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The effects of reoccuring starvation and refeeding of a high carbohydrate diet on blood cellular constituents, vectorcardiograms, blood pressures, and femoral artery stretch-tension characteristics in beagles

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INTRODUCTION

Starvation-refeeding has been investigated in the past. Some evidence exists for postulating that prolonged or repeated periods of starvation-refeeding may lead to the development of hypertension. The role of dietary carbohydrates in the development of hypertension has also been conjectured. It was the primary goal of this project to investigate the effect of reoccurring starvation and refeeding of a high carbohydrate diet on the arterial blood pressure of beagle dogs.

Several other cardiovascular measurements were made to investigate the cause of the hypertension should it develop. Mechanical properties of femoral artery segments were determined by in vitro stretch-tension analysis. A segment of femoral artery was removed from each beagle at the beginning of the experiment and its mechanical properties were compared to the mechanical properties of an arterial segment removed from the opposite leg of the same dog at the end of the experiment. The arterial segments from the control group of beagles were used to determine if any aging effects occurred over the course of the experiment. Femoral arterial segments from the treatment group of beagles were used to determine if any mechanical change occurred due to the treatment regimens. This experimental design required that the mechanical properties of femoral artery segments from the right and left side of a normal dog be statistically indistinguishable. This was investigated in normal untreated mongrel dogs. Their mechanical properties were then compared to those of beagles. Vectorcardiograms obtained by using the McFee-Parungao orthogonal lead system and heart rate were also measured.

Because of the anticipated stress of prolonged starvation, monitoring of blood cellular constituents and total plasma protein concentration was warranted to assess the health of the experimental dogs.

REVIEW OF LITERATURE

Cardiovascular Effects of Starvation and Refeeding

Starvation has occurred frequently throughout the history of mankind; usually resulting from a natural disaster which disrupted food supplies or as a result of war. Bloom (1959) more recently advocated the use of acute starvation for the purpose of weight loss in obese humans. He hypothesized that it could be utilized without ill effects. Starvation-refeeding regimens, however, cannot be considered absolutely safe.

Garnett et al. (1969) reported on a 20 year old female patient who reduced her body weight from 118 kg to 60 kg by starving for 30 weeks. Refeeding was initiated with small meals but cardiac arrest occurred on the seventh day. Treatment was successful but ventricular fibrillation on the next day was fatal. Electron microscopy of the heart revealed paucity and disruption of myofibrils. The publishing of the article by Garnett et al. initiated several letters to the editor. Some of the letters further substantiated the previous article by reporting similar cases. One letter refuted the electron microscopic findings as being artifacts because of the elapsed time between death and fixation of the tissue.

Sandhofer et al. (1973) described the development of circulatory shock and an abnormal electrocardiogram during starvation of a patient. This particular woman had lost 48 kg of weight by starving under medical supervision for 18 weeks. The development of the cardiovascular abnormality prompted the end of the fasting and the patient recovered.

The effect of starvation on the cardiovascular system has been studied by several researchers. The most extensive study was done by Keys et al. (1950) during World War II.

Keys et al. (1950) submitted 32 volunteer men to 24 weeks of semi-starvation. The mean pulse rate for 12 of the men decreased from a control value of 56.1 to 37.8 beats per minute. The systolic blood pressure of these same 12 men decreased from the control value of 105.3 mm Hg to 92.7 mm Hg after the semi-starvation. The diastolic blood pressure declined from a control value of 70.2 mm Hg to 63.3 mm Hg. After 20 weeks of refeeding the values for pulse rate and blood pressure were back to normal. The venous blood pressure in the 12 individuals during starvation was below normal. Upon refeeding it rose to normal values and exceeded normal in three of the 12 subjects studied. This elevated venous pressure indicated incipient heart failure. The atrophic heart of starvation was in some cases unable to cope with the sudden increase in metabolic load of refeeding. One subject of the 32 developed transient heart failure upon refeeding after the 24 weeks of semi-starvation.

Friedberg (1966) cites several works concerned with the effects of undernutrition and starvation on the heart. The cardiac weight loss due to myocardial fiber atrophy was proportionate to or slightly less than the reduction in total body weight. A pronounced sinus bradycardia and a decrease in cardiac output and stroke volume caused a prolonged circulation time. Blood pressure and pulse pressure both declined with the systolic pressure being reduced more than the diastolic. The edema seen in starvation was not due to heart failure but was probably related to a reduced osmotic pressure of the blood. Electrocardiograms (ECG) showed reduced

voltage in all leads, right axis deviations, depressed S-T segments, altered T-waves, and prolongation of the PR intervals or QRS durations. These ECG changes were thought to be due in part to deficient potassium and calcium intake.

Consalazio et al. (1967) starved six men for only 2 days and found that the blood pressures decreased and the mean electrical axis of the heart shifted toward the right.

Balasubramanian and Dhalla (1972) measured the levels of norepinephrine in rat hearts before and after starvation. Even though the cardiac concentration of norepinephrine increased during starvation the authors believed that the proportion available for release from storage was reduced. This could reduce the influence of sympathetic activity on the heart. The lack of norepinephrine release in starvation may account for the increased presence of myocardial glycogen.

Ko and Paradise (1972) found no deleterious effect of starvation on the functional status of the isolated rat atria. The authors believed that starvation increased the storage of readily metabolizable substrate in the heart.

The long term effects of starvation-refeeding and the effects of diet only on the cardiovascular system have been studied.

Brozek et al. (1948) reviewed the literature concerning previous periods of drastic food restriction. One such period was the seige of Leningrad during World War II which lasted from September, 1941, until March, 1942. Semi-starvation during the seige was common and from hospital records the number of patients admitted with hypertension declined.

Two years following the seige, with improved availability of food, hypertension became a major medical problem.

The role of nutrition in hypertension was reviewed by Hartroft (1966). He stated, "...various dietary manipulations may alter, modify, initiate, and even reverse hypertension, particularly that of renal origin."

One of the diets used to lower blood pressure was the Kempner rice diet. Watkin et al. (1950) studied the effect of this diet on 50 hospitalized patients. The diet consisted of 250-350 gm of rice per day plus white sugar or dextrose and 700-1000 ml of fruit juice. It provided about 2000 kilocalories and was low in sodium and devoid of cholesterol. The diet was also rather unpalatable. In this particular study the systolic pressures dropped an average of 29 mm Hg and the diastolic pressures declined, on the average, 16 mm Hg.

Bernardis and Brownie (1965) found that diet restriction retarded the development of hypertension which usually follows adrenal enucleation in rats. Realimentation resulted in great increases of the blood pressure. The dietary restriction did not alter the final hypertension that resulted from adrenal enucleation.

Further work in the area of dietary production of hypertension was done by Wilhelmj and McCarthy (1963). Experiments performed by Wilhelmj and his co-workers are compiled in a compendium by Wilhelmj and McCarthy (1963). They determined that the basal blood pressure of trained dogs using the auscultatory method on the hind leg was less than 140/70 mm Hg. The amount of time necessary to train a dog was from 1 to 4 months and various emotional factors were found to elevate the blood pressure. Such

things as strangers in the laboratory, a change of experimenters, an unusual noise, a change in the established routine, or the presence of a female in estrus while making a determination on a male were found to cause elevations of the basal blood pressure. Thus, one must be very careful and consistent before reliable blood pressure measurements can be made from the canine.

Fasting in the dog had an effect on the blood pressure which could be divided into two stages (Wilhelmj and McCarthy, 1963). Stage one consisted of a blood pressure which fluctuated from day to day while declining towards a stable value. Stage two was the stable fasting level which lasted until refeeding. A representative stable fasting blood pressure was 90/40 mm Hg. The heart rate underwent a similar process but was not as marked. The authors believed the fluctuations of stage one were due to activity of the pituitary-adrenal cortical system counteracting the stress of fasting.

Wilhelmj and McCarthy (1963) refed the dogs with various diets and observed the effect each had on blood pressure and heart rate. Diets high in protein consisted of ground horse meat, beef, or casein. When this diet was fed at a low maintenance level (60 Cal/m^2/hr) there was an immediate return of the blood pressure and heart rate to control levels. The same was true if the high protein diet were refed at a luxus consumption level (120 Cal/m^2/hr). A high carbohydrate diet of cracker meal or white rice fed at the low maintenance level following a starvation caused the blood pressure and heart rate to immediately return to control levels just as the protein diet did. Feeding the high carbohydrate diet at a luxus consumption level, however, would cause the systolic pressure and

heart rate to rise significantly above control levels. The elevated pressure persisted as long as the diet was fed but disappeared as soon as the diet was stopped.

The authors repeated the starvation-refeeding regimens on four dogs which had been surgically sympathectomized. The control blood pressures were within normal limits but upon fasting they did not decline. Instead the blood pressures fluctuated around control levels. Upon refeeding with a luxus consumption of a high carbohydrate diet the blood pressures did not rise but remained at control levels. The authors concluded that the elevated systolic pressures seen with refeeding of a high carbohydrate diet at luxus consumption levels were mediated by the sympathetic-adrenal medullary system.

Wilhelmj and McCarthy (1963) also reported on several starvation-refeeding episodes performed on four trained dogs utilizing a high fat diet or beef suet alone or in combination with unsalted butter. This diet contained 50 percent or more of the calories from fat and was fed at the luxus consumption rate. During a period of 14 months these dogs went through six starvation-refeeding episodes. After four of these episodes the dogs began to show an abnormal response to fasting. Immediately after fasting began the blood pressures would drop to the stable fasting level but then would increase to levels above the control values. The blood pressures would remain elevated until refeeding at which time they returned to normal if the standard kennel diet was fed. When the high fat diet was fed the blood pressures were quite variable. These dogs were then put on a semi-starvation diet of ground raw horse meat interrupted repeatedly by feeding normal amounts of the standard kennel diet. In

three of the four dogs the blood pressures rose during the 16 month period to levels significantly above control. Four years later these three dogs were still showing systolic and diastolic hypertension (approximately 150/120 mm Hg) on a normal kennel diet. The heart rates during these experiments were elevated in only one dog.

Smith et al. (1964) performed starvation-refeeding experiments on young swine. Electrocardiograms during starvation revealed arrhythmias and T-wave inversion. The blood pressure, determined by the auscultatory method on a front leg, and heart rate declined during starvation. Refeeding consisted of several diets but those of glucose or high in glucose content were the most stressful. Diets of the latter type produced tachycardia, arrhythmias, and an elevated blood pressure. Diastolic hypertension became evident after the second starvation-refeeding episode. It persisted after the fourth episode until the end of the experiment 4 to 6 months later. Post-mortem examination revealed mild to moderate ventricular hypertrophy and fibrotic aortic plaques. Histologic examination of the myocardium revealed focal areas of necrosis. The arterioles in many organs had thickening of the media.

Johnson (1966) tried to repeat the experiments of Smith et al. (1964) with very little success. The author concluded that susceptibility of swine to the development of permanent hypertension due to the stress of starvation-refeeding was hereditary.

Hembrough and Link (1968) found a decreased arterial capacitance in swine following starvation-refeeding. They suggest that this could be the cause of the hypertension produced by Smith et al. (1964).

Hembrough and Riedesel (1970) used rats in a starvation-refeeding experiment. Circumferential stretching of the thoracic aorta was used to determine its stiffness. The refeeding diet consisted of either normal rat chow or dextrose. The treated rats did not show an elevated blood pressure at the conclusion of the experiment but did exhibit some aortic stiffness alterations. Those rats which were refed dextrose and sacrificed 3 weeks after the last starvation-refeeding period had a significantly decreased aortic stiffness. However, increased aortic stiffness was found in rats refed dextrose and sacrificed 6 weeks following the last episode. The authors interpreted these opposite findings to indicate initial damage to the aorta due to starvation-refeeding which was apparent as a decreased stiffness at 3 weeks post-treatment. Subsequently the aorta underwent repair which was apparent as an increased stiffness at 6 weeks post-treatment.

Crouch (1968) starved dogs for 3 days and then refed them with commercial dog food for 3 days repetitively five times. This treatment had no effect on arterial capacitance measured in vivo by an indirect technique. Circumferential stretch-tension analysis demonstrated a significant increase in femoral artery stiffness but a decrease in thoracic aorta stiffness. Blood pressures measured directly under anesthesia were not altered by the starvation-refeeding treatment.

Electrolyte and Body Fluid Compartment Changes with Starvation and Refeeding

Keys et al. (1950) found that the plasma volume as determined by the dye dilution technique increased with 24 weeks of semi-starvation from a

control value of 3.165 liters to a value of 3.410 liters. The total blood volume, however, decreased from a control value of 5.936 liters to 5.379 liters with semi-starvation. The increased plasma volume in the presence of a decreased blood volume was at least partially due to a decrease in blood cellular constituents as indicated by the hematocrit which decreased from a control value of 46.78 percent to 36.38 percent. At the end of semi-starvation the extracellular fluid volume was estimated using thiocyanate. The thiocyanate space at this time was 33.98 percent of the body weight while the normal value was considered to be 23.5 percent of the body weight. After 19 weeks of refeeding the excess extracellular fluid had almost completely disappeared. The authors stated that both plasma volume and extracellular fluid volume, when calculated on a per unit of body weight, increased during starvation and decreased during rehabilitation.

Haxhe (1967a) studied four dogs under semi-starvation conditions for 11 weeks. The following changes were noted: body weight decreased 27.5%, blood volume decreased 15.3%, plasma volume decreased 7.9%, hematocrit decreased 14.7%, and cardiac output decreased 30.4%.

Kutscher (1971) studied the water balance of hooded rats during 48- and 96-hour food and water deprivation. The plasma volume and total carcass water decreased while the hematocrit rose and the plasma protein and osmolality remained unchanged.

Bloom (1962) studied sodium excretion in the urine of humans during starvation-refeeding. Normal human subjects developed natriuresis during the early stages of fasting but sodium excretion in the urine decreased to

low levels immediately after carbohydrate ingestion. Refeeding with fat or protein, however, increased the sodium excretion of fasting.

Hall and Hall (1966) found that the quantity of 1% saline consumed and the development of salt hypertension in rats were both enhanced by the addition of either glucose or sucrose in 5% concentration to the drinking water.

Bloom (1967) reviewed the literature concerning carbohydrates and water balance. He did not discover the mechanism but found considerable evidence that carbohydrate diets caused salt and water retention. The mechanism was not related to ketosis but rather, he thought, to carbohydrate metabolism.

Katz et al. (1968) also found that fasting in man was accompanied by natriuresis which lasted for several days or weeks. In their study, however, refeeding with either carbohydrate or protein halted this sodium loss.

Hoffman et al. (1971) found that either intravenous or oral administration of glucose would halt the natriuresis of fasting. The antinatriuretic effect of oral glucose was independent of circulating insulin and free fatty acid levels. Oral glucose had its antinatriuretic effect only in the fasted state but it had an antikaliuretic effect in both fed and fasted subjects. Intravenous glucose had the same effect as its oral administration on sodium but it had no effect on potassium excretion.

Veverbrants and Arky (1969) studied the electrolyte excretion with fasting in humans maintained on a constant fluid and electrolyte intake. They found that 3 days of fasting was accompanied by natriuresis and

kaliuresis. Refeeding a carbohydrate diet rapidly reversed the electrolyte excretion. A protein diet slowly halted the electrolyte loss, and a fat diet aggravated the negative sodium balance. The authors did not believe the action of carbohydrates was secondary to changes in levels of aldosterone, glucocorticoids, or catecholamines.

Boulter et al. (1973) found natriuresis in man during the early stages of starvation (days 2 through 6), which was followed by a positive sodium balance until starvation was halted after 15 days. Even the isocaloric reduction of the carbohydrate in the diet to 50 gms or less resulted in natriuresis. The authors postulated that the mechanism may involve glucagon.

Saudek et al. (1972) also believed that glucagon may be the cause of natriuresis in starvation. They infused five obese humans with glucagon at a rate which simulated the increased levels seen in early starvation. The resulting urinary excretion of sodium increased by a mean of 97 percent.

Indeed, earlier investigators (Elrick et al., 1958) found that glucagon injection in man enhanced sodium, potassium, chloride, and inorganic phosphorus clearance ratios. They believed the effect of glucagon was directly on the tubules of the kidney and was not related to hyperglycemia or carbonic anhydrase inhibition.

Glucagon administration in the dog enhanced the renal excretion of iodide, sodium, potassium, chloride, and phosphate (Staub et al., 1957). This enhancement may have been due, at least in part, to its effect of increasing the glomerular filtration rate (Serratto and Earle, 1959).

Garnett et al. (1973) measured the aldosterone secretion rate and plasma renin activity during starvation and refeeding in man. After 10 days of starvation both measurements rose significantly. The elevation of both may play a role in the reduction of urinary sodium loss observed in late starvation. Refeeding with a carbohydrate diet did not alter these hormone levels but a sudden and significant reduction in urinary sodium was still present. The authors concluded that the antinatriuretic effect seen with the consumption of carbohydrate diets was not due to the renin-aldosterone system.

Boulter et al. (1974) found dissociation of the renin-aldosterone system in fasted man. During the natriuretic phase of fasting aldosterone secretion rate rose while plasma renin activity decreased. Just the opposite was seen during the antinatriuretic phase of refeeding. Another interesting observation made by the authors was that fasting natriuresis was unresponsive to mineralocorticoids. The authors emphasized that metabolic fuels and the hormones which regulate the availability of these fuels were important in controlling sodium excretion by the kidney.

Blood Cellular Constituent Changes with Starvation and Refeeding

Anemia has been a relatively consistent finding with under-nutrition of animals and man. Erythropoiesis was considered to be suppressed or ceased by starvation in some literature reviews (Kjellberg and Reizenstein, 1970; Lucarelli et al., 1970).

Keys et al. (1950) found that the hemoglobin concentration in the 32 men who underwent 24 weeks of semi-starvation fell from 15.1 ± 0.9 gm per

100 ml of whole blood to 11.7 ± 0.8 gm per 100 ml of whole blood. The number of red blood cells per cu mm of whole blood decreased from 5.222 to 3.782 million over the 24 weeks of semi-starvation in the 18 subjects sampled. The hematocrit in those same 18 individuals decreased from 46.78% to 36.38%. These values indicate that moderate macrocytic anemia had developed during the semi-starvation. Return of these values to normal was slow taking about 33 weeks.

Kjellberg and Reizenstein (1970) studied humans undergoing 4 weeks of starvation and found a decrease in the hemoglobin concentration of women. The reticulocyte counts were decreased but not significantly. Serum levels of iron in women and folic acid in men were decreased.

Kornegay et al. (1964) starved swine for 7 days and found increased values for the hematocrit and hemoglobin concentration after the first day while the total plasma and blood volume did not change. The percentage of body weight due to blood increased from a control value of 6.51% to 9.96% at the end of the experiment. The rectal temperatures decreased after 27 hours of starvation.

Furugouri (1973) starved 24 pigs for 15 days and refed them for the same period of time. During starvation the hemoglobin concentration became elevated while the serum protein level remained unchanged. Refeeding was accompanied by a decline in the hemoglobin concentration to normal and a decrease in the serum protein concentration.

Haxhe (1967b) subjected dogs to undernutrition for 11 weeks. The dogs were then transfused with isotope labelled red blood cells and kept on a calorie deficient diet for 4 more weeks. The transfusion resulted in a marked reduction in the volume of autogenous cells. The author

interpreted this to mean that the body did not need as many red blood cells for oxygen transport during undernutrition. Erythropoiesis was apparently slowed down during undernutrition and the transfusion of extra red cells caused a further reduction in erythropoiesis. Three weeks of starvation in the dog have been shown (Penny, 1973) to cause changes in the cellular constituents of bone marrow.

Keys et al. (1950) also followed the total and differential leukocyte counts in seven human subjects during semi-starvation and rehabilitation. Semi-starvation caused leukopenia with the count decreasing from a control value of 6,346 per cu mm of blood to 4,129. The differential counts were not significantly altered by semi-starvation. These results were contrasted by the authors with findings of a relative lymphocytosis by previous researchers. The leukocyte count had returned towards normal after 12 weeks of rehabilitation.

Wilhelmj and McCarthy (1963) followed the total eosinophil counts of dogs during starvation and refeeding. During the early stages of fasting the eosinophil counts fluctuated markedly and gradually diminished to a low fasting level. Refeeding with a high protein diet was accompanied by a return of the eosinophil count to normal. Refeeding with a high carbohydrate diet, however, did not return the eosinophil numbers to normal. Instead they remained at the low level established during fasting. The authors were using the total eosinophil count as an index of pituitary-adrenal cortical activity. They concluded that this system was activated by starvation and the activation was continued by consumption of a diet high in carbohydrates. The same conclusions were drawn by concurrently

measuring the resistance of the capillary wall to external suction which was elevated by starvation and refeeding of a high carbohydrate diet.

Drenick (1971) fasted human patients and found an increase in neutrophils during the first 10 days and then a decrease to 50% of the control values after prolonged starvation which averaged around 44 days. The author postulated that protein deficiency during the late stages of starvation may impair bone marrow activity.

Aschkenasy (1957) authored a paper on the pathogenesis of anemia and leukopenia induced by protein deficiency. He listed the involvement of three factors: 1) a lack of amino acids for new cell production; 2) a reduced store of necessary vitamins, viz., vitamin B_{12}, folic acid, and nicotinic acid; and 3) a hormonal imbalance due to inactive thyroids and gonads with a relatively hyperactive adrenal gland.

Bray (1974) listed numerous vitamins as being reduced in circulating concentration during starvation, viz., thiamine, niacin, biotin, pantothenic acid, folate, riboflavin, and pyridoxine.

Wintrobe (1967) described the effect that various endocrine glands had on red and white blood cell counts. Castration in males led to anemia which was reversed by testosterone administration. Castration in females led to a rise in red blood cell counts which was reversed by administering estradiol. Thyroidectomy led to anemia which may be due to reduced erythropoietin production as a result of decreased oxygen demand by the tissue. Adrenal corticosteroids may sometimes improve erythropoiesis but are consistently associated with producing a relative or absolute neutrophilia, lymphopenia, and eosinopenia. Hyperthyroidism was accompanied by a relative and absolute increase in lymphocytes and eosinophils.

Pospisil et al. (1970) adrenalectomized mice and found that anemia developed which could be reversed by giving cortisol and corticosterone. Starved mice also became anemic but were not as severe as those which were adrenalectomized. The authors postulated that the anemia during starvation may be due to less oxygen demand by the tissues of the body.

Other Effects of Starvation and Refeeding

The effect of starvation and undernutrition on the thyroid and adrenal gland has been studied by several groups of researchers.

Keys et al. (1950) found that the thyroid gland had consistently been reported as being atrophied during starvation.

Alexander et al. (1964) discovered a decrease in thyroid activity with deprivation of food.

While studying the effect of starvation on the thyroid gland Schatz et al. (1967), however, found increased levels of free thyroxine in the blood. This was attributed to a decreased thyroid binding globulin and thyroid binding prealbumin as a result of reduced liver synthesis of these compounds.

Keys et al. (1950) reviewed many morphologic studies on the adrenal gland in starvation. In animals the adrenals hypertrophy during acute starvation but atrophy in chronic starvation.

Schachner et al. (1965) concluded, after fasting humans for 10 to 14 days, that cortisol secretion was reduced by brief periods of fasting.

Sabeh et al. (1969) found that 1 week of starvation in man caused partial inhibition of 11-hydroxylase in the adrenal gland.

Adrenal activity in the starved adult female rabbit was increased for the first 24 to 48 hours but became mildly depressed after 4 to 14 days (Bouille and Assenmacher, 1970).

Carbohydrates, Hypertension, and Heart Disease

There have been occasional attempts to correlate diet with hypertension, arteriosclerosis, and coronary heart disease.

Yudkin (1957) compared diet and mortality due to coronary heart disease utilizing data from many countries for the years 1951-1952. He also looked at dietary changes within the United Kingdom since 1928 and compared them to the increased incidence of coronary heart disease since that time. The author found it hard to support any theory which supposes a single or major dietary cause of coronary thrombosis. He suggested that over consumption of food and reduced physical exercise may be a causative factor.

Using questionnaires as a source of data Yudkin (1964) concluded that subjects with peripheral artery disease or a recent first myocardial infarction consumed significantly more sugar than control subjects. The author believed this to be the first published evidence of a dietary difference in people with atherosclerotic disease.

Masironi (1970) concluded from data on diet and the incidence of arteriosclerotic and degenerative heart disease from many countries that the relationship was still controversial. An interesting statistic in this paper was that between 1955 and 1965 death due to arteriosclerotic and degenerative heart disease increased by 51%. The correlation coefficients between dietary constituents and the increased death rate were:

total calories, +.57; fat, +.18; unsaturated fat, -.27; sucrose, +.43; simple sugar, +.40; complex carbohydrates, +.08; and protein, +.26. It is interesting to note that sucrose has the highest positive correlation coefficient of the dietary constituents.

Animals subjected to high glucose diets have developed cardiovascular pathology. Smith et al. (1964) found ventricular hypertrophy, myocardial necrosis, fibrotic aortic plaques, and arteriolar medial hypertrophy in swine that were starved and refed high glucose diets.

Brooks et al. (1972) induced granulomatous calcareous endocarditis in a high percentage of the swine fed a high sucrose diet (64 or 66%). They occasionally found lesions in the left ventricle also.

Ahrens (1974) reviewed the literature on the role of sucrose in heart disease. He postulated that high sucrose diets lead to renal retention of sodium and water which in turn leads to hypertension. Hypertension combined with elevated dietary sucrose produced liver damage which would lead to high plasma triglyceride concentration. The combined effect of hypertension and elevated plasma triglycerides would consequently cause arteriosclerosis and heart disease.

Physical Properties of Blood Vessels

Roy (1880-1882) was one of the first to investigate the extensibility of arteries. He did this by conducting in vitro pressure-volume studies on the arteries of dogs and by stretching strips of human aortae.

Krafka (1939) stretched the ligament of nuchae from cows and aortae of the dog, man, and cow. He observed that the first stretch of the aorta was different but following this the vessel could be stretched up to 30

times without significant variation. Young's Modulus was calculated at 25, 50, 100, and 200 grams tension using the formula:

$$Y = \frac{W \cdot L \cdot 980}{A \cdot el}$$

where

Y = Young's Modulus, dynes x $10^6/cm^2$

W = tension, grams

L = initial length, cm

A = area, cm^2

el = elongation, cm

980 = conversion, grams to dynes

The Young's Modulus increased in value as more tension was placed on the vessel. By using putrefaction to destroy elastin the author concluded that collagen was responsible for this increase in Young's Modulus.

Remington (1948) in stretching aortic rings removed from humans noted that retraction of the aorta after its removal varied from 2 to 15%. The tension-length data were converted to pressure-volume values and an unsuccessful attempt was made to predict the stroke index from the pulse pressure.

Burton (1954) tried to correlate vessel structure with function. He emphasized that the sigmoid-shaped pressure-volume curves should be converted to tension-radius curves for mechanical analysis of the blood vessel. He found that the Young's Modulus of collagen (1.3 x 10^9 dynes/cm^2) was 400 times that of elastic fibers (3 x 10^6 dynes/cm^2). The elastic properties of smooth muscle were believed to be of little significance in maintaining the elastic tension of the whole wall during steady

state conditions. The elastic fibers produced the maintenance tension which opposed the normal blood pressure in the vessel. Collagenous fibers were stretched only at higher than normal pressures and had a protective supporting role. Elastic tissue and smooth muscle, together, made possible the stable graded contractions of blood vessels. Contraction of the smooth muscle pulled the restrictive collagen jacket inward and would hold it in the face of high pressure.

Remington (1955) studied the hysteresis in the tension-length diagrams of canine aortic rings in vitro. Tension of 25 to 50 grams was necessary to make the walls of the ring parallel. The width of the hysteresis loop depended upon the amplitude of the stretch and, when stretched above a critical level, upon the rate of stretch. The femoral artery displayed a similar hysteresis.

Roach and Burton (1957) investigated the role that elastin and collagen play in determining the shape of the distensibility curves of arteries. The arterial wall was considered heterogeneous. Ninety percent formic acid was used to remove the collagen and crude trypsin was used to remove the elastin. From studies on digested arteries they concluded that the initial slope of the distensibility curve was an index of the state or number of elastin fibers in the artery while the final slope was an index of the state or number of collagenous fibers.

Peterson et al. (1960) and Peterson (1966) devised a method for recording pressure-circumference changes in vivo. The authors did not believe that pressure-strain diagrams provided much information on the mechanical properties of the wall and converted their data to tension-strain units. The resulting curve was fit with a first order differential

equation. In order to make the conversion of units from pressure to tension the authors needed the arterial wall thickness. This was obtained histologically after fixing the vessel with a balloon inside its lumen inflated to 150 mm Hg pressure. The radius/wall thickness ratio for the femoral artery of the dog at 150 mm Hg ranged from 3.4 to 8.8. The coefficients for the first order differential equation were called elastic and viscous coefficients. No attempt was made to correlate the resulting coefficients and vessel structure.

Bergel (1960) made several conclusions after reviewing previous work in the area of the structural and physical properties of arteries. He found that the arterial wall was non-Hookian in its elastic behavior and became stiffer with increasing distension and with the age of the individual vessel. He also concluded that the structural components of the wall, i.e., collagen and elastin fibers, were arranged in parallel.

An electric caliper sutured to the vessel wall was used to study the in vivo pressure-radius changes of canine vessels. Patel et al. (1960) found that the radius change of the pulmonary artery per unit of pressure change decreased 19% after norepinephrine was administered. Contrary to previous assumptions the aorta was found (Patel et al., 1961) to change in length during respiration and the cardiac cycle. The recorded aortic distensibility was found to decrease at high pressures.

Bergel (1961a) measured the static elastic properties of various arteries harvested from dogs. He held the vessels at their in vivo length and obtained a pressure-volume curve. The data were then converted to an incremental modulus which related pressure change to radius change. Arteries, in general, were found to be much more extensible than metals

and did not show a constant ratio between stress and strain but rather became stiffer as they were stretched. The incremental elastic modulus for various vessels at 100 mm Hg pressure in dynes x $10^6/cm^2$ were: thoracic aorta, 4.3; abdominal aorta, 8.7; femoral artery, 6.9; and carotid artery, 6.4. The relative wall thickness/radius ratio for the femoral artery at 100 mm Hg pressure was 11.5 ± 0.96. The percent shortening of the femoral artery after removal from the body was found to be 42.0 ± 0.9 percent.

Bergel (1961b) also performed dynamic studies on segments of arteries. Arteries were found to show properties of creep and stress relaxation. Creep was defined as the continued extension of a vessel under a constant load. Stress relaxation was defined as a decay in the tension of an artery held at a constant length. Bergel's dynamic studies were done in vitro at a mean pressure of 100 mm Hg with a dynamic change of ± 5 to 10 mm Hg. The frequency of the pressure cycle was varied with a maximum of 20 cycles per second. All arteries tested demonstrated an abrupt increase in stiffness between frequencies of zero (i.e., static) and two cycles per second. The increase in stiffness was greatest in magnitude for muscular arteries.

Attinger et al. (1964) found that excised arterial segments from the aorta retracted with the greatest percentage. The percent recoil of the femoral artery was very dependent on the position of the leg when the vessel was excised, but the mechanical properties of the excised artery did not depend on the leg position prior to excision.

Wolinsky and Glagov (1964) described the aorta of the rabbit as functioning like a two-phase material. Low pressures were associated with

straightening and uncrumpling of elastic lamellae and fibrils, alignment of collagen fibers, and restoration of interlamellar spaces to physiological dimensions. High pressures were born primarily by the circumferentially aligned collagen fibers which had a high tensile strength and a high modulus of elasticity. The wall thickness was found to decrease markedly at low tensions but remained relatively constant at high tensions. Structurally, collagen appeared to be arranged in a uniform helix with only a small pitch. The circumferential arrangement of elastin was not as uniform as the collagen because of the numerous branching and connections between elastic fibers.

Wiederhielm (1965) measured the extensibility of arterioles in the frog mesentery and the calculated values of Young's Modulus increased with increasing strain.

Apter et al. (1966) performed stretch relaxation tests on canine aorta and pulmonary artery. From the data they calculated constants to fit a model consisting of a series elastic and viscous element and a parallel elastic element. The viscous and series elastic constants were higher in vessels where muscle content was high and increased markedly when the muscle was tonically contracted. The parallel elastic constant was high when elastin was high and in the presence of contracted muscle. The parallel elastic constant seemed independent of collagen content.

Peterson (1966) discussed the relationship of vascular caliber and blood flow. He stated that the vascular radius was a function of the distending pressure, the geometry of the vessel's wall thickness and radius, and the mechanical properties of the wall.

Fischer and Llaurado (1966) autoclaved arteries to separate collagen and elastin. Collagen and elastin were then quantitated by hydroxyproline analysis and their sum ranged from 58 to 75% of the dry defatted vessel weight. The ratio of collagen to elastin (C/E) was calculated for various vessels:

Coronary	3.12 ± 0.21
Carotid	2.55 ± 0.13
Abdominal aorta	1.58 ± 0.15
Renal	2.46 ± 0.27
Femoral	1.89 ± 0.14

Arteries that were exposed to a high external pressure (e.g., coronary artery) and those leading to pressure sensors (e.g., carotid and renal) had higher C/E ratios.

Wolinsky and Glagov (1967) described the elastic lamellae and the contents of the adjacent interlamellar zone as the unit of function in the mammalian aortic wall. The average tension per lamellar unit in the aortic media was very constant among various species, viz., 1090-3010 dynes/cm.

Tickner and Sacks (1967) were more exacting in their description of the static elastic properties of blood vessels. They stated that the arterial wall behaves as a nonlinear, homogeneous, anisotropic, compressible material. The authors theorized that the mechanical characteristics of an artery could be described by six elastic constants at each level of strain. The six elastic constants could then be used to calculate the Young's Modulus for each of the three planes.

Patel et al. (1969) found that values for the radius/wall thickness ratio of the canine thoracic aorta ranged from 8.1 to 10. This ratio was considered large enough to permit calculation of wall stress based on thin-wall theory.

Patel and Fry (1969) concluded from in vitro tests on the carotid artery and thoracic and abdominal aorta that these segments are cylindrically orthotropic. That is, only elongating strains were found, no shearing strain of significance was measured.

Dobrin and Rovick (1969) found that activation of smooth muscle increased the elastic modulus when it was plotted against strain. But, smooth muscle activation decreased the elastic modulus when plotted as a function of pressure. The discrepancy was due to the decrease in radius seen with smooth muscle contraction.

Dobrin and Doyle (1970) concluded that the carotid artery of the canine was not isotropic at physiologic pressures. The circumferential elastic modulus was greater than the longitudinal modulus.

Azuma and Hasegawa (1971) calculated Young's moduli for various tissues by elongation of strips. They used longitudinal and circular strips from the external iliac and femoral arteries. Their results were:

	$\times 10^6$ dynes/cm^2
Vascular wall	1.72 ± 0.57
Smooth muscle	1.45 ± 0.76
Ligament of nuchae	3.72 ± 0.56
Tendon	4220. ± 1210.

Fung (1972) reviewed the technique of measuring the mechanical properties of soft tissue by elongation. He commented that precondition-

ing (prestretching) was commonly done and its meaning was unclear. He advocated the use of Langrangian stress, obtained from the division of wall tension by the initial cross-sectional area of the tissue. This eliminated having to measure the tissue area at every level of tension. He also suggested plotting the slope of the stress-strain diagram (Young's modulus) against stress. This relationship can then be described by either a linear or a quadratic equation. This technique was applied by Tanaka and Fung (1974) using 5 mm wide strips of canine artery. They concluded that arteries are inelastic (non-Hookian) and the numerical value of Young's modulus increased for vessels located more towards the periphery.

Dobrin and Canfield (1973) did not like the terms Young's modulus or elastic modulus to describe the slope of the stress-strain graph. They consequently used the term stiffness and plotted it against stress. A quick release technique was used to study the series elastic and contractile elements of vascular smooth muscle.

Soden and Kershaw (1974) discussed mechanical testing of biological materials by simple elongation. The increase in stiffness seen in arteries with stretching was thought to be due to a progressive increase in the number of collagen fibers carrying the load. They discussed stretching blood vessels by unaxial tension and concluded it was a valid technique if the strain applied was considerably larger than the deformation induced by bending the tissue into the straight position. The induced deformation comes from straightening the circular blood vessel which stretches the inner surface and compresses the outer surface tending to buckle the tissue. The difference in strain between the inner and outer surfaces

when straightened can be calculated by dividing the wall thickness by the mean radius of curvature.

Arterial Wall Changes with Age and Certain Abnormal Conditions

Various techniques have been used to determine the effect of aging on the extensibility of arteries.

Clark (1933) used plethysmography to study the pressure-volume relationship of veins in vivo. He expressed the mechanical properties as a coefficient of functional elasticity (E). This coefficient, E, was equal to the volume of the vessel divided by the volume change per unit of pressure change. The author found that E decreased with age, an unexpected finding.

Hallock (1934) calculated arterial distensibility from the pulse wave velocity using the formula:

$$\frac{\Delta V}{V \cdot \Delta P} = \frac{12.7}{(\text{pulse wave velocity, meters/sec})^2}$$

where:

ΔV = the change in volume

V = the original volume

ΔP = the change in pressure

The pulse wave velocity was found to increase with advancing age which implied a concomitant decrease in arterial distensibility. Subsequently, Hallock and Benson (1937) determined the arterial distensibility from pressure-volume measurements performed on isolated aortae from normotensive humans. Values for pulse wave velocity calculated from the vessel distensibility were on the average 6.4% lower than those actually measured.

The authors found that arterial rigidity increased with age and with increasing diastolic pressure.

Wilens (1937) froze transverse strips of human aorta for 24 hours to kill the smooth muscle. He then thawed the strips and loaded them with a 300 gm weight, removed 295 gm of the load, and measured the resulting retraction. His measurements indicated that aging of the individual produced a progressive loss of aortic extensibility.

Krafka (1940) utilized elongation of longitudinal aortic strips to evaluate changes with age. He found a gradual loss in extensibility with age throughout life.

Hass (1942) extracted collagen from the human aorta by formic acid digestion. The quantity of elastic tissue that remained per unit volume of aortic wall was nearly constant throughout life. But, the tensile strength and extensibility of this elastic tissue decreased with increasing age. These changes could not be predicted from the morphologic appearance or extensibility of the intact aorta and were considered to be due to discontinuities of the elastic system from disintegration of elastic lamellae (Hass, 1943). Indeed, the low extensibility of the aged dilated intact aorta could have been due, at least in part, to the fact that at zero load the elastic network was already under tension. The decreased extensibility with aging of the individual also occurs in rough proportion to the increase in collagen in the intima and media. Thus, aging of the individual seemed to have two effects on the artery: 1) disintegration of the elastic tissue; and 2) an increase in the collagen content. Both could lead to a decreased extensibility.

Burton (1951) stated that aging caused a decrease in the "elastic constant" and an increase in the rigidity of the "jacket" of fibrous tissue.

Learoyd and Taylor (1966) used the incremental modulus to study changes in arterial wall viscoelastic properties with age. The amount of vessel retraction decreased as age increased and arteries from young people were successively stiffer as one went peripherally but just the opposite was true of the older people. Wall tissue became weaker per unit area with age but the body seemed to compensate by dilatation and thickening of the wall.

Nichol (1955) found that rabbits with atherosclerosis induced by a high cholesterol diet had thoracic aortae which were more distensible than normal at low pressures due to destruction of elastic tissue.

Coulson and Carnes (1962) found that swine placed on a copper deficient diet since birth had an altered aortic stretch-tension curve. They calculated two stretch moduli, one for the lower and one for the upper part of the curve. The copper dificient swine had a reduced modulus for the lower part of the curve which was interpreted to be due to defective elastin. The ultimate tensile strength of the aorta of these swine was reduced fourfold when compared with controls.

Coulson et al. (1965) repeated the study on copper deficient swine but this time the second phase stretch modulus was altered. Elastin was removed from the aorta by digestion to examine the collagen stretch-tension curve. They found, however, that removing elastin not only altered the first part of the stretch-tension curve but it also altered the second phase. The authors remained with their previous conclusion that copper

deficiency altered elastin production. The second phase of the stretch-tension diagram was altered because elastin and collagen had functional interconnections and damage to elastin could alter the second phase of a stretch-tension diagram.

Folkow et al. (1958) found that the lumen of blood vessels of people with essential hypertension at maximum dilatation was narrowed. This, they hypothesized, was due to medial hypertrophy. The increase in wall mass would also cause a proportionally bigger lumen decrease for a given smooth muscle shortening. The increased mechanical advantage that hypertrophy of the media would provide for the smooth muscles could account for the increased reactivity to vasomotor drugs seen in hypertensives.

Furuyama (1962) devised a method of normalizing measurements from histologic sections of blood vessels. He measured the area of the media by planimetry and the length of the wavy internal elastic membrane. He then developed a formula to calculate the vessel radius and medial thickness as if the internal elastic membrane were straight and unwavy. He concluded that hypertensives have hypertrophy of the media layer in the renal and superior mesenteric arteries, the only two vessels that he studied with his technique.

Feigl et al. (1963) measured simultaneously in vivo pressures and diameter changes in the femoral arteries of dogs before and after the development of renal hypertension. Values for the elastic modulus calculated from these data were increased after hypertension was present for 4 weeks. The authors interpreted this to mean that the hypertensive dogs had stiffer vessels after exposure to a high blood pressure. The water

content of the arteries from the hypertensives was also significantly elevated.

Fischer and Llaurado (1967) failed to find an increased C/E ratio in the arteries of renal hypertensive dogs. The increased stiffness seen with aging was due to an increase in collagen and a decrease in elastin content. Thus, the authors concluded that the stiffer arteries of hypertensives was not an accelerated age change.

Greene et al. (1966) found a decreased distensibility in the brachial artery of humans with essential hypertension by performing pressure-volume measurements on a 3 cm segment of surgically exposed artery.

Folkow and Sivertsson (1968) studied the hindleg vascular bed of cats after 3-5 weeks of regional hypotension. The hypotensive vascular bed had a normal threshold to norepinephrine but a reduced response to supra-threshold doses of norepinephrine. The authors interpreted this to mean that the wall thickness of the arterioles was reduced in the hypotensive adapted vascular bed. This interpretation was supported by histologic examination of blood vessels from both normal and hypotensive vascular beds which were fixed at identical radii. The authors concluded that blood vessel walls do adapt to the pressure they are exposed to.

Aars (1968) found that aortic strips from hypertensive rabbits had thicker walls and were less distensible per unit of cross sectional area than aortic strips from normotensive rabbits.

Tobian et al. (1969) studied the water content of arterioles after development of reno-vascular hypertension. An increase in water content per 100 gms of solids was found which could increase the arteriole stiffness.

Wolinsky (1970) studied the adaptation of the tunica media of the aorta to 8 weeks of hypertension. The thoracic aorta was increased in diameter and had an increased thickness and cross-sectional area of the tunica media. The number of lamellar units was unchanged but the absolute amount of both elastin and collagen were increased.

Sivertsson (1970) reviewed the literature on essential hypertension. He found that people with essential hypertension had a normal cardiac output at rest and normal blood viscosity. He found little evidence which pointed to an increase in vasoconstrictor nerve activity or increased levels of circulating pressor substances. Electrolyte alterations and "water logging" of vessels was not well substantiated. Vascular reactivity was increased for many different drugs and the morphologic change of an increased wall thickness to radius ratio was commonly seen in arterioles.

Folkow et al. (1970a) found structural adaptation in the vascular bed of rats as soon as 1 week following the onset of hypertension. The resistance to flow in these hypertensive rats was increased above normal at maximal vasodilation. The authors suggest that the morphologic adaptation raised the peripheral resistance.

Folkow et al. (1970b) found that the perfused hind limbs of hypertensive rats responded in the way one would predict if the tunica media of the arterioles was increased in thickness and encroaching upon the lumen. In particular, the hypertensive rats had a higher resistance to flow than normal rats during maximum vasodilation. Both groups of rats had identical thresholds to norepinephrine. But, the hypertensive rats showed an increased resistance to flow above that seen in normotensive rats with suprathreshold doses of norepinephrine.

Wolinsky (1971) produced hypertension in rats for 10 weeks and then reversed the hypertension for 10 weeks. There was a decrease in the calculated wall stress to normal after reversal of the hypertension. The wall thickness and the area of the tunica media in male rats remained elevated and were similar to measurements from hypertensives even though their blood pressure had returned to normal. These measurements in the female rats had returned to normal when hypertension was reversed. The elastin and collagen content remained elevated after reversal of the hypertension and the author concluded that the decrease in arterial wall thickness was due to a loss of the noncollagenous, alkali-soluble fraction of the arterial wall.

Overbeck et al. (1971) studied the hemodynamics of the canine forelimb before and after renal hypertension. A portion of the elevated vascular resistance was not attributable to neural stimuli. The authors postulated that structural vascular changes may account for part of the increase in limb resistance.

Folkow et al. (1971a) found that reducing the blood pressure of spontaneously hypertensive rats with hydralazine and quanethidine caused partial reversal of the increased wall/lumen ratio.

Folkow et al. (1971b) produced the same result as above by regional hypotension in the spontaneously hypertensive rat. The authors concluded that an altered pressure load, decreased or increased, rapidly affects the wall structure of the resistance vessels.

Wolinsky (1972) found an increase in wall thickness after 16 months of renal hypertension in rats. Concurrent aging changes were qualitatively similar to, but less pronounced, than those seen with hypertension.

Hallback et al. (1972) did hemodynamic studies on the isolated perfused hind quarter of renal hypertensive rats. The renal hypertensives had a higher resistance to flow at maximal vasodilation. The threshold for norepinephrine was unchanged but there was an increased steepness to the resistance curve relating pressure to graded norepinephrine dose in the hypertensives. The authors postulated that the same exaggerated response of the tunica media thickening may be occurring in humans with essential hypertension.

Folkow et al. (1972) prevented the extreme high blood pressure seen in spontaneously hypertensive rats by immunosympathectomy. This procedure, however, did not prevent the vascular adaptive changes seen in hypertensive rats. The authors hypothesized that the spontaneously hypertensive rat may be genetically more prone to develop structural vascular adaptations to increased pressure loads.

Folkow et al. (1973) found that vessels will adapt very rapidly to a change in perfusion pressure. Structural regression was seen in the vessels of spontaneously hypertensive rats within 3 to 7 days following regional hypotension. Renal hypertensives had structural vascular adaptations within 7 to 14 days following constriction of the renal artery. Structural changes in both groups were complete within 3 weeks. The more rapid response of the spontaneously hypertensive rats may be a genetic feature.

Hallback et al. (1974) found a reduced distensibility in resistance vessels of spontaneously hypertensive rats.

Lundgren et al. (1974) reported the rate of appearance of various adaptive changes seen in surgically produced renal hypertensive rats.

Left ventricular hypertrophy appeared within 1 week which was followed by adaptive structural changes in the resistance vessels. The vessel changes were mainly hypertrophy of the tunica media which was complete within 2 to 3 weeks. The aorta did not show an increased water content until 4.5 months after the operation. The vascular changes were considered secondary to an increase in pressure load but were considered important to the development and maintenance of the hypertensive state.

MATERIALS AND METHODS

Animals

Nineteen specific pathogen free beagles were obtained from the Veterinary Medical Research Institute of Iowa State University in October, 1971. The dogs, ten females and nine males, were 8 months old. They were housed in individual cages and vaccinated for canine distemper, hepatitis, and leptospirosis.[1] Fecal examination did not reveal any pathologic parasites.

Carotid Loop Surgery

In November, 1971, left carotid loops were prepared in all 19 beagles. Under general anesthesia and utilizing sterile technique the surgical procedure of O'Brien et al. (1971) was performed, with a few modifications, on the dogs. The loops were subsequently removed from four dogs because of excessive post-operative edema and infection. Of these four dogs the carotid was left intact in two of the dogs and returned to a near normal position in the neck. The artery was completely removed in the other two dogs because it had ruptured in one case and because it was nonfunctional in the other.

Blood Pressure Recording

In April, 1972, after several months of training, blood pressure recording was initiated. The dogs with carotid loops were laid on their

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[1]Fromm Laboratories, Inc., Grafton, Wisconsin.

sides in a "V"-shaped wooden trough. After a few minutes their carotid loop was punctured with a 22 gauge 1 inch hypodermic needle.[1] The needle was connected to a pressure transducer[2] via a saline filled vinyl tubing (I.D.-0.020 in., O.D.--0.036 in.).[3] The entire fluid filled system had a damping ratio of 0.73 and an undamped frequency response of 49 hertz. Paper chart recordings[4] were used to determine the blood pressure and heart rate. Ten consecutive wave forms were averaged to determine the systolic and diastolic blood pressure. The heart rate was determined from a 15 second interval containing the ten wave forms used for the blood pressure determination.

Group Assignments

A control group and a treatment group of dogs with similar initial blood pressures were desirable. Mean blood pressures from the days April 10, 12, 14, 19, and 21 were used to make group assignments. The dogs were arranged by sex in ascending order according to their mean blood pressure over the previously mentioned 5 days and alternately assigned to groups. Those dogs which did not have carotid loops were randomly assigned to groups. The control group contained ten beagles, five females (four of

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[1]Yale hypodermic needle, Becton, Dickinson and Company, Rutherford, New Jersey.

[2]Statham P23dB, Statham Instruments, Inc., Oxnard, California.

[3]Vinyl tubing, Becton, Dickinson and Company, Rutherford, New Jersey.

