M. D. Venttsel', 1965), and also in the human intrauterine foetus (V. L. Karpman et al., personal report indicate to us that sympathetic nervous effects play an important role in the origin of slow waves. /372

In conclusion, we should say a few words concerning the capabilities of the autocorrelation methods of analyzing the cardiac rhythm. Like any other method, autocorrelation analysis has its limitations. Let us note one of these. If we encounter a case in which various chaotic perturbations are superimposed over a periodic process with unchanging frequency, the latent process can be revealed by autocorrelation analysis. The situation becomes considerably more complicated if the frequency of the latent periodic process changes. Then, autocorrelation analysis cannot show anything, even though latent periodicity does occur. It is difficult to assume that the biological processes which, for example, cause the appearance of slow waves, remain unchanged in their frequency characteristics for a long period of time. In

this case, autocorrelation analysis may be fruitless. This is why, although the presence of a periodic component on the autocorrelogram definitely indicates existence of rhythmic fluctuations in the process being investigated, the absence of periodic components on the autocorrelogram should be evaluated more cautiously.

PART III

DISRUPTIONS IN THE ACTIVITY OF THE CARDIOVASCULAR SYSTEMS OF ATHLETES

Problems of the pathology of the heart and vessels in athletes are quite /373
pressing for modern sports medicine. The high volume of training loads, high frequency of competitive stress, the year-round training process -- all of these place high demands on the athlete's organism and particularly on his cardiovascular system. Disruptions in the operation of the circulatory apparatus resulting from various pathological factors are the most frequent causes for failure of athletes of various specialties and levels of qualification. These disruptions may be either nonspecific for the sport involved (for example, disruptions in rhythm, arterial hypertension) or specific, directly related to the sports activity (for example overstress, overtraining).

The third portion of the present monograph is dedicated to a rather broad range of problems of the pathology of the cardiovascular system in athletes. In any case, it discusses the most important and most frequently encountered forms of disruption in the work of the heart.

Particular problems of sports cardiology are the subject of an essay in which certain clinical aspects of sports cardiology are discussed. The points developed in this essay are not used by all sports doctors, although these points can be seriously supported and therefore should be carefully analyzed by a broad range of specialists.

CHAPTER X

SOME CLINICAL PROBLEMS OF SPORTS MEDICINE

ABSTRACT. Factors resulting in disease in athletes are analyzed. These factors include changes in the external medium, improper and ineffective training, and intensive training exercises attempted by persons with insufficient physical conditioning. The relationships between chronic centers of infection, more common in athletes than in non-athletes and cardiac problems are explored. The importance of complete recovery from infectious diseases, dental carries, etc. before resumption of sports training is emphasized.

Sports cardiology today represents a large and interesting chapter both /374
of sports medicine and of contemporary cardiology. Its important sections are: analysis of changes in the arterial pressure, clinical evaluation of specifics of the electrocardiogram of the athlete and disruptions of heart rhythm, as well as investigation of the state represented by overstress of the heart. These problems are briefly discussed in Chapters XI, XII and XIII. Naturally, these problems do not exhaust the entire range of clinical problems in sports cardiology. In these chapters, the approach to the evaluation of changes in the cardiovascular system of persons involved in sports differs from that encountered in the works of other authors (Reindell, Kral, S. P. Letunov), in that the treatment of various changes of the cardiovascular system of the athlete, the presence of which is not denied by anyone today, is performed from the clinical standpoint. Difficulties of this approach include the fact that in sports medicine there are essentially no pathophysiological trends, without which clinical evaluation of changes occurring in the organism of the athlete is quite difficult.

In spite of the fact that the influence of involvement in sports on the state of the cardiovascular system has been studied for some time, there is as yet no single point of view concerning the possibility of pathological changes in the cardiovascular system of the athlete and their clinical evaluation.

In many works both of pathologists and of clinicists, principally performed at the end of the nineteenth century and beginning of the twentieth century, one constant theme was the affirmation of the unfavorable influence of sports activity on the heart. The fact of heart enlargement noted in many /375
athletes was evaluated negatively, and the involvement in sports was used as an attempt to explain the increased incidence of cardiovascular diseases in

many countries. At the same time, certain authors who studied this problem noted the positive influence of involvement in sports on the state of health and on the cardiovascular system in particular.

The term "sports heart" used in the literature was also interpreted differently. While some authors used this term with a definite pathological sense, others did not acknowledge the correctness of the very existence of the term and spoke rather of the "heart of the athlete," which had a considerable capacity for adaptation to great physical loads.

The extreme opinion that any "sports heart" is pathological was countered by the diametrically opposite point of view, according to which a healthy heart could withstand any physical loads without problems, so that if any pathological changes were detected in the athlete's heart, it indicated that it had been damaged before the beginning of the sports activities. These two extreme points of view, as investigations in recent years have shown, cannot be accepted.

The most nearly accurate conception is that of G. F. Lang (1938), who wrote of the physiological and pathological "sports heart" or, as he said, the sports circulatory system.

A proper clinical evaluation of data produced in a study of the cardiovascular system of the athlete is the basis not only for the solution of the question of the state of training, but also for early diagnosis of various prepathological and pathological conditions.

Still, the clinical manifestations and mechanism of formation of various disruptions in the state of health of the athlete resulting from various causes, including improperly organized sports activity, inefficient training methods, improper regime and medical supervision remain little studied in sports medicine (S. P. Letunov, 1963). This is explained, in our opinion, by the considerable separation of sports doctors from clinical practice, as well as by the quite unjustified fear of discrediting sports. Neither of these reasons holds up to criticism, since it is obvious that the sports doctor should be a qualified, clinically literate doctor, always at the contemporary level of development of medical science. As concerns possible discreditation of sports, if the injuries and traumas in sports, which are the subject of the study of sports traumatology, do not discredit sports, it is not clear just why damage to the internal organs should be kept quiet. Both of these /376
aspects must be studied in order to develop means for preventing their occurrence, for their early diagnosis and treatment, since neither injuries nor diseases should occur in sports.

There is no doubt that sports and health are synonymous, and that involvement in sports cannot and should not be a reason for the formation of any diseases of the internal organs. However, this does require certain conditions, which are far from always fulfilled, and to some extent cannot be fulfilled; therefore, athletes still become sick.

The most important reason for the formation of diseases in athletes might consist of changing conditions of the external medium, epidemic outbursts (grippe, etc.), the influence of which cannot be avoided. Therefore, athletes do fall ill with grippe, pneumonia, anginae, etc., and it might be assumed that the high functional state of the athlete's organism determines the well known differences in the course of these "ordinary" diseases and consequently requires different treatment. This problem has been insufficiently studied and requires special analysis.

A second factor which might cause diseases of the internal organs and in particular of the athlete's heart is improper and inefficient training, facilitating the formation of overloads and overstresses (N. D. Grayevskaya, Ye. V. Kukolevskaya, Ya. A. Mel'nikov, 1963; N. D. Grayevskaya, 1964).

It should immediately be stated that "overloads" are not necessarily caused by great and intensive physical loadings. An overload or overstress should be understood to mean a state arising upon the performance of a physical load exceeding the capabilities of the given person at the moment it is performed. Thus, the same physical load may be an overload or an insufficient load not only for two different people, but for the same person depending on his state of training at the moment.

This same factor has an effect on diseases arising in an absolutely healthy athlete as a result of various disruptions in the day's schedule, feeding, as well as bad habits which are still rather widespread, unfortunately.

Finally, a third reason for diseases in athletes is the fact that persons with various health problems are allowed to take part in intensive training exercises. This is a result either of underestimation of the defects which the doctor has found in the state of health or insufficiently precise diagnosis. These defects in the state of health appear or become worse when heavy training loads are attempted.

Thus, if an athlete falls ill, this may be a result of the influence of /377
external unfavorable factors, of involvement in sports by a physically unprepared person, of overloading or overstress resulting from ineffective utilization of training loads and disruption of the training regime. All of these factors may be the basis for formation and development of various types of damage to the athlete's heart.

G. F. Lang stated in 1938 that excessive muscular stresses, both acute onetime stresses and systematic repeated stresses, sooner or later cause pathological changes in the myocardium, as a result of which circulatory deficiency may even arise. The basis of the development of the changes in the myocardium described by G. F. Lang is the relative deficiency of its blood supply which occurs in case of considerable physical stress. As a result of this, the intensive chemical processes occurring in the myocardium during active muscular work are not supplied with sufficient oxygen. This is the reason for the appearance of the phenomenon of fatigue, followed by

overfatigue of the heart, which are based on various biochemical changes in the myocardium, and in this stage are still reversible. Then, if the intensive muscular activity continues, the changes become irreversible and lead to necrosis of the muscular fibers in the myocardium. This may lead to sudden death as a result of ventricular fibrillation occurring upon sharp disruption of the functions of conduction, excitation, and also as a result fo sharp biochemical changes in the myocardium. If this does not occur, at points of necrosis of the muscular fibers, primarily in the left ventricle, connecting scar tissue appears, i.e. cardiosclerosis occurs. This type of change, limiting the working ability of the heart, creates the conditions for the development of chronic cardiac deficiency.

The plan of the development of cardiac deficiency described by G. F. Lang as a result of physical overstress has not lost its significance at the present time. However, as will be seen from the following, it has been slightly clarified on the basis of the latest scientific data in the area of cardiology concerning primarily the biochemistry of the myocardium.

The statement of G. F. Lang (1938) that the transition from physiological phenomena in the "sports heart" to pathological phenomena and the development of cardiac deficiency generally occur gradually and unnoticeably for the athlete is quite important and retains its strength even today. It is obvious from this that simply feeling well can in no case be considered a criterion for evaluation of high functional state of the athlete's heart, nor can high sports results. Therefore, we must agree fully with S. P. Letunov (1949), V. N. Kovalenko (1959), N. D. Grayevskaya (1964) and others that high sports results have only relative value for the diagnosis of the state of training. Unfortunately, these two parameters (good feeling and sports achievements) are frequently decisive in evaluating the state of health of the athlete. /378

Of course, absolute health is generally combined with good feeling, although the reverse is far from always true. The tremendous compensatory capabilities of the young organism frequently mask various types of organic damage to the heart to the extent that the patient feels excellent and manifests a high state of training. However, with intensive physical loads, the heart damage already present is generally worsened and sooner or later appears as clear disruptions in the function. It is obvious from this that a high level of training, like good feeling, can be evaluated positively only if it is based on absolute health, i.e. complete absence of any deviations in the state of health.

A high state of training with various defects in the state of health cannot and should not serve as a basis for granting of permission to so-called heavy sports. If we add to this the frequent cases of dissimulation among athletes, it becomes obvious that one should not base any conclusions in evaluating the state of health on the feelings of the athlete. This evaluation must be based on careful clinical investigation using modern diagnostic apparatus. In this respect, there is considerable interest in a

a work by L. V. Shapkova (1965) who, working with skiers in the lower classifications, showed that a sense of readiness for work corresponded with the objective data only in 55.1% of the cases. Sometimes, this sensation arises before the objective evidence indicates readiness, sometimes later.

Thus, the first and principal task of the doctor is precise diagnosis of the state of the cardiovascular system and especially manifestation of organic damage to the heart. In other words, as concerns the cardiovascular system, the diagnosis: "healthy" must be given. This diagnosis is very difficult and complex, primarily since the diagnosis of muscular changes in the heart and nascent valve damage, particularly damage to the mitral valve, is still far from perfection.

It is not by chance that ever newer methods of investigation are appearing in the arsenal of diagnostic methods available to the doctor. The unnoticeability of the transition of the "physiological sports heart" to a pathological state greatly complicates diagnosis. A feeling of sickness will occur only at a certain level of disruption of the activity of the organism, whereas the doctor should recognize these disruptions earlier (I. A. Kassir- /379
skiy, 1964). Therefore, one of the principal tasks of the sports doctor is early diagnosis of such disruptions in the state of health of the athlete, disruptions which he does not feel and which do not yet influence his sports results. Timely recognition and treatment of these changes will prevent their development, which could lead to irreversible changes and inability to continue sports activity if the proper steps were not taken (S. P. Letunov, 1949; A. G. Dembo, 1958; N. D. Grayevskaya, 1964; etc.).

In recent times, the diagnosis of so-called functional changes is being made quite easily in sports medicine. The interpretation of changes of the cardiovascular system as functional does not eliminate the necessity of diagnosing these changes, which is usually not done. Also, at the present time the problem of the functional and organic changes is being essentially restudied. It is more proper to speak of reversibility and irreversibility of various pathological states, which is hardly identical to the concept of functional and organic.

N. N. Savitskiy (1964) correctly indicates that there is no reason to divide diseases into organic and functional. This division is a result of the organopathological trend in clinical medicine and eliminates the material substrate from a disease. The presence or absence of certain morphological changes hardly answers the question of the origin or of the course of a disease. The reason for disruptions in the function of any organ might be extensive anatomical changes or changes at the molecular level which cannot be determined at the present time due to the imperfection of our investigatory methods.

We can see from the above that the sports doctor should not limit himself to a statement that the various changes he has detected in an athlete are "functional" in nature, and call them therefore "deviations." In each case, the doctor should attempt to determine the genesis of these changes

and make his diagnosis, since even functional diseases should have a concrete and generally recognized significance. Incidentally, this is frequently not done.

In analyzing the principal factors causing the formation of diseases in athletes, which we mentioned above, we must discuss certain problems of significance from the standpoint of sports cardiology. From this point of view, in analyzing the first group of factors we should briefly discuss the centers of chronic infection, particularly chronic tonsillitis.

Chronic centers of infection in athletes should be given considerable significance, since with the great physical loads which they carry, the possibility of a negative influence of these centers increases sharply.

Chronic sources of infection must include chronic cholecystitis, more /380
frequently encountered in athletes than in nonathletes (A. G. Dembo, 1965), teeth with caries and chronic tonsillitis. Each of these sources of infection may cause heart damage.

In clinical practice, changes to the heart arising in patients suffering from chronic cholecystitis are well known (A. L. Myasnikov, V. A. Galkin, 1963). The sports medical literature contains descriptions of interstitial Abramov-Fidler type myocarditis, arising in persons with chronic infections in response to slight physical loads, sometimes resulting in death (Hochrein, Schlicher, Frey, etc.).

V. I. Kovalenko (1959) presents an example with athlete Kh, rated in the first classification as a free-style wrestler. In spite of angina accompanied by a high temperature, he continued intensive training and took part in competition. He was stricken with acute myocarditis and complete transverse dissociation with a pulse frequency of 10-12 beats per minute and Morgan-Adams-Stokes attacks. The athlete was subsequently forced to discontinue his involvement with sports.

Similar examples can be found frequently in the literature (Wolf, 1957; Prokop, 1958; Hantschel, Frank, 1958; Gedlicka, 1954; etc.).

Chronic tonsillitis is particularly significant in sports cardiology. According to the data of many authors (T. M. Yerokhina, 1958; N. D. Grayevskaya, Ye. V. Kukolevskaya and Ya. A. Mel'nikov, 1959; V. N. Kovalenko, 1959; F. A. Iordanskaya, 1960; L. I. Stogova, 1961; etc.), the percentage of athletes with chronic tonsillitis varies from 5 to 48 depending on the make-up of the group investigated. This percentage is considerably higher than the number of persons with chronic tonsillitis among nonathletes (A. G. Dembo, 1958; T. M. Yerokhina, 1958; etc.). The reasons for this, as for the large percentage of persons with dental caries among athletes, cannot be considered quite clear. The possibility of the influence of mouth breathing during training and competition, which excludes the protective role of respiration through the nose, cannot be excluded.

This makes obvious the great significance in sports cardiology of the so-called tonsil-cardiac syndrome (B. A. Yegorov, I. K. Kireyev, 1964; V. M. Kovalevich, Ye. M. Kuperenko, 1964; E. R. Agababova, Z. A. Val'dman and A. L. Serkin, 1964; etc.). This syndrome means the combination of chronic tonsillitis with various mechanisms of changes in the myocardium of nonrheumatic etiology. These combinations are encountered in 10-15% of all patients suffering from chronic tonsillitis (A. A. Kedrov, 1963). Clinically, tonsilogenic intoxication may manifest itself in various ways -- pains in the area of the heart, rapid fatigue, sometimes vague pains in the joints, an inclination to tachycardia disruption of the heart rhythm, subfebrility, hypotension, etc. In patients with chronic tonsillitis, a reduction in the /381
phagocytic capacity of neutrophils and the bactericidic properties of the skin is noted. However, all these symptoms are inconstant and are combined in various ways.

Changes in the electrocardiogram in many cases allow us to note dystrophic changes in the myocardium or nuclear myocarditis resulting in cardiosclerosis. Cases of tonsilogenic interstitial myocarditis have been described (S. A. Gilyarevskiy and S. O. Androsova, 1964, etc.).

Extended tonsilogenic intoxication may lead to disruption of the functions of automatism of excitability and conductivity. Also, stenocardia resulting from tonsilogenic intoxication has been described (I. D. Mishenin, 1965).

The changes occurring in the cardiac muscle during the tonsil-cardiac syndrome are histologically confirmed. Usually, nuclear interstitial myocarditis is noted, along with necrosis of the muscular fibers, myloysis, i.e. manifestations of nonspecific myocarditis.

It is thought that chronic tonsillitis may be accompanied by neurovegetative disfunction with damage to various organs and systems. These disruptions may also be accompanied by changes in the activity of reticular formation (R. G. Mezhebovskiy, 1963).

It is important to state that the changes in the heart in chronic tonsillitis arise independently, regardless of whether it is considered compensatory or decompensatory. According to the data of I. K. Kireyev (1964), changes in the electrocardiogram, as well as changes in the vascular tonus, were encountered in 75% of patients suffering from compensatory and decompensatory tonsillitis. Thus, the concept of compensation in chronic tonsillitis is relative and, of course, cannot solve the problem of the necessity of tonsillectomies.

In sports medicine, the basis for the treatment of chronic tonsillitis should be surgical operation and indications toward surgery should be made by the therapist. The effectiveness of conservative treatment is not great, and in 44% of cases, intensification occurs.

According to the data of many authors (N. A. Bobrovskaya and A. S. Sych, 1952; I. A. Kireyev, 1964; V. A. Yegorov, V. T. Yermolayev and Ye. A. Borshchevskaya; Weicher, Holz, Leonaedelli, 1964), after tonsillectomies, the electrocardiogram normalizes and complaints disappear in 80% of cases. Most authors believe that timely performance of tonsillectomies assures reverse development of changes in the cardiac muscle. If reverse development does not occur, these changes should be considered irreversible (S. B. Trakhtenberg, S. F. Oleynik and Ye. M. Kucherenko, etc.). Such results were observed in 18.8% of cases (I. M. Potashnik, A. M. Nogaller, 1964), while in 0.9% of cases a worsening even occurred.

The data presented indicate the significance of centers of chronic infection in the formation of various types of damage to the myocardium. Centers of infection are even more significant if considerable physical loadings are applied, in particular with involvement in sports, as will be discussed below. /382

Going over to the second factor which may result in diseases in athletes, i.e. overstress and overloading, arising as a result of improper and ineffective training, failure to observe the training regime, etc., we should state first of all that this group of diseases in athletes is the largest group. We are not concerned here with the usage of high training loads in general, without which it is impossible to imagine modern sports, but rather of inefficient utilization of large loads, when they become too great. This depends on the degree of training of the organism.

A loading applied to an organism insufficiently prepared for it, particularly if this is combined with inefficient work and daily living regimes, bad habits and centers of chronic infection, may cause various types of damage to the cardiovascular system, not only reducing the working ability, but, if proper measures are not taken, completely disrupting the organisms of the athlete.

Effective utilization of high training loads by an absolutely healthy athlete prepared for performance of the loads, cannot result in sickness.

The significance of inefficient utilization of high physical loads has been clearly shown experimentally by P. Z. Gudz', O. V. Kachorovskaya and G. A. Kashpurovskiy (1964).

The experimentaldata of these authors confirm fully the idea that large loads performed efficiently and after the proper preparation produce favorable changes in the cardiac muscle. On the other hand, these loads, if used without the proper preparation or used ineffectively, lead to manifest pathological changes in the myocardium. The authors also found that changes in the myocardium which were subject to reverse development upon resting occur again quite easily if the physical loading is renewed. In other words, a heart which has undergone changes as a result of extreme physical loading is more sensitive to physical efforts in the future. Also, at a certain stage in the changes in the myocardium resulting from physical loading,

reverse development may become impossible. These experimental data are of important significance for an understanding of a number of pathological changes in the athlete's heart, arising as a result of extremal and inefficient loadings. Analysis of the diseases of leading athletes performed by N. D. Grayevskaya, Ye. V. Kukolevskaya and Ya. A. Mel'nikov (1963) showed /383
that the reasons for the formation of diseases related to sports are distributed as follows: in 26% -- forced training with maximum loadings without sufficient general physical training, in 18% -- one-sided, narrowly specialized training with very slight utilization of general physical training, in 11% -- sharp transition from low training loads to intensive loads, i.e. no gradual increase in loadings, in 12% -- participation in training exercises or competition while ill or immediately after illness, in 10% -- insufficient rest between training exercises, in 5% -- training without sufficient consideration of young age, in 10% -- improper method of construction of drills and in 8% -- improper conditions in which training was performed, disruption of training regime, etc.

The favorable influence of overloadings has been shown in the works of many authors (A. G. Dembo, 1961, 1963, 1964; L. I. Stogova, 1963; F. A. Iordanskaya, 1964; S. P. Letunov, 1957; etc.).

It is obvious at the present time that involvement in contemporary sports can be permitted only for absolutely healthy persons with no pathological deviations in the state of health. Even the most insignificant deviations in the state of health, not preventing physical culture exercises, may be aggravated by the maximal loads used in training, as a result of which diseases can arise essentially limiting the sports ability and sports life of the athlete (A. G. Dembo, 1959; Ya. A. Mel'nikov, 1962; etc.).

It is obvious therefore that, since sports requires absolute health, the term "practically healthy" widely used in clinical practice cannot be used for athletes.

TABLE 30. MORTALITY FROM CARDIOVASCULAR DISEASES BY AGES (IN PERCENT) FROM 1913 TO 1948 (AFTER SCHMIDT)

Age, years	Athletes	Nonathletes
25-34	8.6	1.85
35-44	8.62	4.36
45-54	10.75	9.49
55-64	25.87	19.99
65-74	29.12	36.55
75 and older	17.04	27.76

Table 30 shows foreign data on the mortality of athletes from cardiovascular diseases by ages in comparison with nonathletes (Schmidt). As we can see from Table 30, the death rate among nonathletes is lower up to 64 years of age than among athletes. This is explained not by the fact that sports causes cardiovascular system diseases, but rather from the absence of the necessary medical control, as a result of which persons are allowed to become involved in sports without preliminary medical examination or with a superficial examination, which is particularly true of foreign sports. The intensive training loads encountered by athletes, if cardiovascular system diseases are present, represent one of the greatest reasons for mortality of athletes abroad. /384

In the light of these data, it is incorrect to think that with certain pathological states of the cardiovascular system, involvement in sports is not only permissible but even to be recommended. The basis for this concept is apparently the fact that some persons with clear pathology of the cardiovascular system, in particular mitral valve deficiency, have not only set records, but have become champions and record holders of the world.

At the present time, it is well known (V. I. Kovalenko, 1959; A. G. Dembo, Zh. A. Teslenko and A. M. Tyurin, 1965; etc.) that high physical working ability with clear pathological changes in the muscle or valves of the heart is achieved by tremendous stress of the compensatory mechanisms and sooner or later the resulting pathological changes always appear, although in a much more threatening, frequently irreparable form. Sometimes this ends in tragedy. This possibility is unfortnuately frequently forgotten by doctors, who allow persons with pathology of the cardiovascular system to become involved in sports.

One factor facilitating the development of the idea of the permissibility of sports involvement by persons with mitral valve deficiency is apparently the hyperdiagnosis of this problem. We know that isolated mitral valve deficiency is encountered quite rarely. However, this diagnosis is widely made in sports medicine. A functional systolic noise frequently heard in athletes is evaluated as an organic problem, i.e. as a result of mitral valve deficiency.

Insufficient familiarity with heart damage in athletes has led to the fact that frequently all deviations in the state of health, in particular in the cardiovascular system, is considered to be a "peculiarity" of the athlete solely on the basis of the fact that the athlete has made no complaints and has set records. As a result of this approach to "peculiarities" of the athlete's heart, all dull tones of the heart, sometimes a result of cardiosclerosis, are included in this category; systolic noises, frequently without sufficient clinical analysis, are considered to be functional; with manifest hypertrophy of the left ventricle, no differential diagnosis is performed to distinguish between physiological and pathological hypertrophy; complaints of pains in the area of the heart are also not given their proper significance -- the possibility of early atherosclerosis of the coronary vessels or changes of the myocardium of noncoronary origin is not considered, etc. /385

It is clear at the present time that in all these cases, before calling these "peculiarities," it is necessary to perform careful clinical investigation using modern diagnostic apparatus, and most importantly we must always recall the possibilities of interpreting these changes not only as peculiarities of the athlete, but also as manifestations of pathological changes in the cardiovascular system.

All of this relates to the large number of various "peculiarities" of the electrocardiogram of athletes described in the literature, and looked upon as variants of the norm. Also, careful clinical analysis performed by a number of athletes for various changes in the electrocardiogram and other deviations looked upon as "peculiarities" of the sports heart, have shown that some of them doubtless are pathological (A. G. Dembo, 1961; L. A. Butchenko, 1963; R. D. Dibner, 1964; M. L. Proektor, 1963; S. P. Letunov and R. S. Motylyanskaya, 1951; Zh. A. Teslenko, 1963; V. N. Kovalenko, 1959; Klemola et al., 1960; etc.).

Suffice it to say that the percentage of athletes with various deviations from the normal electrocardiogram has increased in the last 5-7 years by more than two times (L. A. Butchenko, 1959; M. L. Proektor, 1964). Of course, we are concerned here with comparable changes, since over the past five years many changes in the electrocardiogram have come to be interpreted differently, and the techniques of recording of curves have also changed. For example, the large percentage of low-voltage electrocardiograms in 1959 and its reduction in 1964 are explained by the fact that the EKP-4 apparatus used to record electrocardiograms in 1959 passes frequencies up to 50 Hz, whereas the devices used in 1963-1964 pass frequencies up to 150-800 Hz.

However, in addition to these quantitative changes, qualitative changes in the "peculiarities" of the electrocardiograms of athletes have occurred. This is particularly clearly seen in changes of the electrocardiogram evaluated as manifestations of the syndrome of cardiac overstress, as well as disruptions in the rhythm. This type of analysis of these changes is presented in the following chapters of this section. We need indicate only that an increase in the number of athletes with disruptions of automatism has /386
been noted (from 2.3% in 1957 to 4.4% in 1964), while acute, sinus bradycardia has decreased (from 1.55 to 0.9%). Various disruptions in the atrioventricular conductivity have also increased from 0.8 to 3.48% in 1964. Among the group of arhythmias resulting from disruptions in conductivity, the percentage of athletes with the WPW syndrome has slightly increased (from 0.4 to 0.6%). The number of athletes with changes in the final portion of the ventricular complex, interpreted as the cardiac overstress syndrome, has increased. All of these changes must be related to the significant increase both in the volume and intensity of training loads which has occurred in recent years. This factor alone indicates the necessity of careful clinical examination of each case of deviation from the normal electrocardiogram, before considering these deviations "peculiarities" of the athlete.

These are a few of the clinical problems of sports cardiology. The necessity of further active work in this area is obvious. The materials presented in this chapter, without pretending to be undebatable or to cover the problem completely, can be used as a starting point for development of this important section of sports medicine.

CHAPTER XI

CHANGES IN THE ARTERIAL PRESSURE OF ATHLETES

ABSTRACT. Data available from the rather skimpy investigations into the arterial pressure of athletes are presented. It is pointed out that although arterial pressure has been measured for decades, accurate methods capable of providing reproducible results have only recently become available, and the interpretation of the results produced also varies widely. Individual differential diagnosis most be performed in all cases of abnormal arterial pressure, either high or low.

The importance of the problem of proper clinical evaluation of changes /387
in the arterial pressure of athletes is beyond doubt. We are concerned not only with the possibilities of involvement in sports for persons with different levels of arterial pressure, which is of great practical significance, but also with the manifestation and clinical evaluation of early phases and stages of hyper- and hypotension.

Unfortunately, there is no clear and unified opinion concerning the evaluation of changes in arterial pressure in sports medicine, either as concerns an increase or a decrease in this pressure. For example, hypotension, which a number of authors now evaluate as one of the three classical characteristics of high states of training, in many cases may be a manifestation of disease. On the other hand, an increase in arterial pressure requires differential diagnostic analysis in each individual case. Also, although the method of measurement of arterial pressure has long been widely used by sports doctors, it has not been clear until recently which level is characteristic for persons involved in sports. G. F. Lang (1950), A. L. Myasnikov (1954) and others have considered that both physical labor and sports, if not excessive, facilitate a decrease in the arterial pressure. However, this opinion has not been clearly confirmed, based on massive measurement of the arterial pressure of athletes.

In spite of the fact that massive measurements of arterial pressure have been performed in the USSR on a broad, planned and systematic scale for various groups of the population, professions, etc., this type of investiga- /388
tion has not been performed among athletes for a long time. The works dedicated to this problem have been primarily concerned with the influence of

various intensities of training or functional tests on the level of arterial pressure.

Data on the number of athletes with deviations of arterial pressure from the standard values presented by various authors have been exceptionally inconsistent. At the present time, the norms given by G. F. Lang are considered correct -- 100-139 mm Hg for the maximum and 60-89 mm Hg for the minimum arterial pressure. The correctness of these figures for persons up to 39 years of age has been confirmed by a number of works by the members of the Institute of Therapy of the Academy of Medical Sciences USSR and other authors (A. K. Fedorova, 1955; I. K. Belyayeva, 1955; G. L. Spivak, 1955; V. A. Khlyupin, 1957; etc.). In studies of the arterial pressure of athletes, one should base oneself on these generally accepted figures, as has been done in the works of recent years of N. I. Volnov (1958), L. I. Zharikov (1962, 1963), V. N. Kovalenko (1959), S. P. Letunov and R. Ye. Motylyanskaya (1951), etc.

TABLE 31. COMPARATIVE DATA ON THE ARTERIAL PRESSURE (PERCENTAGES) OF ATHLETES

Arterial pressure, mm Hg	S. P. Letunov and R. Ye. Motylyanskaya, 1951 (1000 persons)	N. I. Volnov, 1958 (5965 persons)
Maximum		
up 99	12.2	3.6
100-119	56.2	56.4(from 100-109-19.8; 110-119-36.6)
120-129	13.8	26.4
130 and higher	11.8	13.6
Minimum		
up 59	11.0	8.2
60-79	64.1	80.1(from 60-69-45.1; from 70-79-35)
80-89	18.5	10.1
90 and higher	6.4	1.6

The problem of clinical evaluation of increased and decreased arterial pressure with involvement in sports cannot be solved without a clear idea of its general level in athletes (Table 31).

We can see from Table 31 that in seven years numerous measurements in the level of arterial pressure were made. The percentage of athletes with

low maximum pressure was decreased over this period of time, while the percentage with high maximum arterial pressure slightly increased (as well as the percentage with pressures at the upper limit of the norm). As concerns the minimum pressure, a slight decrease in the percentage of athletes with low figures, as well as that with figures higher than the normal level was noted, and the percentage of persons with pressures at the lower boundary of the norm was increased. /389

As was indicated above, the question of "sports hypotension" as an indicator of the state of training must be re-analyzed. This is confirmed by the data presented in Table 32, from which we can clearly see that athletes with decreased pressure are encountered almost as frequently as athletes with increased pressure. The idea that hypotension is characteristic of athletes apparently arose since they doubtless have a tendency to decreased arterial pressure, although the figures should not go beyond the limits of the normal level.

TABLE 32. FREQUENCY OF INCREASE OF ARTERIAL PRESSURE IN ATHLETES AND IN PERSONS NOT INVOLVED IN SPORTS BY AGES (IN PERCENT)

Age of test subjects, years	Athletes (data of N. I. Volnov)	Population of Moscow (data of Ye. P. Fedorova)
16-19	10.7	2.4
20-24	11.8	4.1
25-29	15.1	5.4
30-39	17.1	7.1
40-49	24.7	15.4

The data of N. I. Volnov et al. also allow us to affirm that involvement in sports itself is not a factor causing an increase in arterial pressure. The problem is that involvement in sports produces more than the normal number of factors which, acting on persons with increased excitability of the vasomotor centers, result in a tendency to increased arterial pressure.

The problem of the clinical evaluation of changes in pressure in athletes and the behavior of the doctor when they occur is great and complex. Many aspects of this problem cannot be considered finally solved. However, the pressing nature and practical significance of proper evaluation of hypo- and hypertension are beyond doubt.

