CARDIOVASCULAR PARAMETERS AND STATIC ELASTIC MODULI OF THE TURKEY AORTA

Thesis for the Degree of Ph. D.
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Elwood William Speckmann, Jr.
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Elwood William Speckmann, Jr.

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Robert H. Grogan
Major professor

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CARDIOVASCULAR PARAMETERS AND STATIC ELASTIC MODULI OF THE TURKEY AORTA

by

Elwood William Speckmann, Jr.

AN ABSTRACT

Submitted to
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Department of Poultry Science

1962

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ABSTRACT

CARDIOVASCULAR PARAMETERS AND STATIC ELASTIC MODULI OF THE TURKEY AORTA

by Elwood William Speckmann, Jr.

Under commercial rearing conditions, turkeys are spontaneously affected by a fatal condition known as aortic rupture. Investigations in this laboratory have indicated that the cause of the aortic rupture syndrome may be of a physiological nature rather than of a nutritional deficiency. Cardiac and circulatory dynamics were recorded under normal conditions and following epinephrine injection. The association of atherosclerosis with plasma cholesterol and aortic elasticity and the relationship of these values to the cardiovascular parameters was evaluated.

Plasma cholesterol level was found to increase with age in both male and female Broad Breasted Bronze (BBB) turkeys from 4 weeks of age to approximately 16 weeks of age after which age the plasma cholesterol level plateaued. The adult plasma cholesterol level was determined as 249 ± 8 mg percent for commercially purchased poults and 344 ± 13 mg percent for Experiment Station stock. There was no sexual difference in plasma cholesterol levels at any age.

In these experiments atherosclerosis began early in age, for by 8 weeks of age all turkeys had either microscopic or macroscopic atherosclerotic lesions. The males exhibited a greater atherosclerotic severity than did the females. Reserpine administration at the level of 0.1 ppm and 0.2 ppm in the feed did not offer any protection against

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the increase in plasma cholesterol or the severity of atherosclerosis with advancing age.

The cardiac output of untreated mature male BBB turkeys was determined by an isotope dilution technique using radioactive-phosphorus (P^{32}) as the indicator. Common carotid and popliteal arterial blood pressures were measured directly and were recorded simultaneously with the cardiac output determinations pre- and post-epinephrine injection by means of two strain guages connected to a recording polygraph.

From the cardiovascular measurements systemic resistence was calculated. The mean cardiac output of mature male BBB turkeys was 231 ± 18 ml per $Kg^{0.734}$ per minute. The cardiac parameters were not affected by strain but were significantly influenced by atherosclerotic severity. Turkeys with a higher incidence of atherosclerosis had a greater minute volume, cardiac output and stroke volume than turkeys with little or no atherosclerosis. The injection of 0.5 ml of 1/25,000 epinephrine caused a negative cardiovascular response; minute volume, cardiac output and stroke volume decreased.

Blood pressures increased with advancing age in both male and female BBB turkeys. Several of the hemodynamic parameters were influenced both by strain and atherosclerotic severity. The M.S.U. strain of turkey had a significantly higher systolic blood pressure than did the commercial strain of turkeys with the cross between the two strains of turkeys falling in between. The group with the highest atherosclerotic score also had the highest systolic blood pressure. The popliteal arterial blood pressures were consistently lower than their common carotid artery counterparts and were not influenced as

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much by strain and atherosclerotic severity. The injection of epinephrine caused a pronounced elevation of blood pressure. This was accompanied by a negative cardiovascular response and a concomitant increase in systemic resistance which elevated blood pressure. Reserpine significantly reduced blood pressure whereas BAPN and vitamin C had no affect.

A method for the determination of the static modulus of elasticity for the thoracic and abdominal aorta and an equation rearranged for elastance calculations as a function of change in volume were described. The aortic segments were most distensible in the physiological blood pressure range, the pressure-volume curves being sigmoid in nature. The vessel segments also exhibited hysteresis. The thoracic aortas were much more distensible than the abdominal aortas; the elastances of untreated male BBB turkeys being in the neighborhood of 3.28 ± 0.11 and 13.95 ± 0.24 dynes/cm^2 X 10^5 respectively in the physiological blood pressure range. This difference becomes more pronounced at higher pressures.

Partial correlation coefficients of cardiac, hemodynamic and elasticity parameters were calculated to evaluate their interrelationships.

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A THESIS

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1962

This dissertation is dedicated to my father, whose last wish has been fulfilled and to my mother, whose sacrifices and encouragement made this endeavor possible.

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and to Dr. R. J. Evans, Department of Biochemistry, for his constructive review of the thesis.

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Elwood W. Speckmann, Jr.

Candidate for the degree of

Doctor of Philosophy

DISSERTATION:

Cardiovascular Parameters and Static Elastic Moduli of the Turkey Aorta

OUTLINE OF STUDIES:

Major area of study: Poultry Science (Avian Physiology)

Supporting areas of study: Biochemistry, Nutrition

BIOGRAPHICAL ITEMS:

Born: January 10, 1936, Brooklyn, New York

Undergraduate studies: Rutgers University, New Jersey 1953-1957

Graduate studies: Michigan State University, Michigan 1957-1962

EXPERIENCE:

Graduate Research Assistant, Michigan State University 1957-1960

Dr. Salsbury Graduate Research Fellow, Michigan State University, 1960-1962

MEMBER:

Poultry Science Association

Society of Sigma Xi

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INTRODUCTION

Aortic rupture was first reported in turkeys in 1952. This condition probably existed for some time prior to 1952 but had gone unreported and/or unnoticed. Today, the turkey grower and breeder are faced with narrowed margins and reduced income. Therefore, each producer must scrutinize his losses from disease and poor management practices.

Aortic rupture has been of utmost concern to turkey growers and breeders because it is occurring under various systems of management in widely scattered portions of the United States, Canada and England. There are apparently no external symptoms to this malady prior to death yet mortality may be as high as 20 percent. Males of the larger varieties (Broad Breasted Bronze and Broad Breasted White) are predominantly affected and die usually between 8 - 24 weeks of age, representing a significant economic loss to the turkey raiser. Reports from turkey raisers indicate the highest incidence of death occurs following a "stress". In this dissertation, stress denotes a physiological imbalance which causes a release of epinephrine. Jumping off a roost, fighting, sexual play, undue medication, sudden excitement, or other such stresses which are most frequent in growing male birds reaching sexual maturity, are probable contributing agents to aortic rupture.

Aortic rupture has been reported to occur in association with atherosclerosis. The relationship of atherosclerosis to dietary patterns and lipid metabolism, particularly cholesterol, is well

known. Primary investigation of the aortic rupture syndrome in this country, therefore, has been from the nutritional aspect. Considerable literature exists as to the effects of various dietary regimes on aortic rupture artificially produced with beta-aminopropionitrile, few of which were beneficial.

The possible association of stress with aortic rupture, coupled with the high blood pressure reported for the male turkey suggest the cause of this condition to be of a physiological nature. The successful use of reserpine, a hypotensive tranquilizer, in stopping losses due to aortic rupture further documents this assumption. The primary objective of the research reported in this dissertation was to investigate the basic cardiovascular and hemodynamic parameters which may contribute to the cause of aortic rupture in turkeys. In addition, the elasticity of the thoracic and abdominal aortas was measured to ascertain their distensibility under various physiological conditions.

REVIEW OF LITERATURE

Problem of Aortic Rupture in Turkeys

The incidence of aortic rupture in turkeys was first reported in the United States in 1952 (Durrell, et al., 1952). However, some growers have observed this condition since as early as 1940 (Waibel, 1960). More recently, aortic rupture has been reported in Brown Leghorn hens (Siller, 1962a; 1962b; 1962c). Durrell et al. (1952) reported the malady to occur under various systems of management in widely scattered areas of the United States. They found a wide variation in age incidence, with the condition occurring generally in growing male turkeys between the ages of 8 - 24 weeks, of the Broad Breasted Bronze (BBB) or Broad Breasted White (BBW) varieties. Aortic rupture is not a condition of poor health as it is generally observed in the largest males that are experiencing rapid body weight gains and is, therefore, a significant economic loss to the turkey raiser.

