AN ELECTROCARDIOGRAPH STUDY OF TWENTY CHAMPION SWIMMERS BEFORE AND AFTER ONE-HUNDRED AND TEN YARD SPRINT SWIMMING COMPETITION

by

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ABSTRACT

It was believed that a study of athletes under the stress of competition would present a somewhat different challenge than would the same tests held under laboratory conditions. The belief was that actual competition would produce a far greater emotional and physiological stress than could be produced in an artificial setting.

For this reason then, nineteen highly trained and healthy, teen-age swimmers of championship calibre were selected for study before and after sprint swimming competition. One swimmer was studied a second time, two years after the first testing, making a total of twenty sets of observations.

The subjects were studied before the exercise to determine their resting blood pressures and pulse rates and to record their resting electrocardiograms. The swimming races were, for the most part, held in regularly scheduled meets and were distances of one-hundred and ten yards. Four observations were made following official time trials of similar swimming events. The study was to be concerned with the changes and recovery of the electrocardiograms, pulse rates and blood pressures following the races.

In reviewing the literature, no similar study could be found that employed the use of serial electrocardiograms that would present a view of the recovery of the heart during a selected time interval immediately following actual competition. Therefore, this study concerned itself with a thirty minute recovery period beginning three minutes after the race. Each subject, then, had electrocardiograms taken at: three, six, nine, twelve, fifteen, twenty, twenty-five and thirty minute intervals following the "all-out" sprint. Blood pressure readings were also taken at regular intervals and the pulse rate was automatically recorded by the electrocardiograph.

The results of these measurements indicated individual variations in blood pressure and pulse rate. However, general trends were observed in the measurements of the P-R interval, the ST segment and the T wave. The P-R interval, in twelve of the twenty cases, was prolonged. ST depression was seen in eight cases. The T wave was lowered considerably in all of the cases, with T inversion in nine of the athletes. Eighteen of the twenty observations showed one or all of the following major variations: PR prolongation, T inversion, ST depression.

These three changes of the electrocardiogram, if of sufficient degree, are taken as evidence of cardiac disease according to medical tests. Several studies of athletes

observed after exercise have also pointed out that PR prolongation, T wave inversion and ST depression are to be taken as pathological variations.

In the present study, these variations were not present in the resting recordings of any of the subjects. These variations did appear, however, at different times throughout the thirty minute recovery period. The deflections of concern, for the most part, had returned to normal by the end of the test. This evidence might then suggest that such variation, in healthy, young athletes, appearing after strenuous competition, would be indicative of functional adjustment of the myocardium to this situation of physical and emotional stress.

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CHAPTER I

STATEMENT OF THE PROBLEM

The problem suggested itself following a review of the literature (1, 2, 3, 4, 5) concerned with the effect of athletic activity on the heart. The literature contained a great deal of data on athletes studied in many ways.

Some researchers employed the use of the electrocardiogram to study the recovery of the athlete's heart following strenuous exertion (6, 7, 8, 9). However, it was seen that most of the studies were conducted under laboratory conditions (10, 11, 12) and were therefore not indicative of the true strain of competition. Other research (13, 14, 15, 16) did measure the stress of actual competition. However, the time intervals of the graph readings during these reports were either inconsistently spaced or were not taken continuously (17, 18), being limited to but one recovery reading (19, 20),

The problem is to record data as to the recovery of the blood pressures, heart rates and electrocardiograms of champion athletes following actual competition. There is difficulty in gaining facts descriptive of the cardiac recovery during the time interval from immediately after a race until such a time when the electrocardiogram approaches resting levels.

The purpose of this report, therefore, is to undertake a study of nineteen healthy, young athletes, of championship calibre, following actual competition in one-hundred metre swimming races. One subject was studied a second time, two years after his first reading, making twenty sets of observations in all. Electrocardiograms, pulse rates and blood pressures are to be taken at rest and recorded serially from three minutes to thirty minutes following the completion of the one-hundred metre sprint swim.

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CHAPTER II

JUSTIFICATION OF THE PROBLEM

Although there was no record of a serial electrocardiograph study begun immediately after performance in actual competition, many related papers were reviewed (1, 2, 3, 4, 5). These publications were compared with the results of a pilot electrocardiograph study of four champion swimmers before and after competition[1] and were found to be in disagreement.

According to some (6, 7, 8, 9, 10), ST depression and loss of T amplitude in the recovery phase of the cardiac cycle may indicate insufficient coronary artery blood supply if the change is of sufficient magnitude. The pilot study revealed that in all subjects, previously found free of any clinically detectable heart disease, there was ST and T depression and in one case even T wave inversion.

The second result at variance with the literature was in connection with the prolonged PR interval or atrioventricular conduction time. All subjects showed this

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[1]Hunt, E. A.,"An Electrocardiographic Study of Four Champion Athletes Before and After Competition," Unpublished Graduation Essay, University of British Columbia, February, 1957.

prolongation, while it was previously assumed that the increased heart rate would automatically shorten this period (11). There is a shortage of studies concerned with the PR interval following competition.

This pilot study then, cast some doubt on previous reports to be found in the literature. More subjects were obviously required in order to test the reliability of the results shown by the first four swimmers.

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CHAPTER III

REVIEW OF THE LITERATURE

Study of Curves Affected by Exercise.

There are many publications to be found within the literature that attempt to study the effects of exercise upon the electrocardiograms of athletes or of normal, young subjects. However, no report was found that had studied athletes with serial electrocardiograms beginning two or three minutes after the end of actual competition. Therefore, those studies selected for review are admittedly different from the present undertaking. It was believed, however, that the similarities would act as guidelines for the present study.

P Wave. The P wave may show increases in normal subjects who have participated in exercise. This was shown by Cureton (1) as he cited: Fredericq (2) who, in 1927, had studied soldiers after a long march; Messerle (3) who studied subjects after they had performed on a bicycle ergometer in 1928; and Kostjukow and Reiselman (4) who studied athletes in 1931 following weightlifting and heavy exercise programs.

In 1935 Paterson and Paterson (5) reported the results of an electrocardiogram (henceforth referred to as ECG) study of ten subjects who completed a 31 mile walking marathon on a hot day in 1930. The men, whose age ranged between 18 and 40, were studied before the race, approximately 15 minutes after the race and 2 hours following the second reading. The authors reported increased P wave amplitude in all cases. Cooper, O'Sullivan and Hughes (6) recorded increases in the P wave after one mile of strenuous rowing. Butterworth and Poindexter (7) found after a boxing tournament in 1942 that the P wave amplitude of all 35 subjects studied had increased.

In 1943 Barrow and Ouer (8) reported a P wave decrease in one-half of his 100 subjects following participation in hand-ball, swimming and other recreational sports. Cureton (9) in 1951, examined seated athletes one minute after they had run to exhaustion on a graded treadmill. He reported that the average P wave amplitude was lower after exercise than rest and that he believed that good performance would be accompanied by a low P wave. In 1958, Rasch et al. (10) reported that Olympic free style wrestlers displayed a rise in the amplitude of the P wave in four of the five subjects studied approximately 15 minutes after their bout. In 1957, Manning (11) reported no significant change after charting 200 normal subjects in the Masters' two-step test.

A summary of opinion in the literature indicates that with exercise the auricles are subject to increased sympathetic nervous activity as well as to an increase in venous return. These factors increase auricular action, as described in Starling's Law, and thereby force larger amounts of blood into the ventricle at greater rates. This surge of increased work may produce larger action currents resulting in a larger P wave.

PR interval. In 1937, Cooper, O'Sullivan and Hughes (12) found evidence of decrease in the PR interval after a severe rowing race. However, in 1942, Butterworth and Poindexter (13), after studying 35 boys in a Golden Gloves boxing tournament, reported that these subjects showed almost no change in the PR interval after this exercise. Paterson and Paterson (14), studying athletes immediately after a walking marathon of 31 miles, reported no PR interval change in seven subjects and a shortening in three. In 1943, Barrow and Ouer (15) reported PR interval decreases following participation in recreational sport by 100 men.

In 1954 Beckner and Winsor (16) studied 205 marathon runners and reported a decrease in the PR interval in all subjects but one. This subject showed an increase from 0.16 mv. to 0.21 mv.

In 1959 Abugattas (17) studied 54 champion athletes following skip jumping and reported that (18) ". . . in all instances the P-R interval became shorter when the physical activity decreased or when it was abandoned."

The PR interval marks the conduction time from the sinus auricular node to the ventricles of the heart. This time interval usually does not exceed 0.16 seconds. It would seem logical that this conduction time would shorten as the cardiac rate increases.

Q wave. There are few studies that have been concerned with the effect of exercise upon the Q wave. Poppi and Rovigatti (19), in 1939, reported the accentuation of the Q wave in experiments held at 17,712 feet as compared with readings taken at ground level. In 1951, Cureton (20) suggested that a study of the Q wave be undertaken.

R wave. The R wave will vary with exercise. Some studies indicate that increases in blood flow following moderate exercise will cause a rise in the amplitude of the R wave. This anomaly was demonstrated by Hausz (21) in 1933 after studying ten normal subjects. In 1937 Cooper, O'Sullivan and Hughes (22) supported this report.

However in 1938, Doetsch (23) demonstrated with decompression chamber experiments that the R wave can become

decreased during an oxygen deficiency. He presumed that an insufficient oxygen supply to the heart in exercise would produce the same result. Albers (24) supported this theory in 1942 when he showed evidence of R wave decreases during moderately-hard work on the bicycle ergometer. Hoogerwerf (25), studying marathon runners after their event in the 1928 Olympiad, also found a decrease in the R wave.

ST segment. Baum, et al. (26) studied twenty-three normal subjects in 1945. The ECGs were taken sitting down, while at rest, at 18,000 feet and at 2, 4, 6, 8 and 10 minute intervals after the Master's step test. The group T wave average was lowered after the exercise test and the altitude test. In a 1942 boxing tournament, Butterworth and Poindexter (27) found some ST elevation but no ST depression in 35 boys studied in the sitting position as soon as possible after their bouts. Yu, et al. (28), studied normal and diseased groups on the treadmill. The normal group demonstrated no ST depression. In 1954 Beckner and Winsor (29) reported ST depression in the ECG of one of 205 subjects taken 1-5 minutes after a marathon. Abugattas (30) studied fifty-four football players and swimmers in 1959 after an exercise of jumping with feet together for three to six minutes. He reported the observance of ST displacement upwards and attributed this to early repolarization.

Among other things, depression of the ST segment may indicate insufficient oxygen supply to the myocardium under the conditions of work.

T wave. Several studies have shown that an increase of the T wave after moderate to severe exercise is to be expected in normal subjects.

For example, in 1931 Wood and Wolferth (31) reported an increase of T wave potential after testing 100 normal subjects with varying amounts of exertion. The authors stated that:

> In none of our control subjects did exercise produce a deepening of an inverted T wave, a definite inversion of the flat T wave or a deviation of the S-T interval from the iso-electric line.

Hausz (33) reported in 1933 that a study of ten normal subjects after moderate work showed that all produced an increase in T potential. Paterson and Paterson (34) reported increased T waves in ten subjects studied after a 31-mile walking race in Toronto. In 1937, Cooper, O'Sullivan and Hughes (35) found that trained rowers showed an increase in T wave amplitude after rowing for one mile. The authors report that untrained subjects showed lower T waves.

Butterworth and Poindexter (36) studied 35 boys after a boxing tournament in 1942. The authors reported a general decrease in T wave amplitude. In this finding they differed

with Barker, Schrader and Ronzoni (37) who had studied four normal subjects in 1938. The subjects had run up and down stairs for two to four minutes and had ECGs taken immediately after. The researchers concluded that (38) ". . . the exercise is followed by acidosis and by a striking increase in the amplitude of the T waves."

Landen (39) concluded in a 1947 paper that moderate ergometer bicycle work should cause no change in the T waves of normal subjects. However, Walser (40) in 1946 stated that trained athletes had demonstrated initial T wave increase which was followed by a diminution of potential.

Yu, et al. (41) charted in 1951, twenty normal subjects during and following exercise on a graded treadmill. The authors reported that the T wave amplitude (42) ". . . usually became slightly lower during exercise and slightly higher during recovery." It was also observed that (43), "High, peaked T or inverted T waves during exercise were never observed.

In 1953, Wolf (44) concluded that the T waves of untrained subjects decreased after an "all-out" treadmill run but observed that trained persons increased this amplitude after exercise. Beckner and Winsor (45) studied 205 marathoners in 1954 and reported increases in the T waves. No inversion was found.

In 1957, Manning (46) undertook a study to determine the responses of the ECG to exercise. He observed 200 normal subjects before, during and after the Master's two-step test and reported that it was apparent that (47) ". . . minor to marked T-wave changes are of frequent occurrence in the immediate post-exercise tracing in this group of healthy young men."

Cureton (48), in 1958, compiled the results of many subjects from several tests and concluded that the potential of the T wave was amplified by athletic training and wheat germ oil ingestion. He further stated that the T wave may become lowered with very hard and exhaustive work or when the athlete developed"staleness."

The changes that occur in an athlete's ECG following exertion seem to depend to some degree upon the stress of the exercise and how well trained the individual was. Lighter activities appear to cause little alteration in normal subjects or athletes. Untrained and diseased individuals may show a loss of T potential as may those people engaged in very strenuous activity. The T wave alterations have significance in a category very similar to ST depression. It may indicate among other things that an insufficient oxygen supply is available to the myocardium under the condition of work.