Increased Arterial Pressure in Athletes

It is well known that an increase in arterial pressure usually, particularly in young persons, occurs at random. Since these increases are frequently transient and are noted but once, they are frequently not given

the significance which they should receive. We should note here that this has been stated by one of the leading therapists of today, who studied hypertension, G. F. Lang (1950): "It is very difficult to determine the boundary between physiological and pathological states as concerns the first stages of hypertension. The reactivity of the vasomotor apparatus differs in different people, and may vary in an individual during his life depending on a number of internal and external factors; in many persons this reactivity is increased, and this increased reactivity is accompanied by a tendency toward increased arterial pressure."

As we know, the persons found to have high arterial pressure in massive /390
investigations make up a heterogeneous group. In addition to those suffering from hypertension, this group contains persons who are merely predisposed to high blood pressure. Of course, athletes are no exception in this respect.

Some confirmation of the heterogeneity of the group of athletes with high blood pressure can be found in the data of S. P. Letunov and R. Ye. Motylyanskaya published in 1951, according to which sports training causes the blood pressure to normalize in 41% of athletes, to increase in 41% and causes no change in 18%. Although the authors do not explain this, the heterogeneity of the group of athletes with hypertension in their reaction to sports training is obvious. It is also obviously necessary to perform differential diagnosis and proper clinical evaluation of this hypertension.

The data of various authors on the increases in pressure in athletes diverge. Thus, L. G. Serkin (1937-1938) noted only 2%, S. P. Letunov and R. Ye. Motylyanskaya (1951) noted 11.8%, while Moga (1958) noted 6-8% of the athletes studied with high arterial pressure.

Recent works, performed both in our laboratory (N. I. Volnov, 1958; L. I. Zharikov, 1962, 1963; M. Ya. Levin, 1965) and by other authors (V. N. Kovalenko, 1959, etc.), have clarified this problem somewhat.

The data produced at the present time, based on an investigation of the blood pressure of many thousands of athletes of various sports qualifications, ranking, etc., have shown that athletes with high blood pressure make up 11-14% of the total. Among these athletes were people both with constant and with onetime changes in arterial pressure. Also, increases or decreases of the minimum pressure alone, maximum pressure alone or minimum and maximum arterial pressure together were taken into consideration.

First of all, it must be noted that the percentage of persons with high pressure among athletes is greater than among persons not involved in sports. Thus, in comparing the data of N. I. Volnov (15,000 athletes) and Ye. P. Fedorova (40,000 residents of Moscow), it was found that whereas among the population the percentage is 9.9, among athletes it reaches 11.4, and is even greater according to the data of other authors.

It should be noted in all fairness that the data of various authors indicates percentages of persons with high blood pressure among young people

of between 1.7 and 17.3, or even up to 23%. Still, the data of Ye. P. Fedorova seem to us to be most convincing. If we compare the data of N. I. Volnov with those of Ye. P. Fedorova as to ages, we find that up to 49 years in each age group the percentage of athletes with high blood pressure is higher (Table 32).

However, among the older age groups, the population of Moscow shows a sharply increased percentage of persons with high blood pressure.

The sports specialization has a tremendous significance as concerns the frequency of high blood pressure (Table 33).

TABLE 33. FREQUENCY OF OCCURRENCE OF HIGH ARTERIAL PRESSURE IN ATHLETES AS A FUNCTION OF SPORTS SPECIALTY (DATA OF N. I. VOLNOV)

Type of sports	Percentage	Type of sports	Percentage
heavy athletics	21.2	light athletics	10.6
football	16.6	bicycling	9.7
volleyball	15.6	boxing	9.6
skating	14.2	basketball	9.5
rowing	13.6	swimming	9.1
wrestling	12.6	gymnastics	8.4
skiing	11.6		

The relatively low percentage of persons with high blood pressure among boxers, where we would expect to find a much higher percentage, is explained by the fact that the extremely high demands placed on the organism of the athlete by this type of sport do not allow the sports activity to be continued after even the slightest deviations in the state of health are detected. The confirmation of this "cut-off" is the fact that the experience of boxers with high pressure is low -- only one percent of the athletes had over 10 years experience (N. I. Volnov, 1958).

The significance of the degree of nervous stress in the formation of hypertension was clearly confirmed by L. I. Zharikov (1963), who showed that among student athletes the percentage of persons with high arterial pressure /392 was 11.6, while among athletes in other professions the percentage was 7-8.

The data of L. I. Zharikov correspond completely to the literature data and allow us to consider it expedient to decrease the intensity of sports training during examination time significantly (A. G. Dembo, T. P. Fanagorskaya).

Our attention is drawn by the decrease in the percentage of athletes with high blood pressure as the sports classification increases (Table 34). It might be assumed that this depends on two factors: first of all, the elimination of persons with hypertension as their sports mastery increases,

and also the normalization of arterial pressure resulting from properly and efficiently organized training (N. I. Volnov, L. I. Zharikov, etc.).

TABLE 34. DISTRIBUTION OF ATHLETES WITH HIGH ARTERIAL PRESSURE ACCORDING TO SPORTS MASTERY (DATA OF N. I. VOLNOV)

Sports qualification	Masters of sports and first class	Second class	Third class	Novice	Unranked
percentage found	9.9	11.5	12.5	10.0	13.4

These are the basic data available on athletes with high arterial pressure. It should be added that high pressure is noted three times as frequently in male athletes as in female.

The problem of the etiology and pathogenesis of hypertension has been broadly and fully studied in the clinical literature (G. F. Lang, A. L. Myasnikov, etc.), which makes it unnecessary to repeat this information at this time.

However, before going over to the problem of the causes and clinical evaluation of high blood pressure in athletes, we should note the classifications of hypertension existing at the present time.

The founder of the contemporary study of hypertension, G. F. Lang, considered that high arterial pressure is not always a manifestation of hypertension in its various stages. It may be observed in persons with high reactivity of the vasomotor centers, whom G. F. Lang divided into hyperreactors and prehypertensives, and considered healthy persons predisposed to hypertension. The classification of A. L. Myasnikov retains the same distinction with the single difference that the prehypertensives are included /393 in stage I of hypertension.

The hyperreactors and prehypertensives are persons with increased reactivity of the vasomotor apparatus, who react with inadequate increases in pressure both to various emotional factors and to physical stress. These persons are not ill and if the proper conditions are maintained they may not get hypertension. On the other hand, they do fall ill with hypertension more easily than others.

This division is generally accepted and important in principle primarily because early establishment of hyperreactivity or the initial stages of hypertension and performance of the proper measures may prevent or delay the development of hypertension.

In a collection of works of the Central Scientific Research Institute of Physical Culture for 1958, a classification of hypertensive states was

published, suggested by S. P. Letunov for athletes. He divides hypertensive states in athletes into four groups: 1) stage I of hypertension; 2) hypertension resulting from physical stress; 3) juvenile increase in arterial pressure; 4) hypertension resulting from emotional lability.

This classification calls forth considerable debate. It is well known that juvenile hypertension does not exist as a specific pathological form. In other words, both hyperreaction and prehypertensive states, as well as hypertension itself in various stages are encountered in young persons. As concerns the increase in arterial pressure resulting from physical overstress and emotional lability, the inclusion of all possible etiological factors causing hypertension in a classification system is impossible. These factors are many, and cannot be used to construct a classification. This sort of attempt has been made (V. M. Kogan-Yasnyy, etc.) but has met with failure.

The possibility of increased arterial pressure under the influence of physical overstress is well known. However, this increase in arterial pressure may become stable (remain at high level even after completion of training), so that the athlete becomes hypertensive. Physical overstress, as well as emotional influences, are factors causing an inclination to increased arterial pressure. Many athletes who have been subjected to physical overstress do not have high blood pressure. As concerns the high emotional activity characteristic of sports, for example, agitation before competition leads to an inadequate increase in arterial pressure only in some athletes. /394
In both cases, this increase is determined in those persons who have an inadequate reaction to external effects, indicating increased excitability of the nervous vasomotor centers, reacting by tonic constriction of arterioles. Therefore, there is no need for special separation of these factors or for their inclusion as a special classification. They do not differ in principle from many other factors influencing the excitability of the vasomotor centers.

It should be considered that among athletes with increased arterial pressure, there are doubtless both those suffering from hypertension and hyperreactors and prehypertensives.

Speaking of the significance of mental stress in the etiology and pathogenesis of hypertension, it is important to note that there is no direct and quantitative relationship between the strength and nature of the emotion and the level of arterial pressure. Since the range of emotions of each man is unique and individual, it is obvious that evaluations of mental trauma in different persons are impossible. Frequently, weak emotions corresponding to internal fine variations in a given person produce a sharp pressor effect. However, as was stated above, the development of hypertension requires, in addition to mental stress, an increase in the excitability of the vasomotor nervous apparatus, in which the potential capacity for increased vascular tonus is greater than the tendency toward its reduction.

In many cases, when there is an isolated increase in the maximum pressure, as has been shown by the works of V. P. Nikitin (1956),

Yu. I. Kuznetsov (1958, 1963), etc., it is the result of an increase in the hemodynamic impulse, since with so-called systolic hypertension the increase in maximum pressure is not accompanied by an increase in mean or lateral systolic pressure. This indicates that, in contrast to hypertension or prehypertensive states, when an increase in arterial pressure is a result of noncorrespondence between the increase in the blood volume per minute and deficient expansion of the lumen of the arterioles, in these cases the increase in pressure results from a relative increase in the blood volume per beat with normal blood volume per minute and an adequate reaction of the arterioles. However, if the proper attention is not given and the proper measures are not taken for persons with this form of increased pressure, it may lead to development of hypertension.

It is obvious from the above that the problem of increased arterial pressure in athletes is pressing, and that each case requires clinical analysis, first of all to determine a proper diagnosis, i.e. decide whether hypertension is present or not.

The importance of proper evaluation of increased arterial pressure /395
results from the fact that, depending on the diagnosis, the problem must be solved of the degree of physical loading. Also, with proper diagnosis, effective treatment can be used to prevent development of hypertension.

We must emphasize the impermissibility of the casual attitude sometimes seen toward a onetime increase in arterial pressure. This type of one-time, seemingly chance increase in pressure should be carefully studied (G. F. Lang, 1950). In these cases, the athlete should be carefully examined and, if no pathology is detected, kept under particular medical observation. Special investigations performed in this direction (L. I. Zharikov, 1962, 1963) have shown that athletes with onetime increases in arterial pressure have inadequate reactions to emotional factors (examinations, competition), and that it is from this group that hypertensives later come.

It should be noted at this time that, according to many literature references, 2-4 years after discovery of seemingly random increases in arterial pressure in 25% of cases hypertension develops, while 50% require further observation and only in 25% is the hyperreactivity completely liquidated (A. L. Myasnikov, 1954; A. G. Dembo, 1965; etc.).

Figure 108 shows data on the measurement of arterial pressure by years in two athletes. As we can see from this figure, a onetime, seemingly random increase in arterial pressure became stable after a certain time interval. Changes appeared in the fundus oculi, and the diagnosis of stage I of hypertension could be given for these athletes.

Thus, the sports doctor has a complex diagnostic problem. Its solution must rely heavily on the anamnesis, determination of hereditary predisposition, which is of tremendous significance in the development of hypertensive states, etc. The dissimulation common to athletes must be recalled,

as well as the possibility of taking various hypotensive agents before going to the doctor. In clinical examination, attention should be given to the combination of hypertrophy of the left ventricle with an accentuation of tone II in the aorta. Physiological hypertrophy is generally not accompanied by accentuation of tone II in the aorta. The evaluation of the degree of hypertrophy of the left ventricle should be performed on the basis of X-ray, electrocardiographic and particularly vector cardiographic investigation. In the early stages of the development of hypertension, particularly in trained athletes, differential diagnosis between physiological and pathological hypertrophy of the left ventricle is particularly difficult (S. P. Letunov, 1957). The presence of the corresponding changes in the vessels of the fundus oculi, and the reaction to the functional test allow a proper diagnosis to be made in many cases. /396

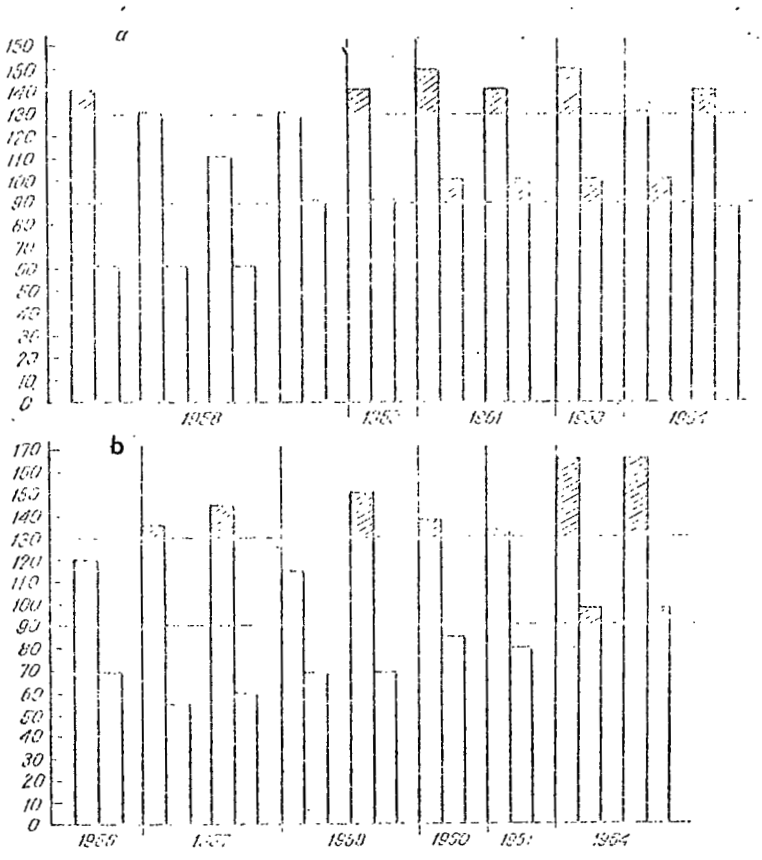


Figure 108. Level of Arterial Pressure in Masters of Sports in Football V. (a) and I. (b) over Several Years

However, sometimes even careful clinical examination does not provide a sufficient basis for diagnosis. In these cases, determination of the residual pressure and the complex of functional pressor and depressor tests can be of assistance.

It is well known that the level of arterial pressure in healthy persons /397 is unstable and fluctuates throughout the course of the day depending on various factors (N. A. Tolubeyeva, 1947; Ye. V. Andrushchenko, 1957; Holnigun, 1955; etc.). These fluctuations usually do not exceed 10 mm Hg, although in persons with elevated excitability of the vasomotor centers they are considerably greater and less stable. This is the reason for calling a pressure measured one time a random pressure. It consists of the main, i.e. stable pressure, plus the supplementary pressure, varying as a function of various situations.

The main pressure is determined under basal metabolism conditions. The pressure is measured each 3-5 min for 20-25 min until stable figures are produced. The stable figure of arterial pressure produced is called the main or basal pressure, and the difference between the random and main pressures is called the supplementary pressure.

The term "residual pressure" also refers to the main pressure, but not measured under basal metabolism conditions, but rather after 15-20 min rest at any time of day.

The value of the supplementary pressure determines the degree of excitability of the nervous apparatus controlling the pressure and will be greater, the more expressed this degree of excitability is. The time required to reach the residual pressure level increases, but in a hyperreactor will still be less than in a hypertensive (G. F. Lang, 1950; N. A. Tolubeyeva, 1926; Ye. S. Bykova, 1950; etc.).

Investigation of the residual pressure aids in distinguishing the hypertonic reaction from stably increased arterial pressure and in determining the degree of the hypertonic reaction (R. S. Ivanov, 1952; V. G. Fleysher, 1953; etc.).

Obviously, without measuring the residual pressure it is impossible to evaluate the increase in arterial pressure in athletes reliably. High figures of supplementary pressure with normal residual pressure should make the doctor be cautious. As we can see from the above, the measurement of the residual, supplementary and random pressures can be of essential diagnostic significance in evaluating high pressure and should be broadly introduced to the practice of sports medicine, particularly since this investigation is relatively easy and can be performed under any conditions.

There is yet another way to insure proper evaluation of increased arterial pressure. This is the usage of the complex of pressor and depressor tests, broadly used in clinical practice. These tests supplement the clinical examination in an important way, although they cannot replace it.

In sports medicine, the most valuable pressor tests are the following: the cold test and the test with breath held, while the most valuable depressor tests are the nitroglycerin test and hyperventilation (N. I. Vasi-l'yeva, 1958). /398

The essence of the cold test is that reflex contraction of the arterioles occurs as the forearm is submerged in a cold bath, and the arterial pressure increases; this increase is greater, the higher the excitability of the vasomotor centers.

As the breath is held, hypercapnia develops, exciting the vasomotor centers and causing an increase in arterial pressure; the increase is greater, the higher the excitability of these centers.

As concerns the depressor tests, the hypocapnia arising upon hyperventilation decreases the arterial pressure, as does the use of nitroglycerin. It should be noted that depressor tests are considerably less effective than pressor tests.

TABLE 35. TABLE FOR CLINICAL EVALUATION OF HIGH ARTERIAL PRESSURE (IN mm Hg) IN ATHLETES (N. I. VOLNOV)

Group of test subjects	Time of arterial pressure decreased to base pressure, min	Cold test		Holding breath		Residual arterial pressure	
		Degree of rise	Level of rise	Degree of rise	Level of rise	Degree of rise	Level of rise
hyperreactors	up to 12	up to 10	usually to 129,89	up to 10	usually to 129/89	up to 10	usually to 119/79
prehypertensives	15—21	15	usually to 139,99	15	usually to 139,99	15	usually to 120—129, 80—89
patients with stage 1 hypertensions	24 or more	20 or more	140/90 and higher	20 or more	140/90 and higher	20 or more	120,80 and higher

Table 35 shows the primary differential diagnostic criteria with respect to residual pressure and pressor tests which can be recommended for practical usage. In combination with clinical examination, they allow a proper evaluation of the increase in arterial pressure.

Thus, when increased arterial pressure is determined, even though only once, the athlete should be carefully examined clinically using a special set /399 of functional tests.

If the diagnosis of hypertension is made, involvement in sports should be forbidden and physical culture exercises should be recommended, under constant medical observation. As concerns prehypertension hyperreactors, the question of involvement of these persons in sports should be answered individually. In many cases, an effectively designed training program can facilitate normalization of the increased excitability of the vasomotor centers. However, involvement of these persons in sports should be subjected to careful medical control, so that training can be halted in time in case of unfavorable influence of the sports on the patient. It should also be considered that training exercises in certain types of sports facilitate increased arterial pressure.

Thus, we cannot consider every increase in arterial pressure a counter-indication for involvement in sports, nor can we allow all athletes with high blood pressure to continue in sports. This question must be answered by careful medical examination. It is important to consider that good feeling and high results in athletes with high blood pressure cannot be used as a criterion to answer the question of the continuation of the involvement in sports. Many examples can be cited when interruption of training resulted in complete normalization of the arterial pressure, while a frivolous attitude toward the nature of the training has forced athletes showing excellent results to leave the sports arena rapidly, becoming hypertensives.

On the other hand, there are many examples of normalization of arterial pressure by properly conducted sports training under careful medical observation.

In conclusion, we must turn our attention to one more important fact. In recent years the clinical literature has turned ever greater attention to the frequency of the combination of hypertension and atherosclerosis. The pathogenesis of these pathological states is considered by many to be largely the same. Thus, for example, the quantity of cholesterol in the blood, like the arterial pressure, increases in cases of nervous stress and decreases in persons involved in mental labor during periods of rest, etc. (A. L. Myasnikov, 1963). Increasing numbers of works are appearing concerning early atherosclerosis of the coronary arteries. It is sufficient to present the data of A. V. Vikhert and A. F. Ushkalov, who found coronary atherosclerosis in 30 of 35 healthy young persons autopsied after death from traumas.

Both hypertension and atherosclerosis can occur without manifesting themselves, particularly in youth. According to the data of Tamayo et al., autopsies of 2000 persons from 10 to 100 years in age showed latent atherosclerosis in 25%. Although we know that effectively performed physical /400
culture exercises and sports facilitate decreased blood cholesterol levels (S. P. Letunov, 1963), apparently influencing lipid metabolism, extreme loads may have the reverse effect.

Also, it should be kept in mind that in young hyperreactors (we are speaking here of persons 18 to 22 years of age not involved in sports), N. I. Shtel'makh (1963) discovered metabolic disruptions leading to atherosclerosis, considered the hyperreactivity to be a prologue to the development not only of hypertension, but also of atherosclerosis.

Low Arterial Pressure in Athletes

Very few works have been written concerning low arterial pressure in athletes. This is understandable, if we consider that hypotension has been thought a sign of good training, and therefore has not attracted the attention of doctors. The investigations available are principally based on studies of the data of highly qualified athletes, and pay little attention to the possibility of the existence of pathological forms of hypotension.

The literature data on the number of persons with low arterial pressure in the population are quite contradictory. According to the information of various authors, who performed many investigations, these figures fluctuate from 0.63 to 22.5%.

As concerns hypotension in athletes, according to the data of recent years (N. I. Volnov, 1958; Ye. V. Shestakov, L. I. Zharikov, 1962, 1963; M. Ya. Levin, 1965), the frequency of hypotension in athletes varies from 10.1 to 16%, the most frequent case being isolated decreases in the maximum pressure (from 2.2 to 6.9%), then a decrease of the minimum pressure only (from 4.3 to 6.1%) and least frequently a total drop in pressure (from 1.5 to 3.5%).

The literature indicates a high percentage of hypotension in athletes -- 24 (V. N. Kovalenko, 1959), while in a study of highly qualified athletes, members of teams representing the entire country, V. Ye. Vasil'yeva (1964) found hypotension in five of ten swimmers, seven of 21 light athletes, three of ten water polo players and six of 24 gymnasts, i.e. from 25 to 50%. However, it should be kept in mind that the very small number of persons investigated does not allow us to make any general conclusions.

The data of V. Ye. Vasil'yeva, produced using a large number of students at the Physical Culture Institute, were 5.8% for the first year and 13.4% for the fourth year, i.e. corresponded to the figures of other authors presented above.

As was already stated, the percentage of athletes with true hypotension, /401
i.e. with a decrease in arterial pressure below 100 and 60 mm Hg, was found to be approximately the same as the percentage of athletes with high pressure. Onetime determination of low arterial pressure was noted in 76.2% of all persons with low pressure, repeated determinations in 19.9%, constant determinations in 3.9%.

All of the above served as the basis for a special study of hypotension in athletes, which indicated that the group of hypotensives was heterogeneous.

There are many classifications of hypotensive states, none of which is generally accepted.

The most popular classification of arterial hypotension divides hypotensives into two large groups -- physiological and pathological (N. S. Molchanov, 1962). Physiological hypotension means a decrease in arterial pressure involving no pathological changes in the organism, which might be the cause of this decrease, with retention of complete working ability and normal feelings. Physiological hypotension is a variant of the norm, so that low arterial pressure in persons with physiological hypotension is observed over the course of years and sometimes throughout the entire life. Physiological hypotension may occur in completely healthy persons as a result of particular climatic conditions. Thus, in persons working in the Antarctic (I. I. Tikhomirov and V. V. Gavrilov, 1959) at temperatures of -86° with an atmospheric pressure of 450 mm, the arterial pressure was measured at 60/30 mm Hg, with full working ability and good feeling retained.

As concerns pathological forms of arterial hypotension, if we do not include the acute hypotension accompanying shock, collapse and fainting, its chronic forms are encountered both as primary hypotension (hypotension, neurocirculatory dystonia of hypotensive type), and secondary hypotension, a symptom of various diseases or intoxications.

The essence of true hypotension is a disruption of the regulation of the circulatory apparatus. Primary hypotension is accompanied by a number of complaints and symptoms, indicating disruption of the functions of various organs and systems of the organism. As concerns secondary hypotension, as we know, a decrease in arterial pressure is a constant symptom of a number of diseases (for example, Addison's disease, infectious diseases) and intoxications. Hypotension frequently occurs at various centers of chronic infection, including latent infections. This is particularly true in the case of arterial hypotension in athletes, since the percentage of persons with centers of infections among athletes is considerable.

A study of groups of athletes with hypotension showed that the sex, age /402
and sports specialty influence the level of arterial pressure. Thus, among female athletes, hypotension is encountered slightly more than twice as frequently as in men (26 and 12.8% respectively, according to the data of M. Ya. Levin).

TABLE 36. FREQUENCY OF DECREASE IN ARTERIAL PRESSURE IN ATHLETES AS A FUNCTION OF SPORTS SPECIALTY (DATA OF M. Ya. LEVIN)

Type of sports	Percent of cases	Type of sports	Percent of cases
gymnastics	30.6	wrestling	12.7
light athletics	25.3	skiing	12.7
basketball	24.0	bicycling	12.2
boxing	18.9	volleyball	8.6
skating	16.7	rowing	8.2
swimming	13.0	football	7.5
heavy athletics	12.7		

As we can see from Table 36, the highest percentage of athletes with hypotension was found among gymnasts (30.6), the lowest among football players (7.5). As concerns age, as we can see from Table 37, the younger the athletes, the more frequently persons with decreased pressures are encountered among them.

TABLE 37. DECREASE IN ARTERIAL PRESSURE AND AGE OF ATHLETES

Age, years	18-19	20-24	25-29	30-34	35-39	40 and older
Percentage of cases	22	17	15.1	15.9	10.8	7.7

These are the general data concerning low arterial pressure in athletes. As we can see, as concerns the average figures there is no difference in the percentage of persons with low blood pressure in athletes and in persons not involved in sports. This confirms again the assumption that the group of athletes with hypotension is heterogeneous and that, in addition to physiological hypotension, pathological forms are encountered among them. Thus, V. N. Kovalenko (1957) showed that in 40% of the athletes with hypotension, an unsatisfactory reaction to the functional test was noted; hypotension in /403
these persons is looked upon as an indication of fatigue. The author found no relationship between increasing state of training and increasing number of hypotensives. He came to the conclusion that hypotension in athletes may be either an indication of high state of training or a manifestation of fatigue. Similar data were presented by Ye. V. Shestakova, who also showed the heterogeneity of the group of athletes with hypotension.

However, along with this it is commonly thought that hypotension is characteristic of high training and in the overwhelming majority of cases should be evaluated as a positive factor. In order to answer this question,

in our laboratory M. Ya. Levin subjected two groups of athletes to clinical and statistical investigation with respect to various parameters.

M. Ya. Levin (1965) compared the data produced in a study of 987 athletes of various sports specialties with hypotension with a similar (as to age, sex, sports specialty, sports mastery, etc.) group of athletes (also 987 persons) with normal arterial pressure figures (Table 38).

TABLE 38. SOME CLINICAL-PHYSIOLOGICAL INDICATORS IN ATHLETES WITH LOW AND NORMAL ARTERIAL PRESSURE (DATA OF M. Ya. LEVIN)

Group	Various complaints	Bradycardia (below 60 beats/min)	Centers of chronic infection (without caries)	Overtraining in anamnesis	Respiratory diseases	
					Angina	Acute upper respiratory infection
athletes with hypotension	11.4	19	30.4	29.7	1.2	3
athletes with normal arterial pressure	1.9	25.4	11.6	12.7	0.1	0.5

It was found that athletes with low arterial pressure are more frequently subject to colds, and more frequently show changes in the heart (dullness of tone I, systolic noise, etc.), have more centers of chronic infection, are more frequently overtrained, show less frequent bradycardia, etc. Also, it was found that the increase in sports results of these persons occurs more slowly than in athletes with normal pressure, the reaction of the cardiovascular system to physical loading is poorer, etc. The difference in the two groups was also quite clearly seen from changes in the electrocardiograms (Table 39).

At the same time, it was found that the highest percentage of athletes /404
with hypotension consisted of masters of sports (Table 40). As concerns the training period, athletes with hypotension were found to be mostly in the period of main training.

All of this confirms the idea that in addition to the hypotension in athletes arising as a result of high states of training, a considerable number of cases of hypotension are pathological. Analysis of the literature data and clinical investigations of a large number of athletes with low arterial pressure allows us to state that secondary hypotension in athletes

can be divided into hypotension as a result of centers of infection and hypotension arising as a result of fatigue.

TABLE 39. SOME ELECTROCARDIOGRAPHIC INDICATORS (IN PERCENT) OF ATHLETES WITH LOW AND NORMAL ARTERIAL PRESSURE (DATA OF M. Ya. LEVIN)

Group	extrasystoles	sinus arhythmia	Elongation of P-Q interval	Left ventricular overstress	Low-voltage EKG	Unsatisfactory reaction to physical load	Diffuse muscular changes	Atrioventricular rhythm
athletes with hypotension	3.9	8	2.9	8.6	2.2	14.2	1.6	3.1
athletes with normal arterial pressure	2	3.9	1.1	3.1	0.2	5.3	0.3	1.1

TABLE 40. DROP IN ARTERIAL PRESSURE IN ATHLETES OF VARIOUS SPORTS QUALIFICATIONS (DATA OF M. Ya. LEVIN)

Sports qualification	Master of sports	Class I	Class II	Class III	Unranked
Percentage found	21	14.7	14.9	3	2.5

The necessity arises of the development of differential diagnostic criteria which would allow a diagnosis to be made for various forms of hypotension. The differential diagnosis should be based first of all on the history, clinical picture, reaction of the pressure to pressor tests, condition of the vasomotor centers, and in both cases similar tests should be used.

Sometimes the question of the pathological nature of reduced arterial pressure can be solved ex juvantibus. Thus, frequently the removal of centers of infection (tonsillectomy, sanitization of the oral cavity) normalizes the pressure, so that its pathological nature is obvious. /405

This can be seen from the following example.

Athlete P, 23 years old, is a class I skater. When examined in February of 1964, he complained of piercing pains in the area of the heart, frequent headaches, rapid fatiguing. Objective examination showed pulse 62 beats per minute. No deviations from the norm in the internal organs were found. Arterial pressure was 90/45 mm Hg. Nine gangrenous teeth were found in the oral cavity, along with enlargement and soreness of the submaxillary lymph nodes on both sides. The electrocardiogram showed initial phenomena of left ventricular overstress, unsatisfactory reaction to physical loads. X-ray investigation of the thorax, analysis of the blood and urine showed no deviations from the norm. The blood flow time over the lung-ear sector was 4.1 sec. The rate of propagation of the pulse wave through the elastic type vessels was 320 cm/sec, through muscular type vessels -- 740 cm/sec. After two minutes run in place, the rate of propagation of the pulse wave increased by 211 cm/sec and 305 cm/sec respectively. Diagnosis: secondary hypotension as a result of chronic infection (massive caries).

In March of 1964, complete sanitization of the oral cavity was performed. When examined in June and December of 1964, the patient had no complaints, arterial pressure was 110/80 mm Hg, blood flow time was 6.5 sec; the rate of propagation of the pulse wave through the elastic vessels was 332 cm/sec (after loading, increased by 85 cm/sec), through muscular type vessels -- 1160 cm/sec (after loading unchanged).

The development of differential diagnostic criteria for a proper evaluation of decreased arterial pressure in athletes is an important and pressing problem. Carefully gathered medical and sports histories, detailed clinical examination with analysis of the nature of complaints and determination of centers of infection allow differential diagnosis to be performed between the four principal forms of hypotension in athletes (A. G. Dembo and M. Ya. Levin, 1963).

In the group of physiological hypotension, or hypotension as a characteristic of a high state of training, we include athletes showing no complaints, no deviations in the state of health (following clinical and instrumental examination), no centers of infection, all combined with good feeling and high working ability. Of the 987 athletes with hypotension whom we studied, this group included 32.3%.

The group with secondary hypotension should include those athletes who have various types of complaints. Some of these have clear centers of chronic infection (30.3%), and in some we could establish a direct relationship between the decrease in pressure and fatigue (29.7%). Finally, in a small group of athletes with various complaints, we could find no centers

of infection, and no connection to fatigue. We diagnosed these as neurocirculatory hypotensive dystonia (7.7%). It is interesting to note that this group included most of the athletes with constantly decreased arterial pressure. The data presented in Table 41 tend to confirm the correctness of this differential diagnosis. /406

TABLE 41. FORMS OF HYPOTENSION AND SPORTS QUALIFICATION (DATA OF A. G. DEMBO AND M. Ya. LEVIN)

Sports qualification	Form of hypotension, percent			
	Physiological hypotension	Secondary hypotension as a result of		Neurocirculatory dystonia
		Infection	Fatigue	
master of sports	41.2	28.8	21.9	8
class I	28.7	31.4	32.8	7
classes II and III	20	27.7	41.5	12.3

As we can see from Table 41, the percentage of athletes with physiological hypotension increases with the sports qualification, whereas the percentage of athletes with hypotension resulting from fatigue decreases. As concerns the remaining two groups, as would be expected, they are independent of the level of sports mastery and therefore remain almost unchanged. The percentage of persons with neurocirculatory dystonia decreases slightly among masters of sports and athletes first class.

Determination of the residual pressure, as well as the complex of pressor tests and determination of the rate of blood flow by oxyhemometry can be an important source of supplementary information for the differential diagnosis (A. G. Dembo and M. Ya. Levin, 1965).