Morrison (1960) indicates that aortic rupture occurs almost every month of the year but is most prevalent from April to October, the time when the largest number of turkeys are being raised. Although aortic rupture usually affects more mature birds, it has been reported by Morrison (1960) to occur as early as 5 weeks of age. He also reported that the condition occurs in both sexes but is more prevalent among male turkeys. Losses from aortic rupture are generally under 5 percent; however, losses as high as 20 percent have been reported (Waibel, 1960). A survey reported by Ringer (1959) indicated that the incidence of death frequently occurred early in the morning when the bird is jumping off a roost or disturbed by the producer.

Possibly other stresses such as fighting, sexual play, undue medication, or moving which are most frequent in growing male birds reaching sexual maturity, are contributing agents to aortic rupture.

The aortic rupture syndrome is characterized by a rupture of the abdominal aorta in the interrenal area resulting in a fatal hemorrhage. McSherry et al. (1954), described the degenerative changes of the aorta in detail and showed it to be precipitated by a dissecting aneurysm. Gottlieb and Lalich (1954), Carnaghan (1955) and Gibson and deGruchy (1955) reported that this syndrome is not only precipitated by a dissecting aneurysm, but occurs in association with spontaneous atherosclerotic plaques. Siller (1962b), however, found aortic rupture with and without atherosclerosis in chickens. Although the exact etiology of aortic rupture remains obscure, the foregoing suggests that a general weakening of the arterial wall may be involved in the condition (Barnett, 1960; Ringer, 1962).

The association between plasma cholesterol and atherosclerosis on one hand and plasma cholesterol and dietary regime on the other is well known (Katz and Stamler, 1953; Kritchevsky, 1958; 1962). Therefore, research on the aortic rupture syndrome in the United States has been primarily from the nutritional standpoint. Waibel (1960) observed blood clots in the body cavity of turkeys which had died from aortic rupture. This suggested that the blood clotting mechanism was involved in aortic rupture. Barnett et al. (1958) reported that anticoagulants added to a beta-aminopropionitrile (BAPN)-containing diet increased, whereas, vitamin K added to this diet reduced, mortality due to aortic rupture. Beta-aminopropionitrile, originally isolated as the principle in sweet pea seeds that

caused lathyrism, is a toxic substance used to produce aortic rupture (Barnett et al. 1957). It will be explained more completely in subsequent passages.

Van Itallie (1957) reviewed work which indicated that atherosclerosis in man is associated with a high level of dietary fat. The incidence of dissecting aneurysms in turkeys has increased in recent years. Also during the past few years the caloric content of the diet has been increased, often by the addition of fat (Barnett and Morgan, 1959). Since Gottlieb and Lalich (1954), Carnaghan (1955) and Gibson and deGruchy (1955) associated atherosclerosis with aortic rupture, the emphasis of nutritional studies switched to fat supplementation. Pritchard et al. (1958) reported that diets high in fat and protein increased the incidence of dissecting aneurysms. Barnett and Morgan (1959) reported that mortality was almost doubled in chicks when 20 percent fat was isonitrogenously added to the diet. This increase in mortality occurred in spite of an approximate 20 percent reduction in BAPN intake on the high energy diet. Waibel and Pomeroy (1959) failed to confirm these results in turkeys; however, they reported that dietary fish meal hastened the appearance of BAPN-induced mortality. Lalich et al. (1957) reported that they could not prevent aortic rupture in turkeys by feeding a high protein (28%) ration in association with BAPN-HCl although rats exhibited a significant decrease in aortic rupture when fed 20 percent protein diet plus BAPN. Stamler et al. (1958), however, observed that high-protein diets appear to protect chicks against atherosclerosis induced by cholesterol feeding. Recently McDonald et al. (1962) reported that the amino acids - L - lysine and L - cystine - and the related compound taurine potentiated

BAPN-induced dissecting aneurysms in turkeys. These amino acids, however, occur in most all proteins and are essential to birds.

Drugs such as nitrofurazone and semicarbazide hydrochloride have been shown to potentiate BAPN-induced aortic rupture (Roy et al. 1960). Work with vitamins by Barnett, as cited by Carlson (1959), showed that vitamins B_6 and B_{12} and fats increased the incidence of aortic rupture. Increasing the vitamin level or lowering protein did not stop losses from aortic rupture (Morrison, 1960). Recently, Thornton (1960) reported that the addition of ascorbic acid at levels as high as 100 mg/lb feed did not improve BAPN-induced aortic rupture in turkeys. Aortic rupture resulting from copper deficiency has been reported in chicks (O'Dell et al., 1961) and in swine (Carnes et al., 1961). Copper deficiency apparently results in a derangement of connective tissue as extensive dissecting aneurysms were readily present in both studies. The relationship of copper to aortic rupture in turkeys is not yet known.

The occurrence of aortic rupture under diverse dietary regimes and the failure of the nutritionists to prevent or stop aortic rupture in the field support the concept that nutrition, other than its association with cholesterol and atherosclerosis, has very little to do with the aortic rupture syndrome. The association of stress with aortic rupture has prompted this author to attack the problem from a physiological standpoint.

Atherosclerosis - Plasma Cholesterol - Hemodynamic Complex

Atherosclerosis

Atherosclerosis is a distinct entity -- one among the arteriosclerosis and by far the most important (Stamler, 1962). It is a syndrome with different pathogenic and etiological mechanisms, yet it is interwoven with hypertension. Atherosclerosis is the chief pathologic lesion in coronary heart disease in humans. It is a disease, and not a manifestation of senescence, and therefore is preventable and to a certain point curable. Further, atherosclerosis is a metabolic disease in which altered cholesterol-lipid-lipoprotein metabolism plays a critical and decisive role (Kritchevsky, 1962). Elevated levels of circulating serum cholesterol-lipid-lipoprotein are cardinal signs of the abnormality and constitute the metabolic prerequisites for atherogenesis in most persons afflicted during middle age (Albrink et al., 1961). The pathological hall-mark of atherosclerosis is the lipid-and cholesterol-containing, focal intimal plaque (Katz and Stamler, 1953). Increased blood pressure is also believed to be involved significantly in the pathogenesis of atherosclerosis in man (Anitschkow, 1933; Rosenthal, 1934; Moschcowitz, 1950; Blumenthal, 1956c; Burch and Phillips, 1960; and Stamler, 1962) and in aves (Weiss and Fisher, 1959). Many theories have arisen concerning the genesis of atherosclerosis (Blumenthal 1956a; 1956b; 1956c).

Of the species commercially used as laboratory animals, spontaneous atherosclerosis has been reported to occur in dogs (Lindsay et al. 1952), cats (Lindsay and Chaikoff, 1955), rabbits (Bragdon, 1952), rats (Humphries, 1957), chickens (Dauber, 1944), turkeys (Carnaghan, 1955), pigeons (Lofland and Clarkson, 1959) and baboons (Gillman and Gilbert, 1957). Of these animals the atherosclerotic lesions of aves

most closely resemble that of man (Dauber, 1944). The normal spontaneous development of avian arteriosclerosis (Fox, 1933; Dauber, 1944; Weiss, 1959), its similarity to human atherosclerosis (Dauber, 1944), and the ease with which cholesterol can induce this condition (Katz and Stamler, 1953; Kritchevsky, 1958) have precipitated another wave of nutritional research designed to produce or control atherosclerosis by altering the plasma cholesterol level. It is not the scope of this dissertation to review the multiferous procedures for producing hypercholesterolemia and atherosclerosis. Excellent reviews on the relationship between nutrition and atherosclerosis are reported by Katz and Stamler (1953) and Katz et al. (1958). More specific experiments in birds are reported by Kestern et al. (1936), Stamler and Katz (1950), Stamler et al. (1957, 1958, 1959), Fisher et al. (1959, 1960), Leveille et al. (1960) and Siller and Bolton (1961). Needless to say, the type of dietary fat and amount of cholesterol influences blood lipids as well as severity of atherosclerosis. Leveille and Fisher (1958) suggested that if fowl have an adequate protein intake they can metabolize excess dietary cholesterol. Stamler et al. (1958) confirmed these results. Wollenberger and Kossler (1960) reported that caged chickens had a higher incidence of aortic and coronary atherosclerotic lesions than did free birds allowed to exercise. Exercise, or rather metabolic rate, may have considerable influence on plasma cholesterol level. When desiccated thyroid was fed to chicks it significantly decreased the incidence and degree of diethylstilbestrol-induced atherosclerosis although it was without sustained effect upon plasma and tissue lipids (Stamler et al., 1950). Thyroxine and thiouracil in small doses were reported to aggravate spontaneous atherosclerosis in cockerels

(Perttala, 1961). Perttala (1961) has an excellent review on the role of the thyroid in atherosclerosis.