U wave. In 1954 Beckner and Winsor (49) reported large U waves superimposed upon the T waves of 165 marathon competitors after five years of training. In 1956 a symposium was held by the Vermont Heart Association and the University of Vermont College of Medicine to summarize the known facts of U wave genesis. During this conference Lepeschkin (50) examined three possible explanations for the cause of the wave. He suggested that the wave was developed by (51):

1. A longer duration of the action potential in some section of the ventricles.
2. After-potentials following the action of potential proper.
3. Potentials elicited by the stretching of ventricular muscle during the period of rapid filling.

Surawicz, Kemp and Bellet (52) have stated (53) that "The U wave is frequently difficult to recognize, its onset and termination are often poorly delineated . . . ." The authors then concluded that (54): "Since the origin of the normal U wave is not clearly understood, it is difficult to attribute the finding of an abnormal wave to any specific physiologic or pathologic event." They also stated that (55): "It has also been found that the U wave cannot be clearly delineated when the heart rate is more than 110/min."

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CHAPTER IV

DESCRIPTION OF THE ELECTROCARDIOGRAM

The electrocardiogram (ECG) is a graph that records certain electrophysiologic phenomena which are expressed by the heart muscle during the phases of contraction and relaxation. This electric potential is recorded by the apparatus known as the electrocardiograph.

The first evidence that electrical currents were developed in the heart during contraction was demonstrated in the middle of the nineteenth century. The currents developed in the organ are small but instruments today are sufficiently sensitive to record them. Modern electrocardiography studies the direction, time relations and magnitude of these currents. It is known that all normal hearts yield graphs which conform in general to a regular pattern. No two individuals are exactly alike of course, but nevertheless there are limits to the variations which occur between individuals in good health (1).

The standards for the various resting readings have been established by experiment and study. These are being recognized or broadened as more research is conducted.

The various deviations of the ink line on the graph represent the electrical events in the heart and these deviations are identified by letters of the alphabet as detailed below. Positive deflections are above the iso-electric line and negative deflections below.

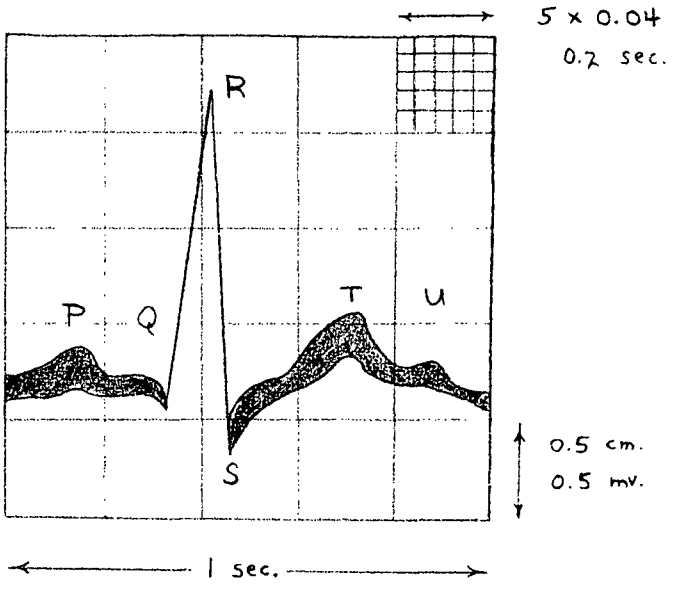

FIGURE I

The Naming of the Graph Segments

P WAVE: This is a small positive summit found in all normal young adults which rarely exceeds two scale divisions in amplitude. The P Wave represents the excitation of the atrial tissue by the sudden surge of electrical force from the sinus node. It is the first upward deflection from iso-electric.

PR INTERVAL: This represents the length of time required for the electrical impulse to travel from its origin in the auricle to the first sign of ventricular action. It does not usually exceed 0.16 seconds but may reach 0.22 seconds.

Q WAVE: A downward deflection indicating the onset of ventricular contraction. It is the first negative movement after P.

QRS COMPLEX: This group of waves represents the time taken for the ventricular conduction. This is completed very quickly having in normal subjects a total duration of no more than 0.1 seconds.

R AND S WAVES: These represent the ventricular activity. The R wave is the first positive deflection showing activity of the ventricular muscles and has a variable voltage of up to 2.0 millivolts.(henceforth referred to as mv.). The S wave is the first downward deflection following the R wave and is variable in depth depending on cardiac position. Variation in the twelve leads is normal as a result of the shifting importance of musculature of the left and right ventricles. Notching of R and S may also occur.

ST SEGMENT: This is the first recovery phase and is important if it fails to return to the iso-electric control line. This segment is usually not more than 0.1mv. above or below the iso-electric line.

T WAVE: This is also a recovery wave which follows the ST segment. It is usually upright in normal, young and healthy subjects in appropriate leads and can be varied in height. In 3, aVR and right side V leads it may be inverted.

QT INTERVAL: This interval represents the time of ventricular action from onset to recovery.

U WAVE: The additional deflection of the U wave is not abnormal and is related to the early stages of diastole (2). It is usually a small and positive wave that appears after the T wave and before the P wave.

Besides the blood pressure and pulse rate, the electrocardiographic observations studied were: the P wave amplitude, the PR interval's duration, the QRS interval, the QT interval, the voltage of R, the depth of S, the ST segment depression and the T wave. Outstanding U waves were also noted.

The most clearly defined cycle from each reading was selected for measurement. If extrinsic factors such as electrical disturbance or muscular movement interfered with the tracing, that lead was not considered. All downward deflections are marked with the negative symbol whole positive deflections are considered without sign.

Some of the considerations observed for accurate measurement were:

(a) When measuring the complexes, the thickness of the inking line must be taken into consideration. The technique must be consistent. This study used measurements from "top to top."

(b) Each small square along the abscissa measuring one millimetre is 0.04 seconds in duration with five to a section totalling 0.2 seconds.

(c) Each small square of the ordinates measures one millimetre. A deflection of ten millimetres is caused by one mv. of current.

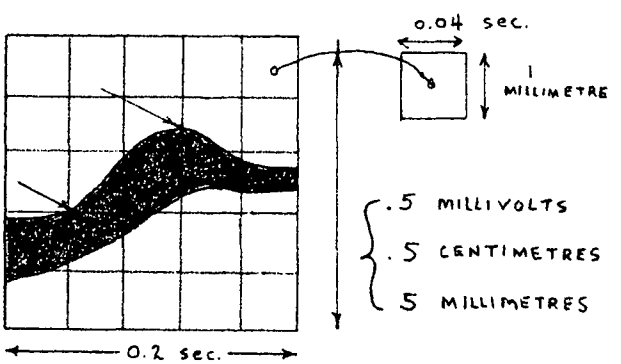

FIGURE II

Measurement of the Electrocardiogram

The function of the various leads can be described as follows:

Lead 1. The contrast of the electric potential of the right arm to the left arm.

Lead 2. The contrast of the potential of the right arm to the left leg.

Lead 3. The contrast of the electric potential of the left arm to the left leg.

Lead aVR. The total potential of the isolated right arm is compared to a zero potential rather than to another limb.

Lead aVL. The total potential of the isolated left arm is compared to a zero potential.

Lead aVF. The total potential of the isolated left foot is compared to a zero potential.

Precordial of V Leads. The potential from six separate positions across the chest is contrasted with a zero potential. These positions are shown in figure three.

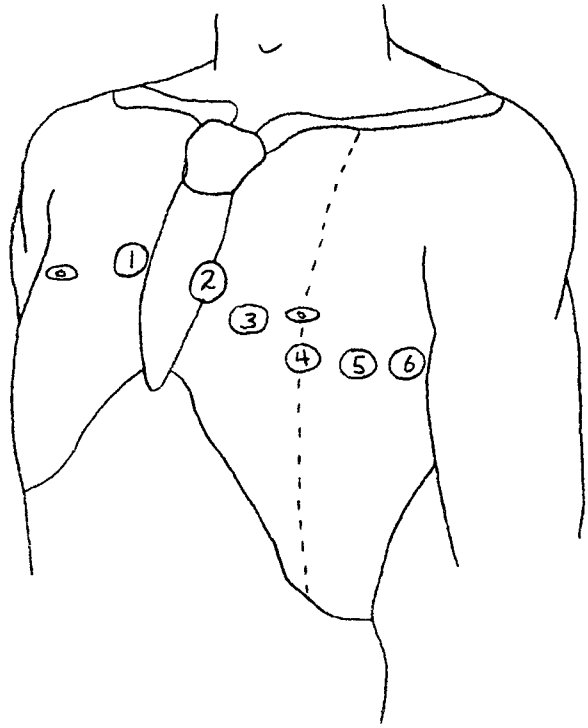

FIGURE III

The Placement of the Chest Electrodes

The location of the precordial electrode for each of the usual six chest leads is diagrammed as recommended by the Standardization Committee of the American Heart Association (3).

Reliability of Electrocardiograms

There are many factors, both intrinsic and extrinsic, that can affect the ECG. There are difficulties of a technical nature, of environmental source and of physical disturbance.

For example, it may be seen that technical errors can develop from static electrical interference or improper positioning of the subject. In order to avoid technical variations concerned with the ECG instrument the technique of the operator must be standardized. The placement of the electrodes must be constant, for one thing, and electrode paste should be employed.

However, it may not be faulty technique alone that disturbs the pattern. Normal individuals are subjected to variations in their environment and different activities may cause deviations from their resting ECG pattern. For example, it was reported in 1946 by Simonson, et al. (4) after studying twelve normal young men, that consistent ECG changes occur following a heavy meal. No changes were abnormal but the authors suggested waiting for at least an hour after a meal before taking an ECG.

Studies by Cureton, et al. (5) resulted in the conclusion that under standardized living conditions the ECG readings of normal resting subjects had little variation even over periods of months. There was agreement, however, with the 1946 research of Sensenbach (6) that indicated that varying environmental conditions such as athletic training, smoking, fatigue, obesity, hypoglycemia and fear could cause alterations in the pattern of the ECG.

Physical disturbances from the subject himself will also alter the graph. Broustet and Eggenberger (7) published in 1936 that they had found a relationship between the pattern of the graphs and the anatomical and functional state of the heart. McFarland, et al. (8), also established that postural changes affect the ECG. They showed that elevation of the diaphragm may cause right or left axis displacement and would therefore alter the graph. They suggested that factors such as deep breathing and change of positions in retest situations should be considered as possible effectors of ECG variation.

It was a major concern of this study to standardize each procedure and to select each subject so that such influences of technique, environment and physique were avoided in the attempt to record the influences of competitive sprint swimming.

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CHAPTER V

METHODS AND PROCEDURE

Nineteen champion swimmers, selected after interview, were invited to participate in the experiment. Each one had demonstrated superiority in his district and all had competed in national championships. One subject was re-tested two years later so that in all, twenty performances were analyzed. The testing procedure was outlined to each volunteer in the presence of his or her parents, at which time all questions about the test were answered.

The female subjects and their parents were given an explanation of the placement of the chest electrodes for the six chest leads. They and their parents were assured that only a physician and a nurse would be present during this phase of the study. For this reason, the number of female subjects was kept at a minimum.

The Canadian Amateur Swimming Association had scheduled the British Columbia Provincial Championships for June 22, 1956, at Empire Pool, Vancouver, British Columbia. The tests were arranged to conform to the regular programme and because more than thirty minutes were required to complete the tests, there was some difficulty in the selection

of the swimmers to be used. As a result, rarely more than three swimmers could be examined in a day. The examination period, then, had to be carried over four summers from 1956 to 1959. The B. C. Championships was used in all cases but three. These latter swimmers were tested following time trials held in conjunction with the Pan-American Games Trials.

Every one of the twenty tests was conducted at the Empire Pool at the University of British Columbia. The same facilities were used and each subject swam in the same lane, standardizing the distance to be covered from the pool's edge to the examining table. The pool temperature was a constant factor at 76°F. and the weather varied little.

A room in the gymnasium adjoining the pool was prepared for all the observations. This room was well isolated and, therefore, complete quiet and privacy were actually maintained throughout the entire proceedings.

The resting pulse rates and blood pressures of all subjects were determined and the resting ECGs were taken. In attempting to establish the resting ECGs of the first two subjects, it was found that the excitement of the race affected such a reading. Therefore it became standard practise to establish the resting levels one or two days after the race.

It was explained to the swimmers that they were to conduct themselves in every manner as had been their custom in past years of racing. Their diet, training, sleep and pre-race procedures were to be as normal as possible.

Immediately following their race, the swimmers were to leave the pool and walk to the examination room. Here they were to lie quietly while ECG readings were taken at three, six, nine, twelve, fifteen, twenty, twenty-five and thirty minute intervals to establish their recovery pattern. Following each ECG reading, a blood pressure reading was taken. H.S. was the first subject tested and therefore established the time intervals for the other competitors. It was especially important to begin the first ECG reading exactly three minutes after the completion of the race.

The ECGs of the first four competitors were recorded by Campbell and Davies.[1] The time intervals were recorded by Howell.[2] The identical procedures undertaken with the first subjects were maintained with all the swimmers. Strict

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Dr. R. Davies, M.D., Fellow of Cardiology at Vancouver General Hospital.

[2]Dr. M. L. Howell, B.A., M.A., Dip. P.E., Ed. D., F.A.C.S.M., Assistant Professor of Physical Education at the University of British Columbia.

adherence to the time schedule was accomplished.

