THE EFFECTS OF ATHLETIC TRAINING ON THE RESTING ELECTROCARDIOGRAPHIC RECORDS OF COLLEGE WRESTLERS AND SWIMMERS

By

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CHAPTER I

INTRODUCTION

Statement of the Problem

Examination of the published material on the circulation of trained athletes reveals that there are several conflicting studies. The only features enjoying general agreement among researchers is that bradycardia can be induced by training and an increase in heart volume does take place. Mellerowicz and others presented the view that the trained circulation at rest is manifested by a low cardiac output, mediated via a slow heart rate and reduced stroke volume.[1] Frick[2], however, reports that an enhanced stroke volume is due to training.

Rautaharju and others have reported[3], that the Rand T amplitudes in the conventional leads of skier's ECG are significantly higher than those of a healthy adult population. Cureton[4] in a study conducted on

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[1]H. Vergleichende Mellerowicz, "Vntersuchungeu uber das Okonomieprinzip in Arbeit und Leistung destrainierten Kreislaufs und seine Bedeutung seine Bedeutung fur die praventive und rehabilitive Medizin," Archiv Fuer Kreislaufforshung, XXIV (1956), p. 70.

[2]M. H. Fricks, Physical Activity and the Heart (Springfield, Illinois, 1967), p. 33.

[3]P. M. Rautaharju et al., "Heart Size of Champion Skiers," Annales Medicinae Internai Fenniae, XXXXVI (1957), p. 169.

[4]Thomas K. Cureton, Physical Fitness of Champion Athletes (Urbana, Illinois, 1951), p. 227.

champion athletes reported similar findings with R and T amplitudes being higher than a group of normal young men. After completing this electrocardiogram study, Cureton[5] suggested the need for careful experimentation on subjects to show whether athletic training affects the transient record in comparison to the permanent or beginning record.

Purpose of Study

The purpose of this study was to evaluate the effects of athletic training on the resting electrocardiographic record of college wrestlers and swimmers. The characteristics of the resting ECG records that were of interest, were the wave amplitudes, time intervals, heart rate, and the QRS axis.

Definition of Terms

Amplitude - The height or amount of a wave above or below the isoelectric line.

Isoelectric line - The horizontal line inscribed on the graph during diastole of the heart beat, also known as the "base line", "diastolic level", and "zero level".

Positive wave - A wave that is above the isoelectric line.

Negative wave - A wave that is below the isoelectric line.

Inverted wave - A wave that is above or below the isoelectric line but normally would be found inscribed in the other direction.

Peak of a wave - That portion of a wave where it reaches its greatest point of deflection from the isoelectric line.

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[5]Ibid.

Complex - A series of waves which are generally considered to be together.

Cycle length - The duration of one complete beat of the heart or the distance on the graph from the one point on a wave to the same point on the following wave of the same kind, such as R wave to R wave.

Leads - The arrangement or way in which the action currents of the heart are led off of the subject's body to the electrocardiographic recorder where it is amplified and recorded on the moving sensitized paper. The Standard Leads were the only leads used in this study, and they were Leads I, II, and III and are shown in Figure 1.

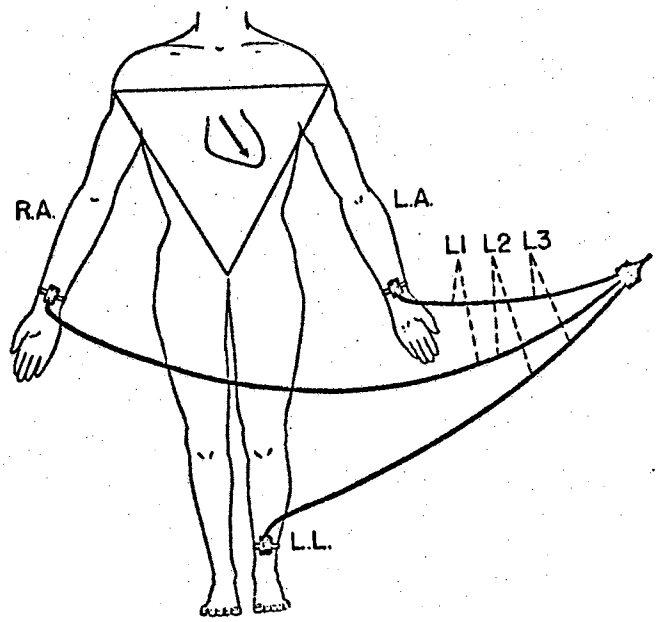

Figure 1. Method of Obtaining Three Standard Leads

R.A. = right arm
L.A. = left arm
L.L. = left leg
L1 = first lead
L2 = second lead
L3 - third lead

(Courtesy of Sigler)

Lead I - The electrode connections are made with the subject's right and left arms.

Lead II - The electrode connections are made with the subject's right arm and left leg.

Lead III - The electrode connections are made with the left arm and left leg.

ECG - The abbreviated form of electrocardiogram.

Bradycardia - A term used to refer to subjects who at rest exhibit a heart rate of 60 to 40 beats/minute.

Inherited Bradycardia - Individuals who have a heart rate below 60 who did not obtain it by any physical training program, but inherited the characteristic.

Training-Induced Bradycardia - A term used to refer to subjects whose heart rate has dropped below 60 beats/minute because of physical training programs.

Hypertrophy - A condition of excessive development of the heart, as from excessive use.

Limitations of Study

Limitations were as follows:

1. The hour of the day for testing was not consistent throughout the study, and according to Simonson[6] this could have a variable effect on results.
2. Almost no attempt was made to control the usage of 110 voltage in

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[6]E. Simonson et al, "The Effects of Meals on the Electrocardiogram in Normal Subjects," American Heart Journal, XXXV (August, 1964), p. 202.

the lab during testing, and its interference does distort ECG recordings.

3. The skin of the subjects was not cleaned before applying the electrodes.
4. The standardization records of the earlier recordings were not saved thus reducing the reliability of the records because the proper standardization could not be checked or corrected.
5. No attempt was made to apply the correction factor for errors in standardization due to the fact that the errors were so minimal that it would not effect the records.
6. No restriction of the subject's activities was made other than to take all recordings prior to practice, and only after a ten minute rest period after the subjects reached the lab.
7. An equipment failure on the last test date of several of the subjects made rescheduling impossible.
8. The testing frequency was not consistent throughout the study due to scheduling problems and the missing of appointments by the subjects.

CHAPTER II

REVIEW OF RELATED LITERATURE

Application of Electrocardiography

Since the reader may not be familiar with the electrocardiograph recorder, this chapter will begin with an explanation. According to Sigler[1], the electrical current generated and propagated in the heart is conducted from the heart to the surface of the body by various tissues. From the surface it is carried off by cables which make a circuit with a conductor of very small dimensions, situated between two electromagnets of the galvanometer. The electrical current from the heart thus passes through the conductor alternately from above downwards and from below upwards, depending upon its direction from moment to moment. This alternate upward and downward flow of current through the conductor creates a variable magnetic field, thus causing rapid movements of the conductor from side to side giving us the electrocardiogram.

"The current generated in the heart is infinitesimal in force, and the vibrations it sets up are therefore very minute. In order to record these vibrations, the electrocardiograph is equipped with a special device for amplifying them. The electrical impulses from the heart are magnified in a sufficient degree to move the stylus of the machine, and

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[1]Louis H. Sigler, The Electrocardiogram, Its Interpretation and Clinical Application (New York and London, 1957), p. 2.

are thus recorded on the paper. The mangitude and direction of such movement depend upon the magnitude and direction of the current coming from the heart at that moment."[2]

Anatomic Basis

The heart is supplied with specialized tissue, as described by Sigler[3], for the creation and propagation of electrical impulses. These are the sino-auricular node, the auriculoventricular node, the auriculoventriculo-bundle, the bundle branches and the ramifications of the latter into the aborization of the purkinje system of fibers, as illustrated diagrammatically in Figure 2. The sino-auricular node is in contact with the vetal and sympathetic nerve fibers. The auriculoventricular node, the bundle branches, and the aborization of the purkinje system of fibers, form a continuous system of special neuromuscular tissue between the base of the auricles and the entire ventricular musculature of both ventricles.

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[2]Ibid., p. 6.

[3]Ibid.

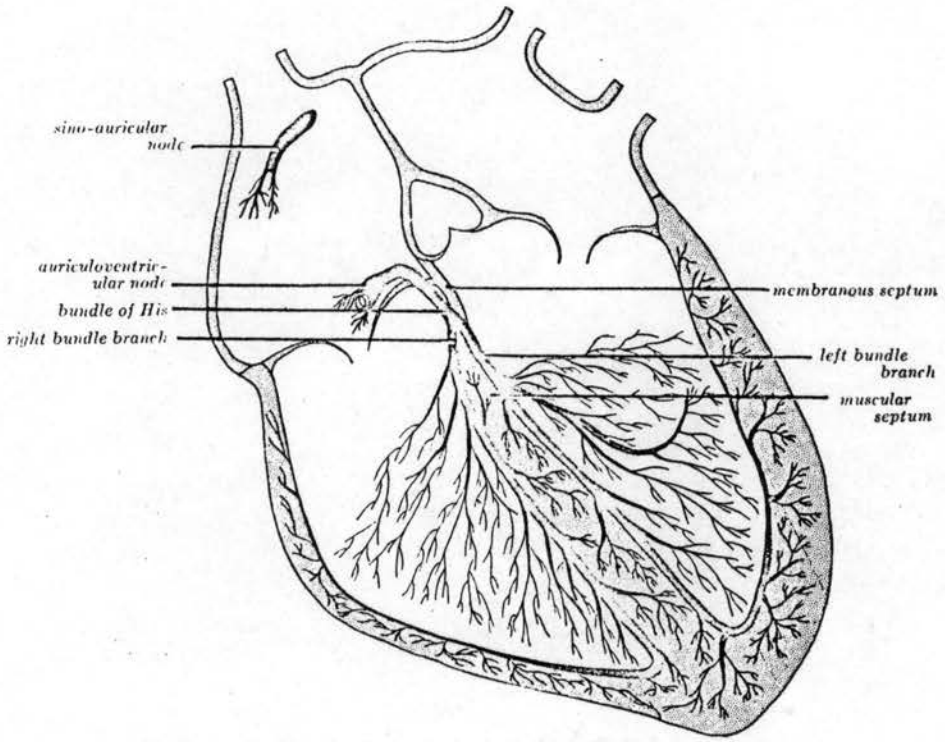

Figure 2. Diagram of Conduction System and Its Relation To Other Parts of Heart

(Courtesy of Sigler)

Initiation and Propagation of the Impulse

According to Sigler[4], the impulse that sets off the electrical activities in the heart originates in the sino-auricular node (pacemaker). This impulse immediately spreads throughout both auricles in a radiating fashion in all directions and reaches the auriculoventricular node (A-V node). There the impulse is slowed somewhat. This slowing is a natural adaptive mechanism to give time for the auricles to end their activity

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[4]Ibid., p. 13.

before the ventricles go into action. From the A-V node the impulse passes downward at an extremely fast rate along the bundle of His, its two branches, and the aborization of the Purkinje system of fibers, ultimately reaching all the ventricular muscle tissue. Thus the spread of the current in the ventricular musculature is from within out, that is, from the endocardial to the pericardial surface. The electrical events are shown diagrammatically in Figure 3. The general direction of the sum total of the current flow in ventricles is from above downward and from right to left.