[4]Beckman R411 dynograph, Beckman Instruments Inc., Schiller Park, Illinois.

which had carotid loops) and five males (three of which had carotid loops). The treatment group consisted of nine dogs, five females (all with carotid loops) and four males (one of which did not have a carotid loop).

Femoral Artery Segments

Prior to the experiment (April 4-8, 1972) and at the conclusion of the experiment (August 21-23, 1972) a segment of femoral artery was surgically removed from each dog. The initial site of removal was randomly selected. Utilizing general anesthesia and sterile technique the femoral artery was exposed as proximal as possible through an incision on the medial side of the leg. Care was taken to preserve the deep femoral branch of the femoral artery so collateral circulation would be established (Perkins and Edmark, 1971). A standard 1 cm length of femoral artery was removed with the assistance of two razor blades separated to that distance by a piece of Plexiglas. The removed arterial segment was immediately placed in saline and frozen until the time when stretch-tension analysis was performed. Previous work has shown that prolonged storage in this fashion does not alter the physical properties significantly (Learoyd and Taylor, 1966) and will effectively kill smooth muscle within 24 hours (Wilens, 1937).

The opposite femoral artery was removed at the end of the experiment when the dogs were euthanatized.

Treatment Regimens

The control group of dogs was left on the normal kennel diet[1] ad libitum throughout the experiment while the treatment group underwent various dietary alterations. Both groups of dogs had continuous access to water during the experiment. The various treatment regimens and their time intervals are shown in Table 1. Blood pressure recording began on April 10, 1972. That date was considered as day #1 of the experiment. The 70% carbohydrate diet[2] was fed at a rate of 120 Cal/m^2/hr during both periods of refeeding. The surface area of each dog was obtained from a nomogram which correlated body weight and length with surface area. Body weights were measured weekly and adjustments in the quantity fed were made. The caloric content of the experimental diet was 678 Cal/lb and its digestibility was estimated by the supplier to be 85%. Seventy percent of the calories came from carbohydrates of which sucrose constituted 27% while rice and other starches accounted for 43% of the diet's energy. The composition of the high carbohydrate diet is summarized in Table 2. Vitamins and minerals were sufficient to meet the requirements of the Committee of Animal Nutrition (1962).

Vectorcardiograms

Towards the end of each feeding regimen a vectorcardiogram (VCG) was created using scalar recordings obtained from each dog. The McFee and

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[1]Friskies dry dog food, Carnation Company, Los Angeles, California.

[2]Theracon, Inc., Topeka, Kansas.

Table 1. Treatment regimens

Regimen	Time continuum (days)	Diet
Control	1-38	Normal kennel diet
Starvation I	39-68	None
Refeeding I	69-88	70% carbohydrate diet
Starvation II	89-105	None
Refeeding II	106-136	70% carbohydrate diet

Table 2. Chemical composition of the high carbohydrate diet

Nutrients	Wet weight percentage
Moisture	60.79
Dry matter	39.25
Protein[a]	8.64
Fat[b]	1.15
Ash	1.53
Calcium	0.41
Phosphorus	0.29
Sodium[c]	0.14
Potassium	0.06
Nitrogen free extract	27.93

[a]Four parts horsemeat and one part casein.

[b]Corn oil.

[c]The sodium contents of the high carbohydrate diet and of the commercial diet were 350 mg/100 gm dry weight and 359 mg/100 gm dry weight, respectively.

Parungao lead system (McFee and Parungao, 1961) was used to obtain scalar recordings of the heart's electrical activity in three planes: X, Y, and Z. Each dog was restrained in sternal recumbency on a wooden table while 11 subdermal electrodes were inserted (see Figure 1) and the scalar recordings made. The X-lead consisted of two electrodes on each side of the chest. The negative pole of the X-lead was formed by two electrodes on the right side of the chest. One was at the level of the costochondral junction in the fifth intercostal space, and the other approximately 3 cm craniad to the first. The positive pole of the X-lead was formed by two electrodes placed on the left side of the chest in an analogous position. Two electrodes were used for the Y-lead. The negative electrode was placed immediately craniad to the point of the left shoulder. The positive electrode was placed on the lateral side of the left hindleg and posterior to the stifle. The Z-lead utilized three electrodes for the negative pole and one electrode for the positive pole. Two of the negative pole electrodes were placed bilaterally at the sternal junction of the seventh rib. The third electrode was placed crainally in the middle of the sternum at such a distance as to create an equilateral triangle with the other two electrodes. The electrode for the positive pole of the Z-lead was positioned dorsal to the spine of the seventh thoracic vertebra. Another electrode was used to ground the animal and was inserted subcutaneously in the right hindleg.

A VCG was produced in each of the three planes of the body, viz., frontal, transverse, and left sagittal (see Figure 2). The X and Y-leads were used to form the frontal plane loop, X and Z-leads to form the transverse plane loop, and Y and Z-leads to form the left sagittal plane loop.

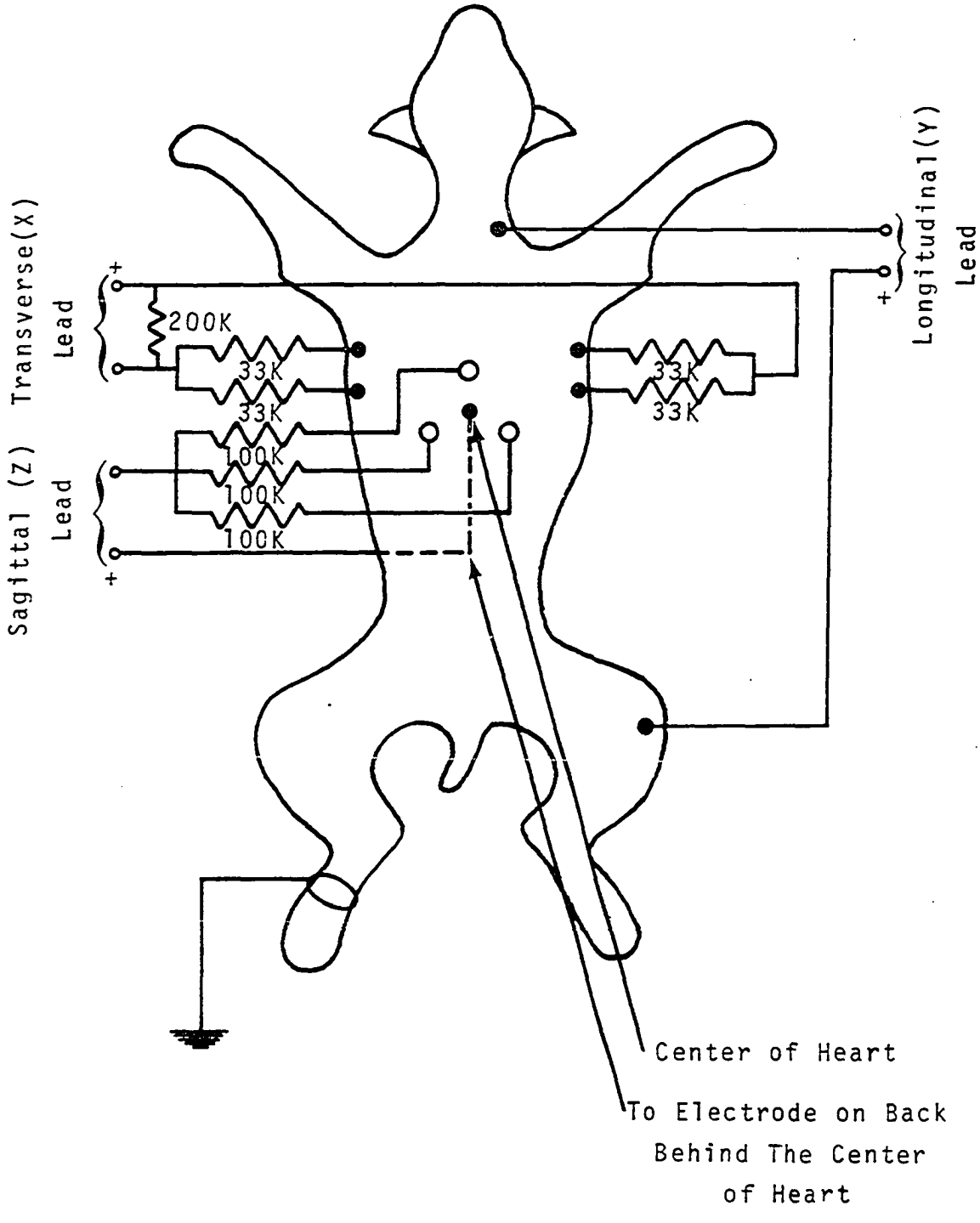

Figure 1. Ventral view of a dog with the electrode placement and circuitry used for recording the McFee-Parungao transverse (X), longitudinal (Y), and sagittal (Z) leads

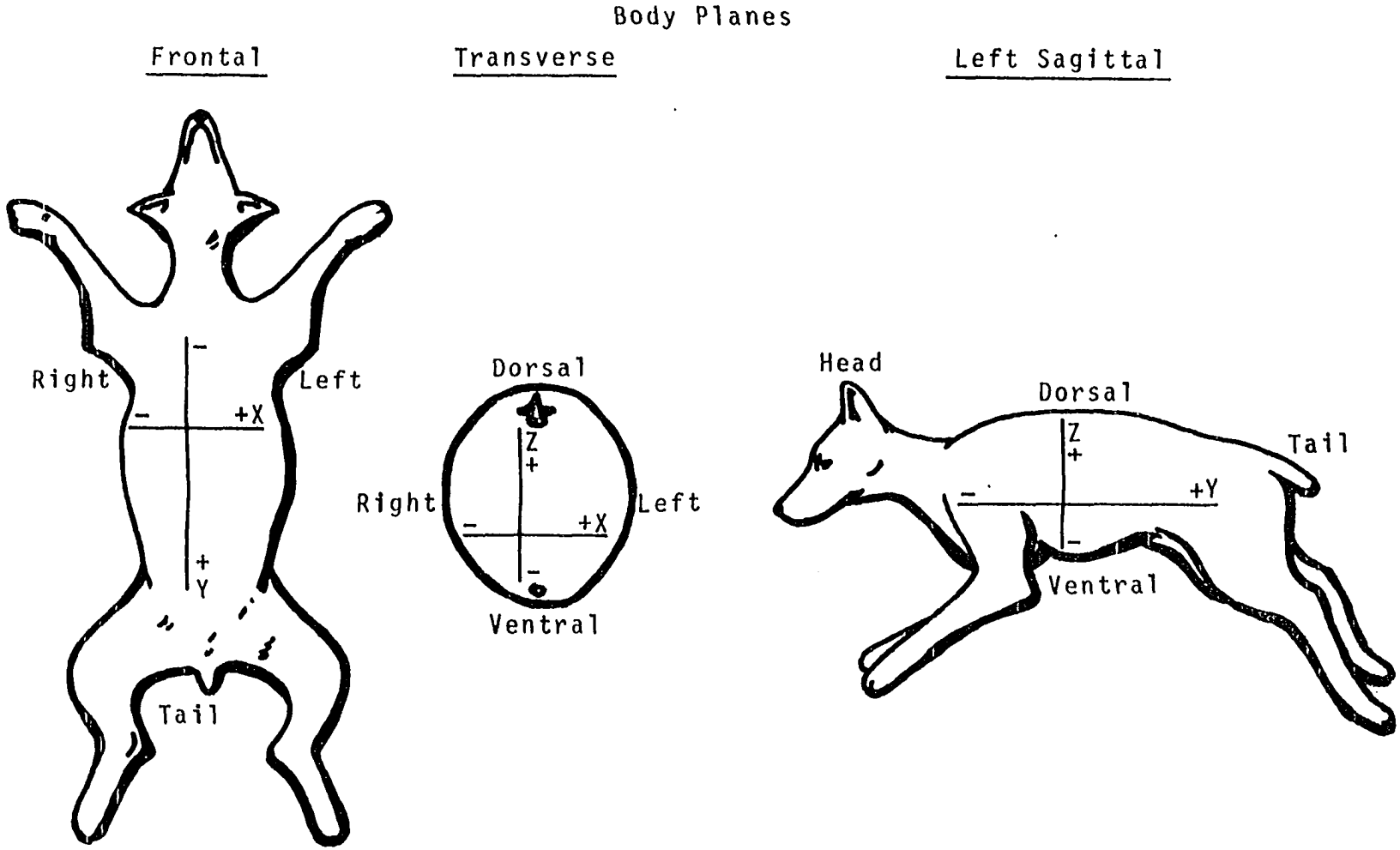

Figure 2. The three body planes in which vectorcardiograms were recorded with the leads and their polarity indicated

All three VCG leads were recorded simultaneously on paper[1] and magnetic tape.[2] The paper recording was used to detect arrhythmias while the magnetic tape was used to form the VCG. The scalar VCG was recorded on the tape at a speed of 60 inches per second (ips). It was later played back at 1 7/8 ips with one lead going into the horizontal and another lead going into the vertical plates of an oscilloscope[3] to monitor the VCG in a particular plane. When a representative loop was viewed, the footage location of the tape was noted and the loops in the other two planes were viewed using scalar recordings from the same cardiac cycle. If all three loops were acceptable they were recorded on an X-Y plotter[4] after the signal was conditioned by a low-pass filter[5] with a break frequency of 50 hertz.

Measurements made from the VCG included the direction of inscription of the QRS loop, the magnitude and angle of the maximum QRS vector in the frontal, transverse, and left sagittal planes, the angle of the maximum T wave vector in all three planes, and the magnitude and angle of the half-area vector of the QRS loop in the transverse and left sagittal planes. The angular scale used was that recommended by the 1967 Committee on Standardization in Electrocardiography of the American Heart Association. The magnitudes were measured from the isoelectric point. The maximum

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[1]Dynograph R411, Beckman Instruments, Schiller Park, Illinois.

[2]Model 5600, Honeywell, Chicago, Illinois.

[3]Type 502, Tektronix, Portland, Oregon.

[4]Model 7004b, Hewlett-Packard, St. Paul, Minnesota.

[5]Model 3202, Krohn-Hite, Cambridge, Massachusetts.

vectors were drawn from the isoelectric point to the point on the loop which was farthest away. The half-area vectors were drawn from the isoelectric point to a point on the loop which would bisect the loop into two equal areas. The half-area vectors were visually estimated and then altered using planimetry until the true vector was found.

Blood Samples

Jugular blood samples were drawn at the termination of each feeding regimen. The packed cell volume, hemoglobin concentration, total red blood cell count, total white blood cell count, differential white blood cell count, and total plasma protein concentration were measured.

The blood was collected and mixed with disodium ethylenediaminetetracetate[1] to prevent coagulation. The packed cell volume was determined by the microhematocrit technique by centrifuging[2] two blood filled capillary tubes[3] plugged with clay.[4] The centrifuge ran at approximately 13,500-25,000 g for 5 minutes. The two values on each dog were averaged.

Plasma protein concentration was estimated using plasma from the microhematocrit tubes and a clinical refractometer.[5]

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[1]Cambridge Chemical Products, Inc., Dearborn, Michigan.

[2]International Equipment Company, Model MB, Needham Heights, Massachusetts.

[3]Capilets, American Hospital Supply Corp., Miami, Florida.

[4]Critoseal, Aloe Scientific, St. Louis, Missouri.

[5]TS meter, American Optical Corp., Buffalo, New York.

Total hemoglobin concentration was measured using cyanmethemoglobin reagent[1] and standard[2] and a spectrophotometer.[3]

Smears of the blood were made and stained with a modified Wright's stain.[4] Microscopic[5] examination of the blood smears under oil immersion (1000X) was used to make the differential cell counts.

Total red and white blood cell counts were made on an electronic cell counter.[6]

Femoral Artery Stretch-Tension Characteristics

Approximately 3 months after the dogs were euthanatized the femoral artery segments were thawed and placed in a constant temperature bath[7] of 38 C (Figures 3 and 4). The segment was then connected to a stationary Plexiglas block by a stainless steel rod passing through the lumen of the vessel. Another rod through the vessel lumen was connected to a movable force-displacement transducer.[8] The vessel was stretched by moving the force-displacement transducer (connected to a rod through the vessel

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[1]Hycel, Inc., Houston, Texas.

[2]Hycel, Inc., Houston, Texas.

[3]Model B, Beckman Instruments, Inc., Fullerton, California.

[4]Camco Quik Stain, Scientific Products Company, Evanston, Illinois.

[5]Series 10, American Optical Company, Buffalo, New York.

[6]Coulter Counter, Coulter Electronic, Inc., Hialeah, Florida.

[7]Model FJ, Haake, Berlin, West Germany.

[8]FtO3c, Grass Instruments, Quincy, Massachusetts.

Figure 3. Equipment used for measuring the stretch-tension characteristics of femoral artery segments

A - motorized micrometer

B - force-displacement transducer

C - constant temperature saline bath

D - function generator for controlling the step rate of the micrometer

E - power supply for the force-displacement transducer

F - strip chart recorder

G - power supply and control panel for the motor driven micrometer

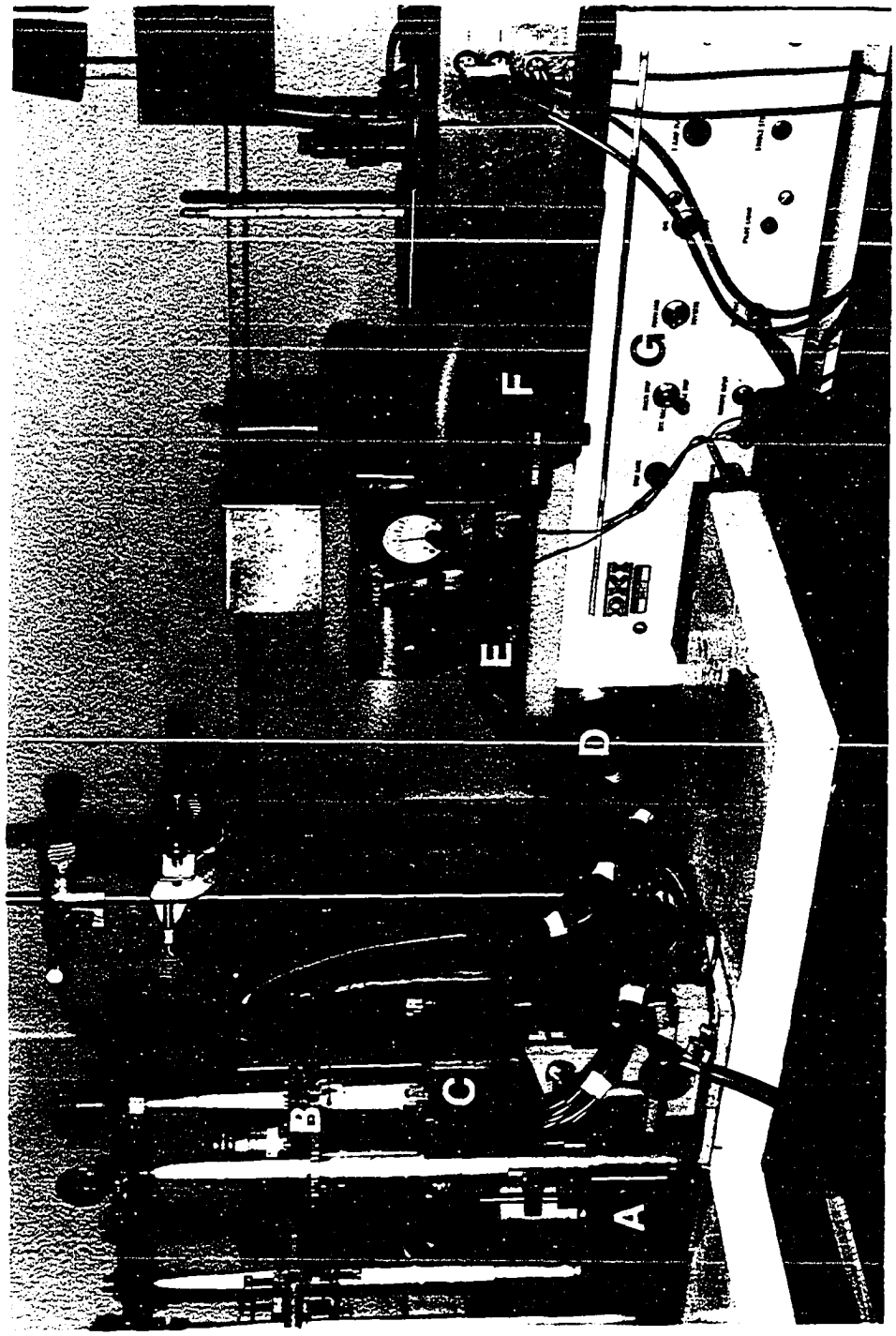

A
B
C
D
E
F
G

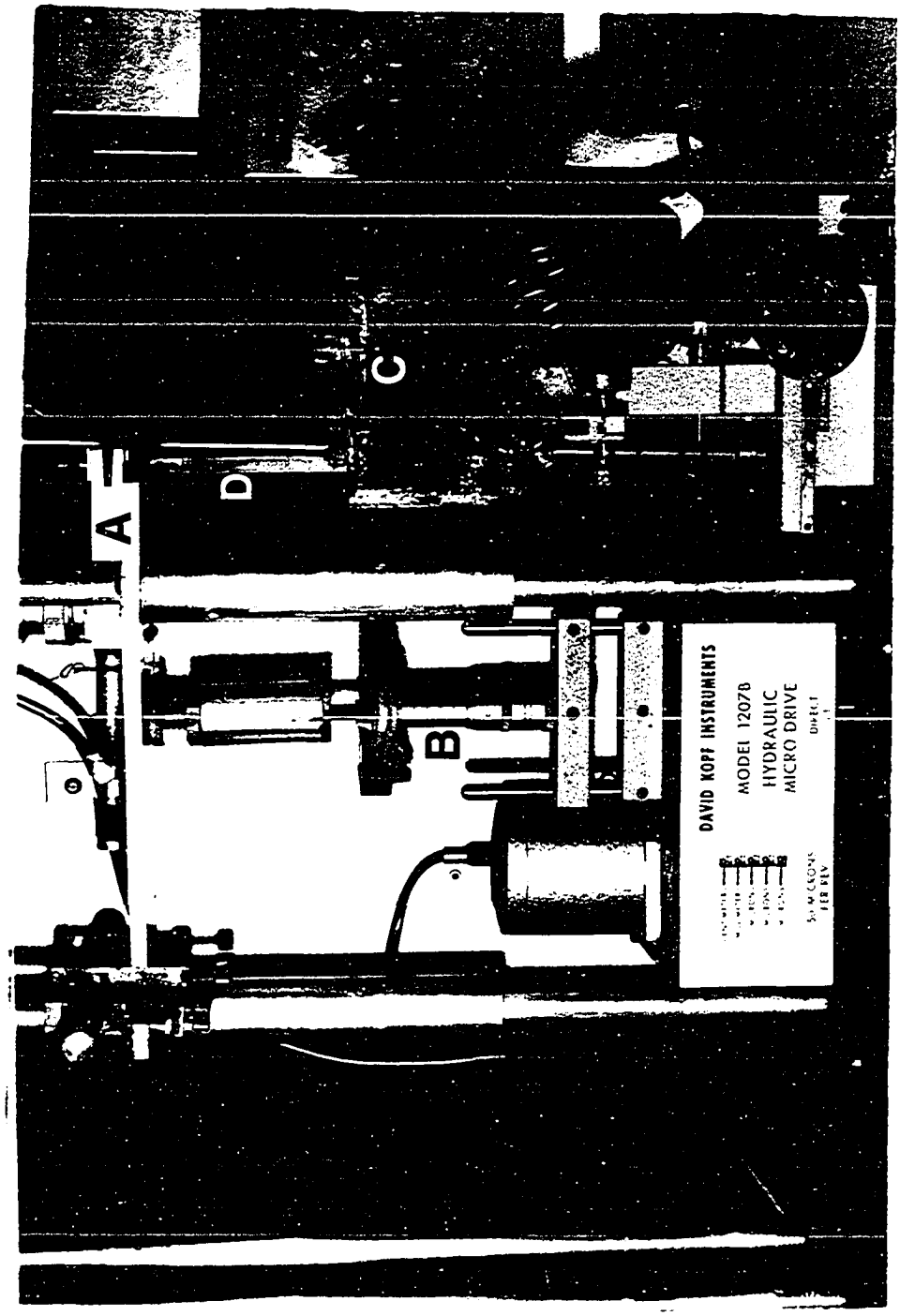
Figure 4. Equipment for stretching and measuring tension in femoral artery segments

A - force-displacement transducer

B - motor drive micrometer

C - constant temperature saline bath

D - Plexiglas arm connecting the force-displacement transducer to the upper rod inside the artery lumen


A
B
C
D
DAVID KOPF INSTRUMENTS
MODEL 1207B
HYDRAULIC
MICRO DRIVE

lumen) at a constant rate (15 microns per second) by a motorized micrometer.[1] The transducer simultaneously measured the amount of tension developed on a strip chart recorder.[2] All vessels were stretched to 100 gm and then rapidly returned to the zero tension position. Following a 5 minute waiting period the vessels were stretched a second time and following another 5 minute wait a third time. The vessel was then removed from the chamber and placed in a saline filled pan with a pin through its lumen to hold the vessel in a vertical position. A dissecting microscope[3] with a filar micrometer eyepiece[4] was used to visually estimate the thickness of the vessel wall at several points on its circumference. The wall-thickness measurements from one vessel were averaged together. The vessel was then slit longitudinally and held flat in the saline bowl by placing a glass slide on top of it. The initial length and circumference of the vessel were then measured visually by the dissecting microscope and filar eyepiece micrometer.

From the strip chart recordings (Figure 5), values of stretch (microns) were calculated at tensions of 1.25 gm increments from 1.25 to 20.0 gm, 2.50 gm increments from 22.5 to 37.5 gm, and 6.25 gm increments from 43.75 to 100.0 gm. The stretch was calculated by knowing the chart speed and thus the time necessary to reach a particular tension and the step rate of the micrometer. These micron values were then corrected for

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[1] 1207S Stepping Micro-drive, David Kopf Instruments, Tujunga, Calif.

[2] Bausch and Lomb, Inc., Rochester, New York.

[3] Series 53, American Optical Company, Buffalo, New York.

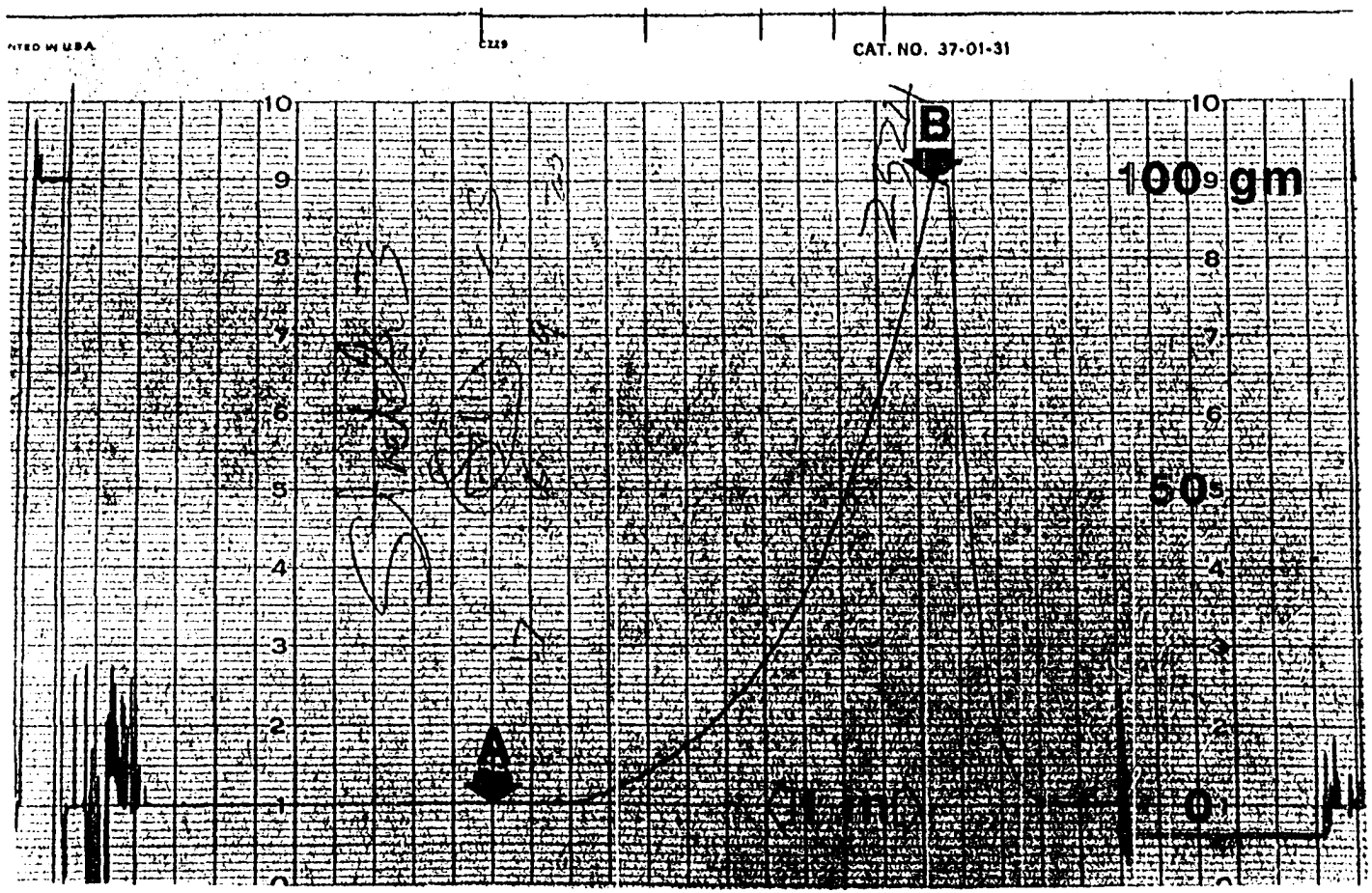
[4] Micrometer eyepiece, Bausch and Lomb, Inc., Rochester, New York.

Figure 5. An example strip chart recording of a femoral artery segment stretch

A - start of the motorized micrometer which performs the stretch

B - stop of the motorized micrometer at a vessel tension of 100 g

Paper speed of the recorder was 1 in/min and the step rate of the micrometer was approximately 15 microns/sec


CAT. NO. 37-01-31
B
A
100 gm
50
10
9
8
7
6
5
4
3
2
1
0

the amount of transducer movement which occurred due to the tension. The microns were then converted into the vessel circumference assuming that the vessel had a "racetrack" configuration (Figure 6).

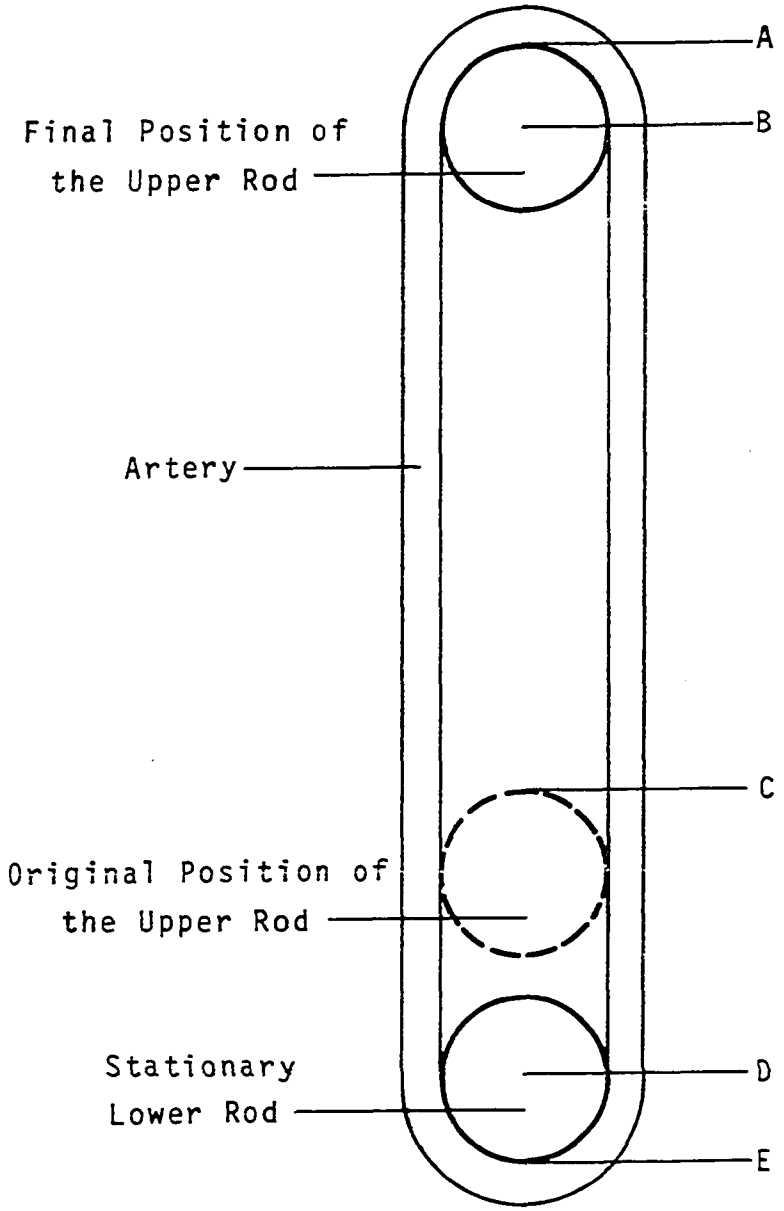
In order to calculate the vessel circumference during the stretch it was necessary to know how far the two stainless steel rods were separated when the micrometer was at zero microns. To do this a piece of stainless steel tubing was placed in the position that a vessel would be and the step micrometer was started. As soon as tension was perceptible the micrometer was read and the difference between the known diameter of the stainless steel tube and the micrometer reading was the distance that the rods were separated. The distance of rod separation was then added to the micron value calculated from the strip chart (minus the transducer movement), doubled, and added to the two hemicircumference of the rods in the vessel lumen to yield the vessel circumference (Figure 6). The vessel circumference measured at each increment of tension was divided by the initial circumference to yield the strain ratio:

Strain ratio = circumference (C) (cm)/initial circumference (Co) (cm)

The tension in grams was converted into Langrangian stress by dividing the tension in grams by the product of the initial wall-thickness and the length of the vessel at zero tension:

$$\text{Stress } (gm/cm^2) = \frac{\text{tension (gm)}}{2[\text{wall-thickness cm x length cm}]}$$

Table 3 lists the graphs made from the stretch-tension data on each vessel. Graphs 1 and 2 utilize the original data without normalization for wall-thickness or the initial circumference. Graphs 3 and 4 examine the physical properties of the entire vessel wall. Graphs 5 and 6 give



Internal circumference=2[(CE) + (AC) - (DE) -(AB)] + π(DE+AB)

AC=(total microns-transducer movement)

Figure 6. An end-on view of an artery in the "race-track" geometry assumed during stretching and the formula for calculating internal circumference

Table 3. Graphs used to examine the stretch-tension data

Graph number	Data plotted on the abscissa	Data plotted on the ordinate
1	Circumference (cm)	Tension (gm)
2	Tension (gm)	Slope I (Slope of Graph 1 per 0.2 mm of circumference increase)
3	Strain ratio	Tension (gm)
4	Tension (gm)	Slope II (Slope of Graph 3 per 0.02 strain ratio increase)
5	Strain ratio	Stress (gm/cm^2)
6	Stress (gm/cm^2)	Slope III (Slope of Graph 5 per 0.02 strain ratio increase)

information about the physical properties of the vessel wall material per unit of area.

The right and left femoral arteries were removed sequentially from five mongrel dogs and frozen using the same technique that was applied to the beagles. These vessels were likewise thawed and stretched to examine whether or not there was a difference in mechanical properties between the femoral arteries from the two sides of the body.

Statistical analysis was performed on all the data with the assistance of the statistical consultants and the analysis of variance and paired and unpaired t-tests.

Circumference-tension relationship

From the circumference-tension data the value of tension in grams was extrapolated for even numbers of hundred microns. That is, the values for grams tension were extrapolated for even values of stretch at increments of 200 microns (e.g., 7000, 7200, 7400, etc.).

The extrapolated values for grams were subtracted from the next higher value to determine slope I or the increment of tension per 0.02 mm (200 microns) of stretch. The difference in grams was then plotted against the greater of the two tension values used to achieve the difference. These values of tension-slope I were fitted by various polynomials from X^1 to X^6. Using X^2 as a reasonable fit without complexity, values of slope I were calculated at 0, 20, 40, 60, 80, and 100 grams tension. Paired and unpaired t-tests were used to evaluate the circumference-tension and the tension-slope I relationships for each group of dogs.

Strain ratio-tension relationship

Values of tension were extrapolated for strain ratio levels from 1.00 at increments of 0.02 to the point where no more data were available for that vessel. Data were present on all vessels up through a strain ratio of 1.14 and no values were available for strain ratios above 1.38. Paired and unpaired t-tests were used to analyze the data.

The difference in tension between one extrapolated value and the next highest value which occurred due to a strain increase of 0.02 was calculated (slope II). The difference in tension at equal increments of strain ratio was plotted against the lower value of tension used to obtain the difference. Using various degrees of polynomial equations it was found that a first degree equation could be used for the data from the beagles

but a second degree was necessary for the data from the mongrels. Utilizing the second degree polynomial for both groups of dogs values of slope II were calculated for tensions of 0, 20, 40, 60, 80, and 100 gm. Paired and unpaired t-tests were performed on these values.

Strain ratio-stress relationship

The tension was converted to Langrangian stress and the circumference to strain ratio. Stress values were extrapolated for values of strain ratio beginning with 1.00 and increasing by increments of 0.02 until the original data were exhausted. Not all dogs were stretched to the same value of strain ratio since 100 gm tension was the stopping point. Thus, there were missing data at strains over 1.14 and none of the vessels were strained over 1.38. The values for stress were then compared at equal increments of strain by a paired t-test.

From the described strain ratio-stress data the increment of stress increase per 0.02 strain ratio increase (slope III) was calculated by subtracting stress values at succeedingly greater strain ratios. This increment in stress per 0.02 strain ratio increase was essentially the slope of the strain ratio-stress relationship at that particular strain ratio and was similar to Young's modulus used by other researchers. A plot was then made of slope III and the lower stress value used to obtain the difference. These data were then fit by polynomials ranging from X^1 to X^6. It was found that a second order polynomial fit the data. Using these polynomials, values for slope III were calculated for the following levels of stress: 0, 500, 1000, 1500, 2000, and 2500 gm/cm^2. Comparisons were made between groups by using paired and unpaired t-tests.

Ventricular Weights

After the dogs were euthanized their hearts were removed from the thorax. The great vessels and atria were removed down to the atrio-ventricular ring. The ventricular cavity was washed free of blood and the right ventricular free wall was cut away at its point of attachment to the septum. The left ventricle and septum were weighed together and the right ventricular free wall was weighed separately. Their combined weight became the total ventricular weight. The following ratios were calculated: right ventricular weight/total ventricular weight; left ventricular and septal weight/total ventricular weight; and total ventricular weight/body weight. Statistical analysis was preformed using the unpaired t-test.

RESULTS AND DISCUSSION

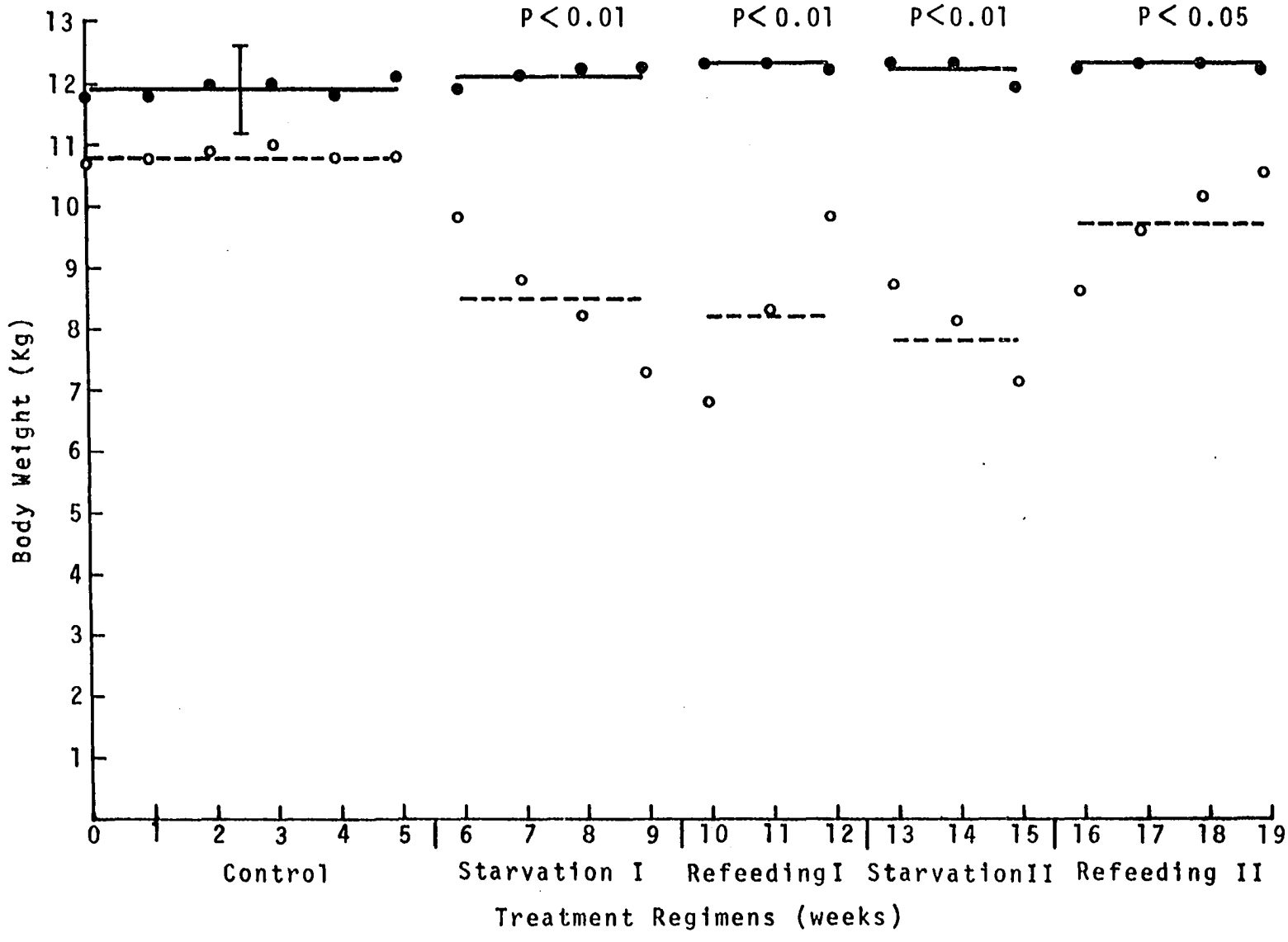
Body Weight

The body weights were determined weekly and the values are listed in Table A-1 and means are shown in Figure 7. The analysis of variance (Table A-2) revealed several significant effects. The males had a consistently greater body weight than females producing a significant ($P<0.01$) sex effect. The control dogs gradually increased in body weight from 11.9 ± 0.7 kg at the control regimen to 12.3 kg at the refeeding II regimen. This increase in the control group body weight was not significant statistically. The treatment dogs started the experiment with a body weight of 10.8 kg during the control regimen and then decreased to a mean of 8.5 kg during starvation I. The treatment dogs then showed a rise in weekly body weights during the refeeding I regimen but the mean value was 8.2 kg, less than the mean for starvation I. Starvation II caused a loss of more body weight to a mean of 7.8 kg and refeeding II produced a rise in body weight to a mean of 9.7 kg. The overall control group mean body weight was significantly greater than that of the overall treatment group during regimens starvation I ($P<0.01$), refeeding I ($P<0.01$), starvation II ($P<0.01$), and refeeding II ($P<0.05$). The overall treatment group mean for the control regimen was significantly greater than the means at all subsequent regimens. The males of the treatment group showed a significant decrease in their mean body weight during the starvation I, refeeding I, starvation II, and refeeding II regimens. The female treatment dogs did not show significant changes in their body weight but did show the

Figure 7. Mean body weights in control dogs maintained on a commercial diet and in treatment dogs starved and refed a high carbohydrate diet; values represent weekly means; the pooled standard deviation is indicated

Control dogs:
Weekly mean ●
Regimen mean ——

Treatment dogs:
Weekly mean ○
Regimen mean - - - -


P < 0.01
P < 0.01
P < 0.01
P < 0.05
Body Weight (Kg)
13
12
11
10
9
8
7
6
5
4
3
2
1
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
Control
Starvation I
Refeeding I
Starvation II
Refeeding II
Treatment Regimens (weeks)

same general pattern of losses during starvation and gains during refeeding that the males did.

The treatment dogs had a 37% decrease in body weight during the 30 day starvation I from 10.8 kg on week #5 to 6.8 kg on week #10. Starvation II (16 days in duration) produced a 28% decrease in the treatment group's body weight (9.8 kg at week #12 to 7.1 kg at week #15). Wilhelmj and McCarthy (1963) reported a similar rate of body weight loss in their dogs and stated that most healthy dogs can fast for as long as 3 to 4 weeks. Comparatively, humans do not lose weight as rapidly as the dog (Keys et al., 1950; Garnett et al., 1969; Sandhofer et al., 1973).

Hematology

The erythrocytes within the circulating blood were evaluated using packed cell volume (PCV), hemoglobin concentration, total red blood cell counts (RBC), mean corpuscular volume (MCV), and mean corpuscular hemoglobin concentration (MCHC). Normal values for the dog are shown in Table 4.

Blood hemoglobin concentration

The individual values and group means for the hemoglobin concentration can be found in Table A-3. The analysis of variance for the hemoglobin concentration is shown in Table A-4. Significant ($P<0.01$) values for mean squares were found for group, regimen, and group-regimen interaction effects. The regimen effect was considered to originate from normal biologic variations that occur from one sampling time to another. The decreased hemoglobin concentrations seen in the treatment group with and subsequent to the first starvation period were believed to lead to the

Table 4. Normal hematological data for dog[a]

Type of measurement	Normal range
PCV (%)	37-55
WBC (cells X 10^3/cu mm)	6-17
Neutrophil, segmented (cells X 10^3/cu mm)	3-11.5
Neutrophil, band (cells X 10^3/cu mm)	0-0.3
Eosinophils (cells X 10^3/cu mm)	0.1-1.2
Basophils (cells X 10^3/cu mm)	Rare
Monocytes (cells X 10^3/cu mm)	0.1-0.4
Lymphocytes (cells X 10^3/cu mm)	1.0-4.8
Hemoglobin (gm/100 ml)	12-18
RBC (cells X 10^6/cu mm)	5.5-8.5
MCV (cu microns)	60-77
MCHC (%)	32-36
Plasma protein (gm/100 ml)	6.0-7.8

[a]Kirk, 1974.

significant group and group-regimen interaction effects found in the analysis of variance.

The mean hemoglobin concentration for the control and treatment groups at the control regimen were 18.5 ± 1.0 and 18.3 gm/100 ml, respectively. The means for the starvation I, refeeding I, starvation II, and refeeding II regimens of the control group were 18.7, 18.4, 18.1, and 17.2 gm/100 ml, respectively. The mean for the refeeding II regimen was

significantly ($P<0.01$) less than the control regimen mean for this group. The mean hemoglobin concentrations for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 17.7, 14.4, 16.9, and 13.0 gm/100 ml, respectively. The treatment group means for the refeeding I, starvation II, and refeeding II regimens were significantly ($P<0.01$) less than the control regimen mean for that group.

The control group means were significantly greater than the treatment group means for the starvation I ($P<0.05$), refeeding I ($P<0.01$), starvation II ($P<0.05$), and refeeding II ($P<0.01$) regimens.

Red blood cell count

Table A-5 shows the individual values and regimen means for the two groups of beagle dogs. The analysis of variance for the RBC counts is given in Table A-6. Significant mean squares were found for group ($P<0.05$) and regimen ($P<0.01$). The regimen effect was considered to indicate that the red blood cell counts varied significantly from one regimen to another. The group effect was interpreted to mean that the values for red blood cell counts were different for the two groups of dogs.

The initial control regimen means for the control and treatment groups were 7.69 ± 0.30 and 7.55 cells X 10^6/cu mm, respectively. The control group showed a significant decline ($P<0.05$) in their red blood cell counts to 6.82 cells X 10^6/cu mm during the starvation I regimen even though they were not starved. The RBC count at the end of refeeding I was 7.32 cells X 10^6/cu mm which was not significantly different from the control regimen mean. The mean values for starvation II and refeeding II

were 6.20 and 6.37 cells X 10^6/cu mm, respectively, and significantly ($P<0.01$) less than the control regimen mean.

The treatment group means for RBC counts at the starvation I, refeeding I, starvation II, and refeeding II regimens were 6.30, 6.24, 5.80, and 5.00 cells X 10^6/cu mm respectively. The control regimen mean for the treatment group was significantly ($P<0.01$) greater than all subsequent mean values for that same group of dogs. The control group means were significantly greater than the treatment group means at the refeeding I ($P<0.05$) and refeeding II ($P<0.01$) regimens.

The significant group effect in the analysis of variance probably stems from the fact that the mean RBC counts for the treatment group were consistently lower than the counts for the control group.

