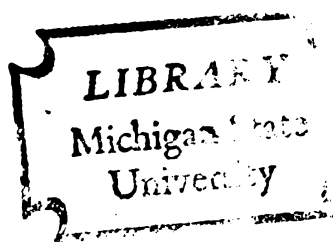


CARDIAC HYPERTROPHY IN POSTPUBERTAL
LABORATORY RATS AFTER CONTROLLED RUNNING
EXERCISE

Thesis for the Degree of M. A.
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THESIS



ABSTRACT

CARDIAC HYPERTROPHY IN POSTPUBERTAL LABORATORY RATS AFTER CONTROLLED RUNNING EXERCISE

By

Kwok-Wai Ho

This study was designed to obtain in vitro anthropometric measurements of the cardiac muscle in postpubertal rats after different intensities of interval training.

Thirty-two male albino rats (Spartan Sprague-Dawley strain) were divided randomly into four equal groups, three exercise groups, each of which was subject to a different interval training program, and one control group. The animals were 71 days old at the start of the experiment.

For two weeks prior to the initiation of the study, the treatment animals were housed in spontaneous activity cages for foot conditioning and acclimatization to the laboratory. During the study, all the rats were maintained in sedentary cages. The three treatment groups were trained once daily, five days per week (Monday-Friday), for eight weeks. Training was performed in small animal controlled-running wheels.

At the end of the training period, the animals were fasted for twenty-four hours. They then were decapitated and their hearts were fixed in a 10 per cent formaldehyde solution. Afterwards, the ventricular length, the ventricular weight, the atrial weight, and the total heart weight of each animal were determined. A transverse slice of each heart was cut at a standard location and stained with Hematoxylin and Eosin. The slide was projected and the outline of the section was traced. From the tracing, the cross-sectional area, the ventricular chamber size, and the ventricular free wall area of the heart were measured with a planimeter.

The exercised animals had significantly larger heart measurements than the control group in regard to the ventricular length, the total heart weight, the ventricular weight, the atrial weight, and the left and right ventricular chamber area ($P = .10$). Among the exercised groups, the long interval training group had the largest right ventricular chamber area ($P = .10$). Measurements of the left ventricular chamber area showed that the long and the short interval training groups had larger chamber areas than the medium interval training group ($P = .10$). The short and the medium interval training groups had greater total heart weight, ventricular length, and ventricular weight in comparison to the long interval training group ($P = .10$). No

significant differences in the above mentioned variables were found between the short and the medium interval training groups.

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CHAPTER I

INTRODUCTION

It is well known that vigorous physical exercise over an effective period may produce cardiac hypertrophy both in animal and human subjects. With hypertrophy, the heart enlarges its dimensions and its weight increases. The muscle fibers of the heart probably remain unchanged in quantity, but increase in diameter and length by an increase in the number of sarcomeres. The detailed biochemical process of cardiac hypertrophy is unknown. It is believed that the hypertrophy develops as a result of an increase in nucleic acids and protein synthesis, a process stimulated by elevated ATP (adenosine triphosphate) metabolism.

If one considers the heart as a muscular pump, its size and shape must affect the mechanics of its operation. There is no doubt that the hypertrophied athlete's heart is highly efficient. However, research in intact organisms on the adaptation of the cardiovascular system to muscular activity is limited by the techniques that can be used without impairing the subject's (particularly in

man) health or performance. An additional problem arises when the researcher attempts to control accurately different intensities of exercise in animal training. The interval training wheel for rats is designed to solve this problem by carefully controlling different intensities of running exercise for rats, as well as varying rest periods during the animals' interval running.

This study was conducted as an attempt to measure, in vitro, the anthropometric changes of the cardiac muscle in postpubertal rats following different intensities of interval training for eight weeks.

CHAPTER II

REVIEW OF LITERATURE

The most important phenomenon in cardiovascular adjustment during muscular activity is the increased blood flow to the exercising muscles. The increased flow is needed for several reasons: to supply more nutrients to the muscles as sources of energy for contraction, to remove waste products which otherwise would accumulate rapidly and impair the function of the muscle, and to permit a greater dissipation of heat produced by muscular activity. If circulation to the muscles does not increase, muscular contraction cannot be sustained for any significant length of time, and consequently exercise must stop. This increased cardiac output is achieved by an increase both in the number of heart beats per minute and in the volume of blood pumped with each beat (stroke volume). Therefore, repeated muscular activities increase work-loads both in ventricles and atria, and vigorous muscular activities over an effective period are believed to constitute the stimuli for the so-called physiological hypertrophy of the cardiac muscle in healthy individuals or animals.

As early as 1628, Harvery stated "the stronger, more muscular, and more substantial the build of men, thicker, heavier, more powerful and fibrous the heart, and the auricles and arteries are proportionally increased in thickness, strength, and all other respects" (37, p. 132).

Early observations of Bergmann (1884), Parrot (1893), Grober (1908), and Hesse (1921) showed that the size of the heart of an animal would reflect its degree of activity. Wild animals possess relatively larger hearts than domesticated animals, and birds which fly great distances or which fly clumsily with more wing actions have unusually large hearts (25, 77).

Dietlen and Schieffer reported that hard military work would cause the heart to increase in size (22, 25).

In 1896 before the use of X-ray, Henschen carried out his measurements by percussion and found an enlargement in the heart of ski runners (22, 25). Among the early researchers, Schott (1897) was the first to employ X-ray and found an unusual enlargement in athletes' hearts following a wrestling bout; however, inaccuracies were shown in his work (22, 25). The introduction of Moritz' (1908) orthodiagraph enabled Schieffer (1916) and Diethen (1919) to improve the accuracy of the observations. They reported that the area of the heart in habitual cyclists was greater than that in occasional cyclists or non-cyclists of the same body size and age (22, 25, 77).

Since, a lot of researches have been conducted to relate muscular activities with heart size. However, there is no agreement between the experimental results. The observations of Lee (1917) on Harvard oarsmen, of Cohn (1920) on returned soldiers, of Farrell (1929) on American trans-continental runners, of Eyster (1930) on athletes with prolonged athletic history, and Keys (1938) on college athletes showed that athletes and heavy workers have the same heart size as normal individuals of the same size and age (21, 27, 30, 53, 58). But many other researchers have found that vigorous athletic activities and hard physical labor produce cardiac muscle hypertrophy in men and animals (8, 22, 23, 24, 25, 38, 39, 41, 47, 48, 49, 59, 60, 77, 78, 82, 84, 85, 93).

Herrmann (1926) weighed the hearts of dogs and found that the average heart-weight ratio (per kilogram of body weight) was 7.98 gm/kg for normal mongrels, 13.4 for ten racing greyhounds, and 17.3 for the best racing greyhounds. He therefore concluded that heart hypertrophy, produced by exercise, is related to the degree of the exercise (39). X-ray studies of athletes' hearts usually have involved measuring the transverse diameter of the heart shadow. After examining several thousand athletes in various sports, Deutsch and Kauf reported that the athletes who participated in rowing, ski-running, and cycling had the largest hearts. They concluded that

the psychic strain and the excitement of certain sports could cause an enlargement of the heart (25). Hodges and Eyster reported that the transverse diameter of the heart had a higher correlation (.5738) with body weight than with age (.2371) or heart (.2140) (40).