Although dietary or hormonal induced avian atherosclerosis resembles the spontaneously occurring malady in both chickens and man, there are important differences in distribution and histological structure between spontaneous and experimental lesions. The spontaneous lesion is primarily fibrotic and tends to localize in the muscular abdominal aorta; whereas, the experimental lesion is characterized initially by lipid deposition, mainly in the elastic thoracic aorta (Dauber and Katz, 1942; 1943; Weiss, 1959). Fisher et al. (1959), however, induced atherosclerotic lesions in the abdominal aorta of chickens that were similar to spontaneous lesions. Since aortic rupture in turkeys concerns the abdominal aorta, spontaneous lesions, or lesions which can be induced in this area of the aorta have the greatest significance.

Lofland and Clarkson (1959) studied spontaneous atherosclerosis in pigeons. These workers found Racing Homers and Show Racers to be resistant to atherosclerosis while the White Carneau and Silver King had a high incidence of spontaneous atheromatous lesions in the aorta. The level of serum cholesterol and phospholipid and the cholesterol: phospholipid ratio, however, were unrelated to the incidence and severity of atherosclerosis. They, therefore, attributed the susceptibility to atherosclerosis to be genetically determined. This concept was also advanced by Prichard et al. (1962) in a more recent paper. In pigeons, there is no sex difference in blood pressure (Ringer et al. 1955) or in susceptibility to atherosclerosis (Lofland and Clarkson, 1960; Prichard et al., 1962); whereas, in chickens (Pick et al., 1952c; Katz and Stamler,

1953; Weiss, 1959) and turkeys (Ringer and Rood, 1959) the male has the higher blood pressure and is more prone to atherogenesis after puberty (Speckmann and Ringer, 1962).

Estrogens of the female pigeon apparently have no prophylatic value against the severity or incidence of spontaneous atherosclerosis in this species (Lofland and Clarkson, 1960; Prichard et al., 1962). The prophylatic effect of estrogens in cholesterol-induced-coronary atherosclerosis has been reported for chickens but it apparently does not apply for the thoracic or abdominal sectors of the aorta, in fact, in these latter areas, estrogen may actually potentiate atherosclerosis (Pick et al., 1952a; 1952b; Stamler et al., 1954; Peck et al., 1961). For further information on the role of hormones in atherosclerosis, an excellent review is given by Pincus (1959).

Cholesterol

The involvement of an altered cholesterol metabolism further complicates the study of the atherogenic syndrome. Excellent reviews on the role of cholesterol in atherosclerosis are given by Katz and Stamler (1953) and Kritchevsky (1958).

A thorough investigation into the sterol composition of atherosclerotic aortas showed that cholesterol was the major constituent (Hardegger et al., 1943). The aortic cholesterol esters are deposited from the plasma resulting in the arterial wall having a similar composition to the plasma (Weinhouse and Hirsch, 1940; Bottcher et al., 1960). Exogenous cholesterol also becomes deposited in the atheroma of cholesterol-fed rabbits (Biggs and Kritchevsky, 1951). The arterial wall of some species is capable of snythesizing cholesterol (Chernick

et al., 1949; Siperstein et al., 1951; Azarnoff, 1958; Field, 1960). This has recently been reported for chickens (Dayton, 1959; 1961). Dayton (1961) reported a decline in the rate of cholesterol synthesis during maturation of the aorta. Perhaps cholesterol synthesis is concomitant with growth rate, being more rapid in the growing chick. In chickens, the age period rather than age itself seems to be a more significant factor in atherogenesis. A report by Rodbard et al., (1951) showed that hypercholesterolemia occurred in chicks during embryonic development, yet no atheromas were found in newly hatched chicks. During the first two months of life the chick is resistant to hypercholesterolemia and atherogenesis. At the eighth week (corresponding to puberty), plasma cholesterol increases markedly, despite an unchanged dietary regime. Following this rapid increase in plasma cholesterol, atherosclerosis proceeds rapidly and by 16 weeks the birds may be severely affected.

Azarnoff (1958) has divided experimental animals into two groups. The first group consists of herbivorous animals such as rabbits, chickens, guinea pigs, swine and calves. The aorta of these animals can incorporate acetate into cholesterol and it is these animals that can be made atherogenic with relative ease by feeding a high cholesterol diet. The second group consists of carnivorous and omnivorous animals such as rats, cats, dogs and humans. The aorta of these animals cannot incorporate acetate into cholesterol and it is difficult to produce atherosclerosis in this group by a high cholesterol intake alone. The role of the arterial wall in the maintenance of atherosclerosis is unknown, yet it may contribute significantly to atherogenesis. The fact that atherosclerotic plaques contain carotenoid pigments (Blankenhorn, 1956)

and linoleic acid (Böttcher et al., 1960) which cannot be synthesized in the body suggest that at least part if not most of the atherosclerotic plaque is derived from the plasma. For further detail on the role of the arterial wall in atherogenesis, the reader is referred to Lansing (1959) and Kask (1962).

Windaus (1910) has shown that the atherosclerotic aorta contains 6 - 7 times as much free cholesterol and 20 - 60 times as much ester cholesterol as does the normal aorta. These findings were confirmed by Katz and Dauber (1945) and Buck and Rossiter (1951). Field (1960) demonstrated a rapid turnover of cholesterol deposited in aortic plaques. Therefore, atherosclerotic plaques may be reversible (Field, 1960). The turnover of cholesterol in the chicken was found to be more rapid in the thoracic than in the abdominal aorta (Dayton, 1959). The lower ability to handle cholesterol in the abdominal aorta may be concomitant with its higher susceptibility to atherogenesis.

Dauber (1944) reported that the incidence of spontaneous atherosclerosis in chickens increases with age. The potentiating effects of high plasma cholesterol levels on the development of atherosclerosis (Katz and Stamler, 1953) suggest that the changes in plasma cholesterol with age may be significant. Serum cholesterol increases with age (after 30 years) in men and women (Keys, 1949; Keys et al., 1950; Jones et al., 1951). Weiss (1957) reported that the average total plasma cholesterol level of female chickens from 8 - 57 months of age was 197 (180 - 210) mg percent and did not vary with age. On the other hand, male chickens had an average plasma cholesterol level of 139 mg percent between 8 - 25 months of age, increased to 155 mg percent by 35 - 38 months and reached a level of 209 mg percent by 45 months of

age. In this particular experiment, cholesterol did not seem involved in the development of atherosclerosis, at least for the first two years of age.

Very little work has been reported concerning the effect of age on the plasma cholesterol of the turkey. Waibel et al., (1960) reported a very slight change in the plasma cholesterol levels of BBB turkeys between the ages of 3 weeks (126 mg percent) and 5 weeks (129 mg percent). Speckmann and Ringer (1962), however, reported that plasma cholesterol increased with age in both male and female BBB turkeys from 8 weeks of age (149 mg percent) to 16 weeks of age (249 mg percent), after which age the plasma cholesterol level plateaued.