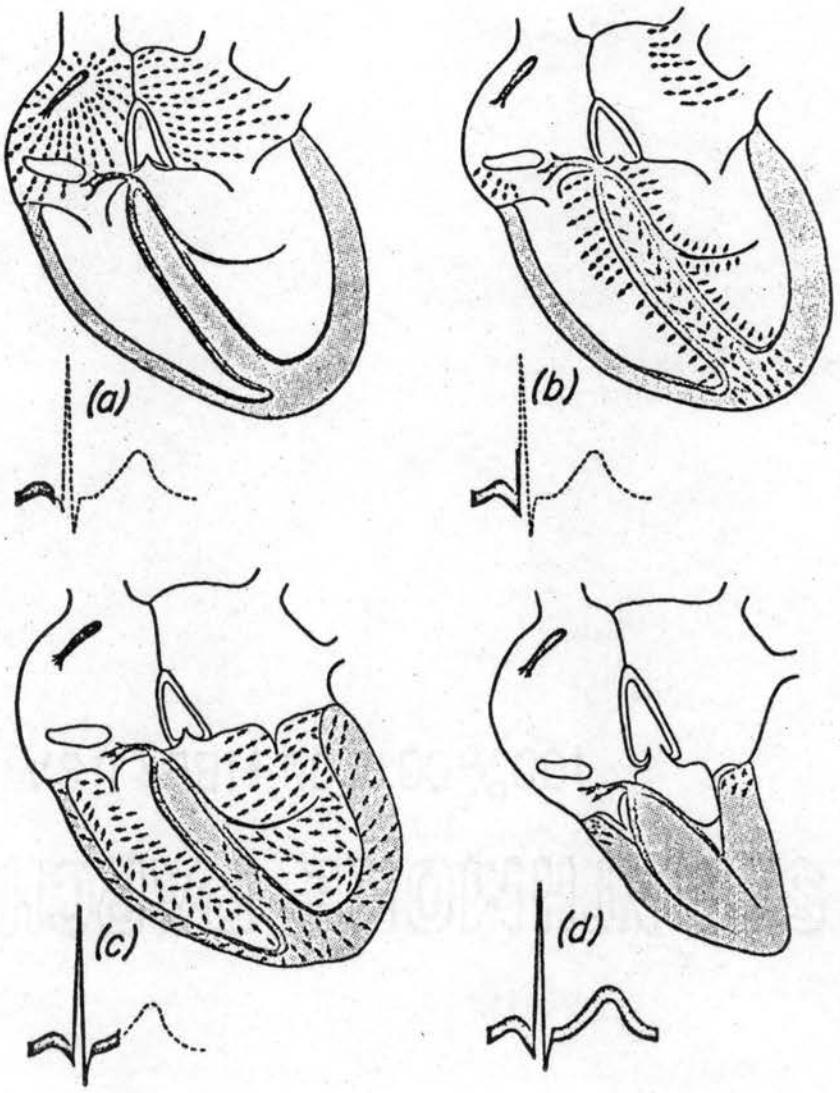

Figure 3. Spread of Impulse in Heart and Inscription of Electrocardiogram

a. The impulse originates in the sino-auricular node and invades the auricles. The P wave is inscribed during this period.
b. The impulse in the auricles has receded, except for isolated areas. The ventricles are beginning to be invaded. Areas closest to the bundle of His and the two bundle branches are first involved. The QRS complex is beginning to form.
c. The entire intraventricular muscle is invaded. The part that was invaded in the earliest stage shows some recession of the impulse. The -QRS complex and the S-T segments are fully inscribed.
d. The impulse has almost entirely receded, except for isolated areas, and the ventricles are in full contraction. The RS-T segment and the T wave are inscribed immediately before and during this stage.

(Courtesy of Sigler)

The Normal Electrocardiogram

The application of the word "normal" as used here is applied to the characteristic of the tracing, and not the condition of the heart. According to Halloway[5], "the normal electrocardiogram is the recording of a series of deflections or waves produced by the upward and downward movement of the stylus. This series of deflections usually is grouped into five consecutive waves arbitrarily designated as P, Q, R, S, T, and sometimes a U wave. Each group of these five or six waves represent a complete heart cycle. The P wave is a reflection of the electrical activity occuring in the auricular muscle and QRST waves are primarily the electrical activity in the ventricular muscles. During diastole there are no upward or downward deflections. The recording during this phase is usually horizontal and is referred to as the isoelectric line."

The Individual Waves

The following description of the components of the normal electrocardiograms are based on reports of Sigler[6], Cureton [7], Halloway[8], and the Criteria Committee[9] of the New York Heart Association. Figure 4 should aid the reader with the explanation of the individual waves.

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[5]Fred T. Halloway, "An Electrocardiographic Study of College Athletes" (unpub. Ph.D. Thesis, University of New York, 1954), p. 4.

[6]Sigler, pp. 33-47.

[7]Cureton, pp. 148-151.

[8]Halloway, p. 6.

[9]The Criteria Committee of the New York Heart Association, Nomenclature and Criteria for Diagnosis of Diseases of the Heart, pp. 131-139.

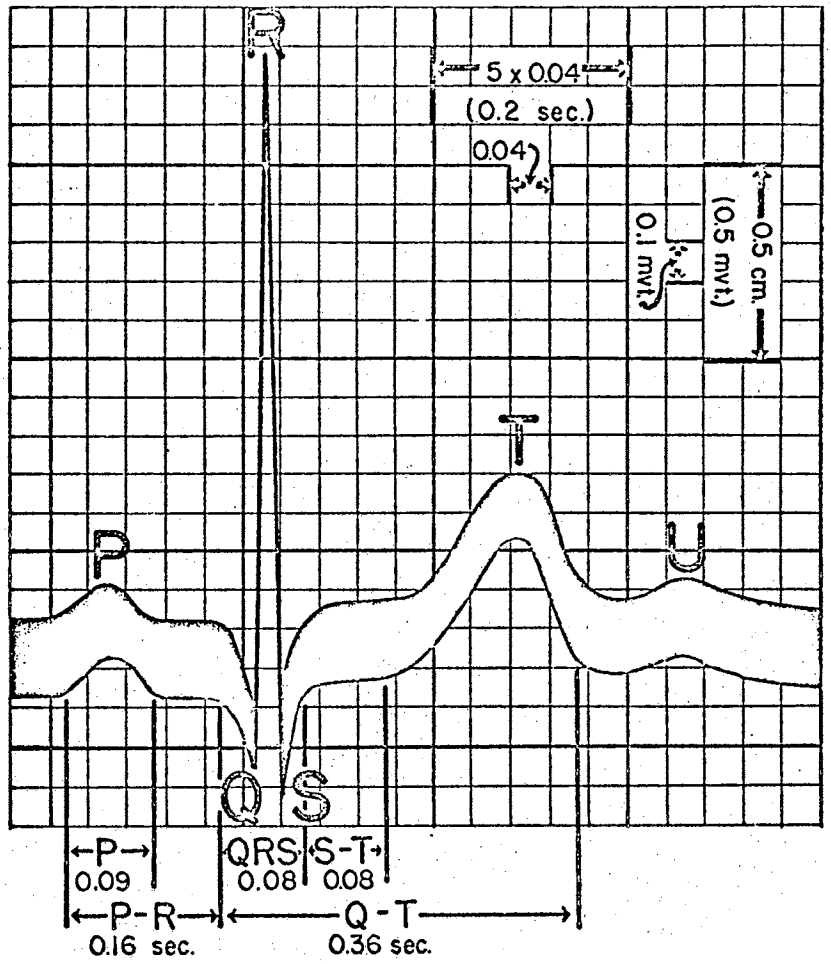

Figure 4. Drawing of Actual Electrocardiogram Enlarged About Five Times

Each single square of the graph represents 1 mm. Each group of 5 mm. is marked off in the horizontal and vertical by thicker lines. Measured in the horizontal, each millimeter represents a time interval of 0.04 second, therefore 5 mm. equals 0.2 second. Measured in the vertical, each millimeter represents 0.1 mv. of current, if we employ the usual standardization, 1 cm. of deflection = 1 mv. of current. Hence, 5 mm. or 0.5 cm. of deflection equals 0.5 mv. of current. The duration of the P wave, P-R interval, QRS interval, S-T interval, and Q-T interval respectively, for this normal electrocardiogram, is shown in the corresponding sections of the tracing.

The P Wave

This wave is inscribed by the electrical activity associated with auricular contraction. It is therefore the first wave in the electrocardiogram. It is usually positive in the three standard leads. The shape of the P wave is usually round but it may be pointed. In about 30 per cent of normal cases, there may be some notching.

In Lead I the amplitude of the P wave ranged from 0.3 to 2.0 mm with a mean of 0.92 mm in a Cureton[10] study of eighty-one normal young men. In this study Lead II was found to have a range from 0.3 to 3.3 mm with a mean of 1.31 mm; and Lead III with a range of 0.2 to 2.8 mm and a mean of 0.96 mm.

The Criteria Committee of the New York Heart Association[11] indicates that the normal limits of the P wave amplitude ranged from 0.5 mm to 2.5 mm in that one of the three leads in which the P wave is highest.

The P-R Interval

It represents the time required for the passage of the electrical impulse from the sino-auricular node (S-A node), through the auricles, the auriculoventricular node (A-V node), and the bundle braches down to the terminal aborization. The greater part of this interval is consumed by the passage of the impulse through the A-V node. This interval is usually isoelectric, because no muscle masses are activated during this period[12].

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[10]Cureton, p. 142.

[11]The Criteria Committee of the New York Heart Association, p. 136.

[12]Sigler, p. 34.

The Criteria Committee[13] indicates the normal upper limits to be 0.20 second in adults with normal heart rates, and would be found in the longest interval of any of the standard leads.

Cureton[14] in his study of eighty-one normal young men found that in Lead I a range of 1.10 to 0.20 second existed with a mean of 1.176 second. In Lead II the limits were between 0.12 and 0.22 second with a mean of 0.177 second, and in Lead III the range was 0.12 to 0.20 and with a mean of 0.175 second.

The R Wave

The range of R wave amplitude in Lead I is from 1.2 to 13.3 with a mean of 5.30 mm, and in Leads II and III it was 4.7 to 23.8 and 1.5 to 18.8 mm with means of 13.19 mm and 9.35 mm, respectively.[15] The Criteria Committee[16] gives the normal limits as not more than 20.0 mm or less than 5.0 mm in the standard lead that contains the highest wave.

The S Wave

The S wave amplitude ranged from 0 to 6.1 mm with an average of 1.26 mm in Lead I. In Lead II it ranged from 0 to 4.3 mm with mean of 1.10 mm, whereas in Lead III the amplitude ranged from 0 to 6.3 mm with an average of 0.75 mm.[17]

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[13]The Criteria Committee of New York Heart Association, p. 136.