In July, during the summer of 1957, three new subjects (A.B., J. Al., and D.G.) were tested in the same manner as had been the previous swimmers. Campbell took the tracings and blood pressures. The competition chosen was the B.C. Open Championships held at the Empire Pool. The same facilities were obtained for the test and the same pool was used.

Two years later, in the summer of 1959, thirteen additional subjects (A.M., D.M., A.S., E.C., B.Sa., B.C., M.S., B.W., J.A.2, B.S., M.H., W.M., D.S.) were observed following the B.C. Open Championships. The same procedure was again followed, the same pool was the scene of the competition, and the same facilities were used.

The electrocardiographs of these thirteen subjects were taken by the author, who had spent two weeks at the Vancouver General Hospital with the staff technicians. They had given the author instruction and had observed his techniques within the wards.

Practice in the ECG techniques, where speed was required, was undertaken with an assistant before any test subject was attempted. The author and the assistant practiced the timing of a swimmer leaving the pool, attaching the electrodes and blood pressure cuff and taking the first

readings. Three practice sessions were held; then the resting ECGs were tested on four subjects (J.A.2, B.Sl., M.H., W.M.) during the same summer at the Empire Pool. There was a variation here, in that no regular competition was utilized. The subjects were tested following a formal time trial. The performances here were being used as a decisive factor in determining selection for a team to be sent to the Pan American Games trials. However, the subjects were not racing other swimmers as in previous tests but were instead "racing the clock."

One of the swimmers (J.A.2), tested after the time trial, had been tested two years earlier following an orthodox race (J.A.1). His results were essentially similar in both cases.

None of the subjects was a smoker nor ever had been. Each felt that he or she had been leading a 'moderate life' with what each considered to be good habits of diet and sleep.

The author and assistant were dressed in white laboratory coats during the examinations, in an attempt to develop a formal atmosphere. The swimmers remained in their wet suits during the examination, but the room was quite warm, and after being lightly dried off, each subject attested that he was comfortable.

The pool water or perspiration did not apparently affect the validity of the readings, as an ample application of ECG jelly was used. The amount of jelly present and the positioning of the electrode were constantly checked.

A description of the subjects may be seen in Table 1.

TABLE I

Description of the Subjects

Subject	Age	Height	Weight	Resting Pulse	Resting Blood Pressure	Event	Test Time	Best Time	Remarks
Males									
1. B. Sa.	13	5'5	123	80	120/60	Free	1:11.2	1:09.7	Provincial Champion
2. D.S.	16	5'6	140	50	120/60-50	Free	1:04.4	1:03.1	Provincial Champion
3. E.C.	17	5'7	143	55	115/70-60	Free	1:03.1	1:02.5	National Junior Record
4. B.B.	19	6'	175	52	120/70	Free	1:07	1:04.3	Collegiate Swimmer and National Finalist
5. A.B.	17	5'11	165	55	130/70	Breast	1:20.5	1:16.1	Pan American Games
6. D.G.	15	5'9	155	75	120/70	Breast	1:25.2	1:22.4	National Junior Champion
7. J.A.1	20	5'11	170	60	130/80	Breast	1:20.8	1:18.9	British Empire Games
8. J.A.2	22	5'11	175	73	130/70	Breast	1:22.1*	1:18.9	British Empire Games
9. A.M.	15	5'6	134	75	118/70	Free	1:03.8	1:03.0	National Junior Record
10. B.W.	17	6'1	160	40	110/60	Back	1:07.4	1:05.2	Olympic Team, Pan-Am. and B.E. Games

* Taken during time trials.

TABLE I

Description of the Subjects
(Continued)

Subject	Age	Height	Weight	Resting Pulse	Resting Blood Pressure	Event	Test Time	Best Time	Remarks
Males									
11. A.S.	15	6'1	149	60	120/70-60	Fly	1:17.2	1:14	Provincial Champion
12. D.M.	16	6'	188	65	120/80-70	Free	58.2	1:00,4	Hawaiian School Boy Champion
13. D.J.	19	6'	180	58	110/60	Breast	1:24.1	1:21.4	British Empire Games
14. B.C.	17	5'10	175	52	112/70	Free	1:02.3	1:01.2	Pan American Games
15. B.S.1	19	5'11	175	44	120/80	Free	1:05.1*	1:00	Olympic Team, B.E. Games
16. W.M.	19	5'9	165	65	120/80	Fly	1:21.2*	1:15.8	German School Boy Champion
17. M.H.	20	5'9	155	47	110/70	Fly	1:22*	1:15.6	English County Champion
Females									
18. M.S.	13	5'1	95	75	100/80-60	Free	1:11.2	1:10.1	Pan American Games
19. H.S.	17	5'8	148	54	110/80	Free	1:08	1:07	Olympic Team, Pan Am. and B.E. Games
20. J.J.	16	5'10	143	80	110/60	Breast	1:36.2	1:27.5	Provincial Champion

* Taken during time trials.

CHAPTER VI

ANALYSIS OF RESULTS

In analyzing the results, several observations of the ECGs were made. The study was also concerned with the pre-exercise and post-exercise pulse rates and blood pressure changes. The resting levels were established by readings that were taken on days other than the tournament date. The recovery levels were measured during the thirty minute period that began three minutes after the competition.

The details of the electrocardiogram measured were: the P wave amplitude, the PR intervals' duration, the QRS interval, the QT interval, the voltage of R, the depth of S, the ST segment depression and the T wave. Outstanding U waves were also noted.

The results of the individual pulse rate, blood pressure and ECG readings are listed below. The time notations shown in relation to the ECG readings are those intervals of the recovery period that show results that are of significance according to medical texts.

1. MALE, B. SAN.

Pulse rate.

The pulse rate increased from a resting of 80 to 115 at the 3 minute reading. By 30 minutes it had returned to 80.

Blood pressure.

The resting normal was 120/60. This reading showed a rise to 160/80-50 at 2 minutes. After thirty minutes of rest, the final reading was 122/82.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 9 minute reading.

P wave.

3 min. The P wave in aVL became inverted -0.05 mv. from a resting position that was slightly positive. Lead 3 increased from 0.05 mv. at rest to 0.2 mv. at 3 minutes. Lead aVF also increased from 0.1 to 0.2 mv.

9 min. Lead 2 had increased from a resting position of 0.15 to 0.2 mv.

30 min. These changes had returned to the resting levels.

PR interval.

No measurable change was seen.

QRS interval.

No measurable change was seen.

ST segment.

No measurable change was seen.

T wave.

3 min. V T1 became inverted from a resting positive reading of 0.5 mv. to a negative reading of -0.15 mv. Inversion of -0.05 mv. was also seen in aVL. There was loss of potential in all other V leads. Lead 1 showed a lowering from 0.2 to 0.1 mv.

6 min. The inversion in V T1 increased at 6 minutes to 0.2 mv. The T wave in the V leads was further lowered by the second reading.

12 min. V T1 began to return to the resting level at 12 minutes.

20 min. All the V leads were returning to normal.

25 min. aVL had recovered from inversion to an iso-electric position.

30 min. The T waves had returned to normal by the final reading.

R and S waves.

No measurable change was seen.

U wave.

Prominent U waves were seen at rest and at 3 minutes in the V leads. This wave was apparent in all readings.

2. MALE, D.S.

Pulse rate.

The resting rate was seen to be 50 beats per minute. This rate had increased to 96 by 3 minutes. The rate was returned to 72 by 30 minutes.

Blood pressure.

The resting reading for this subject was 120/60-50. The pressures rose markedly to 200/80-0 by 3 minutes. The 30 minute reading was 122/80-60.

Electrocardiograms.

Rhythm.

No measurable change was seen.

P wave.

3 min. There appeared to be a slight effect upon the P wave as this unit increased from 0.15 mv. to 0.20 mv. in lead 2; from 0.05 mv. to 0.15 mv. in lead 3; and from 0.1 mv. to 0.20 mv. in aVF at the 3 minute reading.

15 min. Lead 2 had recovered.

30 min. Leads 3 and aVF had not completely recovered by 30 minutes.

PR interval.

3 min. The PR interval increased slightly between the resting and the 3 minute reading from 0.16 seconds to 0.18 seconds.

12 min. This interval had recovered by 12 minutes.

QRS interval.

No measurable change was seen.

ST segment.

3 min. ST segment in the V leads had lowered to iso-electric from an elevated position of 0.1 mv.

9 min. These leads had returned to their resting position.

T wave.

3 min. T in aVL lowered to iso-electric from 0.2 mv.

9 min. There was little other change in T potential until the 9 minute reading when a slight loss of potential was seen. Lead 1 dropped from a resting of 0.4 mv. to 0.2 mv.; lead 2 from 0.5 mv. to 0.3 mv. The V leads showed slight loss of potential.

30 min. By the final reading the tracings had almost returned to their resting levels.

R and S waves.

No measurable change was seen.

3. MALE, E.C.

Pulse rate.

From a low resting reading of 55 this subject rose markedly to 120 beats per minute at 3 minutes. By 30 minutes this had returned to 75.

Blood pressure.

The resting level showed a reading of 115/70-60. This pressure rose to 165/80 by 2 minutes. The 30 minute reading was 120/80 which was approaching the resting reading.

Electrocardiograms.

Rhythm.

A sinus arrhythmia was present at rest. A sinus tachycardia was seen until the 12 minute reading.

P wave.

3 min. Changes in the P wave at this reading were not measurable because of a blending with the T waves.

6 min. A large P wave was developing in leads 2, 3, aVR and aVF.

9 min. By 9 minutes the P wave was 0.2 mv. in lead 2, 0.15 mv. in lead 3, 0.15 mv. in aVF, and 0.1 mv. in aVR.

30 min. These waves were still high at 0.1 mv. by the final reading.

PR interval.

3 min. The resting PR interval was 0.14 seconds. This rose to 0.24 seconds by 3 minutes in lead V6.

15 min. By 15 minutes this time lowered slightly but was still 0.22 seconds.

30 min. The interval had still not recovered by the final reading when it was seen to be 0.20 seconds.

QRS interval.

No measurable change was seen.

ST segment.

3 min. Elevation of the ST segment was present at rest in the V leads up to 0.15 mv. in V4 but by 3 minutes these had lowered to iso-electric.

12 min. The segments began to ascend again.

30 min. The segments had regained their elevated position.

T wave.

3 min. In comparing the T waves of the resting to the first reading there was an apparent general reduction of potential.

9 min. During the 9 minute reading there was more general loss as exemplified by the reduction of T in lead one from a resting of almost 0.3 mv. to 0.1 mv., in lead 2 from 0.4 mv. to 0.2 mv., in lead V2 from 0.7 mv. to 0.3 mv. and in V3 from 0.7 mv. to 0.3 mv.

12 min. These readings began to ascend in amplitude.

30 min. By the final graph reading these T waves showed a pattern of good recovery.

R and S waves.

No measurable change was seen.

4. MALE, B.B.

Pulse rate.

The resting pulse rate of 52 increased to 115 at 3 minutes and was still 96 at 30 minutes. The following day it was again 52.

Blood pressure.

The systolic and diastolic pressures rose to 180/60-30 at 2 minutes following the exercise. This had returned to the pre-competition level of 130/80-70 in 26 minutes but had not returned to the resting average of 120/70 in 31 minutes.

Electrocardiograms.

Rhythm.

The resting rhythm showed a marked sinus arrythmia which remained true in post-exercise readings. There was a dropped beat in lead 1 of the 3 minute tracing. A sinus tachycardia was seen until the 30 minute reading.

P wave.

3 min. The P wave showed a definite increase in size in all leads, as from iso-electric to 0.05 mv. in lead 2.

12 min. The P wave had returned to the resting amplitude by 12 minutes in most leads.

PR interval.

3 min. The PR interval showed a definite increase from 0.16 at rest to 0.20 seconds in duration at 3 minutes in lead aVR.

30 min. The prolongation had not completely recovered by the final reading but had gained the resting level by the following day.

QRS interval.

The ventricular conduction time remained unchanged at 0.10 seconds.

ST segment.

6 min. This segment showed a fairly marked depression and slurring between 0.1 to 0.2 mv. in leads 1, 2, aVF and V4. This was maximal at 6 minutes and was most evident in leads 2, aVF and V4.

30 min. The variation had not completely recovered by the final reading.

T wave.

3 min. The T wave showed a loss of voltage in leads 1, 2, and aVF. Leads 1 and 2 lowered from 0.4 to 0.2 mv. The direction of the T wave was changed in V1.

9 min. Lead 3 inverted slightly from a slightly positive resting level. Leads 1 and 2 were 0.05 mv.

30 min. By the final reading the T waves were still quite small. Lead 2 was iso-electric. Lead 1 was 0.05 mv. Lead 3 was still slightly inverted. After 24 hours the tracings were similar to the control but still the amplitude was not quite so great. T in lead 3 had recovered.

R and S waves.

These waves showed respiratory variation but no measurable change could be discerned.

5. MALE, A.B.

Pulse rate.

The rate did not increase very markedly as it changed from a resting of 55 to 88 by 3 minutes. This fell again to 55 at 30 minutes.

Blood pressure.

The resting levels were 130/70. This rose to 180/40 at 3 minutes. By 11 minutes the pressure had returned to 130/70 and by 30 minutes had reached 120/80.

Electrocardiograms.

Rhythm.

Extra ventricular beats were observed even during the 15 minute reading. An example may be seen in lead 1 at 3 minutes. At 3 minutes in lead 2 there also occurred a dropped beat which was seen again occasionally as in V4 at 20 minutes. There was also a fair degree of sinus arrhythmia to be seen even though a tachycardia was present. This arrhythmia was present in the resting reading.