The essence of this combined investigation of the functional state of the apparatus controlling arterial pressure (Table 42) consists of determination of its reactions to certain physiological processes (test to determine the residual pressure) and to a number of artificial stimuli (water from ±1° to 4° in the cold test and an increase in carbon dioxide in the blood in the breath holding test).

When the residual pressure was determined, attention was turned to the time required for the random pressure to drop to the residual level. In determining the pressor tests, the amplitude of fluctuations in arterial pressure was considered, as well as the time required for return to the initial level.

As the data of Table 42 show, the time for the decrease in arterial pressure to the residual pressure level and fluctuations in residual pressure in athletes with physiological hypotension differ from pathological

hypotension. Similar data are found when the cold test and breath holding test are used, as well as in investigations of the blood flow time determined /408 by oxyhemometry. With pathological hypotension, this latter figure is normal or abbreviated, whereas it is characteristically lengthened for physiological hypotension.

As we can see from Table 42, the data produced in an investigation of athletes with hypotension resulting from fatigue, according to the indications of the cold test and the breath holding test are similar to the data produced upon investigation of athletes with physiological hypotension. When the residual pressure was determined in this group of athletes, data were produced which lay between the physiological and pathological forms of hypotension. As concerns the blood flow time, it was reduced, which indicates a drop in the level of training.

The data produced by using various tests indicate difference in the reactions characterizing the function of the vasomotor centers with physiological and pathological forms of hypotension. Physiological hypotension is characterized by reactions natural to healthy persons with normal arterial pressure. The reactions with hypotension and secondary hypotension resulting from chronic infection are quite similar to each other. Great lability of the residual pressure is characteristic for this group of athletes.

The similarity of the reactions of the apparatus controlling arterial pressure in athletes with pathological forms of hypotension allows us to assume that, although they are developed on different bases, both hypotension and secondary hypotension resulting from centers of chronic infection cause similar changes of the internal organs and higher centers regulating the arterial pressure.

Detailed analysis of the data of athletes with hypotension resulting from fatigue confirms the idea that these are practically healthy persons with slight disruptions of the vasomotor center function.

It seems to us that this method allows us to determine not only manifest changes in the activity of the apparatus controlling arterial pressure, but also its early disruptions. It allows us to perform differential diagnosis between physiological hypotension on the one hand and its pathological forms on the other. However, its significance is only secondary. The basis of differential diagnosis must be carefully collected anamnesis and detailed clinical examination.

TABLE 42. CLINICAL-PHYSIOLOGICAL ANALYSIS OF VARIOUS HYPOTENSIVE STATES IN ATHLETES

Group of test subjects	Residual arterial pressure		Cold test		Breath holding test		Blood flow time, sec.
	Time of decrease, min	Fluctuations, mm Hg	Degree of increase of arterial pressure, mm Hg	Time of recovery of arterial pressure, min	Degree of increase of arterial pressure, mm Hg	Time of recovery of arterial pressure, min	
Norms	6—9	None	5—10	Up to 3	5—10	Up to 3	Depending on age
Physiological hypotension	Up to 6	»	5—10	» 3	5—10	» 3	7,15 (Delay)
Hypotension	9—12	10	No reaction 15—20	6—8	No reaction	—	5,55 Norm or acceleration
Secondary hypotension resulting from chronic infection	9—12	10	No reaction 15—20	6—8	Same	—	5,5 Norm or acceleration
Secondary hypotension resulting from fatigue	6—12	5	5—10	Up to 5	5—10	Up to 5	5,1 Acceleration

The similarity of the reactions in hypotension and secondary hypotension resulting from centers of chronic infection indicates that the decrease in arterial pressure with these forms of hypotension is caused by the same factors. As concerns hypotension resulting from fatigue, it occupies an intermediate position, since it is encountered in most cases in healthy persons [NOTE: This chapter ends here, in the middle of a sentence, in the foreign language original. No page numbers are missing; an undetermined amount of text was simply omitted in the printing -- Tr.]

CHAPTER XII

DISRUPTIONS OF THE CARDIAC RHYTHM IN ATHLETES

ABSTRACT. The frequency and significance of various arhythmias in athletes are analyzed. Case histories are presented to illustrate that many types of arhythmia frequently described as "variations of the norm" in the "sports heart" may in actuality indicate cardiac pathology. The importance of differential diagnosis of all arhythmias is emphasized. And next, attempts at too rapid physical training and conditioning can lead to phemenona of cardiac overstress. Case histories, electrocardiograms and physiological explanations are presented to illustrate the significance of measured, properly applied physical exercise, undertaken only during periods of perfect health, in the prevention of cardiac pathology in athletes.

The problem of disruptions of the heart rhythm is one of the most /409
important problems in contemporary cardiology. Disruptions of the heart rhythm are encountered both in healthy persons, and in persons with widely varied health problems. Also, a number of basic problems in the etiology and pathogenesis of disruptions of the heart rhythm cannot yet be considered solved. Essentially, there is not even a generally accepted classification of rhythm disruptions.

The rhythm of cardiac activity, being an extremely variable parameter, characterizes the functional state of the heart precisely. Therefore, analysis of the rhythm of cardiac activity has important clinical significance. We are concerned here not only with changes in the frequency of heart contractions or various intervals between individual heart contractions, but also disruptions in the rhythm occurring within the rhythmic complex, manifested only upon electrocardiographic investigation -- the so-called eurhythmia.

Normal circulation is determined by smooth coordination of cardiac activity. The precise sequence in the contraction of the atria and ventricles, coordinated with the mechanism of successive closing and opening of the atrioventricular and semilunar valves, is supported by pulses arising rhythmically in the sinus node and the normal function of the conducting system of the heart (atrioventricular node, bundle of His and both of its branches).

An extensive clinical literature exists on disruptions of the heart rhythm, and this literature is generalized in the monographs of A. M. Sigal (1958), I. A. Chernogorov (1962) and others. Still, this problem is not completely clear. This is particularly true of disruptions of the heart rhythm in athletes, the study of which has been very sparse (L. A. Butchenko, /410

1963; V. N. Kovalenko, 1959; V. I. Kalyunov; A. G. Dembo, M. L. Proektor and Ye. I. Kareva, 1965; Kh. Yu. Sil'dmyae, A. A. Viru, S. M. Oya and E. A. Viru, 1964; etc.). However, this problem becomes more pressing with each passing year, since the number of athletes with disruptions of the heart rhythm increases, and there are conclusive data currently available indicating the pathological significance in many cases.

According to present concepts, all disruptions in the rhythm are manifestations of changes in the normal relationships between excitability and conductivity on the one hand and automatism of the specific conductor and muscular tissue of the heart on the other hand (I. A. Chernogorov).

A second essential factor in the origin of disruptions of the rhythm consists of the morphological changes in the myocardium resulting from various causes, including infections, intoxications, disruptions of endocrine metabolism and exogenic disruptions (I. A. Chernogorov, 1962). In the opinion of I. A. Chernogorov, the principal background against which disruptions of the cardiac rhythm develop is the functional state of the higher segments of the vegetative nervous system controlling the principal functions of the heart. Without negating the tremendous significance of organic heart damage, I. A. Chernogorov evaluates them as a stimulant, the degree of the effect of which depends on the functional state of the entire complex system of regulation of the activity of the heart at the moment the stimulant arises.

In recent times, many authors (V. M. Bogolyubov, 1963; L. T. Malaya et al., 1964; Scherf and Schott, 1959; Fox, 1959; etc.) look upon changes in the normal relationships between potassium and sodium as one of the pathogenetic links in the disruption of the rhythm. One confirmation of this is the effectiveness of large doses of potassium in the treatment of arhythmia. It is considered that the effect of potassium is nonspecific; it restores disruptions in the equilibrium between potassium and sodium and depressed the ectopic center of excitation in the myocardium. These ectopic centers of excitation arise in the myocardium as a result of changes in the metabolism, the nature of which may be quite varied. These metabolic changes facilitate disorders in the nervous regulation of the heart, leading sometimes to a reduction in the content of macroergic phosphorous compounds (M. S. Bakumenko).

We know that disruptions in the heart rhythm may be encountered in quite healthy persons as a result of various extracardiac influences (G. F. Lang, A. M. Sigal, etc.). On the other hand, disruptions in the rhythm are frequently manifestations of various pathological changes in the cardiac muscle. In this connection, we must recall the work of F. Z. Meyerson et al. (1964), in which it was shown experimentally that the nervous /411
regulation of the heart changes in cardiac hyperfunction; in particular, the reactivity to the influence of the vagus nerve is decreased. They called this the "emancipation phenomenon" and explained it as an increase in the resistance of the sinus node to the inhibitory influence of the vagus nerve.

One of the factors explaining both the phenomenon of emancipation and the increase in automatism was considered by these authors to be a decrease in the level of potassium in the myocardium as a result of an increase in its elimination during cardiac hyperfunction.

In addition to this, it is thought that the formation of disruptions in the rhythm under the influence of nervous factors can occur only in the presence of certain pathological changes in the myocardium (D. M. Fedorov, 1965; etc.). In any case, it is doubtless that the reaction of the cardiac rhythm to any given stimulus depends on the functional state of the cardiac muscle and that the same stimulus may give different effects.

Among active athletes, we encounter persons with almost all types of arhythmia, depending not only on disruptions of automatism, but also on excitability and conductivity. The frequency of various disruptions in the rhythm are variable -- from individual cases, such as paroxysmal ciliary arhythmia or dissociation with interference, to rather frequently encountered arhythmias caused principally by disruption of excitability and automatism. Still, disruptions of the rhythm in athletes are encountered twice as frequently as in persons not involved in sports (Table 43). In works dedicated to analysis of disruptions in the cardiac rhythm of athletes, these disruptions are frequently interpreted as functional, as a variant of the norm, as a "peculiarity" of the sports heart, etc. In our opinion, each such case should be carefully clinically analyzed.

Table 43 presents data on the frequency of various arhythmias in athletes (A. G. Dembo, Ye. I. Kareva, M. L. Proektor, 1964).

As we can see from Table 43, the percentage of athletes with various arhythmias is 11.28% of the overall total (2742 persons). The arhythmias resulting from disruption of automatism, according to our data, make up 4.4%, almost twice as great as that noted by L. A. Butchenko (1957). It should be noted that this group did not include sinus tachycardia or sinus arhythmia. It did include: acute sinus bradycardia (less than 40 beats per minute), migration of the source of rhythm, atrioventricular rhythm and coronary sinus rhythm. The arhythmias resulting from disruption of conductivity amounted to 3.65%. The principal group among these consisted of atrioventricular block, first degree, frequently encountered in athletes and representing in many cases a normal variant of the sports electrocardiogram. However, this group of arhythmias also includes atrioventricular block, /412
stage II, complete block of the right branch of the bundle of His of the Wilson type and the Wolf-Parkinson-White syndrome. Listing of these changes as a variant of the norm requires convincing proof. As was pointed out above, the remaining types of arhythmia were encountered in a few individual cases.

No convincing data concerning the influence of sex, age and sports mastery on the frequency of arhythmia was discovered.

TABLE 43. DISRUPTIONS OF HEART RHYTHM IN ATHLETES (IN PERCENTAGE OF TOTAL NUMBER OF PERSONS STUDIED) IN 1962 FROM MATERIALS OF LGVFD (A. G. DEMBO, Ye. I. KAREVA, M. L. PROEKTOR)

Type of rhythm disruption	Total no. subjects		Of these			
			Men		Women	
	Absolute	%	Absolute	%	Absolute	%
Arhythmia resulting from disruption of excitability:						
extrasystolic arhythmia	88	3,22	59	2,81	29	4,51
Arhythmia resulting from disruption of automatism	121	4,4	93	5,6	28	4,5
Bradycardia (less than 40 beats per min)	25	0,9	24	1,16	1	0,15
Migration of sinus rhythm	35	1,27	24	1,16	11	1,71
Atrioventricular (nodal) rhythm	18	0,65	11	0,52	7	1,1
Coronary sinus rhythm	43	1,56	34	0,61	9	1,4
Arhythmia resulting from disruptions of conductivity	100	3,65	79	3,52	21	3,1
Atrioventricular block	72	2,64	55	2,62	17	2,64
stage I, stage II	6	0,21	5	0,93	1	0,15
Complete block of right branch of bundle of His, Wilson type	5	0,18	4	0,19	1	0,15
WPW(Wolf-Parkinson-White) syndrome	17	0,62	15	0,71	2	0,31

Tr. Note: Commas indicate decimal points.

There is some interest in a comparison of these data with the data produced by Hiss and Lamb (1962), who studied the electrocardiograms of 122,043 healthy persons -- pilots and soldiers (Table 44).

We can see from Table 44 that, with the exception of migration of the sinus rhythm and complete block of the right branch of the bundle of His, the remaining forms of disruption of the heart rhythm are encountered much more frequently in athletes than in nonathletes.

TABLE 44. COMPARATIVE DATA ON VARIOUS DISRUPTIONS OF CARDIAC RHYTHM ACCORDING TO VARIOUS AUTHORS (IN PERCENT)

Authors, year	Disruption in excitability	Disruption in automatism				Disruption in conductivity					Grand total
		Migration of sinus rhythm	Nodal rhythm	Coronary sinus rhythm	Total	Atrioventricular block Stage I	Atrioventricular block Stage II	Complete block of right branch of bundle of His	WPW syndrome	Total	
A. G. Dembo E. M. Kareva M. L. Proektor 1964	3,22	1,27	0,65	1,76	3,5	2,64	0,21	0,18	0,62	3,66	10,38
Hiss, Lamb, 1962	1,53	1,52	0,01	0,55	2,08	0,65	0,003	0,18	0,15	0,98	4,49

Tr. Note: Commas indicate decimal points.

Disruption in excitability. The greatest interest in sports medicine is felt in the group of arhythmias arising as a result of disruption in excitability, i.e. extrasystolic arhythmia. These arhythmias are quite difficult for differential diagnosis.

Also, the number of athletes with extrasystolic arhythmia has increased considerably in the last 15 years, so that the necessity of determining the origin of these arhythmias is of considerable practical significance. In 1950, the percentage of athletes with extrasystolic arhythmias was 1.5 (S. P. Letunov), i.e. the same as Stewart and Manning (1942) determined for healthy persons. However, in 1959 this percentage had increased to 2.9 among athletes (L. A. Butchenko), while by 1962 extrasystolic arhythmia was noted in 3.22% of the active athletes. This increase must be related to the sharp increase in the degree of training loads which has occurred in recent years, which should caution us once more against considering extrasystolic arhythmia in athletes a variant of the norm.

It is well known that extrasystolic arhythmia, namely individual extrasystoles, is encountered in healthy persons. The causes are many. In many cases, they result from the influence of extracardiac factors and are principally a manifestation of discoordination between the parasympathetic and sympathetic influences, and also a result of various emotional influences.

An example of the latter is master of sports in skating M, 28 years old. /414 He has no complaints. He has high emotional excitability. On the electrocardiogram (Figure 109) taken during a time of considerable nervous excitation, we see bigeminy and trigeminy. A few days later, we see complete

normalization of the electrocardiogram. Subsequently, no disruptions in the electrocardiogram were observed, and the athlete continued to train and enter competition. This observation is interesting also because allorhythmia was always considered to be a manifestation of severe organic changes in the heart.

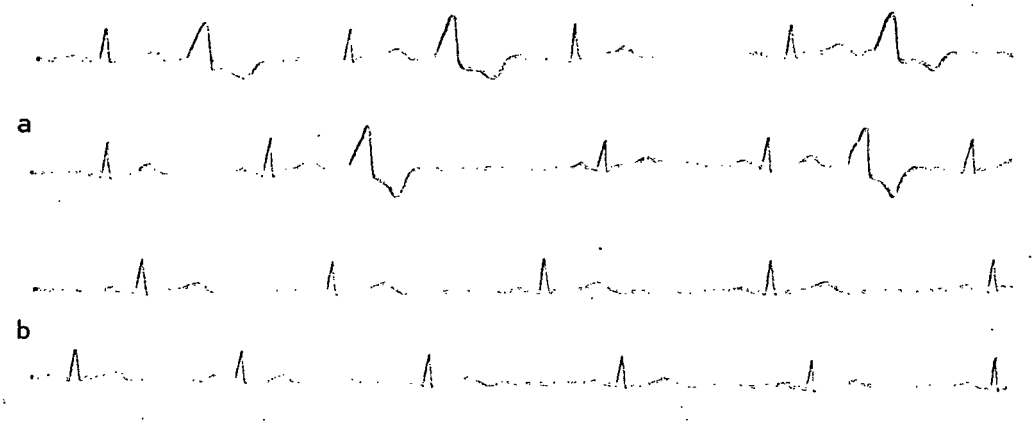


Figure 109. Electrocardiogram of Athlete M. a, During nervous excitation; b, several days later (explanation in text).

The reason for the formation of extrasystolic arhythmia may also be reflector influences arising from the tonsils and biliary tracts (M. A. Eres'ko, 1957), the sexual area (Lecont, 1911), or the respiratory organs (Lamb et al., 1958), etc.

An essential role in the formation of extrasystolic arhythmia is played by dystrophy of the myocardium both during acute infection and as a result of intoxication from various centers of chronic infection (R. G. Mezhebovskiy, 1961; Reindell, 1943; Klemola, 1962; etc.). This should be particularly kept in mind in evaluating extrasystolia in athletes, since the percentage with centers of chronic infection (tonsillitis, abscessed teeth, cholecystitis) is higher than among persons not involved in sports. Convincing confirmation of the significance of tonsillogenic infection in the formation of rhythm disruptions can be found in the disappearance of these disruptions after tonsillectomy.

Let us present an example. Light athlete class I N, 20 years old. No complaints. Clinical examination shows chronic tonsillitis. The electrocardiogram (Figure 110) shows atrial extrasystoles (a), completely disappearing after six months following tonsillectomy (b).

An essential role in the formation of extrasystolic arhythmia in athletes is played by physical overstress. A number of authors (Messerle, 1928; Reindell, 1938; S. P. Letunov, 1950; A. A. Kedrov, 1963; /415

V. I. Kalyunov, 1964; etc.) have described extrasystolic arhythmia as a result of physical overstress.

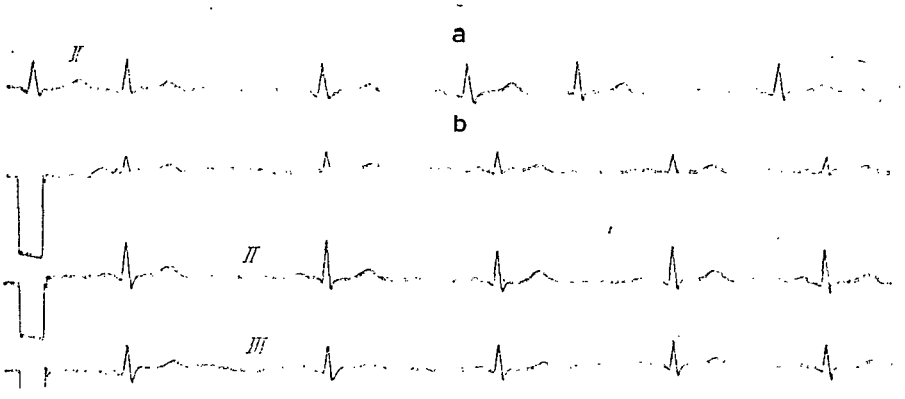


Figure 110. Electrocardiogram of Athlete N (Explanation in Text)

One case of formation of ciliary arhythmia under the influence of weight lifting has been described (L. T. Antonov, 1964). V. N. Morozov (1964) describes various changes in the electrocardiogram, including extrasystoles, depending, in his opinion, on overloading, performed without sufficient rest.

There is some interest in a case of functional polytopic bigeminy noted in a 23-year-old athlete, arising as a result of overfatigue with large training loads (N. S. Yevseyev and Ye. S. Rom-Boguslavskaya, 1963). This bigeminy disappeared completely under the influence of rest and treatment.

1. Master of sports in wrestling A, 22 years old. The electrocardiogram (Figure 111) taken during overtraining, shows atrioventricular rhythm with Wenkebach-Samoylov periods. After one half year's rest, we see complete normalization of the electrocardiogram.

2. Football player first class F, 23 years old. Manifest phenomena of overtraining observed, and the electrocardiogram, previously completely normal, shows atrioventricular block with Wenkebach-Samoylov periods (Figure 112). After two months' rest, all eliminated, and the electrocardiogram is quite normal.

Disruptions in the regimen have some significance in the formation of rhythm disruptions. M. B. Kazakov (1961) presents a case involving heavy athlete L, who, after a severe disruption in regimen (immoderate drinking and /416 smoking), shows ciliary arhythmia. The corresponding treatment succeeded in in achieving restoration of the rhythm, and subsequently no arhythmia was noted for the next three years, although the athlete continued in training.

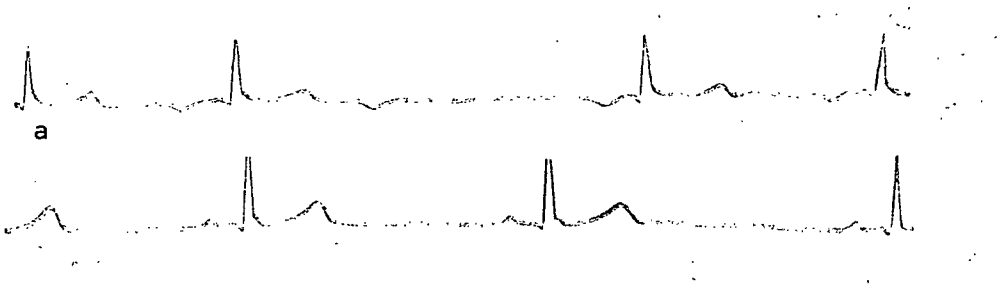


Figure 111. Electrocardiogram of Athlete A (Explanation in text)

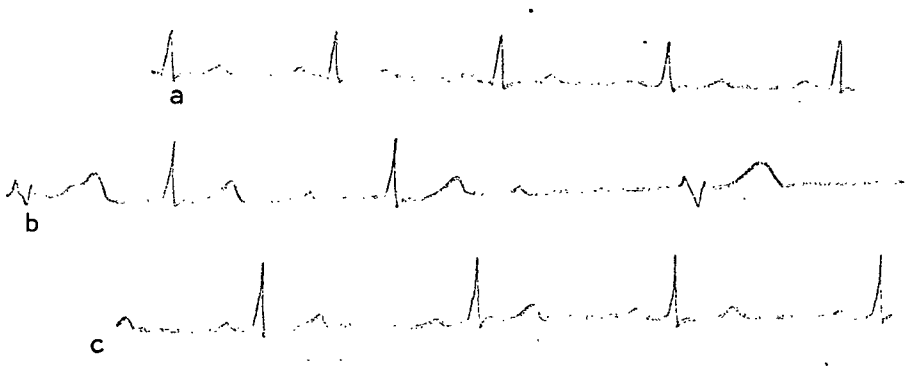


Figure 112. Electrocardiogram of Athlete F (Explanation in text)

Let us present a similar case. Boxer R, 28 years old, master of sports (Figure 113). On 30/3/1957, an electrocardiogram was taken, which proved normal (a). Between 30/3 and 25/4, the athlete was knocked out in a bout, followed after some time by alcoholic intoxication. The electrocardiogram of 25/4 shows ciliary arhythmia (b), disappearing after two weeks' complete rest, not to return (c).

Disruptions in electrolyte metabolism, the significance of which in the formation of rhythm disruptions has been indicated recently in the literature (Friedberg, 1956; Scherf, Schott, 1957; Fisch et al., 1960; L. T. Malaya, 1964; A. V. Sumarukov, 1964), may also have some significance in the formation of arhythmias in athletes. It should be noted that this factor doubtless has some significance in the formation of the cardiac overstress syndrome in athletes (A. G. Dembo, M. L. Proektor, Zh. A. Teslenko, 1962, 1963).

Finally, cardiac rhythm disruptions in athletes may result from cardiosclerosis following past, undiagnosed rheumatic myocarditis or resulting from other causes. /417

Figure 113. Electrocardiogram of Athlete R (Explanation in text)

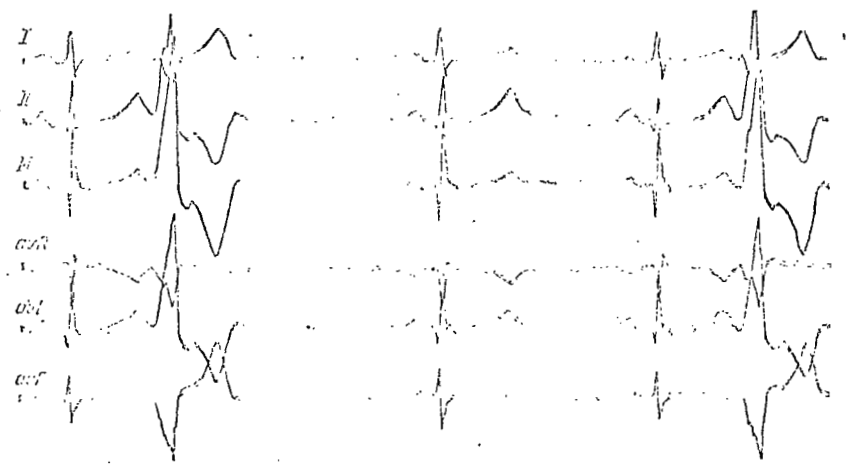


Figure 114. Electrocardiogram of Athlete Zh (Explanation in Text)

Master of sports in skiing Zh, 21 years old. The electrocardiogram (Figure 114) clearly shows bigeminy. This bigeminy is combined with other changes indicating pathology. The changes in the electrocardiogram, data from the anamnesis and clinical examination indicate the following diagnosis: myocarditic cardiosclerosis, further involvement in sports forbidden. Unfortunately, in spite of this, the athlete continued training, and after an intensive training exercise, died in sleep. The diagnosis of myocarditic cardiosclerosis was confirmed by autopsy.

Considering the above, the entire group of extrasystolic arhythmias in athletes can be divided into three groups. The first group includes persons with individual extrasystoles, in which there is reason to believe that the extrasystolia is functional in origin. The second and third subgroups include those types of extrasystolic arhythmia in which there is reason to suspect the possibility of organic damage to the heart (Kienle, 1946; L. A. Butchenko, 1962). The second group includes various allorhythmias, group and polytopic extrasystoles. The third group includes persons in which the extrasystolia is combined with other changes on the electrocardiogram (WPW syndrome, block of the right branch of the bundle of His, etc.). /418

Table 45 presents data on the frequency of various types of extrasystolic arhythmia in the period from 1962 to 1964. Our attention is drawn by the fact that, although the total number of athletes with extrasystolic arhythmias has remained almost unchanged over the past three years, the number of athletes with individual extrasystoles has noticeably decreased, with a corresponding increase in more complex forms of arhythmia. Thus, the combination of extrasystoles with other changes in the electrocardiogram has increased from 11.4% to 23.1%; allorhythmia, polytopic and group extrasystoles have increased from 12.5% in 1962 to 30.6% in 1964.

TABLE 45. FREQUENCY OF VARIOUS TYPES OF EXTRASYSTOLIC ARHYTHMIA BY YEARS (PERCENTAGES OF TOTAL NUMBER OF ATHLETES WITH EXTRASYSTOLIC ARHYTHMIA IN EACH YEAR)

Year	Number of athletes with extrasystolic arhythmias	First Group		Second group			Third group
		Individual extrasystoles	Various allorhythmias	Group extrasystoles	Polytopic extrasystoles	Total	Extrasystolia combined with other changes of EKG
1962	88[1] 100[2]	67 76,1	4 4,6	1 1,1	6 6,8	11 12,5	10 11,4
1963	94 100	57 60,6	11 11,8	1 1,0	7 7,4	19 20,2	18 19,2
1964	95 100	44 46,3	14 14,8	4 4,2	11 11,6	29 30,6	22 23,1

1 Number of test subjects
2 Percentage

Tr. Note: Commas indicate decimal points.

Thus, in comparison with 1953, we see not only an increase in the number of athletes with extrasystolia, but also a change in the nature of the extrasystolic arhythmias. In the light of what we have stated above concerning the increase in intensity of training loads, this cannot be by chance, since high loads, used inefficiently, may be a reason for the formation of pathological changes of the heart. In connection with this, it is even more important to perform obligatory and careful clinical analysis of each case of extrasystolic arhythmia in athletes, particularly since they make no complaints in most cases. /419

Careful clinical analysis of each case of cardiac arhythmia is necessary. Listing disruptions in the rhythm as variants of the norm on the basis of absence of complaints alone, or in combination with high sports results, cannot be done. The reversibility of arhythmias does not always indicate that there is no organic damage to the heart. All of this is true not only of arhythmias resulting from disruptions in excitability, but all disruptions in the cardiac rhythm in athletes.

Disruptions of automatism. The group of arhythmias arising as a result of disruptions in automatism must include first of all manifest sinus bradycardia with a pulse frequency of less than 40 beats per minute.

As was noted above, the classical triad of the state of training (hypotension, bradycardia, hypertrophy) requires further examination. In Chapter XI (see p. 375) we showed the possibility of various origins of hypotension. As concerns bradycardia, first of all it can be looked upon as an indicator of the state of training without special examination of the athlete only to a certain extent. Below this frequency level, we must in each case be sure that it is not a manifestation of pathological changes in the myocardium.

The convincing confirmation of the above is the case of light athlete B, 23 years old, who had manifest bradycardia (28-30 beats per minute). Our attention is drawn by the fact that in the functional test, this athlete's pulse showed very little increase. Electrocardiographic examinations showed complete atrioventricular block as a result of myocarditic cardiosclerosis of rheumatic etiology.

V. N. Kovalenko, L. A. Butchenko, V. I. Kalyunov, M. L. Proektor did not always find a parallelism between bradycardia and high level of training. Thus, V. I. Kalyunov (1963) found poor adaptability to loading in 30% of athletes with bradycardia, along with a decrease in the working ability, fatigue, sleep disruptions and irregular appetite, pains, etc. He assumes that in these cases the bradycardia is a result of overfatigue or infectious-toxic effects.

An example of bradycardia as a result of overloading is presented by the electrocardiogram of athlete V, 19 years old, a class I volleyball player (Figure 115). He has no complaints, trains daily, combining this with study at the institute. The electrocardiogram (Figure 115, A) shows manifest /420

bradycardia (33 beats per minute), combined with ventricular overstress phenomena (see p. 428). For two months, training was stopped. The electrocardiogram taken two months later (Figure 115, B) shows no left ventricular overstress, and the bradycardia has disappeared -- the pulse frequency is 54 beats per minute.

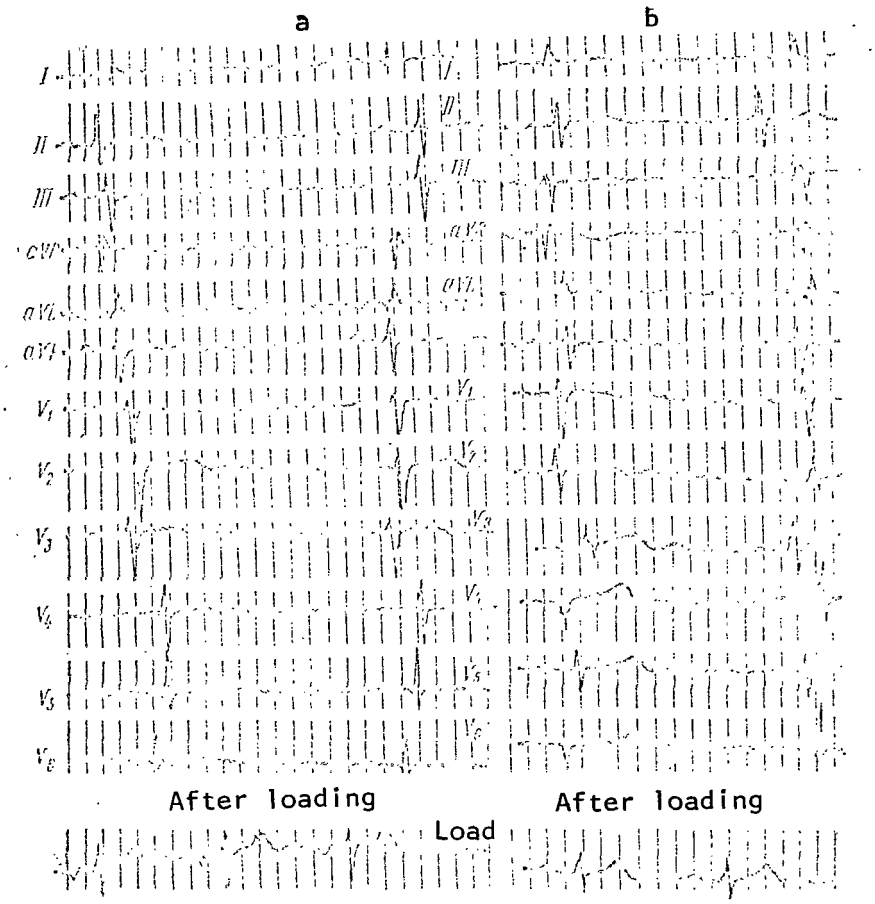


Figure 115. Electrocardiogram of Athlete V (Explanation in Text)

Thus, it is obvious that any case of bradycardia below 40 beats per minute should be the subject of special medical analysis and only if no other changes on the electrocardiogram are found, and if there are no deviations from the norm in the state of health, can the bradycardia be considered a peculiarity of the electrocardiogram of a healthy athlete, indicating a high state of training. /421

Among the disruptions in rhythm arising as a result of disruptions in automatism, we must also include migration of the source of rhythm, i.e. displacement of the rhythm control from the sinus node to the atrioventricular node and back, atrioventricular rhythm and coronary sinus rhythm, one variety of atrioventricular rhythm. These disruptions in rhythm are encountered in a certain percentage of healthy persons, although they are most frequently a result of pathological changes in the myocardium.