[14]Cureton, p. 148.

[15]Ibid.

[16]The Criteria Committee of the New York Heart Association, p. 137.

[17]Cureton, p. 148.

QRS Complex

According to Sigler,[18] "the inscription of this complex begins with the earliest invasion of the ventricular myocardium by the impulse and ends where every part of the ventricular muscle has been completely involved. In as much as certain portions of muscle are invaded slightly earlier than others, and the course of the spread of the impulse points to some extent upward and laterally, although the main direction above downward and from right to left, the QRS complex is not a single uniform wave but is composed of more than one wave. The variations are due to the differences in the direction of the main impulse from moment to moment in relation to the location of electrodes in the given lead. An impulse that has its main direction of flow parallel to a given lead will express itself by a large wave in that lead. On the other hand, an impulse that has a perpendicular direction to the lead will show no wave in that lead."

Cureton[19] found in his study of the normal young men that the mean QRS duration was found to be 0.065 in Lead I, 0.068 in Lead II, and 0.067 in Lead III.

The T Wave

According to Sigler,[20] "in normal cases, the repolarization wave or T wave usually takes the same direction as the main deflection of the

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[18] Sigler, pp. 35-36.

[19] Cureton, p. 148.

[20] Sigler, p. 41.

QRS. There is, however, some inconsistency in the relationship between these two phases of the electrocardiogram. No satisfactory explanation for these inconsistencies has so far been given. They may indicate the process of the depolarization and repolarization in the ventricular muscle does not follow the same sequential routes. A more likely explanation is that there is a variation in the duration of repolarization in different parts of the ventricular musculature. This is evidenced by the fact that the T wave is the most unstable part of the electrocardiogram."

"The height or voltage of the T wave varies considerably in different individuals and in the same individual in different Leads. In the majority of normal hearts its height relationship in the three standard leads is such that height in the second lead equals the combined heights of those of the first and third leads. Where the T wave is positive in all leads, its height will therefore be greatest in the second lead. Where it is negative in Lead III, its height will be greatest in Lead I," according to Sigler.[21]

Cureton,[22] in his study of normal young men found the T-wave amplitude to range from -1.7 to 8.5 mm in Lead I, with a mean of 2.777 mm. In Lead II it ranged from 0.8 to 8.5 mm in amplitude, and with a mean of 4.2 mm. Lead III ranged in amplitude from -1.7 to 14.6 mm and with a mean of 1.87 mm. The Criteria Committee[23] gave the normal amplitude of the T wave as being between 1.0 mm and 5.0 mm in the standard lead of

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[21]Ibid.

[22]Cureton, p. 149.

[23]Criteria Committee of New York Heart Association, p. 139.

highest deflection.

The U Wave

Most of the literature written on this wave is rather garbled. Cureton[24] indicates that it is found only in superior athletes, and appears between the T and P wave. Sigler[25] does not venture a theory about this wave until more is understood about its characteristics. He does, however, give a range of 0.5 to 2.0 mm of amplitude for it, and a time duration of 0.18 to 0.24 second.

Some Factors Affecting the Electrocardiogram

Sensenback[26] presents 47 conditions not due to primary heart disease in which changes occur which are similar to the changes in the QRST complex, and especially the ST segment and T wave due to myocardial disease. These conditions included eleven different drugs, exercise, nine types of acute infections, acute and chronic pericarditis, fourteen types of acute upper abdominal disease, pulmonary embolism, two types of autonomic nervous imbalances.

Simonson[27] showed that consistent changes occur in various fundamental electrocardiographic functions after eating of moderate meals and that the most important effect is the decrease of the T wave. He also

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[24]Cureton, p. 141.

[25]Sigler, p. 45.

[26]W. Sensenback, "Some Common Conditions, Not Due to Primary Heart Disease, that May be Associated with Changes in the Electrocardiogram," Annuals of Internal Medicine, XXV (October, 1946), pp. 632-647.

[27]Simonson, pp. 202-214.

found that body position does have various effects on the fundamentals of the electrocardiogram.

Reliability of the ECG Measurements

The changes occurring from time to time in an individual's record are primarily concerned with: (1) horizontal time intervals such as the P-QR, QRS, work, and rest times; (2) vertical displacements such as the P, Q, R, S, T, and U waves according to Cureton.[28] These measurements being recorded in mm and fractions of a second can vary tremendously if standardization is not obtained before testing. To obtain a good tracing proper standardization of 1 millivolt or 1 centimeter of deflection must be employed within a 0.02 second or less according to Sigler.[29]

Related Studies

Tuttle and Korns[30] conducted a study of forty-eight athletes (four wrestlers, seven gymnasts, nine swimmers, six basketball players, and twenty-two track men) who were electrocardiographed at the beginning and near the end of a training season. Their objective was similar to that of this study. Their results were that no difference was found in forty-three of the forty-eight cases. However, in the remaining five, one had an insignificant difference. Of the four remaining subjects, one subject, a twenty year old gymnast, had a much more definitely

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[28]Cureton, p. 139.

[29]Sigler, p. 25.

[30]W. W. Tuttle and H. M. Korns, "Electrocardiographic Observation on Athletes Before and After a Season of Physical Training," The American Heart Journal, XXI (January, 1941), pp. 104-107.

inverted T wave in Lead III, and yet with no change in Leads I and II. A thirty-one year old basketball player had no change in Lead I, a change to a diaphasic T wave in Lead II, and the degree of inversion of the T wave increased in Lead III. Another subject, a twenty-nine year old swimmer, had an increase in amplitude of the R and T waves in Lead I, no change in Lead II, but had a change from an inverted to an upright P wave in Lead III. The last subject, a twenty-one year old wrestler, had an increase in amplitude of the R and T waves in Lead I, no change in Lead II and a change from isoelectric to an inverted T wave in Lead III.

Halloway's study[31] found that athletes can be grouped by characteristics of their ECG according to the degree of tendency found for the particular sport group or combination of sport groups in which they participated.

In an extensive study on 107 sportsmen, Frick[32] found an enhanced stroke volume due to training which was supported by Wang[33] and associates. It was found that bradycardia due to training also showed a significant increase in heart stroke volumes, the size of the heart, and reduction in the number of beats per minute. However, the heart rate did decrease in the subjects with inherited bradycardia, which implies that this did not directly connect to the other circulatory effects of training but is regulated by other means to complete the picture of

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[31]Halloway, p. 219.

[32]Frick, p. 33.

[33]Y. Wang et al., "Cardiac Response to Exercise in Unconditioned Young Men and in Athletes," Circulation, XXIV (October, 1961), p. 1064.

trained circulation.

Rautaharju,[34] in a related study in the symposium, found that the magnitude of mean QRS and T vectors, determined from the conventional ECG's, were significantly higher in athletes.

From the investigations of Vijer,[35] Mortensen,[36] and Sokolow and Lyon,[37] high amplitudes in ECG's are known to be a common manifestation of hypertrophy of the heart. On the other hand, an increased level of physical activity is known to produce hypertrophy of the heart at least in experimental animals.

In an investigation by Karvonen and associates,[38] the ECG's of a group of nineteen long-distance skiers were characterized in quantitative terms and compared with ECG's of a group of twenty sedentary male subjects. The spatial magnitudes of all vectors of the athletes were significantly larger than those of the normal male subjects. The vector cardiographic patterns of athletes are compatible with the electrocardiographic pattern of a "diastolic overload" of the left ventricle,

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[34]P. M. Rautaharju, "Voltage Changes in Electrocardiogram as Caused by Vigorous Training," Abstracts of Communication (3rd World Congress of Cardiology, Brussels, 1958).

[35]T. Vijer, "Über Die Bedeutung der Grosse der Komplexe des Elektrokardiogramms," Deutsche Medizinische Wocenshriff, I (1938), p. 21.

[36]V. Mortensen, "Die Electrocardiographische Diagnose des Herfinforkts," Archiv Fuer Kreislaufforschung, 1942, p. 115.

[37]M. Sokolow and T. P. Lyon, "The Ventricular Complex in Left Ventricial or Hypertrophy as Obtained by Unipolar Precordial and Limb Leads," American Heart Journal, XXXVII (December, 1949), p. 161.

[38]M. S. Karvonen et al., "Heart Disease and Employment. Cardiovascular Studies on Lumberjacks," Journal of Occupational Medicine, III (February, 1961), p. 49.

according to the concept of Cabrera and Sodi-Pallares.[39]

Rautakarju and Karvonen[40] concluded on the basis of the study on the nineteen long-distance skiers and the group of twenty sendentary male subjects, that the possible mechanism underlying development of increased ECG magnitudes was due to changes in the distance of the heart to the recording electrodes which decreased as a consequence of adaptive dilation. It might, however, be explained by the increased diameter of the muscle fibers with hypertrophy, and possibly also alignment and lengthening of the muscle fibers which are factors that might affect the current dipole moment and cause an increase of the ECG magnitude.

Summary of Related Studies

In a similar study Tuttle and Korns found only slight alterations in the ECG records of wrestlers and swimmers. In the studies conducted by Frick and Wang significant increases were shown in the stroke volume and heart size, while a decrease in heart rate was exhibited. Crueton, Rautaharju, Karvonen and others, on the basis of their studies, indicate that athletes exhibit higher ECG magnitudes than normal young men of the same age. According to Vijer, Mortensen, Sokolow, and Lyon high amplitudes are a common manifestation of hypertrophy of the heart. As to the cause underlying the mechanism of hypertrophy, several theories have been set forth.

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[39]D. Sodi-Pallares, New Bases of Electrocardiography (St. Louis, 1956).

[40]P. M. Rautaharju and M. J. Karvoneu, Physical Activity and the Heart (Springfield, Illinois, 1967), p. 181.

CHAPTER III

METHODOLOGY

The research methodology for this study was the case study with twelve volunteer subjects being tested at various intervals throughout the five to six months of athletic training.

Five of the subjects were wrestlers whereas the remaining seven subjects were swimmers. All of the subjects were athletes at Oklahoma State University who were currently participating in their athletic area during the Fall semester 1966, to the Spring semester 1967. The subjects ranged in age from eighteen to twenty-one years of age, and in weight from 128 to 210 pounds. The skill and experience level of these athletes can be determined from the fact that two of these subjects were National N. C. A. A. Wrestling Champions and one a Big Eight Freestyle Swimming Champion.

The wrestling subjects began their physical training at the beginning of October 1966, whereas the swimmers did not begin their training until the latter days of October 1966. In most cases the ECG testing of the subjects began approximately two weeks after the beginning of their physical training season. The last tests were taken prior to the NCAA Wrestling finals, and prior to the Big Eight Swimming Tournament. The frequency of testing ranged from three to six times throughout the training season.

Instrument of Measurement

The Birtcher Model 335 compact electrocardiogram recorder was the instrument used in this study (see Figure 5). The records made in this study were taken from the first three leads (standard leads). The administration of the testing was done solely by the author with the Birtcher recorder.