Packed cell volume

Table A-7 lists the individual values for the PCV and group means. The group means are also graphically displayed in Figure 8. The analysis of variance is shown in Table A-8. Significant ($P<0.01$) mean squares were found for group, regimen, and group-regimen interaction. Variation from one regimen to another would account for the significant regimen effect. The decrease in the treatment group's PCV during the refeeding I and refeeding II regimens probably accounts for the significant group and group-regimen interaction effects.

The control regimen means for the control and treatment groups of beagle dogs were both 49 ± 3%. The control group means for the starvation I, refeeding I, starvation II, and refeeding II regimens were 50, 51, 48, and 50%, respectively. None of these values were significantly different from the control regimen mean.

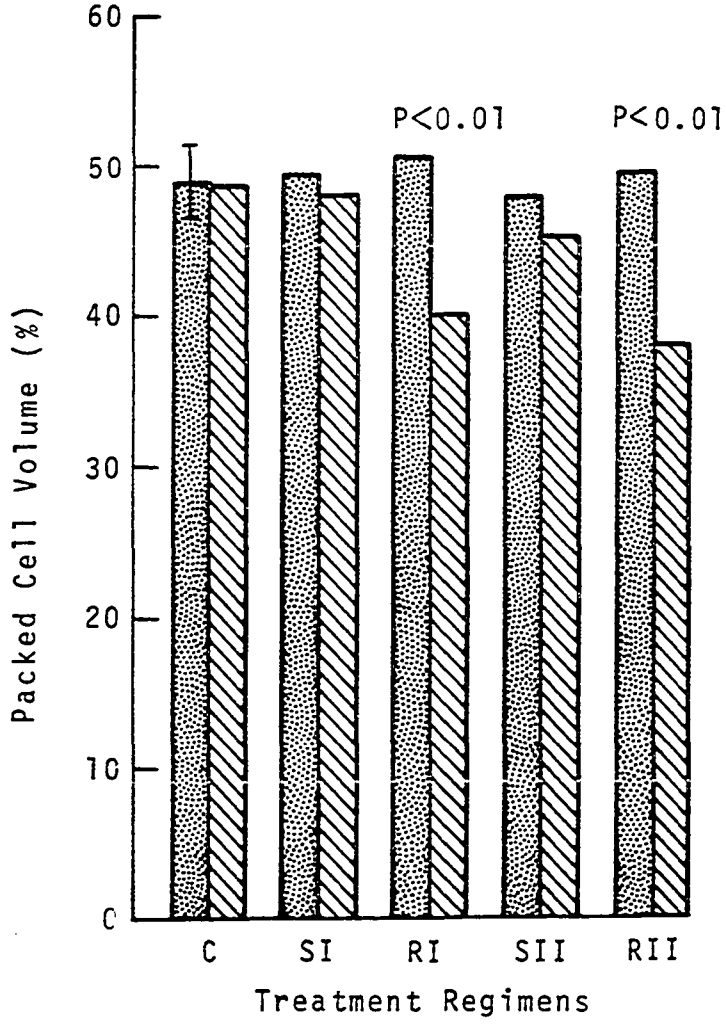


Figure 8. Mean packed cell volumes in control dogs maintained on a commercial diet and in treatment dogs starved and refed a high carbohydrate diet; values represent means at the end of each regimen; the pooled standard deviation is indicated

C - control regimen
SI - starvation I regimen
RI - refeeding I regimen
SII - starvation II regimen
RII - refeeding II regimen
Control dogs ▨
Treatment dogs ▧

The treatment group means for regimens starvation I, refeeding I, starvation II, and refeeding II were 48, 40, 45, and 38%, respectively. The means for refeeding I, starvation II, and refeeding II were significantly ($P<0.01$) less than the control regimen mean for the treatment group.

The control group means for PCV were significantly higher than those for the treatment group at the refeeding I ($P<0.01$), starvation II ($P<0.05$), and refeeding II ($P<0.01$) regimens (see Figure 8).

Undernutrition has been accompanied by anemia in man (Keys et al., 1950). It has been reported that short periods of starvation (7-15 days) will actually elevate the PCV and hemoglobin concentration in swine (Kornegay et al., 1964; Furugouri, 1973). Refeeding of the swine, however, was accompanied by a decline in the hemoglobin concentration (Furugouri, 1973). This elevation during starvation and decline during refeeding seems paradoxical but it is probably the result of blood volume changes. The blood volume of man, dog, and rat has been found to decrease during periods of undernutrition (Keys et al., 1950; Haxhe, 1967a; Kutscher, 1971). The dehydration was related to natriuresis that has been observed during the early stages of fasting (Bloom, 1962; Katz et al., 1968; Veverbrants and Arky, 1969; Boulter et al., 1973). If anemia and dehydration appear concurrently the decreased PCV and hemoglobin concentration associated with anemia may not be evident because of a decreased blood volume.

Refeeding and especially refeeding with carbohydrates will halt the natriuresis of fasting (Bloom, 1962; Katz et al., 1968; Hoffman et al., 1971; Veverbrants and Arky, 1969). Blood volume will return to normal

during refeeding and any anemia will then become apparent. Blood volume in this experiment was not measured. From previous work and the pattern of changes seen in the PCV, hemoglobin concentration, and total red blood cell counts it can be postulated that the blood volume decreased during starvation masking alterations in the above erythrocyte measurements. Refeeding was probably accompanied by a return of the blood volume to near normal levels and the anemic tendency that had developed became apparent.

Mean corpuscular hemoglobin concentration

Table A-9 lists the individual values and the group means for each regimen. Table A-10 shows the analysis of variance for the mean corpuscular hemoglobin concentration (MCHC). Statistical significance was found for mean squares of group ($P<0.05$) and regimen ($P<0.01$). The group effect was probably due to the fact that the values for MCHC in the treatment group were consistently less than the values for the control group. The regimen effect indicated that the MCHC varied significantly from one regimen to another.

The mean values for the control and treatment groups at the control regimen were 38 ± 1 and 37%, respectively. Subsequent values for the control group in the starvation I, refeeding I, starvation II, and refeeding II regimens were 38, 36, 38, and 35%, respectively. The mean values for the refeeding I and refeeding II regimens of the control group were significantly ($P<0.01$) less than the mean MCHC at the control regimen. The mean MCHC for the starvation I, refeeding I, starvation II, and refeeding II regimens of the treatment group were 37, 36, 37, and 34%, respectively. The only regimen mean for the treatment group that was significantly ($P<0.01$) less than the control regimen mean was that for

refeeding II. At no point was there a significant difference between the regimen means for the control and treatment groups.

Mean corpuscular volume

Individual values and group means at each regimen are given in Table A-11. The analysis of variance for the mean corpuscular volume is shown in Table A-12. The only mean square of statistical significance ($P<0.01$) was that for the regimen effect. This was interpreted to mean that the mean corpuscular volume varied significantly from one regimen to another.

The mean values for the control regimen in the control and treatment groups were 65 ± 9 and 66 cu microns, respectively. The mean corpuscular volume (MCV) for the starvation I, refeeding I, starvation II, and refeeding II regimens in the control group of beagle dogs were 72, 71, 80, and 78 cu microns, respectively. The means for the starvation II and refeeding II regimen were significantly ($P<0.01$) greater than the control regimen mean for the control group. The mean values for the treatment group of beagle dogs in the starvation I, refeeding I, starvation II, and refeeding II regimens were 77, 65, 80, and 77 cu microns, respectively. The control regimen mean for the treatment group was found to be statistically less than the subsequent starvation I ($P<0.05$), starvation II ($P<0.01$), and refeeding II ($P<0.05$) regimen means. No difference was found between means for the control and treatment groups at the various regimens.

Although the values for MCV and MCHC varied quite a bit both groups of dogs had similar values. Consequently, starvation-refeeding was not considered to alter either the MCV or the MCHC.

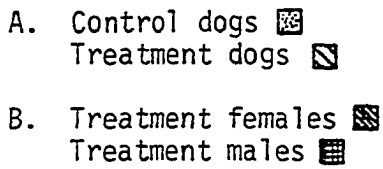
Plasma protein concentration

The individual determinations and group means for the plasma protein concentrations at the end of each regimen are listed in Table A-13. The analysis of variance is shown in Table A-14. The regimen means for the control and treatment groups are also shown in Figure 9A.

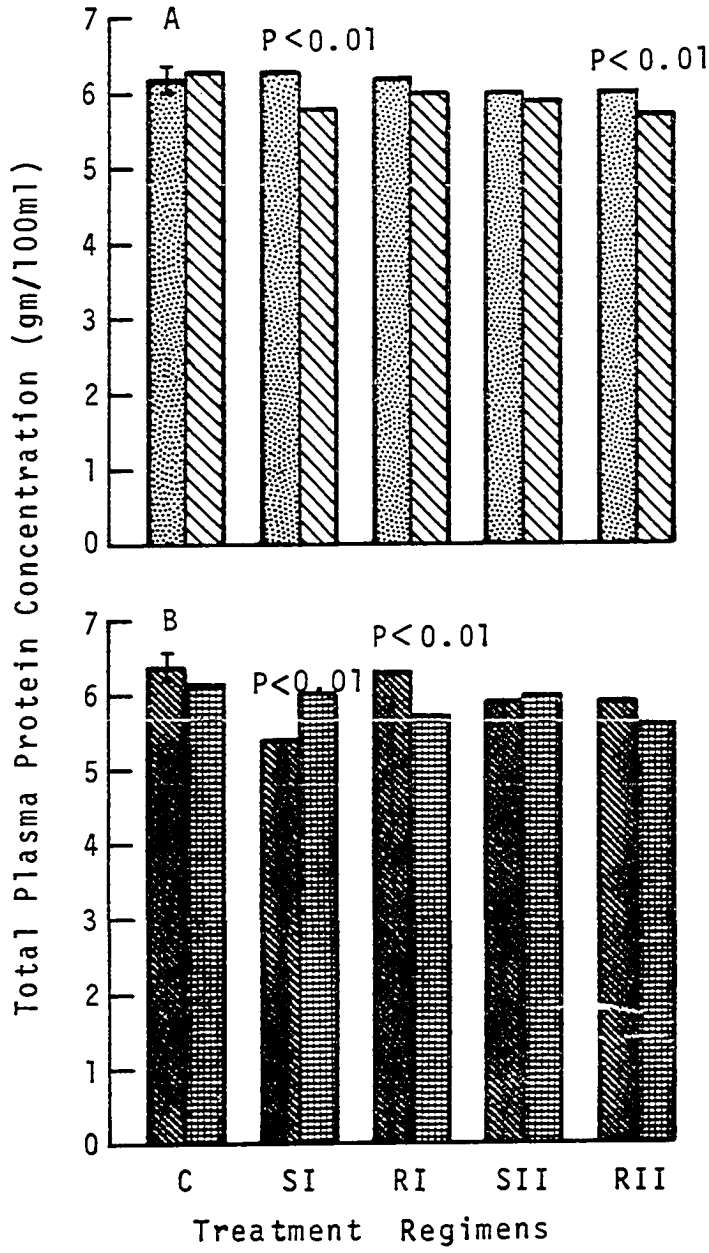
The analysis of variance revealed statistical significance ($P<0.01$) for the following mean squares: regimen; group-regimen; sex-regimen; and sex-group-regimen. The significant regimen effect was interpreted to mean that the plasma protein concentration varied significantly from one regimen to another.

The group-regimen interaction was probably due to the significant ($P<0.01$) decline in the treatment group's plasma protein concentration during the starvation I and refeeding II regimens. The control regimen means for the control and treatment groups were 6.2 ± 0.2 and 6.3 gm/100 ml, respectively. At the end of the starvation I regimen the control group mean was 6.3 gm/100 ml while the mean for the treatment group had declined to 5.8 gm/100 ml. The difference between the control and treatment group means at the end of the starvation I regimen was significant ($P<0.01$). The treatment group's plasma protein concentration rose during the refeeding I regimen towards normal and ended the regimen with a mean of 6.0 gm/100 ml. Starvation II produced another slight fall in the treatment group plasma protein concentration to 5.9 gm/100 ml. The mean for the treatment group at the end of the refeeding II regimen was 5.7 gm/100 ml which was significantly ($P<0.01$) less than the control group mean for the same regimen (6.0 gm/100 ml). The plasma protein concentration for the control group did not change significantly from its control

Figure 9. Mean plasma concentrations of total protein in control dogs maintained on a commercial diet and in treatment dogs starved and refed a high carbohydrate diet; values represent means at the end of each regimen; the pooled standard deviation is indicated



C - control regimen
SI - starvation I regimen
RI - refeeding I regimen
SII - starvation II regimen
RII - refeeding II regimen


A
P<0.01
P<0.01
B
P<0.01
P<0.01
Total Plasma Protein Concentration (gm/100ml)
0
1
2
3
4
5
6
7
C
SI
RI
SII
RII
Treatment Regimens

value of 6.3 gm/100 ml. The treatment group began the experiment with a control regimen mean of 6.3 gm/100 ml. Subsequent regimen means for the treatment group were significantly ($P<0.01$) less than the control regimen mean.

The sex-regimen interaction indicated that the two sexes did not vary in an identical manner from one regimen to another. The sex-group-regimen interaction was interpreted to indicate that the sexes responded differently within each group. The most apparent sex difference was within the treatment group at the starvation I and refeeding I regimens (see Figure 9B). The female and male means for the treatment group at the control regimen were 6.4 and 6.2 gm/100 ml, respectively. Starvation I was associated with a severe decline in the plasma protein concentration for the treatment females to 5.4 gm/100 ml while the males did not vary significantly from their control value with a mean of 6.1 gm/100 ml. A Student's t-test between the two sexes revealed that they were significantly different ($P<0.01$). The refeeding I regimen caused the opposite effects in the two sexes with the female's plasma protein concentration rising to 6.3 gm/100 ml and the male's declining to 5.8 gm/100 ml. The two sex means were again significantly different ($P<0.01$), using the Student's t-test. Starvation II caused a decline in the female's plasma protein concentration to 5.9 gm/100 ml while the male's rose above the preceding regimen mean to 6.0 gm/100 ml. Refeeding II was associated with an unchanged plasma protein concentration in the females and a decline in the males to 5.6 gm/100 ml.

The control regimen mean for the treatment females was significantly ($P<0.01$) greater than the means for starvation I, starvation II, and refeeding II.

The variations in the female and male treatment group's plasma protein concentrations are difficult to explain in a believable manner. It might be reasoned that starvation would either: 1) lead to a decrease in the plasma protein concentration as the result of protein catabolism and reduced synthesis by the liver; or 2) produce dehydration and the reduced blood volume would then tend to elevate the remaining protein concentration. The female treatment dogs seemed to follow the former reasoning of decreased plasma protein concentration during starvation regimens. The male treatment dogs, however, responded in a manner suggestive of the later mechanism with the greatest decline in plasma protein concentration occurring during periods of refeeding and consequent rehydration. Since neither blood volumes nor the albumin and globulin content of the plasma proteins were measured it is not possible to give a definitive reason for the sex difference observed during the starvation I and refeeding I regimens.

Total white blood cell count

The individual dog values and the group means for the total white blood cell (WBC) counts are shown in Table A-15. The analysis of variance for the WBC counts can be found in Table A-16. Normal values for the dog are given in Table 4. The mean squares which were statistically significant were group ($P<0.01$), sex ($P<0.05$), regimen ($P<0.01$), and group-

regimen ($P<0.01$) interaction. The total height of the columns in Figure 10 shows graphically how the WBC counts changed during the experiment.

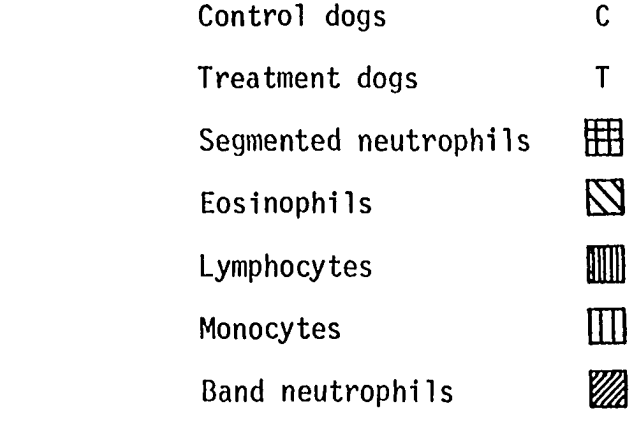
The significant group effect in the analysis of variance indicates that, as a whole, the control group had higher WBC counts than the treatment group. The sex effect, although not highly significant, lends support for the observation that the males usually had higher WBC counts than the females. The significant regimen effect in the analysis of variance was interpreted to mean that the WBC counts changed significantly from one regimen to another. The group-regimen interaction was considered to be the result of unequal group changes with the various regimens.

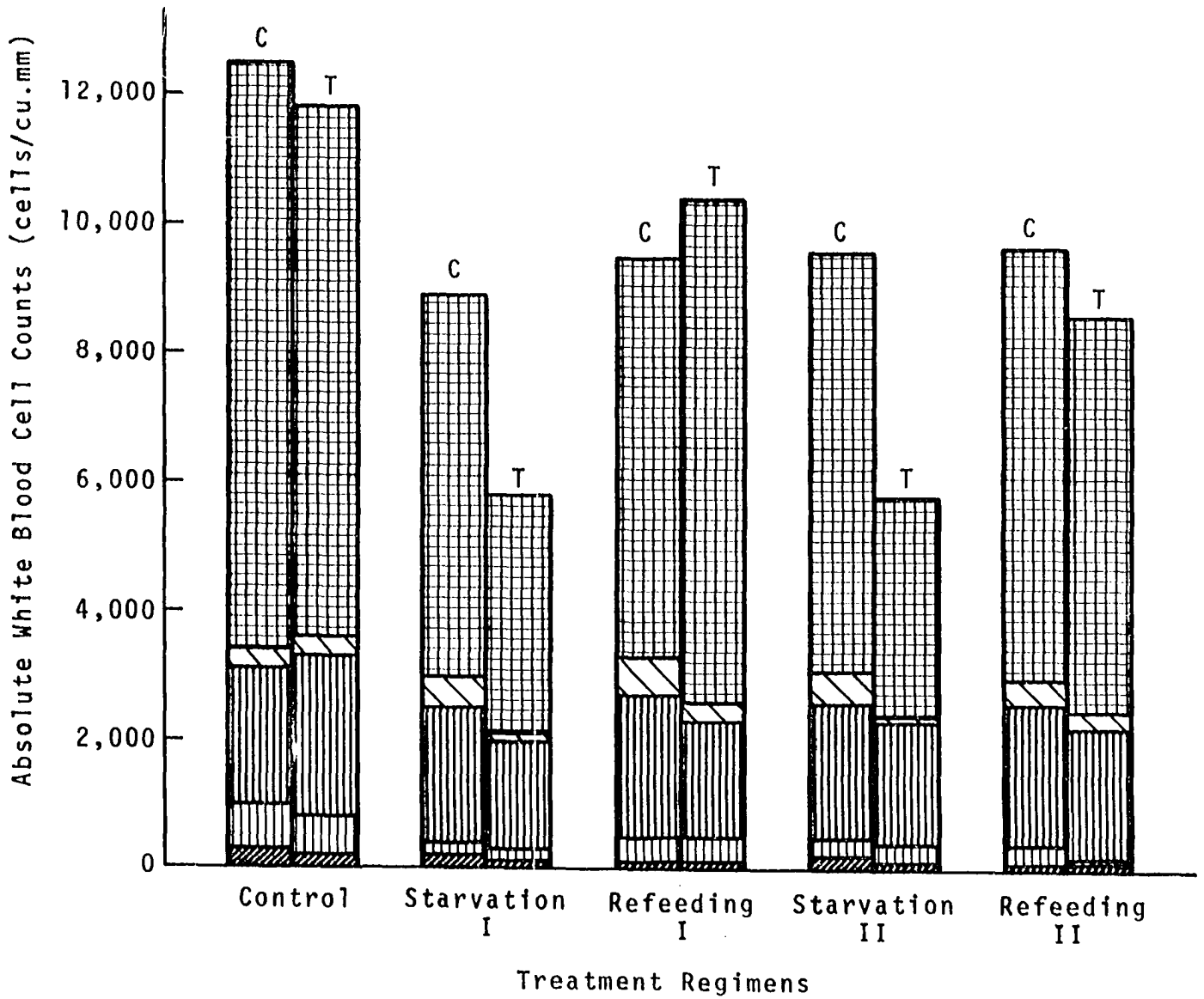
The control regimen mean for the WBC counts of the control and treatment groups were 12,502 ± 1,547 and 11,817 cells/cu mm, respectively. The means for the control group in the starvation I, refeeding I, starvation II, and refeeding II regimens were 8,928, 9,548, 9,604, and 9,749 cells/cu mm, respectively. The control regimen WBC count of the control group was significantly ($P<0.01$) greater than the means for all subsequent regimens in that group. The mean WBC counts for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 5,812, 10,446, 5,761, and 8,583 cells/cu mm, respectively. The treatment group's control regimen mean was significantly ($P<0.01$) greater than the starvation I, starvation II, and refeeding II regimen means. The control group means were significantly ($P<0.01$) greater than the treatment group means at the starvation I and starvation II regimens.

Absolute segmented neutrophil blood cell count

Table A-17 lists the individual values and group means for the segmented neutrophil counts at the various regimens. Table 4 gives the

Figure 10. Mean total white blood cell and absolute differential white blood cell counts in control dogs maintained on a commercial diet and in treatment dogs starved and refed a high carbohydrate diet; values represent mean counts at the end of each regimen




Absolute White Blood Cell Counts (cells/cu.mm)
12,000
10,000
8,000
6,000
4,000
2,000
0
C
T
Control
Starvation I
Refeeding I
Starvation II
Refeeding II
Treatment Regimens

normal range for the dog. The group mean values are also shown graphically in Figure 10. The analysis of variance is given in Table A-18. The mean squares, which were significant, corresponded to group ($P<0.01$), sex ($P<0.05$), regimen ($P<0.01$), and group-regimen interaction ($P<0.01$) effects.

The significant group effect indicates that the control group, as a whole, over the duration of the experiment had a higher segmented neutrophil count than the treatment group. The significant sex effect lends support to the observation that the males usually had a higher segmented neutrophil count than the females. The regimen effect again means that there was significant variation in the cell counts from one regimen to another. The significant group-regimen interaction was probably brought about by the decreased cell counts that occurred in the treatment groups during the two starvation regimens.

The control regimen means for the control and treatment groups were 9110 ± 1365 and 8311 cells/cu mm, respectively. The mean segmented neutrophil counts for the control group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 6075, 6246, 6493, and 6658 cells/cu mm, respectively. The mean for the control group at the control regimen was significantly ($P<0.01$) greater than all subsequent regimen means for that group. The means for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 3760, 7800, 3286, and 6045 cells/cu mm, respectively. The treatment group control regimen mean was significantly ($P<0.01$) greater than the starvation I, starvation II, and refeeding II regimen means for the same group. The control group means were significantly greater than the treatment group

means at starvation I ($P<0.01$) and starvation II ($P<0.01$) and significantly less than the treatment groups and means at the refeeding I regimen ($P<0.05$).

Absolute lymphocyte blood cell count

The individual dog values and group means for the various regimens are shown in Table A-19. Normal values for the dog are given in Table 4. A graphical representation of the mean values by group and regimen and their relationship to the total white blood cell counts can be found in Figure 10. The analysis of variance for the lymphocyte counts is listed in Table A-20. None of the mean squares in the analysis of variance demonstrated statistical significance.

The means for the control and treatment groups at the control regimen were 2113 ± 568 and 2461 cells/cu mm, respectively. The control group means for the starvation I, refeeding I, starvation II, and refeeding II regimens were 2138, 2217, 2121, and 2247 cells/cu mm, respectively. The means for the treatment group at starvation I, refeeding I, starvation II, and refeeding II regimens were 1741, 1823, 1931, and 2006 cells/cu mm, respectively. No statistical difference between regimens or groups was found using the Student's t-test.

Absolute band neutrophil blood cell count

Table A-21 lists the individual dog and group means for the band neutrophil counts at the various regimens. Figure 10 shows the mean values at each regimen and their relationship to the total white blood cell count. Table 4 gives the normal range for the dog. The analysis of variance for the band neutrophil cell count is given in Table A-22. None

of the mean squares in the analysis of variance showed statistical significance.

The control regimen mean for the control and treatment groups were 311 ± 160 and 183 cells/cu mm, respectively. The control group's mean band neutrophil cell counts at the starvation I, refeeding I, starvation II, and refeeding II regimens were 155, 111, 154, and 106 cells/cu mm, respectively. The means for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II were 70, 145, 61, and 121 cells/cu mm, respectively. There was no statistically significant difference between regimen or group means for the band neutrophil cell counts using the Student's t-test.

Absolute monocyte blood cell count

The individual dog values and group means for the monocyte counts at the various regimens are listed in Table A-23. The group means are also shown in Figure 10 with the other components of the total white blood cell counts. The normal range for the dog is given in Table 4. Table A-24 shows the analysis of variance for the monocyte counts. The significant mean squares were for the regimen ($P<0.01$) and sex-regimen ($P<0.05$) interaction effects.

The significant regimen effect in the analysis of variance indicates that the monocyte counts varied significantly from one regimen to another. The sex-regimen interaction was interpreted to mean that the sexes responded differently to the various regimens. It is not obvious from the data why this interaction appeared to be significant.

The control regimen means for the control and treatment groups were 678 ± 212 and 545 cells/cu mm, respectively. The control group means for

the starvation I, refeeding I, starvation II, and refeeding II regimens were 194, 391, 302, and 307 cells/cu mm, respectively. Using the Student's t-test the control group's control regimen mean was found to be significantly ($P<0.01$) greater than all the subsequent regimen means for that group. The means for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 186, 400, 294, and 140 cells/cu mm, respectively. The control regimen mean for the treatment group was significantly greater than the means for starvation I ($P<0.01$), starvation II ($P<0.05$), and refeeding II ($P<0.01$) regimens. No significant difference was found between the mean monocyte counts for the control and treatment groups using the Student's t-test.

Absolute eosinophil blood cell count

Table A-25 lists the individual dog values and group means for the eosinophil counts at the various regimens. Table 4 gives the normal range for the dog. Figure 10 shows the group means at each regimen and their relationship to the total white blood cell count. The analysis of variance is given in Table A-26. The significant ($P<0.01$) mean squares were for the group and group-regimen interaction effects.

The significant group effect in the analysis of variance indicates that the eosinophil counts for the control group were, as a whole, greater than the treatment group eosionphil counts. The group-regimen interaction was considered to mean that the two groups responded differently to the regimens. The group differences are exemplified by the fact that the control group means were significantly ($P<0.01$) greater than the treatment group means at the starvation I, refeeding I, and starvation II regimens using the Student's t-test.

The control regimen mean for the control and treatment groups were 289 ± 224 and 325 cell/cu mm, respectively. The mean eosinophil counts for the control group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 503, 582, 534, and 430 cells/cu mm, respectively. The control group control regimen mean was significantly less than the starvation I ($P<0.05$), refeeding I ($P<0.01$), and starvation II ($P<0.05$) regimen means. The means for the treatment group at the starvation I, refeeding I, starvation II, and refeeding II regimens were 71, 277, 107, and 270 cells/cu mm, respectively.

A decrease in the total white blood cell count with a normal differential count has been observed in human undernutrition (Keys et al., 1950; Drenick, 1971). Wilhelmj and McCarthy (1963) reported that the eosinophil count in the blood of starved canines decreased. In the present study it was found that the total white blood cell count was decreased by starvation. From differential cell counts it was determined that the decreased counts were primarily in those cells whose origin was the bone marrow, viz., neutrophils and eosinophils. The lymphocyte and monocyte counts remained relatively unchanged. The decreased cell counts were probably due to a lack of amino acids for new cell production, a shortage of necessary vitamins, and/or a hormone imbalance due to thyroid, gonad, and adrenal malfunction (Aschkenasy, 1957; Bray, 1974; Wintrobe, 1967; Pospisil et al., 1970; Keys et al., 1950; Alexander et al., 1964; Schatz et al., 1967; Schachner et al., 1965; Sabeh et al., 1969; Bouille and Assenmacher, 1970).

Heart Rate

The heart rate was determined at the same time that the blood pressure was measured in 15 of the beagle dogs. These measurements were made several times weekly and means for each day of measurement can be found in Table A-27 and Figure 11. The regimen means are shown in Table A-28 and Figure 11. The analysis of variance for the heart rate is shown in Table A-29.

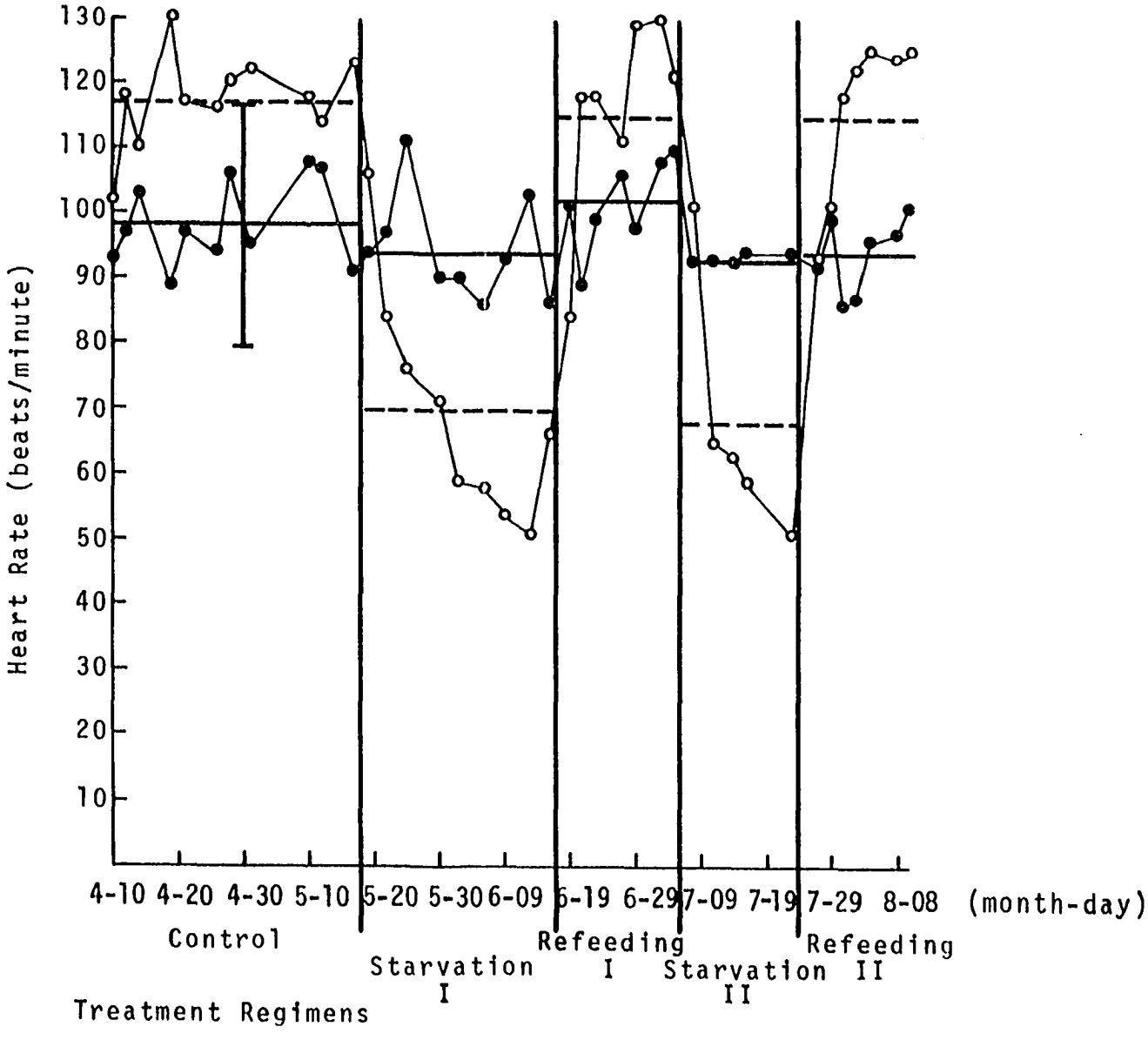
The females consistently had a higher heart rate than the males which produced a significant sex effect in the analysis of variance ($P<0.05$). There was also a significant ($P<0.01$) treatment and group-treatment interaction. These were probably due to the development of bradycardia in the treatment dogs during the starvation I and II regimens.

The mean heart rate for the control group varied from 93 ± 19 beats per minute (bpm) during the starvation II regimen to 102 bpm during the refeeding I regimen.

The initial mean for heart rate of the treatment group during the control regimen was 117 bpm. This was considerably higher than the mean for the control group during the same regimen (98 bpm). This difference was not, however, significant. Starvation I decreased the heart rate drastically to as low as a mean of 51 bpm on 6-13 (month-day). The regimen mean for starvation I was 70 bpm and significantly ($P<0.01$) different from the control regimen mean. Refeeding I brought the heart rate of the treatment dogs rapidly back to normal. The regimen mean was 115 bpm. The second starvation regimen decreased the heart rate, also, to a mean of 68 bpm. This value was significantly ($P<0.01$) different from the control

Figure 11. Mean heart rates in control dogs maintained on a commercial diet and in treatment dogs starved and refed a high carbohydrate diet; values represent day and regimen means; the pooled standard deviation is indicated

Control dog's day mean ●
Treatment dog's day mean ○
Control dog's regimen mean ——
Treatment dog's regimen mean - - - -


Heart Rate (beats/minute)
130
120
110
100
90
80
70
60
50
40
30
20
10
4-10 4-20 4-30 5-10 5-20 5-30 6-09 6-19 6-29 7-09 7-19 7-29 8-08 (month-day)
Control
Starvation I
Refeeding I
Starvation II
Refeeding II
Treatment Regimens

regimen value. The second refeeding again brought the rate back to control levels with a mean of 115 bpm.

The female and male treatment dogs responded qualitatively the same to starvation and refeeding. The females had a quantitatively larger decrease but the percentage decrease of the heart rate in both sexes was similar.

The normal range for heart rate in the untrained dogs is 70-160 beats per minute (bpm) (Ettinger and Suter, 1970). Wilhelmj and McCarthy (1963) reported that the mean heart rates on 13 trained dogs were in the range of 43-91 bpm. The beagles used in the present study were probably not as well trained as those used by Wilhelmj and thus a difference in heart rate exists. The decrease in heart rate with starvation has been reported in dogs (Wilhelmj and McCarthy, 1963), pigs (Smith et al., 1964), and man (Keys et al., 1950).

Along with the bradycardia several dogs (#5, 7, and 25) developed second degree heart block during starvation I and one dog (#9) showed the arrhythmia during starvation II. In both instances the arrhythmia was noticed on electrocardiograms taken towards the end of the starvation regimens. Since only one electrocardiogram was taken during each regimen it is impossible to say how often or when the arrhythmia first appeared. The same arrhythmia was seen in swine during starvation (Smith et al., 1964).

Why starvation brings about bradycardia and second degree heart block is not known. There is evidently a rise in vagal tone and/or a lowering of sympathetic tone in the body. It may be a compensatory mechanism that accompanies the decrease in metabolism of starvation.

Since the body is not using as much oxygen the heart does not need to pump as rapidly as normal.

Blood Pressure

The direct blood pressure was determined for 15 of the beagle dogs (eight control and seven treatment dogs) several times weekly. The means for each day of measurement can be found in Table A-27 and Figure 12. The regimen means are shown in the above mentioned figure and Table A-28.

Systolic blood pressure

The analysis of variance for the systolic blood pressure can be found in Table A-30. There were significant ($P<0.01$) effects due to regimens and the group-regimen interaction. This was interpreted to mean that the systolic blood pressure varied significantly from one regimen to another and that the two groups did not respond to each regimen in the same manner.

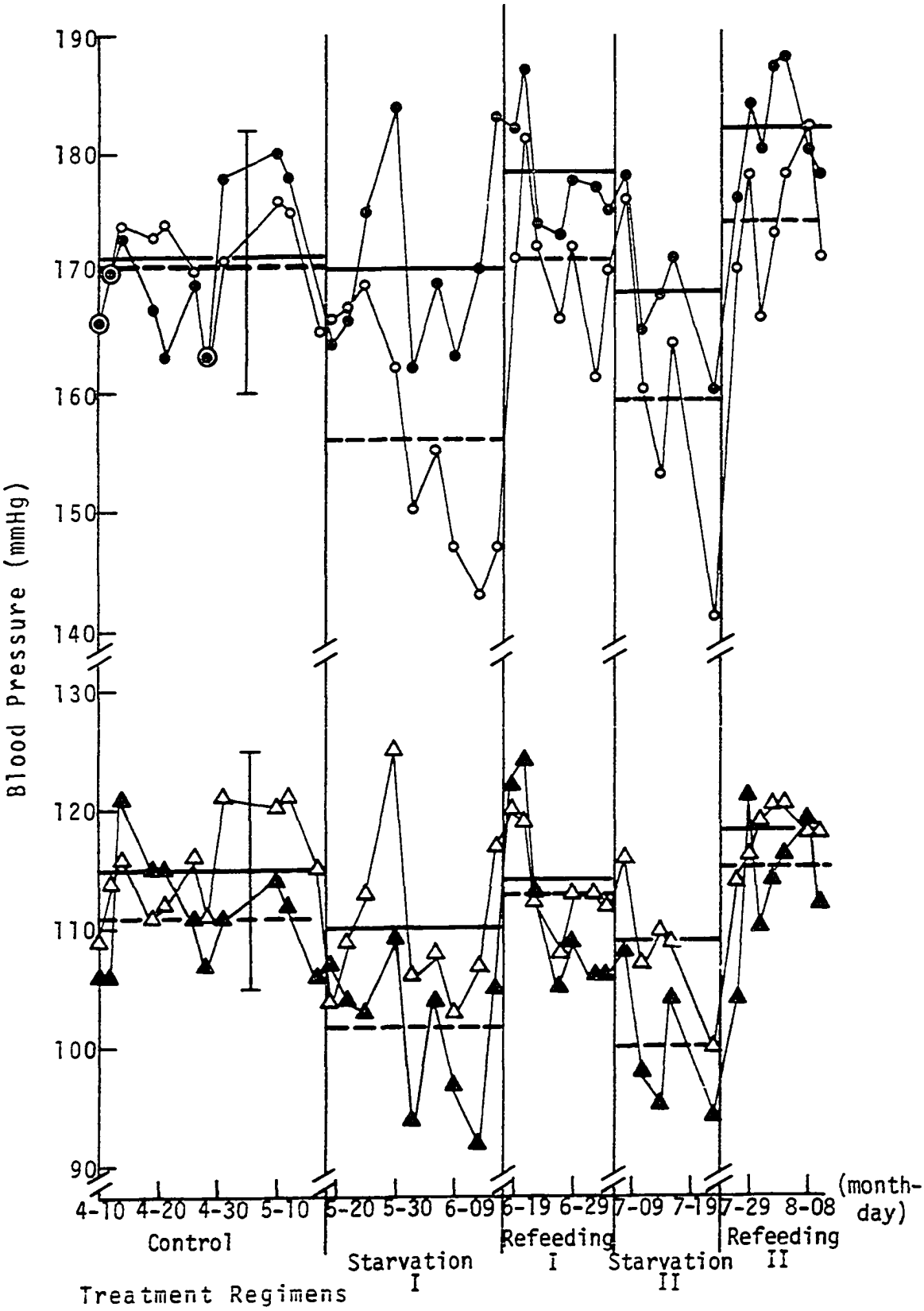
The dogs were assigned to two groups in a manner that both would have very similar control regimen pressures. The mean systolic pressure for both the control and treatment groups during the control regimen was 171 ± 12 mm Hg. The control group mean for subsequent regimens varied between 168 mm Hg during the starvation II and 182 mm Hg during the refeeding II regimen.

The treatment group showed a decrease in their mean systolic pressure from control regimen levels of 171 mm Hg to 156 mm Hg during starvation I. The lowest daily mean was 143 mm Hg on 6-13 (month-day). Refeeding I was immediately accompanied by a return of the systolic pressure to normal values. The mean for the treatment group during the refeeding I regimen

Figure 12. Mean systolic and diastolic blood pressures in control dogs maintained on a commercial diet and in treatment dogs which were starved and refed a high carbohydrate diet; values represent day and regimen means; the pooled standard deviation for systolic pressure is indicated

Control dogs:
- Day mean for systolic pressure ●
- Day mean for diastolic pressure △
- Regimen mean for systolic pressure ——
- Regimen mean for diastolic pressure ——

Treatment dogs:
- Day mean for systolic pressure ○
- Day mean for diastolic pressure ▲
- Regimen mean for systolic pressure ----
- Regimen mean for diastolic pressure ----


Blood Pressure (mmHg)
190
180
170
160
150
140
130
120
110
100
90
4-10 4-20 4-30 5-10 5-20 5-30 6-09 6-19 6-29 7-09 7-19 7-29 8-08 (month-day)
Control
Starvation I
Refeeding I
Starvation II
Refeeding II
Treatment Regimens

was 171 mm Hg. The second starvation regimen again produced a hypotensive trend with the mean for the regimen being 159 mm Hg. The lowest daily mean for the treatment group during the starvation II regimen was 141 mm Hg on 7-23 (month-day). The last refeeding regimen brought the blood pressure back to control levels with the regimen mean being 174 mm Hg.

Student's t-test on the regimen means did not reveal any statistical significance between regimens or groups.

Diastolic blood pressure

The analysis of variance for the diastolic blood pressure is shown in Table A-31. The regimen effect was the only one showing statistical significance ($P<0.01$). This was interpreted to mean that the diastolic pressure varied significantly from one regimen to another. The lack of a significant group-regimen interaction indicated that both groups of dogs responded similarly to each regimen.

The control group of beagles, which remained on a normal kennel diet, had a mean diastolic pressure that varied between 109 ± 10 mm Hg for starvation II to 118 mm Hg for refeeding II.

The treatment dogs began the experiment with a mean diastolic pressure of 111 mm Hg for the control regimen. Starvation I tended to decrease the pressure with the lowest value being 92 mm Hg on 6-13 (month-day). The mean for this regimen was 102 mm Hg which was not significantly different from the control regimen mean. It is interesting to note that every daily mean during the starvation I regimen was less than the overall control regimen mean. Refeeding I was immediately accompanied by 2 recording days with high mean diastolic pressure, 122 mm Hg on 6-19 and 124 mm Hg on 6-21. The subsequent daily means for this regimen were lower and

the mean for the entire regimen was 113 mm Hg. The second starvation regimen caused a trend of decreased diastolic pressure as starvation I did. The starvation II regimen mean for diastolic pressure was 100 mm Hg. The second refeeding regimen seemed to increase the diastolic pressure from the low levels of starvation II to a mean of 115 mm Hg for the last regimen.

The normal canine blood pressure in unanesthesized subjects is not well established. Wilhelmj and McCarthy (1963) considered the upper normal limit for trained dogs to be 140/70 mm Hg (systolic/diastolic) as determined by an auscultatory technique on the hind leg. Detweiler et al. (1968) referenced a study in which the normal direct blood pressure of 127 trained dogs was determined to be 155/80 mm Hg. It cannot be said for certain that the beagles used in this experiment were hypertensive but they seemed to have a resting blood pressure which exceeded published normal values. Even though the beagles in this study were trained they were not very relaxed around human beings. Thus, their elevated blood pressures may have been at least partially due to excitement. If, however, excitement was the sole cause of the elevated blood pressure, it should have declined to normal levels during starvation when the heart rate decreased. The bradycardia observed in this study was similar in magnitude to that reported by Wilhelmj and McCarthy (1963). The blood pressure of the beagles in this study did not, however, decrease to anywhere near the 85/45 mm Hg reported by Wilhelmj and McCarthy (1963) for dogs in starvation. It would seem that the beagles used in this experiment may have been hypertensive in the very beginning.

It had been hypothesized that starvation followed by refeeding of a high carbohydrate diet would cause either a systolic hypertension as observed previously in dogs (Wilhelmj and McCarthy, 1963) or diastolic hypertension as previously observed in swine (Smith et al., 1964). Neither of these effects were evident from the data on the beagles in the present study.

Vectorcardiography

Examples of vectorcardiograms are shown in Figures 13 through 18. These were recorded from dog #8 of the treatment group and depict the vectorcardiogram (VCG) in the frontal, transverse, and left sagittal planes for the control (Figures 13, 15, and 17) and starvation I (Figures 14, 16, and 18) regimens. The P loop which was not measured is on the left side of the figures. The QRS loop is seen in the middle of the figures. A small arrowhead on the QRS loop indicates the direction of its inscription. Faint lines drawn on the QRS loop from the isoelectric point to the periphery are the maximum vector (frontal, transverse, and left sagittal planes) and half-area vector (transverse and left sagittal planes). The T loop is shown towards the right side of the figure. The maximum T loop vector appears as an overlaid line drawn from the isoelectric point through the point on the T loop which was a maximum distance away.

Half-area QRS vector

The regimen means for the half-area QRS vectors of the control and treatment groups in the transverse and left sagittal planes are shown in Figure 19.