P wave.

The large P waves present at rest showed no measurable change following exercise.

PR interval.

3 min. This interval was 0.14 seconds at rest and had increased to 0.16 seconds by 3 minutes.

20 min. The PR interval was once again 0.14 seconds.

QRS interval.

No measurable change was seen.

ST segment.

No change of significance was seen beyond the fact that the ST segment was elevated at rest by 0.1 mv. in leads 2, 3, aVF, V3, V4, V5 and V6. These sections were iso-electric at 3 minutes and did not begin to return to the resting level until 12 minutes. By the 20 minutes reading they had fully recovered.

T wave.

3 min. High peaked T waves were seen in V3, V4, V5 and V6 at the resting level and at 3 minutes. V2 peaked at 3 minutes thereby increasing T amplitude from the resting level.

6 min. There was loss of T amplitude at 6 minutes in most leads. V2 fell from 0.10 mv. to 0.5 mv.; V3 from 0.12 mv. to 0.7 mv.; V5 from 0.11 mv. to 0.8 mv. and V6 from 0.8 mv. to 0.5 mv. T in aVR fell from 0.8 mv. to 0.4 mv.

9 min. At 9 minutes there was slightly more loss of T potential.

12 min. The levels were essentially similar to 9 minutes.

15 min. The levels were essentially similar to 12 min.

20 min. The 20 minute graphs and those following were close to the resting readings.

R and S waves.

No measurable change was seen.

6. MALE, D.G.

Pulse rate.

This subject showed a large increase in rate, as from a resting reading of 75 the pulse became 140 by 3 minutes. After 30 minutes of recovery it had lowered to 84.

Blood pressure.

The resting pressures were 120/70. This became 150/90 by 3 minutes and climbed to 170/50-30 by 5 minutes. At 29 minutes the pressures were 130/70.

Electrocardiograms.

Rhythm.

There was evidence of sinus arrhythmia in the resting readings. A sinus tachycardia was seen until the 20 minute reading.

P wave.

3 min. The P waves in leads 2, 3 and aVF became inverted at -0.1 mv. from a positive reading of 0.05 mv. at rest.

PR interval.

No measurable change was seen.

QRS interval.

No measurable change was seen.

ST interval.

No measurable change was seen.

T wave.

3 min. There was high peaking of the waves in V3, V4, V5 and V6 at 3 minutes.

9 min. The peaking had disappeared and all leads had lost considerable potential. V4 for example moved from a resting of 0.7 mv. to 1.4 mv. at 3 minutes and to 0.2 mv. by 9 minutes.

12 min. Lead 3 had inverted -0.1 mv. from its resting position of 0.15 mv.

15 min. Lead 3 remained inverted -0.1 mv.

30 min. All the leads showed steady return toward resting levels although the resting readings were not quite reached by the final reading.

R and S waves.

No measurable change was seen.

7. MALE, J.A.1

Pulse rate.

From a resting rate of 60 the subject developed an increase to 120 by the 3 minute reading. By 30 minutes this rate had lowered to 88.

Blood pressure.

The resting pressures were seen to be 130/80. These rose to 160/20 by 2 minutes and had returned to 130/80 by 17 minutes. By 30 minutes the pressures were 120/80..

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 20 minute reading.

P wave.

3 min. The P wave showed increased voltage in lead 2 by the 3 minute reading from 0.1 mv. to 0.3 mv.

15 min. P had decreased to 0.2 mv. in lead 2.

30 min. This lead was still elevated at 0.2 mv. by 30 minutes.

PR interval.

3 min. At rest the PR interval was seen to be 0.18 seconds. At 3 minutes this interval had increased slightly to 0.20 seconds.

15 min. The reading had returned to 0.18 seconds.

QRS interval.

No measurable change was seen.

ST segment.

3 min. The subject showed a resting ST segment elevation of 0.2 mv. in lead 2. A resting ST of about 0.1 mv. was seen in lead 3, aVF, V2, V3, V4, V5 and V6. These elevated ST segments at rest had become iso-electric at 3 minutes.

12 min. The segments rose again.

30 min. This segment had not returned to normal by 30 minutes.

T wave.

3 min. There was loss of T amplitude in lead 1 from a resting of 0.4 mv. to 0.3 mv. at 3 minutes. VT1 was positive at 0.15 mv. from an inverted resting of -0.5 mv.

6 min. Lead 1 decreased to 0.2 mv. All other leads had lost potential as well.

9 min. Lead 1 had further diminished to 0.15 mv. at 9 minutes from 0.4 mv. at rest. VT1 became inverted again at 9 minutes.

12 min. T potential showed recovery of potential.

15 min. Further improvement of T potential was seen.

20 min. T in lead 1 began to improve.

30 min. T in lead 1 was still only 0.3 mv. The resting amplitude in most of the leads was not fully gained.

R and S waves.

No measurable change was seen.

8. MALE, J.A.2

Pulse rate.

The resting rate of 73 was increased by the exertion of exercise to 110 at 3 minutes. By 30 minutes this rate had become 96.

Blood pressure.

The resting pressure was 130/70. This rose during the exercise and by 2 minutes was 160/50. The blood pressure had returned to 130/80-70 by the 30 minute examining period.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 25 minute reading.

P wave.

3 min. The P wave in lead 2 rose from a resting of 0.05 mv. to 0.15 mv. Lead 3 increased from 0.05 mv. to 0.1 mv.; and aVF rose from an iso-electric measurement to 0.15 mv.

30 min. These waves had not recovered by the final reading.

PR interval.

3 min. In V1 at resting the PR interval was clearly 0.16 seconds. In the same lead at 3 minutes this interval was 0.18+ seconds.

9 min. These increases showed signs of diminution.

15 min. The interval was once again 0.16 seconds.

QRS interval.

9 min. The QRS complex from its resting of 0.08 seconds increased slightly by the 9 minute mark to 0.10 seconds.

30 min. This complex had returned to resting duration.

ST segment.

3 min. At rest the ST segment was elevated 0.1 mv. in lead 2. It was also elevated in leads aVF, V1, V4, V5 and V6. By 3 minutes this segment was depressed slightly in aVF, V1 and V6. Leads 2 and V5 were iso-electric. In aVR, ST was normally in a slightly depressed position but by 3 minutes this was elevated 0.1 mv.

6 min. All these leads but aVR were iso-electric.

9 min. aVR was iso-electric.

20 min. aVR was at the resting level.

30 min. aVF and V5 were elevated once again. In general, however, the ST segment had not returned to normal by 30 minutes.

T wave.

3 min. In lead 1 the amplitude at rest was 0.3 mv. This reading had lowered to 0.2 mv. by 3 minutes.

6 min. In general most of the waves had lost potential by the 6 minute reading.

9 min. VT1, which was inverted by -0.3 mv. at resting, was -0.05 mv. at 6 minutes and -0.1 mv. by 9 minutes.

20 min. VT1 was recovering erratically, reducing again to -0.05 mv. at 20 minutes.

25 min. VT1 reduced further, becoming iso-electric.

30 min. By 30 minutes VT1 was still only -0.05 mv. in inversion. The resting T potential in other leads had not been reached by the 30 minute period.

R and S waves.

No measurable change was seen.

9. MALE, A.M.

Pulse rate.

The resting rate was 75. This rose quickly to 120 by 3 minutes and fell again to 92 by 30 minutes.

Blood pressure.

The resting blood pressure reading was 115/70. These pressures rose to 160/70 by 2 minutes. By 30 minutes the pressures were 120/80.

Electrocardiography.

Rhythm.

A sinus tachycardia was seen until the 25 minute reading.

P wave.

No measurable change was seen.

PR interval.

No measurable change was seen.

ST segment.

No measurable change was seen.

T wave.

3 min. At 3 minutes the T waves generally became more prominent. V2, V3, V4, and V5, especially, showed the increase.

6 min. By 6 minutes the T waves had begun to diminish.

12 min. There was further general loss of T potential. Lead 3 became iso-electric from a resting position of 0.2 mv. upright. The other leads are generally below their resting levels.

20 min. The waves looked much like their resting counterparts.

30 min. The graphs were very near the resting control.

R and S waves.

No measurable change was seen.

U wave.

a prominent U wave had developed by the 20 minute reading, particularly in V2, V3 and V4. This wave persisted to the 30 minute reading.

10. MALE, B.W.

Pulse rate.

This athlete showed a resting pulse of 40 beats per minute. The rate rose sharply to 130 at the 3 minute reading. By 30 minutes the rate was lowered to 94.

Blood pressure.

From a resting of 110/60 the pressures rose to 170/60-50 by 2 minutes. This dropped to 110/40-20 by 11 minutes and was 115/70-50 at 30 minutes.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 30 minute reading.

P wave.

3 min. The P waves became more prominent, particularly in leads 2, 3, and aVF. These waves were difficult to measure, however, as they were slurred in with the T wave.

30 min. By 30 minutes these P waves were still somewhat more prominent than normal.

PR interval.

3 min. Despite the marked change in rate the PR interval was slightly prolonged from 0.14 seconds to 0.16 seconds.

25 min. The prolongation is apparent until the 25 minute reading when it is once again 0.14 seconds.

QRS interval.

No measurable change was seen.

ST segment.

3 min. There was ST depression of -0.1 mv. in lead 2 and of -0.5 mv. in aVF at 3 minutes.

6 min. These depressions were largely recovered by 6 minutes.

T wave.

3 min. The T waves in V2 and V3 became tall and peaked by 3 minutes. T in aVF was inverting.

6 min. By the 6 minute reading, T in leads 1 and 2 was less prominent than at 3 minutes. T in aVF was inverted -0.1 mv. V5 and V6 were lowered from resting readings of 0.6 mv. to 6 minute levels of 0.2 mv.

12 min. aVF was inverted -0.2 mv. from a resting level of 0.05 mv.

30 min. These T changes had not completely returned to resting by the final reading. T in aVF was still slightly inverted. T in lead 3 was inverted -0.2 mv. at rest and remained so inverted, without change, throughout the entire test.

R and S waves.

No measurable change was seen.

11. MALE, A.S.

Pulse rate.

The resting rate was 60. By 3 minutes after the exercise the rate had increased to 110. This rate had fallen to 84 by 25 minutes.

Blood pressure.

From a resting reading of 120/70-60 the blood pressure rose to 160/60-50 by 2 minutes. By 30 minutes the pressure was again 120/70.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 6 minute reading.

P wave.

3 min. The P waves in leads 2, 3, aVR and aVF at rest were nearly iso-electric but at 3 minutes these had enlarged to 0.2 mv. and 0.3 mv.

20 min. By 20 minutes these waves were back to the resting level with the exception of lead 2.

PR interval.

3 min. The PR interval at rest was 0.14 seconds in lead 2. At 3 minutes this interval had prolonged from 0.16 to 0.18 seconds.

25 min. This interval was not fully recovered by 25 minutes.

QRS interval.

No measurable change was seen.

ST segment.

3 min. There was ST depression in leads 2, V5 and V6. From an elevated resting position of 0.1 mv. V6 is depressed -0.1 mv. by 3 minutes. Lead 2 has variable depression from -0.05 to -0.1 mv.

9 min. This depression was almost completely recovered.

T wave.

6 min. Loss of T potential was apparent at 6 minutes. T in lead 1 dropped from a resting of 0.3 mv. to 0.15 mv., aVL lowered from 0.2 mv. to 0.05 mv.

12 min. General loss continued until V5 showed a 12 minute reading of 0.3 mv. as compared with its resting level of 0.8 mv.

25 min. T potential was still generally lower at the final reading.

R and S waves.

No measurable change was seen.

12. MALE, D.M.

Pulse rate.

From a resting level of 65 the rate increased to 100 at 3 minutes. Twenty minutes was the last reading for this boy and the rate was 75.

Blood pressure.

The resting readings were 120/80-70. This rose to 170/60-40 by 2 minutes and was 130/80-70 by 22 minutes.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 9 minute reading.

P wave.

No measurable change was seen.

PR interval.

No measurable change was seen.

QRS interval.

No measurable change was seen.

ST segment.

No measurable change was seen.

T wave.

3 min. By 3 minutes there was a slight peaking of the T waves in V2, V3 and V4.

6 min. T in lead 3 inverted -0.1 mv. and T in aVR became flattened from a negative position of -0.3 mv. T in aVF was flattened and diphasic from a resting position of 0.2 mv.

9 min. By 9 minutes there was further, although slight, reduction of T potential. T3 remained inverted. T2 had flattened out from a resting reading of 0.3 mv.

15 min. By this recovery period the T wave had shown a steady improvement toward resting levels.

20 min. The resting levels had essentially been reached.

R and S waves.

No measurable change was seen.

13. MALE, D.J.

Pulse rate.

The resting rate of 58 beats per minute had not been recovered in 30 minutes. It rose to 105 in 3 minutes and fell gradually to 85 by the final reading.

Blood pressure.

The pressures had risen to 165/50 at 2 minutes. These had returned pretty well to the resting normal of 110/60 by 17 minutes.

Electrocardiograms.

Rhythm.

A marked sinus arrhythmia in the resting tracing was observed. This was present to a lesser degree following activity. A sinus tachycardia was seen until the 20 minute reading.

P wave.

3 min. These waves showed increase in amplitude from 0.1 to 0.15 mv. in lead 2, and from 0.05 to 0.15 mv. in lead 1. The P wave became biphasic in V1.

12 min. Lead 2 had increased to 0.2 mv.