Testing Procedure

The following test procedure was performed on all twelve cases to evaluate the possible effects of athletic training on the resting ECG records of the athletes. The subjects were tested from three to six times during the duration of the study. The first recording was made in the early weeks of training and continued throughout the season at irregular intervals.

Testing Hour of the Day

Generally, most of the records were recorded prior to the subjects' lunch. However, there were incidents where subjects missed the scheduled hour of testing and then they were recorded just prior to the practice of that day and never after a practice.

Resting ECG Recordings

It was the interest of the author to take a resting ECG of the subjects. Only after an approximate ten minute or more rest period, lying in the supine position on a rather large wood table in the research laboratory were the subjects' ECG recorded. Before the electrodes were

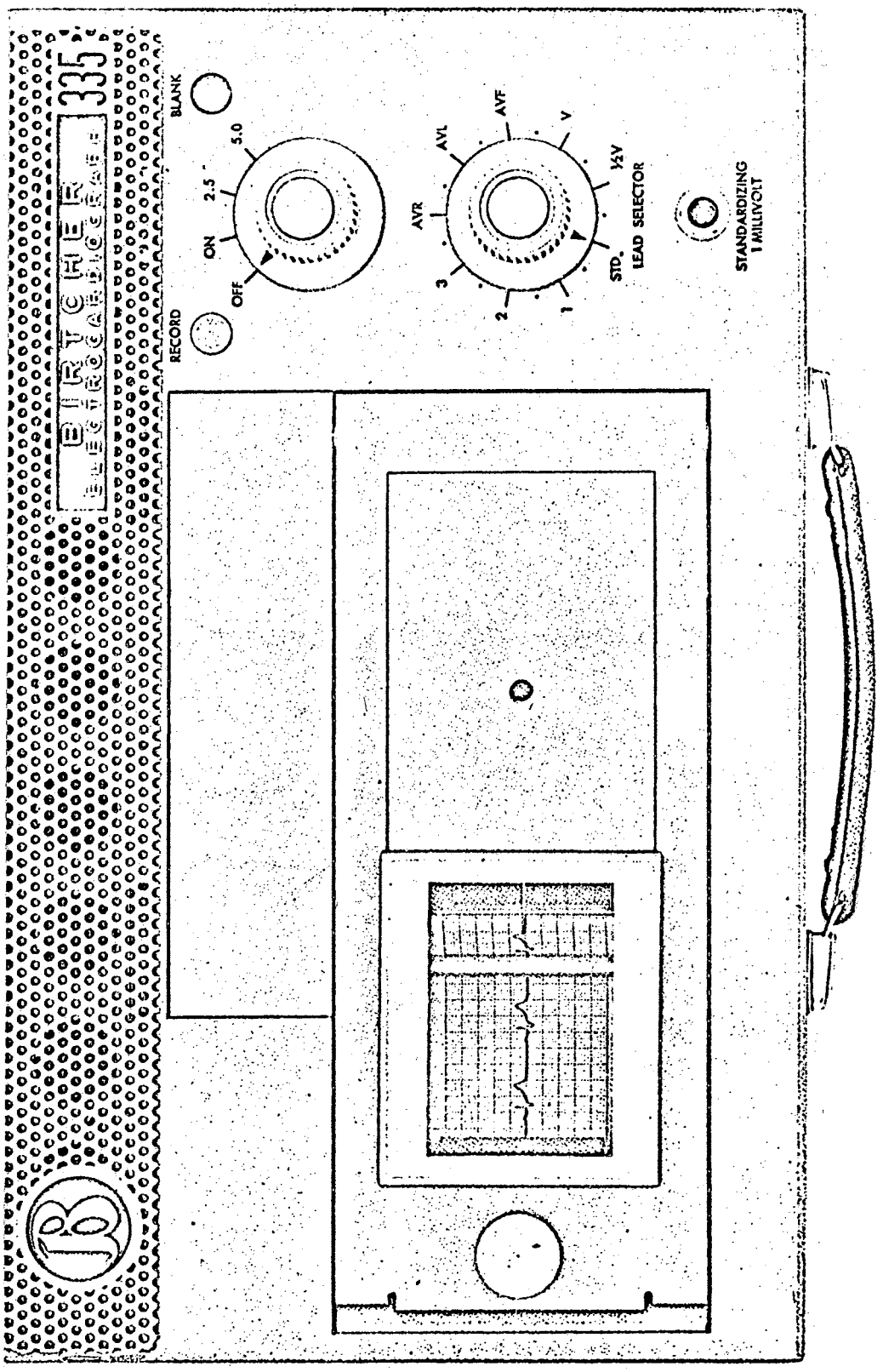

Figure 5. The Birtcher Electrocardiograph 335

placed on the prospective limbs they were cleaned with the cleaning compound, "Comet." No preparation of the skin was made by the author other than by employing the electro-paste suggested by the Birtcher Company to be used in conjunction with the electrodes.

Warm-up and Standardization of the Recorder

The recorder was always turned on from fifteen to thirty minutes prior to testing time to insure ample warm-up time. After the electrodes were connected to the subject, the machine was standardized to the specifications found in the Birtcher Model 335 Operation Manual. This standardization consists of a deflection of one millivolt or one centimeter within an 0.02 second interval duration.

Length of Recordings

A recording was made in each of the three standard leads for a duration of ten seconds.

Method of Measuring the ECG Records

Measurement of the amplitudes and time intervals was obtained in accordance with Cureton[1] method, with the exception that needle points were attached to Vernier calipers to insure accurate readings.

Method of Measuring the Heart Rate

The heart rate was determined by the use of the Birtcher ECG ruler (catalog number 79-1) in which the first two complete cardiac cycles

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[1]Cureton, pp. 138-139.

were measured. In this study, the author always took the first three R waves appearing in the six second samples to determine the rate.

Tabulation of Data

The electrocardiographic records were tabulated under appropriate wave headings and according to the case number. In a case where a particular wave was not present, record of its measurement was tabulated as zero. All waves that appeared above the isoelectric line were tabulated as positive, and those appearing below were tabulated as negative (-) in value. Data for each case was recorded in a table for the entire duration of the study. This table appears with each case presentation in the results. No statistical treatment was employed since the small number of subjects warranted only descriptive case presentation and a comparison of means.

CHAPTER IV

RESULTS

Case One - Gene Davis (Wrestler) - Age 20

Amplitudes

The P amplitudes were only slightly altered in all three leads. The subject's entire QRS complex was drastically changed in Leads I and III. In Lead I the R amplitude was reduced by 15.6 mm, and increased by 31 mm in Lead III. The S amplitudes were, of course, significantly altered too as there was an increase of 3.1 mm and 4.8 mm in Leads II and III. A small change was shown in the T amplitude with Lead III showing the greatest change of 1.8 mm increase in amplitude height.

Time Intervals

The rest/work ratio was the only time element showing any change, and it was increased .94 and 1.04 in Leads II and III respectively.

Heart Rate

His heart rate falls within the bradycardia range, and he showed a reduction of 12 beats/minute from his beginning heart rate average, 54 beats/minute, to his final heart rate average, 42 beats/minute.

QRS Axis

The first axis recorded was a left axis deviation of minus 28 degrees, whereas the final axis was recorded in the direction of the normal axis at 97 degrees. The subject showed a change of some 115 degrees.

TABLE I

RAW DATA, CASE I

	Date						
	10-13-66	10-31-66	11-15-66	12-7-66	12-20-66	2-2-67	3-16-67
LEAD I							
Ampl. of P (mm)	1	1.5	1	0.7	0.8	0.5	0.7
Ampl. of R (mm)	18	17	3.5	1.9	3.2	3.1	2.4
Ampl. of S (mm)	2	0.5	-1.5	-2	-1.5	.99	1.3
Ampl. of T (mm)	4.2	3.9	3.5	2.8	3	3.6	3.4
P-QR int. (sec)	0.240	0.15	0.12	0.12	0.12	0.128	0.148
QRS int. (sec)	0.040	0.06	0.04	0.05	0.05	0.044	0.048
Work (sec)	0.340	0.36	0.36	0.38	0.38	0.392	0.340
Rest (sec)	0.920	1.04	0.81	0.54	0.82	0.980	1.016
Rest/work	2.70	2.88	2.25	1.42	2.17	2.5	2.9
Heart Rate	48	43	50	67	47	42	45
LEAD II							
Ampl. of P (mm)	1	0.8	0.7	1	1	0.9	0.8
Ampl. of R (mm)	3.2	16.2	8.1	20	19	19.95	7.8
Ampl. of S (mm)	-1	1	0	0.5	0.5	2.1	2.1
Ampl. of T (mm)	3.5	3.7	4	3.8	3.2	4.5	3.4
P-QR int. (sec)	0.120	0.16	0.16	0.15	0.12	0.112	0.068
QRS int. (sec)	0.04	0.07	0.07	0.06	0.05	0.068	0.056
Work (sec)	0.35	0.36	0.36	0.37	0.36	0.440	0.380
Rest (sec)	0.64	0.95	0.96	0.78	0.88	1.048	1.096
Rest/work	1.82	2.63	2.69	2.1	2.44	2.38	2.88
Heart Rate	58	45	44	52	45	42	40

TABLE I (Continued)

	Date						
	10-13-66	10-31-66	11-15-66	12-7-66	12-20-66	2-2-67	3-16-67
LEAD III							
Ampl. of P (mm)	-0.2	-0.2	0	0.1	0.1	0	0
Ampl. of R (mm)	-16	0	16.2	18	17	18	15
Ampl. of S (mm)	-3	0	1	0.6	1	2.2	1.8
Ampl. of T (mm)	-0.8	0	1	0.1	0.8	1.5	1
P-QR int. (sec)	0.19	0	0.05	0.12	0.120	0.106	0.108
QRS int. (sec)	0.030	0	0.04	0.04	0.060	0.072	0.072
Work (sec)	0.36	0	0.35	0.33	0.340	0.440	0.340
Rest (sec)	0.68	0	0.88	0.720	0.950	1.136	0.904
Rest/work	1.61	0	2.45	2.18	2.71	2.58	2.65
Heart Rate	54	44	49	53	47	43	41

Case Two - Fred Fozzard (Wrestler) - Age 21

Amplitudes

He had a small change in P amplitudes with the maximum change being .3 mm in both Leads II and III. The R amplitudes increased the greatest in Leads II and III, 1.2 mm and 1 mm respectively. In Lead II an increase of 1 mm was the greatest change in the S amplitudes. The T wave was no exception as the greatest change that occurred was only 1.4 in Lead II.

Time Intervals

His rest/work ratio was the only time interval that showed any change, and that was a reduction of 1.36 in Lead III.

Heart Rate

His heart rate did fall within the bradycardia range, and he showed no significant changes in his heart rate as the beginning heart rate average was 43 beats/minute and his final heart rate average was 44 beats/minute.

QRS Axis

The subject showed an increase of two degrees in his axis from 86 degrees to 88 degrees.