Assuming that the heart was a sphere with radius equal to one-half of the transverse diameter of the heart shadow, Herxheimer calculated the heart volume to weight ratio of hundreds of top athletes (Appendix E) and concluded that exercises of strength, speed, or maximal effort will induce hypertrophy of skeletal muscle, but will only increase the heart mass to a small extent. However, exercises of vigorous endurance will lead to heart hypertrophy but will leave the body muscles unchanged in size (22, 25). Herxheimer's theory was emphasized by Steinhaus and was confirmed recently by Nocher and Reindell (64).

In general, cardiac enlargement (hypertrophy and dilation) appears first in the left heart and then in the right heart (59). In 1923 Herxheimer claimed that the heart would enlarge symmetrically on both sides of its vertical bisector and 1:2.2 was the ratio given of the transverse diameter of the right to the left segment of the heart after the enlargement (22). In 1919 under experimental conditions, Hiramatsu was the first to find hypertrophy in the right side of the heart (25).

Cureton published an extensive study of athletes' hearts in 1951 (22). He reported that track men who competed in long sprints (200, 400, and 800m) and swimmers who competed in the 200m and the 800m relay usually had a larger proportionate enlargement in the right transverse diameter. This is believed to be the result of the larger effort exerted by the right ventricle in ejecting blood into the lungs at times when the chest was constricted. Long distance runners generally had enlargement on the left side and would breath more easily and fully than the sprinters (22).

Other studies showed that hypertrophy developed in animals when they were subjected to induced anoxemia in a decompression chamber (80, 81, 83, 84, 86), and in men living at high altitudes (51, 70). The hypertrophy involved mainly the right ventricle and was produced presumably by the increased work load imposed by the pulmonary hypertension associated with hypoxia (81, 84). It was also shown that comparable anoxemia might result in long sprints, especially in swimming 200m or running 400m (22, 23). Thus, it is obvious that enlargement of the heart depends on the intensity of training, the type of training, and the duration of training. The sports which make the greatest demands on the circulatory system produce the greatest cardiac hypertrophy.

When hypertrophy develops, the number of fibers (nuclei) remains unchanged, but their diameter increases,

and the fibers lengthen by an increase in number of sarcomers (60). This is believed to be the result of an increase in nucleic acids and protein synthesis, a process stimulated by stronger ATP (adenosine triphosphate) metabolism. However, the obvious factor stimulating the development of myocardium hypertrophy is anaerobic ATP resynthesis occurring when the oxidizing phosphorylation of ATP directly corresponds to the needs of the functioning heart (59). In this so-called "physiologic" hypertrophy, which occurs in athletes or hard laborers, the weight of the heart rarely exceeds 500 gm, which has been designated as the critical heart weight by Linzbach (60).

However, when the heart is stimulated to do heavier work for a long time period by some pathological mechanism such as hypertension or aortic valvular disease, it's weight will exceed this physiologic limit and may reach 1,000 gm or more. If the hypertrophy is associated with a normal ventricular chamber and residual blood volume, it is described as concentric and occurs in the early stages of chronic pressure load; e.g., valvular stenosis or arterial hypertension. If the hypertrophy is associated with a ventricular chamber and residual blood volume larger than normal as in valvular regurgitation or myocardial disease, it is described as eccentric. Both concentric and eccentric hypertrophy are generally referred to as pathologic hypertrophy. In concentric hypertrophy, contrary to the classical concept,

there is an absolute increase in the number of fibers (more nuclei), which are only moderately thickened. Apparently, the increase in the number of muscle fibers is due to the longitudinal cleavage between points of anastomosis of the myocardial syncytium. In eccentric hypertrophy, the cardiac chamber is dilated with destruction and fibrosis of some muscle fibers. The surviving fibers undergo splitting and rearrangement, as in the concentric type, but attain a diameter greater than that of the concentric type when hearts of equal weight are compared (6, 60).

Experimental studies of the behavior and basic properties of hypertrophied hearts are few (34, 50). Experimental heart hypertrophy is usually produced by increasing the resistance to the ventricular ejection; e.g., increasing the resistance in the aorta or pulmonary artery. The response is characterized by a marked increase in the contractile function of the myocardium (estimated from the tension developed) with respect to the increase in the resistance (62).

Dieckhoff (1936) used the heart-lung preparation to study hypertrophied cat hearts and found a higher arterial pressure and cardiac output in them than in normal cat hearts (6, 50). Beznak also reported a higher cardiac output (by the direct Fick-method) in hypertrophied rat hearts when the rats were at rest.

However, he found no difference in the infusion rate between hypertrophied and normal rat hearts (9).

Geha, Kerr, Whitehorn and their co-workers claimed that hypertrophied heart muscle was capable of better performance than nonhypertrophied heart muscle (33, 50, 92). For given diastolic lengths, they found that a greater tension developed in the columnar carneae of the left ventricle of rat hearts which had been hypertrophied from repeated swimming to exhaustion (92). The maximal tension (per unit weight of papillary muscle) was also found to be significantly greater in the hypertrophied left ventricle of rats (50). In dogs, it was found that the hypertrophied right ventricle performed more work (per unit gross mass of the myocardium) than the normal right ventricle (33).

However, Grimm and his associates found no differences between hypertrophied and normal rat papillary muscle in tension production (per unit weight), water content, total protein concentration, or actomyosin concentration (35). Sandler and Dodge, who calculated the tension and stress of the inner wall of the left ventricle during a cardiac cycle by measuring the pressure, the dimensions, and the wall thickness of the ventricle from biplane angiocardiograms, also noted that the force per unit area of the ventricular wall in subjects with left cardiac hypertrophy was not different from those with normal left ventricles (73).

Another study reported that the oxygen consumption per minute per unit mass of the hypertrophied left ventricle was within the range of normal values, but the data did not permit the calculation of myocardial oxygen uptake per stroke because heart rates were not recorded (11). However, in dogs, the data of West indicated no difference in oxygen consumption per stroke per unit mass of tissue between normal and hypertensive animals (91). From these data, one might expect that the hypertrophied fibers of the heart would develop greater contractile force because of the increase in mass of the contractile tissues but not because of the increased functional capacity of the cellular elements. This was believed by Badeer (6) and may fall in the "second stage" of the "complex and wear" hypothesis, which states that the process of cardiac hypertrophy occurs in three stages. This hypothesis was proposed by Meerson in 1965 (62).