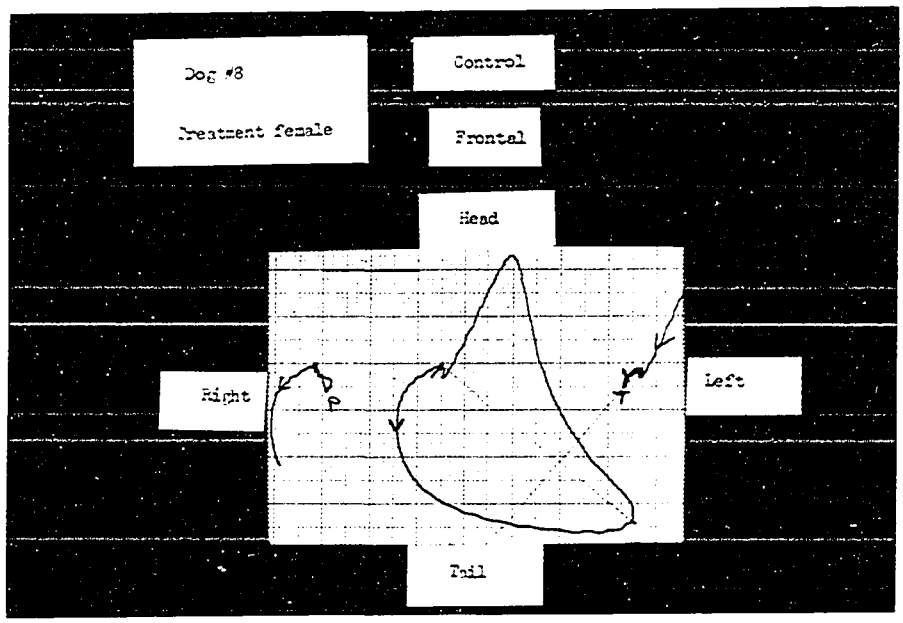


Figure 13. Control regimen vectorcardiogram in the frontal plane for dog 08; calibration is 1 mv/2cm (line)

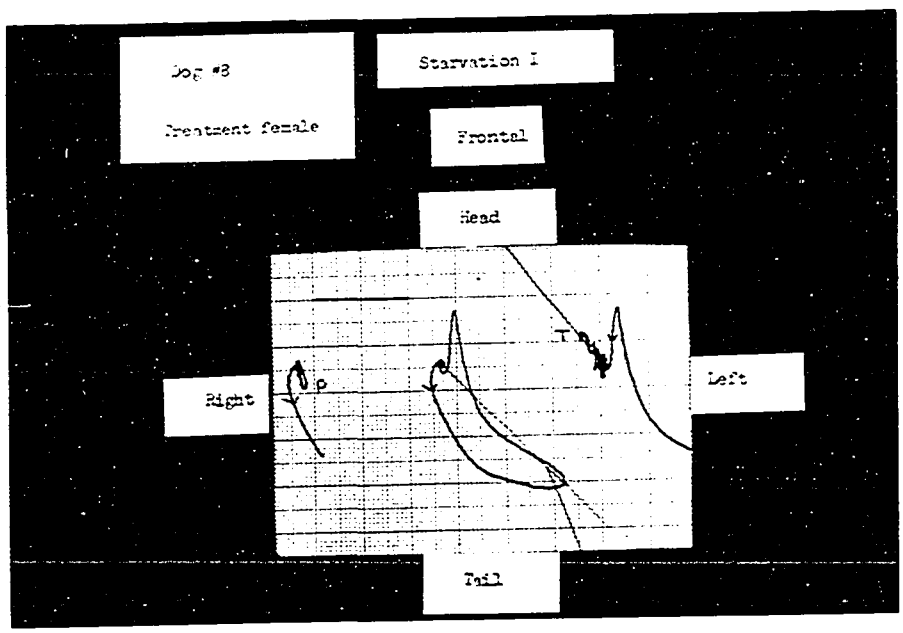


Figure 14. Starvation I regimen vectorcardiogram in the frontal plane for dog 08; calibration is 1 mv/2 cm (line)

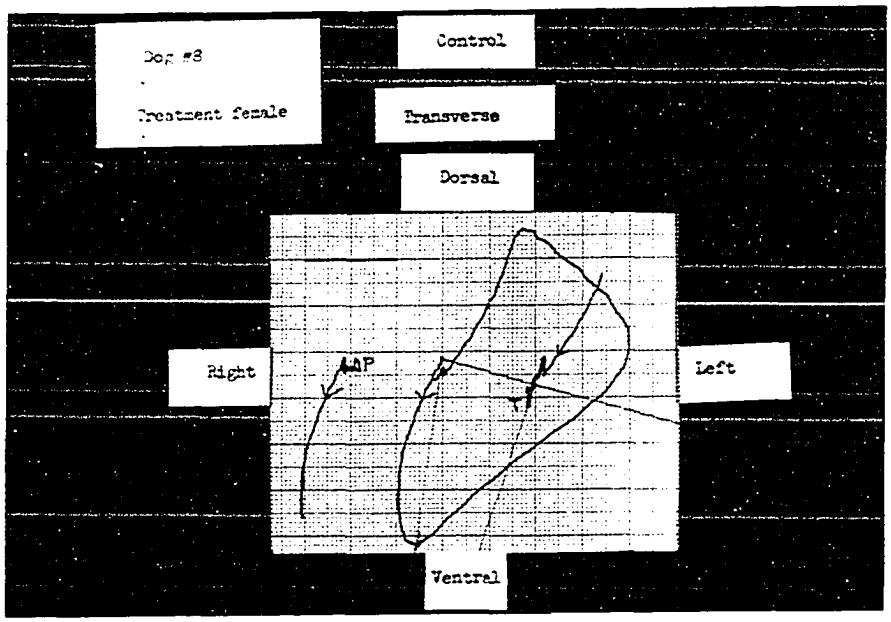


Figure 15. Control regimen vectorcardiogram in the transverse plane for dog 08; calibration is 1 mv/2 cm (line)

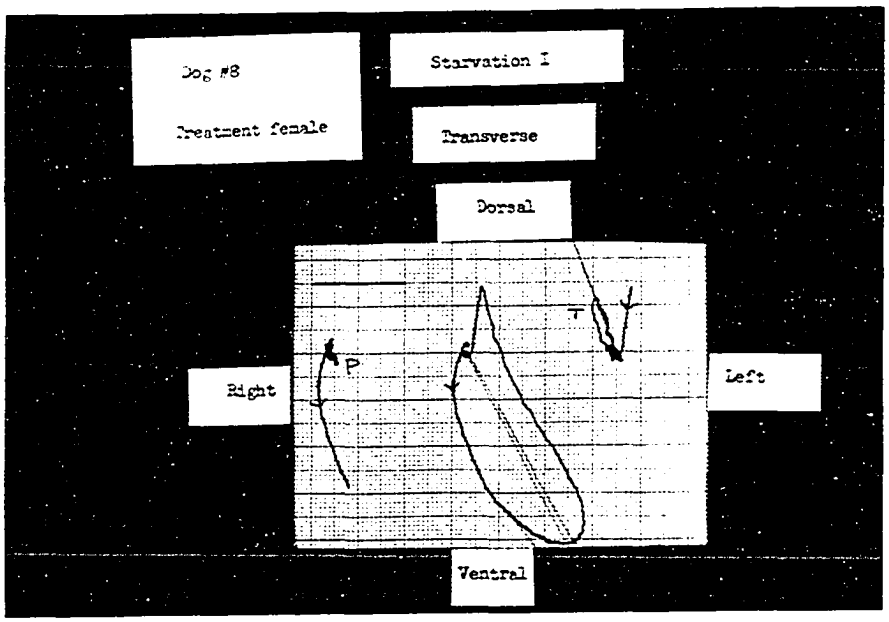


Figure 16. Starvation I regimen vectorcardiogram in the transverse plane for dog 08; calibration is 1 mv/2 cm (line)

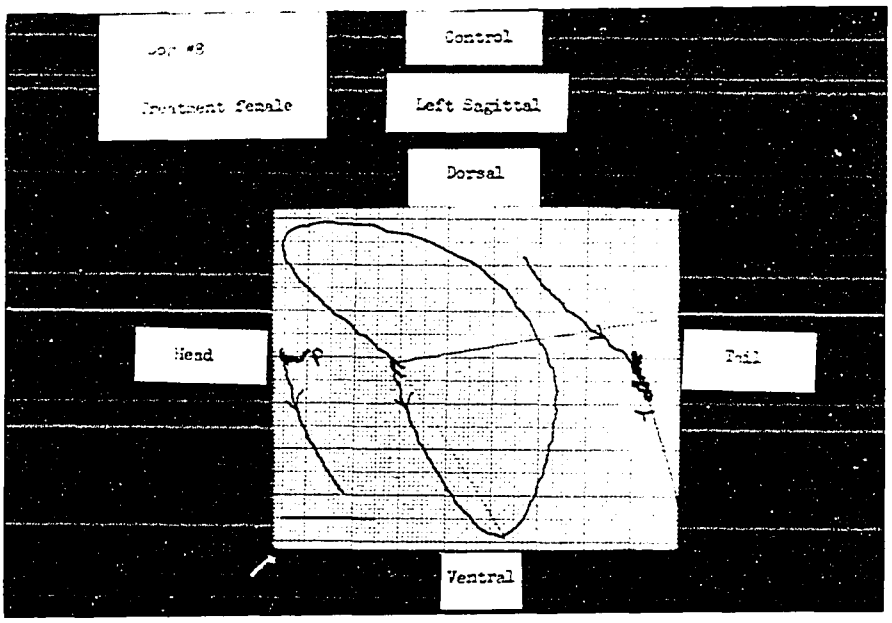


Figure 17. Control regimen vectorcardiogram in the left sagittal plane for dog 08; calibration is 1 mv/2 cm (line)

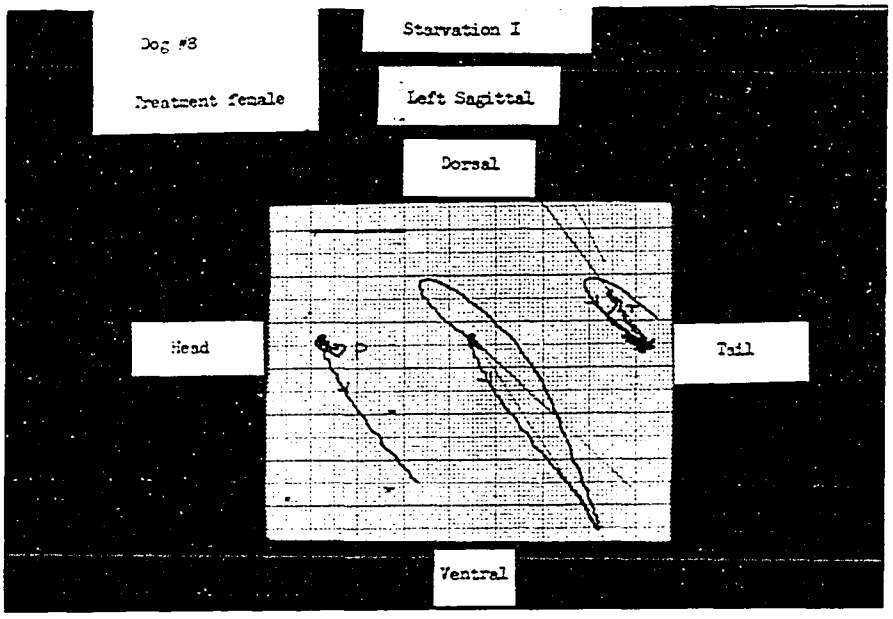
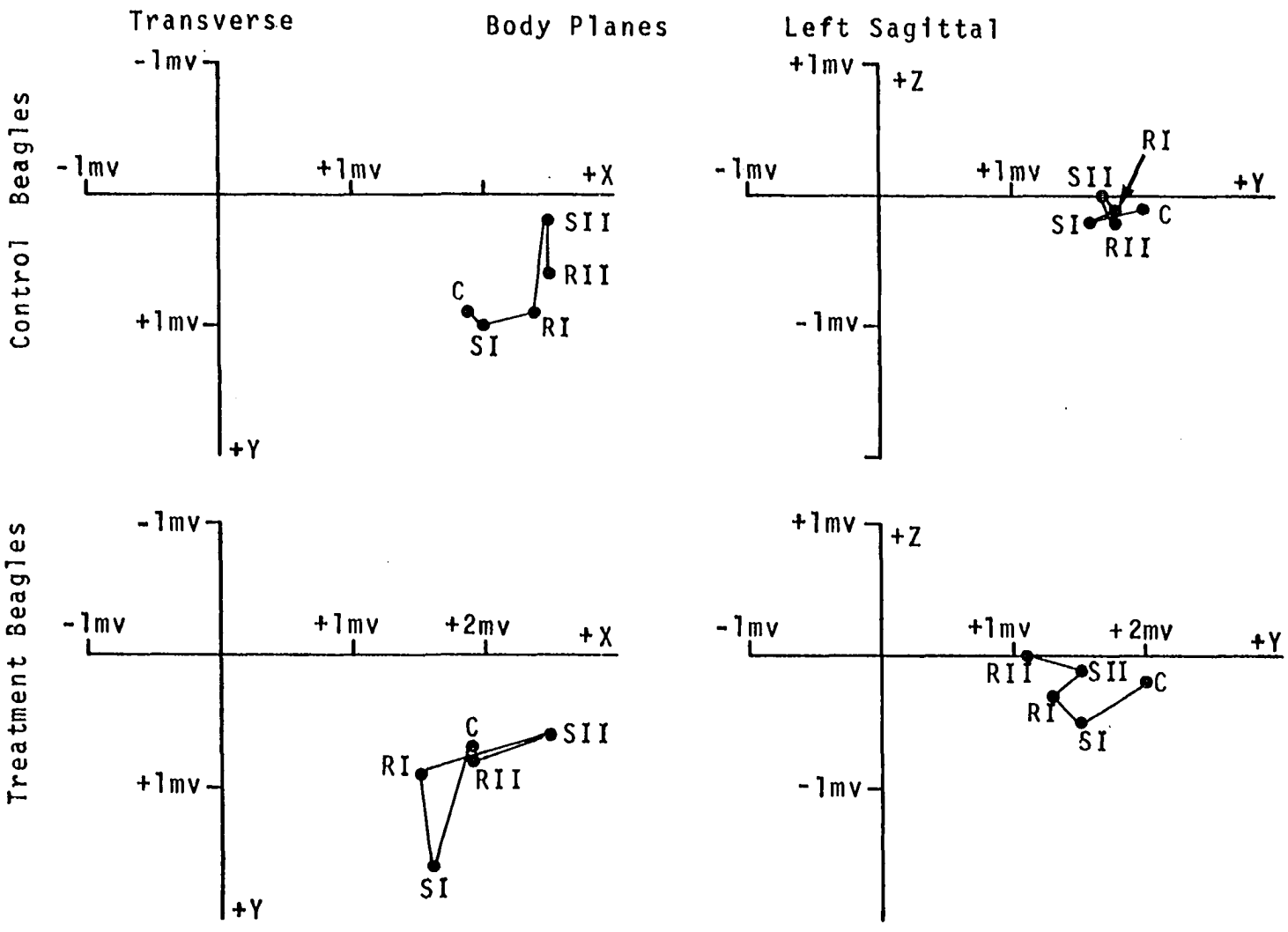


Figure 18. Starvation I regimen vectorcardiogram in the left sagittal plane for dog 08; calibration is 1 mv/2 cm (line)

Figure 19. Mean half-area vectors for the QRS loop in the transverse and left sagittal planes using the McFee-Parungao lead system in control dogs maintained on a commercial diet and in treatment dogs which were starved and refed a high carbohydrate diet; values represent means at the end of each regimen

C - control regimen
SI - starvation I regimen
RI - refeeding I regimen
SII - starvation II regimen
RII - refeeding II regimen


Transverse
Body Planes
Left Sagittal
Control Beagles
Treatment Beagles
-1mv
+1mv
+2mv
+X
+Y
+Z
SII
RII
RI
SI
C

Transverse plane Table A-32 lists the individual and group mean values for the X and Z lead coordinates. Tables A-33 and A-34 show the analyses of variance for the X and Z lead coordinates, respectively. The only mean squares with statistical significance in the analyses of variance were for regimens (X lead - $P<0.01$, Z lead - $P<0.05$). These significant effects were interpreted to mean that the X and Z lead coordinates varied significantly from one regimen to another.

Left sagittal plane The individual and group mean values for the Y and Z lead coordinates of the half-area vector in the left sagittal plane can be found in Table A-35. The analyses of variance for the Y and Z lead coordinates are shown in Table A-36 and A-37. Significant ($P<0.01$) mean squares were found for regimens and group-regimen interaction in the Y lead. None of the mean squares in the Z lead analysis of variance were significant.

The significant regimen effect in the Y lead analysis of variance was thought to be due to variation in this coordinate from one regimen to another. The significant group-regimen interaction was interpreted to mean that the two groups did not change in an identical manner from one regimen to another. This group difference can be visualized in Figure 19. The half-area vectors in the left sagittal plane for the control group are closely grouped with very small variation in either the Y or Z lead coordinates. The treatment group, however, has a much wider variation in both coordinates from regimen to regimen. This difference in variation may have led to the group-regimen interaction.

Maximum QRS vector

The maximum QRS vectors for the control and treatment groups of beagle dogs in the frontal, transverse, and left sagittal planes are shown in Figure 20 for each regimen.

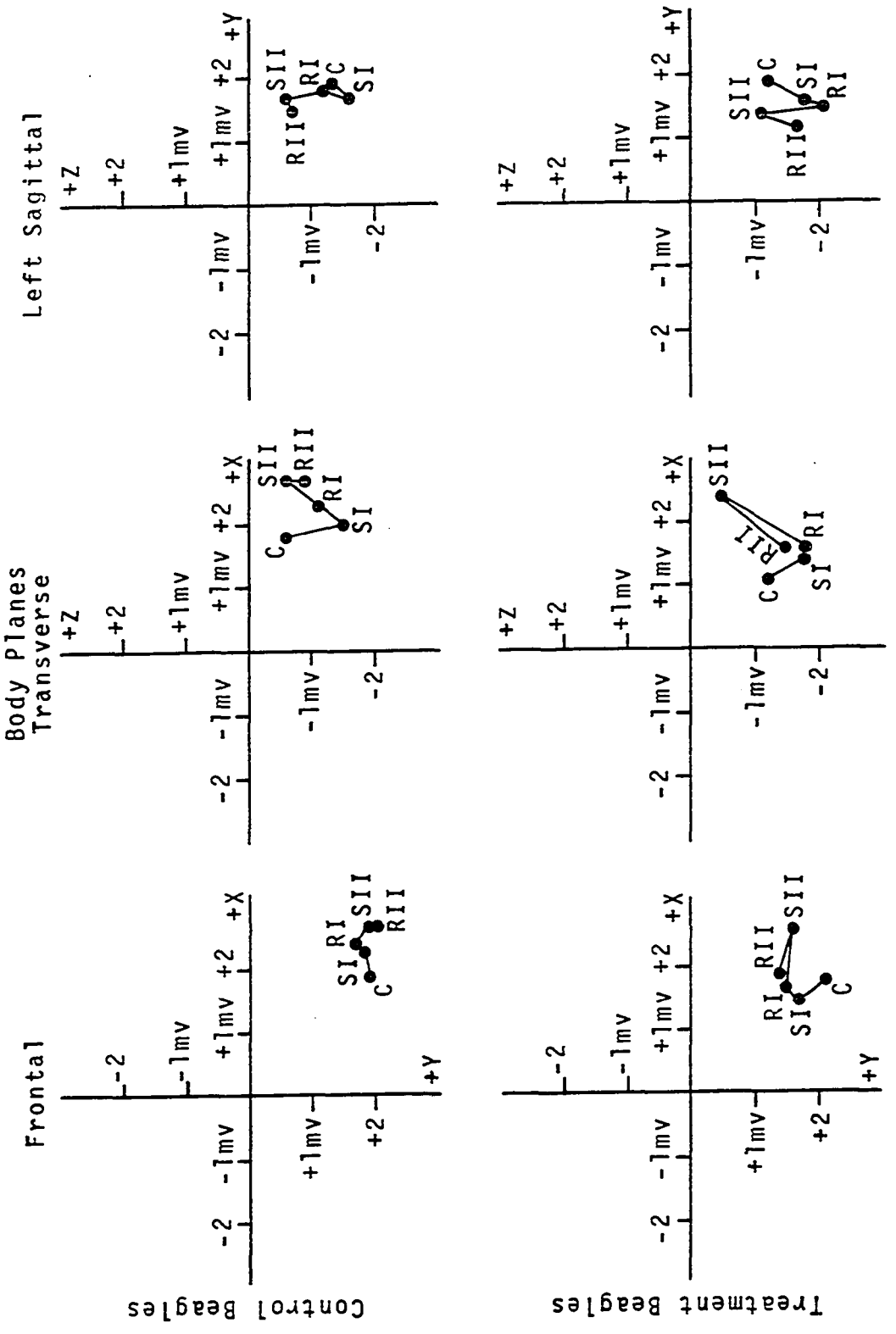
Frontal plane The X and Y lead coordinates of the maximum QRS vectors in the frontal plane for the beagle dogs at each regimen are listed in Table A-38. The analysis of variance for the X lead and Y lead coordinates can be found in Tables A-39 and A-40, respectively. The analysis of variance for the X lead coordinate revealed a significant ($P<0.01$) regimen effect. This was interpreted to mean that this coordinate varied significantly from one regimen to another. No other sources of variation were significant. The analysis of variance for the Y lead coordinate did not reveal any significant sources of variation. A significant ($P<0.01$) difference was found to exist between the X lead coordinates for the mean vector of the treatment group for regimens starvation I and II.

Transverse plane The individual dog values and group means for the X and Z lead coordinates are listed in Table A-41. The analysis of variance for the X and Z lead coordinates are shown in Tables A-42 and A-43. Both leads had significant ($P<0.01$) effects in their analysis of variance due to regimens. This significant effect meant that both coordinates varied significantly from one regimen to another. Student's t-tests on the individual group means for each regimen revealed that the X lead coordinates for the treatment group at the end of the control and starvation II regimens were significantly different ($P<0.01$).

Left sagittal plane Values for the Y and Z lead coordinates of individual dogs and their group means are listed in Table A-44. Tables

Figure 20. Means for the maximum vector of the QRS loop in the frontal, transverse, and left sagittal planes using the McFee-Parungao lead system in control dogs maintained on a commercial diet and in treatment dogs which were starved and refed a high carbohydrate diet; values represent means at the end of each regimen

C - control regimen
SI - starvation I regimen
RI - refeeding I regimen
SII - starvation II regimen
RII - refeeding II regimen


Left Sagittal
Body Planes
Transverse
Frontal
Control Beagles
Treatment Beagles
+Z
+2
+1mv
-1mv
-2
+X
+Y
SII
RII
RI
SI
C

A-45 and A-46 show the analysis of variance for the Y and Z lead coordinates, respectively. A significant ($P<0.05$) sex-regimen interaction was found in the Y lead coordinate analysis of variance. What this interaction may mean is unclear but the mean Y lead coordinate for the females was consistently less than the mean for the males at each regimen. The magnitude of the difference between the regimen means for the males and females varies and this may be the source of the interaction. Student's t-tests on individual regimen means did not reveal any significant differences. The analysis of variance for the Z lead coordinates did not show any significant sources of variation.

Discussion

Maximum and half-area QRS vectors for a group of normal mongrel dogs are shown in Table 5. They are similar in value to those measured for the beagles in the present study. There was no apparent effect of starvation-refeeding on the QRS loops in the three body planes.

Table 5. Coordinates for the mean QRS vector from normal dogs[a]

Vector for QRS loop	Body planes (mv)					
	Frontal		Transverse		Left sagittal	
	X-lead	Y-lead	X-lead	Z-lead	Y-lead	Z-lead
Maximum	2.12	1.91	1.72	-2.46	2.19	-2.19
Half-area	-[b]	-[b]	2.22	-1.62	2.09	-1.07

[a]Chastain et al., 1974.

[b]Half-area vectors in the frontal plane were not determined.

Direction of QRS loop inscription

The direction of inscription of the QRS loop in the frontal, transverse, and left sagittal planes can be found in Tables A-47, A-48, and A-49, respectively.

In the frontal plane the direction of inscription varied and was found to be clockwise, counterclockwise, or a figure-8. The figure-8 configuration was the most common followed by counterclockwise and then clockwise.

The transverse plane QRS loops were usually inscribed in a counterclockwise direction with a few figure-8 patterns being seen.

The direction of inscription in the left sagittal plane was exclusively counterclockwise.

There was no apparent relationship between the treatment regimens and the direction of QRS loop inscription.

Chastain et al. (1974) and Ettinger and Suter (1970) found that the direction of the normal canine QRS loop from the frontal plane may be counterclockwise, clockwise, or figure-8. The predominant directions in the transverse and left sagittal planes were counterclockwise but a few figure-8 patterns were seen (Chastain et al., 1974).

Maximum T wave vector

Frontal plane The X and Y coordinates of the maximum T wave vector in the frontal plane are listed for each dog in Table A-50. The analysis of variance for the X coordinate is shown in Table A-51 and for the Y coordinate in Table A-52. The X coordinate had a statistically significant ($P<0.05$) regimen effect meaning that the coordinate varied significantly from one regimen to another. The Y coordinate had a

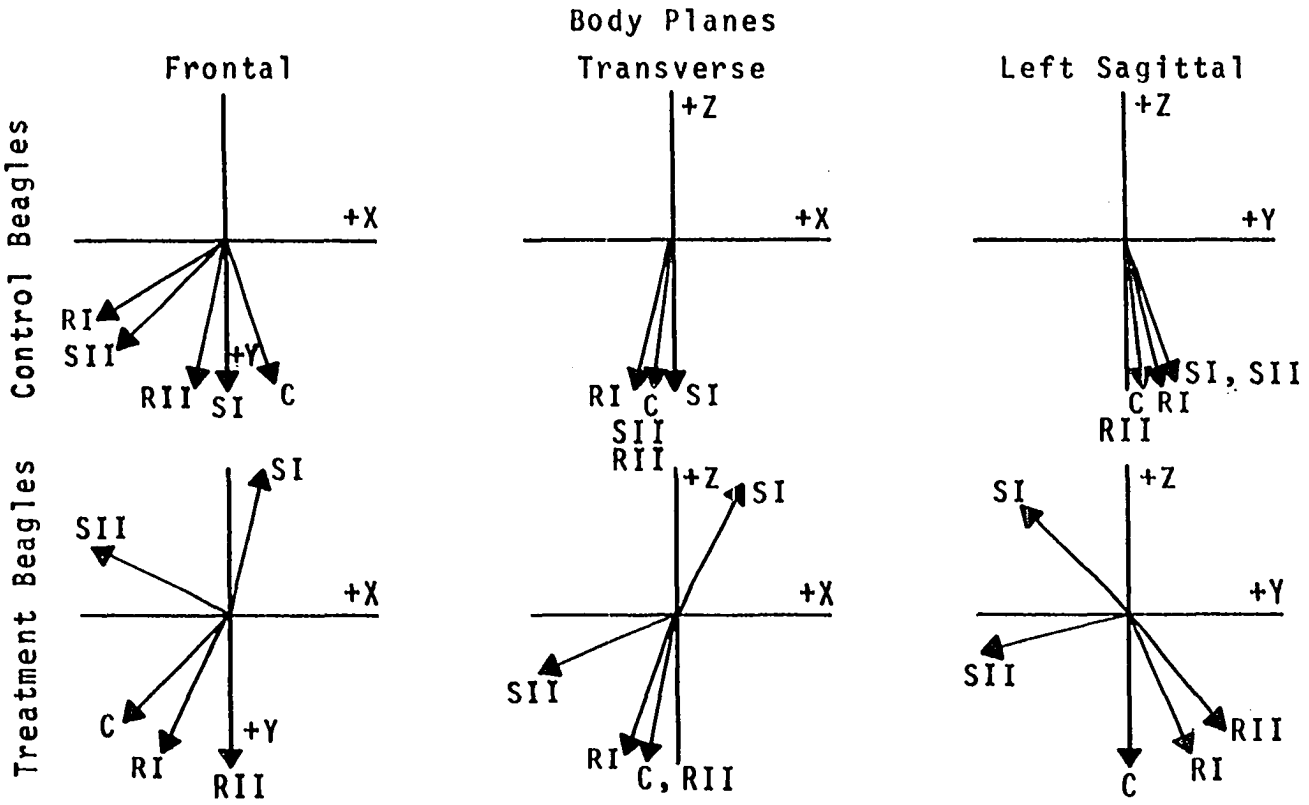
significant ($P<0.05$) regimen effect and a significant ($P<0.01$) group-regimen interaction. This was interpreted to mean that the Y coordinate varied from one regimen to another but the two groups did not change in an identical manner. The difference in Y coordinates for the two groups in the various regimens can be seen by examining Figure 21. The mean Y coordinate for all the regimens in the control beagles was positive in polarity as was the Y coordinate in the treatment beagles for the control, refeeding I, and refeeding II regimens. But, the mean Y coordinate for the treatment beagles during the starvation I and starvation II regimens was negative in polarity.

The maximum T-wave vector was normally directed either to the right or to the left and caudally. During starvation, however, the maximum T wave vector shifted to the cranial direction.

Transverse plane The individual dog values for the X and Z coordinates of the maximum T vector in the transverse plane are shown in Table A-53. The analysis of variance for the X coordinate can be found in Table A-54. There was a significant ($P<0.05$) regimen effect meaning that the X coordinate changed from one regimen to another. The Y coordinate analysis of variance is listed in Table A-55. Significant ($P<0.01$) effects were found for group, regimen, and the group-regimen interaction. The mean values for each regimen are shown in Figure 21. It can be seen that the mean T wave of the treatment beagles in the transverse plane was shifted in the positive Z direction by starvation. The control beagles during the same regimens had T waves which were directed in the negative Z direction.

Figure 21. Mean orientation of the maximum T wave vector in the frontal, transverse, and left sagittal planes using the McFee-Parungao lead system in control dogs maintained on a commercial diet and in treatment dogs which were starved and refed a high carbohydrate diet; values represent means at the end of each regimen

C - control regimen
SI - starvation I regimen
RI - refeeding I regimen
SII - starvation II regimen
RII - refeeding II regimen


Body Planes
Frontal
Transverse
Left Sagittal
Control Beagles
Treatment Beagles
+X
+Y
+Z
RI
SII
RII
SI
C
SI, SII
C RI
C, RII

The mean maximum T wave in the transverse plane was normally directed rightward and ventrad. Starvation shifted the T wave vector dorsally.

Left sagittal plane The Y and Z coordinates of the maximum T wave vector in the left sagittal plane for the individual beagle dogs during the various regimens can be found in Table A-56. The analysis of variance for the Y coordinate is shown in Table A-57. There was a significant regimen ($P<0.05$) and group-regimen interaction ($P<0.01$). The analysis of variance for the Z coordinate is listed in Table A-58 with statistical ($P<0.01$) significance for group, regimen, and the group-regimen interaction. From Figure 21 it can be seen that the normal mean maximum T wave in the left sagittal plane was directed ventrad and caudad. After starvation the T wave was directed more craniad and dorsad than normal.

Discussion Ettinger and Suter (1970) found that the T loop varied considerably among individual dogs. The loop was usually directed ventrad and to the right of the midline but in the frontal plane it could be either cranial or caudal. Although Chastain et al. (1974) did not measure the T loop they described its orientation in a similar manner.

Even though the mean Y coordinate of the maximum T loop in the frontal plane was negative following the starvation regimens it is doubtful that such an orientation can be considered abnormal because six of the ten control beagles had a negative Y coordinate for at least one regimen (Table A-50).

Likewise, the X lead coordinate was quite variable from dog to dog and regimen to regimen. On the other hand, the Z lead coordinate was never found to be positive in the control beagles or the treatment beagles after the control, refeeding I, or refeeding II regimens. It can be

assumed from the normal values in this experiment plus those of Chastain et al. (1974) and Ettinger and Suter (1970) that positive Z coordinates for the T loop as seen following the starvation I and II regimens in the treatment beagles are abnormal.

T wave changes in dogs have been reported due to hyperpotassemia (Ettinger and Suter, 1970; Coulter and Engen, 1972), asphyxia (Coulter and Engen, 1972), right ventricular hypertrophy (Detweiler and Patterson, 1965), and myocardial infarction (Fregin et al., 1972). Changes in the T wave similar to those in this study were found in right ventricular hypertrophy and myocardial infarction involving the descending branch of the left coronary artery. Although McFee-Parungao vectorcardiograms were not recorded in these instances of T wave change, a positive T wave polarity in lead V_{10} which is similar to the Z lead was observed.

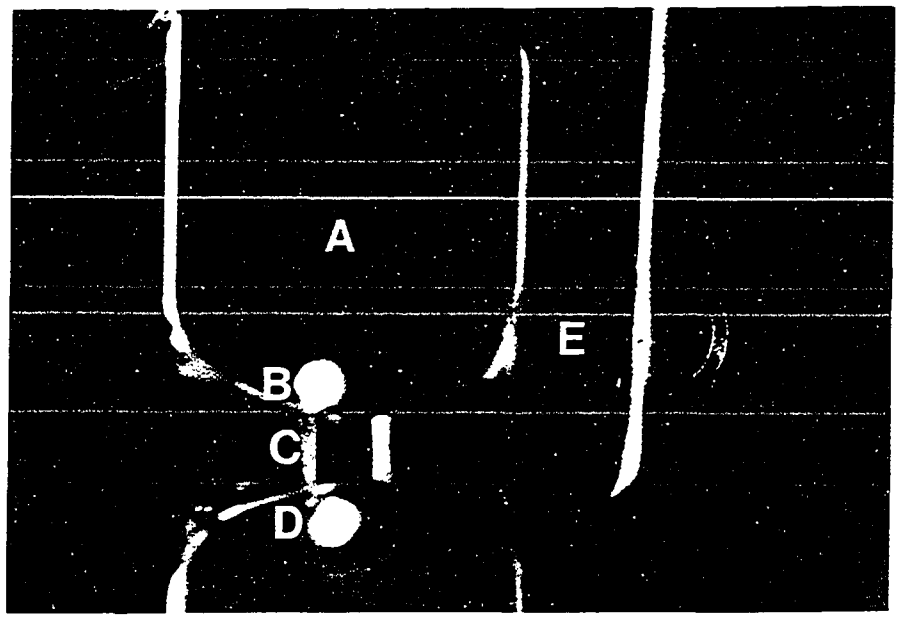
T wave changes with starvation have been seen in man (Keys et al., 1950; Friedberg, 1966) and swine (Smith et al., 1964) but have not been reported in the dog. The cause of such changes is unknown but must relate to altered ventricular repolarization.

Femoral Artery Stretch-Tension Characteristics

All of the blood vessels were stretched to 100 gms tension in the testing apparatus (Figures 3 and 4). The resulting paper chart recording (Figure 5) had a time base for the abscissa and grams tension for the ordinate. The appearance of an artery being stretched in the chamber is shown in Figures 22, 23, 24, 25, and 26. The artery had already taken on a racetrack configuration at 6.25 gms tension. Since the artery's circumference was calculated with the assumption that it had a racetrack

Figure 22. The right femoral artery from dog 01 under 6.25 gm tension; the Plexiglas arm (A) connecting the force-displacement transducer to the upper rod, the movable upper (B) and stationary lower (D) rods inside the artery (C) lumen, and a stainless steel cylinder (E) used as a size reference are shown

Figure 23. The right femoral artery from dog 01 under 25 gm tension; the Plexiglas arm (A) connecting the force-displacement transducer to the upper rod, the movable upper (B) and stationary lower (D) rods inside the artery (C) lumen, and a stainless steel cylinder (E) used as a size reference are shown


A
E
B
C
D

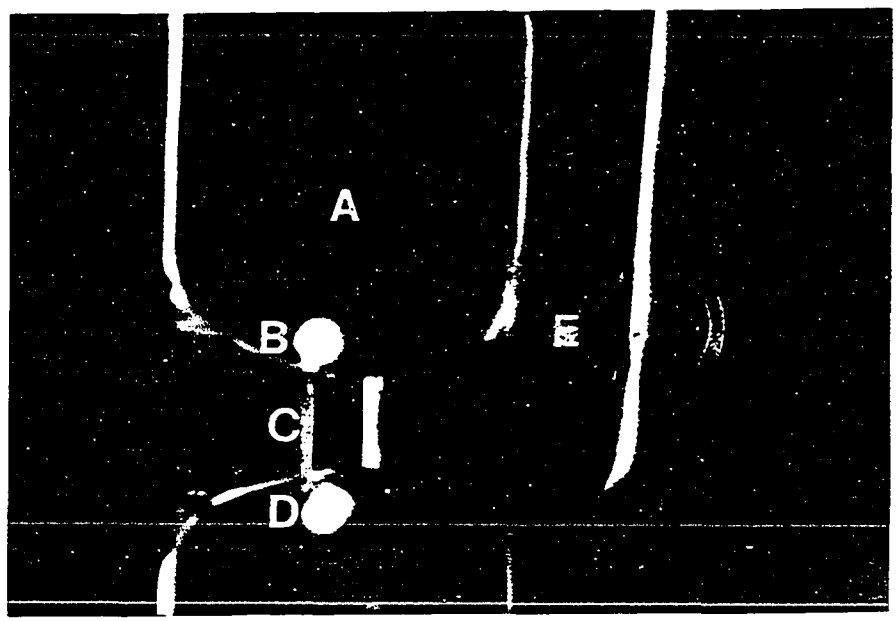
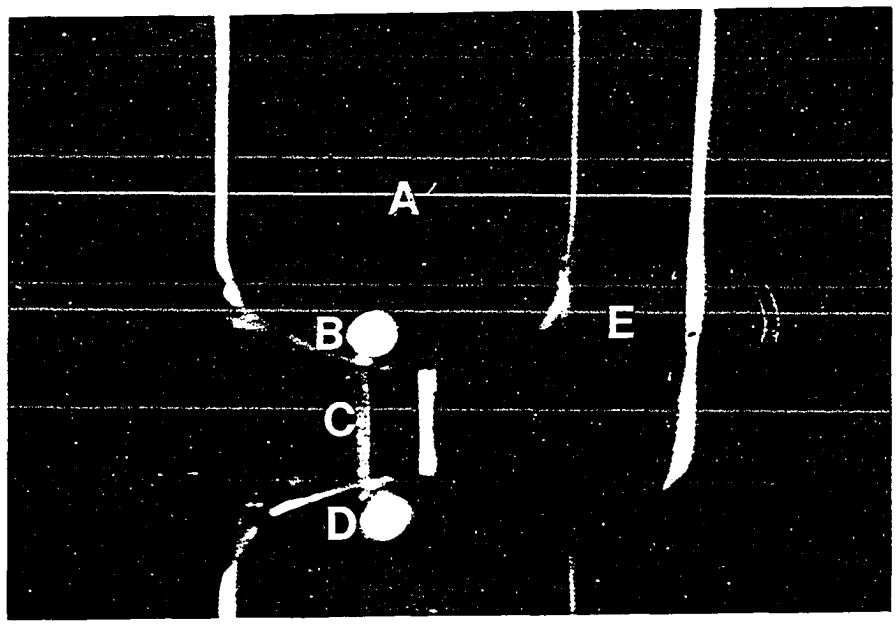
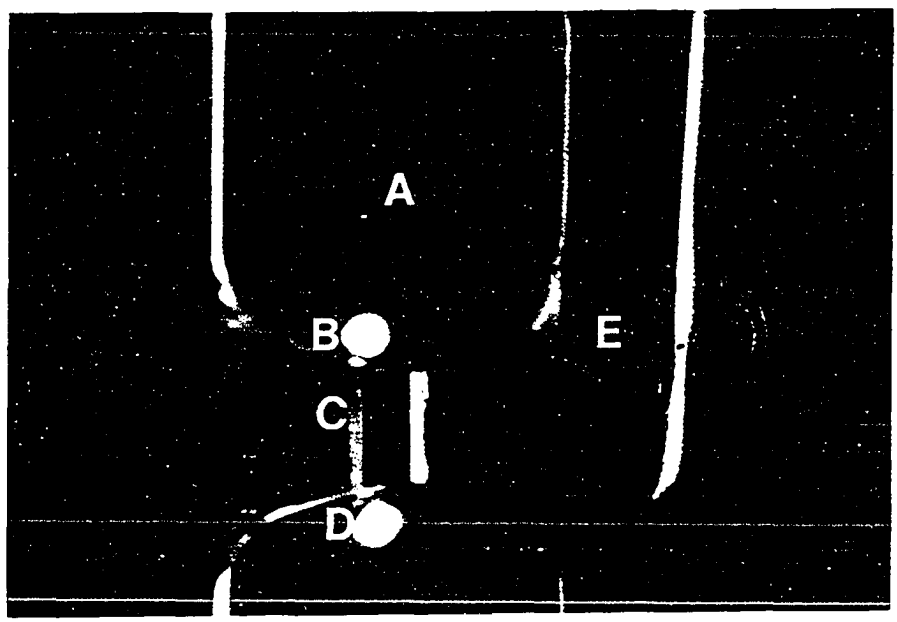

A
B
E
C
D

Figure 24. The right femoral artery from dog 01 under 50 gm tension; the Plexiglas arm (A) connecting the force-displacement transducer to the upper rod, the movable upper (B) and stationary lower (D) rods inside the artery (C) lumen, and a stainless steel cylinder (E) used as a size reference are shown

Figure 25. The right femoral artery from dog 01 under 75 gm tension; the Plexiglas arm (A) connecting the force-displacement transducer to the upper rod, the movable upper (B) and stationary lower (D) rods inside the artery (C) lumen, and a stainless steel cylinder (E) used as a size reference are shown


A
B
C
D
E


A
B
C
D
E

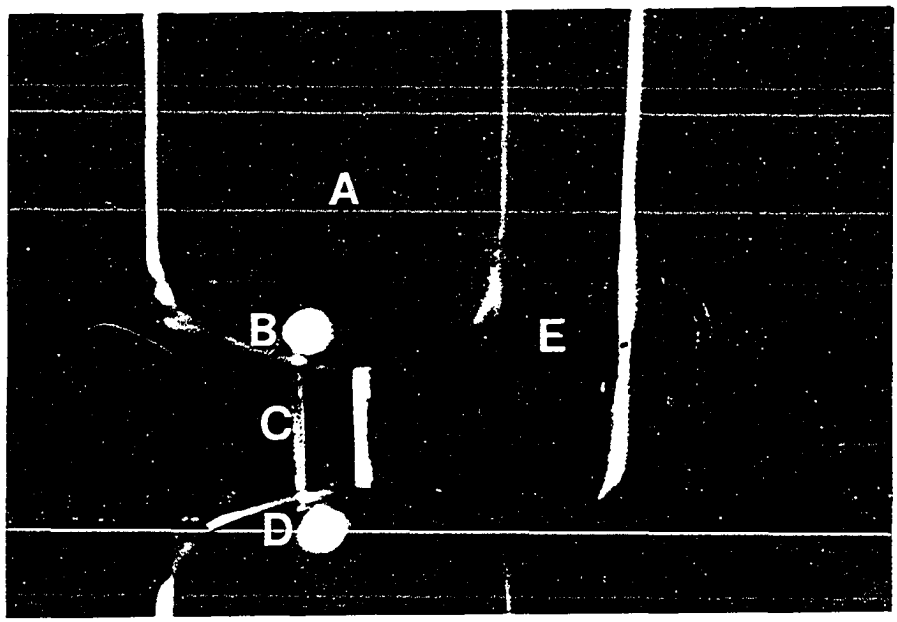


Figure 26. The right femoral artery from dog 01 under 100 gm tension; the Plexiglas arm (A) connecting the force-displacement transducer to the upper rod, the movable upper (B) and stationary lower (D) rods inside the artery (C) lumen, and a stainless steel cylinder (E) used as a size reference are shown

geometry, any calculations prior to the time that the artery achieved the appropriate configuration would be inaccurate. Consequently the initial values for circumference are undoubtedly wrong and there was no way of telling exactly when the vessel achieved the desired geometry. Photographs were taken of all the arteries being stretched in the same manner as those shown in Figures 22, 23, 24, 25, and 26. It was concluded that most vessels had parallel walls at 6.25 gms and all had parallel walls at 25 gms of tension.

Arterial wall measurements

Individual dog values and group means for femoral artery circumference, length, wall-thickness, and radius/wall-thickness ratio can be found in Table 6. The same measurements on femoral arteries from mongrel dogs are listed in Table 7.

The control females were significantly ($P<0.01$) lighter than the males in body weight when both the initial and the final segments were harvested. The treatment females were significantly ($P<0.05$) lighter than the males at the time of the initial segment removal but no difference was found at the time of removal of the final segment. The overall control and treatment group body weight means were not significantly different at the time of the initial segment removal. The control mean was significantly ($P<0.05$) greater than the treatment mean body weight, however, at the end of the experiment.

Using paired t-tests the control females showed no significant change in their body weight while the males increased in weight significantly ($P<0.05$) from the initial to the final segment harvesting dates. The treatment females did not significantly change in body weight while the

Table 6. Femoral artery segment dimensions of dogs

Group	Initial segment: Body weight (kg)	Initial segment: Circumference (cm)	Initial segment: Length (cm)	Initial segment: Wall-thickness (cm)	Initial segment: R/WT[a]	Final segment: Body weight (kg)	Final segment: Circumference (cm)	Final segment: Length (cm)	Final segment: Wall-thickness (cm)	Final segment: R/WT
Control females										
01	9.8	.7540	.65	.01890	6.3	10.5	.8130	.71	.01975	6.5
02	10.5	.8327	.69	.01451	9.1	10.2	.8564	.63	.01659	8.2
04	9.8	.8109	.51	.01690	7.6	10.9	.7851	.50	.01628	7.7
06	10.0	.7594	.69	.01651	7.3	10.5	.7149	.52	.02199	5.2
10	10.9	.9376	.46	.02220	6.7	11.8	.8812	.63	.02238	6.3
Mean	10.2±[b**]	.8189±	.60±	.01780±	7.4±	10.8±[b**]	.8101±	.60±	.01940±	6.8±
	.5[c]	.0743	.11	.00291	1.1	.6	.0650	.09	.00289	1.2
Males										
22	13.6	.8614	.61	.01883	7.3	13.6	.8267	.63	.01813	7.3
23	13.6	.8386	.58	.01782	7.5	14.1	.8574	.58	.01060	6.6
24	13.6	.9030	.55	.02230	6.4	14.5	.8832	.90	.02091	6.7
27	14.1	.8752	.46	.01898	7.3	14.5	.8911	.57	.01898	7.5
28	12.3	.7089	.71	.01852	6.1	12.7	.7772	.53	.01829	6.8

[a]Ratio of radius to wall-thickness.

[b]The control females had a significantly lower body weight than the control males at the time of removal of both the initial and the final segment.

[c]Standard deviation.

**Difference was significant at $P<0.01$.

Table 6. (Continued)

Group	Initial segment: Body weight (kg)	Circum-ference (cm)	Length (cm)	Wall-thickness (cm)	R/WT	Final segment: Body weight (kg)	Circum-ference (cm)	Length (cm)	Wall-thickness (cm)	R/WT
Mean	13.4± .7	.8374± .0755	.58± .09	.01929± .00174	6.9± .6	13.9±[d*] .8	.8471± .0465	.64± .15	.01738± .00395	7.0± .4
Overall control mean	11.8± 1.8	.8282± .0713	.59± .09	.01855± .00239	7.2± .9	12.3± 1.8	.8286± .0567	.62± .12	.01839± .00343	6.9± .8
Treatment females										
03	8.6	.8069	.59	.02269	5.7	---[e]	---	---	---	---
05	9.1	.8376	.61	.02191	6.1	9.1	.8307	.57	.01099	6.3
07	9.1	.8248	.67	.02052	6.4	7.3	.8079	.73	.01821	7.1
08	10.5	.7921	.57	.02037	6.2	10.9	.7861	.51	.01667	7.5
09	10.2	.7812	.73	.01991	6.2	---[e]	---	---	---	---
Mean	9.5±[f*] .8	.8085± .0231	.63± .07	.02108± .00117	6.1± .3	9.1± 1.8	.8082± .0223	.60± .11	.01862± .00219	7.0± .6

[d] A significant increase in body weight occurred between the initial and the final segment removal dates for these groups.

[e] Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

[f] The treatment females had a significantly lower body weight than the treatment males at the time of removal of the initial artery segment.

* Difference was significant at $P<0.05$.

Table 6. (Continued)

Group	Initial segment: Body weight (kg)	Initial segment: Circumference (cm)	Initial segment: Length (cm)	Initial segment: Wall-thickness (cm)	Initial segment: R/WT	Final segment: Body weight (kg)	Final segment: Circumference (cm)	Final segment: Length (cm)	Final segment: Wall-thickness (cm)	Final segment: R/WT
Treatment males										
21	10.0	.8129	.58	.02014	6.3	6.4	.7832	.81	.01620	7.7
25	12.5	.8743	.64	.01636	8.5	10.9	.8911	.67	.01659	8.5
26	13.9	.8119	.60	.01983	6.5	10.9	.8663	.63	.01698	8.1
29	12.5	.8495	.54	.01620	8.3	11.6	.8634	.54	.02106	6.5
Mean	12.2±	.8372±	.59±	.01813±	7.4±	10.0±[g*]	.8510±	.66±	.01771±	7.7±
	1.6	.303	.04	.00214	1.2	2.4	.0469	.11	.00226	.9
Overall treatment mean	10.7±	.8212±	.61±	.01977±	6.7±	9.6±	.8327±	.64±	.01810±	7.4±
	1.8	.0290	.06	.00220	1.0	2.0	.0423	.11	.00209	.8

[g]A significant decrease in body weight occurred between the initial and the final segment removal dates for these groups.

Table 7. Femoral artery segment dimensions of mongrel dogs

	Right femoral					Left femoral			
Group	Body weight (kg)	Circum-ference (cm)	Length (cm)	Wall-thickness (cm)	R/WT[a]	Circum-ference (cm)	Length (cm)	Wall-thickness (cm)	R/WT
Mongrels									
01	13.6	1.0089	.60	.02508	6.4	1.0129	.65	.02407	6.7
02	27.3	1.2327	.60	.02508	7.8	1.2376	.48	.02832	7.0
03	10.5	0.8535	.46	.01906	7.1	0.8624	.57	.01860	7.4
04	11.4	0.8208	.52	.02387	5.5	0.8812	.53	.02755	5.1
05	10.9	0.9238	.48	.01991	7.4	1.0500	.50	.02759	6.1
Mean[b]	14.7± 7.1	.9854±[c*] .1561	.54± .05	.02391±[c**] .00305	6.6± 0.8				

[a]Ratio of vessel radius to wall-thickness.

[b]The right and left femoral artery dimensions were averaged for each dog and this average value was used to determine the mean.

[c]Significantly greater than the overall group means of the beagle dogs for both the initial and final artery segment

[**]Difference was significant at P<0.01.

[*]Difference was significant at P<0.05.

males lost a significant ($P<0.05$) amount. The overall control group showed a significant ($P<0.01$) rise in body weight and the overall treatment group had a significant ($P<0.05$) decrease in body weight between vessel harvesting dates. The mongrel dog's body weights were not found to be significantly different from the beagle's.

The mongrels had a larger vessel circumference ($P<0.05$) than the control and treatment beagles. The circumference of the femoral artery segments in both the control and the treatment group of beagles did not change from the initial to the final sampling period.

No significant difference in vessel length was found between or within groups.

No significant difference between or within the groups of beagle dogs was found. The mongrels, however, had significantly thicker walls than the control and treatment beagles.

No significant difference between or within groups of beagles or mongrels could be found in the femoral artery radius/wall-thickness ratio (R/WT). The values arrived at in this experiment agree fairly well with those reported by Feigl et al. (1963). Their mean value for the R/WT of femoral arteries from 17 normotensive dogs was 6.4 ± 0.3. The measuring technique used by Feigl et al. (1963) was different in that the removed segment was stretched to its in vivo length and inflated by a balloon within its lumen to the dog's "normal" blood pressure. The vessel was then fixed in formalin and its dimensions were measured histologically with a calibrated microscope.

Circumference-tension relationship

Table 8 lists the date and side from which the initial and final femoral arteries were removed from each beagle dog.

The circumference-tension measurements for the individual beagle dogs are shown in Tables A-59 through A-66. The means for the initial femoral artery segments are shown in Table A-67 and for the final segments in Table A-68. The measurements of circumference-tension were also made on femoral segments from mongrel dogs to test the hypothesis that a difference existed between the right and left femoral arteries. The resulting measurements are listed in Tables A-69 and A-70 for the right and left femoral arteries, respectively. The means for the mongrel dog's femoral artery circumference-tension data are given in Table A-71. Figure 27 shows the mean values for the mongrels' right and left vessels and the beagles' initial and final vessels.

Paired t-tests comparing the right and left femoral arteries of the mongrel dogs revealed no significant difference between them.

The control beagles showed a significant ($P<0.05$) decrease in their femoral artery circumference from the initial to the final arterial segment at the higher levels of vessel tension (56.25, 62.50, 68.75, 75.00, 81.25, 87.50, 93.75, and 100.00 gms tension). The circumference-tension relationship was not found to be significantly altered by the experiment. When the mongrels' vessels were compared with the beagles' it was found that the former group had consistently larger values for circumference at all levels of tension ($P<0.05$).