30 min. This increase had largely disappeared by the final reading.

PR interval.

3 min. This interval showed an increase from 0.12 at rest to 0.16 seconds by 3 minutes in lead 2.

20 min. This interval had returned to 0.12 seconds.

QRS interval.

In the resting position the ventricular conduction time showed a reading of 0.10 to 0.12 seconds. The R deflection showed splintering of the R in V1 and showed a typical M shaped R. There was a fairly prominent S wave of 0.7 mv. from the R precordium. There was no significant change in this unusual conduction pattern. The QT interval appeared to be shortened following the exercise.

ST segment.

6 min. This segment showed a significant degree of depression in leads 2, aVF, V5 and V6 which appeared to be maximal at 6 minutes with a depression of -0.1 mv.

25 min. The depression did not disappear until the seventh tracing.

T wave.

12 min. The T wave in lead 1 became inverted -0.2 mv. from a resting of 0.05. T in aVF inverted to -0.5 mv. from a resting reading of 0.2 mv. T in V1 also inverted slightly from a resting level of 0.1 mv.

30 min. The T wave inversion was still present in lead 1 at 30 minutes although it had disappeared from aVF. There was a loss of T wave amplitude in lead 2, V4 and V6 which had not recovered in 30 minutes.

R and S waves.

There was no measurable variation in the height of the R or the depth of the S.

14. MALE, B.C.

Pulse rate.

The normal resting rate was seen to be 52 while after the strain of competition it rose to 118 at 3 minutes. At 30 minutes the rate was still 95.

Blood pressure.

The resting blood pressure was 112/70. This had risen to 180/80-60 at the 2 minute reading. By 19 minutes this had returned to 120/80-60 and by 30 minutes was 120/80.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 25 minute reading.

P wave.

3 min. The P wave was more prominent in the first reading that it was at rest as it rose in aVF from 0.05 mv. to 0.15 mv., in lead 3 from iso-electric to 0.1 mv. and in lead 2 from 0.05 mv. to 0.15 mv.

30 min. The P wave in all these leads had not reached the resting level by 30 minutes.

PR interval.

3 min. The atrio-ventricular conduction time was 0.16 seconds at rest in aVF while at the 3 minute reading it was prolonged to 0.18 seconds.

9 min. The interval had returned to its resting value of 0.14 seconds.

QRS segment.

No measurable change could be seen.

ST segment.

No measurable change could be seen.

T wave.

3 min. There were high peaked waves in V2 and V3. At the same reading there was a loss of T potential in V5 and V6 from a resting of 0.50 mv. to 0.30 mv.

6 min. By this reading the high peaked T waves had rounded and there was further loss of T potential so that T in V5 was then 0.3 mv. and V6 was 0.2 mv. In lead aVF, T had decreased from a resting of 0.3 mv. to 0.15 mv.

9 min. There was further loss of T potential in lead 1 which dropped from a resting of 0.3 mv. to a 9 minute reading of 0.2 mv. Lead 3 dropped from 0.15 mv. at rest to a position of 0.05 mv. Lead aVF declined to 0.1 mv. and V5 and V6 lowered to 0.2 mv.

12 min. This reading was essentially the same as the 9 minute reading.

15 min. The T wave potential was beginning to return.

30 min. The T potential in all leads improved to better than the 6 minute level but had not reached the resting position.

R and S waves.

No measurable change was seen.

15. MALE, B.S.1

Pulse rate.

This subject's rate was very slow at 44. The rate increased to 105 at the 3 minute reading and had lowered to 82 by 30 minutes.

Blood pressure.

The pressure rose from a resting of 120/70-60 to a 3 minute reading of 160/70-40. At 11 minutes it had returned to 120/60-40. By the 30 minute interval it was 110/70-60.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 9 minute reading.

P wave.

3 min. The P waves decreased slightly in the V leads at the 3 minute reading. These waves had flattened out from a resting position of 0.05 mv. Lead 2 remained unchanged.

6 min. By the second reading the P waves were up again in V1 and V2.

9 min. V3 and V4 had returned to normal but V5 and V6 had not returned to the resting level of 0.5 mv.

30 min. The waves had recovered their resting position.

PR interval.

No measurable change was seen.

QRS segment.

3 min. The resting QRS complex showed a reading of 0.08 seconds while at 3 minutes the length was 0.1 seconds.

9 min. This length had decreased slightly.

12 min. The complex had returned to the normal interval of 0.08 seconds.

ST segment.

No change of significance was seen beyond the fact that the ST segment was elevated at rest by 0.1 mv. in the V leads. These sections were iso-electric at 3 minutes and did not begin to return to the resting level until 20 minutes. By the final reading they had fully recovered.

T wave.

3 min. At 3 minutes there could be seen minor wave loss and it was noticed that VT1 became upright from a negative resting to a positive reading of 0.15 mv.

6 min. VT1 was positive by 0.2 mv. Lead 3 flattened to iso-electric from a resting position of 0.1 mv.

9 min. A lowering of aVR could be seen as it dropped from 0.3 mv. to 0.15 mv.

12 min. VT1 remained positive until 12 minutes when it flattened.

30 min. VT1 had not fully recovered to its resting negative deflection.

R and S waves.

No measurable change was seen.

16. MALE, W.M.

Pulse rate.

The resting rate was approximately 65 per minute. This rose to 120 by 3 minutes and fell again to 90 by 30 minutes.

Blood pressure.

The resting pressure was 120/80 which rose to 170/50 by 2 minutes. The subject showed a reading of 110/80-70 at 30 minutes.

Electrocardiograms.

Rhythm.

There was a sinus arrhythmia at rest. A sinus tachycardia was seen until the 25 minute reading.

P wave.

No measurable change was seen.

PR interval.

No measurable change was seen.

QRS interval.

No measurable change was seen.

ST segment.

3 min. There was ST depression in V5 and V6 at 3 minutes as this segment lowered to -0.1 mv. from an iso-electric position. ST is also depressed slightly in leads 1, 2, and aVF.

15 min. For the most part this depression lasted through the 15 minute interval.

30 min. Normal levels were reached by the final reading.

T wave.

3 min. There was general loss of T amplitude in all leads except V3, V4 and V5, which became high and peaked.

6 min. T1 and T2 were further lowered. T3 became slightly inverted from iso-electric to -0.05 mv.

9 min. All T potential showed a reduction except V3, V4 and V5.

12 min. T3 became inverted -0.1 mv. Also aVF became inverted -0.05 mv. from a positive resting reading of 0.1 mv.

30 min. T1, T2 and T3 almost returned to the resting level by the final reading, but aVF remained inverted.

R and S waves.

No measurable change was seen.

17. MALE, M.H.

Pulse rate.

The rate for this subject was low at 47 at rest in the lying position. This rate rose during competition and showed 96 at 3 minutes. By 30 minutes the rate had lowered to 52.

Blood pressure.

The resting reading was 110/70. The 2 minute reading showed a change to 150/50-30. The subject again demonstrated the resting level of 120/70 at 22 minutes.

Electrocardiograms.

Rhythm.

No measurable change was seen.

P wave.

3 min. The P wave in lead 2 rose to 0.15 mv. from a resting level of 0.5 mv. P in other leads did not change.

20 min. Lead 2 had returned to normal.

PR interval.

3 min. The resting PR interval was 0.14 seconds in aVF while at 3 minutes there was an increase to 0.16 seconds.

15 min. This interval had returned to 0.14 seconds.

QRS interval.

No measurable change was seen.

ST segment.

No measurable change was seen.

T wave.

3 min. This subject showed an inverted T wave during rest in leads 3, aVR, aVF, V1, V2 and V3. At 3 minutes T in leads 3 and V1 became positive at 0.1 mv. V2 and V3 became positive at 0.15 mv.

6 min. T1 and T2 were less prominent than at 3 minutes and were only 0.1 mv. positive. T in V1, V2 and V3 were still upright. V4 was depressed from a resting reading of 0.6 mv. to 0.2 mv. V5 was depressed from 0.5 mv. to 0.2 mv. V6 dropped from 0.4 mv. to 0.2 mv.

12 min. There was slight improvement toward resting levels by this recovery period.

15 min. By 15 minutes VT1 and VT3 were becoming inverted once again while T in leads V4, V5 and V6 were rising toward their normal amplitude.

30 min. There was further improvement to the final reading although the T potential did not fully return to the resting graphs.

R and S waves.

No measurable change was seen.

18. FEMALE, M.S.

Pulse rate.

Resting readings showed this subject's pulse rate to be approximately 75 per minute. By 2 minutes this rate had increased to 120. The 30 minute rate showed a return to 86.

Blood pressure.

The resting blood pressures were 100/80-60. Within 2 minutes after the competition this had risen to 150/60-40. The 30 minute reading indicated a return to 110/80-70.

Electrocardiograms.

Rhythm.

There was a marked sinus arrhythmia present throughout the readings. A sinus tachycardia was seen until the 25 minute reading.

P wave.

3 min. The P waves in lead 1 flattened out at 3 minutes from a resting level of 0.1 mv. The P wave in V4, V5 and V6 had flattened out and in aVL had become inverted from an 0.5 reading. P in leads 2 and 3 had, by 3 minutes, become elevated from 0.05 to 0.15 mv. P in aVF also rose.

25 min. All leads showed a return to resting levels.

PR interval.

There was no apparent change in the PR interval despite the rapid rate increase. The resting PR was 0.12 seconds as was the reading at 3, 6, 9 minutes and all further time intervals.

QRS interval.

No measurable change was seen.

ST segment.

3 min. ST depression was seen in lead 3. This lead was depressed -0.1 mv.

6 min. There was depression of -0.1 mv. seen in aVF.

12 min. Both leads had recovered.

T wave.

3 min. The T waves became high and peaked in V2, V3, V4 and V5.

6 min. By this reading the peaking was not nearly so apparent and T in lead 3 had inverted to -0.05 mv. from 0.1 mv. at rest.

9 min. By this reading it could be seen that there had been a general loss of T potential. The resting inversion of T in V1 at -0.3 mv. was less prominent at -0.05 mv. and the normally positive T in lead 3 remained inverted.

30 min. T potential had gradually been regained and was near to the resting level by 30 minutes. T in lead 3 remained inverted.

R and S waves.

No measurable change was seen.

19. FEMALE, H.S.

Pulse rate.

The rate before the race was 80 as contrasted with the normal resting rate of 54. The maximal increase in rate was to 115 in 3 minutes which did not return to the resting anxiety (80) until after the 30 minute reading.

Blood pressure.

The blood pressure rose markedly with a systolic recording of 150 and a diastolic of 40 at 2 minutes. The readings returned to the resting normal of 110/80 at 26 minutes.

Electrocardiograms.

Rhythm.

The resting rhythm showed a marked sinus arrhythmia. A sinus tachycardia developed following the race and persisted until the 25 minute reading.

P waves.

No measurable changes were apparent.

PR interval.

No measurable changes were apparent.

QRS interval.

The ventricular conduction time (QRS interval) tended to be slightly shorter than the increased pulse rate. There did not appear, however, to be a reduction in the duration of the QT interval as would have been expected. Instead it remained the same or somewhat greater than the control tracing. This condition remained until 24 hours after the event when the QT interval was still above the normal value.

ST segment.

3 min. The ST segments showed a depression of -0.1 mv. in all significant leads, being particularly apparent in leads 2, 3, aVF and V4.

6 min. The depression appeared to be maximal at -0.1 mv. in the 6 minute reading.

30 min. The tracing still had not quite returned to the control level. By 24 hours, however, the segments were comparable to the control tracing.

T wave.

3 min. The T waves showed a definite loss of potential from 0.3 to 0.2 mv. in lead 1. 12 min. The loss seemed to be maximal at 0.05 mv. and was most noticeable in leads 1 and aVL. In V1 there was a change from negative to positive and in V2 there was an increase in T wave amplitude.

R and S waves.

There was variation in R amplitude and S depth but the respiratory effort and the technical difficulties of obtaining rapid tracings made these small variations appear unimportant. In V4 the R wave increased from 1.5 mv. at rest to 2.5 by 3 minutes.

20. FEMALE, J.J.

Pulse rate.

The rate increased from a resting of 80 to a maximal 110 and returned close to the control rate in 30 minutes.

Blood pressure.

The pressure rose markedly to 165-60/30 at the 2 minute mark. This returned to the resting normal of 110/80 in 22 minutes.

Electrocardiograms.

Rhythm.

A sinus tachycardia was seen until the 25 minute reading.

P waves.

3 min. The P wave amplitude lowered at 3 minutes from a resting of 0.15 mv. in lead 2 to 0.05 mv. The resting level was regained by 12 minutes.

PR interval.

3 min. This interval lengthened slightly from 0.14 to 0.16 seconds in leads 1 and 2.

12 min. This interval had returned to the resting level.

QRS interval.

3 min. This interval appeared largely unchanged except for a definite increase from 0.36 to 0.40 seconds in lead V4.

ST interval.

3 min. During the 3 minute tracing the ST interval was depressed up to 0.15 mv. in leads 1, 2, V2, V4, V5.

20 min. The depression had returned to normal levels.

T wave.

6 min. The T waves showed a distinct loss of voltage which appeared maximal by the 6th minute. The T wave in 3, became inverted to -0.1 mv. from a resting level of 0.5 mv. V1 changed direction.

20 min. These changes had largely returned to the resting levels by 20 minutes.

R and S waves.

No measurable change was seen.