In man, attempts to control plasma cholesterol by decreasing exogenous cholesterol intake fail because of the high rate of cholesterol synthesis in the liver and other organs (Ruskin, 1960). Triparanol (MER-29), a hypocholesterolemic drug, has been reported to lower serum cholesterol in man (Ruskin, 1960) and in rats and monkeys (Blohm et al., 1959); Blohm and MacKenzie, 1959). When fed to turkey poults triparanol also decreased serum cholesterol levels but it decreased growth and failed to prevent mortality from BAPN-induced aortic rupture (Waibel et al., 1960). Recent studies with triparanol in man have demonstrated that this compound inhibits cholesterol biosynthesis by blocking the reduction of 24-dehydrocholesterol (desmosterol) to cholesterol (Avigan et al., 1960; Steinberg and Avigan, 1960). Desmosterol, however, appears to be more atherogenic in chickens than cholesterol (Wong and Avigan, 1962). The prophylactic effects of triparanol on aortic rupture in turkeys is therefore questioned. The feeding of bile acid binding polymeric organic bases (Tennent et al., 1960) and ferric

chloride, which precipitate bile acids (Siperstein et al., 1953) have been reported to inhibit the increase in serum cholesterol and atherogenesis in cholesterol fed cockerels.

Hemodynamics

That hemodynamics play a role in the etiology of atherosclerosis was first suggested by Anitschkow (1933) and Rosenthal (1934). Although hypertension alone may not lead to atherosclerosis, it is more prevalent in the descending stage of life and then coupled with hypercholesterolemia, may play a decisive role. The rapid increase in blood pressure (Ringer and Rood, 1959) coupled with rapidly increasing levels of plasma cholesterol (Speckmann and Ringer, 1962) in the growing male BBB turkey may therefore be predisposing to the aortic rupture syndrome.

It is also interesting to observe that the lesions of the aorta are generally more severe than those of other arteries. The aorta distends considerably during each systole and recoils during diastole. It is possible that the recurrent stretching of the vessel may facilitate the rapid entry of lipids into the intima and media, especially under the driving force of an increased arterial tension (Aschoff, 1933; Kuroyanagi, 1959). Rosenthal (1934) reported that the deposition of lipid was closely related to the structure of the vessel and to the infiltration and expression of fats. In this respect, the thoracic aorta expresses cholesterol and lipids well; whereas, the abdominal aorta does not (Aschoff, 1933). The higher blood pressure in the aorta than in other areas of the circulation probably enhances lipid infiltration into the vessel wall. Also, the absence of atherosclerotic

plaques in areas of relatively low pressures such as the pulmonary or venous circuits further suggests a hemodynamic-atherogenic involvement.

When cortisone was administered to cholesterol-fed chicks, it produced moderate hypertensive effects concomitant with intensified aortic and coronary atherosclerosis; whereas, the administration of hydrocortisone and adrenocorticotropin to similar animals produced hypercholesterolemia and hyperlipemia yet did not intensify aortic or coronary atherosclerosis (Stamler et al., 1954).

Blumenthal (1956c) believes that atherosclerosis is a reparative process to the wear and tear of an increasing arterial tension (with age) on the vascular system. Weiss and Fisher (1959) demonstrated a relationship between blood pressure and aortic hydroxyproline (connective tissue) concentration in the chicken; whereas, plasma cholesterol was correlated with aortic cholesterol concentration. Thus, blood pressure is a better measure of abdominal atherosclerotic severity and plasma cholesterol a better measure of thoracic atherosclerotic severity. These researchers also suggested that blood pressure was involved in spontaneous avian atherosclerosis. Hemodynamic changes have also been implicated as contributory agents in the aortic rupture syndrome (Gibson and deGruchy, 1955; Ringer and Rood, 1959; Speckmann and Ringer, 1961; Burger, 1962; Ringer, 1962) as well as reduced distensibility due to atherosclerosis (Weiss and Sheahan, 1958).

Aortic Rupture Syndrome

Attempts to Produce Aortic Rupture

The pathogenesis and etiology of the aortic rupture syndrome have already been discussed. Much interest still remains as to the cause of this malady, yet it is difficult to study field problems in view of its sporadic occurrence. That beta-aminopropionitrile (BAPN) produces a similar dissecting aneurysm under predictable and controlled conditions allows research in the laboratory. Ponseti and Baird (1952) were able to show that dissecting aneurysms could be produced in rats by feeding rations containing sweet pea seed (Lathyrus odoratus). Subsequently, McKay et al., (1954) and Schilling and Strong (1955) identified the toxic substance in L. odoratus as beta-(N-gamma-L-glutamyl)-aminopropionitrile. BAPN is also contained in seeds of Caley pea (L. hirsutus) and singletary pea (L. pusillus) as reported by Barnett (1960). The similarity of lesions produced experimentally and those found in field cases of aortic rupture (Pritchard et al., 1958) suggested that BAPN might be contained as some feed ingredient but the analysis of many legumes, common feedstuffs and even the feed used during an outbreak of aortic rupture failed to detect the presence of BAPN (Barnett, 1960).

As time progressed, other investigators (Bachhuber and Lalich, 1954; Bachhuber et al., 1955; Lalich, 1956) demonstrated that dietary BAPN produced aortic rupture and other signs of lathyrism in rats. Mortality resulted from aortic rupture in the thoracic region of the aorta which was accompanied by hock and toe deformities and pericardial and pulmonary hemorrhages. Lalich et al., (1956) have also shown that

dissecting aneurysms as well as skeletal defects could be produced in turkeys by feeding different levels of BAPN. Barnett et al., (1957), Lalich et al., (1957) and Waibel and Pomeroy (1958) likened the naturally occurring condition of aortic rupture in turkeys to the dissecting aneurysms observed in rats fed BAPN. Barnett et al., (1957) observed that turkeys fed BAPN developed lathyrism with symptoms similar to those induced in rats except aortic rupture in turkeys occurred in the abdominal aorta, similar to field cases of the condition. The field cases, however, were not accompanied by hock and toe deformities and other complications (Barnett et al., 1957). The use of BAPN does, however, provide a valuable tool for the study of aortic rupture in the laboratory.

The turkey is 4 - 8 times more susceptible to BAPN than is the rat (Lalich et al., 1957). The faster growth rate and greater feed consumption may also contribute to the greater susceptibility of the turkey to BAPN toxicity (Barnett et al., 1957). Age seems to be a very important factor in susceptibility to BAPN toxicity (Geiger et al., 1933; Ponseti and Shepard, 1954; Walker and Wirtschafter, 1956; Waibel and Pomeroy, 1958). To obtain circulatory damage in rats, BAPN must be fed during the first few weeks after birth when elastogenesis is particularly active. Rats over seven weeks of age are not subject to dissecting aneurysms induced by BAPN (Walker, 1957). Zahor and Machova (1961) noted severe mortality in BAPN-treated chick embryos due to extensive hemorrhaging and dissecting aneurysms. Waibel and Pomeroy (1958) found that higher levels of BAPN are required to produce hemorrhaging when birds become older and the circulatory system is more completely developed. Ringer (1961) feeding 0.04 percent BAPN-

fumarate to 3-week-old turkey poults for seven weeks could not produce aortic rupture.

Waibel and Pomeroy (1958) demonstrated that the severity of BAPN toxicity was proportional to dosage level. Low levels of BAPN (0.01 percent) fed to 4-day-old poults produced no aortic rupture to 21 weeks of age, however, a level of 0.02 percent BAPN fed to 4-day-old turkey poults produced aortic rupture in 4 of 7 turkeys between 7 and 13 weeks of age. Higher dosage levels of BAPN increased and hastened mortality. Sex is apparently not a factor in BAPN toxicity as both sexes die. The males usually die first probably because of their faster growth rate and greater feed consumption. Ringer (1961) observed that dietary BAPN (at 0.04 percent) had no effect on blood pressure. Kowalewski (1960) noted that when BAPN was fed to cockerels it had no effect on lipid content in serum, liver or aorta thus BAPN most likely causes aortic rupture by causing degenerative changes in the aortic wall.