Migration of the source of rhythm in healthy persons may result from an increase in the tonus of the vagus nerve. Among healthy persons (see Table 43) the percentage of persons with this form of rhythm disruption is somewhat higher than among athletes. At the same time, migration of the sinus rhythm may be a result of various intoxications or various types of inflammatory processes in the myocardium.

As concerns atrioventricular rhythm and its varieties, it occurs in cases of complete or partial block of the automatism of the sinus node. This may occur following any influences resulting in inhibition of the automatic functioning of the sinus node, as well as in cases of acute bradycardia (L. I. Fogel'son, 1957; A. M. Sigal, I. A. Chernogorov, 1962; etc.). The influences facilitating an increase in excitability of the atrioventricular node, in addition to those outlined above, include various intoxications and myocarditis.

It is believed that coronary sinus rhythm is encountered so rarely, even in pathological cases, that some authors (I. S. Shnitser, 1963) relat-d to to casuistry. However, as we can see from Table 44, this is not so. Coronary sinus rhythm may have a neurogenic origin, but may also depend on inflammatory or sclerotic changes, as well as ischemia of sectors of the myocardium in the area of the coronary sinus. In spite of the fact that the possibility of formation of atrioventricular rhythm, like coronary sinus rhythm, is obvious with vagotension, the question of their origin in athletes requires careful clinical analysis in each case in order to eliminate possible pathological changes of the myocardium.

Disruptions in conductivity. The percentage of athletes with arhythmia resulting from disruptions in conductivity is 3.66. A particular group among them is made up by those with first stage atrioventricular block, i.e. elongation of the P-Q interval above the upper boundary of the norm (P-Q > 0.21 sec).

It is well known that atrioventricular block may depend on an increase in the tonus of the vagus nerve. Most authors (O. V. Kachorovskaya, 1947; /422
S. P. Letunov, 1950, 1957; V. V. Bulychev, 1962; Herzum, 1940; Reindell, 1949; Slapak, 1956; Lepeschkin, 1957; etc.), considering that the increase in tonus of the vagus nerve is an inherent property of persons involved in sports, considered this disruption in rhythm as a peculiarity of the electrocardiogram of the athlete. However, we should not forget that the elongation of atrioventricular conductivity may be a manifestation of inflammatory or sclerotic processes in the myocardium, a result of

intoxication resulting from a center of chronic infection or, finally, dystrophy of the myocardium, arising upon overfatigue or overtraining. A case has been described of complete atrioventricular block, resulting from severe alcoholic intoxication (L. A. Kuvaldina, 1964).

According to the data of V. I. Kalyunov (1963), 39.1% of all athletes with elongation of the atrioventricular conductivity show this phenomenon as a result of some pathological process.

Thus, it is obvious that this type of arhythmia also requires clinical analysis.

The degree of elongation of the atrioventricular conductivity should not be used as a criterion for solution of the question of the genesis of this elongation (S. P. Letunov, 1957; Reindell, 1949; Lepeschkin, 1957; M. L. Proektor, 1964; etc.).

Differential diagnosis between physiological and pathological elongation of the atrioventricular conductivity can be performed using a test with physical loading and the injection of atropine (S. P. Letunov, M. L. Proektor, Reindell, Herzum, Kienle, etc.). It is believed that if the elongated P-Q interval is shortened under the influence of physical loading, this elongation should be looked upon as physiological, whereas the absence of any change in this interval or its further elongation, and especially appearance of Wenkebach-Samoylov periods, etc., indicate pathological origin of the elongation. However, a number of investigations (S. P. Letunov, Slapak, etc.) have established that elongation of the atrioventricular conductivity during physical loading may occur in absolutely healthy athletes, whereas in heart disease under these conditions it is frequently shortened. It follows from this that the physical loading test cannot be used as a sufficiently accurate criterion for clinical evaluation of elongation of the atrioventricular conductivity.

As concerns the atropine test, if 30-60 min following subcutaneous injection of 1 mℓ of a 0.1% solution of atropine a shortening of a previously elongated P-Q interval occurs, it follows that this elongation is a manifestation of vagotension. With organic damage to the myocardium, the effect of the injection of atropine should not occur. As we know, atropine eliminates the influence of increased tonus of the vagus nerve on the one hand, while expanding the vessels feeding the area of the atrioventricular node on the other hand and, by improving blood supply, normalizes its function. Thanks to this latter property of atropine, it has been found that patients with rheumatic heart damage also may show a shortening of the P-Q interval under the influence of atropine, since the blood supply to the conducting system of the heart is improved (S. G. Moiseyev, Lepeschkin, Messer, etc.). Thus, it is obvious that even this test does not allow reliable diagnosis of functional or organic genesis of elongation of the atrioventricular conductivity. /423

Solution of the problem requires in each individual case the performance of careful clinical analysis using both of these tests. M. L. Proektor (1964), after performing this clinical examination and using various tests for 200 athletes with elongated atrioventricular conductivity, showed that in only 35% could the elongation of the P-Q interval be evaluated as a manifestation of vagotension; in 37.5%, the elongation of the P-Q interval was a result of myocardial dystrophy, arising in connection with overtraining or intoxications from centers of infection; in 27.5%, the disruption of conductivity was a manifestation of myocarditic cardiosclerosis.

Complete block of the right branch of the bundle of His is seen in athletes in 0.18% of all cases, the same percentage as in healthy persons not involved in sports (see Table 43). According to the data of certain authors, this percentage reaches 0.74 among healthy persons.

Under certain unfavorable conditions, complete block of the right branch may occur as a result of inefficient sports exercises.

The following case is a confirmation of this.

Athlete L, 40 years old, is a class I marathon runner. He has no complaints. For the past three years, changes have begun to appear on his electrocardiogram (Figure 116), indicating overstress of the left ventricle. Negative T waves in the V_1 and V_2 leads and a two-phased wave in the V_3 lead (see Chapter V, section on "Electrocardiography"). The athlete was forbidden to train, but continued to train intensively. One month later (Figure 116) the electrocardiogram showed complete block of the right branch of the bundle of His. When training was completely halted (Figure 116) the block of the branch of the bundle of His disappeared.

As concerns partial block of the right branch in athletes, at the present time, due to the vector cardiographic method of investigation (M. B. Tartakovskiy, Zh. A. Teslenko) it has been shown that in the overwhelming majority of cases this electrocardiographic picture is explained by a delay in excitation of the right supraventricular crest (a muscular shaft located at the boundary of the anterior and posterior segments of the right ventricle), and is a variant of the norm.

The changes in the electrocardiogram described in 1930 by Wolf, Parkinson and White (the WPW syndrome) are characterized by a combination of a short P-Q interval (less than 0.12 sec) with an expanded QRS complex (over 0.11 sec). This change in the electrocardiogram is encountered in athletes in 0.62% of cases. It has come to be considered that this syndrome may appear in young persons without organic damage to the heart, although 50-70% of those persons with this syndrome suffer from paroxysmal tachicardia upon occasion. /424 /425

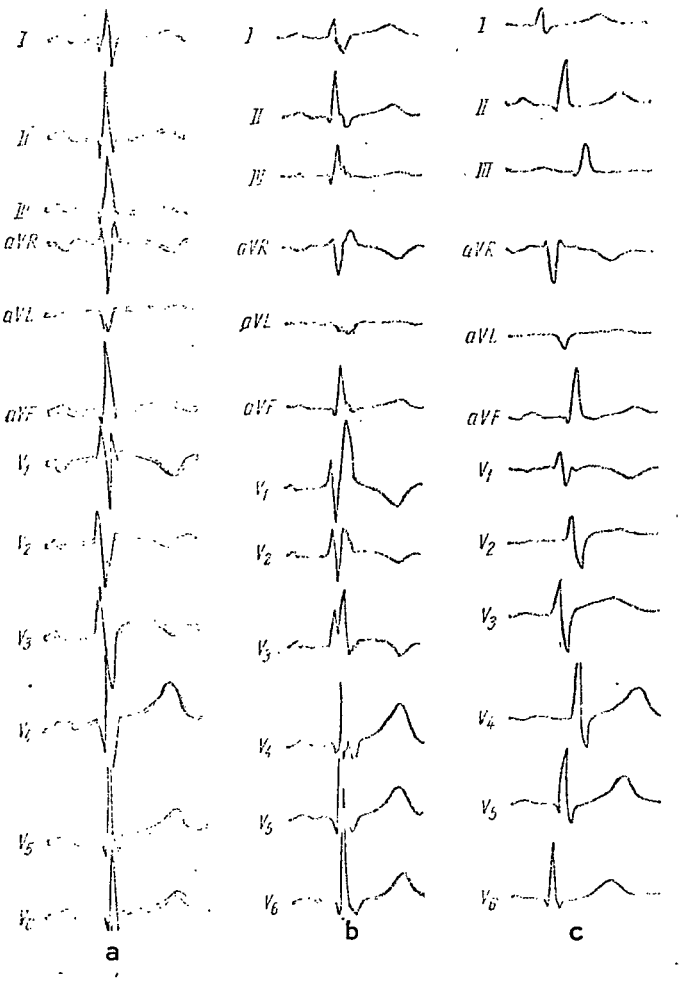


Figure 116. Electrocardiogram of Athlete L (Explanation in Text)

The WPW syndrome has also been described with various heart diseases -- atherosclerotic cardiosclerosis, myocardial dystrophy, various nervous influences, etc. According to the opinion of Z. M. Volynskiy et al. (1964), the WPW syndrome cannot be considered an "electrocardiographic curiosity," as is sometimes the case.

In athletes, individual cases of WPW syndrome have been described (S. P. Letunov, V. V. Bulychev; L. A. Butchenko and M. L. Proektor, V. A. Tishler; Doneff and Sheid; etc.). Most authors look upon this syndrome as a variant of the norm. However, cases have been described in which the WPW syndrome arose after physical overstress and when it has been worsened by the same factor. Cases have been described of the WPW syndrome arising after diphtherial myocarditis in athletes, as well as in persons not involved in sports (V. V. Bulychev, L. A. Butchenko and M. L. Proektor). Bachman (1957)

described two cases of WPW syndrome in athletes and assumed a congenital heart anomaly.

The literature contains indications that the WPW syndrome is a counterindication to acceptance into the armed forces (Monning, 1954; Schmitt, 1964). It is also believed (Friedberg, 1956; 1957; etc.) that physical stress and overfatigue provoke attacks of paroxysmal tachycardia in persons with WPW syndrome, so that they should not be allowed to take part in sports.

Thus, it is obvious that evaluation of the WPW syndrome in athletes in the solution of the problem of permission for training and competition requires great care, since this syndrome is polyetiological and polypathogenetic.

As concerns usage of a test with physical loading and the atropine test to answer the question of the functional or organic genesis of the WPW syndrome, the data are contradictory. Special investigations of athletes with the WPW syndrome which have been performed (L. A. Butchenko and M. L. Proektor) have shown that the reason for its formation in athletes may be either a change in the tonus of the extracardiac nerves, or cardiosclerosis, myocardial dystrophy, or perhaps even a congenital heart anomaly. As concerns the mechanism of development of this syndrome in athletes, it results either from an unusual reaction of parabiotically altered tissue in the conducting system of the heart or from a change in the functional state of the atrioventricular node and the interventricular septum, or the functioning of a congenital additional path between the atria and the ventricles.

One definite difference in the course of the WPW syndrome in athletes and nonathletes is the extreme rareness in the former of attacks of paroxysmal tachicardia. However, these athletes show deficiencies in sports /426
sports achievement, frequent overtraining, often combined with high or low blood pressure.

In solving the problem of whether to allow persons with WPW syndrome to take part in sports, we should primarily perform careful clinical examination. If the WPW syndrome arises from an organic heart disease or is combined with paroxysmal tachicardia, centers of infection or variations in arterial pressure, involvement in sports should be forbidden, since the case apparently involves organic heart damage. Of course, if the WPW syndrome disappears with treatment of the accompanying disease, involvement in sports is not counterindicated.

In cases when clinical investigation allows us to conclude that the WPW syndrome results from changes in the tonus of extracardiac nerves, involvement in sports can be fully allowed, since the syndrome can be considered a peculiarity of the electrocardiogram of the athlete.

In conclusion, we should emphasize once more the importance of the problem of proper evaluation of disruptions in athletes' heart rhythm, their etiology and mechanism of development -- a problem still far from solution and requiring study both by clinicists and by pathophysiologists and pathologists.

CHAPTER XIII

CARDIAC OVERSTRESS IN ATHLETES

ABSTRACT. Methods and techniques for the differential diagnosis of systolic noises are described. The configurations of systolic noises, as well as their responses to physical loading and amylnitrate, can be used for this diagnosis. The techniques outlined allow safe and effective differential diagnosis between accidental and functional noises.

It is hardly necessary to prove today the tremendous significance of /427
physical activity, and consequently physical culture and sports for the maintenance of health, the prevention of various diseases, including cardiovascular diseases and, finally, elongation of life.

Measured, effectively applied and properly used physical exercises are an irreplaceable part of the life of Soviet man. These same principles of the application of physical loads must be the basis of sports training. If they are ignored and ineffective, improper utilization of physical exercises, excessive or forced training without sufficient consideration of recovery, without individualization, etc. are allowed, the result will be untimely deterioration of the system and organs, the development of various pathological states, resulting in invalidism and sometimes death.

Clear examples of the correctness of these alternatives are, on the one hand, Stanley Matthews, who was a great football player at age 50 and the Indonesian rickshaw driver, who commonly died without reaching 30 years of age.

Physical overstress, in addition to the fact that it may be one of the factors in the development of hypertension in persons with predisposition to this condition, has an important influence directly on the heart. The influence of physical overstress on disruptions in heart rhythm has already been discussed. In this chapter, we will discuss the influence of physical overstress on the cardiac muscle.

First of all, we should distinguish between acute and chronic over- /428
stress. The influence of these types of overstress on the heart has an essential difference. Overstress arises when the demands determined by the physical loading and the capability of the organism for their fulfillment

do not correspond (S. P. Letunov, 1950). As a result of this noncorrespondence, clear pathological changes arise in the organism, principally in the heart, which may be the cause of sudden death or severe organic damage to the myocardium (myocardial infarction, myocardial hemorrhage, etc.).

If this noncorrespondence is not too great, reversible biochemical changes occur in the heart muscle, and the corresponding changes are noted in the electrocardiogram. However, if the physical overstress continues for an extended period of time, these reversible changes become irreversible.

With this understanding of physical overstress, it is obvious that it depends not on the absolute volume and power of the physical load, but only on the noncorrespondence of this load to the capabilities of the organism. Obviously, even an apparently small physical load may cause the same changes caused by an extremely large physical load in a trained athlete if the person undertaking the load is incapable of performing it. This fact is important in principle, since it expands the concept of physical overstress, carrying it beyond the limits of sports medicine alone. There is no difference in principle between the formation of various changes in the myocardium in a trained runner, who runs 25-30 km daily in his training, and an untrained person who runs after a bus at a rate too fast for his capabilities for only 300 m. In this case, there are no qualitative differences. The differences here are only quantitative, and the regularities involved in the development of pathological changes during physical loading of the athlete remain completely in force in any situation when the physical load exceeds the capabilities of the organism. This understanding of the significance of physical overstress in the formation of pathological changes in the heart is of particular significance at the present time for sports medicine due to the involvement of older persons in physical culture and sports.

Acute Cardiac Overstress

Acute cardiac overstress may manifest itself in various ways. Sometimes, acute cardiac deficiency arises during or immediately after a physical loading excessive for the person in question. The formation of acute cardiac deficiency is facilitated by various diseases (grippe, angina) during or immediately after which a physical loading is incurred, or the regimen is disrupted (alcohol, nicotine), or weight is lost in an inefficient manner before physical loading, etc.

The clinical picture of acute cardiac deficiency, like acute vascular /429
deficiency (gravitational shock, collapse, etc.) has no difference in principle from the same states encountered in persons not involved in sports. It has been described in detail in the corresponding literature, as have been the medical tactics to be used for these states (S. G. Vaysbeyn, V. K. Dobrovol'skiy, A. A. Luisada and R. M. Rosha, 1964; etc.).

Acute physical overstress may cause either local or diffuse changes in the myocardium. The literature contains descriptions of cases of sudden death as a result of physical overstress (G. P. Shul'tsev, 1962; M. I. Teodori and K. F. Vlasov, 1963; Schmid, Hornof, Kral, 1962; etc.).

Analysis of sudden deaths occurring as a result of diseases of the internal organs resulting from physical overstress, both among athletes and among persons not involved in sports, has shown that the main cause of death in these cases is damage to the myocardium, both that which occurs before physical loading, and that resulting from the overstress. This is facilitated by a combination of physical loading and either cold or heat. Since this problem has been very poorly covered in the literature, we should discuss it further.

All cases of sudden death from diseases of the internal organs among athletes can be divided into three groups:

1) participation in sports training with undiagnosed or underevaluated diseases, which are strengthened by the influence of the exercises and lead to death; as was already noted, the presence of this type of disease will not prevent the setting of sports records for a certain period of time, but sooner or later, when the compensatory mechanisms are exhausted, will appear and may be the reason for death;

2) ineffective, improper, excessive training, causing diseases which lead to death;

3) trauma to the internal organs, both manifest and concealed.

As concerns the cardiovascular system, the first group includes athletes with heart defects, both unrecognized and medically underevaluated, with cardiosclerosis resulting from rheumatic myocarditis or other etiology, etc.

Analysis of 30 cases of sudden death resulting from physical loads in persons between 13 and 32 years of age performed by G. P. Shul'tsev showed that in 12 cases the reason for death was an undiagnosed disease, including seven cases of heart disease. In two cases, the cause was early coronary atherosclerosis with thrombosis of the coronary vessels, and in one partial obliteration of the pericardium as a result of earlier rheumatic carditis, in /430 three cases -- cardiosclerosis of various etiologies, and in one case, hypertrophy of the left ventricle of indefinite etiology. In three of these seven cases, the autopsy revealed hypoplasia of the descending branch of the aorta. As concerns other causes, in two cases of sudden death, diffuse colloid goiter, undiagnosed in life, was the cause, and in two cases -- status thymico-lymphaticus, in one complete obliteration of the pleural cavities was discovered, facilitating the development of acute pulmonary-cardiac deficiency.

The second group of causes of sudden death, directly related to the physical overload, includes primarily so-called acute cardiac deficiency, as well as diffuse and local changes in the myocardium of various origins.

Acute anatomical changes in the heart appearing in cases of sudden death resulting from great physical stress vary considerably, although they are united by pathologists under the term "acute cardiac deficiency." In many cases, an autopsy reveals acute blood shortage in the heart, with full blood supply to the internal organs. It was earlier thought that this results from paradoxical reaction of the coronary vessels, contracting rather than expanding. In the light of contemporary concepts of the role of catecholamines in this type of case, we cannot exclude overexcitation of the sympathetic nervous system and accumulation in the myocardium of adrenaline-like materials such as noradrenaline. As the investigations of Raab have shown (1959), these materials increase the consumption of oxygen by the heart muscles to the extent that the coronary blood flow cannot supply it sufficiently.

According to the data of M. Ye. Raykina (1958), adrenaline does not increase the consumption of oxygen by the heart muscles, but rather decreases its consumption, in spite of the increased work. The formation of changes (decrease in the content of glycogen, increase in lactic acid and absence of formation of ATP) when adrenaline is introduced to the myocardium, in her opinion, result from the depressing effect of the adrenaline on the oxidative enzymes in the tissue and, consequently, result not from a deficiency of oxygen, as Raab assumes, but rather from insufficient oxygen assimilation.

The second group should include deaths of athletes resulting from acute interstitial myocarditis of Abramov-Fidler type, which, in the opinion of many authors, may arise upon physical loading if there are centers of chronic infection.

This same group must include cases of myocardial dystrophy with physical overstress.

As concerns local changes in the myocardium, we are concerned here with necroses which are of coronary genesis in some cases (myocardial infarct), while in other cases they are noncoronarogenic.

The problem of the possibility of noncoronarogenic necroses of the /431
myocardium is of great interest for sports medicine. At the present time, we know that myocardium necroses may occur not only as a result of spasm or mechanical blockage of coronary arteries, but also as a result of disruptions in the metabolism of the cardiac muscle, namely changes in the electrolyte metabolism, effects of toxic metabolic products, various hormonal influences, etc. It is obvious from this that the concept of "necrosis" is broader than the concept "infarct," since the latter is always a necrosis, whereas necroses are not always the result of infarction. Thus, we come to the concept of coronarogenic and noncoronarogenic myocardial necroses.

For the sports doctor, the question of noncoronarogenic myocardial necroses is important, and it is therefore suitable to discuss briefly certain aspects of this problem which may arise in sports medicine.

Without discussing the general classification of noncoronarogenic myocardial necroses (A. L. Myasnikov, 1962), let us indicate some of them.

The first group of noncoronarogenic necroses is related to anoxia of the the cardiac muscle of noncoronarogenic origin. They include anoxemic myocardial necroses, a result of arterial hypoxemia. These necroses have been produced experimentally in the pressure chamber, by carbon monoxide poisoning, etc., when the saturation of arterial blood with oxygen is significantly decreased. Necroses of this etiology may arise with the same physical stresses which are accompanied by sharp decreases in the saturation of arterial blood with oxygen, as well as at high altitudes (in aviation, alpinism). Amenic necroses are a result of the combination of anemia with physical stress. They have been experimentally produced, and this possibility should cause the sports doctor to be more careful concerning the composition of the red blood of the athletes.

Mechanical overloading of the myocardium may also be a cause of the formation of myocardial necroses. Thus, G. P. Shul'tsev has described five cases of sudden death with physical overstress resulting from hemorrhage in the cardiac muscle, clinically occurring as infarction. Four of these occurred during running and one during a bicycle race. In two cases, hemorrhages occurred simultaneously in other organs, while in the others small centers of necrosis and myocardial dystrophy were discovered. The presence of hemorrhaging in the cardiac muscle following physical loadings indicates the possibility of hemorrhage in the myocardium without a lethal result, but resulting in cicatrization, i.e. cardiosclerosis. Clinical observations confirm this possibility. Cases have been described in which athletes have experienced chest pains during stress (horizontal bar work, /432
wrestling, volleyball, etc.), accompanied by changes in the final portion of the ventricular complex in the electrocardiogram, indicating ischemia. These changes were quite rapidly liquidated. This type of change should apparently be evaluated as myocardial necrosis, arising as a result of mechanical overloading -- hemorrhaging or other necroses of noncoronary genesis. These necroses lead to cicatric changes in the myocardium, i.e. to cardiosclerosis. Finally, shock necroses have been described, resulting from vascular collapse, sometimes in athletes.

The second group consists of metabolic necroses, arising due to disruptions in feeding. They may arise with a low protein diet, as a result of excess fat intake, potassium deficiency due to various causes, or alimentary dystrophy. These disruptions in nutrition are particularly significant if they are combined with physical stress.

The group of necroses of hormonal origin is related to an excess of thyroxine, deoxycorticosterone in combination with an excess of steroid hormones and a potassium deficiency, as well as disruption of the

relationship of catecholamines, etc. This group of noncoronarogenic necroses is of particular interest for sports medicine, since all the factors causing their development occur to some extent in sports. This is particularly true of the catecholamines and steroids. We will discuss this problem in somewhat more detail below.

Finally, infectious and allergic myocardial necroses have been described. We are concerned here in particular with the possibility of formation of small necroses in the cardiac muscle after infections, particularly when training is restarted too soon. These myocardial necroses are also of essential significance from the standpoint of sports medicine.

The possibility of formation of noncoronarogenic necroses in athletes is obvious, particularly in connection with the usage of high training loads. However, this problem has not been discussed in the sports medical literature and sports doctors are insufficiently familiar with it. At the same time, when athletes of all ages die from various causes, autopsy frequently finds considerable cicatric changes in the myocardium without atherosclerosis of the coronary vessels and with no occasions of angina pectoris or myocardial infarction in the medical history.

The relationship of cardiac disruptions to physical loading is beyond doubt. Although there are some works in which the relationship between myocardial infarction and physical loading is placed in doubt, it is obvious at the present time that physical stress played an important role in the development of myocardial infarct in one fourth to one third of all heart attack patients (M. I. Teodori and K. F. Vlasov, 1963). Physical overstress is particularly significant in the formation of myocardial infarct if there is some degree of coronary atherosclerosis (M. I. Teodori and K. F. Vlasov, /433
Patterson, 1964, etc.). Considering the possibility of early development of coronary atherosclerosis in young persons, this factor should be kept in mind, particularly since in recent times cases of myocardial infarction in young persons have become more frequent (O. I. Yasakova, 1959; M. A. Gurvich, 1960; Z. M. Volynskiy, P. L. Sukhinin, 1961; etc.). Whereas earlier myocardial infarctions in persons between 30 and 40 years of age were considered casuistic, we now encounter them in 3-12.5% of cases (P. Ye. Lukomskiy and Ye. M. Tareyev, V. Ye. Nezlin, Ye. V. Shestakova, P. L. Sukhinin and V. M. Dvorkina, etc.). According to the data of Yu. D. Romanov (1963), who collected data on 15,666 cases of myocardial infarction, in 0.42% the age of the patients was less than 30 years, and in 4.95% -- less than 40 years. Eight hundred sixty-six cases were described of myocardial infarction of military servicemen of the USA between 18 and 39 years of age (Jater, Traum, Spring, 1948). Doubtless, infarctions have been occurring at younger ages, explained by the fact that the heart of modern man has become more vulnerable, sensitive and reactive.

In recent years, the number of acute myocardial infarctions resulting from physical overstress in athletes has also doubtless increased. S. P. Letunov (1957) observed only one case of acute myocardial infarction in

athletes in 15 years, and two cases of localized changes caused by physical stress (apparently, also myocardial infarcts).

Although we do not have precise statistical data on the frequency of myocardial infarction in athletes, analysis of the domestic literature (G. I. Shul'tsev, 1962; M. A. Gurvich, 1962, 1963; V. P. Kobzev, 1963; M. I. Teodori and K. F. Vlasov, 1963; etc.) indicates an increase. Thus, Ya. P. Griskin and N. F. Petukhov (1963) present a case of myocardial infarction in a 21-year-old athlete, arising after competition. V. P. Kobzev describes a fatal myocardial infarction in a gymnast 20 years of age, occurring 10 min after bar exercise. M. A. Gurvich reported on two cases of death from myocardial infarction: a boy 11 years old after skiing and a boy 16 years old after playing football. The principal etiological factor of involved in myocardial infarct in young persons, including athletes, is physical overstress (A. L. Myasnikov, 1963; etc.).

Also, the psychoemotional factor is tremendously important, particularly the combination of physical overstress with negative emotions. These factors may be the cause of myocardial infarct both with intact coronary arteries and with early coronary atherosclerosis. It should be kept in mind that there is no parallelism between the degree of coronary atherosclerosis and the tendency to myocardial infarct, and that one half of all cases of coronary atherosclerosis involves the so-called painless form (A. L. Myasnikov).

The appearance in recent times of a number of works on myocardial infarction in athletes confirms the significance of physical overstress in its origin. As we know, in physical labors, and consequently in athletes as well, the coronary arteries have more collaterals, and the heart muscle is more richly supplied with blood. This does not facilitate the development of myocardial infarcts (A. L. Myasnikov, 1962). At the same time, we know that physical activity decreases the content of lipids in the blood (N. L. Kipshidze, 1958; S. P. Letunov, 1962), which prevents the development of coronary atherosclerosis. Apparently, physical activity, increasing the energetic processes, creates the conditions for a more complete assimilation of lipids and their decomposition to the end products. All of this hinders the development of hyperlipemia and creates conditions which are unfavorable for the development of atherosclerosis. /434

However, excessive physical loading causes relative deficiency of the metabolism of the myocardium and, consequently, increases the threat of myocardial infarction, since the development of necrotic processes in the myocardium is facilitated (A. L. Myasnikov, 1962). The possibility of the occurrence of myocardial infarction resulting from neurohumoral functional disruptions of morphologically unchanged coronary arteries, namely extended spasm, is ever more widely seen. Closed trauma of the cranium, noted in the anamnesis of a number of young persons with myocardial infarct (according to M. A. Gurvich, in 9 of 70) may have some significance. It is also impossible to exclude the influence of nicotine, and in the opinion of many authors, of alcohol. Although Yu. D. Romanov (1963) believes that in most cases it is

impossible to relate the development of myocardial infarction in young persons with early atherosclerosis, a number of authors indicate that early atherosclerosis of the coronary vessels, although not clinically determined, has decisive significance. Frequently, the autopsy reveals one sclerotic patch -- the cause of coronary artery thrombosis. However, in this case, vasomotor disruptions have a significant role to play.

As one proof of the significance of sharp fluctuations in the tonus of unaltered coronary vessels and their spasm in the genesis of myocardial infarction in young persons, the fact is noted that if the infarction is not serious, coronary blood flow is rapidly and completely restored. However, it should not be forgotten that most authors note a more serious outcome of the myocardial infarction in young persons, although in general the specifics of the course of the myocardial infarction in these persons has not yet been studied sufficiently (A. S. Sidorovich, 1963).

It is interesting to note that of 67 patients with cardiac aneurism after myocardial infarction (R. N. Lebedeva and I. S. Kun, 1964), 11 were between 25 and 34 years of age. The formation of the aneurism depended on insufficient familiarity of the doctors with the possibility of myocardial infarction in young persons, as well as underevaluation of the severity of /435
the condition by the patients, resulting in delayed hospitalization, and failure to observe the subsequent regimen.

Thus, the possibility of formation of the myocardial infarct in athletes is beyond doubt. Although it does occur quite rarely, sports doctors should be aware of the possibility, should be capable of diagnosing an infarct properly, since these cases are relatively frequently fatal.

Gonsales et al. (1954) consider that the normal heart with undisturbed blood supply and undamaged myocardium and coronary vessels may stop as a result of overstress, and explain this by a combination of anoxia and heavy physical stress causing increased work of the heart (cited by M. Plots, 1961).

Finally, a third group of sudden deaths resulting from trauma of the internal organs relates essentially to the competence of the surgeons. However, we should note the possibility of traumatic heart damage after trauma to the thorax (A. L. Vilkovysskiy, 1950; P. M. Kireyev, 1950).

The following case is quite illustrative in this respect (S. M. Gusman, A. A. Itkin, K. S. Karanov). B, 22 years old, was hospitalized from 3/9 through 16/9 for helminthiasis, and was examined completely and pronounced healthy. One week later, while playing volleyball, he was struck sharply in the chest by the ball. He was sent to the hospital, complaining of sharp chest pains, dizziness and general weakness. Immediately after he was struck he lost consciousness briefly. Examination showed visible pulsation of the neck vessels, reinforcement of the apical thrust, enlargement of the boundary of the heart to the left. The pulse was 80-100 beats per minute, arterial pressure 130/0 mm Hg. A loud diastolic noise was heard at the third

intercostal space on the left, and over the femoral arteries a Duroziez/ double murmur and Traube double tone could be heard. The electrocardiogram showed a negative T wave in lead I. X-ray examination showed enlargement of the left ventricle. The diagnosis of aortal valve deficiency of traumatic origin was doubtless correct.

The literature contains descriptions of heart damage resulting from nonpenetrating trauma of the thorax, and this problem should be known to sports doctors. Thus, Uhlenbruck described a case in which, as a result of a strong blow to the anterior chest wall by a football, the intima of the coronary artery was separated, resulting in thrombosis of the coronary artery and death. A similar case, but without the fatal result, was described by M. Ya. Lapiner (1963). During a game, a football player was struck in the area of the heart by an opponent's leg, followed by pain and acute weakness. After three days bedrest, the athlete began to work out again, without having been examined. In one and one-half months, he was engaged in crosscountry running, and skiing up to 20 km. An electrocardiogram taken eight years later showed a scar in the area of the anterior wall and septum.

The problem of sudden death in sports has not been sufficiently studied. /436 For some reason, it is believed that the study of this problem could discredit sports. At the same time, each death in a hospital is the subject of a special analysis at pathological and anatomical conferences, while in sports medicine very little such study is performed.