SUMMARY OF RESULTS

Pulse rate. Each individual showed a fairly marked increase in pulse rate with an average increase of 92.7 per cent above the resting rate. The range of rate increase was from 37 per cent (J.J.) to 225 per cent (B.W.). Only one of the athletes tested (A.B.) returned to his determined resting rate within the 30 minute recovery period. See Table II for summary.

The rate of B.W. showed a large increase from 40 to 130 by 3 minutes. B.C. also had a large rise from 55 to 120. D.G. rose from 75 to 140. The average change was from 60.7 to 112.6.

Blood pressure. Each subject showed a marked increase in pulse pressure. The systolic pressure rose to an average of 165 points at 2 minutes while the diastolic fell to 59 points. The largest increase recorded was shown by D.S. with a reading of 200/80-0 at 3 minutes. See Table III for summary.

Electrocardiogram.

Rhythm. Eight of the subjects tested (E.C., D.J., H.S., M.S., B.B., D.G., A.B., W.M.) showed a marked sinus arrhythmia before and after exertion. All subjects but three (M.H., A.B., D.S.) developed a marked sinus tachycardia after their race. Two subjects (B.B., B.W.) did not show rates below the limit of tachycardia (100 beats per minute) until the final reading at 30 minutes.

TABLE II

Pulse Rate Changes

Subject	Rest	Intervals in Minutes 3	6	9	12	15	20	25	30	Percentage of Change
				Beats per Minute						
Males										
1. B.San.	80	115	100	92	90	86	86	88	80	40
2. D.S.	50	96	90	86	84	76	72	68	72	92
3. E.C.	55	120	110	100	96	96	92	88	75	119
4. B.B.	52	115	105	105	103	103	100	100	96	120
5. A.B.	55	88	86	86	84	80	78	66	55	60
6. D.G.	75	140		103	103	100	94		84	75
7. J.A.1	60	120	105	103	100	102	90	84	88	100
8. J.A.2	73	110	108	102	100	100	100	98	96	65
9. A.M.	75	120	115	110	110	105	100	94	92	86
10. B.W.	40	130	120	110	110	110	102	100	94	225
11. A.S.	60	110	95	90	96	94	88	84		83
12. D.M.	65	100	100	90	88	84	75			54
13. D.J.	58	105	103	100	98	100	96	94	85	770
14. B.C.	52	118	110	110	105	103	100	97	95	125
15. B.S.1	44	105	100	98	94	94	90	92	82	140
16. W.M.	65	120	115	115	110	110	100	95	90	85
17. M.H.	47	96	90	86	80	75	55	52	52	107
Females										
18. M.S.	75	120	110	105	105	100	92	90	86	60
19. H.S.	54	115	105	105	105	100	96	96	90	111
20. J.J.	80	110	105	102	105	105	98	94	90	37
Sum =	1215	2252								1854%
Mean =	60.7	112.6								92.7%

TABLE III

Blood Pressure Changes

Subject	Rest	Intervals in Minutes 2	5	8	11	17	19	22	27	29	32
Males											
1. B.Sa.	120/60	160/60-50	140/78-50	130/80-60	130/80-60	128/80-60	120/84-70	130/80	122/84	122/82	120/80
2. D.S.	120/60-50	200/80-0	190/60-20	150/60-40	150/60-40	140/50-30	158/70-48	140/60-50	130/60-40	128/70	122/80-60
3. E.C.	115/70-60	165/80	150/70	124/60	120/70-60	120/70	130/70	120/75	120/75	120/80	120/80
4. B.B.	120/70	180/60-30	160/60-30	160/60-40	140/60	150/60	150/70-60	150/80-60	150/80-60	140/80-60	130/80-70
5. A.B.	130/70	180/40	170/40	140/50	130/70	130/80	130/80	120/80	120/80	120/80	120/80
6. D.G.	120/70	150/90	170/50-30	140/60-30		130/70-60		130/70	130/70	130/70	
7. J.A.1	130/80	160/20	140/60-40		130/70	130/80	120/80	120/80	120/80	120/80	
8. J.A.2	130/70	160/50	150/60	130/60	130/70	130/70	140/80-70	130/80	130/80-70	126/80	130/80-70
9. A.M.	115/70	160/70	150/80-70	140/80	130/80-70	130/80-70	120/80-70	120/80	115/80	120/80	120/80

TABLE III

Blood Pressure Changes
(Continued)

Subject	Rest	Intervals in Minutes 2	5	8	11	17	19	22	27	29	32
Males											
10. B.W.	110/60	170 60-50	140 40	110 10-20	100 40-20	110 40-30	110 50-30	110 60-40	110 70-50	115 70	115 70-50
11. A.S.	120/70-60	160 60-50	140 60	130 80-70	120 80	120 80-70	130 70	120 80-70	120 80	120 70	120 70
12. D.M.	120/80-70	170 60-40	150 70-40	120 70-60	120 70-60	130 80-60	130 80	130 80-70			
13. D.J.	110/60	165 50	120 40	110 60-40	110 70-60	100 60	110 70	110 70	110 70	110 70	110 80
14. B.C.	112/70	180 80-60	160 60	140 70	120 60	130 70-60	120 80-60	120 80	120 80	120 80	120 80
15. B.S.1	120/70-60	160 70-40	150 60-40	140 60-50	120 60-40	110 60-40	110 60-40	110 60-40	110 70-50	110 70-50	110 70-60
16. W.M.	120/80	170 50	150 60	140 60	170 60-40	140 70-50	150 70-60	140 70	140 80-70	130 80	130 80-70
17. M.H.	110/70	150 50-30	150 60-40	130 60	120 60	120 60	120 70	110 70	110 70	110 80-70	110 80-60

TABLE III

Blood Pressure Changes
(Continued)

Subject	Rest	Intervals in Minutes 2	5	8	11	17	19	22	27	29	32
Females											
18. M.S.	100/80-60	150/60-40	150/60-40	120/60-30	120/60-40	110/70-50	120/70-50	120/70	110/70-60	110/80-70	110/80-70
19. H.S.	110/80	150/40	130/50	120/60	130/60	130/60	120/60	120/60	120/80	110/80	110/80
20. J.J.	110/60	165/60-30	150/50-30	120/40	120/40	120/50	110/50	110/60	110/80	110/80	110/80

	rest	2 min
Sum =	2342/1400	3305/1190
Mean=	117/70	165/59

P wave. Three subjects showed no change in their P wave while thirteen showed an increase in amplitude. Four subjects (J.J., M.S., B.S.1, D.M.) lost P wave potential in some leads. See Table IV for summary.

PR interval. Eight subjects showed no change in their PR intervals despite rapid rate increases. In twelve cases (J.J., B.B., D.J., E.C., J.A.1, J.A.2, B.C., M.H., D.S., A.B., A.S., B.W.) there was a fairly definite prolongation of the atrioventricular conduction time up to 0.04 seconds. E.C. showed an increase of 0.10 seconds, the PR increasing from 0.14 seconds at rest to 0.24 seconds by 3 minutes. In most cases the change was an increase of 0.02 to 0.04 seconds. See Table V for summary.

QRS interval. The ventricular conduction time seemed little affected in most subjects. Fifteen observations showed no changes from the resting interval. Three cases increased the duration slightly (J.J. - 0.36 to 0.4 sec.; J.A.2 - 0.08 to 0.1 sec.; B.S.1 - 0.08 to 0.1 sec.) while two subjects (H.S., D.J.) showed a decrease in time.

ST segment. According to medical texts (1, 2) a significant ST segment depression is defined as a segment 0.1 mv. below the iso-electric line. This segment was significantly depressed in eight subjects (H.S., J.J., B.B., D.J., W.M., M.S., A.S., B.W.). The ST segment was depressed up to 0.2 mv. The depression was maximal in the early

TABLE IV

P Wave Changes

Subject	Lead	Change + 0 -	Rest mv.	3 Min. mv.	Max. Change - mv.		Time min.	Recovery Time	30 Min. mv.
Males									
B.Sa.	2	+	0.15	0.15	0.2	at	9	12	0.15
D.S.	2	+	0.15	0.20	0.20	at	12	15	0.15
E.C.	2	+	0.05	0.05	0.2	at	25		0.15
B.B.	2	+	iso-el.	0.05	0.1	at	9	30	iso-el.
A.B.	2	0	0.2	0.2					0.2
D.G.	2	+	0.2	0.25	0.25	at	3	6	0.1
J.A.1	2	+	0.1	0.3	0.3	at	12	25	0.1
J.A.2	2	+	0.05	0.15	0.15	at	3		0.15
A.M.	2	+	0.2	0.3	0.3	at	6	9	0.15
B.W.	2	+	0.05	0.05	0.1	at	25		0.1
A.S.	2	+	0.05	0.15	0.2	at	12		0.15
D.M.	2	0	0.2	0.2					0.2
D.J.	2	+	0.1	0.15	0.2	at	12		0.15
B.C.	2	+	0.05	0.15	0.2	at	12		0.1

TABLE IV

P Wave Changes
(Continued)

Subject	Lead	Change + 0 -	Rest mv.	3 Min. mv.	Max. Change mv.		Time min.	Recovery Time	30 Min. mv.
Males									
B.S.1	2	0	0.1	0.15					0.1
W.M.	1	0	iso-el.	iso-el.					iso-el.
M.H.	2	+	0.05	0.15	0.15	at	6	25	0.05
Females									
M.S.	2	-	0.05	-0.05	-0.1	at	9	25	0.05
H.S.	1	0	0.05	0.20	0.2	at	3		0.05
J.J.	2	-	0.15	0.05	0.05	at	3	12	0.15

TABLE V

PR Interval Changes

Subject	Lead	Change + 0 -	Rest sec.	3 Min. sec.	Max. Change - sec.		Time min.	Recovery Time	30 Min.
Males									
B.Sa.	2	0	0.16	0.16					0.16
D.S.	2	+	0.16	0.18	0.18	at	3	6	0.16
E.C.	V1	+	0.14	0.24	0.24	at	3		0.16
B.B.	aVR	+	0.16	0.20	0.20	at	9	12	0.16
A.B.	aVF	+	0.14	0.16	0.16	at	12	20	0.14
D.G.	2	0	0.12	0.12					0.12
J.A.1	2	+	0.18	0.20	0.20	at	9	15	0.18
J.A.2	V1	+	0.16	0.18	0.18	at	12	15	0.16
A.M.	2	0	0.16	0.16					0.16
B.W.	2	+	0.14	0.16	0.16	at	9	25	0.14
A.S.	2	+	0.12	0.16	0.16	at	9		0.14
D.M.	2	0	0.12	0.12					0.12
D.J.	2	+	0.12	0.16	0.16	at	9	20	0.12
B.C.	aVF	+	0.16	0.18	0.18	at	3	9	0.16

TABLE V

PR Interval Changes
(Continued)

Subject	Lead	Change + 0 -	Rest sec.	3 Min. sec.	Max. Change - sec.		Time min.	Recovery Time	30 Min.
Males									
B.S.1	aVF	0	0.20	0.20					0.20
W.M.	aVF	0	0.14	0.14					0.14
M.H.	aVF	+	0.14	0.16	0.16	at	3	15	0.14
Females									
M.S.	2	0	0.12	0.12					0.12
H.S.	2	0	0.16	0.16	0.16	at	6	12	0.16
J.J.	2	+	0.14	0.16	0.16	at	6	12	0.14

tracings but persisted for some time in a few subjects. See Table VI for summary.

12 min. A.S., B.W. had recovered.

15 min. W.M. had recovered.

20 min. M.S., J.J. had recovered.

25 min. D.J. had recovered.

30 min. H.S., B.B. had not fully recovered.

Six other subjects (E.C., B.S.1, D.S., A.B., J.A.1, J.A.2) showed ST segments that were up to 0.15 mv. above iso-electric at rest. For these subjects this segment lowered to iso-electric at 3 minutes. This segment had returned to the positive resting level by:

9 min. D.S.

20 min. E.C., A.B.

30 min. B.S.1, J.A.1, J.A.2.

Six subjects showed no significant change in ST value.

T wave. The T waves in all cases showed a marked loss of potential. In ten subjects (J.J., B.B., D.J., B.Sa., W.M., M.S., D.G., M.H., D.M., B.W.) certain leads had actually become inverted and only one (D.M.) had fully recovered by 30 minutes.

Five subjects (B.W., W.M., M.S., B.C., A.B.) showed high peaked T waves at 3 minutes. These diminished quickly and steadily to a position below the amplitude shown in the resting reading. See Table VII for summary.

TABLE VI

ST Segment Changes in Eight Subjects

Subject	Lead	Change + 0 -	Rest mv.	3 Min. mv.	Max. Change mv.		Time min.	Recovery Time	30 Min. mv.
Males									
B.B.	2/V4	-	iso-el.	-0.1	±0.15	at	6		-
D.J.	2	-	iso-el.	-0.1	-0.1	at	6	25	iso-el.
W.M.	2	-	iso-el.	-0.15	-0.15	at	3	9	iso-el.
A.S.	2	-	+	-0.1	-0.1	at	6	12	+
B.W.	2	-	0.1	-0.1	-0.1	at	6	12	0.1
Females									
M.S.	3	-	iso-el.	-0.1	-0.1	at	6	12	iso-el.
H.S.	aVF	-	iso-el.	-0.1	-0.1	at	6		-0.05
J.J.	V4	-	iso-el.	-0.15	-0.15	at	6	20	iso-el.