Investigations into the site of action of BAPN suggest that it acts by interfering with connective tissue metabolism. Enzinger and Warner (1960) suggested that acetoaminonitrile interferes with the formation and maturation of connective tissue fibers. More specifically, Walker (1957) suggested that BAPN acts by inhibiting the formation of elastic fibers rather than acting as a direct elastolytic agent. There is no breakdown in connective tissue when BAPN is administered as is evidenced by its lack of effect on the adult animal. Van Den Hooff et al., (1959) demonstrated that BAPN inhibited the crosslinking whereby tropocollagen is aggregated into insoluble fibers. This evidence was confirmed by Martin et al., (1961) and Wirtschafter

and Bentley (1962).

The lathyritic effects of BAPN may be potentiated by feeding nicotinic derivatives which inhibit the enzyme monoamine oxidase (Roy et al., 1959). Monoamine oxidase catalyzes the metabolic breakdown of BAPN to cyanoacetic acid.

Attempts to Stop Aortic Rupture

Attempts to stop losses due to aortic rupture have been essentially via the feed as this is one of the easiest and most economical ways to treat birds. The failure of voluminous dietary regimes to prevent or stop mortality from aortic rupture have been previously reviewed. Many drugs have been screened in an effort to reduce mortality with but one real success, reserpine.

Reserpine (Rauwolfia serpentina), a hypotensive tranquilizer, has been demonstrated to afford protection against aortic rupture in turkeys (Carlson, 1959; Barnett, 1960; Morrison, 1960; Waibel, 1960). Reserpine has been shown to decrease elevated serum cholesterol levels and inhibit the development of aortic atheromatosis in normotensive and hypertensive rats maintained on an atherogenic diet (Smith and Rossi, 1962). These investigators also demonstrated that reserpine reduced blood pressure only in the hypertensive rats. They also found that the hypertensive state increased the severity of atherogenesis in cholesterolized rats which were not fed reserpine. Although reserpine has been demonstrated to reduce blood pressure in chickens (Sturkie et al., 1958; Bünag and Walaszek, 1962) and turkeys (Speckmann and Ringer, 1961) it has neither an effect on the plasma cholesterol level nor on the incidence of atherosclerosis in growing BBB turkeys (Speckmann and Ringer, 1962).

The central action of reserpine, possibly at the hypothalamic level (Plummer et al., 1955) may be involved in the hemodynamic response to oral administration of reserpine to turkeys. Reserpine is an inhibitor of the serotonin-storing mechanisms of tissues (Shore et al., 1957). According to Shore et al., (1957) reserpine in the body rapidly enters the brain and irreversibly affects the serotonin binding sites and then disappears. Consequently the serotonin in various depots, including the brain, is released and metabolized by monoamine oxidase. The serotonin that continues to be made presents a persistent low concentration of free serotonin to the brain tissue. It is this free serotonin that is considered to exert the actions attributed to reserpine (Cronheim and Gourzis, 1956). Although reserpine disappears quickly, its effects persist for some time until the binding sites have recovered or until new ones are formed. Reserpine administration affects chickens similarly to mammals by releasing bound serotonin (Huber and Link, 1962). Excellent reviews on serotonin are given by Page (1958) and Lewis (1958).

The prophylactic action of reserpine on field cases of aortic rupture may not be due entirely to its hypotensive action. Burger et al., (1961), fed various tranquilizers, a ganglionic blocking agent, and mammalian hypotensive drugs to turkey poults and observed no significant effects on blood pressures with any of the drugs, including reserpine, yet all drugs but the mammalian hypotensive drugs were effective in curtailing mortality due to BAPN toxicity. Krista et al., (1961) treated a natural outbreak of aortic rupture in turkeys and observed that diethylstilbestrol implantation increased the incidence of aortic rupture by more than 60 percent; whereas, daily injection

of testosterone propionate reduced the incidence of aortic rupture by 75 percent. Diethylstilbestrol decreased blood pressure by 16 percent while testosterone had no effects on blood pressure values. Ringer and Rood (1959), however, could not decrease blood pressure in turkeys with large amounts of diethylstilbestrol. Perhaps hypertension is an aggravator rather than a cause in the aortic rupture syndrome.

Physiological Parameters Associated With the Aortic Rupture Syndrome

Cardiac Output

Ever since the discovery of the circulation by Harvey in 1628, there have been voluminous investigations to determine cardiac output under normal and stressing conditions in an attempt to more effectively diagnose cardiac abnormalities. Many methods have been described in the literature, few of which are accurate and constant enough to be considered an infallible estimation of cardiac output. The most publicized methods for determining cardiac output may be classified into five general areas, namely the direct Fick, indirect Fick or foreign gas, the dye dilution, physical methods and thermal methods.

The direct Fick principle was evolved by Fick (1870). It concerns the measurement of arterio-venous oxygen differences with a measurable oxygen consumption under basal conditions. The oxygen content of arterial blood was found by direct puncture whereas the accurate measurement of oxygen content of venous blood proved difficult in man until Forssmann (1929) demonstrated that catheterization of the right heart was possible. Slightly over a decade later Cournand (1945) and Richards (1945) initiated the widespread use of right heart

catheterization for the estimation of cardiac output. This procedure was the most accurate and is still used as a standard today.

The difficulty of applying the direct Fick to man has promoted much investigation into procedures whereby gaseous differences can be determined without direct analysis of the blood. These indirect Fick methods are theoretically successful but their application to man has resulted in limited accuracy. The general developments of indirect methods for measuring cardiac output are described by Hamilton (1945). The most successful of these methods were reported by Christiansen et al., (1914) and Douglas and Haldane (1922) whereby CO_2 arterio-venous differences were obtained without arterial puncture. The acetylene method introduced by Grollman (1932) gave the most accurate results and largely superceded older methods. The chief error in all of these methods seemed to lie in the rebreathing procedure as many investigators believe recirculation occurs and induces underestimation of the pulmonary blood flow during the test period.

Closely correlated to the usage of foreign gasses for cardiac output determinations is the injection of a substance directly into the blood stream and analyzing its dilution in samples of blood. As with the Fick principle, the onset of cardiac catheterization had aided the accurate measurement of dye concentration. Until 1950, the dye injection method was limited in use because the construction of dye dilution curves required laborious, time-consuming spectrophotometric determinations of 30-40 blood samples. The introduction, however, of apparatus for automatic registration of dye concentration in whole blood has greatly facilitated routine application of this method. A complete review of dye injection methods is given by Falholt (1958).

In this researcher's investigation of the aortic rupture syndrome in turkeys it became evident that measurement of cardiac output may elucidate some of the problems associated with this condition. Fundamental studies in determining the cardiac output of animals began as early as 1897. Stewart (1897) initiated the measurement of cardiac output by the injection method using sodium chloride as the indicator. Henriques (1913) introduced a colorimetric procedure for determining cardiac output. Hamilton et al., (1928a; 1928b; 1932), Hamilton (1953) and Kinsman et al., (1929) improved Stewart's technique by using rapid intravenous injection and serial sampling of peripheral arterial blood. This method permitted an emperical replotting of the data to separate the primary dilution curve from subsequent recirculation curves. Moore et al., (1929) reported that the injection method was very accurate when compared with the direct Fick procedure. Since that time, the Stewart-Hamilton dye dilution technique has been employed for many types of flow studies.

Several investigators have utilized radioisotopes as indicators for cardiac output determinations. Hahn and Hevesy (1940) described a method for blood volume determinations using erythrocytes labeled with radiophosphorus (P^{32}). Later Hevesy et al., (1944) applied this method to human subjects. Nylin (1945) and Nylin and Celander (1950) also used erythrocytes labeled with radiophosphorus for cardiac output determinations. They injected the labeled cells into an arm vein and blood samples were taken from the artery of the opposite arm. From the concentration of the indicator as a function of time the cardiac output was determined.