These problems become quite important when middle-aged and elderly persons become involved in sports. Unfortunately, competition is held in these age groups in which the victory is determined by the time required to cover a distance. It is not by chance that the European Symposium on Protecting the Health of Elderly Persons (Kiev, 1964) demanded that competition involving excessive physical and emotional stress be forbidden for these persons.

It should be noted in fairness that in recent times the sports medical society has begun to take an active interest both in problems of the morbidity of athletes and in analysis of sudden deaths (N. D. Grayevskaya, 1964).

Chronic Cardiac Overstress

Chronic physical overstress is manifested in various ways in the activity of the heart. In 1956, S. P. Letunov described the clinical picture of the results of chronic physical overstress in a group of athletes. This group was characterized by various complaints: pain in the area of the heart, "intermissions," rapid fatigue. Also, a number of objective deviations were noted. Changes in the electrocardiograms indicated both diffuse and local changes of the myocardium.

As further investigations showed (A. G. Dembo et al., 1960), significant changes of the final portion of the ventricular complex on the electrocardiogram may occur in athletes with no complaints whatsoever, so that their evaluation represents considerable difficulty.

In 1962, Likoff, Segal, Dreifus described six healthy young persons with such changes in their electrocardiograms. The authors subjected them to detailed examination, including catheterization of the heart and coronary arteriography, determining no pathology. In that same year, Plas and Talbott published observations of 120 bicycle racers during a marathon race. in 15 of their subjects, the authors discovered changes in the electrocardiogram characteristic for myocardial ischemia. None of these athletes had any complaints, and seemed healthy upon examination. The authors gave no explanation for these changes. Concerning the decrease in the S-T segment and the negative T wave noted in the electrocardiograms of young persons, Bollinger (1964) writes that he has no explanation for the phenomenon. /437

Proper clinical evaluation of electrocardiographic indications of myocardial ischemia, i.e. changes in the final portion of the ventricular complex, is of essential significance at the present time in sports cardiology. This type of change is usually evaluated as a result of a disruption in the coronary blood supply. However, it has been found that these electrocardiograms are encountered as a chance finding among active athletes with no complaints, the percentage of which increases with each passing year. For example, whereas in 1955 in a study of many thousands of electrocardiograms of active athletes (L. A. Butchenko) 0.75% had such curves, in 1960 (A. G. Dembo, M. L. Proektor, R. D. Dibner, Zh. A. Teslenko) the percentage was increased to 1.5-2%, and in 1964 -- to 8-10% (A. G. Dembo, /438 M. L. Proektor, Zh. A. Teslenko). In other words, that which was once casuistic has become a problem.

These changes have been noted in clinical examination of highly qualified active athletes, mostly with no complaints. Figure 117 shows the electrocardiogram of an active athlete with typical changes of this type (a) and a patient of 56 years of age, in our clinic suffering from a myocardial infarct (b).

Both electrocardiograms show depressed T waves with double phasing in lead I, negative, one-sided T waves in leads II and III; the right precordial lead shows an elevated S-T sector and a negative T wave.

These changes could justifiably be used to diagnose coronary deficiency. However, whereas this interpretation fully corresponds to the clinical picture of the disease in the analysis of electrocardiogram (b), in the other case, since it belongs to an active athlete, a class I hockey player of 22 years of age, it is difficult to explain it as coronary deficiency. This athlete has no health complaints and shows no deviations from the norm in clinical examination. Thus, interpretation of these electrocardiogram changes as a result of myocardial ischemia does not fit the clinical picture although, it would seem, there is a formal basis for such a diagnosis.

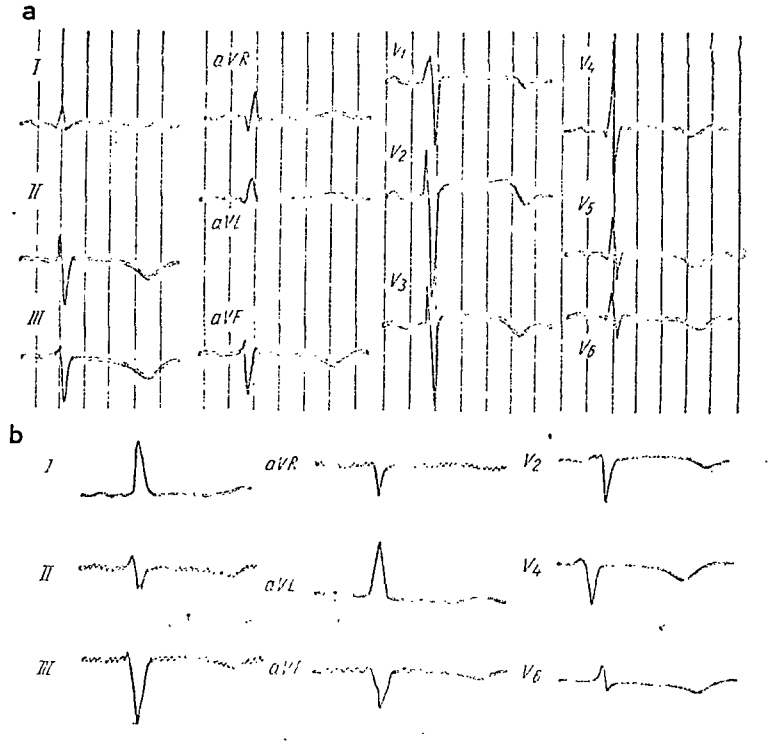


Figure 117. Electrocardiogram of Active Athlete G (a) and Patient S (b) (Explanation in text)

Detailed study of the problem of the origin of these electrocardiographic changes, based on data from the literature and analysis of our own material (about 150 electrocardiograms) has allowed some light to be shed on the genesis of these changes. First of all, it is obvious that they may have varying origins. They may be manifestations of coronary blood supply deficiencies, cardiosclerosis of various etiologies and pathological hypertrophy of the myocardium, frequently leading to uneven blood supply for various sectors of the cardiac muscle.

Finally, they may be a result of cardiac overstress. Clinical practice has long known of acute changes (the heart strain syndrome). These changes arise in the left ventricle with acute nephritis, when as a result of a sudden increase in arterial pressure of the left ventricle it must overcome a sudden large resistance, without being prepared for it. These changes also develop with acute pulmonary heart, in which as a result of embolism of the pulmonary artery, the pressure in the respiratory circulation increases sharply. These changes can result in a diagnosis of myocardial infarct, which is generally not confirmed by autopsy (I. Chernogorski, 1963).

It is natural to assume that the reason for changes in the final ventricular complex of the electrocardiogram revealed without direct connection to physical loading is chronic cardiac overstress, arising as a result of the long-term effects of heavy physical loadings, and, consequently, these changes are not related to disruption of coronary circulation. Some confirmation of the possibility of formation of these changes in the electrocardiogram as a result of chronic overloading of the left ventricle can be found in a number of clinical observations of patients suffering from hypertension in which reserpine treatment and decreased arterial pressure have resulted in disappearance of the electrocardiographic indications of myocardial ischemia. /439

Careful clinical study of all athletes with changes in the final portion of the ventricular complex of the electrocardiogram, in which vector cardiography can be of tremendous diagnostic significance, has confirmed the idea that this group includes persons with coronary deficiency, with pathological left ventricular hypertrophy and with cardiosclerosis. However, in most cases (80-85%) these changes in the electrocardiogram are manifestations of cardiac overstress resulting from chronic physical overloading.

These concepts can be illustrated by the following examples.

1. Athlete Sh, 23 years old, master of sports in football with eight years sports experience, was sent for consultation from Odessa in 1959. Since 1955, he had complained of dull pains in the chest, but continued training and competition. In May of 1958, he was struck by severe chest pains. Since this attack was accompanied by nausea, the doctors considered that it was food poisoning. Although the pains in the chest increased, the athlete took part in 70 games in the fall of 1958 and continued training intensively.

An electrocardiogram was finally taken on 17/9/1958 due to an increase in the attacks, after which the athlete was hospitalized (Figure 118). After 1 1/2 months hospitalization, the electrocardiogram (12/11/1958 and 3/1/1959) improved.

After release from the hospital, Sh once more began training, as a result of which the pains returned and he was sent to Leningrad for Consultation. Objective examination revealed, in addition to weakening of tone I at the apex, no pathological signs. The reaction to the functional test was good. Blood cholesterin content was 120 mg %. X-ray examination revealed slight enlargement of the left ventricle. The electrocardiogram (taken 12/5/1959) showed negative, equal-sided T wave in leads II and III, upon inhalation, in the aVF lead and monopolar precordial lead from V to V_s, a two-phased T wave in lead V_6, depression of the S-T segment below the isoline in leads II, V_6, V_7 and aVF. After two minutes running in place, a weak paradoxical reaction was noted (positive T wave in leads II and aVF). The electrocardiogram taken during 50 deep knee bends showed electrical

alternation of the T waves. Diagnosis: hypertension in the neurogenic stage, coronary angioneurosis (atherosclerosis of coronary vessels not excluded), angina pectoris. Sh was forbidden to continue his involvement in sports and extended sanitarium treatment was recommended. Treatment in a cardiological sanitarium and cessation of physical exercise improved the athlete's health, resulting in disappearance of the pains in the chest. Electrocardiograms taken in July and August of 1959 showed elimination of the negative T wave in lead II and the monopolar precordial lead, replaced by a positive, two-phased, then weakly positive recording in lead aVF. In the fall, the athlete began working as a trainer, and was sometimes disturbed by sudden general weakness. The electrocardiogram taken on 20/11/1959 was unchanged from that taken in August. /441

These changes in the electrocardiogram were evaluated as manifestations of coronary circulation deficiencies in the hypertrophic left ventricle, most clearly shown in the area of the posterior wall of the left ventricle as a result of coronary vessel spasm. This is indicated by the typical sharp, equal-sided negative coronary T waves, which disappeared after extended sanitarium treatment and cessation of physical loading. The electrical alternation which occurred during performance of 50 deep knee bends can indicate deep disruption of metabolic processes in the myocardium under the influence of disruptions in coronary circulation. /442

2. Athlete M, 22 years old, boxer, master of sports, has no complaints, feels fine. In September of 1959 during clinical examination, X-ray studies showed hypertrophy of the left ventricle, accompanied by a slight increase in arterial pressure (150/75 mm Hg) and changes in the electrocardiogram (Figure 119), namely: negative T waves in leads I, II, aVL and V_{3-5}. In spite of a decrease in training intensity, the changes in the electrocardiogram remained.

Analysis of this case led to the conclusion that the electrocardiographic data should be evaluated as a manifestation of pathological hypertrophy of the left ventricle.

On the vector cardiogram (Figure 119), the third period of development of hypertrophy of the myocardium of the left ventricle was determined, characterized by an enlargement of the QRS loops, displacement of the T loop in the coordinate system and unclosed QRS and T loops.

3. Athlete B, 17 years old, kayak rower, unclassed (involved in the sport for two years). The electrocardiogram (Figure 120) showed the following changes: negative T wave in leads II, III, aVF and precordial leads beginning with V_3. Repeated electrocardiographic investigation indicated absence of change. Vector cardiographic data did not indicate hypertrophy of left ventricle; disruptions in repolarization process is noted. Anamnesis indicated extended subfibrility following highmoritis at age 13, tonsillectomy at same age. Following two months bedrest, the patient was sent to the rheumatological sanitorium with a diagnosis of rheumatic /443

myocarditis. At age 15, subfibrility and vague pains in the joints. Objectively: dull heart tones, tone I at apex weaker than II. All of this allows this case to be interpreted as myocarditic cardiosclerosis.

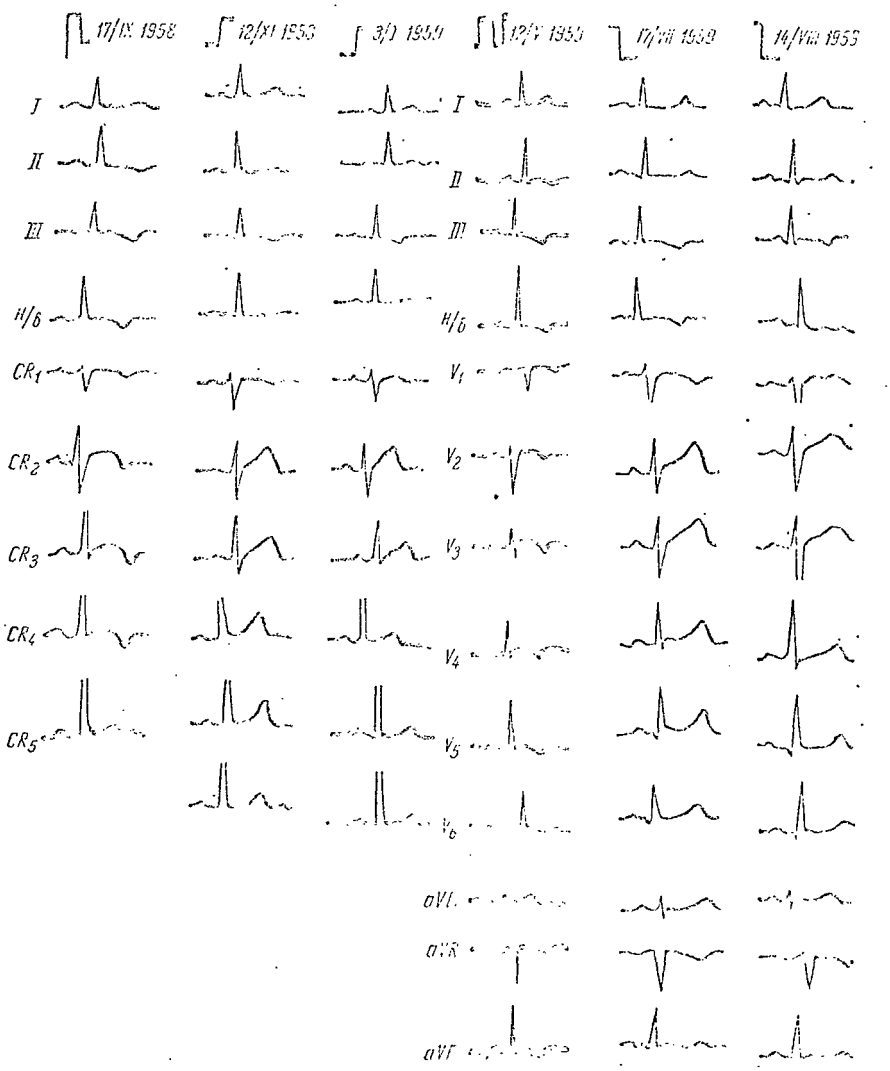


Figure 118. Electrocardiogram of Athlete Sh (Explanation in text)

The reason for the development of cardiosclerosis in athletes may be not only myocarditis, but also fine hemorrhages, centers of necrosis, arising during acute physical overstress. As we can see from the observations described, the role of vector cardiography in the differential diagnosis of this type of condition is difficult to overestimate.

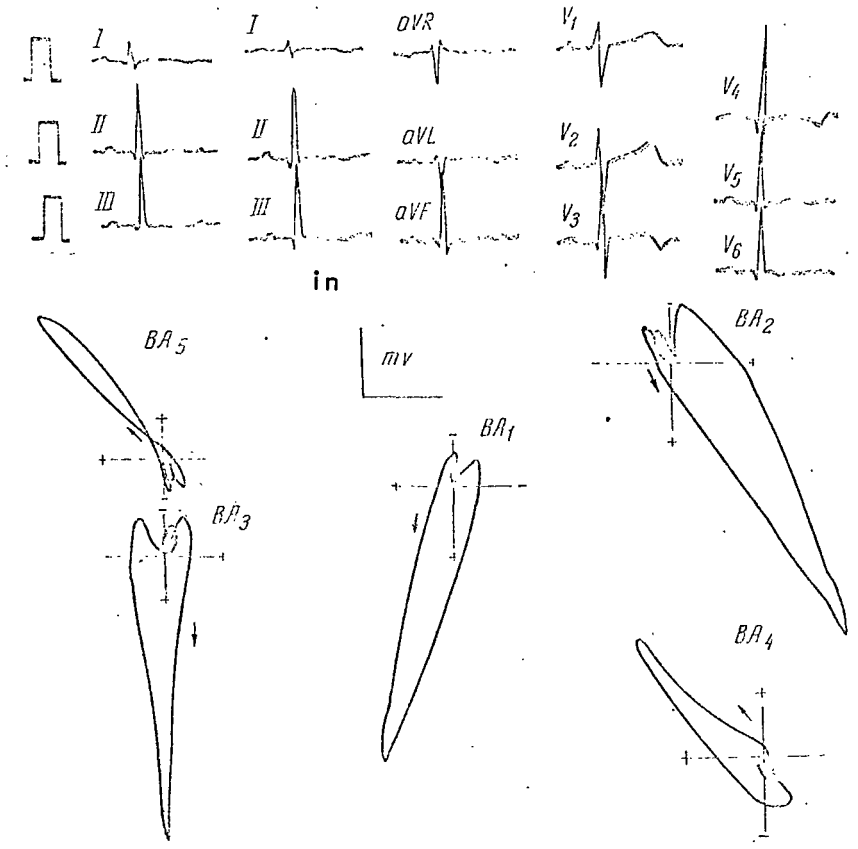


Figure 119. Electrocardiogram and Vector Cardiogram of Athlete V (Explanation in Text)

As concerns athletes with cardiac overstress, it should be emphasized that at the beginning of the development of these changes, they cause no pain, and the electrocardiographic changes will be chance findings. In most cases, these are promising, active athletes, who train a great deal and show excellent results. Unfortunately, this last fact has resulted in a tendency to interpret these changes in the electrocardiogram as so-called peculiarities of the athlete, and in many cases the proper significance has not been attached to them.

Table 46 shows data on the frequency of cardiac overstress in representatives of various types of sports.

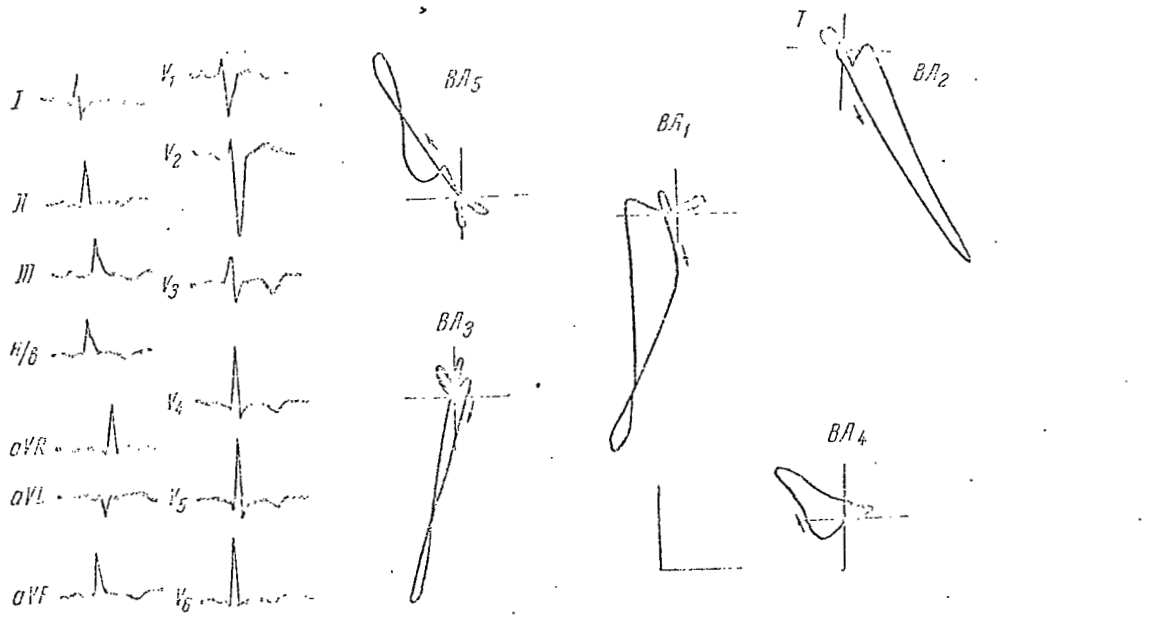


Figure 120. Electrocardiogram and Vector Cardiogram of Athlete B (Explanation in text)

The proof that the reason for the development of the electrocardiographic indicators of cardiac overstress is physical loading lies in the analysis of the factors present when the signs arise, the disappearance of these changes when physical loading is halted and the frequent repeated occurrence of the signs if training is begun again. /445

There are three main causes leading to the appearance of changes in the final portion of the ventricular complex of the electrocardiogram as a result of chronic physical overstress:

1) extreme training and competition loads, not corresponding to the capabilities of the athlete (this latter factor is most important);

2) intensive, but not excessive physical loading if improperly organized, i.e. also acting on an organism not prepared for it;

3) intensive, but not excessive loading combined with intensive work or study or if performed during or soon after any type of disease (grip, angina, etc.).

The presence in athletes of centers of chronic infection (chronic tonsillitis, cholecystitis, dental caries, etc.) aids in the formation of cardiac overstress in all three cases. The first factor is illustrated by the following examples.

TABLE 46. DISTRIBUTION OF ATHLETES WITH CARDIAC OVERSTRESS BY TYPE OF SPORTS

Type of sports	Percentage of athletes investigated in this type of sports	Type of sports	Percentage of athletes investigated in this type of sports
Marathon runners	8,5	Motor racing	3,1
Basketball	8,5	Sports gymnastics	2,2
Football and hockey	6,1	Weight lifting	2,0
Skiing	5,3	Fencing	1,9
Bicycling	4,3	Boxing	1,8
Volleyball	3,8	Wrestling	1,7
Rowing (academic and kayak)	3,5	Swimming	1,7
Skiing	3,3	Light athletics	1,2
Tennis	3,3	Other types	1,9

Tr. Note: Commas indicate decimal points.

1. Athlete Zh, 21 years old, clinically examined at the Leningrad Medical-Physical Culture Dispensary after very intensive physical loading. No deviations in the state of health determined by detailed clinical examination. The electrocardiogram (Figure 121) shows clear changes -- compression of the T_I and T_{II} waves, changes in the T wave in leads V_{3-6}. After three days of complete rest, complete normalization of electrocardiogram was noted.

2. Football player, master of sports K, 23 years old. In July of 1960, electrocardiogram quite normal (Figure 122). In November of the same year, i.e. after an intensive season, the electrocardiogram shows: negative T wave in leads I, II, III, aVF, V_5 and V_6, two-phased T wave in aVL lead and positive T wave in aVR lead, i.e. changes occurred on the posterior lateral wall.

3. Athlete L, 16 years old. Talented tennis player, played in adult team. The electrocardiogram of 4/5 (Figure 123) shows changes indicating damage to anterior lateral and posterior wall of left ventricle, namely smoothed T wave in lead I, depression of S-T interval below isoline and flat

T wave in leads II, aVF and V_6, negative T wave in leads III and V_3 and 5. After complete rest, electrocardiogram is normalized (14/2/1962). Unfortunately, the athlete began training again before permission was granted, and changes on electrocardiogram reappeared (17/11/1962).

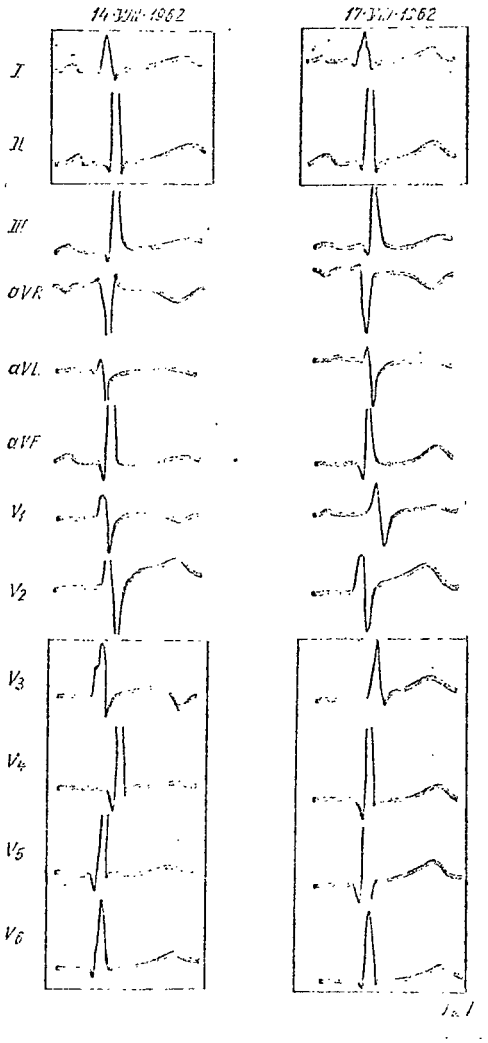


Figure 121. Electrocardiogram of Athlete Zh (Explanation in Text)

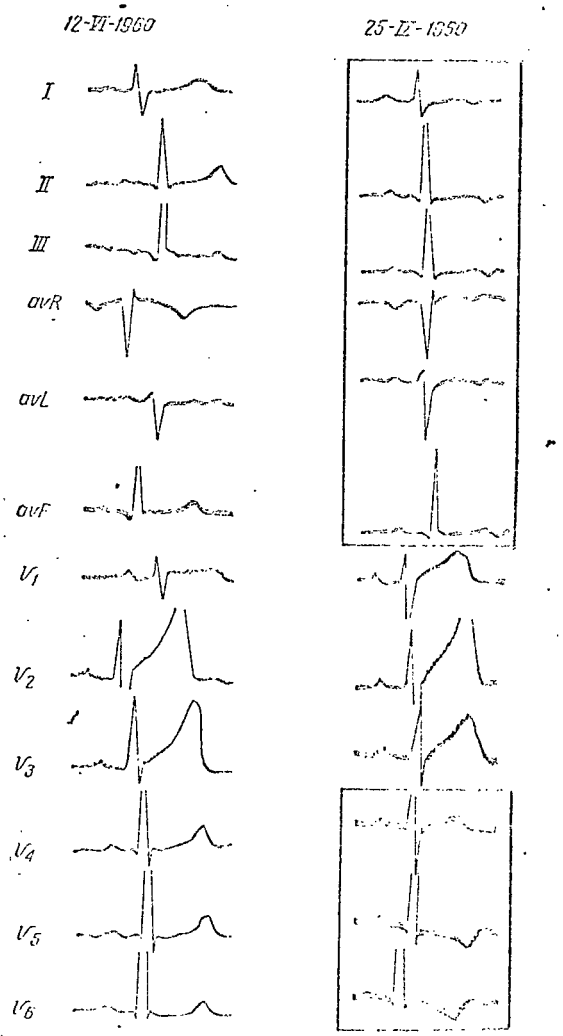


Figure 122. Electrocardiogram of Athlete , (Explanation in Text)

The following examples illustrate the second cause of overstress syndrome.

Gymnast first class M, 28 years old. Healthy, with normal electrocardiogram, traveled to alpine camp and trained intensively as a mountain skier. After this training, the electrocardiogram showed changes indicating cardiac overstress: T wave smoothed in leads I and V_6, negative in leads II,

III, aVF and V_{3-4}, two-phased in lead V_5 (30/3/1962). This indicates damaged /448 anterior and posterior lateral wall. After rest, electrocardiogram normalized (6/7/1962) and the athlete returned to training as a gymnast (Figure 124).

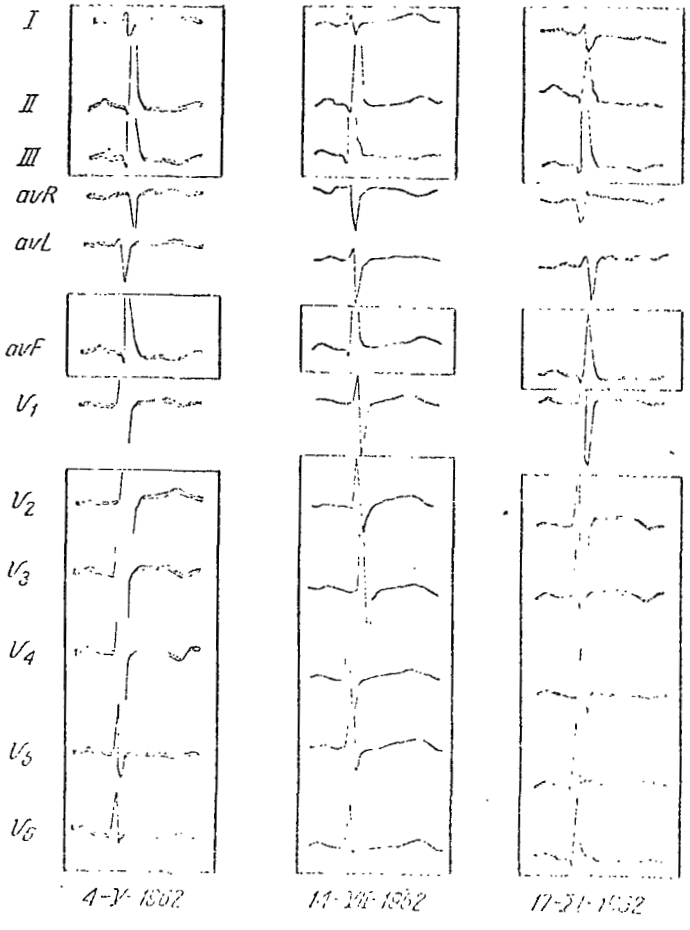


Figure 123. Electrocardiogram of Athlete L (Explanation in Text)

An example of the combination of loading with infection is the electrocardiogram of wrestler T, 17 years old. He began training two days after running a high temperature (grip). The electrocardiogram (Figure 125) shows changes in the anterior septal area, a decrease in the T wave in leads II, V_{4-5}, an isoelectric T wave in leads I and aVL and negative T wave in leads $V_{2,3}$ (17/3/1962). After training was halted, the electrocardiogram was normalized (15/3/1962).

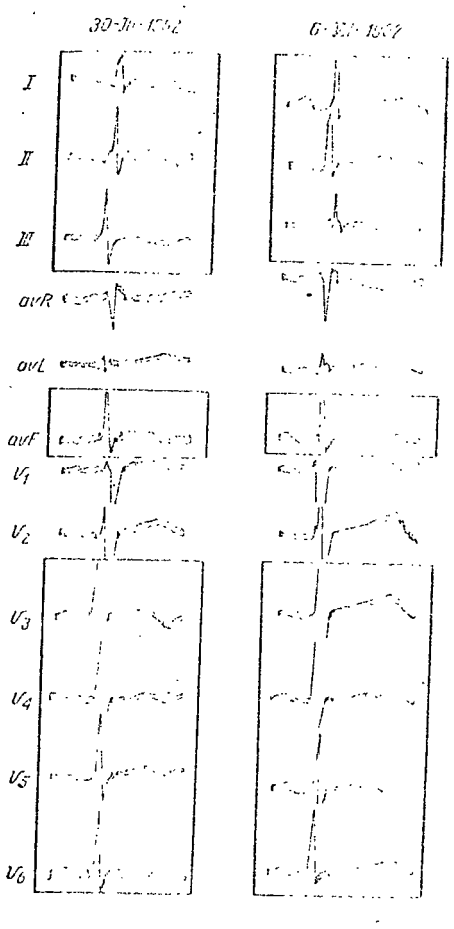


Figure 124. Electrocardiogram of Athlete M (Explanation in Text)

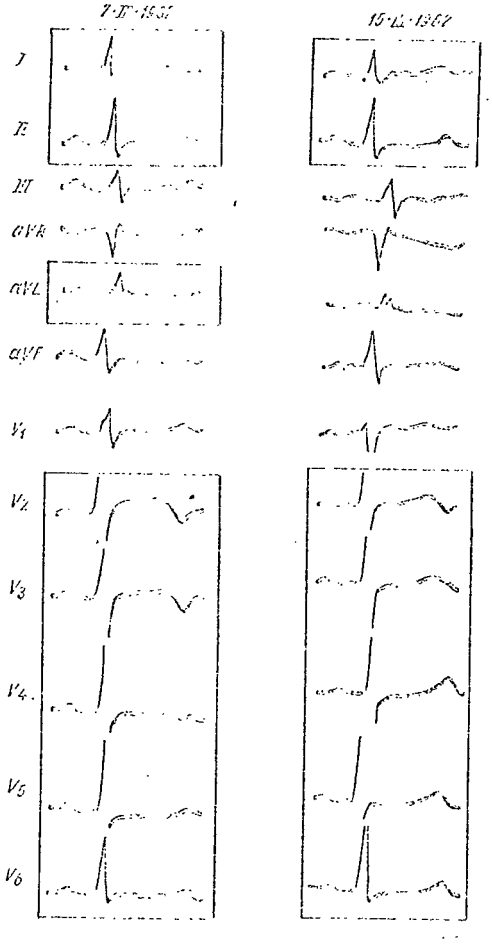


Figure 125. Electrocardiogram of Athlete T (Explanation in text)

The study of the trends in the development of the electrocardiographic changes in chronic cardiac overstress has allowed us to establish three stages of their development, occurring variously depending on the localization (Figure 126).