TABLE II

RAW DATA, CASE 2

	Date					
	10-25-66	11-10-66	12-1-66	12-20-66	2-2-67	3-16-67
LEAD I						
Ampl. of P (mm)	0.5	0.5	0.2	0.2	0.6	0.6
Ampl. of R (mm)	2	2.8	3	2.2	2.9	2.2
Ampl. of S (mm)	-1	-1.2	-1.1	-1.9	-0.7	-1.4
Ampl. of T (mm)	3	3	2.9	2.8	3.6	3.3
P-QR int. (sec)	0.12	0.12	0.13	0.12	0.136	0.136
QRS int. (sec)	0.04	0.055	0.04	0.04	0.048	0.036
Work (sec)	0.35	0.455	0.40	0.36	0.432	0.376
Rest (sec)	0.85	0.81	1.04	0.69	0.980	1.016
Rest/work	2.42	1.80	2.60	1.91	2.03	2.70
Heart Rate	46	52	40	55	43	42
LEAD II						
Ampl. of P (mm)	1	1.5	0.8	0.8	0.6	0.7
Ampl. of R (mm)	10	12.2	10.9	12.9	10.8	11.2
Ampl. of S (mm)	1	0	0.9	1.6	1.2	2
Ampl. of T (mm)	3.5	3.5	4.5	2.8	4.2	4.9
P-QR int. (sec)	0.12	0.16	0.12	0.14	0.144	0.172
QRS int. (sec)	0.04	0.05	0.05	0.04	0.048	0.088
Work (sec)	0.36	0.34	0.40	0.33	0.426	0.448
Rest (sec)	0.88	0.78	1.05	0.69	1.144	1.04
Rest/work	2.44	2.29	2.62	2.09	2.67	2.32
Heart Rate	45	55	40	55	43	44

TABLE II (Continued)

	Date					
	10-25-66	11-10-66	12-1-66	12-20-66	2-2-67	3-16-67
LEAD III						
Ampl. of P (mm)	0.8	0.5	0.5	0.8	0.6	0.5
Ampl. of R (mm)	9	9.2	9	11.9	9.6	10
Ampl. of S (mm)	2	1.3	1.8	1.8	1.5	2.2
Ampl. of T (mm)	0.9	1	1.0	1.1	1.6	1.4
P-QR int. (sec)	0.16	0.15	0.12	0.14	0.156	0.172
QRS int. (sec)	0.04	0.04	0.04	0.05	0.076	0.044
Work (sec)	0.34	0.34	0.34	0.32	0.464	0.428
Rest (sec)	0.86	0.80	1.04	0.72	1.04	0.800
Rest/work	2.52	2.35	3.05	2.25	2.24	1.16
Heart Rate	39	51	41	45	40	45

Case Three - Ellison Beasley (Wrestler) - Age 20

Amplitudes

The subject showed a change in P amplitudes, with a decrease of 1.1 mm in amplitude height in Lead III. He showed important alterations in the R amplitudes of Leads II and III, where there was a decrease of 4.7 mm and 3.2 mm respectively. An increase of 1.2 mm in the S amplitudes was demonstrated in Leads II and III. His T amplitude decreased 1.6 mm in Lead II, and 1.7 mm in Lead III.

Time Intervals

He had no important alterations in any of the time elements or ratios.

Heart Rate

The subject had a significant change in heart rate as his heart rate decreased 11 beats/minute from his beginning average heart rate, 72 beats/minute, to his final average heart rate of 61 beats/minuts. His final heart rate fell above the bradycardia range.

QRS Axis

His beginning QRS axis was 83 degrees and the final QRS axis was 82 degrees.

TABLE III

RAW DATA, CASE 3

	Date					
	10-13-66	11-1-66	11-15-66	12-20-66	2-2-67	3-18-67
LEAD I						
Ampl. of P (mm)	0.1	0.1	0.8	0.1	0.5	0.5
Ampl. of R (mm)	3	3.6	5	3.2	3.3	2.2
Ampl. of S (mm)	-0.8	-0.5	-0.9	-1	-0.7	-0.4
Ampl. of T (mm)	2.2	2.2	3.5	2.5	2.4	2.1
P-QR int. (sec)	0.08	0.12	0.17	0.14	0.112	0.160
QRS int. (sec)	0.04	0.05	0.04	0.04	0.052	0.048
Work (sec)	0.31	0.32	0.28	0.30	0.308	0.360
Rest (sec)	0.48	0.60	0.58	0.49	0.612	0.544
Rest/work	1.54	1.87	1.85	1.63	1.98	1.51
Heart Rate	70	63	70	76	63	62
LEAD II						
Ampl. of P (mm)	1.2	0.6	0.6	0.9	0.7	0.1
Ampl. of R (mm)	15.8	18	17.5	18.7	14.9	11.1
Ampl. of S (mm)	0	0	0	0	0.8	1.2
Ampl. of T (mm)	4.3	4	4.6	5	5.7	2.7
P-QR int. (sec)	0.16	0.17	0.20	0.18	0.184	0.164
QRS int. (sec)	0.04	0.05	0.05	0.04	0.072	0.028
Work (sec)	0.31	0.32	0.28	0.29	0.242	0.340
Rest (sec)	0.50	0.56	0.62	0.44	0.664	0.632
Rest/work	1.61	1.75	2.21	1.51	2.78	1.85
Heart Rate	72	71	68	81	63	60

TABLE III (Continued)

	Date					
	10-13-66	11-1-66	11-15-66	12-20-66	2-2-67	3-18-67
LEAD III						
Ampl. of P (mm)	1.1	0.2	0.4	0.1	0.5	0
Ampl. of R (mm)	12.5	14.8	13	14.6	11.1	9.3
Ampl. of S (mm)	0	0	1	0.5	1.3	1.2
Ampl. of T (mm)	2.7	1.2	2.1	1.8	3	1
P-QR int. (sec)	0.16	0.17	0.20	0.18	0.176	0.160
QRS int. (sec)	0.04	0.05	0.06	0.04	0.072	0.028
Work (sec)	0.28	0.28	0.28	0.28	0.252	0.368
Rest (sec)	0.52	0.48	0.55	0.45	0.644	0.600
Rest/work	1.85	1.71	1.96	1.60	2.59	1.62
Heart Rate	74	60	71	68	61	60

Case Four - Richard Henjyoji (Wrestler) - Age 20

Amplitudes

He had no important alterations in the P amplitude heights. He showed significant changes in his R amplitudes, with reductions of 1.3 mm, 4.4 mm, and 4.1 mm in Leads I, II, and III, respectively. All his S amplitudes increased with Lead I increasing 1.5 mm and Leads II and III increasing 2 mm and 1.4 mm. The only T amplitude that changed to any degree of importance was the decrease in Lead II of 1.6 mm.

Time Intervals

This subject had no significant changes in any of the elements of time.

Heart Rate

No significant alterations occurred in his heart rate. His beginning and final heart rate was just slightly above the bradycardia range, as his average heart rate remained at 61 beats/minute.

QRS Axis

His beginning QRS axis was 71 degrees and the final QRS axis was 68 degrees, thus giving a difference of three degrees.

TABLE IV

RAW DATA, CASE 4

	Date					
	10-18-66	11-10-66	12-1-66	12-20-66	2-16-67	3-18-67
LEAD I						
Ampl. of P (mm)	0.5	0.8	0.2	0.5	0.1	0.8
Ampl. of R (mm)	5.8	5.9	5.7	7.1	5.4	4.5
Ampl. of S (mm)	-2.2	-0.8	-1.5	-1.4	-1.3	-0.7
Ampl. of T (mm)	1.8	2.1	1.8	1.7	2.1	1.5
P-QR int. (sec)	0.12	0.17	0.12	0.14	0.132	0.160
QRS int. (sec)	0.03	0.03	0.04	0.04	0.032	0.04
Work (sec)	0.33	0.28	0.37	0.32	0.368	0.376
Rest (sec)	0.60	0.68	0.80	0.48	0.588	0.584
Rest/work	1.81	2.42	2.16	1.5	1.6	1.55
Heart Rate	62	65	52	73	57	61
LEAD II						
Ampl. of P (mm)	0.5	1	0.3	0.9	0.2	0
Ampl. of R (mm)	14.2	15.3	13	15.8	11.2	9.8
Ampl. of S (mm)	0.5	0.5	0.5	0.3	1	2.5
Ampl. of T (mm)	3	2.3	3.8	2.8	4.3	1.4
P-QR int. (sec)	0.17	0.20	0.16	0.16	0.168	0
QRS int. (sec)	0.07	0.08	0.09	0.08	0.068	0.044
Work (sec)	0.36	0.30	0.47	0.29	0.316	0.408
Rest (sec)	0.62	0.61	0.73	0.49	0.692	0.720
Rest/work	1.72	2.33	1.55	1.68	2.18	1.76
Heart Rate	61	65	48	75	56	61

TABLE IV (Continued)

	Date					
	10-18-66	11-10-66	12-1-66	12-20-66	2-16-67	3-18-67
LEAD III						
Ampl. of P (mm)	0.3	0.5	0.2	0.5	0.1	0.8
Ampl. of R (mm)	9	10.1	9	8.3	6.2	4.9
Ampl. of S (mm)	0	0	0	0	0.7	1.4
Ampl. of T (mm)	0.6	0.5	1.6	1	1	0.8
P-QR int. (sec)	0.20	0.20	0.17	0.18	0.180	0.156
QRS int. (sec)	0.09	0.07	0.09	0.05	0.088	0.052
Work (sec)	0.28	0.28	0.44	0.27	0.360	0.320
Rest (sec)	0.66	0.62	0.64	0.49	0.632	0.668
Rest/work	2.35	2.21	1.45	1.81	1.75	2.15
Heart Rate	61	65	55	75	56	61

Case Five - Ronald Gabbett (Wrestler) - Age 21

Amplitudes

He showed no significant changes in the P amplitudes. An important increase in the R amplitudes was demonstrated in Leads II and II, which amounted to 2.9 mm and 2 mm respectively. He had no important changes in his S amplitudes. The subject showed an increase of 1.6 mm and 1.5 mm in Leads II and III.

Time Intervals

He had no important changes in any of the elements of time.

Heart Rate

The subject had an increased average heart rate of three beats/minute as his beginning heart rate average was 48 beats/minute and his final heart rate average was 51 beats/minute. His heart rate fell within the bradycardia classification.

QRS Axis

His beginning QRS axis was 53 degrees and the final axis was 60 degrees, thus giving an increase of seven degrees.