Cardiac performance is usually investigated by considering the size and shape of the heart which is regarded either as a pump made of muscle or as a muscle acting as a pump. Woods applied Laplace's law to evaluate the heart-wall tension in 1892 (94). Burch assumed a spherical shape for the left ventricle and assigned different volumes to demonstrate changes in the ventricular-wall tension (15, 16, 17). Burton and others also investigated the problem in the same manner (7, 18, 73).

In general, Laplace's law applies to a strained "membrane" separating two spaces of any shape. It states that if a slit is cut in the membrane, the two edges of the slit will be pulled apart with a force proportional to the length of the slit. The wall of the ventricle is a strained "membrane" when the heart contracts; so, a slit in the ventricular wall could be pulled apart. There is also a pressure gradient existing between the inner and outer ventricular wall. The equation is:

$$P = T \left(\frac{1}{R_1} + \frac{1}{R_2} \right) \quad (A)$$

Where P is the ventricular systolic blood pressure; T is the ventricular tension; and R_1 and R_2 are the "principal radii of curvature" at any point on the ventricular wall.

For the special case of a spherical ventricular wall, where the radii of curvature are equal ($R_1 = R_2$), the general equation becomes:

$$P = \frac{2T}{R} \quad (B)$$

Woods assumed that the thickness of the ventricular wall (t) at any point was proportional to the tension developed in the wall during systole; i.e.,

$$t = KT \quad (C)$$

where K is a constant and is equal to the tension in a unit thickness of the wall. Substituting (C) into (B), one obtains:

$$P = \frac{2KT}{R} \quad (18, 94) \quad (D)$$

According to Badeer, the force of contraction of the ventricular wall is defined as the integral of the force developed in the myocardium by a unit length of the circumference and the entire thickness of the wall of a given chamber over the whole period of systole. The mural force during systole is:

$$\text{Mural force} = \bar{T}S = \frac{\bar{P} \cdot \bar{R}}{2} \quad (\#)$$

where:

$\bar{T}$ is the mean force per unit cross-sectional area of the wall,

S is the mean thickness of the wall,

$\bar{P}$ is the mean transmural pressure, and

$\bar{R}$ is the mean radius, during systole.

When hypertrophy develops, the thickness of the chamber wall increases. If the tension in the heart muscle remains unchanged, any increase in the mural force will be due to an increase in the thickness (S) rather than to an increase in the mean force per unit cross-sectional area of the wall (6).

Whether or not the hypertrophied heart will produce a greater tension per unit mass of its muscle than the normal heart is still unknown. However, there is no doubt that the hypertrophied athlete's heart is highly efficient. Trained athletes with heart beats over 200 per minute, systolic pressures up to 240 mm Hg without any disorders in the coronary blood circulation, stroke volumes as large as 200 ml and minute output as much as 35 liters have been recorded. Astrand has reported a maximum oxygen absorption of $5,800 \text{ cm}^3$ in a well-trained athlete working under a 400-Watt load. The heart rate of a well-trained athlete can reach its maximum value within 5 to 8 seconds after the beginning of work, while in untrained subjects maximum rate occurs only after 30 to 40 seconds. Towards the end of the first second of work the trained athlete's heart rate reaches 75 per cent of its eventual maximum rate (59).

It is believed that the increased residual volume of the left ventricle is secondary to physiologic hypertrophy, in that it provides for an immediate increase in circulating blood when the athlete is put under stress. Therefore, the increase in the residual volume is not a sign of cardiac weakness (59, 60). Nocker, Reindell and Kenl showed that about one-third of the heart's energy is derived by oxidizing lactic acid when the subject is at rest. This fraction, however, can run up to 40, 61, and 56 per cent when the subject is subjected

to moderate work, strenuous work, and recovery respectively. Since the amount of lactate consumption depends on the mass of the heart muscle, the larger heart of the distance runner, by its increase in bulk, further contributes relief to the metabolic machinery by burning off the acid metabolites produced by the muscles (64).

CHAPTER III

METHODS

Thirty-two male albino rats (Spartan Sprague-Dawley strain) were randomly divided into four equal groups at 71 days of age:

1. Group S--Short-duration (high-intensity endurance) training group.
2. Group M--Medium-duration (moderate-intensity endurance) training group.
3. Group L--Long-duration (low-intensity endurance) training group.
4. Group C--Control (sedentary) group.

For two weeks prior to the initiation of the study, the treatment animals were housed in spontaneous activity cages for foot conditioning and acclimatization to the laboratory. During the study, all of the animals were maintained in sedentary cages which offered no opportunity for exercise. Water and Wayne Lab Blocks were available ad libitum. The cage quarters and training facilities were kept at a constant temperature of 72°F; however, no attempt was made to control the humidity.

The treatment animals were trained in small animal controlled-running wheels once daily, five days per week (Monday-Friday), for eight weeks. The animals learned to run by avoidance-response-operant-conditioning. The controlled-running wheels and interval training programs were developed by the Human Energy Research Laboratory at Michigan State University.

Groups S, M, and L were trained with different training programs. A detailed breakdown of the training data is shown in Appendices A, B, and C. The performance data of each animal were recorded daily. Several animals were destroyed due to leg injury during training. The lighting in the training room was maintained at a dim level so that the light in each wheel (the conditioning stimulus) would have a more dramatic effect. All unnecessary noise was avoided during the daily training bouts.

When the eight-week training period was completed, the animals in all groups were fasted for twenty-four hours. After the twenty-four-hour fast, the animals were weighed and then decapitated. The heart with a small stem of the aorta still attached was quickly removed. The other great vessels were roughly trimmed away. The blood was expelled from the chambers, and the heart was washed free of blood with distilled water. Each heart then was suspended by a thread from the aortic stem and fixed in a 10 per cent formaldehyde solution. After the fixation period of four days, the great vessels of each heart were

carefully trimmed. The surface of the heart was flushed, and the atria were removed as cleanly as possible by careful dissection along the atrioventricular groove. The length of the ventricle from the apex to the entrance of the pulmonary artery was measured by calipers. The heart was transversely dissected along a line which was 55 per cent of the ventricular length from the apex. All measurements and dissections were performed on the ventral aspect of the heart.

The heart sections were blotted dry, and the total heart weight was determined. Then, the atriums were removed from the balance and total ventricular weight was determined. The total atrial weight was obtained by subtraction.

The sections containing the apex of the heart were then dehydrated in graded concentrations of alcohol. Following dehydration, they were embedded in paraffin and sectioned on a microtome at eight microns per slice starting from the end opposite to the apex. The first ten slices of each heart were discarded while the eleventh slice was stained with Hematoxylin and Eosin.