The first row across the diagram shows the normal electrocardiogram. If changes occur in lead I, first of all the T wave is depressed or dualized, followed by a depression in the S-T interval, the T wave becomes two-phased, /449 and subsequently negative. In leads III and aVF, the T wave becomes isoelectric or depressed, then the S-T interval is depressed, and the T wave becomes two-phased and then isoelectric. In leads V_{2-3}, first the S-T

interval is elevated, the T wave is dualized, then becomes two-phased and negative. As concerns the left precordial leads, the changes here are analogous to the changes in leads I and II.

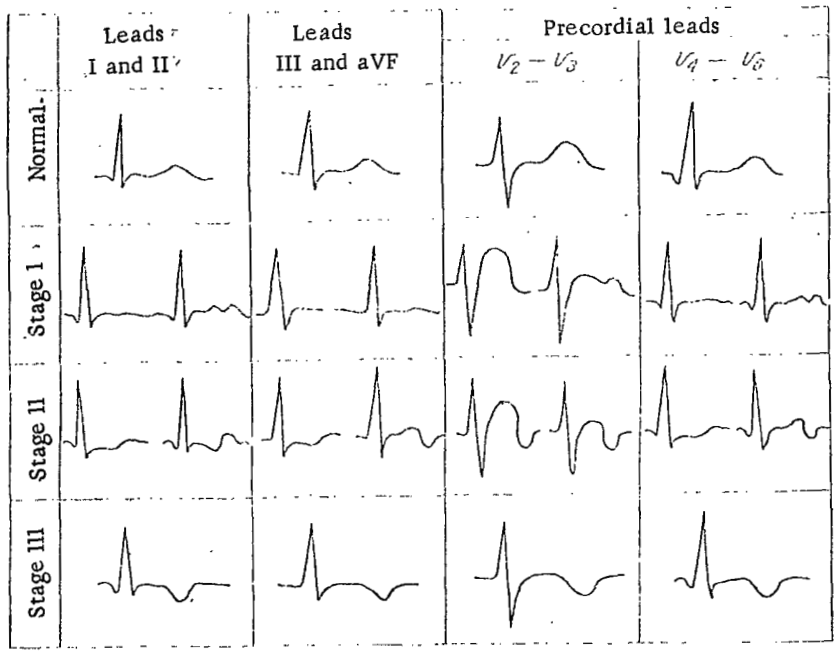


Figure 126. Diagram of Development of Changes in Electrocardiogram During Cardiac Overstress Syndrome

The changes in various leads are frequently combined, then their development passes through the three stages just described variously. Most frequently in an identical percentage of cases (46%) these changes are encountered on the posterior and anterior walls, less frequently (5%) they are localized only in the septum: total changes are encountered in 3% of cases. /450

The following figure (Figure 127) shows the nature of the changes and paths of their development. If the primary changes are noted in the posterior wall (a), usually the next area involved is the anterior lateral wall, followed by the septum. If the first damage is located on the anterior wall (b), changes then occur in the lateral wall. If the first changes are noted in the septum (c), they are next noted in the left precordial leads and encompass all segments of the heart. The final stage of these changes, i.e. total changes, is shown on Figure 127, c. /451

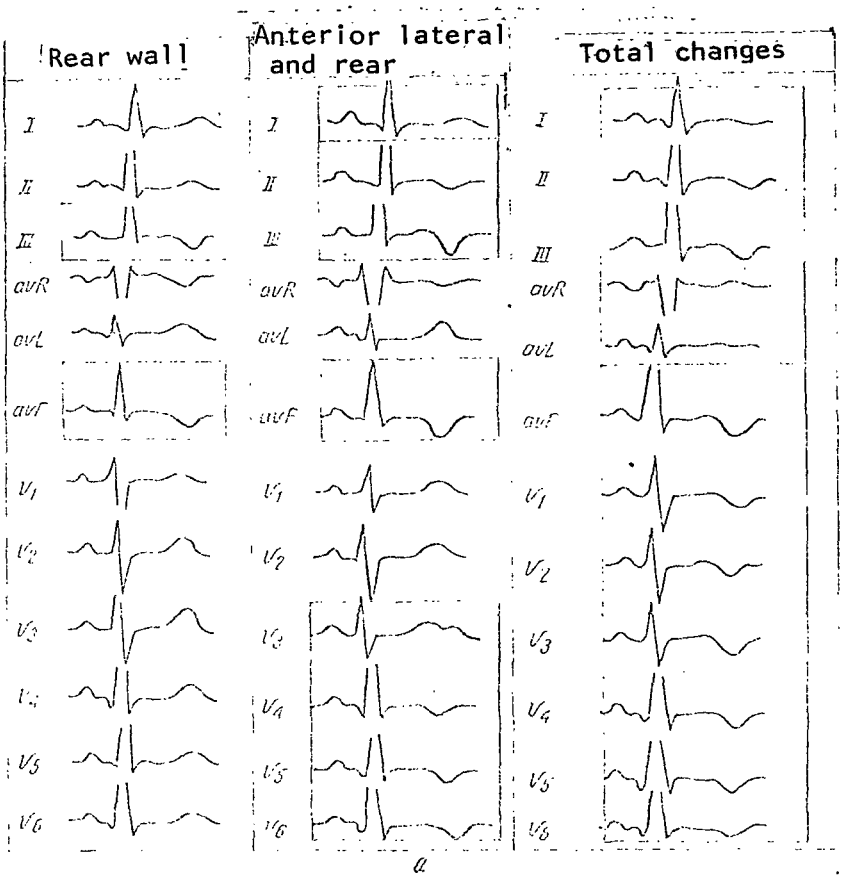
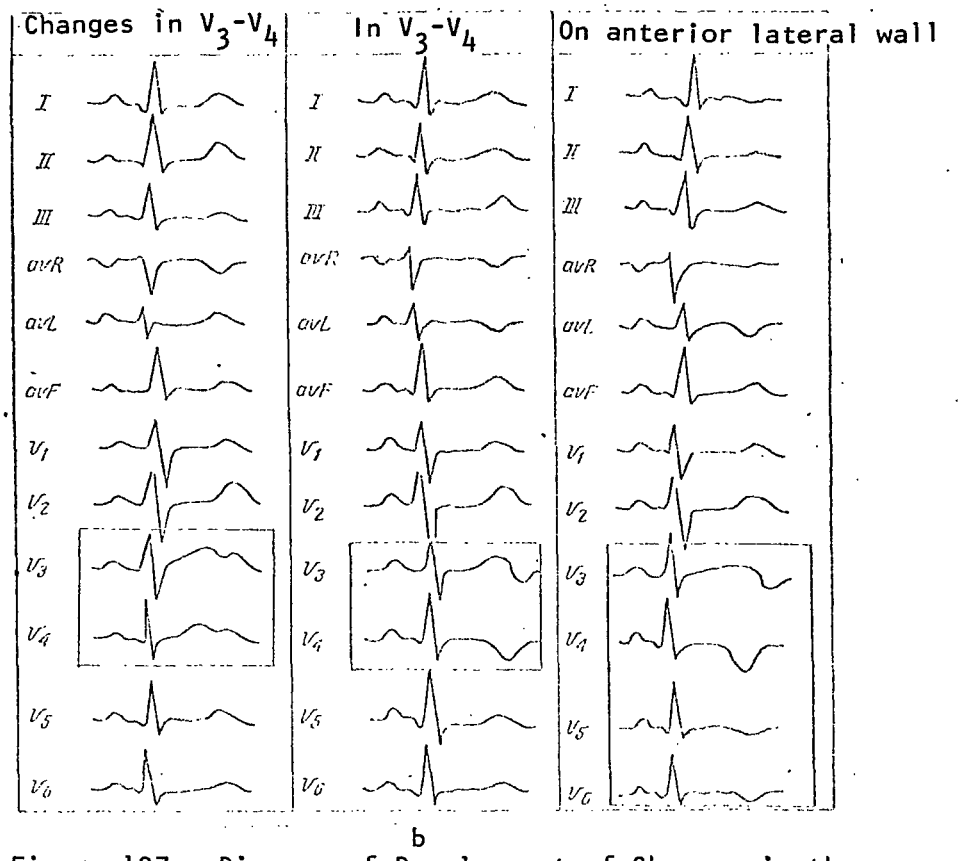


Figure 127. Diagram of Development of Changes in the Electrocardiogram with Cardiac Overstress Syndrome As a Function of Localization. а, With primary damage in area of rear wall

These are the types and stages of electrocardiographic changes characteristic for the chronic cardiac overstress syndrome. Their establishment /452 has both theoretical and great practical significance. We speak here of the importance and necessity of diagnosing the early stages of chronic overstress, i.e. proper interpretation of electrocardiographic changes, to which insufficient attention has yet been turned. Also, early diagnosis of these changes, their proper evaluation and application of the corresponding steps are of decisive significance in their liquidation and maintenance of sports performance ability. If insufficient attention is turned to these changes, as will be shown below, complete disengagement from sports must result.



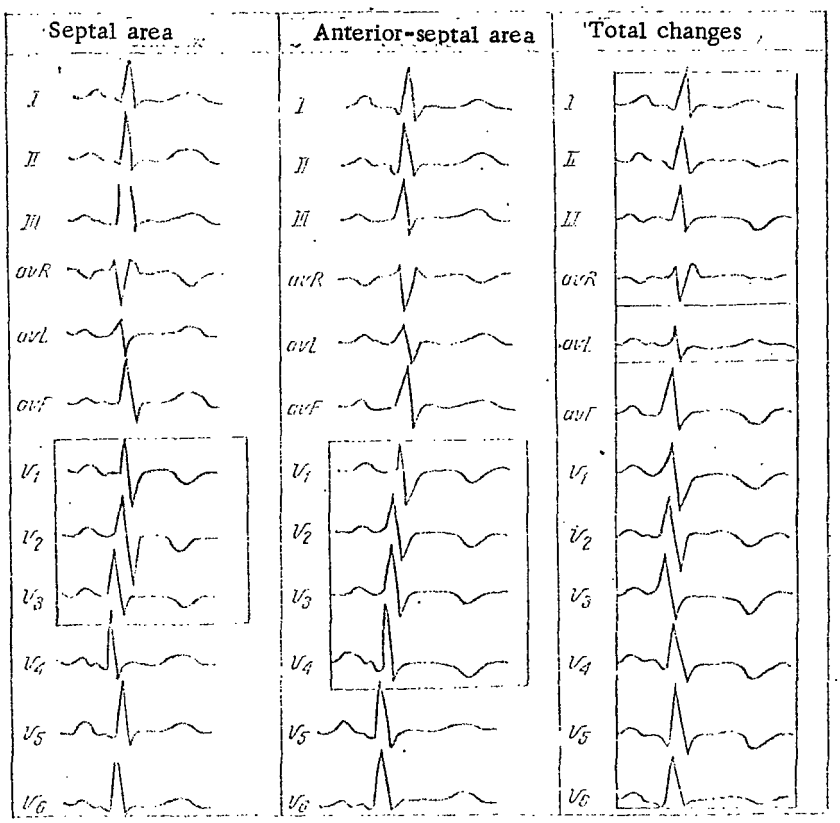
b

Figure 127. Diagram of Development of Changes in the Electrocardiogram with Cardiac Overstress Syndrome As a Function of Localization. b, With primary damage in area of anterior wall

The principal factor in normalization of the electrocardiogram is complete cessation of exercise with physical loads and sufficient duration of this interruption. The earlier the cardiac overstress is diagnosed, the less the initial changes and the better, more rapidly and more completely the normalization of the electrocardiogram occurs, and most important, the more stable is this normalization. In most cases, normalization is achieved, but the time required for this normalization to occur is measured in weeks or months. This is the best variant of the result of the overstress syndrome.

Thus, it is obvious that when the above-noted changes are observed in active athletes, in spite of the fact that the athlete has no complaints and is setting records, it is necessary to halt training completely. Experience has shown that a partial cessation of training is not effective -- deterioration unavoidably results. Complete cessation of training usually leads to normalization of the electrocardiogram, and resumption of training can only

be considered after complete normalization. Resumption of training with incomplete normalization always causes deterioration. Training should be resumed quitre carefully, and the load level should be increased gradually under careful medical observation and electrocardiographic control. When these steps are taken, in a significant portion of cases the normal electrocardiogram can be retained while increasing the intensity of training loads, i.e. complete sports performance ability can be maintained.



c

Figure 127. Diagram of Development of Changes in Electrocardiogram with Cardiac Overstress Syndrome As a Function of Localization. c, With first damage in area of septum

Athlete V, 33 years old, master of sports in canoeing. Sports experience 15 years. During the summer of 1960, he trained quite intensively, combining rowing with running. During a pre-Olympic contest, while continuing intensive training, he failed to observe his rest regimen. A staged, hypotensive reaction to loading was noted, along with a slight increase in arterial pressure (130/80 mm Hg). Objectively -- some dullness of the heart tones, moderate hypertrophy of the left ventricle.

The electrocardiogram taken in February of 1960 showed no deviations from the norm; in July of 1960, during intensive training before the Olympic Games, negative T waves were noted in the aVF lead, along with displacement of the S-T segment above the isoline in leads V_{2-5} with transition to negative T wave. Three to four weeks after interruption of training, normalization of the electrocardiogram was noted. After 1 1/2 months, V resumed training, and according to the information available, his electrocardiogram is still normal. Vector cardiographic data confirm the correctness of the interpretation of the electrocardiographic changes as overstress of the left ventricle. /453

The improper understanding of the significance of these changes and failure to take timely steps to correct them unavoidably lead to progressive worsening of these changes and threaten the capacity to reverse them.

A second possible result is normalization after cessation of training, but unavoidable reappearance of changes when physical loading is resumed.

Athlete K, master of sports in football, 22 years old. Sports experience eight years. No complaints, feels fine. In the past, he had scarletina, inflammation of the middle ear (recurring each year from three to twelve years), angina. During the last year he has twice suffered from angina (January and August) but paid no attention to it and continued training. Objectively: dull heart tones, inclination to increased arterial pressure (to 150/90 mm Hg). Fundus oculi shows moderate degree of constriction of retinal vessels; dental caries. X-ray examination shows moderate hypertrophy of left ventricle. In February of 1960, overtraining occurred, during which changes appeared in the electrocardiogram, which had been normal up to that time (Figure 128). We can see depression of the T wave in lead I, two-phased T wave in leads II, III, aVF and aVL, negative T wave in V_4 and V_6 leads. Training was halted. The electrocardiogram taken in May of 1960 shows improvement, while in July complete normalization is noted. The athlete resumed training that summer without permission of the doctor. Electrocardiographic investigation at the end of the sports season (September 1960) once more showed changes in the electrocardiogram similar to those noted in February.

In these cases, the changes in the myocardium do not retain their completely reversible nature, since they are more marked than in the first case, apparently as a result of their delayed discovery.

Therefore, in the second variant of the result, we must first of all increase the rest time, and the athlete should resume the training process gradually. If, in spite of this, the changes in the electrocardiogram return, involvement in sports must be forbidden and physical culture must be continued under careful medical observation.

The same decision should be made in those cases when the electrocardiographic changes do not disappear, in spite of complete and extended cessation

of training. In these cases, we are involved with the transition of what were reversible changes in the myocardium into irreversible changes. The absence of positive dynamics of the electrocardiogram generally occurs with a combination of cardiac overstress and pathological hypertrophy of the left ventricle. This combination is observed in approximately 20% of the cases of chronic cardiac overstress.

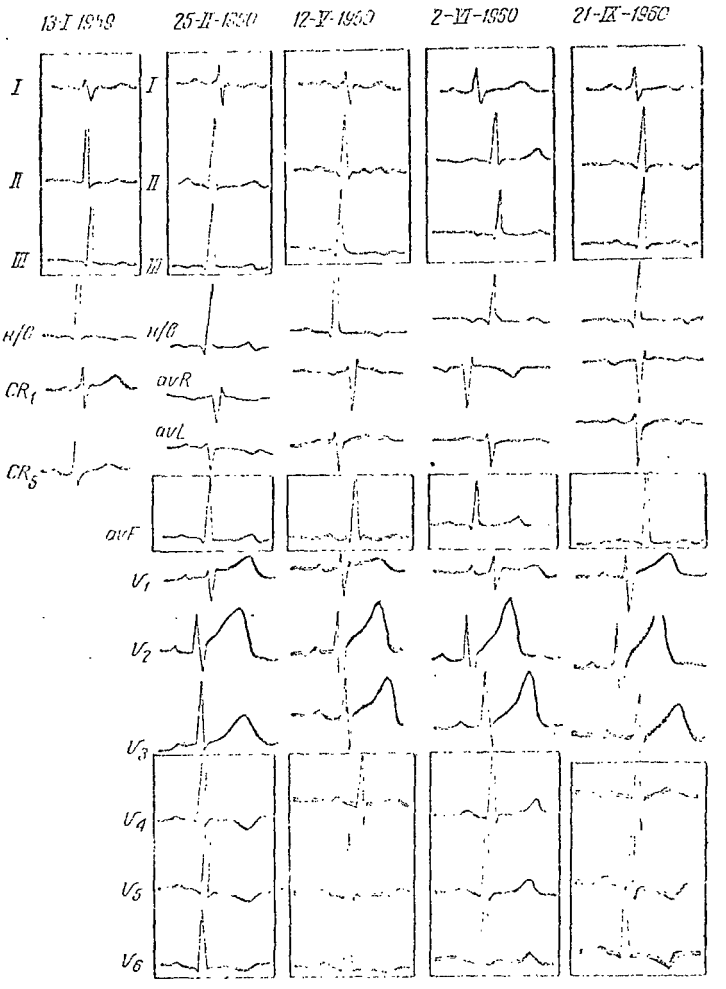


Figure 128. Electrocardiogram of Athlete K. Explanation in text.

It is obvious at the present time that failure to take the proper measures and continuation of training with the overstress syndrome unavoidably leads to stable changes in the myocardium, gradual appearance of a number of complaints and later to the development of circulatory deficiency. All of this, considering the increase in the number of cases of overstress syndrome in athletes, requires proper and early diagnosis by the doctor and /455

forbids a careless attitude toward these changes, based on the absence of complaints and the achievement of high sports results, often attained by these athletes.

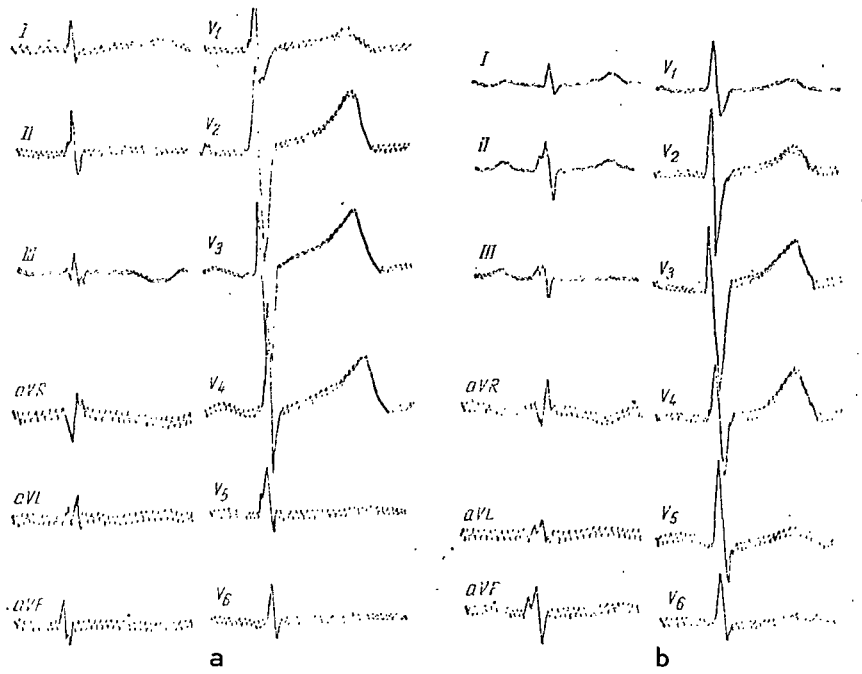


Figure 129. Normalization of Pathologically Changed Electrocardiogram After Two Minutes Run. a, Before loading; b, after loading. Explanation in text.

It should be kept in mind that under the influence of physical loading, changes in the electrocardiogram of the overstress syndrome type frequently can be normalized. For example, Figure 129 shows that a two-minute run in place at 180 steps per minute liquidates the changes in the electrocardiogram.

Similar changes in the electrocardiogram, i.e. normalization in athletes with cardiac overstress syndrome, occur when the hypoxemic test is run (Figure 129). At the same time, athletes with normal electrocardiograms generally show certain negative changes when the level of arterial blood /456
saturation with oxygen is reduced to 60-70% of the norm. In other words, both physical loading and a decrease in the oxygen content of arterial blood in athletes with changes of the electrocardiogram will produce a paradoxical reaction, i.e. normalization of the electrocardiogram. This paradoxical reaction, in our opinion, should be looked upon as unfavorable. It indicates the appearance of other changes in the myocardium which, in combination with

the primary changes, neutralize them and result in apparent normalization of the electrocardiogram. The fact of normalization upon appearance of a second pathological center in the myocardium is well known in clinical practice (M. B. Tartakovskiy, M. G. Udel'nov, Chernogorski and Dushek, etc.).

For example, a patient suffering from acute infarct of the anterior wall may show complete normalization of the electrocardiogram after infarction of the posterior wall. Four such cases have been described by Chernogorski and Dushek (1963). With an infarct of the posterior wall, the electrocardiographic changes were relieved by incipient embolism of the pulmonary artery (Chernogorski and Dushek). Some authors (Evans, 1961) call these changes with repeated infarction, which greatly hinder diagnosis, the "richochet effect," or "cardiographic richochet." The question arises as to the reasons for this type of change in the electrocardiograms of healthy, active athletes, who have no pain.

First of all, this once more confirms the notion that the transition from physiological to pathological sports heart, in the terminology of G. F. Lang, occurs unnoticed. As concerns the genesis of these changes, they can hardly be related to coronary circulatory deficiency.

It must be stated that recent works have essentially changed the current conception of the obligatory significance of changes in coronary blood flow in the genesis of changes in the myocardium. The development of the biochemistry of the myocardium has shown that a number of concepts formulated in cardiology without knowledge of the principal biochemical regularities of the cardiac muscle require re-examination. We have already mentioned the possibility of the development of noncoronarogenic myocardial necroses. In fairness, it should be noted that as long ago as the 1930's, leading domestic clinicists, the founders of the Soviet therapeutic school, G. F. Lang and N. D. Strazhesko clearly understood the significance of biochemistry in the solution of the problem of muscular heart damage. G. F. Lang wrote in 1936: "... in the area of diseases of the myocardium, we should stop attempting to find a morphological substrate for functional changes of the cardiac muscle, and begin seeking a biochemical substrate." The data produced in recent /457
times concerning the biochemistry of the myocardium and its significance, and the evaluation of its condition both by Soviet and by foreign authors (V. N. Vinogradov, A. L. Myasnikov, M. G. Udel'nov, Raab, Lenzi, Hegglin, etc.) provide clear confirmation of the correctness of this investigative trend, suggested 25 years ago by G. F. Lang.

In this chapter, it is impossible to illuminate the current state of the problem of the biochemistry of the myocardium. This is a large, complex and specialized problem. We can discuss only a few of the principal points, without an understanding of which it is impossible to evaluate changes in the cardiac muscle properly.

First of all, we must study not only the biochemistry of the myocardium, but the activity of the regulatory mechanisms controlling the various directions of the biochemical processes. It is obvious at the present time

that the metabolism of the cardiac muscle can change in various directions under the influence of the nervous system. As we know, the Pavlov stimulating nerve causes an increase in the force of cardiac contractions without a simultaneous increase in coronary blood supply, by directly increasing the trophical system.

Disruptions in metabolism in the myocardium may result from two principal causes. These are first of all overloading of the myocardium, i.e. primary myocardial damage, during which metabolic processes in the cardiac muscle are disrupted. With further development of this process, general disruptions in metabolism occur. The myocardium may be damaged secondarily with general disruption of metabolism in the organism, principally in the enzyme systems.

The pathology of metabolism in the myocardium is particularly important among all disruptions of metabolism, since in the other organs loading beyond permissible limits does not play the important role that it does for the myocardium. In no other organ but the heart are the compensatory mechanisms arising during overstress capable of worsening the blood supply to the entire organism and thereby worsening the metabolic disruption itself.

These are a few of the general points involved in the contemporary approach to evaluation of various changes in the cardiac muscle. As concerns problems of biochemistry directly, we should note quite briefly the significance of three elements in myocardial metabolism: the neural hormones of the sympathetic and parasympathetic nervous systems, electrolyte equilibrium and the hormones in the suprarenal cortex.

Normal functioning of the myocardium requires equilibrium between the neurohormones of the sympathetic (adrenaline and noradrenaline -- the so-called adrenosympathetic catecholamines) and parasympathetic (acetylcholin) systems. Disruption of this equilibrium is decisive in the pathogenesis of functional and degenerative heart diseases. Thus, when the level /458 of adrenosympathetic catecholamines increases excessively, beyond the compensation of the acetylcholin, they act as toxic agents, causing hypoxia of the myocardium, extending as far as disseminated necroses of the myocardium, principally in the papillary muscles and in the and in the subendocardial layer of the myocardium (Raab, 1959).

This is used at the present time to explain certain cases of sudden death of healthy young persons after extreme physical or emotional stress, in cases where autopsy reveals no morphological changes in the myocardium. Earlier, these cases were explained by coronary vessel spasm. At the present time, it has been proven that the reason consists in the accumulation of adrenosympathetic catecholamines. Acetylcholin is antagonistic to these substances and increases the effectiveness of the work of the heart.

How does this concern the changes in the hearts of athletes? It has long been established that both mental and physical stress result in

increased adrenaline in the blood. This is evaluated as a manifestation of stimulation and adaptation to the increased work not only of the skeletal musculature and heart muscles, but of other organs and systems of the organism.

Physical overstress may cause so great an increase in adrenosympathetic catecholamines that myocardial necroses occur. It is possible that this is the explanation for the discovery of cicatric changes of the myocardium in athletes who have died from other causes, while the anamnesis shows no indication of myocardial infarction or angina pectoris.

An excess of adrenosympathetic catecholamines, which has a toxic effect on the cardiac muscle, can be determined by the quantity of cholinergic neurohormones. The greater this quantity, the less the quantity of adrenosympathetic neurohormones capable of having a toxic effect on the cardiac muscle. As we know, systematic sports training creates a predominance of vagus cholinergic influences. The gradually developed predominance of cholinergic influences adapts the heart to increased activity of the sympathetic system. However, if physical exercises are excessive and the organism is not prepared for them, the adrenosympathetic catecholamines may have a toxic effect on the cardiac muscle, as a result of the inadequate cholinergic influences.

It can be thought that the capabilities of any heart, including the sports heart, are determined by the level of cholinergic influences, which systematically increase with proper and effective training and are capable of retaining equilibrium with the adrenosympathetic catecholamines, the quantity of which is determined by the intensity of the physical stress. In other words, the capabilities of the heart are determined by predominance of /459
cholinergic influences, which manifests itself either as reinforced liberation of acetylcholin by the endings of the vagus nerve, or by an increase in the reserves of proacetylcholin in the myocardium, or by an increase in the influence of enzymes on acetylcholin metabolism.

It is possible that the changes in the electrocardiogram described above reflect a noncorrespondence between the quantity of adrenosympathetic catecholamines resulting from great physical loads and the quantity of acetylcholin, insufficient for maintenance of equilibrium, due to the failure to achieve the corresponding level of training, which would provide sufficiently powerful vagus cholinergic influence.

The trained heart in the foreign literature is contrasted to the detrained heart, which is commonly called the "busy loafer's heart." This means the heart of modern man, occupied primarily with mental labor, not involved in physical culture or sports (V. Raab, 1959, etc.).

Of no less significance in the formation of changes in the healthy heart is the relationship of the potassium and sodium ions, which have an important role to play in the contractive activity of the myocardium.

Speaking of the significance of the relationship of electrolytes in the metabolism of the myocardium, we can recall the experiments of M. G. Udel'nov who, by applying a segment of necrotic tissue to an undamaged myocardium, produced an electrocardiogram typical for myocardial infarct. It was found that the essential element was the disrupted relationship of potassium and sodium created in the healthy muscular tissue upon contact with the necrotic, decomposing cell. Thus, it is obvious that these disruptions have an essential negative influence on the metabolism of the myocardium, extending to the formation of necrotic sectors. It is interesting that changes in the electrocardiogram occurring with hypopotassemia under experimental conditions are similar to changes observed in athletes with the overstress syndrome. It should be kept in mind that the important thing is not the absolute changes in concentration of these electrolytes, but rather their relationship. A decrease in potassium with a simultaneous decrease in sodium does not disrupt this relationship and is not reflected in the electrocardiogram.

Hypopotassemia may be one of the factors causing changes in the electrocardiogram, particularly since problems of electrolyte metabolism are usually not considered in the dietetic regimen of athletes, or are insufficiently considered. Furthermore, the phosphoric acid salts of sodium are recommended for athletes, and these salts, like glucose, facilitate the development of hypopotassemia. Obviously, these problems require re-examination in the light of current knowledge. As will be seen from the following portions of this report, the phosphoric acid salts of sodium can /461
cause dystrophy of the myocardium, or acting in combination with corticosteroids, can even cause necrosis.

We should discuss the problem of the widespread usage of glucose. As we know, 100-200 mℓ glucose are widely used by athletes both before competition in order to increase performance, and for rapid recovery of energy resources at the finish line (N. N. Yakovlev). However, there are indications in the literature that some athletes feel worse after taking glucose, and performance decreases. Those athletes who take glucose no later than 15 min following competition or 1-1/2 hr before competition generally feel better.

It should be noted that the idea that glucose has "miraculous" effects is so widespread among athletes that it is taken essentially without limitation and in large quantities both before and after training and competition, as well as outside of competition and training.

However, under clinical conditions, practice has long shied away from broad usage of glucose without special direct indications for its application. This is explained by the fact that glucose has a number of negative influences on the organism. Data are available indicating that it increases the mass of circulating blood, increases the viscosity and accelerates the coagulability of the blood, increases the prothrombin index, causes hypopotassemia, etc. Sometimes glucose can cause a change in the end portion of the ventricular complex on the electrocardiogram. As a result of all of this, in clinical practice, for example, cardiac glucosides are injected not

with glucose, as has been done in the past, but in a physiological solution.

In recent times, investigations have been undertaken on the influence of glucose during physical loading. The data of V. P. Pravosudov (1965) have shown clearly that taking glucose without subsequent physical work causes negative changes in the electrocardiogram. Figure 130 shows three electrocardiograms of a rabbit: A, at rest; B, one minute after injection of 5 mℓ of a 40% glucose solution (some depression of the T waves is seen); C, 15 min after injection of glucose. A clear depression of the T wave is seen in leads II, III, aVF and $V_{3,4,5}$, along with splitting of the P wave in Leads II and V_{3-5}, a depression of the S-T segment in leads $V_{1,2}$. Tachycardia increased from 200 (A) to 230 (C).

The injection of glucose after work also causes negative changes in the electrocardiogram. This can be seen from Figure 131 (data of V. P. Pravosudov, 1965). On Figure 131 we see four electrocardiograms of one dog: the first (A) was taken at rest, the second (B) immediately after 2 1/2 hr running on a treadmill (without any essential change in the electrocardiogram), the third (C) one hour after taking 200 g glucose with milk (clear negative changes of the T wave are seen in leads II, III, aVF, V_{2-5}), and the fourth (D) -- one hour after ingestion of 5 g potassium chloride (complete normalization). /463

The data of V. P. Pravosudov are preliminary, although they are of considerable interest, since they confirm the possibility of a negative influence of glucose (in particular, the formation of hypopotassemia under the influence of the injection of glucose) and confirm the assumption made above that a more careful approach to the broad utilization of glucose by athletes must be taken.

Potassium and sodium (particularly the latter) are of essential significance in an analysis of the influence of steroid hormones on the myocardium. This is a third possible path for the development of changes in the myocardium, which should be discussed. The hormones of the cerebral and cortical sections of the adrenal gland have an essential role to play in the development of cardiac muscle damage.

In 1958, a monograph was published by G. Selier, the author of the well known theory of adaptation, dedicated to the problem of chemical prevention of myocardial necroses. The principal thought of this monograph, supported by a mass of experimental material, is that intensive endogenic secretion of corticoids, which occurs with any nonspecific stress (including cold, heat, various emotions, physical stress, etc.) or artificial injection of these compounds into the organism may, under certain conditions, particularly with an excess of sodium salts, be of pathogenic significance for the formation of various types of cardiopathy, in particular myocardial necroses. Selier called this ESCN, which stands for: electrolytes, steroids,

cardiopathy and necrosis. This condition arises only upon injection of sodium salts in combination with corticosteroids. Other cations do not cause necroses in the myocardium. The most active sodium salts in this respect are those formed from phosphoric acid and sulfuric acid (E. P. Vereshchakova, 1965).