TABLE V

RAW DATA, CASE 5

	Date			
	10-18-66	11-4-66	12-1-66	2-9-67
LEAD I				
Ampl. of P (mm)	.8	.8	1.	1.1
Ampl. of R (mm)	9.9	10.2	10.9	9.9
Ampl. of S (mm)	-1.8	-1.5	-2.	-1.8
Ampl. of T (mm)	1.1	1.1	1.2	1.7
P-QR int. (sec)	.13	.15	.14	.108
QRS int. (sec)	.05	.06	.05	.072
Work (sec)	.32	.33	.35	.400
Rest (sec)	.87	.54	.82	.932
Work/Rest	2.75	1.65	2.34	2.33
Heart Rate	48	49	50	55
LEAD II				
Ampl. of P (mm)	.9	0.5	0.9	1.2
Ampl. of R (mm)	14.	16.	18.	16.9
Ampl. of S (mm)	-2.	-1.5	-2.5	-1.6
Ampl. of T (mm)	2.	2.1	3	3.6
P-QR int. (sec)	.12	.12	.14	.132
QRS int. (sec)	.08	.06	.08	.080
Work (sec)	.32	.33	.36	.368
Rest (sec)	.84	.81	.71	.816
Rest/Work	2.62	2.45	1.95	2.21
Heart Rate	49	49	55	53
LEAD III				
Ampl. of P (mm)	.3	-.8	-0.6	.4
Ampl. of R (mm)	5.	6.4	8.2	7.
Ampl. of S (mm)	.3	0.2	0	.8
Ampl. of T (mm)	.4	.5	1	1.9
P-QR int. (sec)	.120	.148	.112	.112
QRS int. (sec)	.04	.04	.05	.048
Work (sec)	.29	.32	.36	.332
Rest (sec)	.73	.89	.60	.892
Rest/Work	2.51	2.77	1.66	2.68
Heart Rate	48	50	64	45

Case Six - David Johnson - Age 20

Amplitudes

He showed a reduction in all the P amplitudes with Lead I being reduced by .4 mm, and Leads II and III being reduced by 1.1 mm and .2 mm. His R amplitude was increased by 1.1 mm in Lead I, and decreased by 1.8 mm in both Leads II and III. The subject's S amplitude showed no important alterations. Al of his T amplitudes were reduced, with the greatest reduction being 1 mm in Lead II.

Time Intervals

No significant changes were exhibited in any of the elements of time.

Heart Rate

He had an increase of four beats/minute in his average heart rate, as his average beginning heart rate was 54 beats/minute and his average final heart rate was 58 beats/minute. The subject's heart rate fell within the bradycardia classification.

QRS Axis

The subjects beginning QRS axis was 81 degrees and his final axis was 76 degrees, thus giving a reduction of five degrees.

TABLE VI

RAW DATA, CASE 6

	Date		
	11-11-66	12-2-66	2-24-67
LEAD I			
Ampl. of P (mm)	.9	.3	.5
Ampl. of R (mm)	3.6	3	4.7
Ampl. of S (mm)	-.9	-.9	-.8
Ampl. of T (mm)	1.7	.8	1.1
P-QR int. (sec)	.128	.120	.112
QRS int. (sec)	.048	.060	.032
Work (sec)	.348	.380	.332
Rest (sec)	.822	.672	.692
Rest/Work	2.36	1.76	2.08
Heart Rate	51	56	55
LEAD II			
Ampl. of P (mm)	1.3	.4	.2
Ampl. of R (mm)	18.6	18.2	16.8
Ampl. of S (mm)	.8	.8	0
Ampl. of T (mm)	4.	4.8	3.
P-QR int. (sec)	.136	.140	.092
QRS int. (sec)	.052	.052	.036
Work (sec)	.448	.372	.336
Rest (sec)	.588	.680	.588
Rest/Work	1.31	1.82	1.75
Heart Rate	58	48	61
LEAD III			
Ampl. of P (mm)	.3	.1	.1
Ampl. of R (mm)	13.7	8.2	11.9
Ampl. of S (mm)	.4	.5	.6
Ampl. of T (mm)	3.	3.8	2.1
P-QR int. (sec)	.128	.160	.128
QRS int. (sec)	.068	.044	.063
Work (sec)	.376	.376	.396
Rest (sec)	.688	.766	.584
Rest/Work	1.88	2.03	1.47
Heart Rate	54	51	59

Case Seven - George Phillips (Swimmer) - Age 20

Amplitudes

The subject showed no significant changes in either the P or R amplitude heights. A reduction of .8 mm in Lead II was the only notable change that occurred in the amplitude of the S wave. He had a reduction of 1.3 mm in height of the T amplitude in Lead I.

Time Interval

A decrease of 1.19 in the rest/work ratio was the only important time alteration that he had, and it took place in Lead III.

Heart Rate

His heart rate falls within the bradycardia range, and he showed an increase of six beats/minute from his beginning average heart rate, 49 beats/minute, to his final average heart rate, 55 beats/minute.

QRS Axis

The subject's beginning QRS axis was 58 degrees and the final QRS axis was 57 degrees.

TABLE VII

RAW DATA, CASE 7

	Date					
	11-3-66	11-17-66	12-1-66	12-20-66	1-13-67	2-27-67
LEAD I						
Ampl. of P (mm)	.8	.6	.5	.7	.5	.5
Ampl. of R (mm)	4.8	3.7	3.9	4.4	4.	4.3
Ampl. of S (mm)	-1.1	-.9	-1.2	-1.2	-1.	-.7
Ampl. of T (mm)	2.7	1.5	2.1	2.	1.3	1.4
P-QR int. (sec)	.116	.112	.152	.168	.152	.132
QRS int. (sec)	.060	.044	.052	.076	.068	.052
Work (sec)	.356	.308	.328	.260	.340	.364
Rest (sec)	.800	.540	.612	.692	.464	.620
Rest/work	2.24	1.75	1.86	2.66	1.36	1.7
Heart Rate	50	62	58	55	71	52
LEAD II						
Ampl. of P (mm)	.7	.5	.3	.5	.5	.3
Ampl. of R (mm)	8.2	9.	8.8	8.7	9.6	8.3
Ampl. of S (mm)	-2.6	-2.3	-2.1	-2.6	-1.8	-1.8
Ampl. of T (mm)	1.4	1.5	1.7	1.	1.4	.8
P-QR int. (sec)	.136	.156	.156	.120	.152	.116
QRS int. (sec)	.060	.080	.064	.072	.064	.068
Work (sec)	.352	.300	.320	.308	.320	.372
Rest (sec)	.708	.600	.708	.680	.624	.660
Rest/work	2.01	2.2	2.27	2.2	1.94	1.8
Heart Rate	48	58	54	57	61	59

TABLE VII (Continued)

	Date					
	11-3-66	11-17-66	12-1-66	12-20-66	1-13-67	2-27-67
LEAD III						
Ampl. of P (mm)	-.6	-.6	-.4	-.7	-.4	
Ampl. of R (mm)	5.	4.3	5.	5.5	4.4	4.8
Ampl. of S (mm)	-1.7	-1.7	-2.1	-2.	-1.5	-1.7
Ampl. of T (mm)	-1.	-.7	-0.4	-.5	-.7	0
P-QR int. (sec)	.128	.100	.120	.128	.132	.060
QRS int. (sec)	.072	.060	.084	.080	.072	0
Work (sec)	.292	.276	.284	.308	.312	0
Rest (sec)	.964	.728	.688	.608	.660	0
Rest/Work	3.30	2.63	2.42	2.2	2.11	0
Heart Rate	48	56	58	57	62	54

Case Eight - William Lemaster - Age 19

Amplitudes

This subject had very slight alterations in the P, R, S, and T amplitude heights in all three Leads.

Time Intervals

He had a decrease of .88 and 1.05 in his rest/work ratios in Leads I and II respectively.

Heart Rate

He showed no change from his original average heart rate of 52 beats/minute. His heart rate falls within the bradycardia range.

QRS Axis

The subject's beginning and final QRS axis were the same, 69 degrees.

TABLE VIII

RAW DATA, CASE 8

	Date				
	11-3-66	11-17-66	12-1-66	12-20-66	1-13-67
LEAD I					
Ampl. of P (mm)	.5	.7	.6	.6	.5
Ampl. of R (mm)	4.	4.2	5.5	5.7	4.2
Ampl. of S (mm)	-.5	0	-.5	-.3	-.6
Ampl. of T (mm)	1.7	2.1	1.9	1.5	1.
P-QR int. (sec)	.138	.160	.148	.144	.164
QRS int. (sec)	.036	.040	.036	.036	.040
Work (sec)	.312	.328	.296	.304	.364
Rest (sec)	.872	.580	.644	.820	.692
Rest/Work	2.78	1.76	2.7	2.66	1.9
Heart Rate	55	68	59	58	56
LEAD II					
Ampl. of P (mm)	.7	.9	1.5	1.7	1.5
Ampl. of R (mm)	11.7	10.3	10.5	13.4	11.8
Ampl. of S (mm)	-1.5	-.8	-1.8	-1.7	-2.2
Ampl. of T (mm)	2.3	2.3	2.5	2.4	2.3
P-QR int. (sec)	.156	.124	.188	.180	.192
QRS int. (sec)	.060	.056	.064	.064	.064
Work (sec)	.352	.300	.344	.288	.352
Rest (sec)	.804	.688	.768	.780	.836
Rest/Work	2.28	2.27	2.23	2.7	2.34
Heart Rate	53	54	56	54	49

TABLE VIII (Continued)

	Date				
	11-3-66	11-17-66	12-1-66	12-20-66	1-13-67
LEAD III					
Ampl. of P (mm)	.9	.9	1.1	1.3	1.1
Ampl. of R (mm)	7.3	5.5	5.5	7.6	7.8
Ampl. of S (mm)	-.7	-.9	-.7	-1.1	-1.2
Ampl. of T (mm)	1.	1.1	1.2	1.5	.7
P-QR int. (sec)	.136	.160	.180	.172	.164
QRS int. (sec)	.056	.056	.056	.068	.048
Work (sec)	.376	.316	.368	.272	.268
Rest (sec)	.948	.880	.800	.856	.960
Rest/Work	2.51	2.81	2.18	3.14	3.56
Heart Rate	48	55	62	53	52

Case Nine - James Denney (Swimmer) - Age 20

Amplitudes

The subject showed slight alterations in the amplitude height of the P waves. His only important change in the height of the R amplitudes occurred in Lead I, which amounted to a decrease of 1.3 mm. The height of his S amplitudes showed more important alterations with Leads II being decreased by 1.1 mm, and Lead III being increased by 3.4 mm. In Lead I he showed an increase of 1.2 mm in the T amplitude, while the remaining two Leads showed only slight changes.

Time Intervals

He showed no important changes in any of the time components.

Heart Rate

His average heart rate decreased seven beats/minute to the final heart rate average of 59 beats/minute. His heart rate fell within the bradycardia range.

QRS Axis

He had an increase of 34 degrees in the QRS axis, as his beginning axis was 19 degrees and the final axis was 53 degrees.