After staining, the slides were projected and magnified approximately 10.76 times on a solid background, 46 inches away, by a Sawyer 500R slide projector. The outline of each cross section was traced and the respective areas were measured by planimeter. Figure 1 shows how the various ventricular areas were arbitrarily

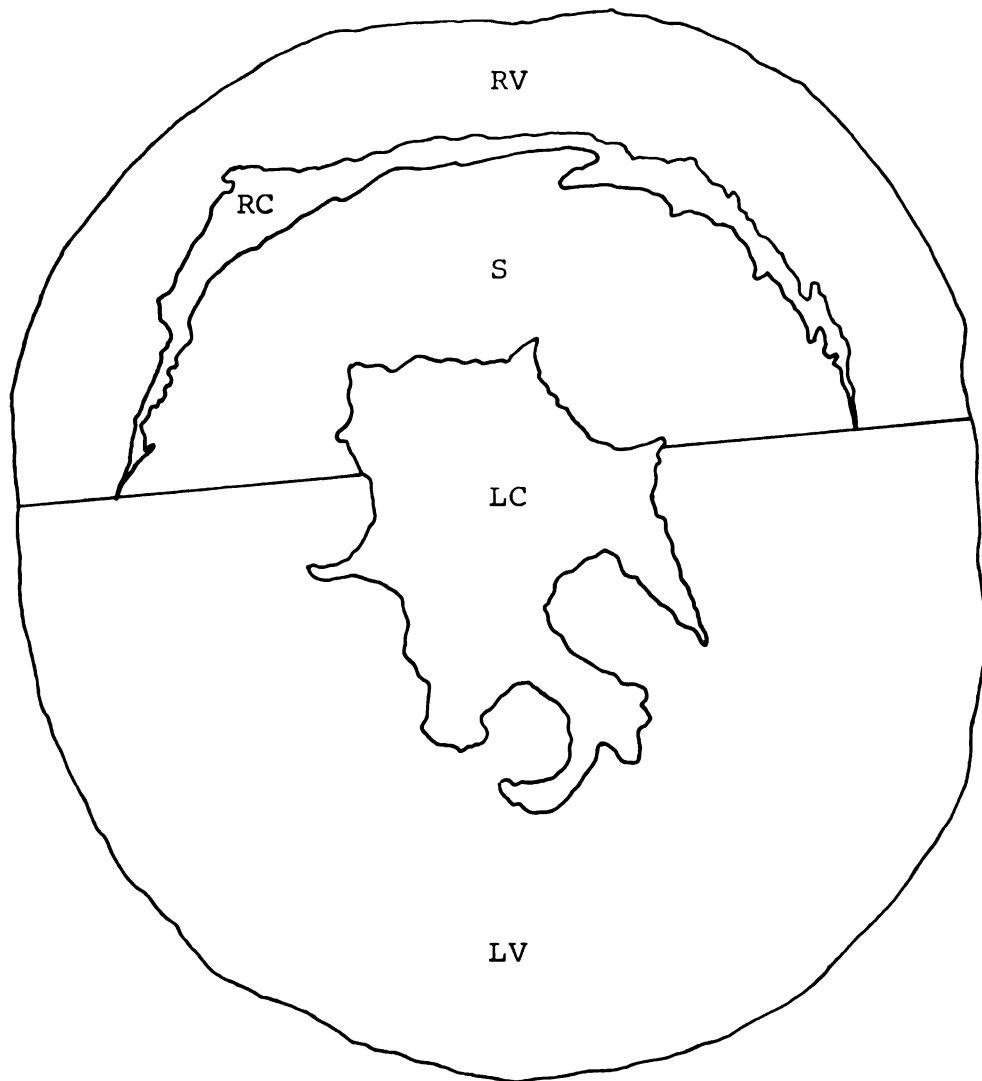


Figure 1.--Sample of the Projected Cross Section of Animal No. S-1.

RV = Right Ventricular Free Wall Area
RC = Right Ventricular Chamber Area
S = Septum Area
LC = Left Ventricular Chamber Area
LV = Left Ventricular Free Wall Area

separated for measurement purposes by an extended straight line drawn through the two extremities of the right ventricular chamber. Although in this way the left ventricular free wall and the septum would seem to be separated, they were considered and measured as one area (the left ventricle-septum area) as there is no anatomical evidence for such a line of demarcation (32, 52). Fulton and his associates have emphasized the usefulness of the ratio of the left ventricular free wall plus septum divided by right ventricular free wall ($LV + S/RV$), for the septum increases in proportion to the increase in the weight of the free wall of either ventricle (32, 52).

The method of separation used in this study provides technical simplification and creates no ambiguities when different ratios are compared, for the technique yields quite uniform results (32, 52).

Mean values of the data for each treatment group were calculated. Analysis of variance was used to analyze the data. Student-Newman-Keuls statistical technique was used to compare the differences between individual groups due to training. The probability of making a type I error was held to the .10 level for this investigation; however, due to the small sample size the same observation was made whenever $P \leq .25$. All raw data are given in Appendix D.

CHAPTER IV

RESULTS

The measurement of ventricular volume has long been a technical problem. Many efforts have been made since the discovery of the X-ray, but none have been satisfactory to all investigators (75). Such a technique is extremely important, for without it one cannot determine the overall power and performance of the heart muscle. Indeed, Starling concluded that when the heart was free from its hormonal and nervous influences, its energy during systole was directly proportional to the diastolic volume (76).

The data of the present study agree with the general opinion that vigorous physical exercise over an effective period produces cardiac hypertrophy. The data in Table 1 show that in general hypertrophied hearts increase in weight, length, total cross-sectional area, and both left and right ventricular chamber areas.

The total heart weight of all exercised animals, both on an absolute and a relative basis, was 22 per cent greater than that of the controls. Such a difference,

TABLE 1.--Table of mean values for all groups.

Groups	Ventricle Length	Total Heart Weight	Ventricle Weights	Atria Weight	Total Heart Weight $\times 10^{-3}$	Ventricle Weights $\times 10^{-3}$	Atria Weights $\times 10^{-3}$
S	1.894 cms.	1.616 gms.	1.454 gms.	.162 gms.	4.021	3.618	.403
M	1.887	1.632	1.467	.165	4.059	3.648	.410
L	1.862	1.565	1.403	.162	4.002	3.587	.414
C	1.734	1.395	1.274	.121	3.292	3.006	.286
F	12.580*	21.351*	7.323*	15.421*	23.225*	20.009*	20.076*
S.N.K. Test	C < S	C < S	C < S	C < S	C < S	C < S	C < S
	C < M	C < M	C < M	C < M	C < M	C < M	C < M
	C < L	C < L	C < L	C < L	C < L	C < L	C < L
	L < S	L < S	L < S				
	L < M	L < M	L < M				

*P < .10.

S.N.K. Test = Student-Newman-Keuls Test.

TABLE 1 (cont'd).