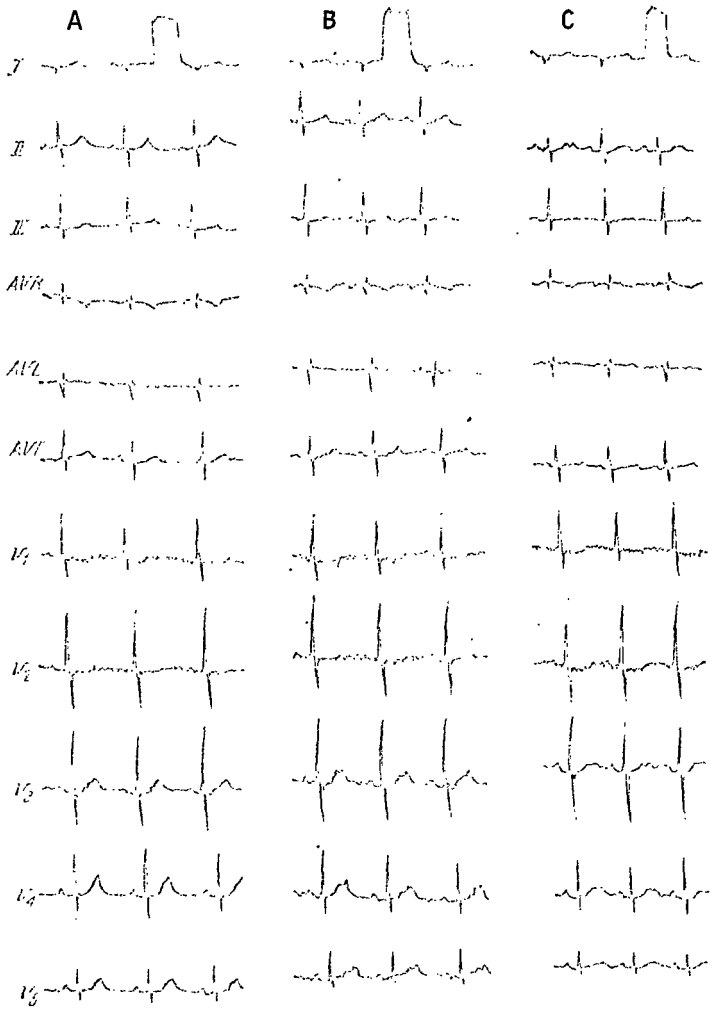


Figure 130. Rabbit Electrocardiogram. A, at rest; B, 1 min following injection of glucose; C, 30 min following injection of glucose. Explanation in text.

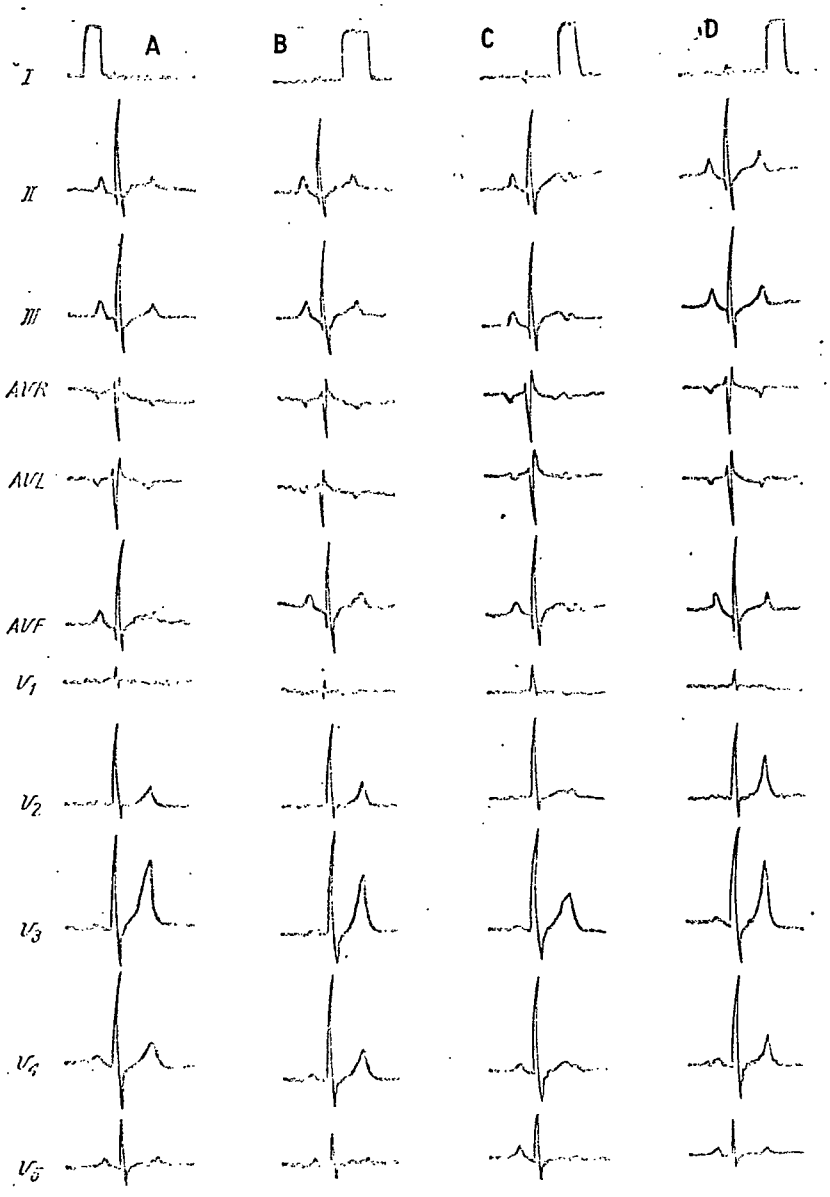


Figure 131. Electrocardiograms of Dog. A, at rest; B, after running; C, 1 hr after taking glucose; D, after taking potassium chloride. Explanation in text.

As concerns the corticoids, the experiments of G. Selier have shown that when sodium phosphates are ingested, myocardial necroses can arise even without the injection of corticoids, since the production of the corticoid hormones by the adrenal cortex is sufficient to cause necrosis. Centers of necroses, arising principally in the right ventricle, atria and the

subendocardial layers of both ventricles do not depend on interruption of coronary blood supply, and result in cicatrization. The electrocardiographic pictures characteristic for localized myocardial damage.

Of great importance for clinical practice and particularly for sports medicine is Selier's conclusion that glucose reinforces ESCN as a result of the hypopotassemia which it causes. A diet rich in vegetable or animal /464
fats has the same effect. Emotion, cold, heat and physical stress create conditions under which doses of sodium ineffective alone may cause widespread myocardial necrosis.

It is also important to note that all of these data were produced not only with rats, but using rabbits, dogs and, which is particularly interesting, apes. In the apes, ECSN was caused by combined injection of cortisone acetate -- the basic human glycocorticoid -- and sodium phosphorus salts.

Selier (1958) considers that ECSN amounts to nonspecific myocardial damage, which may occur in connection with increased production of hormones during nonspecific stress (which may include physical loading) or introduction of the hormones to the organism by various electrolytes always present in the food. In particular, cooking salt takes an active part in the presence of the corticoids.

The data of Selier shed light on the pathogenesis of many pathological states which had not been satisfactorily explained heretofore.

Experimental centers of necrotic myocarditis arising in connection with the injection of large doses of digitalis, in cases of low potassium diet and, finally, with high physical stress have been described. It is interesting that these changes in the myocardium can be prevented by injecting potassium or magnesium chloride, or reinforced by preliminary introduction of the corresponding hormones.

The development of cardiac muscle damage is quite variable and depends on many uncontrollable conditions. However, in experiments with animals prepared by synthetic hormones, many agents, among which muscular stress is by no means the least significant, have caused myocardial necrosis.

Selier's conception of the nonspecific necrotic damage of the myocardium which we have just outlined is of significant interest and allows us to provide some explanation of the changes in the end portion of the ventricular complex of the electrocardiograms of athletes. This conception, furthermore, should cause sports doctors to stop and think before using hydrocortisone for any sports trauma. This conception indicates means of preventing these states (injection of potassium and magnesium salts). It also warns against the usage of glucose, which has facilitated the formation of myocardial necroses under experimental conditions. Finally, it should not be forgotten that the toxic effects of adrenosympathetic catecholamines is increased by the adrenocorticoids.

Of course, we should hardly accept the concepts of Selier completely in their present form, but they can be used as a basis for work in this area.

Returning to changes in the electrocardiogram noted in a comparatively large number of athletes, it should be noted that, since the concept of coronary blood flow cannot explain these changes, it is more proper to interpret them as manifestations of disruptions in the metabolism of the heart muscle. This may be either a problem of hypopotassemia, or the toxic influence of predomination of adrenosympathetic catecholamines over cholinergic substances or, in many cases, appearance of ECSN, i.e. Selier myocardial necroses. /465

Sufficient data is not yet available to give a final answer to these questions, since problems of the biochemistry of the myocardium have not yet been studied in the light of today's concepts in sports. Also, without intensive study of the neurovegetative metabolic influences in the origin of changes in the cardiac muscle, there can be no progress in the understanding and prevention of these states.

Any therapy can be intelligent and purposeful only if the mechanisms of the disruption of the function of the myocardium are known. This is the area in which further investigative work should be performed. However, even today these problems have important practical significance for sports. They should force us to reanalyze many problems of the nutrition of athletes. Furthermore, they allow us to take a new approach to the understanding of the state of training and its proper evaluation, and allow a proper interpretation of changes in the electrocardiograms of athletes which have not yet been explained.

CHAPTER XIV

SYSTOLIC NOISES IN ATHLETES

ABSTRACT. Analysis of phonocardiographic data produced under rest conditions and with various functional tests indicates that accidental systolic noise does not indicate mitral valve deficiency. The usage of phonocardiography for differential diagnosis of accidental and functional noise is described. Case histories are described, and sample phonocardiograms are presented.

The analysis of various systolic heart noises is an important and /466
pressing, but also frequently quite difficult problem. Three types of heart noises are distinguished: organic, functional and accidental (Holldack, Wolf, 1956, 1958, 1960, 1962, 1964).

Organic noises refer to those resulting from congenital and acquired heart defects.

Functional noises are those which, although they may be related to organic damage of the heart and even frequently appear along with organic noises, are not a direct result of a primary disruption in the integrity of a valve or presence of apertures in interior walls of the heart and large vessels. They indicate changes in functioning of the entire heart or of any of its sections, frequently related to acceleration of blood flow, changes in blood viscosity or other factors. With all of this, the cause of each noise is always known. The functional noises include widely varied noises -- from systolic noises in anemia and thyrotoxicosis to systolic and diastolic noises with relative angina pectoris and deficiency of various heart valves.

The accidental noises are nonorganic, nonpathological noises, differing in principle from noises of the first two groups in that they are encountered under ordinary conditions in persons with completely healthy cardiovascular systems, but also may arise in various physiological states. The differentiating specific feature of accidental noises, in addition to their detection in persons with healthy hearts, is the fact that neither the /467
mechanism of their formation nor the point where they arise is known in each concrete case.

In the hundred fifty years which have passed since the time of publication of the investigations of Layenek, in which the possibility of

detection of noises arising during the operation of the heart with the stethoscope was first reported, many hypotheses have appeared concerning the origin of accidental systolic noises. N. N. Savitskiy (1927) explains the appearance of most nonorganic systolic noises as relative deficiency of the mitral valve resulting from too strong stretching of the chords during hypertension of the papillary muscles. He related hypertension of the papillary muscles to increased tonus of the sympathetic segment of the vegetative nervous system. Some authors, agreeing with the opinion of N. N. Savitskiy on the possibility of existence of nonorganic systolic noise as a result of changes in the tonus of the papillary muscles, attribute it to the influence of the vagus nerve or disruption of tonic equilibrium of the two segments of the vegetative nervous system (I. Ya. Lokshina, 1930; K. F. Mikhaylov and V. G. Popov, 1931). A. I. Smirnov (1958), then later V. V. Antipov (1955), showed experimentally that the formation of nonorganic systolic noise actually may be related to changes in the tonus of various segments of the vegetative nervous system. However, in contrast to N. N. Savitskiy, V. V. Antipov believes that this type of systolic noise arises not in the area of the mitral valve, but in the initial segments of the large vessels. In this respect, he sets forth the point of view stated long ago by Sahli (1896). A change in the condition of the vegetative nervous system results in an increase in the force of contraction of the ventricles of the heart, which in turn causes acceleration of the blood flow and the formation of vortex movements.

The idea of a causal connection between accidental (physiological) systolic noises and an increase in the blood volume per beat and blood flow rate through the mouths of the large vessels (in particular the pulmonary artery), leading to the formation of vortices with turbulent blood movement[1] is actively supported by, among other investigators, B. P. Kushelevskiy (1941). This author, disagreeing with N. N. Savitskiy and negating any realistic connection between a-cidental systolic noises and relative deficiency of the mitral valve, bases his argument on many years observations /468 of young men of draft age and athletes.

The results of our phonocardiographic investigations, performed both under conditions of relative quiet and with various functional tests (see below) give us no reason to believe that the accidental systolic noise found in healthy children and adults (athletes and persons not involved in sports) result from relative mitral deficiency. On the contrary, our data force us to agree with Holldack and Wolf (1962) that "the majority of accidental systolic noises are similar to noises arising as fluids folw out of tubes, and result from the formation of vortices caused by the inconstancy of the blood flow conditions from the ventricles, which are quite normal."

[1] A turbulent fluid flow arises arises in those cases when the critical velocity defined by the Reynolds number (Reynolds, 1883) -- VD/μ (V is the velocity, D the diameter and μ the kinematic viscosity) Exceeds 1900. For blood, according to Coulter and Pappenheimer (1949), the corresponding Reynolds number is 1940 ± 160.

Convincing support for this position can be found in the results of investigations performed in recent years using the method of intracardiac phonocardiography (Lewis et al., 1957, 1959; etc.). The data produced by these authors indicate that systolic noise can be recorded by this technique in the pulmonary artery in all healthy persons. As concerns the mechanism of formation of this noise, which is called pulmonary expulsion noise in the modern literature, there are a number of ideas. Thus, Tourniaire et al. (1962), in discussing the reasons for formation of nonorganic systolic pulmonary noise, refer to Chisholm (1937) who showed that the infundibulum and body of the pulmonary artery are easily extendable, there are no fibrous rings at the level of the semilunar valves, while the free ends of the valves are inelastic. During expulsion, these free edges may not lie adjacent to the walls of the vessel, but may remain extended, forming a triangle inscribed in the lumen of the pulmonary artery. The edges of the triangle vibrate like vocal cords.

Pulmonary expulsion noise apparently represents a considerable share of the accidental systolic noises heard on the anterior surface of the thorax (best and most frequently in the second or third intercostal space at the left edge of the thorax or parasternally, but frequently along the entire left edge of the thorax, or under the apex of the heart) in a large number of healthy children and young people, and frequently in adults as well. In addition to the accidental systolic noises related to expulsion of blood from the heart, there are accidental systolic noises apparently arising in the ventricles themselves.

The formation of intraventricular systolic noises under conditions of pathology has been described by I. I. Savchenkov (1960). According to his data, this type of noise results from oscillations of rheumatically damaged tendonous cords and papillary muscles. E. A. Melik-Gyul'nazaryan (1927), on /469 the basis of the results of clinical and pathological comparisons, concluded the possibility of formation of systolic noise as a result of true oscillations of undamaged chordal structures as well. This is also mentioned by S. F. Oleynik (1961), etc.

We cannot exclude intraventricular origin for the "accidental systolic" vibration noise frequently encountered in healthy children of all age groups, particularly preschool and school-age, and sometimes in adults as well. This accidental systolic noise, described in 1909 by Still, appears most clearly along the lower left boundary of the chest or over the middle precordial area or the apex of the heart. One essential characteristic of the "vibration" noise is its musical nature. On the phonocardiogram, it is frequently present as true sinusoidal oscillations, the frequency of which is relatively low. According to the data of certain authors (Castle, Durham, 1961; Zuckerman, 1963), this Still noise may arise in the efflux path of the right ventricle. Welles (1957), analyzing the mechanism of formation of accidental systolic noise in the precordial area, appearing on the phonocardiogram in the form of regular oscillations, the formation of which does not include a high-frequency component, indicates that this noise arises as a result of vibration of the walls of the heart. A. I. Koblents-Mishke (unpublished

work), without contradicting the point of view of Welles, suggest that it is more probable that this noise is formed by tendonous cords. D. D. Lebedev and A. I. Pimenov (1965), reporting on systolic noise detected in healthy children over the apex of the heart and along the left edge of the lower half of the thorax, are inclined to believe that it is caused by vortices in the blood during systolic movement along the internal surface of the wall of the left ventricle, which is covered by muscular trabeculae. The description of the noise and phonocardiogram which these authors present indicate that the noise is "vibrational" in nature. Finally, McKusick (1959) states that the point of formation of Still noise is not in the ventricles, but in the area of the semilunar valves, relating it to vibration of free edges of the triangle described by Chisholm. The data presented indicate that the true source of this "vibrational" noise is still unknown.

Thus, the literature contains discussions of a number of concepts of the formation of accidental systolic noises, each of which, apparently, requires further study. It is quite probable that these noises are polyetiological, and quite possible that frequently the formation of even a single individual accidental noise is the result of the participation of a number of factors. /470
Simultaneous presence of several accidental systolic noises in one person is also quite possible.

The absence of sufficiently complete knowledge concerning the mechanisms of formation of accidental systolic noises should not prevent intensive search for criteria for reliable diagnosis and differentiation between them and noises related to pathology of the heart and large vessels. These investigations seem particularly necessary since accidental systolic noises are encountered in a large number of practically healthy persons and particularly frequently in children, adolescents and young people. Thus, G. M. Slutskaya (1963), R. E. Mazo (1964), A. I. Pimenov (1964), S. I. Shatunovskaya (1964), etc. succeeded in finding them in more than 50% of the children which they investigated in ages up to 14-15 years. Holldack and Wolf (1956 and later) indicate that in children accidental systolic noises are almost always present. According to Mannheimer (1955), modern apparatus allows weak systolic noises to be recorded in all children. Accidental systolic noises are noted somewhat less frequently in adults: in the investigations of R. B. Minkin (1963) they were noted in 28%, according to the data of V. V. Solov'yev, V. V. Bulychev (1962), in 48% of those studied.

According to the data presented in the literature (R. Ye. Motylyanskaya and S. P. Letunov, 1949; R. D. Dibner, 1960; A. G. Kim, 1964; Yu. A. Chizh, 1965; Emmrich et al., 1961; etc.) and our own data, accidental systolic noises are noted in athletes no less frequently, and according to the opinion of V. V. Solov'yev and V. V. Bulychev (1962), approximately twice as frequently as in practically healthy persons of the same age but not involved in sports. Also, there is no reason to consider that the presence of organic heart defects is an extreme rarity in athletes. Thus, the results of observations of athletes with heart defects are presented by

R. Ye. Motylyanskaya (1949, 1963), S. P. Letunov (1957), R. D. Dibner (1962, 1963, 1964).

In recent years, we have studied over 40 athletes in active training with congenital and acquired heart defects, most of these athletes being rated class I or II. Some of these persons had no idea that they had heart disease before special phonocardiographic investigations. Furthermore, even the doctors who had examined many of these persons before had not suspected anything -- the systolic noises resulting from organic heart defects were erroneously interpreted as nonorganic (functional) noises. This was apparently facilitated by the complete absence of any pain and the clear medical history, the rather stable sports results, the satisfactory reaction of the /471
pulse and blood pressure to tests involving physical loading and the normal electrocardiographic and X-ray data, as well as by the skeptical attitude of certain doctors to the possibility of presence of organic heart defects in actively training athletes.

These data indicate the necessity of a very cautious approach to the evaluation of systolic noises in athletes. An insufficiently careful approach to this problem, accompanied by failure to utilize certain important differential characteristics, sometimes leads to the fact that individual athletes with organic heart defects continue to undergo constant, heavy physical loads, which may result in their untimely invalidization.

Although the literature presents descriptions of individual cases in which high results and even records have been achieved by athletes with organic heart defects, at today's level of knowledge we should acknowledge the obligatory nature of eliminating these persons from heavy sports competition. However, it should be kept in mind that many athletes are taken out of competition due to improper interpretation of accidental systolic noises as organic noises.

Phonocardiography in the Differential Diagnosis of Systolic Noises

In sports medical practice, accidental systolic noises are most frequently falsely assumed to be organic systolic noises resulting from mitral deficiency. On this basis, in the following we will discuss the differential diagnosis between accidental systolic noises and regurgitational noise resulting from organic mitral deficiency exclusively.

It should be noted that differentiation between accidental systolic noises and organic mitral deficiency noises in athletes is more difficult than in persons not involved in sports. This is because, in addition to the systolic noise, these persons more frequently have all the characteristics which have been interpreted up to the present time as more or less pathognomonic for mitral valve deficiency (we have in mind weakening of tone I and the presence of tone III over the apex of the heart, more or less expressed splitting and accentuation of tone II over the pulmonary artery, expansion of the boundary of the heart to the left and hypertrophy of the left ventricle).

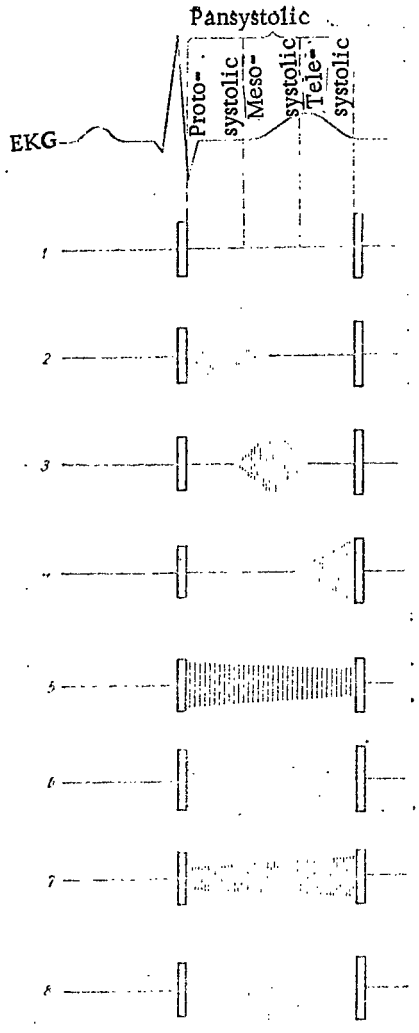


Figure 132. Form ("Configuration") and Position in Time of Systolic Heart Noises (Diagram). 1, Position of tones I and II when recorded at high-frequency characteristic; 2, early systolic (protosystolic) decrescendo noise; 3, mesosystolic rhomboid noise; 4, Telesystolic crescendo noise; 5, pansystolic decrescendo noise; 6, pansystolic band-shaped noise; 7, pansystolic crescendo noise; 8, pansystolic rhomboid noise.

Differential diagnostic phonocardiographic evaluation of noises is usually performed using the following characteristics: amplitude, duration, location in the cardiac cycle, configuration (Figures 132, 133, 134, 135), the frequency characteristic, point of best perception, presence of irradiation of the noise and its direction. /472

The contradictory nature of the information relating both to these characteristics for various types of noises, and particularly to the significance of each of these for differential diagnostic purposes stimulated us to analyze the material available to us, encompassing the auscultative and phonocardiographic investigation of healthy athletes with accidental systolic noises and patients suffering from organic mitral deficiency.

With the test subject lying on his back, accidental systolic noises are usually heard simultaneously at several auscultative points, frequently both over the base of the heart and over the apex. Most frequently, the punctum maximum of the accidental systolic noises is localized in the second intercostal space at the left edge of the thorax, less frequently in the third intercostal space at the left edge of the thorax.

In a smaller number of cases, the epicenter of the accidental systolic noises is located over the apex of the heart or flightly (1-2 cm) higher and more medial (along the peristernal line). /475

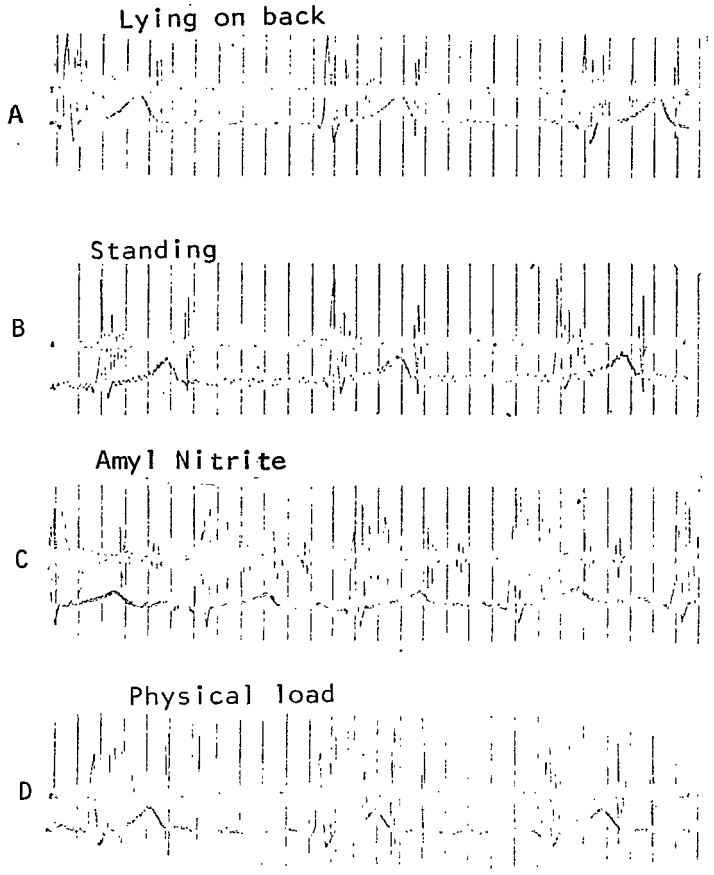


Figure 133. Athlete M-v, 18 Years Old, Class II Skier. Healthy. Phonocardiograms recorded at the apex of the heart (frequency characteristic C_2), show: A, with subject lying on back, tone I split, short, mesosystolic functional musical noise of low amplitude, approximating spindle shape; B, in standing position, noise disappears completely; C, reaction to inhalation of amyl nitrite, increase in pulse frequency, clear increase in amplitude of tone I, weakening of tone II, beginning of which is clearly earlier than end of T wave on electrocardiogram, appearance of tone III and considerable increase in amplitude of systolic noise; D, physical loading, in addition to sharp increase in systolic noise, causes increase in tones I and II and appearance of of tone III, the amplitude of which is quite high.

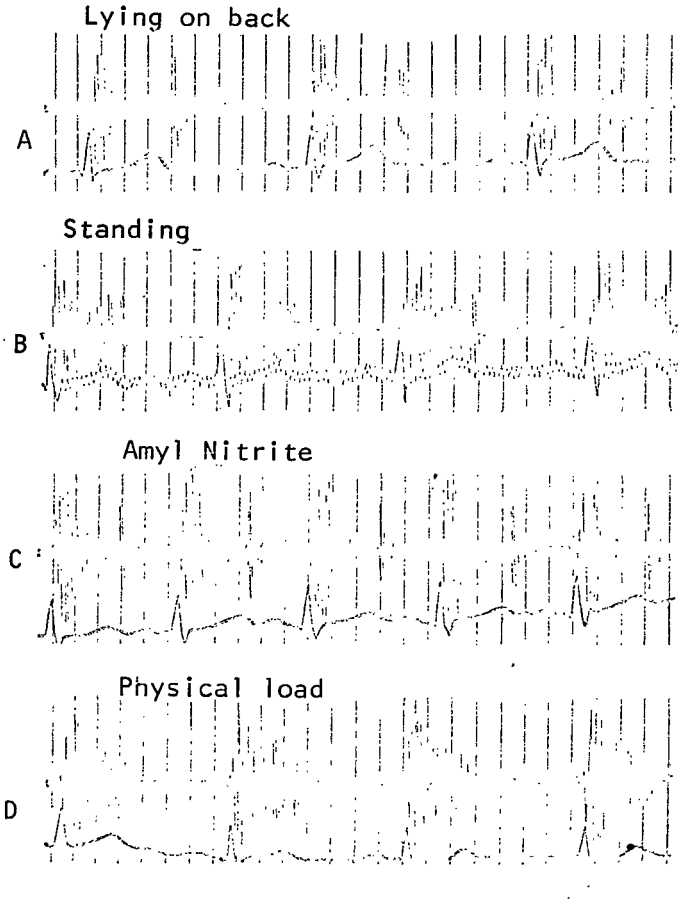


Figure 134. Athlete Z-n, 23 Years Old, Class I (Table Tennis). Mitral deficiency. Phonocardiograms recorded at the apex of the heart (frequency characteristic C_2) showed: A, with test subject in lying position on back, tones without peculiarities, pansystolic noise, increasing toward tone II; B, in standing position, amplitude of pansystolic noise increases markedly without essential change in its form; C, after inhalation of amyl nitrite, configuration and amplitude of noise change markedly: during first half of systole, noise increases; during second half, markedly decreases, which is particularly noticeable in the third and fifth cycles, where the noise practically disappears by the end of the systole; D, reaction to physical loading appears in the form of increase in noise in both first and second half of systole.

The data which we produced in relation to the most frequent localization of the punctum maximum of accidental systolic noises in athletes at the second intercostal space on the left side of the thorax agree with the results of the investigations of Emmrich et al. (1961). These accidental noises appearing most clearly in the second intercostal space at the left side of the thorax corresponded in localization of the epicenter (at the point of projection of the pulmonary artery valves) and all other characteristics to pulmonary expulsion noise. The point of best perception of the systolic noise resulting from organic mitral deficiency in all the subjects whom we investigated (children, adolescents and adults) was the area of the apex of the heart.

Thus, the epicenters of organic systolic noise resulting from mitral deficiency and most accidental noises encountered in athletes do not correspond.

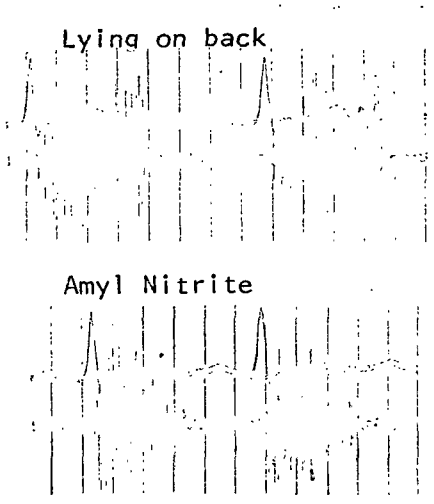


Figure 135. Patient K-a, 34 Years Old, Mitral Deficiency. Phonocardiograms recorded at apex of heart (frequency characteristic C_2) show pansystolic noise of high amplitude in the lying position on the back. After inhalation of amyl nitrite, the amplitude of the noise decreases sharply in the second half of the systole without noticeable change during the first half.

The direction of irradiation of the noise is quite important for differentiation between organic mitral regurgitation noise and accidental systolic noise. Even strong accidental systolic noises, which irradiated downward along the left edge of the thorax and to the apex of the heart or, with maximum expression at the apex, radiated to the base of the heart, did not appear clearly in the left axillary area.

In contrast to this, systolic noise resulting from organic mitral deficiency, although it might be traced weakly from the apex to the base of the heart, was principally irradiated into the left axillary area, where it was always recorded quite clearly.

Thus, the direction of irradiation /476
of accidental systolic noises and of organic mitral deficiency noise also differs.

In phonocardiographic characterization of heart noises, particularly for purposes of differential diagnosis, considerable attention is usually given to data on their intensity (amplitude) and duration.

Some investigators emphasize that the amplitude of accidental systolic noises is low, and clearly less than the amplitude of organic mitral deficiency noise.

In most of our investigations, the organic noise from mitral deficiency was more intensive than accidental systolic noise. However, in a number of persons the amplitude of this noise when recorded with the low frequency (H) and middle-frequency (C_1 and C_2) characteristics is quite low, and is low in relation to certain accidental systolic noises with epicenters in the area of projection of the pulmonary artery valves, as well as the area of the apex of the heart.

Thus, the amplitude characteristic of the noise cannot be used as a reliable criterion for differential diagnosis of various systolic heart noises (I. A. Kassirskiy and G. I. Kassirskiy, 1964; Holldack, Wolf, 1956 and later; Castle, Durham, 1961).

The differential diagnostic significance of the duration of the noise, like the intensity of the noise, is evaluated variously in the literature. Castle and Durham (1961) emphasize that the most important characteristic aiding in differentiation between apical accidental systolic noises and organic mitral deficiency noise is the pansystolic nature of the latter. However, A. G. Salim'yanova (1960), I. A. Kassirskiy and G. I. Kassirskiy (1964) have noted short regurgitation noise, in particular with mitral valve deficiency not too sharply expressed.

In our phonocardiographic investigations with organic mitral deficiency, we have never recorded a short systolic noise, and in fact it has been only as a rare exception that the noise has failed to occupy the entire systole, with a very short free interval noted between its end and the aortal component of tone II. Thus, in almost all cases the organic noise was pansystolic (see Figures 132, 134).