TABLE VII

T Wave Changes

Subject	Lead	Change + 0 -	Rest mv.	3 Min. mv.	Max. Change - mv.	Time min.	Recovery Time	30 Min. mv.
Males								
B.Sa.	aVL	-*	+	-0.05	-0.5 at	6	25	+
D.S.	aVL	-	0.2	iso-el.	iso-el. at	3		0.1
E.C.	2	-	0.4	0.3	iso-el. at	12	30	0.4
B.B.	3	-*	0.05	0.1	-0.05 at	20		-
A.B.	aVR	-	0.6	0.7	0.2 at	9	20	0.6
D.G.	3	-*	0.15	0.25	-0.1 at	12		0.05
J.A.1	3	-	0.2	0.2	iso-el. at	15		0.1
J.A.2	1	-	0.3	0.2	0.1 at	25		0.15
A.M.	3	-	0.15	0.2	iso-el. at	25		0.05
B.W.	aVF	-*	0.05	-	-0.2 at	12		-0.05
A.S.	V5	-	0.8	0.6	0.3 at	12		0.4
D.M.	3	-*	0.1	-	-0.1 at	9	15	+
D.J.	1	-*	0.05	-	-0.20 at	12		-0.05
B.C.	3	-	0.15	0.1	0.05 at	12		0.1

* indicates inversion.

TABLE VII

T Wave Changes
(Continued)

Subject	Lead	Change + 0 -	Rest mv.	3 Min. mv.	Max. Change - mv.	Time min.	Recovery Time	30 Min. mv.
Males								
B.S.1	3	-	0.1	0.1	iso-el.	at 9		+
W.M.	3	-*	0.15	-0.05	-0.1	at 12	20	0.1
M.H.	3	-†	-0.15	0.5	0.5	at 6	9	-0.1
Females								
M.S.	3	-*	0.1	0.1	-0.1	at 12		-0.05
H.S.	1	-	0.3	0.2	0.05	at 6		0.15
J.J.	3	-*	0.1	0.15	-0.05	at 6	20	0.1

* indicates inversion. † change of sign - negative to positive.

R and S waves. Only one subject (H.S.) showed significant variation in the R and S waves. There was variation in R height and S depth from 1.5 mv. at rest to 2.5 mv. by 3 minutes.

U wave. In the section of his book concerned with the electrocardiograms of champion athletes, Cureton (3) stated that the U wave ". . . in general, is found only in superior athletes." A review of the data for this wave was undertaken, subsequently revealing a resting U wave in all the subjects in this report. Two subjects (A.M., B.Sa.) showed prominent U waves in the recovery graphs although finer measuring criteria, as recommended by Holzmann (4), were not used at this time.

All waves considered. Eighteen out of the twenty performances studied, produced at least one of the following variations: PR prolongation, T inversion, or ST depression. Two subjects (A.M., B.S.l) showed insignificant changes in these areas.

Observations concerning the results. Every subject cooperated fully so that there was no disturbance of routine except for one case (D.M.). This boy's father insisted upon taking this subject home to bed following the 20 minute reading. He claimed the boy was being kept out too late. No other complications beyond missing the last two readings were encountered.

The time intervals between each ECG reading were successfully maintained for each of the twenty tests. None of the tests was disturbed by undue excitement or noises. The only interruption was in the case of D.M. and this was merely a halting of the proceedings without any excitement.

Ten of the subjects were disappointed with their times during the test. These subjects (J.A.2, D.G., B.C., B.Sa., B.W., A.S., M.H., W.M., A.B.) felt that they could have gone "harder' or complained of poor tactics such as 'starting off too fast'. Six subjects (J.A.2, B.S.1, B.W., A.S., M.H., W.M.) felt that they were not in 'good enough' condition.

The electrocardiograms of all subjects are on file in the main library of the University of British Columbia.

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CHAPTER VII

DISCUSSION OF RESULTS

From careful perusal of the data two findings of note seem to emerge. The first significant result was found in the atrioventricular conduction time. The PR interval is that time as measured from the first sign of auricular activity (P wave) to the first sign of ventricular activity (R wave). The PR interval was prolonged in twelve of the twenty cases up to 0.24 seconds. These findings were quite out of keeping with previous reports (1, 2, 3) although a similar study of maximal exertion followed by serial electrocardiograms was not found in the literature. In fact Barrow and Ouer (4), in 1943, stated that elongated PR intervals were indicative of cardiac disease.

The prolongation of this interval was doubly significant because of the increased heart rate that these subjects displayed. It was ordinarily assumed that increased heart rate would be accompanied by shortened atrioventricular conduction time.

The second significant finding lay in the change of the recovery phases of the ventricular activity, namely the degree of ST depression, the loss of T wave amplitude and

even T wave inversion. Generally speaking, the literature does not support these findings when studying healthy, normal young people following exercise, as will be shown in the following discussion.

In 1952, Morin et al. (5) described the ECG changes of eight rugby players, boxers and swimmers that were recorded as soon as possible (five to ten minutes) after their events had taken place. The authors found that the T waves had decreased over the resting control in seven of the eight subjects. They believed that these changes had ". . . passed beyond the limits of simple physiological adaptation" (6) and they described the T inversion of one man in leads one and two as being frankly pathological. Rasch, et al. (7) in 1958, also reported a subject who showed changes ". . . suggesting subendocardial injury following competition." (8). The authors had studied six wrestlers fifteen minutes before and fifteen minutes following competition. Five subjects showed increased T wave amplitude while one gave a T inversion described as being ". . . in striking contrast to those observed in the tracings of the other subjects." (9). Jouve, et al. (10) had also reported suspected pathological variation of the T wave in five of their seventy-two subjects following a long foot race in 1957.

Lozada and Tempone (11) reported in the 1958 Acta Cardiologica, their study of twenty normal young men and women after running in place for one minute at the rhythm of 150-160 "movements" per minute. The authors stated that they had frequently observed either an increase or a decrease of the T wave amplitude as a standard response. They add, however, that (12) " . . . noticeable depression as a late response--after five minutes--has a great pathological value."

In the present study it was seen that all of the twenty subjects tested showed a decrease in the T wave amplitude following swimming competition. Furthermore, nine of these healthy, young athletes showed T wave inversion. Perhaps these variations were observed to be so prevalent in this study because of the severity of the exercise and the promptness in beginning the serial electrocardiograms. Each subject was observed first at exactly three minutes after the completion of the race.

In 1953, Simonson (13) studied the effects of fifteen minutes of treadmill walking on the ECGs of thirty normal young (18 to 28) men and 100 normal, middle-aged (48-58) men. Recovery graphs were recorded one and three minutes following exercise. The older group showed PR elongation, ST segment depression and loss of T potential which the author took to

be demonstrated evidences of coronary insufficiency. However he believes these signs merely to indicate the decrease of physical fitness with age.

The present study reports that eighteen of the twenty subjects, all in their late teens, have shown the same variations of PR interval, ST segment and T wave that this older group displayed.

ST segment depression has been discussed by many researchers. Among them were Barrow and Ouer (14) who chose at random, in 1943, one hundred men ranging in age from 21 to 67 who were participating in a variety of recreational sports. The men were divided into two age groups, one averaging 30.7 years and the other 49.1. These men had ECGs recorded before and as soon as possible after participating in their recreation. In this study there were reported only three cases of ST depression or inversion of the T wave following exercise. These three cases had been ruled out of the study because of a history of heart disorder. The authors concluded that (15); "We should therefore be suspicious of intrinsic myocardial damage when distortion of the ST segment or frank inversion of the T wave in significant leads is produced by exercise."

Beckner and Winsor (16) also studied ST depression in 205 marathon runners taken one to five minutes after their

race. They reported ST depression in one subject but the infrequency of this response led them to believe that it was ". . . a pathologic response to exercise" (17). Lozada and Tempone (18) studied twenty normal subjects after running in place for one minute and stated that (19), ". . . no normal individual showed ST modifications after five minutes [after] the exercise was finished."

In the present study of twenty swimmers it was seen that nine demonstrated ST segment depression up to 0.2 mv. following one-hundred metre sprint competition.

There are differences of opinion to be found within the literature as to the gravity of such changes that have been demonstrated by the twenty subjects reviewed here. Not all reviewers attach grave meaning to T inversion and ST depression, as will be shown below.

In reporting their "ECG of the Month" in 1958 Gardberg and Rosen (20) reviewed the tracings revealed by a 33 year old airplane pilot. After vigorous bicycling exercises this subject showed T inversion in lead one although there had been no previous history to suggest cardiac lesion. The authors took these tracings to be normal with the increased rate as they had seen this in other normal adults 18-28 years of age. They added the statement:

"However, the incidence of this finding among normals under ordinary circumstances is so small that it is bound to be held suspect under any circumstances" (21). The authors also stated that there was a traditional prejudice against T inversion but that they believed that this was not necessarily caused by the disease.

Seldon (22) reported in 1958 that many causes other than ischaemic heart disease can produce inversion of the T wave. Normal physiological variation is developed through fear, thyrotoxicosis, increased sympathetic tone due to tension or the ingestion of large amounts of glucose. He warns against misinterpreting such variations as he states that ". . . the most common error in electrocardiographic diagnosis is to attach grave significance to normal physiological variations in the T wave" (23). It was his belief that deeply inverted and symmetrical T waves were characteristic of cardiac infarction. However, he further believed that T waves that are slightly inverted, diphasic or asymmetrical have other causes.

McCristal (24) agrees with Seldon's innocent T wave inversion theory and while Freeman (25) argues with certain points concerned with cause, both believe that the T wave can be unreliable as a diagnostic signpost. They feel that T inversion is not necessarily an ominous sign. In 1959 Abugattas (26) supported this view as he felt that T wave

changes in trained subjects were reflecting more of an adaptation to stress than a disturbance.

It can be seen, then, that there has been a gradual change in feeling about T wave depression following exercise (27, 28, 29) although there are still those who attach pathologic significance to such change (30, 31, 32, 33). It would appear that there is also still much concern about PR interval prolongation and ST depression. Many researchers are of the opinion that these anomalies are indicative of myocardial injury (34, 35, 36, 37).

Our findings would, perhaps, cast some doubt on such summary conclusions because they fail to take into consideration individual general health, physical training, nervous reaction and whether or not the amount of exertion done is a customary experience. There was no doubt that the nineteen individuals involved in this test were free of clinically detectable heart disease. They were all in their late teens, were all in excellent general condition, and were all well trained in their events. Yet they have shown prolongation of their AV conduction time and marked ST segment and T wave changes. In this setting, the variations can only be interpreted as manifestations of functional alterations in the heart following such maximal competitive exertion. There was no suggestion that these functional changes were harmful,

although recovery in many cases was delayed beyond 30 minutes.

The exact cause for these changes could have developed from many sources. The excitement and anxiety of competition should be mentioned first of all. These emotions, it is known, have far-reaching physiological effects. Sympathetic stimulation of the adrenal cortex and the adrenal medulla are only two of those effects worthy of mention. Next, the breath holding of speed swimming may be a factor of some importance as it may tend to increase the oxygen lack and carbon-dioxide retention to a greater degree than other competitive sports.

Another factor to be considered as a possible cause for ECG alteration is the great oxygen debt that would develop within the musculature during severe exertion. This condition would most likely be accompanied by increased lactic acid concentration and carbon-dioxide accumulation. All of these mentioned conditions will tend to cause acidosis, and the resulting change in the blood pH may affect the cardiogram. It should also be noted that during such sudden, severe muscle strain there is almost always an important shift of electrolytes (particularly potassium) out of the muscle cell.

Morin, et al. (38) placed the cause for ECG variations observed in their 1952 study directly upon insufficiency of oxygen. They also observed however, that (39):

> It is well known today that the impregnation of the myocardium with poisons, such as digitalis, or its alteration by a disturbance of the blood affecting its oxygen carrying ability, the concentration of the calcium or potassium ions or the vitamin B1 content bring about modifications of the phase of repolarization.

Causes of T wave decreases in amplitude have long been a point of discussion for physiologists. In 1938, Barker, Schrader and Ronzoni (40) experimented with the ECG after ingestion of ammonium chloride to produce acidosis, and sodium bicarbonate to produce alkalosis. They concluded that, "The observations indicate that alkalosis is accompanied by a decrease and acidosis by an increase in the amplitude of the T-waves" (41).

Butterworth and Poindexter (42), in 1942, believed that shortening of the diastolic filling period might impede coronary blood flow. ECG variation was discussed in 1949 by Hecht (43) who believed that ST segment shifts and T changes are governed by four basic factors:

1. Slowing the rate of repolarization results in inversion of the terminal portion of T in the epicardial leads.
2. Slowing the rate of repolarization endocardially results in an increase in the height of T.
3. Lesions result in an incomplete repolarization of the region injured.

4. Subepicardial leads are indicated by elevation of RS-T in epicardial leads while RS-T depression is the result of alterations of the endocardial surface.

With induced hypoxemia and graded treadmill walking, Yu, et al. (44) in 1951 found that ST depression occurred with a frequency that varied directly with the stress that six different groups were subjected to. In 1953, Simonson (45) also conducted experiments correlating the ECG and anoxia. He found that at rest, oxygen transportation to the subendocardial layers was poorer than to the subepicardial layers. The physiologist concluded that following exercise then, the subendocardial supply would be quite inadequate. This oxygen lack, he believed, would affect ST segment and T waves because they ". . . depend to a much greater degree on metabolic factors" (46).

Beckner and Winsor (47) studied 10 marathon runners in 1954 and could find no positive correlation between the increased height of the T waves and the serum potassium level determined immediately after the race. However, two subjects were studied 8, 21 and 30 hours after running where it was seen that ". . . as the serum potassium rose the T waves became smaller" (48).