Groups	Total Section Area	Left Chamber Area	Right Chamber Area	L & R Ventri- cles and Septal Area (Total Section Minus L & R Chamber Areas)	Right Ventricle Area	L Ventricle and Septum Area (L & R Ventricle Area Minus R Ventricle Area	L Ventricle and Septum Area R Ventricle Area
S(Short Duration)	138.0 cm ³ s.	14.1 cm ³ s.	6.8 cm ³ s.	117.1 cm ³ s.	23.2 cm ³ s.	93.9 cm ³ s.	4.0796
M(Medium Duration)	137.3	11.6	6.3	119.5	23.9	95.6	4.3326
L(Long Duration)	137.8	13.5	8.2	116.1	25.2	91.0	3.6129
C(Control)	127.0	8.9	4.7	113.4	24.8	88.6	3.5847
F	1.9452#	2.5008*	2.7634*	0.4934	1.0280	0.9671	2.1366#
S.N.K. Test		C < S C < M C < L M < S M < L	C < S C < M C < L M < L S < L				

*P < .10.

#P < .25 but P > .10.

S.N.K. Test = Student-Newman-Keuls Test.

however, is far less than those which have been found in some pathologically enlarged human hearts. If a general comparison can be made between rats and men, this difference would fall below the "critical heart weight" of Linzbach (60).

The cross-sectional areas of the hearts of the exercised animals also show that enlargements of the left and right ventricular chamber areas are associated with increased total heart weight or ventricle weight. These cross-sectional differences do not occur in concentric or eccentric types of pathological hypertrophy.

Among the exercised groups, the long-duration running group had significantly smaller absolute ventricular length, total heart weight, and ventricular weight than either the medium- or the short-duration running groups ($P \leq .10$) (Table 1). This group also had a significantly larger mean right ventricular chamber area ($P \leq .10$) and a somewhat lower ratio of left ventricular free wall plus septum area divided by right ventricular free wall ($P \leq .25$). Thus, it may be hypothesized from the data of this study that there may have been a tendency toward right ventricle domination in the long-duration running group.

The exercised animals showed significantly different values from those of the control group in regard to the ventricular length, the total heart weight, the ventricular weight, the atrial weight, and the left and

right ventricular chamber area ($P = .10$). Among the exercised groups, the long interval-training group had a larger mean right ventricular chamber than either the short or the medium group ($P = .10$). Measurements of the left ventricular chamber showed that the long and short groups had larger chambers than the medium interval-training group ($P = .10$). Both the short and the medium interval-training groups had greater total heart weight, ventricle length, and ventricle weight than the long training group ($P = .10$).

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This study was designed to obtain in vitro anthropometric measurements of the cardiac muscle in postpubertal rats after different intensities of interval training.

Thirty-two male albino rats (Spartan Sprague-Dawley strain) were divided randomly into four equal groups--three exercise groups, each of which was subject to a different interval training program, and one control group. The animals were 71 days old at the start of the experiment.

For two weeks prior to the initiation of the study, the treatment animals were housed in spontaneous activity cages for foot conditioning and acclimatization to the laboratory. During the study, all the rats were maintained in sedentary cages. The three treatment groups were trained once daily, five days per week (Monday-Friday), for eight weeks. Training was performed in small animal controlled running wheels.

At the end of the training period, the animals were fasted for twenty-four hours. They then were decapitated and their hearts were fixed in a 10 per cent formaldehyde solution. Afterwards, the ventricular length, the ventricular weight, the atrial weight, and the total heart weight of each animal were determined. A transverse slice of each heart was cut at a standard location and stained with hematoxylin and eosin. The slide was projected and the outline of the section was traced. From the tracing, the cross-sectional area, the ventricular chamber size, and the ventricular free wall area of the heart were measured with a planimeter.

Mean values of the data for each treatment group were calculated. Analysis of variance was used to analyze the data. Student-Newman-Keuls statistical technique was used to compare the differences between individual groups due to training. The probability of making a type I error was held to the .10 level for this investigation; however, due to the small sample size the same observation was made whenever $P \leq .25$.

Conclusions

1. The results of this study indicate that vigorous physical exercise over an eight-week period produces cardiac hypertrophy in postpubertal laboratory rats.

2. The hypertrophied hearts increase in weight, length, total cross-sectional area, and both left and right ventricular chamber areas.
3. Among the exercised groups, the long-duration running group had significantly smaller absolute ventricular length, total heart weight, and ventricular weight than either the medium- or the short-duration running group. This group also had a significantly larger mean right ventricular chamber area and a somewhat lower ratio of the left ventricular free wall plus septum area divided by right ventricular free wall.

Recommendations

1. The physiological and mechanical details pertaining to running in rats are unknown. It will be necessary, therefore, to use a variety of different training programs in order to better define aerobic and anaerobic types of exercise for rats. Such definition is needed in order to compare the effects of different exercise programs on cardiac hypertrophy.
2. Experience has shown that the ability of rats to learn to run in a forced-exercise program and their performance while running on a

particular program may be strain specific. Learning and training programs developed for a particular strain may not be suitable for another strain of rats. The researcher should bear this in mind if replication is attempted.

3. The method used to dissect the ventricles transversely was not ideal due to errors in sectioning. Thus, measurement on the total cross-sectional area may not have been absolutely accurate. A better method must be sought.
4. In addition, only twenty slides were satisfactory and complete enough to illustrate the total cross-sectional area from the total of twenty-seven hearts which were sectioned and stained. Therefore, modification of the histology technique is needed.

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APPENDICES

APPENDIX A

STANDARD EIGHT-WEEK, SHORT-DURATION, HIGH-
INTENSITY ENDURANCE TRAINING PROGRAM FOR
POSTPUBERTAL AND ADULT MALE RATS IN
CONTROLLED-RUNNING WHEELS

TABLE A-1.--Standard eight-week, short-duration, high-intensity endurance training program for postpubertal and adult male rats in controlled-running wheels.