As concerns the duration of the accidental systolic noises and their position in time, according to our observations proto- and protomesosystolic noises, i.e. noises occupying the beginning portion as well as the beginning and middle portions of the systole predominate over the base of the heart in the athletes, while when recording is made over the apex of the heart, /477
the proto-, protomeso- and mesosystolic noises predominate, noises with the epicenter over the apex being usually shorter than noises with the epicenter in the area of the projection of the pulmonary artery valves. This "time location" of the accidental systolic noises corresponds to that usually noted in the literature. Also, in addition to these relatively short systolic noises, we noted longer accidental noises in athletes, occupying two thirds or three fourths of the systole, sometimes recorded over the apex of the heart, and not too rarely recorded at the second intercostal space on the left side of the chest. These relatively longer accidental systolic noises may simulate organic systolic noise resulting from mitral deficiency. However, if we compare the duration of the various accidental systolic noises

recorded with several frequency characteristics to the duration of the organic mitral deficiency noise recorded at the same frequency characteristics, a clear difference is seen between them, particularly in comparisons of the high-frequency phonocardiograms. Thus, with high-frequency recording (B_1) the pansystolic nature of the organic mitral deficiency noise is retained. In contrast to this, the accidental systolic noises, even though they may be quite long at the middle-frequencies, are clearly shorter when recorded at frequency characteristic B_1.

Thus, we might consider that the presence of pansystolic noise over the apex, particularly if it is recorded on the high-frequency phonocardiogram, can be a very serious argument for assumption of organic mitral deficiency.

When the phonocardiograms are recorded with various frequency characteristics, the dependence of amplitude of the noise recorded on its frequency composition is clearly seen. Therefore, by comparing the amplitudes of recordings at a number of frequency characteristics, we can perform simple frequency analysis of the noise. Thus, if the amplitude of any noise is greatest when recorded at characteristic H and C_1, decreasing noticeably when recorded at C_2, and being absent or almost absent at the high-frequency (B_1) characteristic, we can consider that in this case, a low-frequency noise has been recorded. When the amplitude of the noise when recorded with characteristics C_2 and B_1 is not only no less than the amplitude of the recording at the lower frequency characteristics, but even greater, we can state that this is a high-frequency noise. When the noise appears strongest at the medium-frequency characteristics (C_1 and C_2), it can be defined as a medium-frequency noise. Finally, if the amplitude of the noise is considerable when recorded at all frequency characteristics, including the high-frequency characteristic (B_1), it can be considered that the noise has a broad spectrum[1]. /478

The investigations which we have performed have convinced us that when phonocardiograms are recorded under conditions of relative quiet, data on the frequency composition of the noise are quite important and a quite reliable criterion for differentiation between accidental systolic noise and organic noise resulting from mitral deficiency. The amplitude of the recording of accidental systolic noises is usually greatest when recorded with the middle-frequency characteristics (C_1 and C_2), but at the high-frequency

[1] In defining the frequency composition of a noise by comparing the amplitudes of its recordings with various frequency characteristics, the amplification used for each characteristic must be the same.

characteristic (B_1) the noise is generally considerably weaker or completely absent. Consequently, accidental systolic noises are middle-frequency noises. In contrast to this, organic mitral defect noise, which appears very clearly when recorded at characteristics C_1 and C_2, in all cases was clearly recorded at the high-frequency characteristic (B_1) as well. This indicates the broad spectrum of organic mitral deficiency noise and indicates that higher frequency components take part in its formation.

As an important differential characteristic, we should note also the fact that in many cases organic systolic noise resulting from mitral deficiency can be recorded at the high-frequency characteristic (B_1) not only at its epicenter over the apex of the heart, but in the left axillary area, to which, as we have noted, it is always clearly irradiated.

We emphasize the differential diagnostic value of the high-frequency (B_1) recording also due to the fact that it is not used in many works. A number of authors limit themselves to a description of data produced for for recordings at the lowest frequency characteristics, and therefore fail to give the proper significance to an evaluation of the frequency composition of the noise.

On the basis of the above, we consider that the diagnosis and differential diagnosis of various systolic noises cannot be performed using phonocardiographs with lever type recorders (pen recorders, heat recorders and other apparatus whose recording system limits the pass band to frequencies up to 100-150 Hz or lower).

Phonocardiographic differentiation of accidental systolic noises from organic mitral noises can also be greatly aided by the evaluation of the form of the noise. In this respect, as in evaluation of the amplitude and duration of the noise, it should be recalled that the graphic configuration of /479
the noise is notably influenced by the frequency characteristic of the recording. Therefore, here also we must constantly consider the frequency range of the recording, and in comparing the forms of various noises we must use only phonocardiograms with identical frequency characteristics. The change in the form of the graphically recorded noise oscillations when various frequencies are used can be demonstrated on the example of the organic mitral deficiency noise. If this noise does not occupy the entire systole, it is generally a decrescendo noise when recorded at all frequency characteristics (see Figure 132). The pansystolic mitral deficiency noise, when recorded at all frequency characteristics, is most frequently recorded as a band-shaped, and less frequently as a decrescendo or crescendo noise from tone I to tone II (see Figure 132). Also, frequently at lower frequency characteristics the pansystolic noise is recorded as a band-shaped or decrescendo noise, while at higher frequency characteristics a clear telesystolic crescendo of the noise is noted. These changes in the configuration

of the noise, revealed by recordings at various frequency characteristics, can be related to the change in the frequency composition over the course of the noise.

In spite of the fact that organic mitral deficiency noise may vary widely in its configuration (band-shaped, decrescendo, crescendo) a clearly outlined oval (funnel-shaped) or particularly rhomboid form is not characteristic for it (at any frequency range of recording). In contrast to this, accidental systolic noises may have no definite configuration, being recorded on the phonocardiogram as noncharacteristic oscillations of varying amplitude, or may approach the decrescendo shape, but they are frequently recorded as a more or less clearly expressed rhomboid or spindle.

Particularly frequently, the spindle-shaped configuration appears with so-called musical accidental systolic noises (see Figure 133). Since various investigators interpret the significance of the musical nature of the noise differently, we should discuss this problem further.

Comparatively recently, particularly in the years preceding the broad introduction of phonocardiography to clinical practice, the opinion was commonly held that the discovery of a musical heart noise was a clear indication of organic (generally valve) pathology. This point of view, based on data of auscultation, has not only been seen in the works of individual investigators, but has been presented in textbooks and guides on cardiology. Even in some investigations performed in recent years, for example the /480
monograph of I. A. Kassirskiy and G. I. Kassirskiy (1964), it is stated that musical noises, "as a rule are valve type organic noises," and that "in most cases musical noises are combined with valve noises and their origin is related to a valve defect" and, finally, that "musical systolic noises are a rather reliable diagnostic indicator of mitral deficiency, since they generally indicate an organic defect." These authors consider it necessary to differentiate these musical noises only from one form of nonvalve noise, namely from extracardiac cardiopulmonary noise.

However, in addition to this evaluation of musical heart noises, there is another opinion, based to a considerable extent on the data of phonocardiography. In order for a noise recorded on a phonocardiogram to be called musical, it must correspond to quite precise requirements. A musical noise is only a noise which is present on the recording as a true sine wave oscillation of 150-100 Hz or less (low-frequency musical noise) or 300-500 Hz or more (high-frequency musical noise). Holldack and Wolf (1958 and later) indicate that musical heart noises may be either organic or accidental. The organic heart noises will have a low-frequency musical character only as a rare exception. At the same time, musical (particularly low-frequency musical) noises, in the opinion of Holldack and Wolf, are characteristic for accidental systolic noises and are an important indicator of these noises in children. The musical nature of certain accidental systolic noises, particularly frequently the Still noise (vibrational noise) has been noted by a number of authors (M. Luts et al., 1964; P. S. Moshich, 1965; Vogelpoel et al., 1959; Castle, Durham, 1961; etc.). Some investigators (RuDusky,

1963) include the definition of "musicality" among those characteristics which they use in the differentiation of accidental systolic noises from organic noises.

On the basis of our own investigations, we fully and completely agree with the opinion of the authors just presented. "Musicality," which manifests itself clearly at the medium frequencies, is one of the most important, frequently decisive characteristics of accidental systolic noises. These noises are particularly frequently noted in children of preschool or primary school age. The "musicality" of a systolic noise can be noted throughout its entire duration. Sometimes, one or several groups of sinusoidal oscillations arise during the course of a noise, giving the impression of a musical chord in auscultation.

In addition to the acknowledgment of the very frequent presence of /481
accidental musical systolic noises, we should emphasize that, although we have also infrequently heard musical noises with certain organic heart defects, none of the many phonocardiograms which we have recorded for patients with pure mitral deficiency or combined mitral defects (children, adolescents, adults, athletes and nonathletes) has the organic systolic noise appeared musical. Including oscillations of various, particularly high frequencies ("disordered frequency character," according to M. N. Tumanovskiy and Yu. D. Safonov, 1964) and various amplitudes, organic mitral deficiency noise was never fully present as true high-frequency or low-frequency sinusoidal oscillations (only in one case during pansystolic noise were two short groups of regular oscillations detected). We have also never noted musicality of this noise in auscultation. Usually, such noises could be defined as blowing, "pouring," clearly high-frequency.

The data which we have produced agree with the results of the investigations of Holldack and Wolf (1964) who state that "with mitral deficiency involving calcification of the valve surface, musical noises should be heard more frequently," although they do note: "This has not been confirmed by our experience."

Analyzing and comparing various characteristics of accidental systolic noises and organic mitral deficiency noise, we should indicate that phonocardiographic recording quite frequently shows clear variability of the former from cycle to cycle, as well as with the phase of respiration.

The change in accidental systolic noises from cycle to cycle is expressed in that their configuration may vary rather clearly. Noise appearing in one cycle as a clearly outlined spindle with no marked peak in amplitude may appear in another cycle as near rhomboid in form, and in the third cycle may have a configuration near decrescendo. In various cycles, the location of the amplitude peak of the noise may vary, as well as the location of the group of musical oscillations, if such a group is present. Also, certain variations in the total duration and amplitude of the noise may be noted. Respiratiory changes in accidental systolic noises frequently

appear as a clear decrease in the amplitude and duration during the inhalation phase as compared to the exhalation phase.

As concerns organic mitral deficiency noise, its general configuration does not change markedly from cycle to cycle.

Sharp changes in amplitude are also generally not seen, and the duration of this noise generally does not vary as a function of respiration phase. /482

In addition to all of the differences between accidental systolic noises and organic mitral deficiency noise, their differentiation can also be facilitated by evaluation of the changes in intensity of the noise during extrasystolic arhythmia. This was first noted by Holldack and Wolf (1960). As these authors note, the intensity of accidental systolic noises decreases during extrasystolic contractions, then noticeably increases in the post-extrasystolic cycles following the compensatory pause. In contrast to this, organic mitral deficiency noise does not change noticeably in the extrasystolic and postextrasystolic contractions.

The data which we have produced confirm this statement. The clear change in the intensity and duration of accidental systolic noises during extrasystolic arhythmia emphasize their relationship to the volume per beat.

As one of the characteristic differential signs, frequent usage is made of the relationship of the beginning of a noise to tone I of the heart. According to the data of a number of investigators (R. D. Dibner, 1960, 1962, 1963; A. L. Mikhnev et al., 1963; Holldack, Wolf, 1958 and later; etc.), accidental systolic noises in most cases are separated from tone I by a rather short interval (from 0.02 to 0.08 sec). Certain authors (G. M. Slutskaya, 1963; A. G. Kim, 1964; etc.) note that accidental systolic noises fall directly adjacent to tone I in only a few cases.

We do not have sufficient basis to consider the presence of this interval as a constant indicator of accidental systolic noises, since in a large number of cases, the phonocardiograms of healthy persons have shown a direct transition from the final oscillations of tone I to the initial oscillations of accidental noise. Since, however, organic mitral deficiency noise always contacts tone I or (with higher frequency recordings) even replaces it, the presence of an interval between tone I and the beginning of the noise can be used as a basis to interpret the noise as accidental. Thus, the value of this differential criterion is relative: the presence of an interval between tone I and the beginning of the noise may indicate against organic mitral deficiency, but the absence can hardly exclude accidental systolic noise and does not necessarily indicate that the noise is organic in nature. The beginning of the accidental systolic noise immediately after tone I was noted more frequently directly over the pulmonary artery and less frequently over the apex of the heart or its center. The apical and parasternal musical noises were separated from tone I by a rather clear free interval more frequently than the other accidental systolic noises. /483

Many authors, differentiating various systolic noises, indicate the retention of tone I over the apex of the heart with accidental noises and its weakening in cases of organic mitral deficiency. However, phonocardiographic investigations have indicated that in patients with organic mitral deficiency, tone I may be retained and weakened only in the more expressed stages of the defect. At the same time, a weakening of tone I in healthy persons with relatively elongated atrioventricular conductivity has been noted (A. B. Belotserkovskiy, 1965; Holldack, Wolf, 1956; Ueda et al., 1961). Thus, according to the data of Z. B. Belotserkovskiy, produced in the examination of healthy athletes, a perticularly clear decrease in the amplitude of tone I was noted in persons with a P-Q interval on the electrocardiogram exceeding 0.175 sec; in approximately one fourth of these test subjects, no tone I oscillations were noted on the high-frequency phonocardiogram.

In patients with mitral deficiency, we noted a weakening of tone I also only in those cases when elongation of the P-Q interval was noted on the electrocardiogram. Thus, evaluation of the intensity of tone I over the apex of the heart is not an essential criterion for differentiation of these systolic noises.

The more or less manifest accentuation and splitting of tone II over the pulmonary artery cannot be used as a clear indicator for this type of differentiation, nor can the presence of tone III over the apex of the heart, since they are rather frequently encountered not only with mitral deficiency, but also in persons with accidental noises. In particular, the splitting of tone II with an interval between its components mainly from 0.025 to 0.045 sec has been quite frequently observed on the phonocardiograms of healthy athletes. Tone III in the area of the apex heart in the position with the test subject on his left side was detected by us in 95% of young athletes studied.

Functional Tests in the Differentiation of Systolic Noises

Even careful phonocardiographic investigation does not always make it possible to answer clearly the question: may a given noise be looked upon as a reliable indicator of mitral deficiency or should it be considered accidental? In these cases, it is useful to study the details of the acoustic picture of the heart not only in the state of relative quiet, but also with various physiological influences. /484

In order to aid differential diagnosis of various systolic noises and their proper interpretation, the study of the altered sounds of the heart produced in various functional tests, most commonly the tests in which the position of the body is changed or physical loading is applied and less frequently certain pharmacological tests, are broadly used. However, it is necessary to note that the data available in the literature on this question, produced by aucsultation, are contradictory, and phonocardiographic investigations are few. In the area of sports cardiology, in particular

domestic sports cardiology, there are very few works dedicated to phonocardiographic analysis of the changes in accidental systolic noises of the heart during certain functional tests (Ya. I. Ul'brikh, 1960; R. D. Dibner, 1960, 1962; V. V. Solov'yev and V. V. Bulychev, 1962; A. G. Kim, 1964). As concerns comparison of the changes of accidental noises in athletes with the changes in organic mitral deficiency noise during certain functional tests, we can only point out the investigations of R. D. Dibner (1962, 1963).

Considering the contemporary state of the problem of differential diagnosis of various systolic heart noises and its great significance in sports medical practice, we performed a special phonocardiographic analysis and comparison of changes in accidental systolic noises and organic mitral deficiency noise during a number of functional tests.

The functional phonocardiography consisted of a comparison of the acoustical picture of the heart recorded at rest with the changes in this picture in response to changes in the body position (rotation to the left side and orthostatic test), with physical loading (20 deep knee bends during 20-25 sec), as well as the test of inhalation of amyl nitrite vapors. In our investigations, the changes in the sounds of the heart in response to the tests were recorded using two identical microphones, one of which was installed over the apex of the heart, the other -- in the second intercostal space along the left edge of the thorax. This synchronous recording allows us to produce reliable phonocardiographic characteristics of the heart noises over the projections of various valves and thereby to clarify to some extent whether they are independent or conductive.

The test with rotation to the left side generally showed no clear differences in the degree and direction of changes of the acoustical features of the heart in test subjects with accidental noises in comparison to those who were found to have organic mitral deficiency noise.

The only changes noted were an increase in the amplitude and duration of /485
the heart tones and of systolic noise.

With the test subjects in the standing position in most cases tone I became stronger, which was true both of persons with accidental systolic noises and of persons with organic defects. The changes in tone II were irregular; tone III was not recorded in a single case with the test subjects in the standing position.

The change in the intensity and duration of noises as the test subject is shifted to the vertical position is quite important for differentiation. In the overwhelming majority of cases, accidental noises disappeared or their amplitude and duration decreased markedly (Figure 133). Organic mitral deficiency noise retained its pansystolic nature in the vertical position of the test subject, while its amplitude, if it did decrease, decreased to a lesser extent than the amplitude of accidental noises. We should note the fact that in many cases with the test subject in the standing position,

organic mitral deficiency noise not only did not decrease, but even became more intensive than when recorded in the lying position (see Figure 134).

There is doubtless interest in phonocardiographic comparison of changes in heart noises with organic mitral deficiency noise to changes in the acoustical picture with accidental systolic noises in response to the test with physical loading. The investigations performed showed that after physical loading the amplitude of tone I constantly increased, which was true both of persons with accidental noises and of subjects with organic mitral deficiency noise. The changes in the amplitude of tone II, as in the test with varying body position, were not constant. After physical loading, tone III was observed in a larger percentage of cases than on the phonocardiograms at rest. Its amplitude generally increased.

The duration of accidental systolic noises after loading increased relatively, while the interval between the end of the noise and beginning of tone II became shorter. Organic mitral deficiency noise remained pansystolic after loading.

The amplitude of systolic noise, both accidental and organic, after physical loading increased regularly (see Figures 133, 134). However, the degree of the increase of accidental noises was considerably greater than that of organic noises. Thus, whereas organic mitral deficiency noise increased after loading by generally up to 70% of its initial value, the increase in the amplitude of an accidental noise under these conditions was generally markedly greater (from 70 to 300%). The increase in noise was /486
particularly sharp for recordings over the pulmonary artery. Thus, the results which we produced confirmed data on increased amplitudes of accidental systolic noises after loading.

On the basis of this, we assume that the physical loading test cannot be used as a reliable criterion for differential diagnosis of accidental systolic noises from organic mitral deficiency noise, since the influence on these noises moves in the same direction, and the difference in the reaction is only quantitative.

In seeking a functional test which could allow qualitative differences to be determined in the reaction of organic mitral deficiency noise and that of accidental systolic noises in athletes, we considered the test with inhalation of amyl nitrite vapors. It should be noted that in 1961, when we began to use amyl nitrite for differential diagnosis of various systolic noises, and up to the present time, we have not succeeded in finding a single report dedicated to the usage of this functional test in the practice of sports medicine. Furthermore, even clinical data indicating the value of utilization of amyl nitrite for the diagnosis and differential diagnosis of heart noises, has begun to appear in special guides on phonocardiography and auscultation only since 1960-1963 (Holldack, Wolf, 1960; Zuckerman, 1963). However, the first work dedicated to this problem was written in 1918 (Morison). In the domestic literature, only quite recently have individual reports begun to appear (G. K. Korchan, 1963, 1965; P. S. Moshchich, 1963,

1965; E. Sh. Khalfen, 1964), in which the authors present their own data on clinical studies of the reaction of heart noises to the amyl nitrite test.

The basis for the utilization of the test involving inhalation of amyl nitrite vapors for differential diagnosis of accidental systolic noises from organic mitral deficiency noise was for us the investigation of Kahler (1932), Barlow and Shilingford (1958), Vogelpoel et al. (1959 and later), Endrys and Bartova (1960-1961). According to the data of these authors, the inhalation of amyl nitrite vapors results in a clear difference in the reaction of the noises of expulsion and regurgitation, if the latter is related to the left segment of the heart. The regurgitation noise from organic mitral deficiency weakens in the test, which is a result of the great and rapid decrease in arterial pressure and, consequently, decrease in the intake resistance of the systemic circulation. Noises of expulsion during this test become stronger, due to the increased rate of emptying of the ventricle.

Considering this type of data, we turned particular attention to the /487
amyl nitrite test and analysis of the results produced in this test.

The investigations which we performed showed that following the inhalation of amyl nitrite (five deep breaths in 15 sec), a clear increase in the amplitude of tone I and a noticeable decrease in the amplitude of tone II are observed. Tone changes of this type appear both in persons with accidental noises and in test subjects with organic mitral deficiency noise. Persons with accidental noises, in addition to this, a reinforcement of tone III is noted, and in some cases this tone appears although tone III was not present on the initial phonocardiogram.

The reaction of accidental systolic noises and organic mitral deficiency noise to the inhalation of amyl nitrite vapors differs qualitatively. The amplitude of accidental systolic noises increases markedly in all cases, exceeding the initial amplitude by an average of 2 1/2 times. The duration of these noises also increases. The interval from the noise to tone II regularly decreases. The clearest increase in amplitude of accidental noises is noted during the first 20 sec following completion of inhalation (see Figure 133).

In contrast to the increase in the intensity of accidental systolic noises, the amplitude of organic mitral deficiency noise decreases after inhalation of amyl nitrite, which is particularly clearly seen in the second half of the systole. During the first half of the systole, an increase in the amplitude of the noise is sometimes seen, although the degree of this increase is clearly less than in the case of accidental noises (the amplitude exceeds the initial amplitude by not more than half). The amplitude of the pansystolic noise decreases in the second half of the systole by more than two times in comparison with the initial amplitude. In many cases in the last third of the systole the noise disappears completely, which results in a shortening of the total duration of the noise. These changes in the noise

are most clearly seen during the first 20 sec after completion of the inhalation cycle (see Figure 134).

Thus, we can conclude that although it is necessary in each case of differential diagnosis between accidental systolic noises and organic mitral deficiency noise to consider the entire complex of phonocardiographic characteristics in the acoustical picture of the heart, some of these characteristics can serve as quite reliable and constant criteria, facilitating proper evaluation of the noise. These criteria include the frequency characteristic of the noise, the direction of its irradiation and in most cases the duration of the noise. An evaluation of the location of the /488
epicenter of the noise and its configuration, the relationship between the beginning of the noise and tone I of the heart, as well as its appearance on the phonocardiogram as true sinusoidal oscillations can be a significant aid in differentiation.

Organic mitral deficiency is characterized by a noise which constantly and clearly appears in the high-frequency phonocardiogram, covering the entire systole (pansystolic), merged with tone I of the heart and easily traced from its epicenter over the apex to the left axillary area. The configuration of this noise may be either band-shaped or decreasing, or even increasing from tone I to tone II.

Accidental systolic noises, appearing more or less clearly in the recording with the middle-frequency characteristic, are sharply weaker in the high-frequency characteristic, or disappear entirely. Their epicenter may be located either in the area of the pulmonary artery or over the apex or medial portion, although they never irradiate clearly into the left axillary area. These noises are not pansystolic and, covering at a maximum up to one third to three fourths of the systole, are generally located in the proto-, protomeso- and mesosystole. Accidental systolic noises may have no clear configuration, changing from cycle to cycle and as a function of the phase of respiration, however quite frequently they are recorded as more or less well defined rhomboids or spindles. For many accidental noises, musicality is characteristic, manifesting itself in the phonocardiogram as true sinusoidal oscillations, particularly clearly expressed when recorded at the middle-frequency characteristic. The presence of a more or less clearly expressed interval between the beginning of the noise and tone I of the heart is an important, although hardly constant characteristic of accidental systolic noises.

In cases when differentiation between accidental systolic noises and organic mitral deficiency noise is difficult at rest, it seems useful and important to use functional phonocardiography.

The investigations which have been performed have shown that the most valuable of the various functional tests available is the test using inhalation of amyl nitrite, since it allows constant, clear and, which is most important, qualitative differences in the reaction of accidental

systolic noises and organic mitral deficiency noise to be revealed. These purposes can be served to a lesser extent by the orthostatic test.

The usage of the test with physical loading for differentiation of accidental systolic noises from organic mitral deficiency noise is not effective.

CHAPTER XV

ORGANIZATION OF CARDIOLOGICAL SERVICE IN SPORTS MEDICINE

ABSTRACT. The combined methodology of medical examination of those involved in sports is based on broad utilization of various methods allowing complete determination of the functional capacities of the organism. A system of the cardiological service in sports medicine includes sports dispenseries, periodic obligatory physical examinations of all athletes and the organization of scientific investigations. The organization of this system is described, and needs for its future development are noted.

Physical culture and sports are deeply embedded into the daily life of /489
Soviet man. With each year, ever more and more millions of people become involved in physical culture and sports. The morning gymnastics exercise and physical tempering, gymnastics in production areas, obligatory physical training sessions at all educational institutions, sports involvement in volunteer sports societies, tourist trips, exercises by middle-aged and elderly persons in "health groups," the tremendous number of sports competitions and games, holidays -- all of these are the individual links in a unified system of physical culture and sports, developed in our country, with the purpose of physical development, improvement of the health and lengthening of the life of our population.

At the present time, one of every four people in our country is involved in the area of physical culture. These include students, workers, and the intelligensia. In recent years, a significant number of middle-aged and elderly persons have become involved in physical culture. Massive physical culture, involving literally every person throughout his entire life is one of the facets of the development of the future communist society, a manifestation of the way that communism corresponds to the nature of man.

In the program of the CPSU, physical culture and sports are looked upon as one of the most important means of development of the Soviet man, harmonically combining spiritual wealth, moral purity and physical perfection.

The broad development of physical culture and sports in the USSR has been aided by the development of medical control over active athletes, as /490
well as the formation and growth of a new branch of knowledge -- sports medicine. During the years of Soviet power and particularly since the Second

World War, sports medicine has been developed into an independent medical discipline, occupying one of the most important positions in the preventative area of Soviet public health.

Sports medicine studies the effects of various forms of involvement in physical culture and sports on the human organism, performs constant medical testing of the physical development and health of athletes, determines and performs preventative and treatment measures in case of disease and trauma in athletes, defines the most effective sanitary and hygienic conditions for physical development and sports.

In our country, a scientifically well founded system of sports medicine has been developed, and the organization, content and direction of work have been defined; the network and structure of medical-physical culture institutions has been constructed. At the present time, a highly progressive system of medical support has been developed for those involved in physical culture and sports, based on the dispensary method. With each year, the number of medical-physical culture offices and dispensaries increases, the forms and methods of their work become more effective, their material support improves, the number of doctors specializing in physical culture increases and the quality of their training improves.

Sports medicine, enriched with new data, contains a great deal of valuable material for related areas of medical science. For example, sports medicine and sports physiology broadly investigate problems of the morphology and functioning of the human organism, the range of its functional capacities with various working regimens, in hypodynamia, under varied conditions of the external medium, in premorbid conditions, etc.

The attempt to approach more closely to the study of the whole organism has led to the creation of a combined methodology of medical examination of those involved in physical culture and sports, which has expanded the capacities to determine changes in the functional state of the organism during the process of training, has opened the path for active participation of the doctor in sports competition, has allowed the state of training to be determined, overfatigue and overtraining to be revealed.

The combined methodology of medical examination is constructed on the basis of broad utilization of various methods allowing the most complete determination of the functional capacities of the organism. The most important place in this combination is taken by investigation of the cardiovascular system, since its condition largely determines the level of functional capacities of the organism, the possibility of achieving highest results and the adaptability of the organism to work under varying conditions /491
of the external medium. The functional capability of the circulatory apparatus is particularly significant in physical loading in sports and determines to a considerable extent the adaptability of the organism to loads.

In sports medicine, it has been historically found that investigations of the circulatory apparatus have occupied the central position in the medical examination of athletes. The data from examination of the cardiovascular system are essentially the basic data used in determining changes in the functional state and state of training of the organism of the athlete. At the present time, over 10,000 doctors working in the ambulatory and policlinic network, performing examinations of physical culture activists. These examinations, performed as a part of the required "medical-test card of the physical culture activist" (Form No. 227), include particular investigations of the cardiovascular system using functional tests, electrocardiography and X-ray examination.

Each year, over 40 million physical culture activists must undergo one medical examination (and some persons two and three examinations) in which the greatest significance is attached to investigation of the circulatory apparatus. It is obvious from this that the problem of sports cardiology is the most important section of sports medicine. This statement becomes even more important if we consider the dispensary services provided to athletes.

The dispensary method of observation has become a strong part of sports medicine and at the present time is obligatory for leading athletes, included in the teams representing cities, oblasts, republics and the country. Dispensary examination of athletes is performed at main medical-physical culture dispensaries, of which there are approximately 200 in our country. The dispensary method includes regular medical examination (3-4 times per year), school medical observation under training and competition conditions. These examinations and observations include a large number of investigations of the circulatory apparatus.

In the dispensary examination of athletes, in addition to the generally accepted methods of clinical examination, the three-moment combined functional test, electrocardiography, phonocardiography, oscillography and roentgenkymography are used, and in recent times at a number of dispensaries polycardiography with phase analysis of the cardiac cycle has been introduced.

The usage of various methods of investigation during dispensary examination allows a deeper study of the functional state of the cardiovascular system of athletes, who must perform training loads of high volume and intensity.

The scale and volume of work being performed on the investigation of the /492
circulatory apparatus of physical culture enthusiasts and athletes has required the creation of laboratories and offices of sports cardiology at a number of leading medical-physical culture dispensaries in the country -- at Moscow, Sverdlovsk, Kiev, Tbilisi, etc. Analysis of the activity of these laboratories and offices shows that they are quite important and necessary links in the practical, methodological and scientific work being performed on sports medicine.

Based on four to five years experience of the work of these laboratories and offices, it can be considered that special laboratories of sports cardiology should be created at large medical-physical culture dispensaries (noncategorized and category I). At the remaining dispensaries, offices of sports cardiology should be organized, consisting of functional diagnostics sections or medical inspection departments.

The creation of laboratories and offices of sports cardiology has been found necessary at medical-physical culture offices as well. Thus, at the large medical-physical culture offices at the following stadiums: Dinamo in Moscow, the Republic stadium in Kiev, the stadium imeni S. M. Kirov in Leningrad, Dinamo in Tbilisi, etc. -- departments of functional diagnostics have been created, principally designed for investigation of the circulatory apparatus. Thus, practical necessity has resulted in the creation in many dispensaries and offices of new subdivisions for sports cardiology. Certain practical conditions have been set up for the creation of a cardiological service in sports medicine. In this service, the laboratories and offices of sports cardiology should be the principal and basic links in the organization of the system as a whole through the country.

A second important aspect in the development of the cardiological service is the organization of scientific investigations. In this area, a great deal of scientific investigation work is being done, and tremendous amounts of scientific material have been accumulated. Methods are being developed for investigation of the cardiovascular system at rest and directly in the process of muscular work at the Department of Medical Testing and the Laboratory of Sports Cardiology of the State Central Order of Lenin Institute of Physical Culture. For example, the following methods have been developed to answer the demands of sports medicine: polycardiography with phase analysis of the cardiac activity, electrocardiography under conditions of intensive muscular effort, determination of the volume of an athlete's heart, electrokymography, etc. Here also, problems in the organization of the cardiological service in the country are being developed.

In the sector of sports medicine of the All-Union Scientific Research Institute of Physical Culture, many years effort have gone into the development of problems in the area of the study of the functional state of the circulatory apparatus and the diagnosis of the state of training. In the /493
sector of sports physiology of this same institute, problems of the influence of various conditions and loads on the cardiovascular system are being studied. At the Department of Sports Medicine of the Leningrad Institute of Physical Culture, problems related to disruptions of the activity of the cardiovascular system in athletes are being studied.

Problems of sports cardiology are also being studied at many departments of medical testing at medical institutes and institutes of physical culture. If we compare the totality of all scientific investigations on sports cardiology performed at various institutes, their volume is much greater than the volume of investigations on all remaining problems of sports medicine. We can say without exaggeration that at the present time, the scientific

foundations for sports cardiology have been laid. Consequently, objective prerequisites have been established for the creation of an ordered cardiological service in sports medicine. In connection with this, the important problem of organization of the cardiological service on a nationwide scale arises, since this service must be an organic part of the system for the organization of work in sports medicine as a whole which has already been structured and is now being developed.

However, in order to create and develop a well ordered system of organization of the cardiological service, it seems to us necessary to solve a number of problems. One of the most important problems is the system of training of medical cadres in the area of sports cardiology. In this area, a necessary condition is preparation of new programs of courses for the improvement and specialization of physical culture doctors in the system of institutes for improvement of doctors. A useful form of increase in the knowledge of doctors working in the area of sports cardiology should include seminars, conferences, symposiums, etc.

The work on sports cardiology being performed both in practical and in scientific institutions requires constant generalization on a nationwide scale. This creates the necessity of setting up a special division or laboratory for sports cardiology at the leading institute, to act as a planning, coordinating and controlling institution for these scientific problems.

In conclusion, we can state that the term "sports cardiology" has come to reflect one of the most important trends in scientific investigation and in practical work in sports medicine.

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