Recently Sapirstein and Hartman (1959) reported the cardiac output of sodium pentobarbital anesthetized chickens as 218 ml per Kg of body weight per min. These investigators used rubidium (Rb^{86}) as an isotope tracer. Also in 1959, the cardiac outputs of adult male and female chickens were reported as 143 and 173 ml per Kg of body weight per min, respectively, by Sturkie and Vogel (1959). The latter authors used Evans Blue (T-1824) in a dye dilution technique. Lawson et al., (1952) found very little difference in cardiac outputs calculated from dye (T-1824) or radiophosphorus (P^{32}) labeled erythrocyte dilution curves. Sapirstein and Hartman (1959) and Sturkie and Vogel (1959) collected numerous arterial blood samples to obtain a dilution curve. MacIntyre et al., (1951, 1952), and Pritchard et al., (1952) outlined a procedure for the determination of cardiac output by injecting radioiodinated (I^{131}) human serum albumen (RISA) and continuously recording the dilution curve. Milnor et al., (1953) described a photoelectric ear densitometer for continuously recording the arterial concentration of T-1824 in the dye dilution method. Sturkie is using a linear densitometer now to obtain continuous measurements of changes in indicator (T-1824) concentration (Personal Communication).

The physical methods of measuring cardiac output include mainly ballistocardiography and the pulse contour method. Ballistocardiography was first introduced by Henderson and Haggard (1925) but the method was developed more fully by Starr et al., (1939), Starr and Schroeder (1940), and Starr (1944, 1945). The principle involved in this method was that forces originating in the movement of blood were transmitted to the body and could therefore be recorded. Cardiac output was determined by the application of proper formulae to the ballistocardiograph.

When compared to standard methods of determining cardiac output under resting conditions in normal subjects, it was quite accurate. However the departure from basal conditions deprives this method of its accuracy. The pulse contour method of determining cardiac output has been in the literature since 1904, when Erlanger and Hooker (1904) suggested that the output of the heart varies approximately as the product of pulse pressure and pulse rate. The pulse contour method has been difficult to apply and yields highly variable results. The distensibility of the aorta varies and affects the pulse wave velocity; a constant must be calculated to determine cardiac output. A review of the development of this procedure is described by Hamilton (1945). Hamilton and Remington (1947) designed a procedure which rests on the principle that the uptake of blood during systole by various segments of the arterial tree may be estimated directly from the aortic pressure pulse. According to Huggins et al., (1948; 1949) the pulse contour method checks well with determinations by the Fick method; however, Brotmacher (1957) and Bernstein and Evans (1962), making similar comparisons, believe this method is only good for relative values but is not reliable for accurate estimations of cardiac output.

Another method used for determining cardiac output is the thermal dilution method (Evonuk et al., 1961; Richardson et al., 1962). The method depends upon accurate recording of the changes in blood temperature which occur in the pulmonary artery and the arch of the aorta when cool isotonic saline is injected into the superior vena cava. This method compares favorably with the Fick and dye dilution procedures (Fegler, 1954; Goodyer et al., 1959).

Elasticity

The elastic stretching of the human vascular wall has long been the subject of extensive studies. Many physical methods have been developed for examining the elasticity of the vascular wall (Hallock and Benson, 1937; Nichol, 1955; Lawton, 1955; Weiss and Linde, 1960; Bergel, 1961; and Balkrishna et al., 1961). More recently, elastance has been determined in vivo in dogs (Remington, 1962) and estimated from total perfusion of animals (Johnson et al., 1962). To the author's knowledge there have been no elasticity studies in Aves.

OBJECTIVES

1. To investigate the possible causes of aortic rupture in an attempt to minimize losses due to this condition in the field.
2. To determine the normal plasma cholesterol level of the BBB turkey.
3. To determine the cardiac output of mature male BBB turkeys under normal conditions.
4. To determine the modulus of elasticity of thoracic and abdominal aortic segments of mature male BBB turkeys.
5. To evaluate comparative blood pressure measurements, i.e., from the carotid and popliteal arteries.
6. To study the effects of age, sex, and oral reserpine administration on plasma cholesterol, hemodynamic parameters and atherosclerotic severity.
7. To study the effects of injecting a known quantity of epinephrine, simulating a stress, upon cardiovascular parameters.
8. To study the effects of atherosclerosis on plasma cholesterol, cardiac output, elastance and hemodynamic parameters.
9. To investigate the effects of feeding reserpine, BAPN, vitamin C and BAPN + reserpine and the effects of heat on cardiac output, thoracic and abdominal elasticity and carotid and popliteal blood pressures.
10. To determine the effect of strain on blood pressure, cardiac output, and elastance.

EXPERIMENTAL PROCEDURE

General:

The same general procedures were employed throughout the entire research project. All of the experimental animals were turkeys of the Broad Breasted Bronze (BBB) variety. All animals were hatched in Michigan State University Poultry Science Department incubators except for the cholesterol experiment where purchased commercial BBB turkeys were utilized. All turkeys were reared in a commercial type house and had access to a sunporch. All birds, treatments, and locations were randomized, where appropriate, as completely as possible by the selection of numbers from a box.

Cholesterol Determination:

The quantitative estimation of cholesterol utilizing the Liebermann-Burchard reaction was reported by Zlatkis et al. (1953). Modification of this procedure for the estimation of plasma cholesterol in turkeys is reported in the Appendix.

Blood Pressure Determination:

An identical method for determining arterial hemodynamic parameters was used throughout the experiments, namely, the direct technique. Direct measurements were taken in unanesthetized animals held in a supine position on a wooden frame.

Hemodynamic measurements from the right common carotid artery were recorded in a manner similar to that described by Weiss and Sturkie (1951). The common carotid artery was exposed surgically by a longitudinal incision in the neck. The artery was clamped and the cranial end tied permanently. A small nick was made in the artery between the tie and the clamp. A polyethylene tube (i.d. 0.045 in. X o.d. 0.062 in. for young birds or i.d. 0.066 in. X o.d. 0.095 in. for mature birds) which was 30 cm long was inserted into the artery and tied in place. The cannula system was connected to a Statham pressure transducer, Model P-23 156-255, which in turn was connected to a low level DC pre-amplifier of a Grass Model 5 polygraph. The cannula was filled with 10 percent sodium citrate solution to prevent clotting. The pressure equipment was calibrated through the same transducer by means of a "three-way" valve which permitted connection of a pocket model aneroid sphygmomanometer thus establishing a closed air system. The pressure was increased and the levels were recorded on the polygraph.

Popliteal arterial blood pressures were determined via a percutaneous vessel puncture technique similar to that described by Ringer et al. (1955). The cannula (18 gauge hypodermic needle) was connected to polyethylene tubing. The tubing in turn was connected to a separate channel of the polygraph in the manner described for common carotid arterial measurements, thus common carotid and popliteal arterial hemodynamic parameters were recorded simultaneously.

Systemic resistance units for the common carotid and popliteal arteries were calculated in terms of relative units by dividing mean blood pressure (in mm Hg) by milliliters flow per minute. Units were

calculated either on a bird or on a $Kg^{0.734}$ basis. Mean blood pressure was determined by dividing systolic plus diastolic blood pressure by two.

Cardiac output determination:

The well-known Stewart-Hamilton dilution principle was adapted for using radiophosphorus (P^{32}) as a tracer and measuring its dilution, similar to a procedure described by MacIntyre et al. (1951). Unanesthetized BBB turkeys were immobilized in a supine position on a rigid frame throughout the recording.