TABLE IX

RAW DATA, CASE 9

	Date					
	11-4-66	11-17-66	12-1-66	12-20-66	1-13-67	2-27-67
LEAD I						
Ampl. of P (mm)	1.	1.	1.1	.9	.6	.7
Ampl. of R (mm)	6.6	4.9	4.8	5.8	4.7	3.3
Ampl. of S (mm)	-1.3	-.9	-1.5	-1.4	-1.	-.7
Ampl. of T (mm)	3.5	4.6	4.4	4.	4.7	4.7
P-QR int. (sec)	.160	.160	.160	.144	.148	.152
QRS int. (sec)	.060	.060	.028	.040	.052	.040
Work (sec)	.352	.322	.384	.320	.356	.484
Rest (sec)	.576	.542	.668	.584	.656	.544
Rest/Work	1.63	1.62	1.73	1.82	1.84	1.21
Heart Rate	65	75	60	69	55	60
LEAD II						
Ampl. of P (mm)	1.2	1.2	1.2	.9	.8	.8
Ampl. of R (mm)	9.2	8.6	9.3	10.1	8.	8.7
Ampl. of S (mm)	-1.3	-.9	-1.2	-.8	-.9	-.2
Ampl. of T (mm)	2.5	2.1	2.5	1.9	3.1	2.8
P-QR int. (sec)	.168	.172	.164	.124	.148	.140
QRS int. (sec)	.072	.056	.056	.056	.056	.060
Work (sec)	.312	.292	.360	.308	.336	.372
Rest (sec)	.572	.580	.624	.708	.632	.608
Rest/Work	1.83	1.95	1.73	2.03	1.88	1.63
Heart Rate	67	66	60	58	61	56

TABLE IX (Continued

	Date					
	11-4-66	11-17-66	12-1-66	12-20-66	1-13-67	2-27-67
LEAD III						
Ampl. of P (mm)	.7	.6	.3	.4	.5	.5
Ampl. of R (mm)	3.	3.8	3.9	4.	3.6	3.5
Ampl. of S (mm)	-4.	-3.4	-3.	-3.5	-.3	-.6
Ampl. of T (mm)	-1.4	-1.1	-1.5	1.6	-1.3	-.9
P-QR int. (sec)	.148	.148	.124	.124	.164	.132
QRS int. (sec)	.032	.028	.024	.032	.024	.036
Work (sec)	.268	.316	.252	.304	.295	.304
Rest (sec)	.660	.632	.652	.708	.628	.760
Rest/Work	2.46	2.	2.54	2.03	2.15	2.5
Heart Rate	66	66	61	59	61	62

Case Ten - William Keene (Swimmer) - Age 20

Amplitudes

The subject showed very small changes in the amplitude heights of the P waves, with a decrease of .9 mm in Lead II being the greatest change. His R amplitude heights were reduced by 2.6 mm, 1.7 mm, and .7 mm in Leads I, II, and III, respectively. There were absolutely no changes in his S amplitudes. His T amplitude heights were decreased by 1.4 mm in Lead I, and increased by 1.2 mm in Lead III.

Time Intervals

He had no important alterations in any of the time elements recorded.

Heart Rate

His average heart rate decreased three beats/minute to the final heart rate average of 68 beats/minute. His heart rate fell above the bradycardia range.

QRS Axis

The subject's beginning QRS axis was 66 degrees and the final axis was 76 degrees, thus he exhibited an increase of ten degrees.

TABLE X

RAW DATA, CASE 10

	Date					
	11-17-66	12-1-66	12-20-66	1-13-67	2-17-67	2-28-67
LEAD I						
Ampl. of P (mm)	.4	.3	.5	.3	.1	.1
Ampl. of R (mm)	5.3	4.2	5.3	6.1	4.7	2.7
Ampl. of S (mm)	0	.4	.5	-.2	.1	0
Ampl. of T (mm)	2.4	2	.1	2.3	1.9	1.
P-QR int. (sec)	.104	.100	.120	.112	.064	.048
QRS int. (sec)	.024	.024	.048	.032	.028	.028
Work (sec)	.280	.300	.288	.300	.284	.304
Rest (sec)	.512	.548	.632	.516	.576	.580
Rest/Work	1.82	1.82	2.19	1.72	2.03	1.9
Heart Rate	72	68	62	70	73	65
LEAD II						
Ampl. of P (mm)	1.4	.3	1.4	1.4	0	.5
Ampl. of R (mm)	13.8	8.4	13	14.9	12.9	12.1
Ampl. of S (mm)	0	0	.3	-.2	.2	0
Ampl. of T (mm)	1.7	2.9	1.4	2.9	2.2	2.9
P-QR int. (sec)	.112	.092	.104	.092	.068	.112
QRS int. (sec)	.032	.032	.040	.032	.044	.036
Work (sec)	.280	.300	.308	.300	.320	.336
Rest (sec)	.552	.580	.548	.536	.540	.484
Rest/Work	1.97	1.93	1.8	1.78	1.68	1.44
Heart Rate	72	68	63	68	61	67

TABLE X (Continued)

	Date					
	11-17-66	12-1-66	12-20-66	1-13-67	2-17-67	2-28-67
LEAD III						
Ampl. of P (mm)	.9	-.3	.5	1.1	0	.4
Ampl. of R (mm)	7.9	9	7.4	8.1	6.9	7.2
Ampl. of S (mm)	0	-.1	0	-.1	1.2	0
Ampl. of T (mm)	.5	1.1	.3	.5	0	.5
P-QR int. (sec)	.116	.104	.112	.112	.092	.116
QRS int. (sec)	.048	.028	.036	.044	.032	.040
Work (sec)	.268	.280	.312	.320	.300	.292
Rest (sec)	.500	.600	.676	.508	.544	.520
Rest/Work	1.86	2.13	2.16	1.9	1.76	1.78
Heart Rate	69	74	69	70	70	73

Case Eleven - Stephen Autry (Swimmer) - Age 18

Amplitudes

He demonstrated relatively little change in the height of his P amplitudes, however, in Lead III the amplitude increased 1.3 mm. The subject showed a decrease in R amplitude of 3 mm and 1.3 mm in Leads II and III, respectively. Only one notable change occurred in his T amplitudes and that was a decrease of 1.3 mm in Lead III. All of his S amplitudes increased, but none notably.

Time Intervals

The subject had no important changes in any of the time elements.

Heart Rate

His final average heart rate falls within the bradycardia range, as he had an increase of ten beats/minute from his beginning average heart rate, 53 beats/minute, to his final average heart rate, 63 beats/minute.

QRS Axis

His beginning and final QRS axis remained the same 80 degrees.

TABLE XI

RAW DATA, CASE 11

	Date				
	11-17-66	12-1-66	12-20-66	1-28-67	2-15-67
LEAD I					
Ampl. of P (mm)	.7	.5	.5	1.2	.9
Ampl. of R (mm)	3.6	3.9	3.5	4.	3.6
Ampl. of S (mm)	-1.2	-1.7	-1.	-1.5	-1.3
Ampl. of T (mm)	2.	2.5	2.1	1.7	2.3
P-QR int. (sec)	.120	.108	.108	.116	.112
QRS int. (sec)	.032	.032	.052	.052	.056
Work (sec)	.340	.404	.372	.484	.332
Rest (sec)	.760	.808	.656	.756	.600
Rest/Work	2.23	2.	1.76	1.56	1.81
Heart Rate	53	50	66	47	62
LEAD II					
Ampl. of P (mm)	.9	.9	.7	1.4	1.5
Ampl. of R (mm)	14.3	14.1	14.5	13.9	10.3
Ampl. of S (mm)	.5	.4	.5	0	.9
Ampl. of T (mm)	1.8	2.3	1.7	2.5	2.5
P-QR int. (sec)	.096	.116	.128	.120	.128
QRS int. (sec)	.040	.040	.060	.056	.040
Work (sec)	.364	.488	.488	.436	.332
Rest (sec)	.748	.644	.508	.584	.632
Rest/Work	2.05	1.30	1.16	1.34	1.90
Heart Rate	53	51	58	58	61

TABLE XI (Continued)

	Date				
	11-17-66	12-1-66	12-20-66	1-28-67	2-15-67
LEAD III					
Ampl. of P (mm)	-.3	-.4	-.4	.4	1.
Ampl. of R (mm)	10.8	10.5	12.2	12.3	9.5
Ampl. of S (mm)	1.	.6	.5	1.	1.7
Ampl. of T (mm)	-.5	-.4	-.6	-.8	-.5
P-QR int. (sec)	.108	.104	.112	.112	.136
QRS int. (sec)	.024	.052	.068	.036	.072
Work (sec)	.304	.324	.312	.372	.272
Rest (sec)	.720	.704	.692	.932	.600
Rest/Work	2.39	2.17	2.18	2.5	2.2
Heart Rate	54	53	58	50	65

Case Twelve - Jake Cunningham (Swimmer) - Age 19

Amplitudes

The subject demonstrated important decreases in the amplitude heights of the P waves in Leads II and III, which were reduced by 1.7 mm and 1.2 mm respectively. His R amplitudes were reduced notably in both Leads II and III by 2.2 mm. No important changes occurred in his S wave amplitude. He showed important reductions in the T amplitudes of Leads II and III, which amounted to 2.6 mm and 1.8 mm, respectively.

Time Intervals

He had a notable decrease in the rest/work ratio of .91 in Lead I.

Heart Rate

The subject's heart rate remained above the bradycardia range, and his average heart rate increased five beats/minute to the final average heart rate of 69 beats/minute.

QRS Axis

His beginning QRS axis was 77 degrees, and his final axis was 75 degrees.

TABLE XII

RAW DATA, CASE 12

	Date		
	11-11-66	12-2-66	2-27-67
LEAD I			
Ampl. of P (mm)	.9	.8	.4
Ampl. of R (mm)	2.4	3.2	2.9
Ampl. of S (mm)	0	0	-.6
Ampl. of T (mm)	3.6	3	2.8
P-QR int. (sec)	.172	.172	.124
QRS int. (sec)	.052	.040	.052
Work (sec)	.312	.320	.356
Rest (sec)	.720	.604	.496
Rest/Work	2.30	1.88	1.39
Heart Rate	66	68	72
LEAD II			
Ampl. of P (mm)	1.3	.9	-.4
Ampl. of R (mm)	11.3	11.8	9.1
Ampl. of S (mm)	-1.2	-1.	-1.4
Ampl. of T (mm)	5.3	5.1	2.7
P-QR int. (sec)	.148	.164	.132
QRS int. (sec)	.040	.064	.052
Work (sec)	.320	.324	.346
Rest (sec)	.564	.580	.492
Rest/Work	1.76	1.79	1.41
Heart Rate	65	65	68
LEAD III			
Ampl. of P (mm)	1.	1.	-.2
Ampl. of R (mm)	8.5	8.1	6.3
Ampl. of S (mm)	-.7	-.9	0
Ampl. of T (mm)	1.8	1.2	0
P-QR int. (sec)	.184	.180	.116
QRS int. (sec)	.054	.068	.024
Work (sec)	.340	.348	.292
Rest (sec)	.692	.580	.652
Rest/Work	2.03	1.66	2.26
Heart Rate	62	65	68

CHAPTER V

DISCUSSION OF THE RESULTS

Type of Analysis

The data in this study was not given any statistical analysis, because one of the subjects exhibited changes which were sufficient enough to make the small group appear as though something statistically significant had occurred. Instead the alterations brought about in the resting electrocardiogram recording, as a result of wrestling or swimming, were presented in the form of group changes for each of the areas of interest, and then a comparison of the beginning and final means of the same areas was made.