Table 8. Dates of femoral artery segment removal from beagles

Dog	Sex	Initial segment		Final segment	
		Date	Side	Date	Side
01	female	4-8-72	left	8-21-72	right
02	female	4-5-72	left	8-22-72	right
03[a]	female	4-8-72	left		
04	female	4-8-72	right	8-22-72	left
05	female	4-8-72	right	8-21-72	left
06	female	4-6-72	left	8-23-72	right
07	female	4-6-72	left	8-22-72	right
08	female	4-8-72	right	8-23-72	left
09[a]	female	4-6-72	left		
10	female	4-4-72	left	8-23-72	right
21	male	4-5-72	right	8-21-72	left
22	male	4-8-72	left	8-21-72	right
23	male	4-7-72	right	8-22-72	left
24	male	4-7-72	left	8-22-72	right
25	male	4-8-72	right	8-22-72	left
26	male	4-6-72	right	8-22-72	left
27	male	4-4-72	right	8-23-72	left
28	male	4-6-72	right	8-23-72	left
29	male	4-7-72	left	8-23-72	right

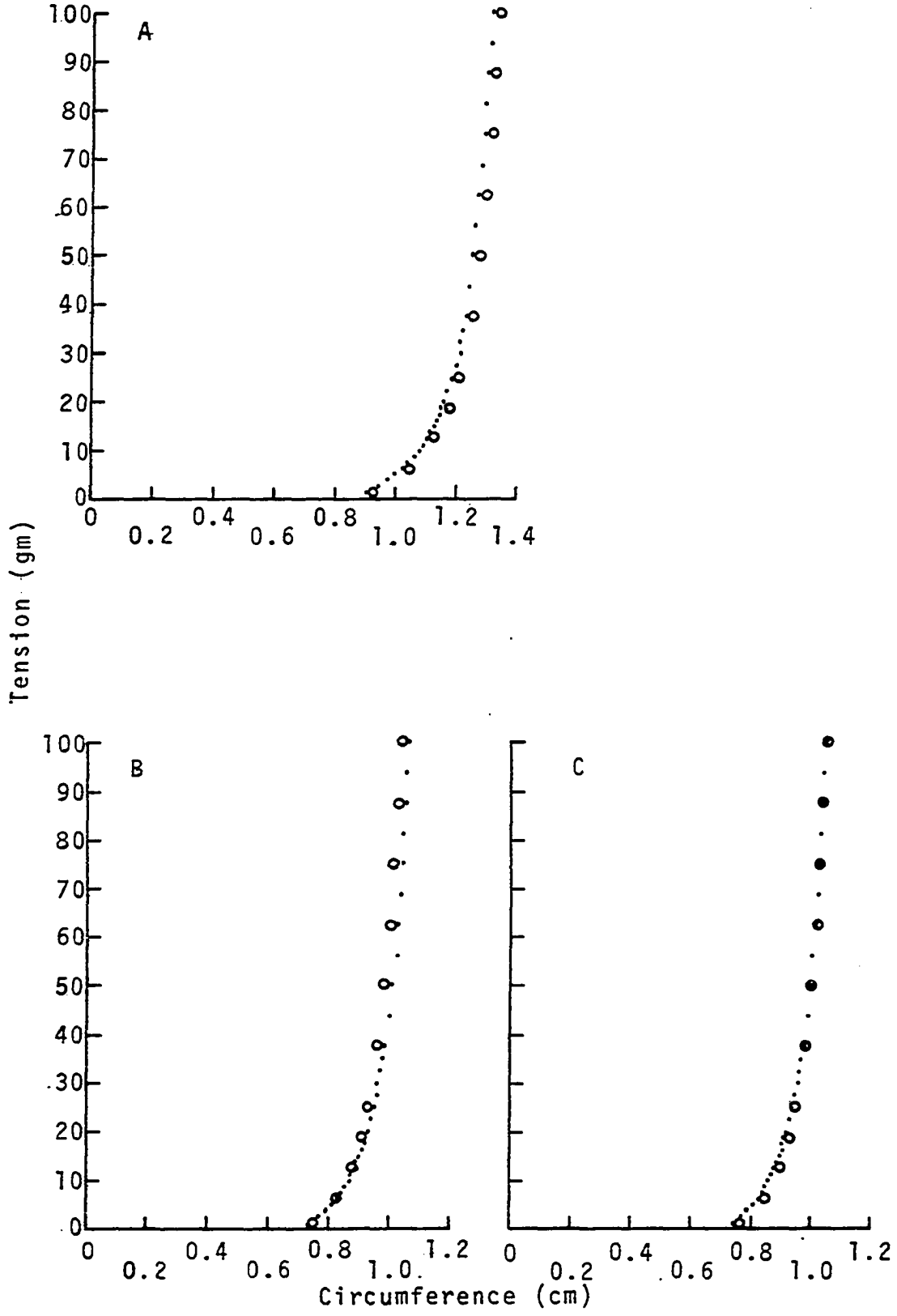
[a]Dogs 03 and 09 died during the experiment and a final arterial segment was not obtained.

Figure 27. Mean circumference-tension values for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet; some of the values in the graphs represented by 0 have been omitted

A. Mongrel dogs
   Right femoral artery •
   Left femoral artery O

B. Control dogs
   Initial femoral artery •
   Final femoral artery O

C. Treatment dogs
   Initial femoral artery •
   Final femoral artery O


A
B
C
Tension (gm)
Circumference (cm)
100
90
80
70
60
50
40
30
20
10
0
0.2
0.4
0.6
0.8
1.0
1.2
1.4

Slope I

The slopes of the circumference-tension graphs (slope I) for the initial and final artery segments of the beagles are given in Tables A-72 and A-73. The values for slope I from the mongrel dogs are listed in Table A-74. The mean values for all three groups of dogs are shown in Figure 28.

Utilizing paired t-tests, no difference was found in the slope I between the initial and final vessels from the control group of beagles. The values for slope I of the final segments of femoral artery from the treatment beagles were found to be significantly ($P<0.05$) greater than the value for the initial vessel at 20 gms tension. No significant difference was found at any other level of tension tested. The right and left femoral artery measurements from mongrel dogs were not significantly different from one another.

A comparison of the means for slope I between the mongrels and the beagles was made using the Student's t-test. It was found that the former group had a significantly lower value than the control beagles' final segment at 20 gms ($P<0.05$) and the treatments beagles' final segment at 20 gms ($P<0.01$) and 40 gms ($P<0.05$) of tension.

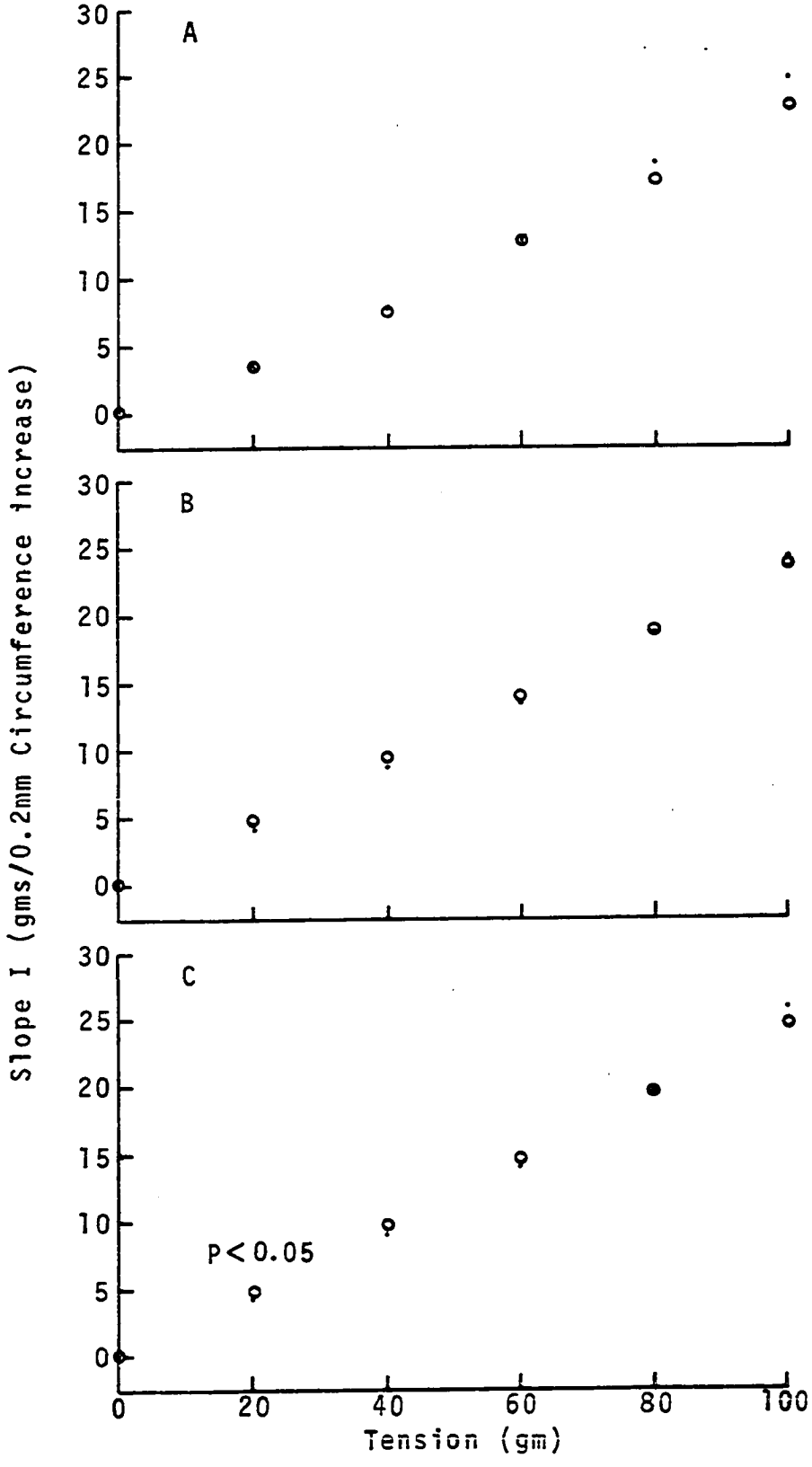
The increase in slope I from the initial to the final segment for the treatment beagles at 20 gms may indicate an increase in arterial stiffness at that level of tension. The higher values for slope I in the control and treatment group of beagles may indicate that they had stiffer arteries than the mongrel dogs at the lower levels of tension.

Figure 28. Mean slope I values (slope of the circumference-tension graph) for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet

A. Mongrel dogs
   Right femoral artery •
   Left femoral artery O

B. Control dogs
   Initial femoral artery •
   Final femoral artery O

C. Treatment dogs
   Initial femoral artery •
   Final femoral artery O


A
B
C
30
25
20
15
10
5
0
P < 0.05
0
20
40
60
80
100
Tension (gm)
Slope I (gms/0.2mm Circumference Increase)

Strain ratio-tension relationship

The individual dog values and group means for the strain ratio-tension relationship in beagles are given in Tables A-75 through A-84. The mean values are shown in Figure 29. The values for the strain ratio-tension relationship from the mongrel dogs are given in Tables A-85 through A-87.

Paired t-tests failed to reveal any significant difference between the right and left femoral arteries of mongrel dogs. Likewise no change was found to occur in the control or treatment group of beagles from the initial to the final artery segment.

There was an observed trend for the arteries of mongrel dogs to have less wall tension at equal values of strain ratio than arteries from both groups of beagles. This trend may indicate that the arteries from the beagles were either stiffer or had thicker walls than those from the mongrels. The mongrels were found to have significantly thicker walls than the beagles at zero tension (Table 7).

Slope II

The slope of the strain ratio-tension graphs (slope II) for the individual beagle dogs as well as the group means are listed in Tables A-88 and A-89. Figure 30 shows the mean values for each group. The values for slope II from the mongrel dogs are given in Table A-90.

Paired t-tests did not reveal a significant difference between the right and left femoral arteries of mongrel dogs. Likewise no significant change was detected in the arteries of the beagle dogs taken initially when compared to those taken at the end of the experiment.

Figure 29. Mean strain ratio-tension value for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet

A. Mongrel dogs
   Right femoral artery •
   Left femoral artery O

B. Control dogs
   Initial femoral artery •
   Final femoral artery O

C. Treatment dogs
   Initial femoral artery •
   Final femoral artery O

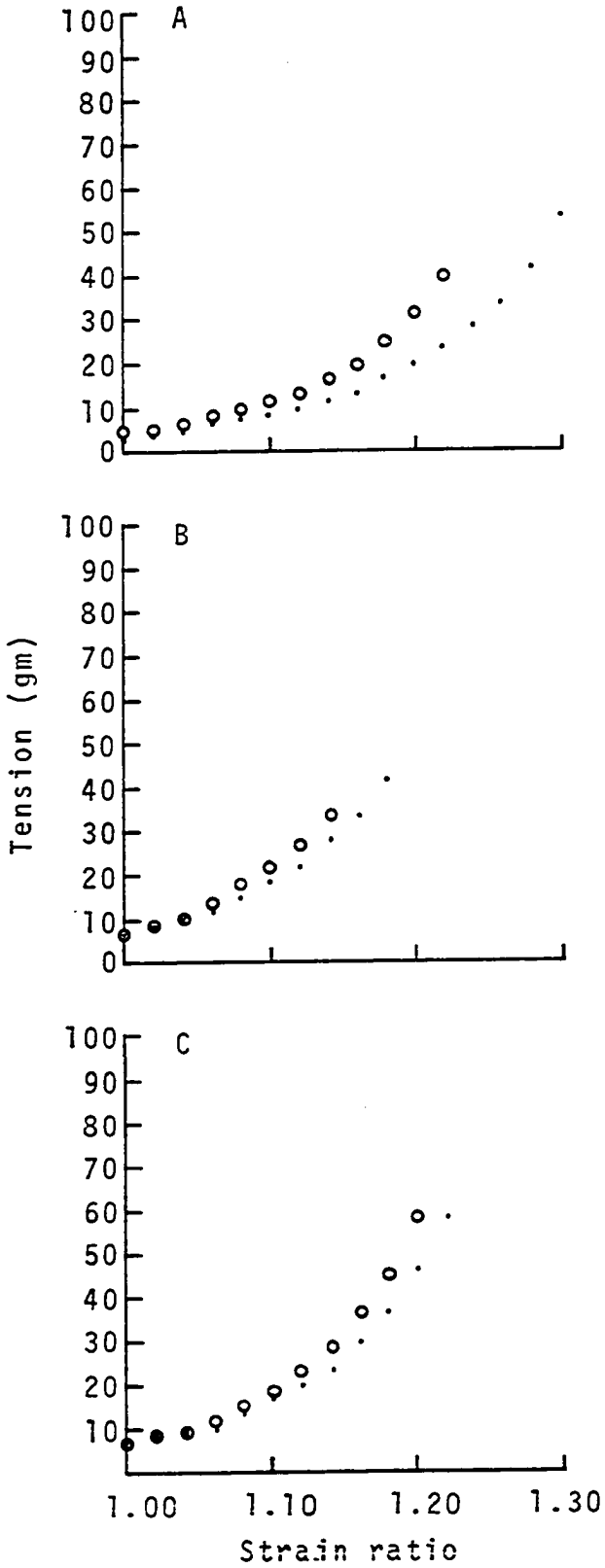
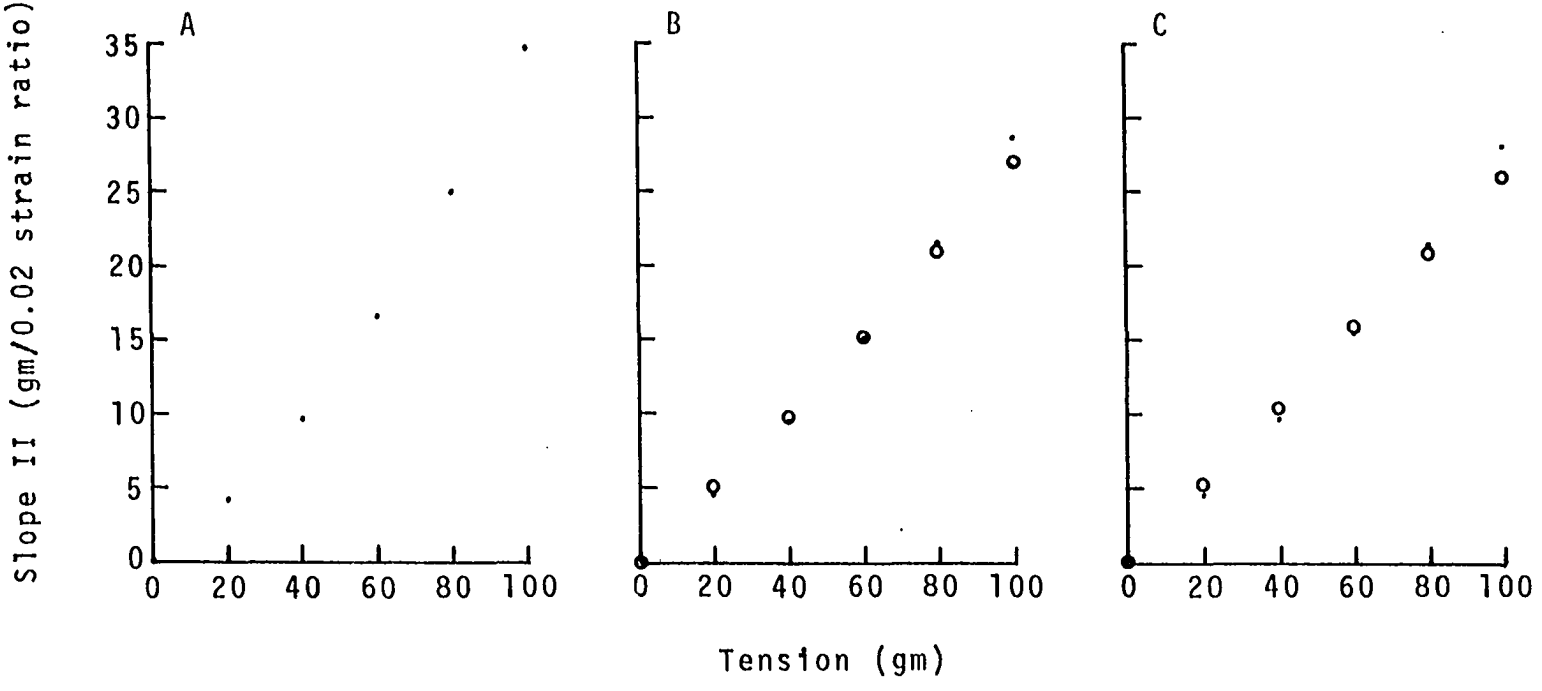

A
B
C
100
90
80
70
60
50
40
30
20
10
0
Tension (gm)
1.00
1.10
1.20
1.30
Strain ratio

Figure 30. Mean slope II values (slope of the strain ratio-tension graph) for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet

A. Mongrel dogs
Right femoral artery •
(Left femoral artery values were omitted from the graph because they were almost identical to the right femoral artery values)

B. Control dogs
Initial femoral artery •
Final femoral artery O

C. Treatment dogs
Initial femoral artery •
Final femoral artery O


A
B
C
35
30
25
20
15
10
5
0
0 20 40 60 80 100
Slope II (gm/0.02 strain ratio)
Tension (gm)

A trend existed in which the beagles had higher values for slope II at 20 gms and lower values at 100 gms tension than the mongrels. This trend was substantiated by a Student's t-test on group means which revealed that the slope II of the treatment group's final artery was significantly ($P<0.05$) different from the mongrel dogs' mean at 20 and 100 gms in the directions indicated above.

Strain ratio-stress relationship

The strain ratio-stress data for the individual vessels and the means for each group of beagle dogs are shown in Table A-91 through A-100. The mean values are graphically illustrated in Figure 31. Measurements made on the right and left femoral arteries of mongrel dogs are listed in Tables A-101 and A-102 and the overall means for the mongrels are given in Table A-103.

Paired t-tests between the right and left femoral arteries of mongrels failed to distinguish a significant difference. It was concluded that the right and left femoral arteries were similar in their strain ratio-stress relationship.

Comparisons between the initial and final artery segments of beagles in the control and treatment groups using a paired t-test did not reveal a significant change in either group over the course of the experiment.

There was a trend for the beagles to have higher stress values than mongrels at the same strain ratio.

Slope III

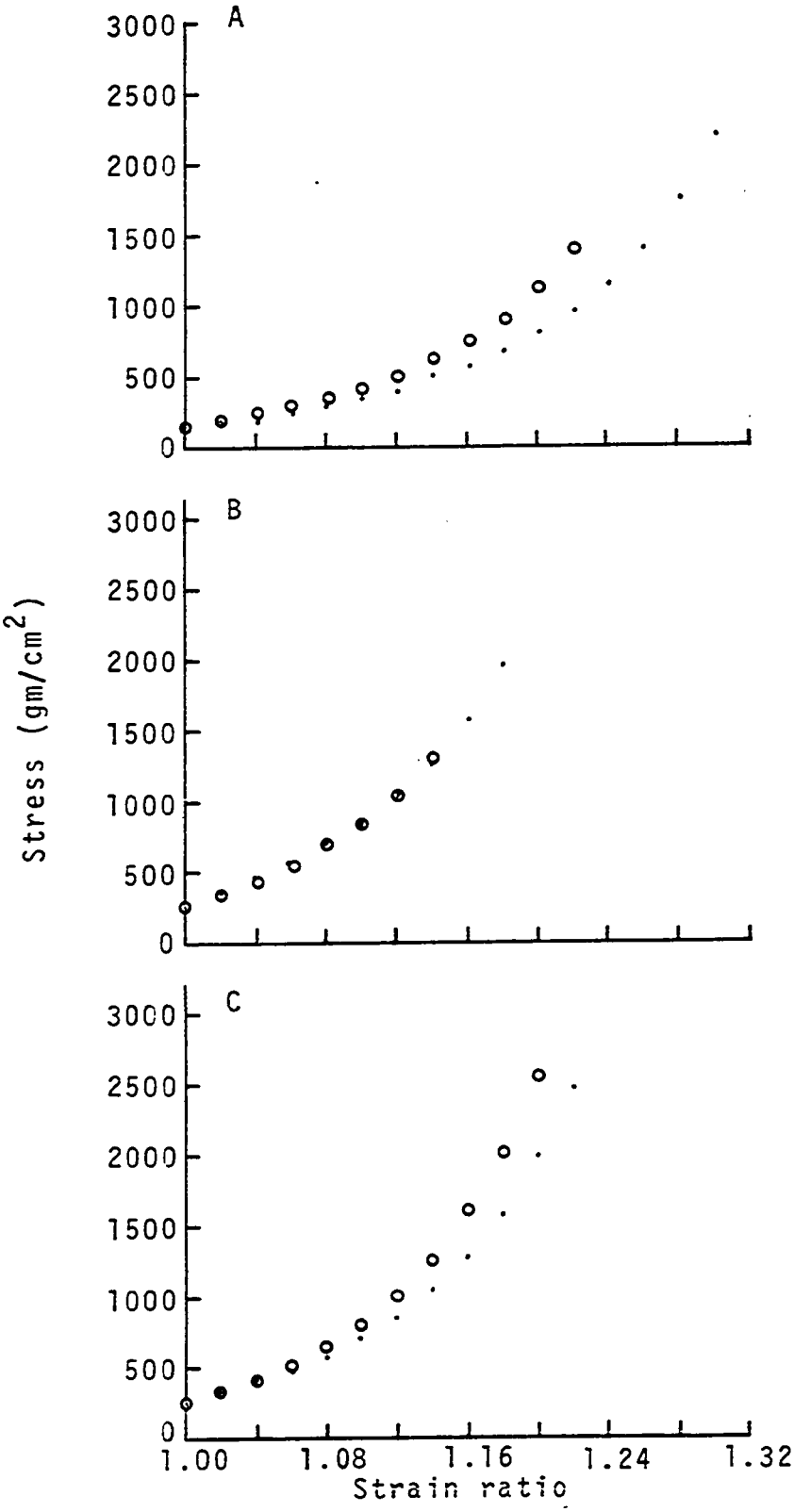
The increment in stress for a 0.02 increase in the strain ratio was determined and the individual dog and group means for the beagles and

Figure 31. Mean strain ratio-stress values for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet

A. Mongrel dogs
   Right femoral artery •
   Left femoral artery O

B. Control dogs
   Initial femoral artery •
   Final femoral artery O

C. Treatment dogs
   Initial femoral artery •
   Final femoral artery O


A
B
C
3000
2500
2000
1500
1000
500
0
Stress (gm/cm^2)
1.00
1.08
1.16
1.24
1.32
Strain ratio

mongrels are listed in Tables A-104 through A-106. The group means for the mongrels, control beagles, and treatment beagles are graphed in Figure 32.

Paired t-tests between the right and left femoral arteries of the mongrel dogs did not reveal a significant difference. Similar tests between the initial and final segments of the control and treatment beagles were also performed. The mean value for slope III in the final segment was found to be significantly ($P<0.05$) greater than the mean value for slope III in the initial segment at 500 gm/cm^2 stress for the treatment beagles. No other significant changes in slope III in the beagles was found to have occurred over the course of the experiment.

Student's t-test was used to compare means for the beagles and mongrels. A trend existed for the beagles to have higher values for slope III at the smaller stress levels and lower values for slope III at larger stress levels than the mongrel dogs. This trend was substantiated by the t-tests which revealed that the mean for slope III at 500 gm/cm^2 in the final segments of the control and treatment beagles were significantly ($P<0.05$) larger than the slope III of mongrel dogs at the same stress. Likewise, the slope III for the final segment of arteries from the treatment beagles at 1000 gm/cm^2 was significantly ($P<0.05$) greater than the slope III for the mongrel dogs at the same stress.

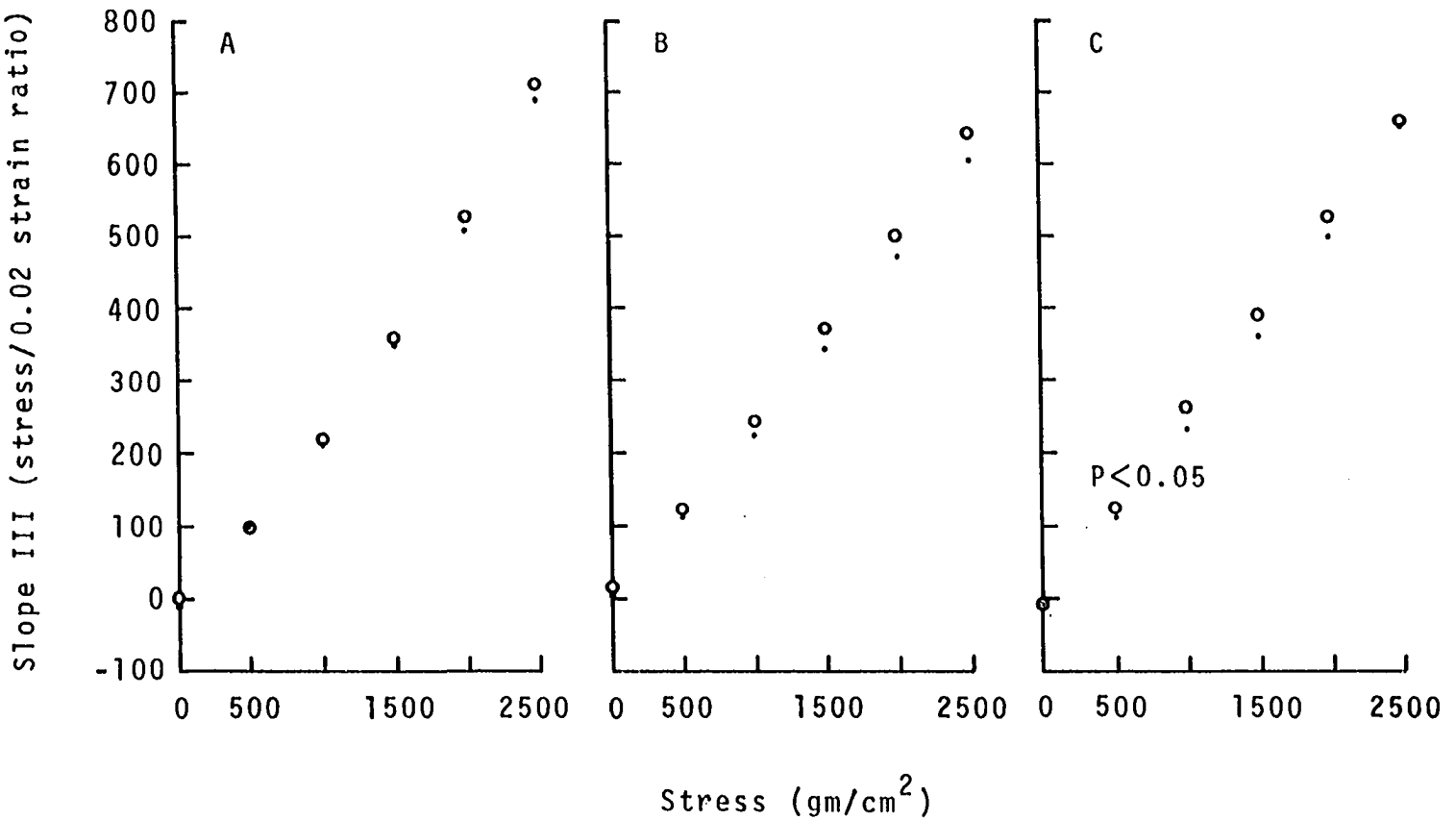
These data could be interpreted to mean that the arteries of the treatment beagles were stiffer at a stress of 500 gm/cm^2 after undergoing the starvation-refeeding regimens. Since this stiffness increase was not found at other levels of stress it is of doubtful significance.

Figure 32. Mean slope III values (slope of the strain ratio-stress graph) for mongrel dogs, control dogs maintained on a commercial diet, and treatment dogs which were starved and refed a high carbohydrate diet

A. Mongrel dogs
   Right femoral artery •
   Left femoral artery O

B. Control dogs
   Initial femoral artery •
   Final femoral artery O

C. Treatment dogs
   Initial femoral artery •
   Final femoral artery O


Slope III (stress/0.02 strain ratio)
800
700
600
500
400
300
200
100
0
-100
A
B
C
P<0.05
0
500
1500
2500
Stress (gm/cm^2)

One could also conclude that a difference existed between the beagles and the mongrels. The beagles tended to be stiffer at low stresses while the mongrels tended to be stiffer at high stresses.

Discussion

The technique of removing one femoral artery from a dog prior to some experimental procedure and using that vessel as a control for comparison with the opposite femoral artery removed at a later date has been used previously (Feigl et al., 1963). No experiments have been conducted to determine if any differences exist in the mechanical properties of the bilateral femoral arteries. In the present study the femoral arteries of five mongrel dogs were bilaterally removed and compared. There was no difference between the right and left femoral arteries in any of the following measurements: circumference, length, R/WT, wall-thickness, circumference-tension, slope I, strain ratio-tension, slope II, strain ratio-stress, and slope III. The use of this technique would seem very advantageous because each dog serves as its own control thus eliminating many variables which would otherwise have to be considered when comparing vessel measurements, viz., size, age, and sex of the dogs.

Aging is generally associated with increased stiffness of arteries (Hallock, 1934; Wilens, 1937; Hass, 1942; Burton, 1951; Learoyd and Taylor, 1966; Fischer and Llaurado, 1967).

The control beagles were used to study what effect 140 days of aging may have on arterial stiffness. None of the slopes were altered but the circumference-tension graph was shifted to the left at the higher levels of tension (56.25-100.00 gms tension). This shift of the graph indicated

that the arteries were stretching a smaller amount with equal tension after aging for 140 days. An increase in vessel stiffness would produce the same affect. It was expected that if the graph was shifted at the higher levels of tension there should be a change in slope prior to that point. Such a change in slope I was not found, although the relatively few samples taken for the slope determination may have missed it. Also, if the control beagles' arteries became stiffer during the 140 day experiment the other graphs (strain ratio-tension and strain ratio-stress) would have also been altered. It would thus seem unlikely that aging occurred to any significant degree in this experiment.

Starvation-refeeding regimens have been found to cause an increase in arterial stiffness in swine (Hembrough and Link, 1968), rats (Hembrough and Riedesel, 1970), and dogs (Crouch, 1968). The present study found an increased stiffness in the femoral arteries of starved-refed beagles at tensions of 20 gms in slope I and 500 gm/cm^2 in slope III. These slope values are similar to elastic moduli and an increase in their value indicates an increase in vessel stiffness. A significant change in the slope of a graph should alter the subsequent points on that graph. However, the graphs from which slope I and slope III were determined did not show any changes in the area of or beyond the area from which the altered slope was obtained. This makes it very difficult to conclude definitely that starvation-refeeding altered the stiffness of the femoral artery.

The most consistent observation made from the stretch-tension data was that the arteries from beagles were mechanically quite different from arteries of mongrels. Part of this difference was due to the zero circum-

ference and wall thickness which were both larger in the mongrels ($P<0.05$) than in the beagles. But, conversion from circumference to strain ratio did not completely remove the difference. Likewise, conversion from wall tension to stress should have removed the effect of a larger wall thickness but some differences still remained. It can be concluded that the femoral arteries of the beagles were stiffer at lower levels of stretch than the mongrels. The lower portion of a stretch-tension graph is due to elastic fibers (Roach and Burton, 1957) and their damage with collagen replacement could cause the altered slopes. Differences between beagles and mongrels have been observed previously by Crouch (1968).

It is rather hard for a physiologist to relate to values like grams tension or stresses of gm/cm^2. A valid question is how do the stresses applied by stretching relate to an internal pressure within the vessel in mm Hg. Unfortunately there seems to be a controversy in the literature on this subject but one method is to use the formula (Peterson, 1966):

$$P = T \times \frac{WT}{R} \times 980 \times 7.5 \times 10^{-4}$$

where

P = pressure in mm Hg

T = tension in gm/cm^2 of wall area

WT = wall thickness in cm

R = vessel radius in cm

980 = conversion factor for grams into dynes

7.5×10^{-4} = conversion factor for dynes into mm Hg

Unfortunately, my data do not include the wall thickness/radius ratio at all levels of tension. For calculation, as an example, a representative value (0.15) at zero tension was used:

$$P = 1000 \text{ gm/cm}^2 \times 0.15 \times 980 \times 7.5 \times 10^{-4}$$

$$= 110 \text{ mm Hg}$$

Most vessels were stretched to a tension of 2500 gm/cm^2 and some to as high as 5400 gm/cm^2.

Another way of calculating the pressure is to use the formula (Peterson, 1966):

$$P = (T'/R) \times 980 \times 7.5 \times 10^{-4}$$

where

P = pressure in mm Hg

T' = tension in gm/cm of circumference

R = radius in cm

To apply this formula to the data the mean circumference at 100 gm tension for the control beagles was used:

$$P = \frac{100 \text{ gm}}{1.00592 \text{ cm}} \times \frac{1}{.1686} \times 980 \times 7.5 \times 10^{-4}$$

$$= 412 \text{ mm Hg}$$

No matter which of the above two relationships is more correct it would appear that most of the vessels underwent a stretch equal to an internal lumen pressure of 200-400 mm Hg. The experiment was, thus, conducted within and slightly beyond physiologic limits of vessel stretch when related to the blood pressure necessary to cause comparable wall tensions.

Heart Weights

The mean weights of the ventricles and their relationship to body weight are shown in Table A-107. The individual values for each dog can be found in the same table.

The control group had a significantly greater ($P<0.05$) body weight at the time of post mortem than the treatment group. This was probably due to starvation and the fact that the treatment dogs had just not been fed long enough to regain all the weight they lost.

The control male body weights were significantly greater than the females ($P<0.01$). The males also had significantly ($P<0.01$) heavier right and left ventricles. Since the percentage of each ventricle and the ratio of total heart weight to body weight were not altered an abnormal hypertrophy was not considered to be present.

The significant reduction in the means for total ventricular weight, right ventricular weight, and left ventricular weight found in the overall treatment group when compared to the control group was probably due to differences in body size. This was concluded because the percentages of each ventricle and the ratio of total ventricular weight to body weight were not significantly altered.

The values for the ratio of total ventricular weight to body weight found in this experiment agree with the normal values reported by Bishop and Cole (1969) which ranged from 5.0 to 11.0 gm/kg.

CONCLUSIONS

1. Starvation initiated normocytic normochromic anemia, neutropenia, eosinopenia, and hypoproteinemia.

2. The cardiovascular effects of starvation include a decrease in heart rate, decrease in systolic and diastolic blood pressures, and dorso-cranial deviation in the orientation of the maximum T wave vector.

3. Starvation and refeeding of a high carbohydrate diet neither produced blood pressures above control levels nor significantly altered the stretch-tension characteristics of the femoral artery.

4. Mongrel dogs had femoral arteries with a larger circumference and wall thickness than those from beagles. Femoral arteries from mongrels tended to be less stiff at low tensions and more stiff at high tensions than those from beagles.

SUMMARY

The role of starvation-refeeding in producing hypertension was unclear. A review of the literature suggests that starvation followed by refeeding will lead to hypertension (Brozek et al., 1948; Wilhelmj and McCarthy, 1963; Smith et al., 1964). Other experiments, however, have failed to demonstrate a definite relationship (Johnson, 1966; Crouch, 1968; Hembrough and Riedesel, 1970).

High sucrose diets have been incriminated as causing hypertension (Ahrens, 1974). Carbohydrate diets have been found to decrease the natriuresis of starvation (Bloom, 1962, 1967; Katz et al., 1968; Hoffman et al., 1971; Veverbrants and Arky, 1969). A decrease in natriuresis could theoretically lead to hypertension.

The present experiment was conducted to investigate the effects of starvation and refeeding of a high carbohydrate diet on the cardiovascular system of the dog. Nineteen beagle dogs were divided into two groups which had similar systolic and diastolic blood pressures. Ten of the dogs (five males and five females) served as a control group and remained on a commercial canine diet throughout the experiment. Nine of the dogs (four males and five females) were in the treatment group which underwent two starvation-refeeding regimens. The length of the starvation I, refeeding I, starvation II, and refeeding II regimens were 30, 20, 17, and 31 days respectively. Two of the treatment group females died during the refeeding I regimen. Post mortem examination failed to reveal the cause of death. The refeeding diet provided 70% of its calories from carbohydrates (27% from sucrose and 43% from rice and other starches).

The control dogs slowly increased in body weight during the experiment. The treatment dogs decreased in body weight during the starvation periods and gained weight during the refeeding periods.

The starvation-refeeding regimens led to the development of a normocytic normochromic anemia. Starvation was accompanied by neutropenia and eosinopenia which were reversed by refeeding.

The heart rate of the treatment group was decreased significantly by starvation and a few second degree heart blocks were found. The bradycardia was rapidly reversed by refeeding.

Starvation was also associated with a decrease in systolic and diastolic blood pressures. The magnitude of decrease was greater for the systolic pressure than the diastolic pressure. Refeeding returned the pressures to control levels and did not produce hypertension.

McFee-Parungao vectorcardiograms on the beagles were analyzed. The maximum and half-area QRS vectors were unchanged by either starvation or refeeding. The orientation of the maximum T wave vector was, however, shifted cranially and dorsally from its normal ventrad caudad position. Refeeding returned the orientation to the control position.

The stretch-tension characteristics of femoral artery segments were determined in vitro. The characteristics of a 1 centimeter segment removed from one leg prior to the beginning of the experiment were compared to a segment of the opposite femoral artery removed at the end of the experiment. Various plots were used to analyze the stretch-tension data including:

	abscissa	ordinate
1.	circumference (cm)	tension (gm)
2.	tension (gm)	slope of the circumference-tension graph (gm/0.02 mm circumference increase)
3.	strain ratio	tension (gm)
4.	tension (gm)	slope of the strain ratio-tension graph (gm/0.02 strain ratio increase)
5.	strain ratio	stress (gm/cm^2)
6.	stress (gm/cm^2)	slope of the strain ratio-stress graph (gm/cm^2/0.02 strain ratio increase)

No definite change in the femoral artery stretch-tension characteristics due to aging or starvation-refeeding could be found.

The right and left femoral arteries from five mongrel dogs were sampled and compared to determine if the two sides were similar in stretch-tension characteristics. No significant difference was found between sides when compared in the same manner as were the segments from the beagle dogs.

There was a trend for the arteries from the mongrel dogs to be less stiff at low tensions and more stiff at high tensions than those from beagles.

The femoral artery segments from the beagles and mongrels were also measured to determine their circumference, length, wall-thickness, and radius:wall-thickness at zero tension. No significant effects due to aging or starvation-refeeding were found in the beagle dogs. No significant difference between the right and left sides was found in the mongrel dogs. But, the mongrel's vessels had significantly larger values

for circumference and wall thickness even though there was not a significant difference in body weight between the two groups.

Heart weights measured on each beagle at their post mortem following the experiment failed to detect any hypertrophy.

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APPENDIX

Table A-1. Body weights of dogs

Group	Regimen									
	Control						Starvation I			
	0	1	2	3	4	5	6	7	8	9
	(weeks)						(weeks)			
Control females	(kg)									
01	9.8	9.5	9.8	9.8	9.3	9.5	9.1	10.0	10.5	10.0
02	10.5	10.0	10.5	10.5	10.0	10.2	10.0	10.0	10.5	10.0
04	9.8	9.8	10.0	10.5	10.0	9.8	10.0	10.5	10.2	10.5
06	10.0	10.0	10.2	10.2	10.2	10.5	10.0	10.0	10.5	10.0
10	10.9	11.4	11.4	11.8	11.4	11.8	12.3	12.3	11.8	12.3
Mean	10.3 ± 0.7[a]						10.5			
Males										
22	13.6	13.6	13.6	13.6	13.4	13.6	13.6	13.4	13.6	14.1
23	13.6	13.4	14.1	13.9	13.9	14.5	13.6	14.1	13.9	14.1
24	13.6	13.6	13.9	14.1	14.1	13.9	13.6	14.1	14.1	14.1
27	14.1	13.9	14.5	13.6	13.9	14.1	14.1	14.1	14.3	14.5
28	12.3	12.3	12.3	12.3	12.3	12.7	12.3	12.3	12.7	12.7
Mean	13.5						13.7			
Overall control mean	11.9						12.1[b**]			

[a]Standard deviation, derived from the error mean squares of the analysis of variance. The pooled standard deviation applies to all mean values in the table.

[b]Mean was significantly different from the overall treatment mean for the same regimen.

**Difference was significant at $P<0.01$.

*Difference was significant at $P<0.05$.

Regimen									
Refeeding I			Starvation II			Refeeding II			
10	11	12	13	14	15	16	17	18	19
(weeks)			(weeks)			(weeks)			
				(kg)					
10.5	10.0	10.2	10.0	10.0	9.5	10.0	10.5	10.2	10.0
10.0	10.5	10.5	10.9	10.5	10.0	10.7	10.2	10.5	10.5
10.5	10.5	10.0	10.0	10.2	10.0	10.9	10.9	11.4	11.1
10.0	10.5	10.2	10.5	10.0	10.2	10.0	10.5	10.5	10.0
11.8	11.8	11.8	11.8	12.3	12.0	11.8	11.8	12.0	11.8
10.6 ± 0.7			10.5			10.8			
13.6	13.6	13.4	13.6	13.6	13.4	14.1	13.6	14.1	13.6
14.5	13.9	13.9	14.1	13.9	13.6	13.6	14.1	13.6	13.9
15.0	14.8	14.5	14.5	15.0	14.3	14.5	14.5	15.0	15.0
14.8	14.5	14.5	14.5	14.5	13.6	14.3	14.5	13.6	13.6
12.3	13.2	12.7	12.7	12.7	12.7	12.3	12.7	12.5	12.5
	13.9			13.8			13.8		
	12.3^{b**}			12.2^{b**}			12.3^{b*}		

Table A-1. (Continued)

Group	Regimen: Control 0	1	2	3	4	5	Starvation I 6	7	8	9
			(weeks)					(weeks)		
Treatment females					(kg)					
03	8.6	8.9	9.1	8.9	8.9	8.6	8.0	7.3	6.8	6.1
05	9.1	8.9	9.1	9.1	8.9	9.1	8.2	7.3	6.1	5.9
07	9.1	9.1	9.1	8.9	9.1	9.1	7.7	6.8	6.8	5.9
08	10.5	11.1	11.1	10.9	11.4	10.9	10.0	9.1	8.4	8.0
09	10.2	10.7	10.5	10.9	10.2	10.5	9.1	8.2	7.7	6.4
Mean			13.5					7.5		
Males										
21	10.0	10.5	10.2	10.5	10.5	10.5	9.1	8.2	7.7	7.0
25	12.5	11.8	12.3	13.0	12.3	12.3	11.8	10.0	9.3	8.2
26	13.9	13.6	14.1	13.9	13.9	14.1	12.5	11.4	10.9	9.3
29	12.5	12.7	12.5	13.2	12.3	12.3	11.4	10.5	10.0	8.9
Mean			12.3					9.8[d*]		
Overall treatment mean			10.8					8.5[d**]		

[c]Dogs 03 and 09 died during the refeeding I regimen.

[d]Mean was significantly different from the control regimen mean for the same group.

Regimen									
Refeeding I			Starvation II			Refeeding II			
10	11	12	13	14	15	16	17	18	19
(weeks)			(weeks)			(weeks)			
				(kg)					
5.7	7.0	-[c]	-	-	-	-	-	-	-
5.0	8.0	9.1	7.7	6.6	6.6	8.0	9.1	9.1	9.8
5.5	7.0	7.5	6.8	5.7	5.2	6.4	7.3	8.0	8.6
7.5	9.5	10.5	9.5	8.2	8.2	9.9	10.9	11.1	11.4
5.2	6.6	-[c]	-	-	-	-	-	-	-
	7.2			7.2			9.1		
5.5	5.9	7.5	6.4	5.5	4.1	5.5	6.4	6.8	6.8
8.4	9.5	10.0	10.0	8.6	8.6	9.8	10.0	11.6	12.3
9.1	10.7	11.4	10.5	9.3	8.4	10.0	10.9	11.8	12.5
9.1	10.7	11.4	10.0	9.5	8.6	10.7	11.6	12.0	12.3
	9.2[d*]			8.3[d**]			10.1[d*]		
	8.2[d**]			7.8[d**]			9.7[d*]		

Table A-2. Analysis of variance plan and observed mean squares for body weight in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	693.64**
Sex	1	610.80**
Sex · group	1	40.48
Dog (sex · group) error	15	22.60
Regimen	4	56.78[a]**
Group · regimen	4	41.36[a]**
Sex · regimen	4	1.73[a]
Sex · group · regimen	4	1.66[a]
Error	329	0.43[a]
Total	363	

[a]A more conservative estimate of significance was used in which the d.f. were divided by the treatment regimen d.f.

**Statistical significance is at $P<0.01$.

Table A-3. Blood hemoglobin concentration in dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(gm/100 ml)		
Control females					
01	17.6	19.2	21.0	19.6	18.0
02	19.5	21.2	20.8	21.3	19.5
04	17.8	19.9	18.8	18.3	16.0
06	17.7	16.2	15.9	16.2	17.6
10	17.8	16.0	16.1	15.6	15.1
Mean	18.1 ± 1.0[a]	18.5	18.5	18.2	17.2
Males					
22	19.5	18.3	18.1	17.3	17.8
23	19.5	20.9	19.2	19.6	19.0
24	19.7	18.2	17.7	17.8	15.2
27	18.7	18.2	18.3	18.1	16.8
28	17.3	18.6	17.7	17.6	17.0
Mean	18.9	18.8	18.2	18.1	17.2[b**]

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at $P<0.01$.

Table A-3. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(gm/100 ml)		
Over control mean	18.5	18.7[c*]	18.4[c**]	18.1[c*]	17.2[bc**]
Treatment females					
03	16.4	16.0	-[d]	-	-
05	16.2	14.4	13.8	14.8	13.9
07	17.7	18.8	14.4	17.3	11.9
08	18.0	16.6	15.2	16.4	13.6
09	20.2	22.4	-[d]	-	-
Mean	17.7	17.6	14.5	16.2[b*]	13.1[b**]
Males					
21	18.3	17.8	13.4	17.1	10.3
25	17.8	18.8	14.0	16.6	13.6
26	20.5	17.6	14.6	18.3	13.9

[c]Overall control mean was significantly larger than the overall treatment mean at the same regimen.

[d]Dogs 03 and 09 of the treatment group died during refeeding I.

*Difference was significant at $P<0.05$.

Table A-3. (Continued)

Group	Control	Starvation I	Refeeding I (gm/100ml)	Starvation II	Refeeding II
29	17.9	16.7	15.1	18.0	13.7
Mean	18.6	17.7	14.3[b**]	17.5	12.9[b**]
Overall treatment mean	18.3	17.7	14.4[b**]	16.9[b**]	13.0[b**]

Table A-4. Analysis of variance plan and observed mean squares for hemoglobin concentration in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	100.83**
Sex	1	1.42
Sex · group	1	0.32
Dog (sex · group) error	15	8.10
Regimen	4	26.73**
Group · regimen	4	11.10**
Sex · regimen	4	1.62
Sex · group · regimen	4	0.41
Error	54	1.06
Total	88	

**Statistical significance is at $P<0.01$.

Table A-5. Red blood cell counts in the dog

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells x 10^6/cu mm)		
Control females					
01	6.48	7.42	7.63	7.15	6.45
02	7.60	7.02	9.29	6.88	7.04
04	8.25	6.81	6.84	3.80	5.69
06	6.96	6.81	7.14	6.47	6.72
10	7.27	5.64	5.87	4.84	5.49
Mean	7.31 ± 0.30[a]	6.74	7.36	5.82[b*]	6.27
Males					
22	8.19	7.40	7.32	6.41	6.73
23	7.48	7.22	9.19	6.79	7.15
24	10.90	6.22	7.52	6.65	5.65
27	6.58	6.95	5.31	6.36	6.64
28	7.15	6.74	7.03	6.67	6.31
Mean	8.07	6.90	7.28	6.58[b*]	6.47[b*]

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly less than the control regimen mean for the same group.