The right common carotid artery was exposed and cannulated with polyethylene tubing (i.d. 0.070 in. X o.d. 0.110 in.). The tubing was tied into each end of the cut artery and arranged in a loop so that continuous blood flow could be maintained. The loop of tubing was passed beneath an end-window G-M (Geiger-Mueller) tube (Nuclear Chicago) and taped in place so that identical counting geometry could be used for counting the standard (Fig. I). The G. M. tube was connected in series to a Model 1620 rate meter (Nuclear Chicago) which, in turn, was connected to an Esterline-Angus recorder. The arrangement of the equipment is illustrated in Fig. II. The rate meter scale was set for a sensitivity of 3 K and a time constant of 0.5 sec. The operating voltage was determined as 1050 V. The recorder was calibrated with the rate meter and set at the fastest gear ratio (12 in. per min). It had a curvilinear deflection of 6 in. Standard counts were determined by a Model 182 A scaling unit (Nuclear Chicago) via the same G-M tube and bore of polyethylene tubing used for the actual determinations. Approximately 40-60 μc of P^{32} (contained in an aliquot

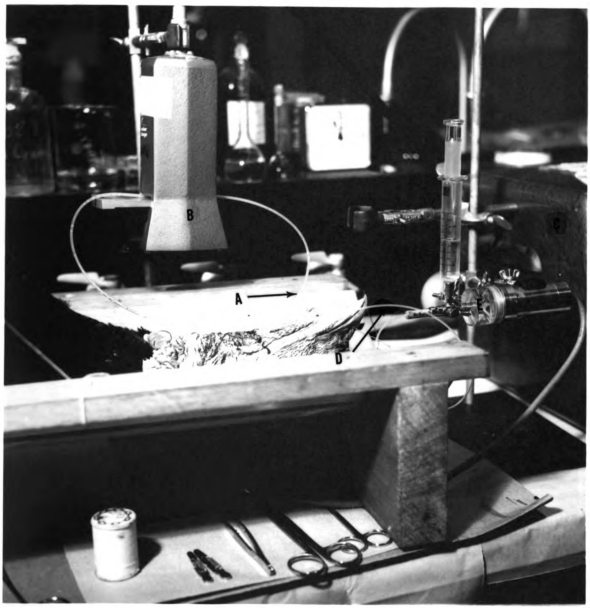


Figure 1. Photograph illustrating polyethylene tubing (A) inserted into the right common carotid artery. Continuous blood flow is directed via tubing beneath G-M tube (T) which is connected to a rate meter (C). Also note polyethylene cannula (D) in left common carotid artery for blood pressure determinations. The pressure transducer (E) is pictured at lower right.

Figure II. Photograph illustrating equipment for detection and recording of radioactivity. From left to right: G-M tube (A), rate meter (C), direct writing ink recorder (D) automatic time-clock (E) and scaler unit (F). Note pressure transducer (B) used for blood pressure determinations.

ranging from 0.01-0.10 ml) were injected into each bird via the brachial vein and close to the body to give maximum deflection of the rate meter. It was therefore possible in one experiment to simultaneously record cardiac output and common carotid and popliteal hemodynamic parameters.

A typical primary dilution curve is shown in Fig. III. The curve shows a rapid upswing reaching a peak in 3-6 seconds; the declining limbs are not interrupted by recirculation for at least 10 seconds. Unfortunately, recirculated blood generally appears before the down-swing of the primary curve is completed, necessitating extrapolation of the primary down-stroke. Since the down-stroke is assumed to closely approximate exponential clearance of the isotope from the heart, extrapolation on semilogarithmic paper completes the dilution curve and if the original tracing is extended the area or average counts of the curve may be accurately determined.

Cardiac output was determined by modifying the standard Stewart-Hamilton dilution technique. It was established that:

$$MV = \frac{(\text{cps inj})}{\frac{(\text{cps})(\text{min})}{(\text{ml})}}$$

Where MV = Minute volume

cps = Counts per second

cps = Average counts per second of primary dilution curve per milliliter of blood

$$CO = \frac{MV}{BW^{0.734}}$$

min = Duration of primary curve in minutes

CO = Cardiac output

BW = Body weight in kilograms

CHART NO. 4309–D

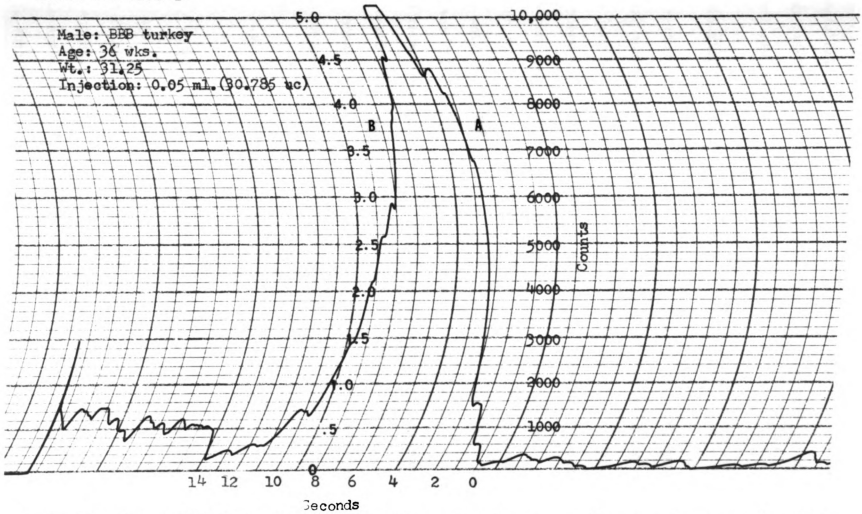


Figure III. Photograph illustrating a typical primary dilution curve. The curve demonstrated a rapid upswing (A) reaching a peak in 5 seconds. The declining limb (B) is not interrupted by recirculation for at least 14 seconds.

Since metabolic activities such as cardiac output do not increase at a linear rate with body size or weight, body weight raised to the 0.734 power was used (Brody, 1945).

Elastance Determination:

A method was devised whereby the elasticity of thoracic and abdominal aortic segments of BPB turkeys could be accurately determined. A 20 mm length of the aortic segment was tied at each end to rigid polyethylene tubing. At one end the tubing was connected to a syringe and Statham pressure transducer by means of a "three-way" valve. At the other end of the segment the tubing was tied onto a tuberculin syringe which in turn was attached to a micrometer. When the entire system was filled with liquid, a calibrated volume of liquid was delivered to the vessel segment, the pressure recorded and the external radius measured. Fig. IV shows a photograph of the apparatus.

An equation for the calculation of elastance (E) of an isotropic tube which does not change in length upon inflation was given by Love (1927):

$$E = \frac{\Delta p}{-R_o} \quad X \quad \frac{2(1-\sigma^2)R_i^2 R_o}{R_o^2 - R_i^2}$$

Where ΔR_o is the change in external radius following a pressure change Δp, R_i is the internal radius and σ is known as Poisson's ratio. Poisson's ratio is the ratio of transverse to longitudinal strain and has been taken as 0.5 for the arterial wall which has been shown to extend isovolumetrically. The measurement of internal radius, however, when the aorta was in a closed system, could not be easily determined. Love's equation was rearranged (see Fig. V) so that

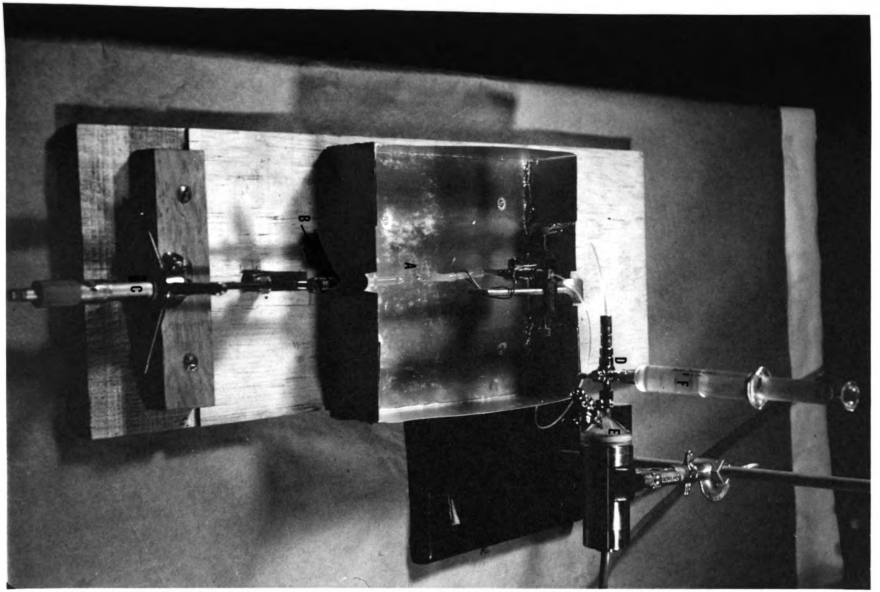


Figure IV. Photograph illustrating apparatus used to determine elastance. The vessel segment is attached at both ends by polyethylene tubing at (A). At one end the tubing is connected to a tuberculin syringe (E) which in turn is attached to a micrometer (C). At the other end the tubing is attached to a "3-way" valve (D) which permits pressure measurements through a transducer (D) or addition of more liquid to the system via a syringe (F).