Seldon (49) had attributed similar T wave changes to carbohydrate ingestion, fear and increased sympathetic tone. Freeman (50) in 1957 agreed in part with Seldon's main point but disputed fear and increased tone as a cause. He supported Gardberg and Rosen (51) who attribute such ". . . innocent T change to postural change in the heart's electrical position"(52).

Cureton (53), in 1958, related diminishing T wave amplitude with hard work and the resultant lowering of vitamin B1 and glycogen reserve. He believed that this was consistent with the findings of Simonson (54) who had demonstrated loss of T wave potential with semistarvation. Cureton believed that the T wave reflects ". . . several aspects of circulatory and nutritive fitness as it is probably associated with build-up of glycogen reserves"(55).

A report in 1959 by Abugattas (56) agreed with Cureton's thesis (57) that there was a relation between the magnitude of the T wave changes and the degree of physical activity. Abugattas ascribed ST segment displacement to ". . . early repolarization" (58).

Following such a literature review, then, it was hypothesized that the ECG changes observed are in general likely to be due to the excessive sympathetic system and glandular stimulation, as well as to transient oxygen lack,

changes in blood pH, and fluctuating electrolyte values. This experiment was not designed to define the actual cause of any observed changes but was meant only to observe the facts. The changes here are probably physiological under this condition of maximal effort with healthy, young, trained individuals. It is thought that before any such changes be considered significant of heart trouble such studies as these of the normal subject should be reviewed.

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27. Seldon, loc. cit.

28. McCristal, loc. cit.

29. Abugattas, loc. cit.

30. Morin, et al., loc. cit.

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CHAPTER VIII

SUMMARY AND CONCLUSIONS

Nineteen teen-age, highly trained, championship calibre athletes were studied by serial electrocardiograph and blood pressure readings at the University of British Columbia, before and immediately following 110 yard competitive swimming. One swimmer was studied a second time, two years after the first testing, so that a total of twenty performances was analyzed.

It was concluded that:

1. Pulse rates were increased rather sharply and did not return to normal by 30 minutes.

2. There was a marked increase in pulse pressure with the systolic reading as high as 200 and the diastolic as low as 30.

3. The 12 lead serial electrocardiogram taken at 3, 6, 9, 12, 15, 20, 25 and 30 minute intervals revealed prolongation of the atrioventricular conduction time, or PR interval, in twelve of the twenty cases observed.

There were also unexpected changes in the recovery phase of the cardiogram. Namely, ST segment depression was seen in eight of the twenty cases and T inversion in nine of the twenty races analyzed.

All twenty subjects lost T wave potential following the exercise.

4. The PR interval prolongation in three of the twelve subjects showing this variation had not returned to their resting levels by the thirty minute reading.

Four of the eight subjects showing ST depression had not fully recovered by thirty minutes of lying recovery time.

Only one of the nine subjects that showed T inversion had fully recovered his resting T potential by the final reading.

Thirteen of the twenty cases studied showed that the T wave potential had not returned to the resting levels in thirty minutes of lying recovery time.

5. PR prolongation, ST depression or T inversion was seen in eighteen of the twenty performances analyzed.

6. Some of the reasons for the alterations, which are considered physiological under the terms of this experiment, are discussed.

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APPENDIX

DEFINITION OF TERMS

1. Tachycardia. Excessive rapidity of the heart's action is described as a tachycardia and is usually applied to a pulse rate above 100 beats per minute.

2. Bradycardia. The term applied to an abnormal slowness of the heart beat is bradycardia. This condition is defined as a pulse rate of less than 60 beats per minute.

3. Extrasystole. An extrasystole involves a premature contraction of the heart which is independent of the normal rhythm and arises in response to an impulse in some part of the heart other than the sino-auricular node.

4. Fibrillation. Fibrillation is defined as an independent contraction not under the control of a motor nerve.

5. Arrhythmia. Any irregularity in the rhythm of the heart beat is described as an arrhythmia.

6. Champion swimmers. Those subjects who have represented their country in international competition or who have competed in national championships shall be termed champion swimmers for the purposes of this study.

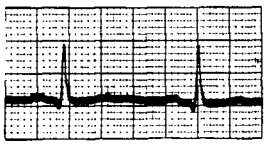
APPENDIX

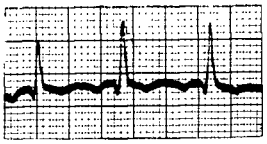
ELECTROCARDIOGRAM SAMPLES

FROM EACH OF THE TWENTY PERFORMANCES

1. B. San. T Inversion.

lead aVL





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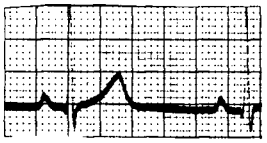
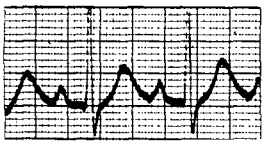
2. D.S. PR Prolongation.

lead 2





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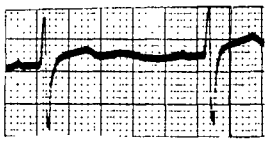
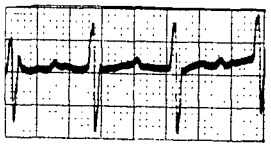
3. E.C. PR Prolongation.

lead V1





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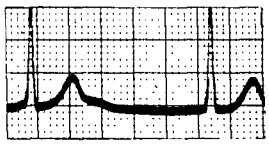
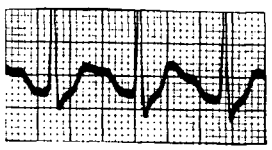
4. B.B. ST Depression.

lead 2

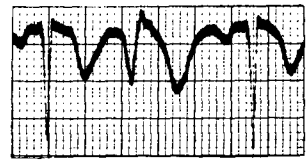




rest ST: iso-el 3 min. ST: -0.1 mv.

5. A.B. Extra Ventricular Beat.

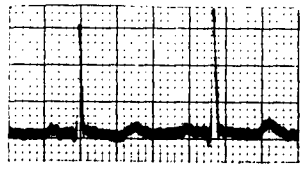
lead aVR

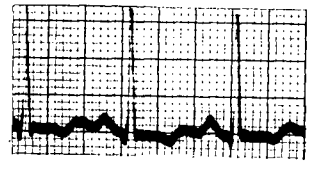


rest 3 min.

6. D.G. T Inversion.

lead 3





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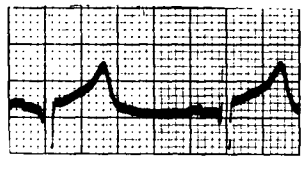
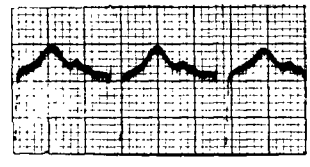
7. J. A1. Lowered ST and T Potential.

lead V5

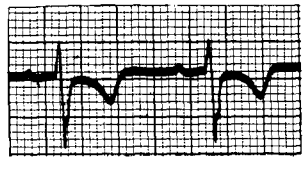


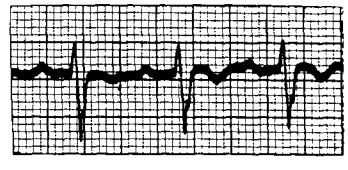


rest ST: 0.15 mv. 6 min. ST: iso-el

8. J. A2. PR Prolongation.

lead V1

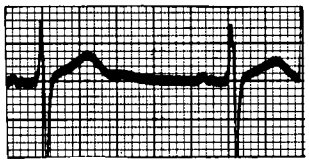


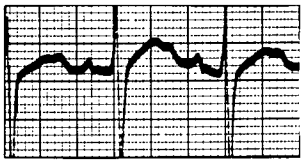


rest PR: 0.16 sec. 3 min. PR: 0.18

9. A.M. Development of Prominent U Wave.

lead V2

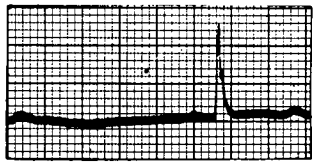


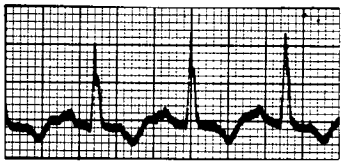


rest 20 min.

10. B.W. T Inversion.

lead aVF

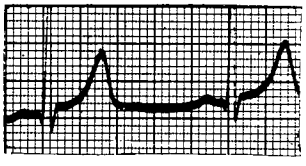


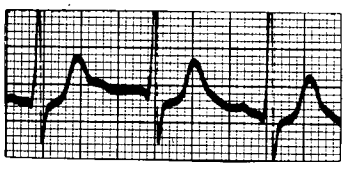


rest T: 0.05 mv. 12 min. T: -0.2 mv.

11. A.S. ST Depression.

lead V6

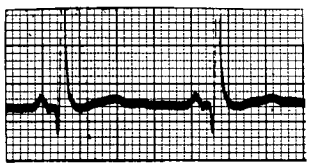


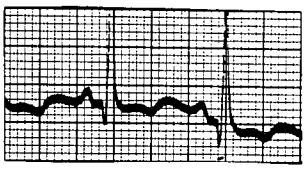


rest ST: 0.1 mv. 3 min. ST: -0.1 mv.

12. D.M. T Inversion.

lead 3

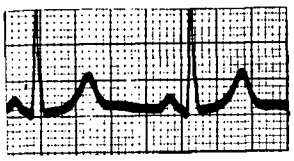


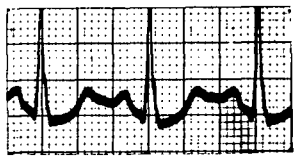


rest T: 0.05 mv. 6 min. T: -0.1 mv.

13. D.J. ST Depression.

lead 2

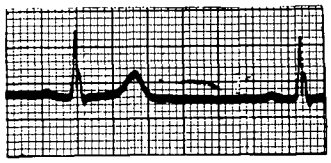


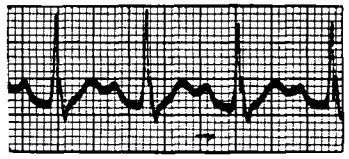


rest ST: iso-el 6 min. ST: -0.1 mv.

14. B.C. PR Prolongation. P Wave Increase.

lead aVF

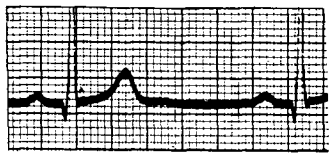


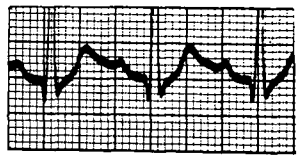


rest PR: 0.16 sec. 3 min. PR: 0.18 sec.

15. B. Sl. QRS Segment Prolongation.

lead 2

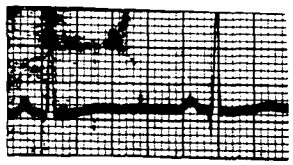


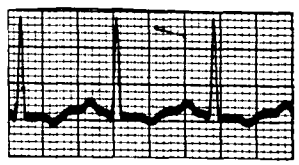


rest QRS: 0.08 sec. 3 min. QRS: 0.1 sec.

16. W.M. T Inversion.

lead 3

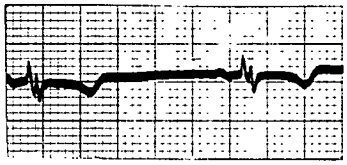


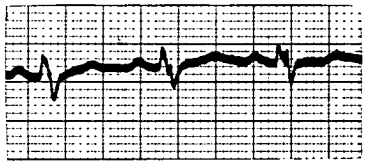


rest T: iso-el 12 min. T: -0.1 mv.

17. M.H. Change of Sign in T Wave.

lead 3

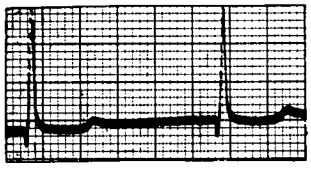


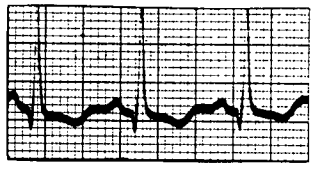


rest T: -0.1 mv. 3 min. T: 0.1 mv.

18. M.S. T Inversion.

lead 3

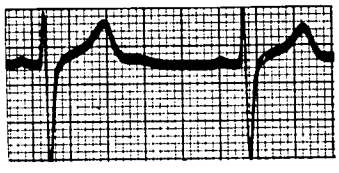


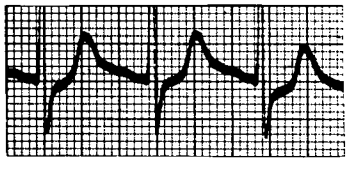


rest T: 0.1 mv. 12 min. T: -0.1 mv.

19. H.S. ST Depression.

lead V2

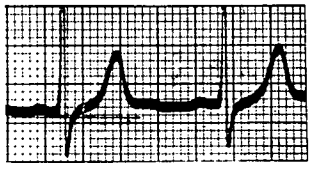


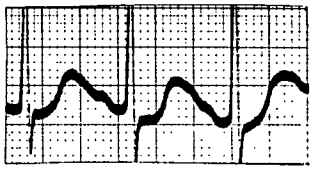


rest ST: iso-el 6 min. ST: -0.1 mv.

20. J.J. ST Depression.

lead V4





rest ST: iso-el 3 min. ST: -0.15 mv.