*Difference was significant at $P<0.05$.

Table A-5. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells x 10^6/cu mm)		
Overall control mean	7.69	6.82[b*]	7.32[c*]	6.20[b**]	6.37[bc**]
Treatment females					
03	6.13	5.61	-[d]	-	-
05	6.45	5.36	5.85	3.84	5.42
07	8.78	6.36	5.50	4.82	4.72
08	6.91	6.14	7.56	7.08	5.23
09	8.15	7.87	-[d]	-	-
Mean	7.27	6.26	6.30	5.25[b**]	5.12[b**]
Males					
21	10.12	6.87	5.65	6.19	4.18
25	6.42	7.20	7.19	5.90	5.13
26	7.38	5.14	6.12	6.52	4.81

[c]Overall control mean was significantly larger than the overall treatment mean at the same regimen.

[d]Dogs 03 and 09 of the treatment group died during refeeding I.

[**]Difference was significant at $P<0.01$.

Table A-5. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells x 10^6/cu mm)		
29	7.39	6.18	5.83	6.25	5.38
Mean	7.83	6.34^{b*}	6.20^{b*}	6.22^{b*}	4.90^{b**}
Overall treatment mean	7.55	6.30^{b**}	6.24^{b**}	5.80^{b**}	5.00^{b**}

Table A-6. Analysis of variance plan and observed mean squares for red blood cell count in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	10.87*
Sex	1	2.02
Sex · group	1	0.06
Dog (sex · group) error	15	1.54
Regimen	4	9.31**
Group · regimen	4	0.95
Sex · regimen	4	0.76
Sex · group · regimen	4	0.06
Error	54	0.89
Total	88	

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-7. Packed cell volume in dogs

Group	Control	Starvation I	Refeeding I (0/0)	Starvation II	Refeeding II
Control females					
01	46	52	56	53	52
02	52	57	59	54	54
04	48	48	49	48	47
06	44	44	46	44	48
10	46	44	44	39	46
Mean	47 ± 3[a]	49	51[b*]	48	49
Males					
22	52	52	51	48	52
23	52	54	53	51	54
24	51	47	49	46	45
27	52	50	51	48	49
28	47	47	50	48	49
Mean	51	50	51	48	50

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

*Difference was significant at $P<0.05$.

Table A-7. (Continued)

Group	Control	Starvation I	Refeeding I (0/0)	Starvation II	Refeeding II
Overall control mean	49	50	51[c**]	48[c*]	50[c**]
Treatment females					
03	45	46	-[d]	-	-
05	44	39	39	40	42
07	48	49	38	46	37
08	48	44	43	44	39
09	54	62	-[d]	-	-
Mean	48	48	40[b**]	43[b*]	39[b**]
Males					
21	50	50	38	46	30
25	46	50	37	44	39
26	54	48	44	50	41

[c]Overall control mean was significantly larger than the overall treatment mean at the same regimen.

[d]Dogs 03 and 09 of the treatment group died during refeeding I.

**Difference was significant at $P<0.01$.

Table A-7. (Continued)

Group	Control	Starvation I	Refeeding I (0/0)	Starvation II	Refeeding II
29	49	44	42	47	40
Mean	50	48	40[b**]	47	38[b**]
Overall treatment mean	49	48	40[b**]	45[b**]	38[b**]

Table A-8. Analysis of variance plan and observed mean squares for packed cell volume in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	601.43**
Sex	1	18.93
Sex · group	1	0.71
Dog (sex · group) error	15	58.53
Regimen	4	52.93**
Group · regimen	4	90.56**
Sex · regimen	4	14.32
Sex · group · regimen	4	3.59
Error	54	6.83
Total	88	

**Statistical significance is at $P<0.01$.

Table A-9. Mean corpuscular hemoglobin concentration in dogs

Group	Control	Starvation I	Refeeding I (0/0)	Starvation II	Refeeding II
Control females					
01	38	37	38	37	35
02	38	38	35	39	36
04	37	42	38	38	34
06	40	36	35	36	36
10	38	36	37	40	33
Mean	38 ± 1[a]	38	36	38	35[b**]
Males					
22	38	35	36	36	34
23	38	39	36	39	35
24	39	39	36	38	34
27	36	36	36	37	34
28	37	40	36	37	35
Mean	38	38	36	38	34[b**]

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at $P<0.01$.

Table A-9. (Continued)

Group	Control	Starvation I	Refeeding I (0/0)	Starvation II	Refeeding II
Overall control mean	38	38	36[b**]	38	35[b**]
Treatment females					
03	36	35	-[c]	-	-
05	37	37	35	37	33
07	37	38	37	38	32
08	37	37	35	38	35
09	37	36	-[c]	-	-
Mean	37	37	36	38	33[b**]
Males					
21	37	36	35	37	34
25	39	37	38	38	35
26	38	37	33	36	34
29	37	38	36	38	34
Mean	38	37	36	37	34[b**]
Overall treatment mean	37	37	36	37	34[b**]

[c]Dogs 03 and 09 died during the refeeding I regimen.

Table A-10. Analysis of variance plan and observed mean squares for mean corpuscular hemoglobin concentration in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	9.90*
Sex	1	0.06
Sex · group	1	3.24
Dog (sex · group) error	15	2.13
Regimen	4	36.79**
Group · regimen	4	.27
Sex · regimen	4	.38
Sex · group · regimen	4	.38
Error	54	1.65
Total	88	

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-11. Mean corpuscular volume for dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cubic microns)		
Control females					
01	72	69	73	73	81
02	68	80	63	79	77
04	58	70	72	128	82
06	64	65	64	69	72
10	64	78	75	81	83
Mean	65 ± 9[a]	72	69	86[b**]	79[b*]
Males					
22	63	70	69	74	77
23	70	75	58	75	76
24	47	76	65	70	80
27	78	72	96	76	73
28	66	70	71	72	77
Mean	65	73	72	73	77

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at $P<0.01$.

*Difference was significant at $P<0.05$.

Table A-11. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cubic microns)		
Overall control mean	65	72	71	80[b**]	78[b**]
Treatment females					
03	73	82	-[c]	-	-
05	67	73	67	103	77
07	54	77	70	94	78
08	70	72	57	61	75
09	67	79	-[c]	-	-
Mean	66	77	65	86[b**]	77
Males					
21	49	73	67	74	73
25	72	70	51	74	76
26	72	93	72	77	84
29	66	71	72	75	75
Mean	65	77	66	75	77
Overall treatment mean	66	77[b*]	65	80[b**]	77[b*]

[c]Dogs 03 and 09 from the treatment group died during the refeeding I regimen.

Table A-12. Analysis of variance plan and observed mean squares for mean corpuscular volume in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	0.92
Sex	1	123.18
Sex • group	1	0.60
Dog (sex • group) error	15	122.22
Regimen	4	706.76**
Group • time	4	40.18
Sex • time	4	129.76
Sex • group • time	4	3.04
Error	54	89.06
Total	88	

**Statistical significance is at $P<0.01$.

Table A-13. Total plasma protein concentration in dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(gm/100 ml)		
Control females					
01	6.2	6.3	6.2	5.9	5.8
02	6.0	6.4	6.0	6.1	6.7
04	6.1	5.9	5.9	5.8	5.6
06	6.6	6.1	6.1	5.8	6.5
10	6.2	6.5	6.7	6.0	6.2
Mean	6.2 ± 0.2[a]	6.3	6.2	5.9	6.0
Males					
22	6.3	6.3	6.2	6.2	6.0
23	6.6	6.2	6.1	6.0	6.0
24	6.0	6.2	6.0	6.0	5.9
27	6.3	6.2	6.0	6.0	5.7
28	6.1	6.7	6.6	6.6	6.6
Mean	6.3	6.3	6.2	6.2	6.0

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-13. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(gm/100 ml)		
Overall control mean	6.2	6.3[b**]	6.2	6.0	6.0[b**]
Treatment females					
03	6.5	5.6	-[c]	-	-
05	6.3	5.6	6.3	6.0	5.6
07	6.3	5.1	5.9	5.6	5.9
08	6.6	5.8	6.8	6.1	6.1
09	6.3	5.1	-[c]	-	-
Mean	6.4	5.4[de**]	6.3[e**]	5.9[d**]	5.9[d**]

[b]Overall control mean was significantly larger than the overall treatment mean at the same regimen.

[c]Dogs 03 and 09 of the treatment group died during refeeding I.

[d]Mean was significantly different from the control regimen mean for the same group.

[e]Mean was significantly different from the mean for the males of the treatment group at the same regimen.

**Difference was significant at $P<0.01$.

Table A-13. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(gm/100 ml)		
Males					
21	6.5	6.4	5.5	5.6	5.1
25	5.7	6.0	6.1	6.0	5.5
26	6.4	6.0	5.9	6.1	5.8
29	6.4	6.0	5.6	6.2	5.8
Mean	6.2	6.1	5.8^{d**}	6.0	5.6^{d**}
Overall treatment mean	6.3	5.8^{d**}	6.0^{d**}	5.9^{d**}	5.7^{d**}

Table A-14. Analysis of variance plan and observed mean squares for total plasma protein concentration in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	0.768
Sex	1	0.005
Sex · group	1	0.115
Dog (sex · group) error	15	0.161
Regimen	4	0.450**
Group · regimen	4	0.230**
Sex · regimen	4	0.270**
Sex · group · regimen	4	0.195**
Error	54	0.050
Total	88	

**Statistical significance is at $P<0.01$.

Table A-15. Total white blood cell counts in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	11,100	8,325	7,650	8,270	8,270
02	13,800	7,575	11,000	10,500	10,400
04	13,800	8,825	8,840	7,660	12,400
06	9,200	9,875	11,400	10,300	9,700
10	9,220	7,300	7,400	11,000	9,150
Mean	11,424 ± 1,547[a]	8,380[b**]	9,258[b*]	9,546	9,984
Males					
22	13,600	8,725	10,800	8,000	8,370
23	11,400	8,400	9,180	8,810	9,100
24	14,900	8,750	12,200	9,900	9,400
27	13,000	9,700	7,610	12,100	11,200
28	15,000	11,800	9,400	9,500	9,500

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at $P<0.01$.

*Difference was significant at $P<0.05$.

Table A-15. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Mean	13,580	9,475[b**]	9,838[b**]	9,662[b**]	9,514[b**]
Overall control mean	12,502	8,928[b**]	9,548[b**]	9,604[b**]	9,749[b**]
Treatment females					
03	13,200	6,575	-[c]	-	-
05	9,300	4,700	9,610	5,880	9,680
07	10,000	6,775	9,000	6,050	7,230
08	10,700	5,500	9,200	3,600	4,790
09	10,600	6,825	-[c]	-	-
Mean	10,760	6,075[b**]	9,270	5,177[b**]	7,233[b**]
Males					
21	14,200	6,225	11,200	6,400	7,640
25	13,100	5,425	15,900	5,800	12,800
26	12,800	4,450	9,310	7,000	10,400
29	11,400	6,100	8,900	5,600	7,540
Mean	12,785	5,550[b**]	11,238	6,110[b**]	9,506[b**]

[c]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-15. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Overall treatment mean	11,817	5,812[bd**]	10,446	5,761[bd**]	8,582[bd**]

[d]Mean was significantly different from the overall control mean for the same regimen.

Table A-16. Analysis of variance plan and observed mean squares for total white blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	59,249,505.86**
Sex	1	23,752,600.05*
Sex · group	1	2,718,619.41
Dog (sex · group) error	15	3,742,781.00
Regimen	4	67,396,989.65**
Group · regimen	4	15,176,490.87**
Sex · regimen	4	2,681,886.54
Sex · group · regimen	4	206,088.32
Error	54	2,393,881.73
Total	88	

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-17. Absolute counts of segmented neutrophils in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	7,992	6,327	4,820	6,120	5,376
02	11,040	5,075	6,490	7,770	6,760
04	9,660	5,648	6,188	4,826	9,800
06	5,980	5,826	7,182	6,798	6,596
10	6,085	4,745	3,848	7,480	6,130
Mean	8,151 ± 1,365[a]	5,524[b**]	5,706[b**]	6,599	6,932
Males					
22	10,472	6,194	7,560	5,200	6,444
23	9,690	5,628	5,967	6,078	6,370
24	12,069	5,862	8,662	6,633	6,204
27	8,060	6,596	5,631	8,228	6,160
28	10,050	8,850	6,110	5,795	6,745

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at $P<0.01$.

Table A-17. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Mean	10,068	6,626[b**]	6,786[b**]	6,387[b**]	6,385[b**]
Overall control mean	9,110	6,075[b**]	6,246[b**]	6,493[b**]	6,658[b**]
Treatment females					
03	7,075	2,928	-[c]	-	-
05	7,161	3,525	7,111	4,174	6,388
07	7,000	5,216	6,840	3,448	4,916
08	8,239	4,235	6,716	2,196	2,778
09	7,738	5,187	-[c]	-	-
Mean	7,443	4,218[b**]	6,889	3,273[b**]	4,694[b**]
Males					
21	10,792	3,548	9,744	3,968	5,654
25	8,646	3,689	12,084	2,900	10,368
26	8,960	2,314	6,144	4,130	7,384
29	8,322	3,660	5,963	2,184	4,826

[c]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-17. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Mean	9,180	3,303[b**]	8,484	3,296[b**]	7,058[b*]
Overall treatment mean	8,311	3,760[bd**]	7,800[d*]	3,286[bd**]	6,045[b**]

[d]Mean was significantly different from the overall control mean for the same regimen.

[*]Difference was significant at $P<0.05$.

Table A-18. Analysis of variance plan and observed mean squares for segmented neutrophil blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	27,594,196.00**
Sex	1	14,258,947.56*
Sex • group	1	461,133.79
Dog (sex • group) error	15	2,548,252.32
Regimen	4	42,292,693.65**
Group • regimen	4	13,493,212.10**
Sex • regimen	4	2,945,591.56
Sex • group • regimen	4	3,332,043.85
Error	54	1,863,491.11
Total	88	

**Statistical significance is at $P<0.01$.

*Statistical significance is at $P<0.05$.

Table A-19. Absolute counts of lymphocytes in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	1,554	2,737	2,142	1,489	1,902
02	1,932	1,439	2,750	1,680	3,120
04	2,346	2,383	1,768	1,840	1,985
06	2,116	2,469	2,394	2,266	1,940
10	1,659	2,044	2,590	2,530	2,379
Mean	1,921 ± 568[a]	2,214	2,329	1,961	2,265
Males					
22	2,176	1,832	2,376	1,440	1,506
23	684	1,848	2,111	1,762	2,275
24	1,788	2,275	2,806	2,574	1,880
27	4,030	2,231	1,446	2,783	3,584
28	2,850	2,124	1,786	2,850	1,900
Mean	2,306	2,062	2,007	2,282	2,229
Overall control mean	2,113	2,138	2,217	2,121	2,247

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-19. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Treatment females					
03	5,412	3,353	-[b]	-	-
05	1,116	1,081	1,442	1,470	2,807
07	2,300	1,152	1,800	2,480	1,952
08	749	880	1,564	1,224	1,437
09	1,908	1,160	-[b]	-	-
Mean	2,297	1,525	1,602	1,725	2,065
Males					
21	2,130	2,054	784	1,920	1,528
25	3,275	1,573	2,703	1,856	1,920
26	2,816	1,824	2,420	1,820	2,288
29	2,280	2,379	2,047	2,744	2,111
Mean	2,625	1,957	1,988	2,085	1,962
Overall treatment mean	2,461	1,741	1,823	1,931	2,006

[b]Dogs 03 and 09 died during the refeeding I regimen.

Table A-20. Analysis of variance plan and observed mean squares for lymphocyte blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	729,669.68
Sex	1	618,875.91
Sex • group	1	265,540.95
Dog (sex • group) error	15	1,472,564.75
Regimen	4	383,765.60
Group • regimen	4	360,794.50
Sex • regimen	4	396,083.42
Sex • group • regimen	4	307,953.00
Error	54	322,693.05
Total	88	

Table A-21. Absolute counts of band neutrophils in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	666	83	153	83	248
02	0	0	0	105	0
04	414	176	0	153	124
06	0	296	114	309	97
10	92	73	74	220	0
Mean	234 ± 160[a]	126	68	174	94
Males					
22	136	174	108	80	84
23	456	168	92	88	0
24	149	175	122	99	0
27	0	291	76	121	224
28	1,200	118	376	285	285
Mean	388	185	155	134	119
Overall control mean	311	155	111	154	106

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-21. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Treatment females					
03	0	66	-[b]	-	-
05	279	47	192	59	96
07	0	68	90	60	72
08	535	165	92	0	96
09	0	68	-[b]	-	-
Mean	163	83	125	40	88
Males					
21	568	186	112	128	229
25	131	0	159	0	128
26	0	44	279	70	0
29	114	0	89	112	226
Mean	203	58	160	78	146
Overall treatment mean	183	70	145	61	121

[b]Dogs 03 and 09 died during the refeeding I regimen.

Table A-22. Analysis of variance plan and observed mean squares for band neutrophil blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	61,718.78
Sex	1	39,988.78
Sex • group	1	4,190.00
Dog (sex • group) error	15	49,943.86
Regimen	4	74,580.99
Group • regimen	4	14,711.45
Sex • regimen	4	5,829.19
Sex • group • regimen	4	11,984.19
Error	54	25,633.89
Total	88	

Table A-23. Absolute counts of monocytes in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	888	166	382	248	414
02	552	152	220	210	0
04	1,104	88	354	383	248
06	644	395	684	0	485
10	738	219	518	110	92
Mean	785 ± 212[a]	204[b**]	432[b*]	190[b**]	248[b**]
Males					
22	680	87	216	480	251
23	570	84	550	616	364
24	894	175	122	297	658
27	260	97	304	484	560

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at P<0.01.

*Difference was significant at P<0.05.

Table A-23. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
28	450	472	564	190	0
Mean	571	183[b**]	351	413	367
Overall control mean	678	194[b**]	391[b**]	302[b**]	307[b**]
Treatment females					
03	396	263	-[c]	-	-
05	372	47	576	59	0
07	600	338	0	60	72
08	963	165	736	72	192
09	636	409	-[c]	-	-
Mean	593	244[b*]	437	64[b**]	88[b**]
Males					
21	0	124	560	384	76
25	393	54	477	290	128
26	1,024	267	186	910	208

[c]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-23. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
29	570	61	267	280	302
Mean	497	127[b*]	373	466	179[b*]
Overall treatment mean	545	186[b**]	400	294[b*]	140[b**]

Table A-24. Analysis of variance plan and observed mean squares for monocyte blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	98,492.59
Sex	1	12,350.99
Sex • group	1	7,540.34
Dog (sex • group) error	15	44,262.77
Regimen	4	532,961.58**
Group • regimen	4	28,371.41
Sex • regimen	4	141,847.70*
Sex • group • regimen	4	13,348.23
Error	54	44,876.84
Total	88	

**Statistical significance is at $P<0.01$.

*Statistical significance is at $P<0.05$.

Table A-25. Absolute counts of eosinophils in the blood of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
Control females					
01	0	250	153	331	331
02	276	909	1,540	735	520
04	276	530	530	459	248
06	460	889	1,026	927	582
10	645	365	370	660	549
Mean	332 ± 224[a]	589	724[b**]	622[b*]	446
Males					
22	136	436	540	800	84
23	0	672	459	264	91
24	0	262	488	297	658
27	650	485	152	484	672

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

[b]Mean was significantly different from the control regimen mean for the same group.

**Difference was significant at P<0.01.

*Difference was significant at P<0.05.

Table A-25. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
28	450	236	564	380	570
Mean	247	418	440	445	415
Overall control mean	289	503[b*]	582[b**]	534[b*]	430
Treatment females					
03	396	132	-[c]	-	-
05	372	0	288	118	387
07	100	0	270	0	216
08	214	55	92	108	287
09	318	0	-[c]	-	-
Mean	280	37	217	75	297
Males					
21	710	311	0	0	152
25	655	108	477	174	256
26	0	0	279	70	520

[c]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-25. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			(cells/cu mm)		
29	114	0	534	280	75
Mean	370	105	322	131	251
Overall treatment mean	325	71[bd**]	277[d**]	107[d**]	270

[d]Mean was significantly different from the overall control mean for the same regimen.

Table A-26. Analysis of variance plan and observed mean squares for eosinophil blood cell counts in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	1,446,373.57**
Sex	1	48,218.93
Sex · group	1	223,252.31
Dog (sex · group) error	15	99,090.66
Regimen	4	54,531.45
Group · regimen	4	175,740.30**
Sex · regimen	4	6,834.59
Sex · group · regimen	4	22,155.90
Error	54	49,991.92
Total	88	

**Statistical significance is at $P<0.01$.

Table A-27. Means values for systolic and diastolic blood pressure and heart rate for the control and treatment beagles on each day of measurement

Date (month-day)	Regimen	Control group n[a]	Control group Systolic pressure (mmHg)	Control group Diastolic pressure (mmHg)	Control group Heart rate (bpm)	Treatment group n[a]	Treatment group Systolic pressure (mmHg)	Treatment group Diastolic pressure (mmHg)	Treatment group Heart rate (bpm)
4-10	Control	7	166±12[b]	109±10[b]	93±19[b]	8	166	106	102
4-12	Control	7	170	114	97	8	170	106	118
4-14	Control	7	173	116	103	8	174	121	110
4-19	Control	7	167	111	89	8	173	115	130
4-21	Control	7	163	112	97	8	174	115	117
4-26	Control	7	169	116	94	8	170	111	116
4-28	Control	7	163	111	106	8	163	107	120
5-01	Control	7	178	121	95	8	171	111	122
5-10	Control	7	180	120	108	7	176	114	118
5-12	Control	7	178	121	107	8	175	112	114
5-17	Control	6	171	115	91	5	165	106	123
5-19	Starvation I	7	164	104	94	8	166	107	106
5-22	Starvation I	7	166	109	97	8	167	104	84

[a]n equals the number of dogs sampled.

[b]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-27. (Continued)

Date (month-day)	Regimen	Control group n	Control group Systolic pressure (mmHg)	Control group Diastolic pressure (mmHg)	Control group Heart rate (bpm)	Treatment group n	Treatment group Systolic pressure (mmHg)	Treatment group Diastolic pressure (mmHg)	Treatment group Heart rate (bpm)
5-25	Starvation I	7	175	113	111	8	169	103	76
5-30	Starvation I	7	184	125	90	8	162	109	71
6-02	Starvation I	7	162	106	90	8	150	94	59[c*]
6-06	Starvation I	7	169	108	86	8	155	104	58[c*]
6-09	Starvation I	7	163	103	93	8	147	97	54[c*]
6-13	Starvation I	7	170	107	103	8	143	92	51[c*]
6-16	Starvation I	7	183	117	86	8	147	105	66
6-19	Refeeding I	7	182	120	101	8	171	122	84
6-21	Refeeding I	7	187	119	89	8	181	124	118
6-23	Refeeding I	7	174	115	99	8	172	113	118
6-27	Refeeding I	7	173	108	106	7	166	105	111
6-29	Refeeding I	7	178	113	98	7	172	109	129
7-03	Refeeding I	7	177	113	108	7	161	106	129
7-05	Refeeding I	7	175	112	110	7	170	106	121

[c]Mean was significantly lower than the treatment group mean for the culture control regimen.

*Difference was significant at $P<0.05$.

Table A-27. (Continued)

Date (month-day)	Regimen	Control group n	Control group Systolic pressure (mmHg)	Control group Diastolic pressure (mmHg)	Control group Heart rate (bpm)	Treatment group n	Treatment group Systolic pressure (mmHg)	Treatment group Diastolic pressure (mmHg)	Treatment group Heart rate (bpm)
7-08	Starvation II	7	178	116	93	6	176	108	101
7-11	Starvation II	7	165	107	93	6	160	98	65
7-14	Starvation II	7	168	110	93	6	153	95	63
7-16	Starvation II	7	171	109	94	6	164	104	59
7-23	Starvation II	7	160	100	94	6	141	94	51[c*]
7-27	Refeeding II	7	176	114	92	6	170	114	93
7-29	Refeeding II	7	184	116	99	6	178	121	101
7-31	Refeeding II	7	180	119	86	6	166	110	118
8-02	Refeeding II	7	187	120	87	6	173	114	122
8-04	Refeeding II	7	188	120	96	6	178	116	125
8-08	Refeeding II	7	180	118	97	6	182	119	124
8-10	Refeeding II	7	178	118	101	6	171	112	125

Table A-28. Regimen means for systolic and diastolic blood pressure and heart rate of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
			Systolic blood pressure (mmHg)		
Control					
Females	170 (44)[a]	167 (36)	173 (28)	164 (20)	177 (28)
Males	173 (32)	176 (27)	185 (21)	175 (15)	188 (21)
Overall	171 (76)	170 (63)	178 (49)	168 (35)	182 (49)
Treatment					
Females	170 (54)	153 (45)	170 (31)[b]	159 (15)	180 (21)
Males	173 (30)	161 (27)	172 (21)	158 (15)	168 (21)
Overall	171 (84)	156 (72)	171 (52)	159 (30)	174 (42)
			Diastolic blood pressure (mmHg)		
Control					
Females	115	106	110	104	114
Males	115	116	120	114	123
Overall	115	110	114	109	118

[a]Numbers in parentheses represent the number of observations composing the mean.

[b]Two female treatment dogs (03 and 09) died during the refeeding I regimen.

Table A-28. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
Treatment					
Females	111	99	111[b]	102	120
Males	113	106	115	97	111
Overall	111	102	113	100	115
			Heart rate (bpm)		
Control					
Females	100	98	105	103	101
Males	96	90	98	81	86
Overall	98	94	102	93	94
Treatment					
Females	127	76[c**]	121[b]	78[c*]	126
Males	99	59	107	57	105
Overall	117	70[c**]	115	68[c**]	115

[c]Mean was significantly less than the control regimen mean for the same group.

**Difference was significant at P<0.01.

*Difference was significant at P<0.05.

Table A-29. Analysis of variance plan and observed mean squares for heart rate in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	785.63
Sex	1	32,711.45*
Sex · group	1	4,436.29
Dog (sex · group) error	11	5,071.57
Regimen	4	21,716.33[a]**
Group · regimen	4	14,738.00[a]**
Sex · regimen	4	271.06[a]
Sex · group · regimen	4	905.41[a]
Error	521	375.10[a]
Total	551	

[a]A more conservative estimate of significance was used in which the d.f. were divided by the treatment regimen d.f.

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-30. Analysis of variance plan and observed mean squares for systolic blood pressure in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	7,780.66
Sex	1	3,129.87
Sex · group	1	1,637.35
Dog (sex · group) error	11	7,125.54
Regimen	4	3,963.26[a]**
Group · regimen	4	1,129.42[a]**
Sex · regimen	4	221.86[a]
Sex · group · regimen	4	337.26[a]
Error	521	133.22[a]
Total	551	

[a]A more conservative estimate of significance was used in which the d.f. were divided by the treatment regimen d.f.

**Statistical significance is at $P<0.01$.

Table A-31. Analysis of variance plan and observed mean squares for diastolic blood pressure in dogs that were starved and refed a high carbohydrate diet

Source of variation	d.f.	Mean squares
Group	1	3,487.50
Sex	1	2,076.80
Sex · group	1	1,065.86
Dog (sex · group) error	11	3,513.43
Regimen	4	2,631.16[a]**
Group · regimen	4	299.37[a]
Sex · regimen	4	285.80[a]
Sex · group · regimen	4	291.73[a]
Error	521	109.67[a]
Total	551	

[a]A more conservative estimate of significance was used in which the d.f. were divided by the treatment regimen d.f.

**Statistical significance is at $P<0.01$.

Table A-32. Coordinates of the half-area QRS vector in the transverse plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
					(mv)					
Control females										
01	0.0	-1.2	.6	-0.1	0.8	-0.6	1.3	-0.4	1.0	0.3
02	2.0	-1.2	2.2	-1.9	1.6	-2.3	2.3	0.6	2.8	-0.6
04	3.1	-0.6	3.2	-0.3	2.6	-2.7	3.1	-1.1	2.7	-1.3
06	3.4	0.3	2.6	-0.6	5.1	0.4	3.1	0.4	3.2	0.2
10	1.2	-1.5	1.6	-1.9	1.8	-1.3	1.9	-1.9	1.3	-1.5
Mean	1.9 ±6.1[a]	-0.8 ±7.2[a]	2.0	-1.0	2.4	-1.3	2.4	0.5	2.2	-0.6
Males										
22	1.0	-1.3	1.0	-1.6	1.9	-1.0	2.5	-1.5	2.2	-1.3
23	1.9	0.1	2.1	-1.0	1.1	-0.4	1.2	-0.4	2.2	-0.3
24	1.1	-1.3	1.9	-2.6	2.4	-1.5	2.5	-1.2	2.4	-1.3
27	2.4	-0.9	2.7	1.3	3.0	0.6	3.5	0.0	3.9	0.5
28	3.3	-1.0	1.7	-1.0	3.5	0.0	3.9	0.7	3.9	0.5
Mean	1.9	-0.9	1.9	-1.0	2.4	-0.4	2.7	0.1	2.7	-.7

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-32. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
					(mv)					
Overall control mean	1.9	-0.9	2.0	-2.0	2.4	-0.9	2.5	-0.2	2.5	-0.6
Treatment females										
03	2.9	-1.5	4.1	-1.6	-[b]	-	-	-	-	-
05	0.4	-0.8	1.1	-3.0	0.6	-2.1	2.2	0.0	1.2	-0.2
07	3.2	-0.3	1.2	-1.8	2.0	-0.8	2.4	-0.4	2.3	-0.7
08	1.6	-0.5	1.1	-2.0	2.1	-1.0	2.7	-1.4	1.8	-1.2
09	1.6	0.0	0.2	0.2	-[b]	-	-	-	-	-
Mean	1.9	-.6	1.5	-1.7	1.6	-1.3	2.4	-.6	1.8	-.7
Males										
21	1.2	-.7	0.9	-1.3	-0.7	2.0	1.6	-0.9	0.2	0.0
25	2.0	-1.0	1.5	-1.7	1.9	0.7	2.8	0.3	3.7	-0.9
26	1.3	-0.9	1.8	-2.1	1.8	-1.7	2.0	-1.2	1.7	-1.1
29	2.6	-1.1	2.3	-1.4	2.6	-1.9	4.1	-0.9	2.4	-1.1
Mean	1.8	-0.9	1.6	-1.6	1.4	-0.6	2.6	-0.7	2.0	-0.9
Overall treatment mean	1.9	-0.7	1.6	-1.6	1.5	-0.9	2.5	-0.6	1.9	-0.8

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-33. Analysis of variance plan and observed mean squares for the X-lead coordinate of the half-area QRS vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	176.671
Sex	1	0.045
Sex • group	1	46.305
Dog (sex • group) error	15	392.550
Regimen	4	175.206**
Sex • regimen	4	13.871
Group • regimen	4	46.020
Sex • group • regimen	4	12.052
Error	54	36.934
Total	88	

**Statistical significance is at $P<0.01$.

Table A-34. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the half-area QRS vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	17.575
Sex	1	0.901
Sex • group	1	31.556
Dog (sex • group) error	15	194.631
Regimen	4	169.289*
Sex • regimen	4	58.033
Group • regimen	4	47.370
Sex • group • regimen	4	2.831
Error	54	52.358
Total	88	

*Statistical significance is at $P<0.05$.

Table A-35. Coordinates of the half-area QRS vector in the left sagittal plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
Control females										
01	0.5	0.9	-0.2	1.8	0.4	1.5	0.1	1.1	0.1	1.2
02	1.6	0.2	1.4	-0.5	1.6	-0.8	1.0	1.1	1.5	0.3
04	2.6	-0.3	1.8	0.0	2.0	-1.5	1.7	-0.7	1.7	-0.7
06	2.2	0.2	1.9	-0.2	2.4	0.1	2.2	0.6	2.1	-0.1
10	2.5	-1.3	2.1	-1.8	2.0	-1.6	2.1	-1.9	2.4	-1.1
Mean	1.9 ±2.6[a]	-0.1 ±4.5[a]	1.4	-0.1	1.7	-0.4	1.4	0.0	1.6	-0.1
Males										
22	2.8	-0.8	2.8	-0.9	2.9	-0.6	2.9	-0.9	2.6	-1.1
23	0.9	1.0	0.9	0.2	0.7	0.5	0.6	0.4	0.6	0.6
24	1.8	-0.3	1.7	-1.2	1.7	-0.5	1.8	-0.6	1.8	-0.8
27	2.7	-0.9	1.7	0.4	1.2	1.2	2.8	-0.2	2.5	-0.6
28	2.3	0.0	2.2	-0.4	2.4	0.1	1.5	0.7	2.3	0.7
Mean	2.1	-0.2	1.8	-0.4	1.8	0.1	1.9	-0.1	2.0	-0.2

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-35. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
Overall control mean	2.0	-0.1	1.6	-0.2	1.8	-0.1	1.7	0.0	1.8	-0.2
Treatment females										
03	2.5	-1.4	2.2	-1.1	-[b]	-	-	-	-	-
05	1.6	0.0	0.7	0.0	0.7	0.3	0.8	0.9	0.5	0.6
07	2.2	0.5	1.3	-0.5	1.4	-0.1	1.2	0.1	1.0	0.1
08	1.6	0.3	0.9	-0.9	0.5	0.2	1.0	-0.6	0.5	0.0
09	1.5	1.2	.6	1.1	-[b]	-	-	-	-	-
Mean	1.9	0.1	1.1	-0.3	0.9	0.1	1.0	0.1	0.7	0.3
Males										
21	2.4	-0.6	2.1	-0.8	1.8	-0.2	2.1	-0.6	1.4	0.4
25	2.2	-0.2	1.8	-0.9	1.2	-0.2	1.6	0.4	1.3	0.1
26	2.4	-0.7	2.0	-1.4	1.8	-1.1	1.8	-0.6	1.2	-0.7
29	2.1	-0.4	1.8	-0.4	1.8	-1.1	2.0	-0.5	1.6	-0.3
Mean	2.3	-0.5	1.9	-0.9	1.6	-0.6	1.9	-0.3	1.4	-0.1
Overall treatment mean	2.0	-0.2	1.5	-0.5	1.3	-0.3	1.5	-0.1	1.1	0.0

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-36. Analysis of variance plan and observed mean squares for the Y-lead coordinate of the half-area QRS vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	134.858
Sex	1	442.909
Sex • group	1	44.905
Dog (sex • group) error	15	195.777
Regimen	4	90.781**
Sex • regimen	4	7.966
Group • regimen	4	38.164**
Sex • group • regimen	4	2.258
Error	54	6.564
Total	88	

**Statistical significance is at $P<0.01$.

Table A-37. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the half-area QRS vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	6.789
Sex	1	168.145
Sex · group	1	141.230
Dog (sex · group) error	15	249.947
Regimen	4	37.131
Sex · regimen	4	5.902
Group · regimen	4	17.237
Sex · group · regimen	4	22.218
Error	54	20.388
Total	88	

Table A-38. Coordinates of the maximum QRS vector in the frontal plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
					(mv)					
Control females										
01	-0.2	-1.7	1.8	1.3	0.1	-1.7	1.5	1.4	1.5	1.6
02	2.1	1.5	2.1	1.5	1.9	1.6	2.4	1.0	2.8	1.5
04	3.1	2.7	3.2	2.0	2.9	2.1	3.1	1.9	2.9	1.8
06	3.4	2.2	2.7	1.9	5.2	2.5	3.1	2.2	3.3	2.1
10	1.4	2.5	2.1	2.1	2.2	1.9	2.3	2.2	1.5	2.6
Mean	2.0 ±.7[a]	1.4 ±.5[a]	2.4	1.8	2.5	1.3	2.5	1.7	2.4	1.9
Males										
22	0.5	3.6	1.7	2.7	2.3	3.0	3.1	2.8	3.0	2.9
23	1.7	1.1	2.2	1.6	1.1	1.3	1.2	1.2	2.3	1.2
24	1.1	1.7	1.9	1.7	2.1	1.5	2.9	1.8	2.3	1.9
27	3.1	2.9	2.8	1.4	2.8	2.1	3.5	2.8	3.5	2.6
28	3.2	2.2	1.9	2.2	3.5	2.5	4.1	1.6	4.1	2.2
Mean	1.9	2.3	2.2	1.9	2.4	2.1	3.0	2.0	3.0	2.2

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-38. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
					(mv)					
Overall control mean	1.9	1.9	2.3	1.8	2.4	1.7	2.7	1.9	2.7	2.0
Treatment females										
03	2.9	2.7	4.2	2.3	-[b]	-	-	-	-	-
05	-0.3	2.2	1.1	1.7	.4	1.7	2.1	1.3	1.2	1.4
07	3.2	1.9	1.2	.9	1.9	1.4	2.3	1.2	2.4	1.1
08	2.0	1.7	1.3	1.3	2.0	0.9	3.2	1.3	1.7	1.0
09	1.3	1.5	-.1	1.1	-[b]	-	-	-	-	-
Mean	1.8	2.0	1.5	1.5	1.5	1.3	2.5	1.2	1.8	1.1
Males										
21	1.3	2.5	0.8	1.9	1.0	1.6	2.0	2.1	0.2	1.4
25	2.1	2.2	0.9	2.2	1.9	1.3	2.8	1.6	3.7	1.5
26	1.4	2.3	2.0	1.9	1.8	1.9	2.0	2.0	1.7	1.4
29	2.6	2.2	2.4	2.1	2.6	1.9	4.1	2.0	2.4	1.8
Mean	1.8	2.3	1.5	2.1	1.8	1.7	2.7	1.9	2.0	1.5

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-38. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
					(mv)					
Overall treatment mean	1.8	2.1	1.5[c**]	1.7	1.7	1.5	2.6	1.6	1.9	1.4

[c]Mean was significantly different from the mean for the starvation II regimen of the same group.

**Difference was significant at $P<0.01$.

Table A-39. Analysis of variance plan and observed mean squares for the X-lead coordinate of the maximum QRS vector in the frontal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	3.543
Sex	1	.101
Sex · group	1	.105
Dog (sex · group) error	15	3.580
Regimen	4	2.132**
Sex · regimen	4	.118
Group · regimen	4	.575
Sex · group · regimen	4	.189
Error	54	.461
Total	88	

**Statistical significance is at $P<0.01$.

Table A-40. Analysis of variance plan and observed mean squares for the Y-lead coordinate of the maximum QRS vector in the frontal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	.507
Sex	1	3.418
Sex · group	1	.029
Dog (sex · group) error	15	1.740
Regimen	4	.339
Sex · regimen	4	.064
Group · regimen	4	.465
Sex · group · regimen	4	.228
Error	54	.276
Total	88	

Table A-41. Coordinates of the maximum QRS vector in the transverse plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
					(mv)					
Control females										
01	0.8	-2.8	1.8	-2.2	-0.1	-0.3	1.4	-1.9	1.6	-2.3
02	2.2	-1.0	2.0	-2.4	1.8	-2.2	2.4	0.5	2.8	-.7
04	2.9	2.5	3.2	-0.7	2.7	-2.6	3.1	-1.1	2.9	-1.1
06	3.5	0.5	2.7	-0.2	5.2	0.9	3.1	0.2	3.2	0.7
10	1.4	-1.5	2.0	-1.9	1.7	-1.1	2.3	-1.9	1.5	-1.3
Mean	2.0 ±.8[a]	-.4 ±.9[a]	2.3	-1.5	2.3	-1.6	2.4	-0.8	2.4	-0.9
Males										
22	0.2	-1.6	1.7	-1.4	2.3	-0.7	3.2	-1.2	3.1	-0.8
23	1.3	1.5	1.7	-1.9	0.7	-1.7	0.8	-1.1	2.3	-0.9
24	0.5	-2.1	1.6	-2.5	2.1	-2.2	2.9	-0.5	2.0	-2.2
27	3.0	-0.3	2.8	1.1	2.9	1.3	3.6	0.2	3.6	-0.4
28	3.3	-0.8	0.6	-2.3	3.5	0.2	4.2	0.5	3.9	0.0
Mean	1.6	-0.7	1.7	-1.4	2.3	-0.6	2.9	-0.4	3.0	-0.9

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-41. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
					(mv)					
Overall control mean	1.8	-0.6	2.0	-1.5	2.3	-1.1	2.7	-0.6	2.7	-0.9
Treatment females										
03	2.9	-1.5	4.1	-1.5	-[b]	-	-	-	-	-
05	-0.7	-2.3	1.0	-3.0	0.3	-3.0	0.6	2.3	0.7	-1.5
07	3.4	-0.1	1.3	-1.8	2.0	-0.9	2.3	-0.5	2.3	-0.8
08	-0.3	-2.1	1.2	-2.1	2.0	-0.9	3.1	-1.1	1.8	-1.2
09	-0.2	1.7	-0.8	2.6	-[b]	-	-	-	-	-
Mean	1.0	-0.8	1.4	-1.2	1.4	-1.6	2.0	0.2	1.6	-1.2
Males										
21	0.6	-2.1	0.8	-3.4	1.2	-2.5	1.9	-2.2	0.4	-1.7
25	2.0	-0.9	1.5	-1.7	1.9	-0.7	2.8	0.2	3.7	-1.0
26	0.4	-1.9	1.7	-2.2	1.5	-2.3	1.9	-1.3	1.1	-1.9
29	2.2	-1.9	1.8	-2.8	2.3	-2.4	4.1	-1.1	1.6	-2.3
Mean	1.3	-1.7	1.5	-2.5	1.7	-2.0	2.7	-1.1	1.7	-1.7

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-41. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
					(mv)					
Overall treatment mean	1.1	-1.2	1.4	-1.8	1.6	-1.8	2.4[c**]	-0.5	1.6	-1.5

[c]Mean was significantly different from the mean for the control regimen of the same group.

**Difference was significant at $P<0.01$.

Table A-42. Analysis of variance plan and observed mean squares for the X-lead coordinate of the maximum QRS vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	7.569
Sex	1	.103
Sex · group	1	.065
Dog (sex · group) error	15	4.194
Regimen	4	2.726**
Sex · regimen	4	.256
Group · regimen	4	.230
Sex · group · regimen	4	.359
Error	54	.619
Total	88	

**Statistical significance is at P<0.01.

Table A-43. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the maximum QRS vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	0.513
Sex	1	6.474
Sex · group	1	13.660
Dog (sex · group) error	15	4.645
Regimen	4	4.070**
Sex · regimen	4	0.342
Group · regimen	4	1.079
Sex · group · regimen	4	0.599
Error	54	0.883
Total	88	

**Statistical significance is at $P<0.01$.

Table A-44. Coordinates of the maximum QRS vector in the left sagittal plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
					(mv)					
Control females										
01	1.0	-2.6	1.2	-2.3	1.4	-3.0	1.1	-1.9	-1.7	1.9
02	0.8	-1.8	1.2	-2.5	1.3	-2.4	0.3	1.8	0.8	-1.8
04	2.0	-3.0	1.7	-2.2	2.0	-2.9	1.8	-2.4	1.8	-2.1
06	2.2	1.5	1.8	-1.5	2.0	1.6	1.9	2.3	2.0	1.7
10	2.3	-1.3	2.1	-1.8	2.0	-1.6	2.1	-1.9	2.5	-1.0
Mean	1.7 ±.8[a]	-1.4 ±1.2[a]	1.6	-2.1	1.7	-1.6	1.4	-1.4	1.1	-0.3
Males										
22	3.7	-0.3	2.7	-0.9	3.2	0.1	3.1	-0.7	3.2	-0.5
23	1.1	-1.9	1.4	-1.9	1.1	-1.7	1.1	-1.1	1.1	-1.4
24	1.1	-2.1	1.6	-2.7	1.1	-2.2	1.3	-2.3	1.4	-2.4
27	2.2	-2.7	1.5	1.9	1.8	1.7	2.3	-1.9	2.1	-2.3
28	2.3	1.2	1.9	-2.0	2.2	-1.9	1.6	1.9	2.0	1.3
Mean	2.1	-1.1	1.8	-1.1	1.9	-0.8	1.9	-0.8	1.9	-1.1

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-44. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
					(mv)					
Overall control mean	1.9	-1.3	1.7	-1.6	1.8	-1.2	1.7	-0.6	1.5	-0.7
Treatment females										
03	2.5	-2.3	2.2	-1.9	-[b]	-	-	-	-	-
05	1.9	-2.3	1.7	-2.9	1.8	-2.9	-0.6	2.4	1.3	-1.5
07	2.3	1.0	1.1	-1.8	1.4	-1.3	1.3	-1.2	1.0	-1.2
08	1.2	-1.8	1.4	-2.1	0.9	-1.5	1.3	-1.5	1.0	-1.6
09	1.6	1.6	0.5	2.2	-[b]	-	-	-	-	-
Mean	1.9	-0.8	1.4	-1.3	1.4	-1.9	0.7	-0.1	1.1	-1.4
Males										
21	2.2	-1.3	1.8	-3.4	1.5	-2.5	2.0	-2.1	0.9	-1.6
25	1.5	-1.7	1.6	-1.8	1.3	-1.3	1.6	-0.8	1.4	-1.9
26	2.2	-1.8	1.7	-2.2	1.8	-2.4	1.8	-1.7	1.3	-1.8
29	2.1	-2.2	2.2	-2.8	2.0	-2.8	2.2	-2.8	1.8	-2.5
Mean	2.0	-1.7	1.8	-2.5	1.6	-2.2	1.9	-1.9	1.3	-1.9
Overall treatment mean	1.9	-1.2	1.6	-1.8	1.5	-2.1	1.4	-1.1	1.2	-1.7

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-45. Analysis of variance plan and observed mean squares for the Y-lead coordinate of the maximum QRS vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	0.011
Sex	1	7.637
Sex · group	1	0.099
Dog (sex · group) error	15	1.817
Regimen	4	0.608
Sex · regimen	4	2.021*
Group · regimen	4	0.674
Sex · group · regimen	4	0.664
Error	54	0.723
Total	88	

*Statistical significance is at $P<0.05$.

Table A-46. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the maximum QRS vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	2.805
Sex	1	7.871
Sex · group	1	12.800
Dog (sex · group) error	15	5.384
Regimen	4	2.312
Sex · regimen	4	2.018
Group · regimen	4	0.308
Sex · group · regimen	4	0.850
Error	54	1.565
Total	88	

Table A-47. Direction[a] of QRS loop inscription in the frontal plane of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
Control females					
01	8	8	8	8	8
02	CW	CW	8	CW	CW
04	CCW	8	CCW	CCW	CCW
06	8	8	8	8	8
10	8	CCW	CCW	8	8
Males					
22	CW	8	CW	8	8
23	CCW	8	CCW	CCW	8
24	CW	CW	CW	CCW	CW
27	CCW	CCW	8	8	8
28	8	8	8	8	8
Treatment females					
03	CCW	CCW	-[b]	-	-

[a]8 = figure eight; CW = clockwise; CCW = counterclockwise.

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-47. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
05	CCW	8	CCW	CCW	CCW
07	8	CW	CW	8	8
08	CCW	CCW	8	8	CCW
09	8	8	-[b]	-	-
Males					
21	8	CW	CW	CW	CW
25	8	CW	CCW	CCW	CCW
26	CCW	CW	CW	8	8
29	8	8	CCW	8	CCW

Table A-48. Direction[a] of QRS loop inscription in the transverse plane of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
Control females					
01	8	CCW	8	CCW	CCW
02	CCW	CCW	CCW	CCW	CCW
04	CCW	CCW	CCW	CCW	CCW
06	CCW	CCW	CCW	CCW	CCW
10	8	8	CCW	8	8
Males					
22	8	8	CCW	CCW	CCW
23	CCW	CCW	CCW	CCW	CCW
24	8	8	CCW	CCW	CCW
27	CCW	CCW	CCW	CCW	CCW
28	CCW	CCW	CCW	CCW	CCW
Treatment females					
03	CCW	CCW	-[b]	-	-

[a]8 = figure eight; CW = clockwise; CCW = counterclockwise.

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-48. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
05	CCW	8	CCW	CCW	CCW
07	CCW	8	CCW	CCW	CCW
08	CCW	CCW	CCW	CCW	CCW
09	8	8	-[b]	-	-
Males					
21	CCW	CCW	CCW	CCW	CCW
25	CCW	8	CCW	CCW	CCW
26	CCW	8	CCW	CCW	CCW
29	CCW	CCW	CCW	CCW	CCW

Table A-49. Direction[a] of QRS loop inscription in the left sagittal plane of dogs

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
Control females					
01	CCW	CCW	CCW	CCW	CCW
02	CCW	CCW	CCW	CCW	CCW
04	CCW	CCW	CCW	CCW	CCW
06	CCW	CCW	CCW	CCW	CCW
10	CCW	CCW	CCW	CCW	CCW
Males					
22	CCW	CCW	CCW	CCW	CCW
23	CCW	CCW	CCW	CCW	CCW
24	CCW	CCW	CCW	CCW	CCW
27	CCW	CCW	CCW	CCW	CCW
28	CCW	CCW	CCW	CCW	CCW
Treatment females					
03	CCW	CCW	-[b]	-	-

[a]8 = figure eight; CW = clockwise; CCW = counterclockwise.