Wk.	Day of Wk.	Day of Tr.	Acceleration Time (sec)	Work Time (min:sec)	Rest Time (sec)	Repetitions per Bout	No. of Bouts	Time Between Bouts (min)	Shock (ma)	Run Speed (ft/sec)	Total Time of Prog. (min:sec)	Total Exp. Revolutions TER	Total Work Time (sec) TWT
1	1=M	1	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	2=T	2	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	3=W	3	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	4=T	4	1.0	00:10	10	40	3	5.0	1.2	2.5	39:45	750	1200
	5=F	5	1.0	00:10	10	40	3	5.0	1.2	2.5	39:45	750	1200
2	1=M	6	1.0	00:10	10	40	3	5.0	1.2	2.5	39:45	750	1200
	2=T	7	1.0	00:10	10	40	3	5.0	1.2	3.0	49:30	900	1200
	3=W	8	1.0	00:10	10	40	3	5.0	1.2	3.0	49:30	900	1200
	4=T	9	1.0	00:10	10	40	3	5.0	1.2	3.0	49:30	900	1200
	5=F	10	1.0	00:10	10	40	3	5.0	1.2	3.0	49:30	900	1200
3	1=M	11	1.0	00:10	10	40	3	5.0	1.2	3.0	49:30	900	1200
	2=T	12	1.5	00:10	15	40	3	5.0	1.0	3.5	59:15	1050	1200
	3=W	13	1.5	00:10	15	40	3	5.0	1.0	3.5	59:15	1050	1200
	4=T	14	1.5	00:10	15	40	3	5.0	1.0	3.5	59:15	1050	1200
	5=F	15	1.5	00:10	15	40	3	5.0	1.0	3.5	59:15	1050	1200
4	1=M	16	1.5	00:10	15	40	3	5.0	1.0	3.5	59:15	1050	1200
	2=T	17	1.5	00:10	20	34	3	5.0	1.0	4.0	60:00	1020	1020
	3=W	18	1.5	00:10	20	34	3	5.0	1.0	4.0	60:00	1020	1020
	4=T	19	1.5	00:10	20	34	3	5.0	1.0	4.0	60:00	1020	1020
	5=F	20	1.5	00:10	20	34	3	5.0	1.0	4.0	60:00	1020	1020
5	1=M	21	1.5	00:10	20	34	3	5.0	1.0	4.0	60:00	1020	1020
	2=T	22	1.5	00:10	25	22	4	2.5	1.0	4.5	57:10	990	880
	3=W	23	1.5	00:10	25	22	4	2.5	1.0	4.5	57:10	990	880
	4=T	24	1.5	00:10	25	22	4	2.5	1.0	4.5	57:10	990	880
	5=F	25	1.5	00:10	25	22	4	2.5	1.0	4.5	57:10	990	880
6	1=M	26	1.5	00:10	25	22	4	2.5	1.0	4.5	57:10	990	880
	2=T	27	1.5	00:10	30	14	5	2.5	0.8	5.0	54:10	875	700
	3=W	28	1.5	00:10	30	14	5	2.5	0.8	5.0	54:10	875	700
	4=T	29	1.5	00:10	30	14	5	2.5	0.8	5.0	54:10	875	700
	5=F	30	1.5	00:10	30	14	5	2.5	0.8	5.0	54:10	875	700
7	1=M	31	1.5	00:10	30	14	5	2.5	0.8	5.0	54:10	875	700
	2=T	32	2.0	00:10	35	10	6	2.5	0.8	5.5	54:00	825	600
	3=W	33	2.0	00:10	35	10	6	2.5	0.8	5.5	54:00	825	600
	4=T	34	2.0	00:10	35	10	6	2.5	0.8	5.5	54:00	825	600
	5=F	35	2.0	00:10	35	10	6	2.5	0.8	5.5	54:00	825	600
8	1=M	36	2.0	00:10	35	10	6	2.5	0.8	5.5	54:00	825	600
	2=T	37	2.0	00:10	40	7	7	2.5	0.8	6.0	51:10	735	490
	3=W	38	2.0	00:10	40	7	7	2.5	0.8	6.0	51:10	735	490
	4=T	39	2.0	00:10	40	7	7	2.5	0.8	6.0	51:10	735	490
	5=F	40	2.0	00:10	40	7	7	2.5	0.8	6.0	51:10	735	490

APPENDIX B

STANDARD EIGHT-WEEK, MEDIUM-DURATION, MODERATE-
INTENSITY ENDURANCE TRAINING PROGRAM FOR
POSTPUBERTAL AND ADULT MALE RATS IN
CONTROLLED-RUNNING WHEELS

TABLE B-1.--Standard eight-week, medium-duration, moderate-intensity endurance training program for postpubertal and adult male rats in controlled-running wheels.

Wk.	Day of Wk.	Day of Tr.	Acceleration Time (sec)	Work Time (min:sec)	Rest Time (sec)	Repetitions per Bout	No. of Bouts	Time Between Bouts (min)	Shock (ma)	Run Speed (ft/sec)	Total Time of Prog. (min:sec)	Total Exp. Revolutions TER	Total Work Time (sec) TWT
1	1=M	1	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	2=T	2	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	3=W	3	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	4=T	4	1.0	00:10	10	28	4	5.0	1.2	2.5	51:40	700	1120
	5=F	5	1.0	00:10	10	28	4	5.0	1.2	2.5	51:40	700	1120
2	1=M	6	1.0	00:10	10	28	4	5.0	1.2	2.5	51:40	700	1120
	2=T	7	1.0	00:10	10	27	4	5.0	1.2	3.0	50:20	810	1080
	3=W	8	1.0	00:10	10	27	4	5.0	1.2	3.0	50:20	810	1080
	4=T	9	1.0	00:10	10	27	4	5.0	1.2	3.0	50:20	810	1080
	5=F	10	1.0	00:10	10	27	4	5.0	1.2	3.0	50:20	810	1080
3	1=M	11	1.0	00:10	10	27	4	5.0	1.2	3.0	50:20	810	1080
	2=T	12	1.5	00:10	10	26	4	5.0	1.0	3.5	49:00	910	1040
	3=W	13	1.5	00:10	10	26	4	5.0	1.0	3.5	49:00	910	1040
	4=T	14	1.5	00:10	10	26	4	5.0	1.0	3.5	49:00	910	1040
	5=F	15	1.5	00:10	10	26	4	5.0	1.0	3.5	49:00	910	1040
4	1=M	16	1.5	00:10	10	26	4	5.0	1.0	3.5	49:00	910	1040
	2=T	17	1.5	00:15	15	19	4	5.0	1.0	3.5	52:00	997	1140
	3=W	18	1.5	00:15	15	19	4	5.0	1.0	3.5	52:00	997	1140
	4=T	19	1.5	00:15	15	19	4	5.0	1.0	3.5	52:00	997	1140
	5=F	20	1.5	00:15	15	19	4	5.0	1.0	3.5	52:00	997	1140
5	1=M	21	1.5	00:15	15	19	4	5.0	1.0	3.5	52:00	997	1140
	2=T	22	1.5	00:15	15	14	5	5.0	1.0	4.0	53:45	1050	1050
	3=W	23	1.5	00:15	15	14	5	5.0	1.0	4.0	53:45	1050	1050
	4=T	24	1.5	00:15	15	14	5	5.0	1.0	4.0	53:45	1050	1050
	5=F	25	1.5	00:15	15	14	5	5.0	1.0	4.0	53:45	1050	1050
6	1=M	26	1.5	00:15	15	14	5	5.0	1.0	4.0	53:45	1050	1050
	2=T	27	1.5	00:20	20	11	5	5.0	0.8	4.0	55:00	1100	1100
	3=W	28	1.5	00:20	20	11	5	5.0	0.8	4.0	55:00	1100	1100
	4=T	29	1.5	00:20	20	11	5	5.0	0.8	4.0	55:00	1100	1100
	5=F	30	1.5	00:20	20	11	5	5.0	0.8	4.0	55:00	1100	1100
7	1=M	31	1.5	00:20	20	11	5	5.0	0.8	4.0	55:00	1100	1100
	2=T	32	1.5	00:25	25	9	5	5.0	0.8	4.0	55:25	1125	1125
	3=W	33	1.5	00:25	25	9	5	5.0	0.8	4.0	55:25	1125	1125
	4=T	34	1.5	00:25	25	9	5	5.0	0.8	4.0	55:25	1125	1125
	5=F	35	1.5	00:25	25	9	5	5.0	0.8	4.0	55:25	1125	1125
8	1=M	36	1.5	00:25	25	9	5	5.0	0.8	4.0	55:25	1125	1125
	2=T	37	1.5	00:30	30	8	5	5.0	0.8	4.0	57:30	1200	1200
	3=W	38	1.5	00:30	30	8	5	5.0	0.8	4.0	57:30	1200	1200
	4=T	39	1.5	00:30	30	8	5	5.0	0.8	4.0	57:30	1200	1200
	5=F	40	1.5	00:30	30	8	5	5.0	0.8	4.0	57:30	1200	1200