Amplitude Results

In the group of wrestlers only one subject showed a prominent change in P amplitudes, and this was a decrease of 1.1 mm in Lead III. There was no common trend in the group's R amplitudes, because one subject increased in an amplitude height as much as 4.4 mm, while another subject decreased in amplitude height as much as 4.7 mm. Two of the members of the group showed only slight variation in R amplitude. One subject expressed an uncommon change in the entire QRS complex in both Lead I and III. In Lead III his amplitudes changed from inverted to upright, and in Lead I the amplitudes were drastically reduced. The increase in his R amplitude in Lead III was 31 mm, and the decrease in

Lead I was 15.6 mm. Only one of the wrestlers did not show an increase in the height of the S amplitudes. The group's greatest increase was 2 mm, however the exceptional subject showed an increase of 4.8 mm in Lead III. There was no common expression of the T amplitudes within the wrestling group, as two of the subjects showed decreases, which were off-set by the remaining subjects exhibiting increases in amplitude height.

The group of swimmers showed no common expression in the P amplitudes. Most of the changes were slight, however one subject did increase 1.3 mm while another decreased 1.7 mm in amplitude height. Only slight changes were observed in the R amplitudes, however there was a tendency for the amplitudes to be reduced in height. One of the swimmers showed a reduction as great as 3 mm in the height of his R amplitude. In the S amplitudes a sundry of results were exhibited, which ranged from increases to no change to decreases in the amplitudes. Three of the seven swimmers exhibited a trend of having inverted T wave amplitudes in Lead III. Any other common expression of the T amplitudes did not exist as the results were diverse.

In considering the results of all the ECG records it would be difficult to say on the basis of these results that the athletic training programs had any common effect upon the amplitudes of these athletes' resting ECG records. It would appear that the cause of higher wave amplitudes is due to either inheritance or is a product of previous athletic training because there was no common expression in this study to suggest that athletic training causes an increase in the wave amplitudes.

High amplitudes in the ECG records are known to be a common manifestation of hypertrophy of the heart. In experimental animals an increased level of physical activity is known to produce hypertrophy, however the results of this study did not indicate such findings due to the inconsistent changes in ECG amplitudes.

Time Interval Results

There were two wrestlers that showed definite changes in the rest/work ratio, whereas the remaining three wrestlers showed no significant alterations. One of these subjects showed an increase in ratio value of 1.04 in Lead III and .94 in Lead II, whereas the other subject decreased in ratio value of 1.36 in Lead III.

There were three swimmers that showed definite changes in the rest/work ratio, while the remaining four subjects exhibited no important alterations. One of the subjects showed a reduction in ratio value of 1.19 in Lead III while another subject exhibited a reduced ratio value of .91 in Lead I. The last subject showed a combination of changes, as he had an increase in ratio value of 1.05 in Lead III and a decrease in ratio value of .88 in Lead I.

No other elements of time in the ECG records exhibited important changes in either group.

Heart Rate Results

Most of the subjects in this study revealed sinus arrhythmia in their ECG recordings. No consistent trend was exhibited by either group in their heart rates other than that seven of the subjects fell within the bradycardia range. Only one of these subjects, a swimmer, exhibited

the characteristics of an induced bradycardia. The remaining six subjects that fell within this classification were there because of either an inherited bradycardia or an induced bradycardia, which was the result of previous physical training or preseason training. The four subjects that remained above the bradycardia range demonstrated no common heart rate expression, either. These results can be seen in the following Table XIII.

QRS Axis Results

Only two subjects out of the twelve subjects showed any important QRS axis deviations. According to Sigler,[1] an axis deviation does not become important until it passes beyond a range of ten degrees, either plus or minus, of the original axis. One of the wrestlers increased 119 degrees from a left axis deviation into the normal axis range. The swimmer that showed an important axis deviation increased some 34 degrees to the right. Both of these subjects' increases were to the right but neither of them moved out of the normal axis range.

Sigler,[2] Kossman and others[3] feel that the physiological and anatomical reasons for deviations of the electrical axis to the right might be caused by variations of ventricular preponderance. On the basis of these reports it would seem reasonable to conclude that the wrestler must have increased a large amount in the muscle mass of his right

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[1]Sigler, p. 57.

[2]Ibid.

[3]C. E. Kossman et al., "An Analysis of Right Axis Deviation Based Partly on Endocardial Potentials of the Hypertrophied Right Ventricle," American Heart Journal, XXXV (February, 1948), pp. 309-335.

TABLE XIII

THE INDIVIDUAL AVERAGE HEART RATES FOR THE BEGINNING AND FINAL RECORDINGS

Case Number	Beginning	Final
1	54	42
2	43	44
3	72	61
4	61	61
5	48	51
6	54	58
7	49	55
8	52	52
9	66	59
10	71	68
11	53	63
12	64	69

ventricle to cause such a massive change in the QRS axis. As to the cause of this hypertrophy of the right ventricle, it can only be assumed that it was due to an increase in the diameter of the muscle fibers in the right ventricle. This increase in diameter does seem reasonable, due to the known effects of physical training upon various muscle fibers.

Comparison of Mean Scores

The mean P wave amplitudes, as seen in Table XIV, were lower than the mean P wave amplitudes of the 81 normal young men of Cureton's study.[4] Generally, there was a trend for the mean P wave amplitude to decrease in size from the beginning mean amplitude. According to Cureton,[5] low P amplitudes are associated with endurance in treadmill running.

The wrestlers' mean R wave amplitudes decreased in Lead I, and increased in Lead III. The reason for this is due to the drastic alterations made by the one subject in his entire QRS complex. Apparently this subject's alterations were so great in magnitude that it made the combined mean in Lead III appear to increase, as seen in Table IV. The group mean R wave amplitudes were all below those means of Cureton's normal young men.[6]

All of the mean S wave amplitudes were negative in Leads I and II, as seen in Table XV. There was a slight trend for them to reduce in

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[4]Cureton, Physical Fitness of Champion Athletes, p. 148.

[5]Thomas K. Cureton, "The Hearts of Athletes," Illinois Medical Journal, 99 (March, 1951), pp. 143-146.

[6]Cureton, Physical Fitness in Champion Athletes, p. 148.

TABLE XIV

THE MEAN SCORES OF THE P AND R AMPLITUDES

	Amplitude of P Wave						Amplitude of R Wave					
	Leads											
	I		II		III		I		II		III	
	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2
Group												
Wrestlers	.64	.74	.92	.56	.46	.14	7.74	4.24	11.44	11.36	3.9	9.24
Swimmers	.74	.5	1.1	1.7	.3	.2	4.3	4.0	12.4	11.0	8.6	7.3
Combined	.88	.60	.10	.12	.035	.027	5.75	4.07	12.02	11.15	6.64	8.1
Cureton's 81 Normal Young Men	.92		1.31		.96		5.3		3.94		9.35	

TABLE XV

THE MEAN SCORES OF THE S AND T AMPLITUDES

	Amplitude of S Wave						Amplitude of T Wave					
	Leads											
	I		II		III		I		II		III	
	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2
Group												
Wrestlers	-.8	-.6	.1	.1	.4	1.1	2.5	2.4	3.3	3.2	.6	1.3
Swimmers	-.07	-.5	-.9	-.9	.2	.3	2.2	2.0	2.7	1.8	.4	.2
Combined	-.7	-.54	-.48	-.13	.3	.6	2.31	2.2	2.95	2.4	.5	.7
Cureton's 81 Normal Young Men	-1.26		-1.10		-.75		2.78		4.2		1.87	

magnitude toward the positive. The negative amplitudes in the first two leads were less than the mean S wave amplitudes of Cureton's 81 normal young men, while the third Lead was positive and above the negative mean of the normal group.[7]

The mean T wave amplitudes in all three leads were below those of Cureton's normal young men,[8] as seen in Table XV. Of course, this is due to the fact that four of the subjects began the study with inverted T waves, and that three of these subjects continued to exhibit these characteristics to the very end of the study. There was not much change shown throughout the study, but there was a slight trend for the mean amplitudes to decrease.

The mean P-QR and QRS intervals remained relatively constant, as seen in Table XVI. The duration of the P-QR interval was less than the mean deviation of Cureton's normal young men.[9] According to Cureton,[10] short P-QR intervals are correlated with endurance.

The mean rest/work ratios showed only a slight decrease in Lead I, as seen in Table XVII. The groups mean rest/work ratios were less than the mean rest/work ratios of Cureton's 81 normal young men.[11]

The group's mean heart rate showed slight changes from the beginning mean heart rates. The swimmers' final mean heart rates were higher than the wrestlers' final mean heart rate, as seen in Table XVII. The

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[7]Ibid., p. 149.

[8]Ibid.

[9]Ibid., p. 148.

[10]Cureton, Illinois Medical Journal, pp. 143-146.

[11]Cureton, Physical Fitness of Champion Athletes, p. 149.

TABLE XVI

THE MEAN SCORES OF THE P-QR AND QRS INTERVALS

	P-QR Interval						QRS Interval					
	Leads											
	I		II		III		I		II		III	
	Beginning and Final Means											
	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2
Group												
Wrestlers	.139	.142	.138	.141	.166	.142	.040	.049	.054	.059	.049	.049
Swimmers	.134	.135	.136	.130	.135	.132	.045	.042	.066	.062	.069	.067
Combined	.136	.138	.137	.135	.148	.136	.043	.045	.061	.061	.060	.059
Cureton's 81 Normal Young Men	.176		.177		.175							

TABLE XVII

THE MEAN SCORES OF THE REST/WORK RATIO AND HEART RATE

	Rest/Work Ratio						Heart Rate					
	I		II		III		I		II		III	
	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2	x^1	x^2
Group												
Wrestlers	2.04	2.19	2.04	2.2	2.16	2.05	.55	.53	.49	.52	.55	.50
Swimmers	2.19	1.7	1.9	1.8	2.2	2.3	.59	.60	.59	.60	.56	.62
Combined	2.13	1.9	1.95	1.94	2.18	2.17	.57	.57	.55	.57	.56	.57
Cureton's 81 Normal Young Men	2.549		2.514		2.6		64.5		64.9		65.8	

group's combined mean heart rates were lower than Cureton's normal young men.[12]

[12]Ibid., p. 149.

CHAPTER VI

CONCLUSIONS

In this study no substantial tendency has been shown, that the athletic training of college wrestlers and swimmers does have an effect upon their resting electrocardiographic records.

The findings of this experiment justify the following conclusions in regard to these twelve subjects.

1. Generally, the resting electrocardiograms of these wrestlers and swimmers were not strikingly different from those reported in the literature for normal young men of the same age.

2. There was no evidence from this study to indicate that arduous training of the wrestlers and swimmers produced any significant increase or decrease in wave amplitudes.

3. Two of the subjects exhibited QRS axis deviation to the right, which was interpreted as right ventricular hypertrophy.

4. The mean heart rates showed relatively no change.

5. No significant changes in time elements or rest/work ratios were demonstrated by these athletes.

Recommendations for Further Study

Further study is needed on younger subjects as they are beginning their athletic careers, to determine if higher amplitudes are brought about by athletic training or inheritance.

Further study is needed on a larger number of athletes, taking only a beginning and final ECG record, who have not participated in any pre-season athletic or pre-season training to determine the effects of their athletic training on their resting ECG records.

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