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-49. (Continued)

Group	Control	Starvation I	Refeeding I	Starvation II	Refeeding II
05	CCW	CCW	CCW	CCW	CCW
07	CCW	CCW	CCW	CCW	CCW
08	CCW	CCW	CCW	CCW	CCW
09	CCW	CCW	-[b]	-	-
Males					
21	CCW	CCW	CCW	CCW	CCW
25	CCW	CCW	CCW	CCW	CCW
26	CCW	CCW	CCW	CCW	CCW
29	CCW	CCW	CCW	CCW	CCW

Table A-50. Coordinates of the maximum T-wave vector in the frontal plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
Control females										
01	.866	-.500	.574	.819	-.866	.500	.105	.995	.469	.883
02	.998	-.070	.766	.643	-.951	-.309	.559	.829	.559	.829
04	-.875	-.485	-.990	.139	-.970	-.242	-.995	.105	-.990	-.139
06	.891	.454	.669	.743	-.799	-.602	-.875	-.485	-.669	-.743
10	-.017	1.000	.809	.588	.766	.643	.629	.777	.052	.999
Mean	.4 ±.5[a]	.1 ±.5[a]	.4	.6	-.6	0.0	.1	.4	-.1	.4
Males										
22	.242	.970	-.999	.035	-1.000	0.000	-.965	.276	-.616	.788
23	.326	.946	.682	.731	.292	.956	.259	.966	.559	.829
24	.515	.857	.242	.970	.530	.848	-.848	.530	.342	.940
27	-.985	.174	-.985	.174	-.629	.777	-.961	-.276	-.174	.985
28	-.906	-.423	-.391	.921	-.961	.276	-.966	.259	-.995	-.105
Mean	-.2	.5	-.3	.6	-.4	.6	-.7	.4	-.2	.7

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-50. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
Overall control mean	.1	.3	0.0	.6	-.5	.3	-.4	.4	-.1	.5
Treatment females										
03	-.920	-.391	-.934	-.358	-[b]	-	-	-	-	-
05	-.999	.035	.259	.966	-.829	.559	-.951	.309	-.052	.999
07	-.848	-.530	.309	-.951	-.469	.883	-.914	-.407	-.995	.105
08	-.643	.766	-.616	-.788	-.208	.978	-.755	-.656	.225	.974
09	.914	.407	.407	-.914	-[b]	-	-	-	-	-
Mean	-.5	.1	-.1	-.4	-.5	.8	-.9	-.3	-.3	.7
Males										
21	.707	.707	-.225	-.974	.819	-.574	-.669	-.743	.899	-.438
25	-.819	-.574	-.342	-.940	-.961	.276	-.695	-.719	-.961	.276
26	0.0	1.000	.988	-.156	-.156	.988	-.914	-.407	.423	.906
29	-.438	.899	.669	.743	-.070	.998	.788	.616	.242	.970
Mean	-.1	.5	.3	-.3	-.1	.4	-.4	-.3	.2	.4

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-50. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Y	X	Y	X	Y	X	Y	X	Y
Overall treatment mean	-.3	.3	.1	-.4[c**]	-.3	.6	-.6	-.3[d**]	0.0	.5

[c]Mean was significantly different from the means for the control, refeeding I, and refeeding II regimens of the same group and the overall control mean for the same regimen.

[d]Mean was significantly different from the means for the refeeding I and refeeding II regimens of the same group and the overall control mean for the same regimen.

**Difference was significant at $P<0.01$.

Table A-51. Analysis of variance plan and observed mean squares for the X-lead coordinate of the maximum T-wave vector in the frontal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	.025
Sex	1	.003
Sex · group	1	2.148
Dog (sex · group) error	15	1.331
Regimen	4	0.798*
Sex · regimen	4	0.115
Group · regimen	4	0.291
Sex · group · regimen	4	0.181
Error	54	0.254
Total	88	

*Statistical significance is at $P<0.05$.

Table A-52. Analysis of variance plan and observed mean squares for the Y-lead coordinate of the maximum T-wave vector in the frontal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	1.516
Sex	1	0.322
Sex · group	1	0.236
Dog (sex · group) error	15	0.831
Regimen	4	0.706*
Sex · regimen	4	0.150
Group · regimen	4	1.195**
Sex · group · regimen	4	0.196
Error	54	0.218
Total	88	

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-53. Coordinates of the maximum T-wave vector in the transverse plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
Control females										
01	.070	-.998	.530	-.848	-.643	-.766	.191	-.982	.500	-.866
02	.242	-.970	.191	-.982	-.358	-.934	.156	-.988	0.000	-1.000
04	-.707	-.707	-.391	-.921	-.956	-.292	-.602	-.799	-.927	-.375
06	.174	-.985	-.017	-1.000	-.174	-.985	-.242	-.970	-.139	-.990
10	-.156	-.988	.500	-.866	.588	-.809	.559	-.829	-.017	-1.000
Mean	-.1 ±.4[a]	-.9 ±.4[a]	.2	-.9	-.3	-.8	0.0	-.9	-.1	-.8
Males										
22	-.087	-.996	-.500	-.866	-.545	-.839	-.358	-.934	-.087	-.996
23	.105	-.995	.485	-.875	.259	-.966	.574	-.819	.616	-.788
24	.259	-.966	.087	-.996	.391	-.920	-.485	-.875	.174	-.985
27	-.407	-.914	-.454	-.891	-.342	-.940	-.500	-.866	-.122	-.993
28	-.358	-.934	-.105	-.995	-.530	-.838	-.407	-.914	-.883	-.469
Mean	-.1	-1.0	-.1	-.9	-.2	-.9	-.2	-.9	-.1	-.8

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-53. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
Overall control mean	-.1	-.9	0.0	-.9	-.2	-.8	-.1	-.9	-.1	-.8
Treatment females										
03	-.777	-.629	-.682	.731	-[b]	-	-	-	-	-
05	-.588	-.809	.225	-.974	-.883	-.469	-.788	-.616	-.515	-.857
07	-.438	-.899	.883	.469	-.530	-.848	.407	-.914	-.629	-.777
08	-.326	-.946	-.342	.940	-.122	-.993	-.966	.259	-.682	-.731
09	.407	-.914	.985	.174	-[b]	-	-	-	-	-
Mean	-.3	-.8	.2	.3	-.5	-.8	-.4	-.4	-.6	-.8
Males										
21	.438	-.899	-.156	.988	.242	-.970	-.777	.629	.208	-.978
25	-.052	-.999	-.454	.891	-.743	-.669	-.990	-.139	-.423	-.906
26	0.000	-1.000	.616	.788	-.156	-.988	-.956	.292	.788	-.616
29	-.208	-.978	.616	-.788	-.070	-.998	.695	-.719	.052	-.999
Mean	0.0	-1.0	.2	.5	-.2	-.9	-.5	0.0	.2	-.9

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-53. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	X	Z	X	Z	X	Z	X	Z	X	Z
Overall treatment mean	-.2	-.9	.2[c**]	.4	-.3	-.8	-.5[d**]	-.2[e**]	-.2	-.8

[c]Mean was significantly different from the means for the control, refeeding I, and refeeding II regimens of the same group and the overall control mean for the same regimen.

[d]Mean was significantly different from the mean for the starvation I regimen of the same group and the overall control mean for the same regimen.

[e]Mean was significantly different from the means for the control, starvation I, refeeding I, and refeeding II regimens of the same group.

**Difference was significant at $P<0.01$.

Table A-54. Analysis of variance plan and observed mean squares for the X-lead coordinate of the maximum T-wave vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	0.157
Sex	1	0.152
Sex • group	1	0.447
Dog (sex • group) error	15	0.660
Regimen	4	0.422*
Sex • regimen	4	0.279
Group • regimen	4	0.120
Sex • group • regimen	4	0.059
Error	54	0.128
Total	88	

*Statistical significance is at $P<0.05$.

Table A-55. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the maximum T-wave vector in the transverse plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	2.967**
Sex	1	0.013
Sex · group	1	0.001
Dog (sex · group) error	15	0.210
Regimen	4	1.189**
Sex · regimen	4	0.105
Group · regimen	4	1.554**
Sex · group · regimen	4	0.113
Error	54	0.122
Total	88	

**Statistical significance is at $P<0.01$.

Table A-56. Coordinates of the maximum T-wave vector in the left sagittal plane of dogs

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
Control females										
01	.139	-.990	.695	-.719	.423	-.906	.883	-.469	.719	-.695
02	0.035	-.999	.070	-.998	-.035	-.999	.309	-.951	.139	-.990
04	-.500	-.866	.139	-.991	-.656	-.755	.087	-.996	-.326	-.946
06	-.156	-.988	.122	-.993	-.191	-.982	-.070	-.998	-.139	-.990
10	.454	-.891	.342	-.940	.342	-.940	.602	-.800	.500	-.866
Mean	0.0 ±.3[a]	-.9 ±.3[a]	.3	-.9	0.0	-.9	.4	-.8	.2	-.9
Males										
22	.156	-.988	.156	-.988	-.017	-1.000	.070	-.998	.309	-.951
23	.438	-.899	.515	-.857	.656	-.755	.875	-.485	.643	-.766
24	.375	-.927	.423	-.906	.602	-.799	.326	-.946	-.469	-.883
27	.105	-.995	.122	-.993	.276	-.961	-.156	-.988	.105	-.995
28	-.208	-.978	.156	-.988	.122	-.993	.052	-.999	-.174	-.985
Mean	.2	-1.0	.3	-.9	.3	-.9	.2	-.9	.1	-.9

[a]Standard deviation, derived from the error mean squares of the analysis of variance.

Table A-56. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
Overall control mean	.1	-1.0	.3	-.9	.2	-.9	.3	-.9	.1	-.9
Treatment females										
03	-.276	-.961	-.500	.866	-[b]	-	-	-	-	-
05	.052	-.999	.875	-.485	.719	-.695	.375	-.927	.695	-.719
07	-.342	-.940	-.940	.342	.139	-.990	-.017	-1.000	.242	-.970
08	.309	-.951	-.602	.800	.500	-.866	-.883	.469	.695	-.719
09	.174	-.985	-.995	.105	-[b]	-	-	-	-	-
Mean	0.0	-1.0	-.4	.3	.5	-.9	-.2	-.5	.5	-.8
Males										
21	.454	-.891	-.616	.788	.017	-1.000	-.829	.559	-.259	.966
25	0.000	-1.000	-.839	.545	.375	-.927	-.998	.070	.087	-.996
26	-.839	.545	-.208	.978	.530	-.848	-.777	.629	.990	-.139
29	.342	-.940	.643	-.766	.629	-.777	.629	-.777	.423	-.906
Mean	0.0	-.6	-.3	.4	.4	-.9	-.5	.1	.3	-.3

[b]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

Table A-56. (Continued)

Group	Control		Starvation I		Refeeding I		Starvation II		Refeeding II	
	Y	Z	Y	Z	Y	Z	Y	Z	Y	Z
Overall treatment mean	0.0	-.8	-.4[c**]	.4[d**]	.4	-.9	-.4[c**]	-.1[c**]	.4	-.5

[c]Mean was significantly different from the means for the refeeding I and refeeding II regimens of the same group and the overall control mean for the same regimen.

[d]Mean was significantly different from the means for the control, refeeding I, starvation II, and refeeding II of the same group and the overall control mean for the same regimen.

[e]Mean was significantly different from the means for the control and refeeding I regimens of the same group and the overall control mean for the same regimen.

**Difference was significant at $P<0.01$.

Table A-57. Analysis of variance plan and observed mean squares for the Y-lead coordinate of the maximum T-wave vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	.460
Sex	1	.064
Sex • group	1	.001
Dog (sex • group) error	15	0.503
Regimen	4	0.361*
Sex • regimen	4	0.150
Group • regimen	4	0.898**
Sex • group • regimen	4	0.038
Error	54	0.100
Total	88	

**Statistical significance is at P<0.01.

*Statistical significance is at P<0.05.

Table A-58. Analysis of variance plan and observed mean squares for the Z-lead coordinate of the maximum T-wave vector in the left sagittal plane in dogs that were starved and refed a high carbohydrate diet

Source of variance	d.f.	Mean squares
Group	1	4.648**
Sex	1	0.185
Sex · group	1	0.244
Dog (sex · group) error	15	0.266
Regimen	4	1.318**
Sex · regimen	4	0.075
Group · regimen	4	1.261**
Sex · group · regimen	4	0.094
Error	54	0.093
Total	88	

**Statistical significance is at $P<0.01$.

Table A-59. Circumference-tension data from the initial femoral artery segments of the control group female beagle dogs

Tension	Control females					
	01	02	04	06	10	Mean
	Circumference (cm)					
1.25	.6768	.7354	.7291	.8331	.8331	.7261±.0687[a]
2.50	.6998	.7702	.7500	.7011	.8624	.7567±.0666
3.75	.7249	.7939	.7696	.7165	.8785	.7767±.0652
5.00	.7390	.8079	.7933	.7381	.8932	.7943±.0636
6.25	.7585	.8261	.8101	.7522	.9121	.8118±.0645
7.50	.7691	.8415	.8186	.7620	.9261	.8235±.0664
8.75	.7804	.8556	.8368	.7761	.9381	.8374±.0661
10.00	.7965	.8668	.8453	.7825	.9480	.8478±.0658
11.25	.8078	.8774	.8510	.7903	.9523	.8558±.0640
12.50	.8183	.8859	.8644	.8037	.9643	.8673±.0637
13.75	.8261	.8993	.8701	.8101	.9714	.8754±.0642
15.00	.8360	.9057	.8751	.8165	.9764	.8819±.0631
16.25	.8452	.9114	.8816	.8229	.9822	.8887±.0623
17.50	.8530	.9199	.8880	.8273	.9879	.8952±.0625
18.75	.8601	.9270	.8965	.8344	.9922	.9020±.0615
20.00	.8673	.9342	.9008	.8394	1.0014	.9086±.0629
22.50	.8767	.9442	.9102	.8461	1.0094	.9173±.0632
25.00	.8895	.9536	.9169	.8527	1.0167	.9259±.0628
27.50	.8968	.9630	.9249	.8600	1.0261	.9342±.0638
30.00	.9069	.9689	.9280	.8659	1.0328	.9405±.0636
32.50	.9135	.9756	.9353	.8712	1.0380	.9467±.0634
35.00	.9215	.9829	.9406	.8778	1.0418	.9529±.0624
37.50	.9302	.9888	.9451	.8816	1.0471	.9586±.0626
43.75	.9430	1.0016	.9530	.8951	1.0584	.9702±.0621

[a]Standard deviation.

Table A-59. (Continued)

Tension	Control females 01	02	04	06	10	Mean
	Circumference (cm)					
50.00	.9536	1.0136	.9623	.9037	1.0691	.9805±.0630
56.25	.9629	1.0222	.9723	.9123	1.0791	.9898±.0634
62.50	.9729	1.0294	.9802	.9188	1.0863	.9975±.0632
68.75	.9815	1.0352	.9860	.9239	1.0935	1.0040±.0637
75.00	.9894	1.0404	.9918	.9284	1.1007	1.0101±.0644
81.25	.9959	1.0469	.9963	.9342	1.1052	1.0157±.0640
87.50	1.0017	1.0527	1.0014	.9400	1.1110	1.0214±.0641
93.75	1.0069	1.0579	1.0045	.9465	1.1147	1.0261±.0633
100.00	1.0127	1.0623	1.0089	.9482	1.1220	1.0308±.0651

Table A-60. Circumference-tension data from the initial femoral artery segments of the control group male beagle dogs

Tension	Control males					
	22	23	24	27	28	Mean
	Circumference (cm)					
1.25	.7446	.7674	.8198	.8194	.6223	.7547±.0810[a]
2.50	.7766	.7980	.8442	.8487	.6419	.7819±.0840
3.75	.7927	.8273	.8638	.8828	.6559	.8045±.0900
5.00	.8164	.8475	.8826	.9023	.6685	.8235±.0927
6.25	.8360	.8720	.8946	.9212	.6798	.8407±.0952
7.50	.8493	.8881	.9086	.9505	.6890	.8571±.1008
8.75	.8648	.9007	.9213	.9652	.7072	.8718±.0989
10.00	.8739	.9175	.9305	.9799	.7191	.8842±.0997
11.25	.8825	.9267	.9383	.9905	.7283	.8933±.0999
12.50	.8958	.9373	.9454	.9990	.7389	.9033±.0990
13.75	.9008	.9520	.9532	1.0151	.7467	.9136±.1017
15.00	.9100	.9598	.9617	1.0271	.7580	.9233±.1013
16.25	.9185	.9649	.9647	1.0342	.7651	.9295±.1008
17.50	.9225	.9706	.9697	1.0420	.7736	.9357±.1002
18.75	.9335	.9770	.9734	1.0464	.7807	.9423±.0991
20.00	.9392	.9848	.9777	1.0514	.7885	.9483±.0980
22.50	.9479	.9956	.9843	1.0649	.8042	.9594±.0965
25.00	.9580	1.0036	.9944	1.0736	.8177	.9695±.0946
27.50	.9653	1.0144	.9997	1.0837	.8319	.9790±.0928
30.00	.9726	1.0210	1.0070	1.0924	.8427	.9871±.0918
32.50	.9785	1.0276	1.0122	1.0983	.8521	.9937±.0904
35.00	.9844	1.0336	1.0181	1.1057	.8608	1.0005±.0898
37.50	.9918	1.0388	1.0213	1.1130	.8702	1.0070±.0886
43.75	1.0038	1.0502	1.0334	1.1236	.8919	1.0206±.0844

[a]Standard deviation.

Table A-60. (Continued)

Tension	Control males					
	22	23	24	27	28	Mean
	Circumference (cm)					
50.00	1.0145	1.0601	1.0399	1.1350	.9081	1.0315±.0823
56.25	1.0231	1.0701	1.0478	1.1436	.9229	1.0415±.0801
62.50	1.0310	1.0780	1.0571	1.1515	.9336	1.0502±.0791
68.75	1.0375	1.0852	1.0629	1.1580	.9436	1.0574±.0779
75.00	1.0440	1.0917	1.0694	1.1638	.9557	1.0679±.0756
81.25	1.0498	1.0955	1.0759	1.1697	.9636	1.0709±.0747
87.50	1.0571	1.1006	1.0804	1.1748	.9715	1.0769±.0736
93.75	1.0622	1.1044	1.0862	1.1806	.9794	1.0826±.0727
100.00	1.0673	1.1102	1.0899	1.1865	.9845	1.0877±.0730

Table A-61. Circumference-tension data from the initial femoral artery segments of the treatment group female beagle dogs

Tension	Treatment females					Mean
	03	05	07	08	09	
	Circumference (cm)					
1.25	.7473	.7557	.7730	.7148	.7127	.7407±.0263[a]
2.50	.7856	.7732	.7988	.7523	.7351	.7690±.0255
3.75	.8045	.7921	.8073	.7726	.7547	.7862±.0223
5.00	.8227	.8179	.8304	.7894	.7666	.8054±.0267
6.25	.8395	.8326	.8458	.8180	.7793	.8230±.0265
7.50	.8500	.8487	.8557	.8410	.7926	.8376±.0257
8.75	.8579	.8593	.8690	.8627	.8004	.8499±.0280
10.00	.8733	.8692	.8754	.8746	.8096	.8604±.0285
11.25	.8797	.8818	.8853	.8852	.8243	.8713±.0264
12.50	.8861	.8924	.8973	.8972	.8315	.8809±.0280
13.75	.8939	.8995	.9058	.9140	.8379	.8402±.0302
15.00	.8983	.9108	.9115	.9218	.8464	.8978±.0299
16.25	.9061	.9158	.9186	.9317	.8535	.9051±.0303
17.50	.9111	.9243	.9216	.9374	.8620	.9113±.0291
18.75	.9176	.9335	.9308	.9452	.8677	.9190±.0303
20.00	.9226	.9372	.9345	.9537	.8728	.9242±.0308
22.50	.9320	.9480	.9445	.9645	.8842	.9346±.0305
25.00	.9400	.9580	.9512	.9718	.8943	.9431±.0296
27.50	.9452	.9633	.9592	.9853	.9009	.9508±.0314
30.00	.9525	.9692	.9644	.9961	.9096	.9584±.0316
32.50	.9550	.9772	.9717	1.0020	.9169	.9646±.0315
25.00	.9616	.9831	.9763	1.0093	.9243	.9709±.0313
37.50	.9662	.9884	.9822	1.0139	.9316	.9765±.0304
43.75	.9748	1.0004	.9942	1.0280	.9416	.9878±.0321

[a]Standard deviation.

Table A-61. (Continued)

Tension	Treatment females 03	05	07	08	09	Mean
			Circumference (cm)			
50.00	.9834	1.0111	1.0035	1.0387	.9529	.9979±.0320
56.25	.9913	1.0183	1.0128	1.0466	.9615	1.0061±.0318
62.50	.9978	1.0248	1.0186	1.0552	.9701	1.0133±.0317
68.75	1.0036	1.0300	1.0245	1.0624	.9787	1.0198±.0312
75.00	1.0081	1.0365	1.0310	1.0689	.9852	1.0259±.0315
81.25	1.0132	1.0430	1.0361	1.0747	.9904	1.0315±.0318
87.50	1.0162	1.0474	1.0413	1.0799	.9969	1.0363±.0316
93.75	1.0194	1.0533	1.0478	1.0836	1.0013	1.0411±.0319
100.00	1.0244	1.0584	1.0522	1.0874	1.0058	1.0456±.0316

Table A-62. Circumference-tension data from the initial femoral artery segments of the treatment group male beagle dogs

Tension	Treatment males				Mean
	21	25	26	29	
	Circumference (cm)				
1.25	.7240	.7878	.7153	.7771	.7511±.0367[a]
2.50	.7471	.8060	.7501	.8064	.7774±.0333
3.75	.7666	.8290	.7655	.8259	.7968±.0355
5.00	.7800	.8423	.7851	.8434	.8127±.0349
6.25	.7940	.8598	.8074	.8575	.8297±.0339
7.50	.8081	.8732	.8201	.8673	.8422±.0329
8.75	.8228	.8824	.8334	.8828	.8554±.0318
10.00	.8327	.8930	.8454	.8940	.8663±.0319
11.25	.8426	.9001	.8567	.9032	.8756±.0306
12.50	.8469	.9072	.8679	.9117	.8834±.0313
13.75	.8575	.9205	.8757	.9182	.8930±.0314
15.00	.8646	.9276	.8835	.9260	.9004±.0314
16.25	.8710	.9348	.8907	.9338	.9076±.0319
17.50	.8788	.9391	.8964	.9381	.9131±.0303
18.75	.8832	.9435	.9056	.9432	.9189±.0297
20.00	.8861	.9513	.9120	.9454	.9237±.0305
22.50	.8976	.9607	.9228	.9555	.9342±.0296
25.00	.9056	.9680	.9315	.9642	.9423±.0295
27.50	.9143	.9760	.9395	.9722	.9505±.0292
30.00	.9209	.9812	.9454	.9802	.9569±.0292
32.50	.9262	.9858	.9520	.9868	.9627±.0292
35.00	.9321	.9938	.9600	.9928	.9697±.0296
37.50	.9380	1.0004	.9646	.9980	.9752±.0297
43.75	.9480	1.0131	.9780	1.0101	.9873±.0306

[a]Standard deviation.

Table A-62. (Continued)

Tension	Treatment males 21	25	26	29	Mean
			Circumference (cm)		
50.00	.9580	1.0210	.9894	1.0193	.9969±.0297
56.25	.9652	1.0310	1.0001	1.0279	1.0060±.0306
62.50	.9738	1.0382	1.0100	1.0352	1.0143±.0298
68.75	.9824	1.0434	1.0172	1.0431	1.0215±.0288
75.00	.9896	1.0506	1.0231	1.0503	1.0284±.0289
81.25	.9934	1.0557	1.0289	1.0568	1.0377±.0298
87.50	.9978	1.0629	1.0354	1.0619	1.0395±.0306
93.75	1.0043	1.0674	1.0419	1.0677	1.0453±.0299
100.00	1.0088	1.0718	1.0485	1.0729	1.0505±.0300

Table A-63. Circumference-tension data from the final femoral artery segments of the control group female beagle dogs

Tension	Control females					
	01	02	04	06	10	Mean
	Circumference (cm)					
1.25	.7218	.7458	.7339	.6815	.8020	.7370±.0437[a]
2.50	.7358	.7902	.7680	.6962	.8146	.7610±.0464
3.75	.7491	.8084	.7883	.7158	.8307	.7785±.0461
5.00	.7687	.8294	.7975	.7354	.8503	.7963±.0461
6.25	.7800	.8483	.8150	.7501	.8602	.8107±.0461
7.50	.7913	.8623	.8235	.7607	.8721	.8220±.0470
8.75	.8011	.8791	.8320	.7699	.8820	.8328±.0488
10.00	.8083	.8883	.8439	.7763	.8877	.8409±.0492
11.25	.8175	.8975	.8531	.7862	.9025	.8514±.0503
12.50	.8287	.9074	.8609	.7933	.9124	.8605±.0510
13.75	.8351	.9145	.8687	.7983	.9202	.8674±.0520
15.00	.8416	.9230	.8717	.8034	.9259	.8731±.0527
16.25	.8459	.9322	.8767	.8063	.9303	.8783±.0544
17.50	.8516	.9386	.8832	.8134	.9374	.8848±.0545
18.75	.8608	.9450	.8861	.8192	.9459	.8914±.0548
20.00	.8645	.9494	.8912	.8221	.9495	.8953±.0552
22.50	.8718	.9588	.8985	.8288	.9603	.9036±.0568
25.00	.8798	.9675	.9079	.8368	.9669	.9118±.0566
27.50	.8871	.9741	.9124	.8399	.9756	.9178±.0582
30.00	.8937	.9814	.9204	.8459	.9843	.9251±.0591
32.50	.9004	.9873	.9243	.8511	.9937	.9314±.0601
35.00	.9063	.9939	.9295	.8549	.9962	.9362±.0602
37.50	.9122	.9999	.9334	.8588	1.0021	.9413±.0609
43.75	.9250	1.0133	.9419	.8667	1.0155	.9525±.0630

[a]Standard deviation.

Table A-63. (Continued)

Tension	Control females					
	01	02	04	06	10	Mean
	Circumference (cm)					
50.00	.9350	1.0226	.9512	.8760	1.0262	.9622±.0633
56.25	.9449	1.0312	.9571	.8839	1.0355	.9705±.0637
62.50	.9515	1.0384	.9657	.8918	1.0462	.9787±.0644
68.75	.9600	1.0456	.9722	.8962	1.0534	.9855±.0652
75.00	.9652	1.0528	.9787	.9021	1.0620	.9922±.0663
81.25	.9724	1.0600	.9845	.9072	1.0685	.9985±.0669
87.50	.9789	1.0666	.9883	.9109	1.0736	1.0037±.0677
93.75	.9847	1.0743	.9934	.9147	1.0795	1.0085±.0679
100.00	.9885	1.0768	.9972	.9191	1.0832	1.0130±.0683

Table A-64. Circumference-tension data from the final femoral artery segments of the control group male beagle dogs

Tension	Control males					
	22	23	24	27	28	Mean
	Circumference (cm)					
1.25	.7439	.8027	.7679	.8312	.7268	.7745±.0426[a]
2.50	.7752	.8278	.7840	.8514	.7408	.7958±.0439
3.75	.7934	.8473	.7959	.8613	.7638	.8123±.0406
5.00	.8067	.8745	.8058	.8795	.7813	.8296±.0445
6.25	.8256	.8844	.8157	.8970	.7926	.8431±.0453
7.50	.8348	.9061	.8270	.9055	.8046	.8556±.0471
8.75	.8454	.9242	.8382	.9223	.8144	.8689±.0509
10.00	.8608	.9362	.8474	.9343	.8216	.8801±.0523
11.25	.8693	.9468	.8552	.9428	.8321	.8892±.0524
12.50	.8785	.9560	.8589	.9554	.8406	.8979±.0545
13.75	.8849	.9617	.8667	.9667	.8457	.9151±.0557
15.00	.8886	.9723	.8724	.9724	.8521	.9116±.0570
16.25	.8971	.9808	.8775	.9781	.8565	.9180±.0579
17.50	.9028	.9872	.8825	.9825	.8622	.9234±.0579
18.75	.9085	.9936	.8869	.9910	.8679	.9296±.0590
20.00	.9143	.9994	.8898	1.0009	.8702	.9349±.0616
22.50	.9237	1.0101	.9013	1.0117	.8768	.9447±.0627
25.00	.9324	1.0195	.9093	1.0197	.8855	.9533±.0628
27.50	.9397	1.0262	.9159	1.0304	.8907	.9606±.0642
30.00	.9456	1.0342	.9219	1.0384	.8967	.9674±.0653
32.50	.9508	1.0408	.9264	1.0457	.9047	.9737±.0656
35.00	.9575	1.0474	.9330	1.0510	.9085	.9795±.0660
37.50	.9627	1.0519	.9389	1.0555	.9117	.9841±.0660
43.75	.9734	1.0647	.9482	1.0697	.9224	.9957±.0678

[a]Standard deviation.

Table A-64. (Continued)

Tension	Control males					
	22	23	24	27	28	Mean
	Circumference (cm)					
50.00	.9840	1.0754	.9596	1.0831	.9310	1.0066±.0690
56.25	.9926	1.0867	.9703	1.0924	.9382	1.0160±.0699
62.50	1.0019	1.0939	.9789	1.1010	.9467	1.0245±.0695
68.75	1.0077	1.1005	.9854	1.1082	.9540	1.0312±.0695
75.00	1.0143	1.1063	.9926	1.1140	.9612	1.0377±.0689
81.25	1.0194	1.1114	.9984	1.1198	.9656	1.0434±.0698
87.50	1.0238	1.1165	1.0042	1.1257	.9708	1.0482±.0693
93.75	1.0304	1.1224	1.0087	1.1322	.9780	1.0543±.0692
100.00	1.0369	1.1269	1.0166	1.1366	.9824	1.0599±.0685

Table A-65. Circumference-tension data from the final femoral artery segments of the treatment group female beagle dogs

Tension	Treatment females					
	03	05	07	08	09	Mean
	Circumference (cm)					
1.25	-[a]	.7543	.7391	.7778	-[a]	.7571±.0195[b]
2.50		.7926	.7587	.8264		.7926±.0339
3.75		.8121	.7679	.8550		.8117±.0436
5.00		.8393	.7868	.8731		.8331±.0435
6.25		.8547	.8057	.8948		.8517±.0446
7.50		.8667	.8162	.9026		.8618±.0434
8.75		.8863	.8289	.9139		.8764±.0434
10.00		.8969	.8353	.9286		.8869±.0474
11.25		.9054	.8445	.9399		.8966±.0483
12.50		.9159	.8537	.9470		.9055±.0475
13.75		.9217	.8601	.9548		.9122±.0481
15.00		.9329	.8665	.9591		.9195±.0477
16.25		.9414	.8695	.9683		.9264±.0511
17.50		.9472	.8746	.9727		.9315±.0509
18.75		.9543	.8817	.9750		.9370±.0490
20.00		.9586	.8853	.9793		.9411±.0494
22.50		.9687	.8954	.9880		.9507±.0489
25.00		.9760	.9020	.9960		.9580±.0495
27.50		.9826	.9107	1.0040		.9658±.0489
30.00		.9907	.9146	1.0092		.9715±.0501
32.50		.9973	.9198	1.0152		.9774±.0507
35.00		1.0032	.9251	1.0204		.9829±.0508
37.50		1.0098	.9303	1.0256		.9986±.0511

[a]Dogs 03 and 09 died during refeeding I and their final femoral arteries were not tested.

[b]Standard deviation.

Table A-65. (Continued)

Tension	Treatment females 03	05	07	08	09	Mean
			Circumference (cm)			
43.75		1.0205	.9382	1.0363		.9983±.0527
50.00		1.0312	.9489	1.0421		1.0074±.0510
56.25		1.0370	.9568	1.0494		1.0144±.0503
62.50		1.0442	.9640	1.0573		1.0218±.0505
68.75		1.0507	.9705	1.0631		1.0281±.0503
75.00		1.0579	.9770	1.0703		1.0351±.0507
81.25		1.0638	.9821	1.0754		1.0404±.0509
87.50		1.0696	.9866	1.0806		1.0456±.0514
93.75		1.0747	.9903	1.0864		1.0505±.0524
100.00		1.0799	.9941	1.0894		1.0545±.0525

Table A-66. Circumference-tension data from the final femoral artery segments of the treatment group male beagle dogs

Tension	Treatment males				Mean
	21	25	26	29	
	Circumference (cm)				
1.25	.7061	.8002	.7821	.7986	.7718±.0445[a]
2.50	.7263	.8240	.8003	.8244	.7938±.0464
3.75	.7383	.8456	.8254	.8460	.8138±.0513
5.00	.7523	.8569	.8402	.8566	.8265±.0501
6.25	.7691	.8771	.8535	.8755	.8438±.0510
7.50	.7776	.8863	.8662	.8902	.8551±.0827
8.75	.7882	.8955	.8747	.9042	.8656±.0531
10.00	.7953	.9054	.8852	.9155	.8754±.0548
11.25	.8031	.9125	.8951	.9212	.8830±.0543
12.50	.8137	.9217	.9015	.9276	.8911±.0528
13.75	.8222	.9288	.9107	.9389	.9002±.0533
15.00	.8272	.9366	.9144	.9453	.9059±.0540
16.25	.8350	.9424	.9174	.9525	.9118±.0533
17.50	.8394	.9488	.9231	.9575	.9172±.0539
18.75	.8437	.9518	.9261	.9584	.9200±.0527
20.00	.8502	.9561	.9339	.9655	.9264±.0525
22.50	.8589	.9662	.9439	.9763	.9363±.0534
25.00	.8655	.9735	.9526	.9822	.9434±.0534
27.50	.8728	.9787	.9579	.9944	.9510±.0542
30.00	.8787	.9861	.9631	1.0010	.9572±.0546
32.50	.8847	.9906	.9690	1.0048	.9623±.0538
35.00	.8913	.9972	.9736	1.0128	.9687±.0541
37.50	.8965	1.0018	.9788	1.0181	.9738±.0540
43.75	.9079	1.0131	.9909	1.0281	.9850±.0536

[a]Standard deviation.

Table A-66. (Continued)

Tension	Treatment males 21	25	26	29	Mean
			Circumference (cm)		
50.00	.9165	1.0238	1.0015	1.0380	.9950±.0544
56.25	.9258	1.0303	1.0101	1.0487	1.0037±.0543
62.50	.9337	1.0382	1.0174	1.0566	1.0115±.0543
68.75	.9395	1.0440	1.0232	1.0652	1.0180±.0551
75.00	.9453	1.0499	1.0290	1.0731	1.0243±.0557
81.25	.9511	1.0557	1.0348	1.0775	1.0298±.0553
87.50	.9570	1.0622	1.0414	1.0834	1.0360±.0554
93.75	.9628	1.0660	1.0479	1.0899	1.0416±.0553
100.00	.9665	1.0718	1.0538	1.0964	1.0471±.0565

Table A-67. Group means for circumference-tension data from the initial femoral artery segments from beagle dogs

Tension	Overall control mean	Overall treatment mean
	Circumference (cm)	
1.25	.7404 ± .0724[a]	.7453 ± .0297
2.50	.7693 ± .0727	.7727 ± .0276
3.75	.7906 ± .0755	.7909 ± .0274
5.00	.8089 ± .0765	.8086 ± .0287
6.25	.8263 ± .0782	.8260 ± .0282
7.50	.8403 ± .0824	.8396 ± .0272
8.75	.8546 ± .0814	.8523 ± .0279
10.00	.8660 ± .0819	.8630 ± .0282
11.25	.8745 ± .0815	.8732 ± .0265
12.50	.8853 ± .0807	.8820 ± .0276
13.75	.8945 ± .0827	.8914 ± .0288
15.00	.9026 ± .0825	.8989 ± .0286
16.25	.9091 ± .0819	.9062 ± .0290
17.50	.9154 ± .0816	.9121 ± .0277
18.75	.9221 ± .0805	.9189 ± .0281
20.00	.9285 ± .0804	.9240 ± .0287
22.50	.9384 ± .0801	.9344 ± .0282
25.00	.9477 ± .0791	.9427 ± .0276
27.50	.9566 ± .0787	.9507 ± .0285
30.00	.9638 ± .0784	.9577 ± .0286
32.50	.9702 ± .0777	.9637 ± .0286
35.00	.9767 ± .0771	.9704 ± .0286
37.50	.9828 ± .0767	.9759 ± .0282
43.75	.9954 ± .0747	.9876 ± .0294
50.00	1.0060 ± .0742	.9975 ± .0291

[a]Standard deviation.

Table A-67. (Continued)

Tension	Overall control mean	Overall treatment mean
	Circumference (cm)	
56.25	1.0156 ± .0734	1.0061 ± .0293
62.50	1.0238 ± .0730	1.0137 ± .0289
68.75	1.0307 ± .0728	1.0206 ± .0283
75.00	1.0375 ± .0722	1.0270 ± .0285
81.25	1.0433 ± .0718	1.0325 ± .0290
87.50	1.0491 ± .0713	1.0377 ± .0292
93.75	1.0543 ± .0708	1.0430 ± .0291
100.00	1.0592 ± .0718	1.0478 ± .0290

Table A-68. Group means for circumference-tension data from the final femoral artery segments from beagle dogs

Tension	Overall control mean	Overall treatment mean
	Circumference (cm)	
1.25	.7558 ± .0452[a]	.7655 ± .0343
2.50	.7778 ± .0569	.7932 ± .0382
3.75	.7954 ± .0447	.8129 ± .0441
5.00	.8129 ± .0462	.8293 ± .0435
6.25	.8269 ± .0463	.8472 ± .0445
7.50	.8388 ± .0478	.8580 ± .0451
8.75	.8509 ± .0507	.8702 ± .0455
10.00	.8604 ± .0521	.8803 ± .0479
11.25	.8703 ± .0524	.8888 ± .0480
12.50	.8792 ± .0535	.8973 ± .0470
13.75	.8862 ± .0546	.9053 ± .0472
15.00	.8923 ± .0556	.9117 ± .0477
16.25	.8981 ± .0570	.9181 ± .0485
17.50	.9041 ± .0568	.9233 ± .0487
18.75	.9105 ± .0574	.9273 ± .0477
20.00	.9151 ± .0589	.9327 ± .0475
22.50	.9242 ± .0604	.9425 ± .0477
25.00	.9325 ± .0604	.9497 ± .0480
27.50	.9392 ± .0620	.9573 ± .0482
30.00	.9462 ± .0628	.9633 ± .0489
32.50	.9525 ± .0634	.9688 ± .0487
35.00	.9578 ± .0638	.9748 ± .0488
37.50	.9627 ± .0640	.9801 ± .0489
43.75	.9741 ± .0658	.9907 ± .0491

[a]Standard deviation.

Table A-68. (Continued)

Tension	Overall control mean	Overall treatment mean
	Circumference (cm)	
50.00	.9844 ± .0667	1.0003 ± .0489
56.25	.9933 ± .0675	1.0083 ± .0485
62.50	1.0015 ± .0677	1.0159 ± .0485
68.75	1.0083 ± .0680	1.0223 ± .0489
75.00	1.0149 ± .0681	1.0289 ± .0494
81.25	1.0207 ± .0683	1.0343 ± .0492
87.50	1.0259 ± .0687	1.0401 ± 1.494
93.75	1.0314 ± .0690	1.0454 ± .0497
100.00	1.0364 ± .0691	1.0503 ± .0503

Table A-69. Circumference-tension data from the right femoral artery segments of mongrel dogs

Tension	Mongrel dogs					
	01	02	03	04	05	Mean
	Circumference (cm)					
1.25	.9221	1.0960	.8389	.7905	.8948	.9085±.1165[a]
2.50	.9679	1.1273	.8917	.8315	.9255	.9488±.1116
3.75	1.0020	1.1580	.9175	.8566	.9485	.9765±.1143
5.00	1.0264	1.1942	.9371	.8845	.9805	1.0045±.1183
6.25	1.0536	1.2144	.9615	.9103	.9946	1.0269±.1170
7.50	1.0725	1.2354	.9707	.9237	1.0176	1.0440±.1204
8.75	1.0872	1.2653	.9923	.9412	1.0358	1.0644±.1246
10.00	1.1123	1.2835	1.0029	.9559	1.0491	1.0807±.1272
11.25	1.1271	1.2996	1.0142	.9665	1.0590	1.0933±.1296
12.50	1.1425	1.3171	1.0247	.9798	1.0710	1.1070±.1320
13.75	1.1579	1.3277	1.0298	.9869	1.0788	1.1162±.1342
15.00	1.1671	1.3452	1.0376	.9933	1.0914	1.1269±.1382
16.25	1.1811	1.3620	1.0461	1.0032	1.0992	1.1383±.1416
17.50	1.1931	1.3712	1.0518	1.0083	1.1050	1.1459±.1436
18.75	1.2009	1.3839	1.0589	1.0154	1.1128	1.1544±.1458
20.00	1.2094	1.3944	1.0626	1.0225	1.1157	1.1609±.1481
22.50	1.2250	1.4142	1.0720	1.0312	1.1300	1.1745±.1525
25.00	1.2420	1.4305	1.0800	1.0392	1.1387	1.1861±.1565
27.50	1.2556	1.4434	1.0831	1.0507	1.1453	1.1956±.1590
30.00	1.2670	1.4597	1.0912	1.0587	1.1554	1.2064±.1624
32.50	1.2750	1.4711	1.0971	1.0653	1.1606	1.2138±.1647
35.00	1.2837	1.4791	1.0989	1.0698	1.1679	1.2199±.1667
37.50	1.2931	1.4899	1.1041	1.0758	1.1732	1.2272±.1691
43.75	1.3093	1.5123	1.1141	1.0857	1.1845	1.2412±.1744

[a]Standard deviation.

Table A-69. (Continued)

Tension	Mongrel dogs					
	01	02	03	04	05	Mean
	Circumference (cm)					
50.00	1.3255	1.5327	1.1227	1.0971	1.1945	1.2545±.1790
56.25	1.3376	1.5475	1.1299	1.1050	1.2031	1.2646±.1822
62.50	1.3483	1.5596	1.1343	1.1136	1.2124	1.2736±.1845
68.75	1.3562	1.5709	1.1395	1.1208	1.2182	1.2811±.1866
75.00	1.3648	1.5802	1.1453	1.1273	1.2233	1.2882±.1882
81.25	1.3720	1.5902	1.1490	1.1318	1.2292	1.2944±.1906
87.50	1.3778	1.6002	1.1528	1.1369	1.2350	1.3005±.1928
93.75	1.3857	1.6088	1.1565	1.1427	1.2394	1.3066±.1946
100.00	1.3922	1.6160	1.1596	1.1458	1.2446	1.3116±.1964

Table A-70. Circumference-tension data from the left femoral artery segments of mongrel dogs

Tension	Mongrel dogs					
	01	02	03	04	05	Mean
	Circumference (cm)					
1.25	.9311	1.1320	.8223	.8099	.9661	.9323±.1305[a]
2.50	.9534	1.1772	.8571	.8308	1.0092	.9655±.1385
3.75	.9903	1.2057	.8774	.8587	1.0191	.9902±.1202
5.00	1.0105	1.2551	.9087	.8762	1.0525	1.0206±.1496
6.25	1.0453	1.2857	.9359	.8916	1.0831	1.0483±.1539
7.50	1.0663	1.3094	.9568	.9119	1.0992	1.0687±.1549
8.75	1.0810	1.3373	.9799	.9204	1.1188	1.0875±.1604
10.00	1.0999	1.3500	.9932	.9289	1.1280	1.1000±.1612
11.25	1.1160	1.3723	1.0059	.9374	1.1448	1.1153±.1662
12.50	1.1273	1.3967	1.0261	.9431	1.1616	1.1310±.1717
13.75	1.1448	1.4094	1.0374	.9516	1.1694	1.1425±.1728
15.00	1.1567	1.4199	1.0487	.9587	1.1779	1.1524±.1736
16.25	1.1687	1.4326	1.0579	.9638	1.1857	1.1617±.1760
17.50	1.1779	1.4439	1.0636	.9695	1.1928	1.1695±.1783
18.75	1.1871	1.4600	1.0769	.9746	1.2055	1.1808±.1816
20.00	1.1990	1.4726	1.0834	.9789	1.2105	1.1889±.1846
22.50	1.2140	1.4882	1.0964	.9862	1.2213	1.2013±.1871
25.00	1.2296	1.5025	1.1063	.9928	1.2300	1.2122±.1899
27.50	1.2452	1.5181	1.1171	1.0002	1.2408	1.2243±.1928
30.00	1.2553	1.5296	1.1258	1.0033	1.2495	1.2327±.1956
32.50	1.2640	1.5431	1.1338	1.0092	1.2589	1.2418±.1983
35.00	1.2741	1.5518	1.1404	1.0152	1.2648	1.2493±.1994
37.50	1.2814	1.5626	1.1470	1.0197	1.2700	1.2561±.2016
43.75	1.2997	1.5829	1.1584	1.0290	1.2842	1.2708±.2059

[a]Standard deviation.

Table A-70. (Continued)

Tension	Mongrel dogs					
	01	02	03	04	05	Mean
	Circumference (cm)					
50.00	1.3159	1.5991	1.1683	1.0397	1.2955	1.2837±.2083
56.25	1.3272	1.6140	1.1769	1.0476	1.3069	1.2945±.2110
62.50	1.3386	1.6274	1.1821	1.0541	1.3176	1.3040±.2141
68.75	1.3479	1.6374	1.1886	1.0592	1.3275	1.3121±.2160
75.00	1.3558	1.6480	1.1937	1.0657	1.3348	1.3196±.2178
81.25	1.3623	1.6553	1.1989	1.0702	1.3413	1.3256±.2188
87.50	1.3702	1.6638	1.2033	1.0746	1.3464	1.3317±.2206
93.75	1.3746	1.6724	1.2071	1.0812	1.3536	1.3378±.2216
100.00	1.3805	1.6803	1.2101	1.0849	1.3594	1.3430±.2235

Table A-71. Mean circumference-tension data from the average of the right and left femoral artery segments of mongrel dogs

Tension	Mean Circumference (cm)	Tension	Mean Circumference (cm)
1.25	.9204±.1226[a]	25.00	1.1992±.1716
2.50	.9572±.1234	27.50	1.2100±.1742
3.75	.9834±.1252	30.00	1.2196±.1772
5.00	1.0126±.1328	32.50	1.2278±.1796
6.25	1.0376±.1340	35.00	1.2346±.1812
7.50	1.0564±.1366	37.50	1.2417±.1834
8.75	1.0759±.1414	43.75	1.2560±.1882
10.00	1.0904±.1431	50.00	1.2691±.1916
11.25	1.1043±.1467	56.25	1.2796±.1945
12.50	1.1190±.1504	62.50	1.2888±.1971
13.75	1.1294±.1521	68.75	1.2966±.1990
15.00	1.1396±.1546	75.00	1.3039±.2007
16.25	1.1500±.1574	81.25	1.3100±.2024
17.50	1.1577±.1595	87.50	1.3161±.2044
18.75	1.1676±.1622	93.75	1.3222±.2057
20.00	1.1749±.1648	100.00	1.3430±.2235
22.50	1.1879±.1683		

[a]Standard deviation.

Table A-72. Slope of the circumference-tension graph (slope I) for the initial femoral artery segments from beagle dogs

Group	Tension (gm) 0	20	40	60	80	100
	Slope per 0.2 mm circumference increase (gm)					
Control females						
01	.30	3.76	7.65	11.96	16.69	21.85
02	-.03	4.18	8.72	13.58	18.78	24.32
04	.03	4.68	9.75	15.24	21.15	27.47
06	.14	4.70	9.73	15.22	21.16	27.56
10	.25	4.55	9.10	13.89	18.93	24.21
Mean	.14 ±.14[a]	4.37 ±.40	8.99 ±.87	13.98 ±1.36	19.34 ±1.88	25.08 ±2.43
Males						
22	-.11	4.30	8.78	13.35	18.00	22.74
23	.06	3.92	8.54	13.92	20.07	26.97
24	.02	5.15	10.12	14.94	19.60	24.11
27	-.09	3.86	8.33	13.33	18.86	24.92
28	.92	3.12	5.86	9.12	12.93	17.27
Mean	.16 ±.43	4.07 ±.74	8.33 ±1.55	12.93 ±2.23	17.89 ±2.88	23.20 ±3.65
Overall control mean	.15 ±.30	4.22 ±.58	8.66 ±1.23	13.46 ±1.83	18.62 ±2.42	24.14 ±3.09
Treatment females						
03	.16	4.80	10.28	16.59	23.74	31.72
05	.23	4.13	8.93	14.65	21.27	28.50
07	.22	4.59	9.41	14.69	20.42	26.61

[a]Standard deviation.