APPENDIX C

STANDARD EIGHT-WEEK, LONG-DURATION, LOW-INTENSITY
ENDURANCE TRAINING PROGRAM FOR POSTPUBERTAL
AND ADULT MALE RATS IN CONTROLLED-
RUNNING WHEELS

TABLE C-1.--Standard eight-week, long-duration, low-intensity endurance training program for postpubertal and adult male rats in controlled-running wheels.

Wk.	Day of Wk.	Day of Tr.	Acceleration Time (sec)	Work Time (min:sec)	Rest Time (sec)	Repetitions per Bout	No. of Bouts	Time Between Bouts (min)	Shock (ma)	Run Speed (ft/sec)	Total Time of Prog. (min:sec)	Total Exp. Revolutions TER	Total Work Time (sec) TWT
1	1=M	1	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	2=T	2	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	3=W	3	1.0	00:10	10	40	3	5.0	1.2	2.0	39:45	600	1200
	4=T	4	1.0	00:20	10	30	2	5.0	1.2	2.0	34:40	600	1200
	5=F	5	1.0	00:30	15	20	2	5.0	1.2	2.0	34:30	600	1200
2	1=M	6	1.0	00:40	20	15	2	5.0	1.2	2.0	34:20	600	1200
	2=T	7	1.0	00:50	25	12	2	5.0	1.2	2.0	34:10	600	1200
	3=W	8	1.0	01:00	30	10	2	5.0	1.2	2.0	34:00	600	1200
	4=T	9	1.0	02:30	60	4	2	5.0	1.2	2.0	31:00	600	1200
	5=F	10	1.0	02:30	60	4	2	5.0	1.2	2.0	31:00	600	1200
3	1=M	11	1.0	02:30	60	4	2	5.0	1.2	2.0	31:00	600	1200
	2=T	12	1.0	05:00	0	1	5	2.5	1.0	2.0	35:00	750	1500
	3=W	13	1.0	05:00	0	1	5	2.5	1.0	2.0	35:00	750	1500
	4=T	14	1.0	05:00	0	1	5	2.5	1.0	2.0	35:00	750	1500
	5=F	15	1.0	05:00	0	1	5	2.5	1.0	2.0	35:00	750	1500
4	1=M	16	1.0	05:00	0	1	5	2.5	1.0	2.0	35:00	750	1500
	2=T	17	1.0	07:30	0	1	4	2.5	1.0	2.0	37:30	900	1800
	3=W	18	1.0	07:30	0	1	4	2.5	1.0	2.0	37:30	900	1800
	4=T	19	1.0	07:30	0	1	4	2.5	1.0	2.0	37:30	900	1800
	5=F	20	1.0	07:30	0	1	4	2.5	1.0	2.0	37:30	900	1800
5	1=M	21	1.0	07:30	0	1	4	2.5	1.0	2.0	37:30	900	1800
	2=T	22	1.0	07:30	0	1	5	2.5	1.0	2.0	47:30	1125	2250
	3=W	23	1.0	07:30	0	1	5	2.5	1.0	2.0	47:30	1125	2250
	4=T	24	1.0	07:30	0	1	5	2.5	1.0	2.0	47:30	1125	2250
	5=F	25	1.0	07:30	0	1	5	2.5	1.0	2.0	47:30	1125	2250
6	1=M	26	1.0	07:30	0	1	5	2.5	1.0	2.0	47:30	1125	2250
	2=T	27	1.0	10:00	0	1	4	2.5	0.8	2.0	47:30	1200	2400
	3=W	28	1.0	10:00	0	1	4	2.5	0.8	2.0	47:30	1200	2400
	4=T	29	1.0	10:00	0	1	4	2.5	0.8	2.0	47:30	1200	2400
	5=F	30	1.0	10:00	0	1	4	2.5	0.8	2.0	47:30	1200	2400
7	1=M	31	1.0	10:00	0	1	4	2.5	0.8	2.0	47:30	1200	2400
	2=T	32	1.0	10:00	0	1	5	2.5	0.8	2.0	60:00	1500	3000
	3=W	33	1.0	10:00	0	1	5	2.5	0.8	2.0	60:00	1500	3000
	4=T	34	1.0	10:00	0	1	5	2.5	0.8	2.0	60:00	1500	3000
	5=F	35	1.0	10:00	0	1	5	2.5	0.8	2.0	60:00	1500	3000
8	1=M	36	1.0	10:00	0	1	5	2.5	0.8	2.0	60:00	1500	3000
	2=T	37	1.0	12:30	0	1	4	2.5	0.8	2.0	57:30	1500	3000
	3=W	38	1.0	12:30	0	1	4	2.5	0.8	2.0	57:30	1500	3000
	4=T	39	1.0	12:30	0	1	4	2.5	0.8	2.0	57:30	1500	3000
	5=F	40	1.0	12:30	0	1	4	2.5	0.8	2.0	57:30	1500	3000

APPENDIX D

BASIC DATA--ALL ANIMALS

TABLE D-1.--Basic data--all animals.

Animal No.	Body Weight	% of Training Program Completed	Ventricle Length	Total Heart Weight	Ventricle Weight
S-1	408.5 gms.	89%	1.875 cms.	1.615 gms.	1.463 gms.
S-2	408.0	56%	1.946	1.668	1.515
S-3	379.0	84%	1.831	1.495	1.340
S-4	400.4	76%	1.875	1.521	1.370
S-5	414.3	62%	1.850	1.690	1.512
S-6	421.4	80%	1.844	1.638	1.459
S-7	386.3	72%	2.004	1.698	1.528
S-8	398.3	64%	1.925	1.602	1.443
M-9	383.1	84%	1.847	1.555	1.392
M-11	361.4	92%	1.872	1.589	1.427
M-12	437.5	71%	1.905	1.665	1.507
M-15	417.6	70%	1.898	1.632	1.459
M-16	416.7	52%	1.915	1.718	1.551
L-17	406.0	94%	1.880	1.669	1.476
L-18	371.4	95%	1.875	1.578	1.407
L-19	415.0	93%	1.854	1.572	1.416
L-20	382.8	80%	1.819	1.501	1.347
L-21	380.0	73%	1.816	1.563	1.415
L-23	416.4	74%	1.963	1.572	1.410
L-24	369.3	92%	1.826	1.498	1.347
C-26	451.5		1.631	1.364	1.240
C-27	416.4		1.819	1.361	1.247
C-28	444.9		1.800	1.356	1.356
C-29	413.5		1.760	1.480	1.336
C-30	394.0		1.681	1.409	1.295
C-31	428.5		1.737	1.388	1.242
C-32	424.8		1.707	1.407	1.300

TABLE D-1 (cont'd).