Table A-72. (Continued)

Group	Tension (gm) 0	20	40	60	80	100
	Slope per 0.2 mm circumference increase (gm)					
08	.03	3.59	7.94	13.06	18.98	25.67
09	.51	4.10	8.26	12.99	18.29	24.16
Mean	.23 ±.18	4.24 ±.47	8.96 ±.93	14.40 ±1.48	20.54 ±2.14	27.33 ±2.91
Males						
21	.14	4.51	9.22	14.28	19.68	25.42
25	.09	4.52	9.17	14.06	19.18	24.54
26	.14	4.06	8.24	12.66	17.33	22.26
29	.06	4.65	9.19	13.69	18.14	22.54
Mean	.11 ±.04	4.44 ±.26	8.96 ±.48	13.68 ±.72	18.58 ±1.05	23.69 ±1.54
Overall treatment mean	.18 ±.14	4.33 ±.38	8.96 ±.72	14.07 ±1.20	19.67 ±1.94	25.75 ±3.01

Table A-73. Slope of the circumference-tension graph (slope I) for the final femoral artery segments from beagle dogs

Group	Tension (gm) 0	20	40	60	80	100
	Slope per 0.02 mm circumference increase (gm)					
Control females						
01	.19	4.59	9.03	13.49	17.98	22.50
02	-.14	4.45	8.96	13.40	17.77	22.06
04	-.28	5.59	10.81	15.40	19.34	22.65
06	-.42	5.72	11.37	16.54	21.22	25.43
10	.37	4.25	8.34	12.64	17.16	21.90
Mean	-.06 ±.33[a]	4.92 ±.68	9.70 ±1.31	14.29 ±1.62	18.69 ±1.62	22.91 ±1.44
Males						
22	.27	4.45	9.11	14.26	19.91	26.04
23	.12	3.98	8.47	13.57	19.30	25.65
24	.58	4.90	9.28	13.72	18.22	22.78
27	.61	3.78	7.90	12.96	18.97	25.92
28	-.14	5.29	10.29	14.86	19.01	22.73
Mean	.29 ±.32	4.48 ±.63	9.01 ±.90	13.87 ±.72	19.08 ±.61	24.62 ±1.71
Overall control mean	.12 ±.35	4.70 ±.66	9.36 ±1.12	14.08 ±1.20	18.89 ±1.17	23.77 ±1.75
Treatment females						
03	-[b]					

[a]Standard deviation.

[b]Dogs 03 and 09 died during the experiment and a final artery segment was not obtained.

Table A-73. (Continued)

Group	Tension (gm) 0	20	40	60	80	100
	Slope per 0.02 mm circumference increase (gm)					
05	-.07	4.38	9.18	14.31	19.77	25.58
07	.10	5.14	10.11	15.02	19.86	24.64
08	-.31	5.11	10.35	15.42	20.32	25.03
09	-[b]					
Mean	-.09 ±.21	4.88 ±.43	9.88 ±.62	14.92 ±56	19.98 ±30	25.08 ±47
Males						
21	.14	4.87	9.61	14.36	19.12	23.89
25	.04	4.87	9.77	14.76	19.84	25.00
26	.08	4.84	9.64	14.50	19.40	24.34
29	.18	4.53	8.94	13.41	17.93	22.51
Mean	.11 ±.06	4.78 ±.17	9.49 ±.37	14.26 ±.59	19.07 ±.82	23.94 ±1.05
Overall treatment mean	.02 ±.17	4.82 ±.28	9.66 ±.49	14.54 ±.63	19.46 ±.77	24.43 ±1.00

Table A-74. Slope of the circumference-tension graph (slope I) for the femoral artery segments from mongrel dogs

Group	Side[a]	Tension (gm) 0	20	40	60	80	100
		Slope per 0.02 mm circumference increase (gm)					
Mongrels							
01	R	.19	2.77	6.17	10.40	15.46	21.35
02	R	.20	2.48	5.21	8.40	12.03	16.12
03	R	-.09	4.63	10.52	17.56	25.75	35.11
04	R	-.09	4.06	8.71	13.86	19.51	25.66
05	R	-.09	4.02	8.63	13.75	19.37	25.50
Mean		.02 ±.16[b]	3.59 ±.92	7.85 ±2.14	12.79 ±3.53	18.42 ±5.13	24.75 ±6.98
01	L	.23	2.75	6.09	10.25	15.21	20.99
02	L	-.07	2.61	5.58	8.83	12.36	16.18
03	L	.03	3.36	8.15	14.39	22.09	31.24
04	L	-.09	4.06	8.71	13.86	19.51	25.66
05	L	-.07	3.64	7.53	11.59	15.84	20.26
Mean		-.04 ±.20	3.52 ±1.05	7.58 ±1.97	12.15 ±2.85	17.22 ±4.03	22.80 ±5.73
Overall mongrel mean		-.01 ±.15	3.56 ±.88	7.72 ±1.89	12.47 ±3.05	17.82 ±4.48	23.78 ±6.27

[a]R = right femoral artery; L = left femoral artery.

[b]Standard deviation.

Table A-75. Strain ratio-tension data for the initial segment of femoral artery from beagle dogs

Strain ratio	Control females					
	01	02	04	06	10	Mean
	Tension (gm)					
1.00	5.96	6.78	6.37	7.17	8.70	7.00±1.05[a]
1.02	7.50	8.20	8.08	8.62	11.67	8.81±1.65
1.04	9.04	9.91	9.71	11.16	14.67	10.90±2.24
1.06	10.30	12.02	12.05	12.75	18.97	13.22±3.34
1.08	12.02	13.76	15.12	15.71	23.59	16.04±4.45
1.10	14.16	16.92	18.09	18.98	29.47	19.52±5.85
1.12	16.15	19.73	21.96	24.19	39.17	24.24±8.86
1.14	18.65	23.84	27.36	29.91	49.85	29.92±11.91
1.16	21.96	28.73	35.04	37.02	63.64	37.28±15.87
1.18	25.05	34.90	46.33	44.49	82.54	46.67±21.78
1.20	29.48	42.61	56.88	55.53		46.12±12.83
1.22	34.49	51.66	72.27	72.33		57.69±18.27
1.24	39.82	65.86	95.21	89.07		72.49±25.18
1.26	47.89	83.72				65.80±25.34
1.28	57.62					
1.30	67.80					
1.32	80.63					
1.34	97.48					
1.36						
1.38						

[a]Standard deviation.

Table A-76. Strain ratio-tension data for the initial segment of femoral artery from beagle dogs

Strain ratio	Control males					
	22	23	24	27	28	Mean
	Tension (gm)					
1.00	8.48	4.45	7.00	3.47	8.93	6.47±2.42[a]
1.02	10.69	5.40	8.73	4.39	10.53	7.95±2.91
1.04	12.52	6.26	11.40	5.52	12.30	9.60±3.42
1.06	15.45	7.58	14.33	6.53	14.27	11.63±4.22
1.08	18.38	9.12	19.29	7.28	16.32	14.08±5.51
1.10	22.40	10.67	24.72	8.54	18.58	16.98±7.12
1.12	27.33	12.66	32.10	10.03	20.87	20.60±9.39
1.14	33.97	14.39	41.71	12.31	23.24	25.12±12.61
1.16	41.37	17.92	56.02	13.76	25.81	30.98±17.53
1.18	51.43	21.09	71.29	15.99	28.56	37.67±23.17
1.20	65.08	25.63	90.97	19.71	32.12	46.70±30.34
1.22	82.17	30.78		23.31	36.08	43.08±26.58
1.24		38.09		27.94	40.05	35.36±6.50
1.26		47.80		34.01	44.25	42.02±7.16
1.28		58.85		41.77	49.72	50.11±8.55
1.30		73.49		52.00	55.67	60.39±11.50
1.32		96.50		66.11	63.83	75.48±18.24
1.34				85.02	72.03	78.52±9.19
1.36					81.68	
1.38					92.89	

[a]Standard deviation.

Table A-77. Strain ratio-tension data for the initial segment of femoral artery from beagle dogs

Strain ratio	Treatment females					
	03	05	07	08	09	Mean
	Tension (gm)					
1.00	3.92	6.64	4.70	5.12	6.43	5.36±1.16[a]
1.02	5.03	8.17	5.89	5.81	8.17	6.61±1.46
1.04	6.23	10.19	7.70	6.56	10.24	8.18±1.93
1.06	8.34	11.96	9.77	7.42	11.91	9.88±2.05
1.08	9.85	14.31	11.82	8.33	14.61	11.78±2.74
1.10	12.73	17.06	14.07	9.65	17.11	14.12±3.14
1.12	15.87	20.22	17.79	11.45	20.47	17.16±3.71
1.14	19.32	24.21	21.44	12.93	24.07	20.39±4.64
1.16	23.76	30.75	26.75	14.53	29.01	24.96±6.39
1.18	29.86	37.50	33.35	16.91	34.16	30.36±7.99
1.20	39.03	46.50	41.42	19.53	41.17	37.53±10.43
1.22	50.83	59.66	51.83	23.15	50.11	47.12±13.93
1.24	65.47	77.05	66.90	26.92	61.47	59.56±19.13
1.26	88.41	96.31	85.05	30.82	74.13	74.94±25.92
1.28				37.50	91.81	64.66±38.40
1.30				44.75		
1.32				55.44		
1.34				67.90		
1.36				84.31		
1.38						

[a]Standard deviation.

Table A-78. Strain ratio-tension data for the initial segment of femoral artery from beagle dogs

Strain ratio	Treatment males				
	21	25	26	29	Mean
			Tension (gm)		
1.00	7.91	7.65	6.69	5.54	6.95±1.08[a]
1.02	9.55	9.86	8.25	7.39	8.76±1.15
1.04	12.07	12.70	9.89	8.83	10.87±1.82
1.06	14.49	14.84	11.69	10.87	12.97±1.99
1.08	17.35	18.88	13.93	13.61	15.94±2.59
1.10	21.75	22.87	16.78	16.44	19.46±3.33
1.12	26.39	29.05	19.48	21.49	24.10±4.39
1.14	32.72	36.11	23.30	26.32	29.61±5.85
1.16	40.59	44.58	28.48	31.96	36.40±7.46
1.18	51.07	56.82	34.38	39.78	45.51±10.26
1.20	63.72	73.78	42.01	50.03	57.38±14.14
1.22	78.56	92.75	50.66	63.47	71.36±18.26
1.24	98.93		60.44	77.98	79.12±19.27
1.26			74.91	96.94	85.92±15.58
1.28			91.15		
1.30					
1.32					
1.34					
1.36					
1.38					

[a]Standard deviation.

Table A-79. Strain ratio-tension data for the final segment of femoral artery from beagle dogs

Strain ratio	Control females					
	01	02	04	06	10	Mean
	Tension (gm)					
1.00	10.64	6.97	3.55	3.69	8.65	6.70±3.10[a]
1.02	12.61	8.34	5.24	4.61	10.94	8.35±3.48
1.04	16.14	10.32	6.48	5.69	13.15	10.36±4.42
1.06	19.07	12.57	8.78	7.16	16.92	12.90±5.11
1.08	24.45	15.26	10.54	9.18	20.50	15.99±6.49
1.10	30.21	18.17	12.93	11.29	25.69	19.66±8.14
1.12	36.80	22.61	16.75	14.34	30.70	24.24±9.44
1.14	44.90	28.26	21.32	17.84	38.65	30.19±11.43
1.16	55.09	34.80	26.57	22.66	47.65	37.35±13.78
1.18	68.24	42.46	33.53	29.04	58.78	46.41±16.68
1.20	84.32	53.69	43.86	36.90	71.70	58.09±19.65
1.22		68.04	56.80	47.43	89.04	65.33±17.91
1.24		83.07	70.04	58.29		70.47±12.40
1.26			88.76	73.61		81.14±10.64
1.28				94.27		
1.30						
1.32						
1.34						
1.36						
1.38						

[a]Standard deviation.

Table A-80. Strain ratio-tension data for the final segment of femoral artery from beagle dogs

Strain ratio	Control males					
	22	23	24	27	28	Mean
	Tension (gm)					
1.00	6.40	4.21	17.70	5.83	4.70	7.77±5.62[a]
1.02	8.50	5.00	22.41	7.76	6.26	9.99±7.08
1.04	9.92	6.67	28.60	9.21	7.97	12.47±9.10
1.06	12.20	7.69	36.34	11.43	10.27	15.60±11.75
1.08	15.63	8.93	46.84	13.27	12.31	19.40±15.53
1.10	18.93	10.82	57.16	16.84	15.81	23.91±18.82
1.12	23.14	13.44	72.04	19.64	20.10	29.67±23.94
1.14	28.66	15.76	91.16	23.81	25.24	36.93±30.68
1.16	35.72	18.96		28.51	31.52	28.68±7.12
1.18	45.00	22.92		35.28	40.66	35.96±9.56
1.20	55.82	28.35		43.60	51.45	44.81±12.08
1.22	69.54	34.48		52.72	64.17	55.23±15.51
1.24	88.71	43.01		65.95	78.57	69.06±19.70
1.26		52.73		84.40	95.57	77.57±22.22
1.28		65.89				
1.30		85.14				
1.32						
1.34						
1.36						
1.38						

[a]Standard deviation.

Table A-81. Strain ratio-tension data for the final segment of femoral artery from beagle dogs

Strain ratio	Treatment females					
	03	05	07	08	09	Mean
			Tension (gm)			
1.00	-[a]	4.60	6.51	1.46	-	4.19±2.55[b]
1.02		5.65	8.27	1.87		5.26±3.22
1.04		7.21	10.67	2.27		6.72±4.22
1.06		8.38	13.02	2.80		8.07±5.12
1.08		10.04	17.00	3.49		10.18±6.76
1.10		12.24	20.83	4.42		12.50±8.21
1.12		14.72	25.81	5.42		15.32±10.21
1.14		17.46	33.07	6.47		19.00±13.37
1.16		21.23	42.94	8.53		24.23±17.40
1.18		26.58	53.53	9.91		30.01±22.01
1.20		32.33	67.78	11.86		37.32±28.29
1.22		39.62	86.16	14.97		46.92±36.15
1.24		49.35		18.64		34.00±21.72
1.26		64.87		23.28		44.08±29.41
1.28		80.75		28.55		54.65±36.91
1.30				35.72		
1.32				45.17		
1.34				59.42		
1.36				73.95		
1.38				92.06		

[a]Dogs 03 and 09 died during refeeding I and their final femoral arteries were not tested.

[b]Standard deviation.

Table A-82. Strain ratio-tension data for the final segment of femoral artery from beagle dogs

Strain ratio	Treatment males				
	21	25	26	29	Mean
	Tension (gm)				
1.00	8.16	8.15	7.52	5.45	7.32±1.28[a]
1.02	10.57	10.62	9.81	6.69	9.42±1.86
1.04	12.62	13.38	12.39	8.19	11.64±2.34
1.06	15.47	16.68	16.45	9.97	14.64±3.16
1.08	19.16	21.56	20.43	13.04	18.55±3.80
1.10	23.50	28.00	25.14	15.77	23.10±5.23
1.12	29.35	35.44	33.17	20.35	29.58±6.65
1.14	35.75	45.34	42.04	25.43	37.14±8.76
1.16	44.21	58.90	52.44	30.36	46.48±12.31
1.18	55.18	76.74	67.73	37.96	59.40±16.81
1.20	69.13	97.34	85.78	48.77	75.26±21.11
1.22	85.93			59.92	72.92±18.39
1.24				73.03	
1.26				91.82	
1.28					
1.30					
1.32					
1.34					
1.36					
1.38					

[a]Standard deviation.

Table A-83. Group means for strain ratio-tension data from the initial femoral artery segments from beagle dogs

Strain ratio	Overall control mean	Overall treatment mean
	Tension (gm)	
1.00	6.73±1.78[a](10)[b]	6.07±1.34 (9)
1.02	8.38±2.28 (10)	7.57±1.69 (9)
1.04	10.25±2.81 (10)	9.38±2.26 (9)
1.06	12.42±3.68 (10)	11.25±2.50 (9)
1.08	15.06±4.84 (10)	13.63±3.33 (9)
1.10	18.25±6.29 (10)	16.50±4.12 (9)
1.12	22.42±8.82 (10)	20.25±5.24 (9)
1.14	27.52±11.84 (10)	24.49±6.87 (9)
1.16	34.13±16.11 (10)	30.05±8.81 (9)
1.18	42.17±21.72 (10)	37.09±11.63 (9)
1.20	46.45±22.85 (9)	46.35±15.45 (9)
1.22	50.39±22.51 (8)	57.89±19.63 (9)
1.24	56.58±26.93 (7)	66.90±20.44 (8)
1.26	51.53±18.86 (5)	78.08±22.74 (7)
1.28	51.99±7.92 (4)	73.49±31.17 (3)
1.30	62.24±10.09 (3)	
1.32	76.77±15.11 (3)	
1.34	84.84±12.73 (2)	
1.36		
1.38		

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-84. Group means for strain ratio-tension data from the final femoral artery segments from beagle dogs

Strain ratio	Overall control mean	Overall treatment mean
	Tension (gm)	
1.00	7.23±4.32[a](10)[b]	5.98±2.41 (7)
1.02	9.17±5.33 (10)	7.64±3.18 (7)
1.04	11.42±6.84 (10)	9.53±3.95 (7)
1.06	14.25±8.66 (10)	11.82±5.11 (7)
1.08	17.69±11.36 (10)	14.96±6.52 (7)
1.10	21.78±13.86 (10)	18.56±8.26 (7)
1.12	26.96±17.40 (10)	23.47±10.72 (7)
1.14	33.56±22.12 (10)	29.37±13.85 (7)
1.16	33.50±11.61 (9)	36.94±17.84 (7)
1.18	41.77±14.27 (9)	46.80±23.44 (7)
1.20	52.19±17.23 (9)	59.00±30.01 (7)
1.22	60.28±16.42 (8)	57.32±30.68 (5)
1.24	69.66±15.68 (7)	47.01±27.27 (3)
1.26	78.99±16.70 (5)	59.99±34.53 (3)
1.28	80.08±20.07 (2)	54.65±36.91 (2)
1.30		
1.32		
1.34		
1.36		
1.38		

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-85. Strain ratio-tension data for the right femoral artery from mongrel dogs

Strain ratio	Mongrel dogs					
	01	02	03	04	05	Mean
	Tension (gm)					
1.00	4.10	7.34	1.60	2.17	2.43	3.53±2.32[a]
1.02	5.12	8.42	2.00	2.78	3.41	4.35±2.55
1.04	6.05	9.90	2.40	3.60	4.23	5.24±2.92
1.06	7.30	11.75	3.13	4.35	4.95	6.30±3.40
1.08	8.87	14.01	4.02	5.09	6.42	7.68±3.97
1.10	9.87	15.80	5.09	5.89	7.42	8.81±4.31
1.12	11.48	18.43	5.97	7.09	8.67	10.33±4.98
1.14	13.12	21.37	7.63	8.36	10.51	12.20±5.56
1.16	15.29	24.91	8.62	9.68	12.60	14.22±6.52
1.18	17.23	29.22	10.47	11.45	14.87	16.65±7.53
1.20	20.20	35.02	12.44	13.41	18.07	19.83±9.08
1.22	23.36	41.39	15.54	16.02	21.98	23.66±10.51
1.24	26.66	48.72	18.64	19.17	27.55	28.15±12.22
1.26	31.30	59.19	23.57	23.44	33.65	34.23±14.68
1.28	37.03	73.40	30.56	27.49	42.62	42.22±18.39
1.30	44.61	89.18	40.91	33.46	54.68	52.57±21.85
1.32	53.21		53.42	42.31	70.22	54.79±11.52
1.34	65.38		73.28	52.18	91.57	70.60±16.47
1.36	81.38			64.18		73.10±11.71
1.38				82.37		

[a]Standard deviation.

Table A-86. Strain ratio-tension data for the left femoral artery from mongrel dogs

Strain ratio	Mongrel dogs					
	01	02	03	04	05	Mean
	Tension (gm)					
1.00	5.09	4.56	2.83	5.40	4.91	4.56±1.01[a]
1.02	5.81	5.30	3.84	6.69	5.76	5.48±1.05
1.04	6.73	6.32	4.53	8.17	6.94	6.54±1.32
1.06	8.13	7.61	5.25	10.76	8.38	8.03±1.97
1.08	9.61	8.72	6.04	13.76	10.45	9.72±2.80
1.10	11.11	10.64	7.01	17.46	12.01	11.65±3.77
1.12	13.01	11.96	7.99	22.77	14.72	14.09±5.45
1.14	14.79	13.93	9.06	30.53	17.91	17.24±8.08
1.16	17.10	16.58	10.71	39.16	21.73	21.06±10.85
1.18	19.60	18.79	11.98	50.11	27.08	25.51±14.75
1.20	22.74	22.00	13.47	66.57	32.97	31.55±20.76
1.22	25.99	26.18	15.47	87.91	42.35	39.58±28.68
1.24	30.20	30.93	18.04		53.56	33.18±14.82
1.26	35.75	36.76	20.60		65.90	39.75±18.93
1.28	42.67	44.21	24.36		84.57	48.95±25.40
1.30	50.50	54.11	28.67			44.43±13.76
1.32	61.64	66.40	34.24			54.09±17.36
1.34	76.44	83.52	42.24			67.40±22.08
1.36	96.86		53.28			75.07±30.82
1.38			70.59			
1.40			94.37			

[a]Standard deviation.

Table A-87. Mean strain ratio-tension data from the average of the right and left femoral artery segments from mongrel dogs

Strain ratio	Mean Tension (gm)
1.00	4.04 ± 1.37[a] (5)[b]
1.02	4.91 ± 1.43 (5)
1.04	5.89 ± 1.67 (5)
1.06	7.16 ± 1.99 (5)
1.08	8.70 ± 2.32 (5)
1.10	10.23 ± 2.68 (5)
1.12	12.21 ± 3.31 (5)
1.14	14.72 ± 4.25 (5)
1.16	17.64 ± 5.51 (5)
1.18	21.08 ± 7.19 (5)
1.20	25.69 ± 9.91 (5)
1.22	31.62 ± 13.48 (5)
1.24	31.79 ± 10.54 (4)
1.26	38.34 ± 13.05 (4)
1.28	47.43 ± 16.80 (4)
1.30	51.33 ± 18.72 (3)
1.32	50.63 ± 9.61 (2)
1.34	64.34 ± 9.30 (2)
1.36	
1.38	
1.40	

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-88. Slope of the strain ratio-tension graph (slope II) for the initial femoral artery segments from beagle dogs

Group	Tension (gm) 0	20	40	60	80	100
	Slope II (tension per 0.02 strain ratio increase) (gm)					
Control females						
01	.21	3.35	7.10	11.45	16.40	21.96
02	.26	3.98	9.21	15.96	24.24	34.03
04	.28	4.82	10.52	17.40	25.44	34.65
06	-.76	4.80	10.05	14.98	19.60	23.91
10	.47	5.52	11.20	17.51	24.44	32.00
Mean	.09 ±.49[a]	4.49 ±.84	9.62 ±2.48	15.46 ±2.48	22.02 ±3.87	29.31 ±5.94
Males						
22	-.05	4.42	9.67	15.70	22.52	30.12
23	.31	3.94	9.31	16.42	25.27	35.87
24	-.88	6.24	12.26	17.17	20.97	23.67
27	.03	4.00	9.56	16.70	25.43	35.74
28	.84	2.50	4.77	7.65	11.15	15.27
Mean	.05 ±.63	4.22 ±1.34	9.11 ±2.71	14.73 ±3.99	21.07 ±5.86	28.13 ±8.76
Overall control mean	.07 ±.53	4.36 ±1.07	9.36 ±2.11	15.09 ±3.16	21.55 ±4.71	28.72 ±7.08
Treatment females						
03	.30	5.08	11.63	19.93	30.00	41.83
05	-1.21	4.73	10.41	15.82	20.97	25.85
07	.04	4.82	10.28	16.41	23.21	30.69

[a]Standard deviation.

Table A-88. (Continued)

Group	Tension (gm) 0	20	40	60	80	100
	Slope II (tension per 0.02 strain ratio increase) (gm)					
08	-.33	3.46	8.25	14.05	20.86	28.66
09	.46	3.89	8.17	13.28	19.23	26.01
Mean	-.15 ±.66	4.40 ±.69	9.75 ±1.50	15.90 ±2.59	22.85 ±4.24	30.61 ±6.59
Males						
21	-.16	4.64	9.65	14.88	20.32	25.97
25	-.54	5.07	10.60	16.07	21.47	26.81
26	-.12	4.05	8.44	13.05	17.88	22.92
29	-.14	4.96	9.89	14.65	19.24	23.66
Mean	-.24 ±.20	4.68 ±.46	9.64 ±.90	14.66 ±1.24	19.73 ±1.53	24.84 ±1.85
Overall treatment mean	-.19 ±.49	4.52 ±.58	9.70 ±1.20	15.35 ±2.09	21.46 ±3.55	28.04 ±5.68

Table A-89. Slope of the strain ratio-tension graph (slope II) for the final artery segments from beagle dogs

Group	Tension (gm) 0	20	40	60	80	100
	Slope II (tension per 0.02 strain ratio increase) (gm)					
Control females						
01	.27	4.56	9.16	14.06	19.25	24.75
02	-1.01	4.86	9.96	14.26	17.78	20.52
04	-.06	5.56	10.82	15.73	20.29	24.50
06	.17	5.13	10.44	16.10	22.10	28.45
10	.43	4.66	9.19	14.01	19.14	24.56
Mean	-.04 ±.57[a]	4.95 ±.40	9.91 ±.74	14.83 ±1.00	19.71 ±1.61	24.56 ±2.81
Males						
22	.40	4.66	9.76	15.69	22.46	30.06
23	.37	4.13	9.49	16.47	25.04	35.23
24	1.90	5.63	10.14	15.42	21.47	28.30
27	.84	4.07	9.09	15.90	24.49	34.88
28	-.32	5.48	10.31	14.16	17.04	18.94
Mean	.64 ±.82	4.79 ±.73	9.76 ±.49	15.53 ±.86	22.10 ±3.18	29.48 ±6.62
Overall control mean	.30 ±.76	4.87 ±.56	9.84 ±.60	15.18 ±.95	20.91 ±2.69	27.02 ±5.45
Treatment females						
03	-[b]					
05	-.58	4.74	10.16	15.67	21.27	26.97

[a]Standard deviation.

[b]Dogs 03 and 09 died during refeeding I and their final femoral arteries were not tested.

Table A-89. (Continued)

Group	Tension (gm) 0	20	40	60	80	100
	Slope II (tension per 0.02 strain ratio increase) (gm)					
07	.00	5.27	10.63	16.10	21.67	27.34
08	-.37	5.32	10.55	15.32	19.63	23.47
09	-[b]					
Mean	-.32 ±.29	5.11 ±.32	10.45 ±.25	15.70 ±.39	20.86 ±1.08	25.93 ±2.14
Males						
21	-.06	4.71	9.64	14.72	19.95	25.34
25	-.82	5.77	11.75	17.10	21.83	25.94
26	.15	5.47	10.84	16.25	21.71	27.22
29	.22	4.81	9.60	14.59	19.78	25.17
Mean	-.13 ±.48	5.19 ±.51	10.46 ±1.04	15.66 ±1.22	20.82 ±1.10	25.92 ±.93
Overall treatment mean	-.21 ±.39	5.16 ±.41	10.45 ±.75	15.68 ±.89	20.83 ±1.00	25.92 ±1.40

Table A-90. Slope of the strain ratio-tension graph (slope II) for the femoral artery segments from mongrel dogs

Group	Side[a]	Tension (gm) 0	20	40	60	80	100
		Slope II (Tension per 0.02 strain ratio increase) (gm)					
Mongrels							
01	R	.42	3.20	7.78	14.15	22.32	32.27
02	R	-.28	3.50	7.90	12.90	18.52	24.76
03	R	-.04	5.30	13.09	23.34	36.04	51.19
04	R	.00	4.13	9.37	15.72	23.19	31.78
05	R	-.11	4.67	10.56	17.55	25.64	34.84
Mean		.00 ±.26[b]	4.16 ±.85	9.74 ±2.19	16.73 ±4.08	25.14 ±6.61	34.97 ±9.81
01	L	.38	3.16	7.71	14.03	22.12	31.98
02	L	.24	3.48	8.29	14.68	22.64	32.16
03	L	.00	3.66	9.95	18.87	30.42	44.61
04	L	-.14	5.85	12.26	19.11	26.38	34.09
05	L	-.24	4.62	10.05	16.07	22.65	29.81
Mean		.05 ±.26	4.15 ±1.09	9.65 ±1.78	16.55 ±2.35	24.84 ±3.55	34.53 ±5.84
Overall mongrel mean		.02 ±.22	4.16 ±.78	9.70 ±1.68	16.64 ±2.97	24.99 ±4.89	34.75 ±7.56

[a]R = right femoral; L = left femoral.

[b]Standard deviation.

Table A-91. Strain ratio-stress data for the initial segment of femoral artery from beagle dogs

Strain ratio	Control females					
	01	02	04	06	10	Mean
	Stress (gm/cm^2)					
1.00	243	339	369	314	426	338±68[a]
1.02	306	409	469	378	571	427±100
1.04	369	495	563	490	718	527±128
1.06	420	600	699	560	928	641±189
1.08	490	687	877	689	1154	780±250
1.10	577	845	1049	833	1442	949±322
1.12	658	985	1274	1062	1916	1179±468
1.14	760	1191	1587	1313	2439	1458±624
1.16	895	1435	2033	1625	3113	1820±830
1.18	1022	1743	2688	1953	4038	2289±1144
1.20	1201	2128	3300	2437		2267±866
1.22	1406	2580	4192	3175		2838±1164
1.24	1623	3289	5523	3909		3586±1612
1.26	1952	4181				3067±1576
1.28	2349					
1.30	2764					
1.32	3287					
1.34	3974					
1.36						
1.38						

[a]Standard deviation.

Table A-92. Strain ratio-stress data for the initial segment of femoral artery from beagle dogs

Strain ratio	Control males					
	22	23	24	27	28	Mean
	Stress (gm/cm^2)					
1.00	369	215	285	199	340	282±75[a]
1.02	465	261	356	251	401	347±92
1.04	545	303	465	316	468	419±105
1.06	672	367	584	374	543	508±134
1.08	800	441	786	417	621	613±182
1.10	975	516	1008	489	707	739±246
1.12	1190	613	1308	575	794	896±336
1.14	1478	696	1700	705	884	1093±466
1.16	1801	867	2283	788	982	1344±663
1.18	2239	1020	2906	916	1086	1634±891
1.20	2833	1240	3708	1129	1222	2026±1178
1.22	3577	1489		1335	1372	1943±1091
1.24		1842		1600	1523	1655±167
1.26		2313		1948	1682	1981±316
1.28		2847		2392	1890	2376±478
1.30		3555		2978	2117	2883±724
1.32		4668		3786	2427	3627±1129
1.34				4869	2739	3804±1506
1.36					3106	
1.38					3532	

[a]Standard deviation.

Table A-93. Strain ratio-stress data for the initial segment of femoral artery from beagle dogs

Strain ratio	Treatment females					
	03	05	07	08	09	Mean
	Stress (gm/cm^2)					
1.00	146	248	171	220	221	201±42[a]
1.02	188	306	214	250	281	248±48
1.04	233	381	280	283	352	306±60
1.06	312	448	356	320	410	369±59
1.08	368	536	430	359	502	439±79
1.10	476	638	512	416	588	526±89
1.12	593	756	647	493	704	639±102
1.14	722	906	780	557	828	758±131
1.16	887	1150	973	626	998	927±193
1.18	1115	1403	1213	728	1175	1127±248
1.20	1458	1740	1506	841	1416	1392±332
1.22	1898	2232	1885	997	1724	1747±458
1.24	2445	2883	2433	1159	2115	2207±646
1.26	3302	3603	3093	1327	2550	2775±896
1.28				1615	3158	2387±1091
1.30				1927		
1.32				2388		
1.34				2924		
1.36				3630		
1.38						

[a]Standard deviation.

Table A-94. Strain ratio-stress data for the initial segment of femoral artery from beagle dogs

Strain ratio	Treatment males				
	21	25	26	29	Mean
			Stress (gm/cm^2)		
1.00	339	365	281	317	326±36[a]
1.02	409	471	347	422	412±51
1.04	517	606	416	505	511±78
1.06	620	709	491	622	610±90
1.08	743	901	585	778	752±130
1.10	931	1092	705	940	917±159
1.12	1130	1387	818	1228	1141±240
1.14	1401	1724	979	1504	1402±313
1.16	1737	2129	1197	1827	1722±388
1.18	2186	2713	1445	2274	2154±526
1.20	2728	3523	1765	2860	2719±725
1.22	3362	4429	2129	3628	3387±953
1.24	4234		2540	4457	3744±1048
1.26			3148	5540	4344±1692
1.28			3831		
1.30					
1.32					
1.34					
1.36					
1.38					

[a]Standard deviation.

Table A-95. Strain ratio-stress data for the final segment of femoral artery from beagle dogs

Strain ratio	Control females					
	01	02	04	06	10	Mean
	Stress (gm/cm^2)					
1.00	377	334	218	162	307	280±88[a]
1.02	447	399	322	201	388	351±95
1.04	572	494	398	249	466	436±122
1.06	676	602	539	313	601	546±139
1.08	867	730	648	402	727	675±172
1.10	1071	869	794	494	911	828±212
1.12	1305	1082	1029	627	1089	1026±247
1.14	1592	1352	1309	780	1370	1281±301
1.16	1953	1665	1632	991	1690	1586±356
1.18	2419	2031	2059	1270	2084	1973±423
1.20	2990	2569	2694	1614	2543	2482±517
1.22		3255	3489	2074	3158	2994±629
1.24		3974	4302	2549		3608±932
1.26			5446	3219		4330±1575
1.28				4122		
1.30						
1.32						
1.34						
1.36						
1.38						

[a]Standard deviation.

Table A-96. Strain ratio-stress data for the final segment of femoral artery from beagle dogs

Strain ratio	Control males					
	22	23	24	27	28	Mean
	Stress (gm/cm^2)					
1.00	280	176	470	269	243	288±110[a]
1.02	372	209	595	358	323	372±140
1.04	434	279	760	426	411	462±178
1.06	534	322	965	528	530	576±236
1.08	684	374	1244	614	635	710±322
1.10	829	453	1519	778	815	879±390
1.12	1013	562	1914	908	1037	1087±500
1.14	1255	659	2422	1100	1302	1348±652
1.16	1564	793		1318	1626	1325±379
1.18	1970	959		1631	2097	1664±510
1.20	2443	1186		2015	2654	2075±649
1.22	3044	1443		2436	3310	2558±829
1.24	3883	1800		3048	4053	3196±1029
1.26		2206		3901	4929	3679±1375
1.28		2757				
1.30		3563				
1.32						
1.34						
1.36						
1.38						

[a]Standard deviation.

Table A-97. Strain ratio-stress data for the final segment of femoral artery from beagle dogs

Strain ratio	Treatment females					
	03	05	07	08	09	Mean
	Stress (gm/cm^2)					
1.00	-[a]	192	245	86	-[a]	174±81[b]
1.02		236	311	110		219±102
1.04		301	401	134		279±135
1.06		350	490	165		335±163
1.08		420	639	205		421±217
1.10		512	784	260		518±262
1.12		615	971	319		635±326
1.14		730	1244	380		785±434
1.16		887	1615	502		1001±565
1.18		1111	2013	583		1236±723
1.20		1351	2549	697		1533±939
1.22		1656	3241	880		1926±1203
1.24		2063		1096		1580±683
1.26		2711		1369		2040±949
1.28		3375		1679		2527±1199
1.30				2101		
1.32				2657		
1.34				3495		
1.36				4349		
1.38				5414		

[a]Dogs 03 and 09 died during refeeding I and their final femoral arteries were not tested.

[b]Standard deviation.

Table A-98. Strain ratio-stress data from the final segment of femoral artery from beagle dogs

Strain ratio	Treatment males				
	21	25	26	29	Mean
			Stress (gm/cm^2)		
1.00	311	366	352	240	317±57[a]
1.02	403	478	458	294	408±82
1.04	481	602	579	360	506±110
1.06	590	750	769	438	637±155
1.08	730	970	955	573	807±191
1.10	896	1260	1175	694	1006±260
1.12	1118	1594	1550	895	1290±340
1.14	1362	2040	1964	1118	1621±452
1.16	1685	2650	2451	1335	2030±623
1.18	2103	3452	3166	1669	2597±848
1.20	2634	4379	4009	2144	3292±1072
1.22	3274			2634	2954±452
1.24				3211	
1.26				4037	
1.28					
1.30					
1.32					
1.34					
1.36					
1.38					

[a]Standard deviation.

Table A-99. Group means for strain ratio-stress data from the initial femoral artery segments from beagle dogs

Strain ratio	Overall control mean	Overall treatment mean
	Stress (gm/cm^2)	
1.00	310 ± 73[a](10)[b]	256 ± 75 (9)
1.02	387 ± 100 (10)	321 ± 98 (9)
1.04	473 ± 124 (10)	397 ± 126 (9)
1.06	575 ± 170 (10)	476 ± 145 (9)
1.08	696 ± 224 (10)	578 ± 192 (9)
1.10	844 ± 292 (10)	700 ± 236 (9)
1.12	1037 ± 412 (10)	862 ± 311 (9)
1.14	1275 ± 554 (10)	1044 ± 400 (9)
1.16	1587 ± 752 (10)	1280 ± 501 (9)
1.18	1961 ± 1026 (10)	1584 ± 654 (9)
1.20	2133 ± 996 (9)	1982 ± 861 (9)
1.22	2391 ± 1149 (8)	2476 ± 1092 (9)
1.24	2759 ± 1541 (7)	2783 ± 1089 (8)
1.26	2415 ± 1012 (5)	3223 ± 1264 (7)
1.28	2370 ± 391 (4)	2868 ± 1136 (3)
1.30	2854 ± 594 (4)	
1.32	3542 ± 937 (4)	
1.34	3860 ± 1070 (3)	
1.36		
1.38		

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-100. Group means for strain ratio-stress data from the final femoral artery segments from beagle dogs

Strain ratio	Overall control mean	Overall treatment mean
	Stress (gm/cm^2)	
1.00	284 ± 94[a](10)[b]	256 ± 98 (7)
1.02	362 ± 114 (10)	327 ± 131 (7)
1.04	449 ± 144 (10)	408 ± 164 (7)
1.06	561 ± 183 (10)	507 ± 216 (7)
1.08	692 ± 244 (10)	642 ± 276 (7)
1.10	853 ± 297 (10)	797 ± 353 (7)
1.12	1056 ± 373 (10)	1009 ± 464 (7)
1.14	1314 ± 480 (10)	1263 ± 604 (7)
1.16	1470 ± 369 (9)	1589 ± 776 (7)
1.18	1836 ± 462 (9)	2014 ± 1032 (7)
1.20	2301 ± 581 (9)	2538 ± 1324 (7)
1.22	2776 ± 720 (8)	2337 ± 1045 (5)
1.24	3373 ± 932 (7)	2123 ± 1058 (3)
1.26	3940 ± 1301 (5)	2706 ± 1334 (3)
1.28	3440 ± 965 (2)	2527 ± 1199 (2)
1.30		
1.32		
1.34		
1.36		
1.38		

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-101. Strain ratio-stress data for the right femoral artery from mongrel dogs

Strain ratio	Mongrel dogs 01	02	03	04	05	Mean
	Stress (gm/cm^2)					
1.00	136	243	92	87	127	137±63[a]
1.02	170	279	115	111	178	171±68
1.04	200	328	138	144	221	206±77
1.06	242	389	180	174	259	249±87
1.08	294	464	231	203	336	306±103
1.10	327	523	293	235	388	353±110
1.12	380	610	343	283	454	414±126
1.14	434	708	439	334	550	493±142
1.16	506	825	495	386	659	574±170
1.18	570	968	602	457	778	675±200
1.20	669	1160	715	535	945	805±248
1.22	774	1371	893	639	1150	965±294
1.24	883	1614	1072	765	1441	1155±362
1.26	1036	1960	1355	936	1760	1409±445
1.28	1226	2431	1756	1097	2229	1748±590
1.30	1477	2954	2352	1335	2860	2196±758
1.32	1762		3070	1689	3672	2548±982
1.34	2165		4212	2083	4789	3312±1392
1.36	2695			2587		2641±76
1.38				3288		
1.40						

[a]Standard deviation.

Table A-102. Strain ratio-stress data for the left femoral artery from mongrel dogs

Strain ratio	Mongrel dogs					
	01	02	03	04	05	Mean
	Stress (gm/cm^2)					
1.00	163	167	134	186	176	165±19[a]
1.02	187	194	183	231	206	200±19
1.04	216	232	215	282	249	239±28
1.06	261	279	250	371	301	292±48
1.08	309	319	287	474	375	353±75
1.10	357	390	333	602	431	422±107
1.12	418	438	380	785	528	510±163
1.14	475	510	431	1052	643	622±253
1.16	550	607	509	1350	780	759±346
1.18	630	688	570	1727	972	917±478
1.20	731	805	641	2294	1183	1131±682
1.22	835	959	736	3029	1520	1416±952
1.24	970	1133	858		1922	1221±481
1.26	1149	1346	980		2365	1460±622
1.28	1371	1619	1158		3035	1796±847
1.30	1623	1981	1363			1656±310
1.32	1981	2431	1628			2013±403
1.34	2456	3058	2008			2508±527
1.36	3112		2534			2823±409
1.38			3357			
1.40			4487			

[a]Standard deviation.

Table A-103. Mean strain ratio-stress data from the average of the right and left femoral artery segments from mongrel dogs

Strain ratio	Mean Stress (gm/cm^2)
1.00	151 ± 34[a] (5)[b]
1.02	185 ± 33 (5)
1.04	222 ± 38 (5)
1.06	270 ± 44 (5)
1.08	329 ± 51 (5)
1.10	388 ± 59 (5)
1.12	462 ± 77 (5)
1.14	557 ± 110 (5)
1.16	667 ± 152 (5)
1.18	796 ± 211 (5)
1.20	968 ± 302 (5)
1.22	1190 ± 426 (5)
1.24	1236 ± 359 (4)
1.26	1494 ± 453 (4)
1.28	1853 ± 605 (4)
1.30	1958 ± 467 (3)
1.32	2110 ± 338 (2)
1.34	2711 ± 565 (2)
1.36	
1.38	
1.40	

[a]Standard deviation.

[b]The numbers in parentheses are the number of observations composing the mean.

Table A-104. Slope of the strain ratio-stress graph (slope III) for the initial femoral artery segments from beagle dogs

Group	Stress (gm/cm^2) 0	500	1000	1500	2000	2500
	Slope III (stress per 0.02 strain ratio increase) (gm/cm^2)					
Control females						
01	9	84	169	263	367	479
02	13	97	199	320	461	620
04	16	121	239	369	512	668
06	-33	107	244	375	503	626
10	23	146	276	414	561	716
Mean	6±22[a]	111±24	225±42	349±58	481±73	622±88
Males						
22	-2	105	224	354	495	648
23	15	95	198	323	471	641
24	-36	147	314	463	596	712
27	2	90	195	318	458	615
28	32	71	120	179	248	327
Mean	2±25	102±28	210±70	327±102	454±127	588±151
Overall control mean	4±22	106±25	218±55	338±79	467±99	605±118
Treatment females						
03	11	124	265	436	637	868
05	-45	104	250	390	527	659
07	2	118	247	387	539	702
08	-14	75	179	298	431	579
09	16	99	197	310	439	583

[a]Standard deviation.

Table A-104. (Continued)

Group	Stress (gm/cm^2) 0	500	1000	1500	2000	2500
	Slope III (stress per 0.02 strain ratio increase) (gm/cm^2)					
Mean	-6±25	104±19	228±37	364±59	515±84	678±118
Males						
21	-7	112	234	359	487	619
25	-26	115	254	393	531	668
26	-5	98	204	314	427	543
29	-8	121	247	372	495	617
Mean	-11±10	111±10	235±22	360±33	485±43	612±51
Overall treatment mean	-8±19	107±15	231±30	362±46	502±67	648±96

Table A-105. Slope of the strain ratio-stress graph (slope III) for the final femoral artery segments from beagle dogs

Group	Stress (gm/cm^2) 0	500	1000	1500	2000	2500
	Slope III (stress per 0.02 strain ratio increase) (gm/cm^2)					
Control females						
01	9	116	227	344	466	594
02	-48	103	244	376	496	607
04	-4	139	279	415	547	676
06	8	130	257	389	526	668
10	15	120	230	345	465	591
Mean	-4±26[a]	122±14	248±21	374±30	500±36	627±41
Males						
22	18	120	234	360	497	647
23	15	101	211	345	503	685
24	50	143	254	383	531	696
27	39	109	204	323	466	633
28	-16	135	274	402	518	622
Mean	21±26	122±17	236±29	363±31	503±24	657±32
Overall control mean	9±28	122±15	242±25	368±29	502±29	642±38
Treatment females						
03	-[b]	-	-	-	-	-
05	-24	108	242	378	515	653
07	0	131	264	399	535	673

[a]Standard deviation.

[b]Dogs 03 and 09 died during refeeding I and their final femoral arteries were not tested.

Table A-105. (Continued)

Group	Stress (gm/cm^2) 0	500	1000	1500	2000	2500
	Slope III (stress per 0.02 strain ratio increase) (gm/cm^2)					
08	-22	124	265	400	531	657
09	-[b]	-	-	-	-	-
Mean	-15±13	121±12	257±13	392±13	527±11	661±11
Males						
21	-2	116	238	361	488	616
25	-37	131	291	442	585	718
26	7	140	273	407	542	677
29	10	123	240	359	481	606
Mean	-6±22	128±10	260±26	392±40	524±49	655±53
Overall treatment mean	-10±18	125±10	259±20	392±29	525±35	657±38

Table A-106. Slope of the strain ratio-stress graph (slope III) for the femoral artery segments from mongrel dogs

Group	Side	Stress (gm/cm^2) 0	500	1000	1500	2000	2500
		Slope III (stress per 0.02 strain ratio increase) (gm/cm^2)					
Mongrels							
01	R[a]	14	78	176	308	474	673
02	R	-9	84	188	303	431	570
03	R	-2	114	257	426	622	845
04	R	0	98	213	346	496	664
05	R	-6	107	232	371	523	688
Mean		-6±9[b]	96±15	213±33	351±51	509±72	688±99
01	L	12	77	176	309	477	680
02	L	9	84	185	314	469	651
03	L	0	76	186	331	511	725
04	L	-5	143	299	463	635	814
05	L	-9	111	240	379	529	688
Mean		1±9	98±29	217±52	359±64	524±66	712±63
Overall mongrel mean		0±7	97±18	215±37	355±44	517±53	700±66

[a]R = right femoral; L = left femoral.

[b]Standard deviation.

Table A-107. Body weights and cardiac ventricular weights obtained at post mortem on the control and treatment groups of beagles

Dog	Body weight (kg)	Total ventricular weight (gm)	Right ventricle		Left ventricle		Heart:body weight ratio (gm/kg)
			Weight (gm)	Percent (%)	Weight (gm)	Percent (%)	
Control females							
01	10.5	85	21	24.7	64	75.3	8.10
02	10.2	67	16	23.9	51	76.1	6.57
04	10.9	74	17	23.0	57	77.0	6.79
06	10.5	80	19	23.8	61	76.2	7.62
10	11.8	75	17	22.7	58	77.3	6.36
Mean	10.8±0.7[a]	76±7	18±2	23.5±0.7	58±4	76.4±0.9	7.09±0.38
Males							
22	13.6	102	24	23.5	78	76.5	7.50
23	14.1	98	24	24.5	74	75.5	6.95
24	14.5	100	26	26.0	74	74.0	6.90
27	14.5	97	22	22.7	75	77.3	6.69
28	12.7	89	20	22.5	69	77.5	7.01

[a]Mean ± standard deviation.

Table A-107. (Continued)

Dog	Body weight (kg)	Total ventricular weight (gm)	Right ventricle Weight (gm)	Right ventricle Percent (%)	Left ventricle Weight (gm)	Left ventricle Percent (%)	Heart:body weight ratio (gm/kg)
Mean	13.9±0.9[b**]	97±4[b**]	23±2[b**]	23.8±1.3	74 2[b**]	76.2±1.3	7.01±0.29
Overall control mean	12.3±1.9[c**]	87±13[c*]	21±[c**]	23.7±0.9	66±9[c*]	76.3±0.9	7.05±0.54
Treatment females							
03	-[d]	-	-	-	-	-	-
05	9.1	70	16	22.8	54	77.2	7.69
07	7.3	53	11	20.8	42	79.2	7.26
08	10.9	61	13	21.3	48	78.7	5.60
09	-[d]	-	-	-	-	-	-

[b]Significantly different from the control females.

[c]Significantly different from the overall treatment mean.

[d]Dogs 03 and 09 of the treatment group died during the refeeding I regimen.

**Difference was significant at P<0.01.

*Difference was significant at P<0.05.

Table A-107. (Continued)

Dog	Body weight (kg)	Total ventricular weight (gm)	Right ventricle		Left ventricle		Heart:body weight ratio (gm/kg)
			Weight (gm)	Percent (%)	Weight (gm)	Percent (%)	
Mean	9.1±1.7	61±9	13±2	21.6±1.0	48±5	78.4±1.0	6.85±1.11
Males							
21	6.4	54	14	25.9	40	74.1	8.44
25	10.9	73	17	23.3	56	76.7	6.70
26	10.9	81	17	21.0	64	79.0	7.43
29	11.6	87	20	23.0	67	77.0	7.50
Mean	10.0±2.8	74±14	17±2	23.3±2	57±12	76.7±2.0	7.52±0.72
Overall treatment mean	9.6±2.1	68±12	15±3	22.6±1.8	53±9	77.4±1.8	7.23±0.90