Animal No.	Atria Weight	Total Heart Wt. (10^{-3}) $\frac{\text{Body Wt.}}{\text{Body Wt.}}$	Ventri- cle Wt. (10^{-3}) $\frac{\text{Body Wt.}}{\text{Body Wt.}}$	Atria Wt. (10^{-3}) $\frac{\text{Body Wt.}}{\text{Body Wt.}}$	Ventricle Length (10^{-3}) $\frac{\text{Body Wt.}}{\text{Body Wt.}}$
S-1	0.152 gms.	3.953	3.581	0.372	4.590
S-2	0.153	4.088	3.713	0.375	4.770
S-3	0.155	3.945	3.536	0.409	4.831
S-4	0.151	3.799	3.422	0.377	4.683
S-5	0.178	4.079	3.650	0.430	4.465
S-6	0.179	3.887	3.462	0.425	4.376
S-7	0.170	4.396	3.955	0.440	5.188
S-8	0.159	4.022	3.623	0.399	4.833
M-9	0.163	4.059	3.634	0.425	4.821
M-11	0.162	4.397	3.946	0.448	5.180
M-12	0.158	3.806	3.445	0.361	4.354
M-15	0.173	3.908	3.494	0.414	4.545
M-16	0.167	4.123	3.722	0.401	4.596
L-17	0.193	4.111	3.635	0.475	4.631
L-18	0.171	4.249	3.788	0.460	5.048
L-19	0.156	3.788	3.412	0.376	4.467
L-20	0.154	3.921	3.519	0.402	4.752
L-21	0.148	4.113	3.724	0.389	4.779
L-23	0.162	3.775	3.386	0.389	4.714
L-24	0.151	4.056	3.647	0.409	4.944
C-26	0.124	3.021	2.746	0.275	3.612
C-27	0.114	3.268	2.995	0.274	4.368
C-28	0.100	3.048	2.823	0.225	4.046
C-29	0.144	3.579	3.231	0.348	4.256
C-30	0.114	3.576	3.287	0.289	4.266
C-31	0.146	3.239	2.899	0.341	4.054
C-32	0.107	3.312	3.060	0.252	4.018

TABLE D-1 (cont'd).

Animal No.	Total Section Area	Left Chamber Area	Right Chamber Area	L & R Ventricles and Septal Area (Total Section Area--L & R Chamber Area)
S-1	143.3 cm ³ s.	14.7 cm ³ s.	5.4 cm ³ s.	123.2 cm ³ s.
S-2	134.6	14.1	6.7	113.8
S-3	125.3	17.2	5.3	102.8
S-6	149.2	15.6	7.5	126.1
S-7	144.0	11.6	9.3	123.1
S-8	131.8	11.6	6.7	113.5
M-9	123.8	11.8	7.7	104.3
M-11	136.7	11.2	5.5	120.0
M-12	140.4	13.1	7.0	120.3
M-15	133.3	8.6	3.0	121.7
M-16	152.2	13.1	8.1	131.0
L-17	143.1	12.8	8.7	121.6
L-18	138.7	17.5	8.2	113.0
L-20	137.2	9.6	8.4	119.2
L-23	132.0	14.8	7.4	109.8
L-24	137.2	12.9	8.2	117.1
C-27	119.8	9.2	3.5	107.1
C-28	132.4	9.3	6.2	116.9
C-29	131.3	9.5	4.8	117.0
C-32	124.6	7.6	4.4	112.6

TABLE D-1 (cont'd).

Animal No.	Right Ventricle Area	Left Ventricle Area and Septum Area (L & R Ventricle Area and Septum Area - R Ventricle Area)	Left Ventricle Area and Septum Area	
			Septum Area	Right Ventricle Area
S-1	26.1 cm ³ s.	91.7 cm ³ s.	3.7241	
S-2	24.7	89.1	3.6072	
S-3	23.2	79.6	3.4301	
S-6	22.3	103.8	4.6547	
S-7	21.2	101.9	4.8066	
S-8	21.6	91.9	4.2546	
M-9	21.8	82.5	3.7844	
M-11	24.3	95.7	3.9382	
M-12	21.1	99.2	4.7014	
M-15	23.8	97.9	4.1134	
M-16	28.3	102.7	3.6289	
L-17	26.4	95.2	3.6060	
L-18	25.0	88.0	3.5200	
L-20	25.5	93.7	3.6745	
L-23	24.7	85.1	3.4453	
L-24	24.3	92.8	3.8189	
C-27	22.2	84.9	3.8243	
C-28	27.2	89.7	3.2977	
C-29	26.2	90.8	3.4656	
C-32	23.7	88.9	3.7510	

APPENDIX E

HERXHEIMER'S HEART SIZE DATA ON THE
1928 OLYMPIC ATHLETES

TABLE E-1.--Herxheimer's heart size data on the 1928 olympic athletes.

Classification	N	Average Heart	Average Right	Average Left	Average Total
		Volume	Diameter	Diameter	Diameter
<u>1922 Material</u>					
Cross-country skiers	16	1/50.8			
Long-distance runners	12	1/57.2			
Middle-distance runners	19	1/60.3			
Heavy athletes	29	1/63.8			
Swimmers	15	1/63.8			
Decathlon men	17	1/67.2			
Boxers	16	1/72.5			
Six day cyclists	12	1/40.3			
<u>1928 Material</u>					
Marathon and distance runners	45	1/46.1	4.9	9.0	13.9
Long-distance cyclists	14	1/48.7	5.2	9.2	14.4
Oarsmen	26	1/51.7	5.2	9.4	14.6
Boxers	18	1/52.7	4.5	8.9	13.4
Sprint cyclists	16	1/55	4.7	9.2	13.9
Middle-distance runners	24	1/55.1	4.6	8.9	13.5
Weight lifters	28	1/56.8	4.8	9.6	14.4
Long-distance runners	18	1/60.8	4.6	9.3	13.9
Sprinters (track)	33	1/62.3	4.4	8.7	13.1
Decathlon men	22	1/63.3	4.7	9.2	13.9
Sprinters (swimming)	9		4.6	8.7	13.3

*Where the volume/weight ratio using volume equal to a sphere calculated from a radius equal to one-half the transverse diameter of the heart shadow.

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