Figure V Mathematical Re-arrangement of Love's Formula Substituting Volume for Radius

$$E = \frac{\Delta p}{\Delta R_o} \times \frac{2(1-\sigma^2)R_\lambda^2 R_o}{(R_o^2 - R_\lambda^2)}$$

1. $V_o = \pi R_o h$

2. $\Delta V_o = 2\pi h R_o \Delta R_o$

3. $\Delta R_o = \frac{\Delta V_o}{2\pi h R_o}$

4. Substituting:

$$E = \frac{\Delta p}{\frac{\Delta V_o}{2\pi h R_o}} \times \frac{2(1-\sigma^2)R_\lambda^2 R_o}{(R_o^2 - R_\lambda^2)}$$

5. $$E = \frac{\Delta p}{\Delta V_o} (4\pi h)(1-\sigma^2) \frac{R_\lambda^2 R_o^2}{R_o^2 - R_\lambda^2}$$

6. $$\frac{R_o^2 R_\lambda^2}{R_o^2 - R_\lambda^2} = \frac{\frac{R_o^2 R_\lambda^2}{R_o^2 R_\lambda^2}}{\frac{R_o^2 - R_\lambda^2}{R_o^2 R_\lambda^2}} = \frac{1}{\frac{R_o^2 - R_\lambda^2}{R_o^2 R_\lambda^2}} = \frac{1}{\frac{R_o^2}{R_o^2 R_\lambda^2} - \frac{R_\lambda^2}{R_o^2 R_\lambda^2}} = \frac{1}{\frac{1}{R_\lambda^2} - \frac{1}{R_o^2}} = \left(\frac{1}{R_\lambda^2} - \frac{1}{R_o^2}\right)^1$$

7. $$\frac{1}{R_\lambda^2} = \frac{\pi h}{V_{in}}$$

8. $$E = \left(\frac{\Delta p}{\Delta V_o}\right)\left(4\pi h\right)\left(1-\sigma^2\right)\left(\frac{\pi h}{V_{in}} - \frac{1}{R_o^2}\right)^{-1}$$

elastance became a function of the change in volume with a given pressure change. Changes in volume and pressure were accurately measured with this apparatus. The final rearranged equation, for the determination of elastance (E), is given as:

$$E = \left(\frac{\Delta p}{\Delta V_o}\right)\left(4\pi h\right)\left(1-\theta^2\right)\left(\frac{\pi h}{V_{in}} - \frac{1}{R_o^2}\right)^{-1}$$

Where ΔV_o is the change from initial volume V_{in} with a pressure change Δp. The h refers to height or length of the vessel and R_o is the outside radius.

Atherosclerosis Determination:

Following each experiment, the abdominal aortas were removed and visually scored for atherosclerotic plaques in the manner described by Katz and Stamler (1953). Scores range from 0 (no incidence) to 4 (high incidence).

Statistical Analysis:

All of the data were statistically analyzed using the Analysis of Variance or the Student "t" test (Snedecor, 1956). The treatment means were compared by the Duncan Multiple Range and Multiple F test (Duncan, 1955). Other data were subjected to correlation analysis (Simpson et al., 1960).

RESULTS AND DISCUSSION

Experiment I: Age, Plasma Cholesterol and Atherosclerosis

The first experiment was conducted on 360 commercially purchased BBB turkey poults. These turkeys were utilized to study plasma cholesterol, atherosclerosis and hemodynamic parameters together with growth. There were three treatments: 0.1 ppm and 0.2 ppm reserpine administered in the feed as Serpasil (reserpine, CIBA) and untreated controls. Each treatment was separated into a male group and a female group and these groups again divided into two replicates resulting in twelve pens of thirty birds each. The position of the treated groups was randomly selected. After the birds were sexed, they were sorted and assigned to treatments based on a one percent level of non-significance in initial body weights. The experiment was initiated on February 25, 1959, at which time the poults were hatched. Serpasil was incorporated into the feed at one day of age and continued until September 10, 1959 when the birds were sacrificed. The turkeys were fed starter, grower and finisher rations from 2-8, 9-16, and 17-27 weeks of age, respectively. The turkeys were raised on the floor throughout the entire experiment, had access to a sunporch during the summer months and received feed and water ad libitum. Individual body weights and feed weights were recorded monthly and feed efficiencies were calculated from these data.

At the same time that the monthly body weight recordings were taken, a two ml. sample of blood was withdrawn from each of eight birds in each replicate for plasma cholesterol determinations.

At the end of the experiment (27 weeks of age) blood pressures were taken directly from the common carotid artery of unanesthetized birds. Only those birds from which plasma cholesterol samples had been withdrawn were used. Following the blood pressure determinations, the abdominal aortas were removed and visually scored for atherosclerotic plaques.

The initial and final body weights, the monthly weight gain and feed efficiencies are shown in Table 1. The initial body weights and the final body weights between treatments within sex were non-significantly different at the 5 percent level. Analyses of these data revealed that differences between the weight gains of the replicates were not statistically significant. Therefore, the replicates were pooled within each treatment.

The results of the monthly plasma cholesterol determinations by sex are presented in Table 2. The data were pooled as the differences between replicates were not statistically significant. Plasma cholesterol levels of both males and females rose from 8 weeks of age to 16 weeks of age, after which age the plasma cholesterol level plateaued. At any particular age, however, the plasma cholesterol levels of the two sexes were not statistically different. The differences between plasma cholesterol levels in any of the treatments were not statistically significant at any age.

The mean common carotid hemodynamic parameters and atherosclerotic scores of the abdominal aorta are presented in Table 3. The blood pressure values obtained differed from those previously reported for BBB turkeys (Ringer and Rood, 1959) but were similar to blood pressures reported for Jersey Buff turkeys (Weiss and Sheahan, 1958).

Also in this study, male and female hemodynamic parameters were not statistically different. The males demonstrated a higher incidence of atherosclerotic plaques in the abdominal aorta than did the females. This is in agreement with evidence obtained in humans and in chickens. There was no difference between the control group and the Serpasil-treated groups within sex. In this study, Serpasil at the levels employed did not significantly influence plasma cholesterol, hemodynamic parameters or atherosclerotic severity.

Key for Tables

A	=	Abdominal
Aor Sc	=	Aortic score
BAPN	=	Beta-aminopropionitrile
BBB	=	Broad Breasted Bronze
BPM	=	Beats per minute
BVT	=	Blood vessel thickness
BW	=	Body weight
C	=	Carotid artery
Chol	=	Cholesterol
CO	=	Cardiac output
Comm	=	Commercial
DBP	=	Diastolic blood pressure
E	=	Elastance
F	=	Female
HR	=	Heart rate
Ht wt	=	Heart weight
M	=	Male
MP	=	Mean pressure
MV	=	Minute volume
PP	=	Pulse pressure
SBP	=	Systolic blood pressure
SRU	=	Systemic resistance units
S.E.M.	=	Standard error of mean
SV	=	Stroke volume
T	=	Thoracic
$\triangle$	=	Change

Table 1. Determinations of initial and final body weights, the monthly weight gains and the feed efficiency of BBB turkeys fed Serpasil 1/ from 1 day to 24 weeks of age

Treatments	Sex	No. birds	Initial BW (gms)	Weight gains 1-day 4-wk (kgm)	4-8 wk (Kgm)	8-12 wk (Kgm)	12-16 wk (Kgm)	16-20 wk (Kgm)	20-24 wk (Kgm)	Final BW (Kgm)	Mortality (percent)
1 Untreated	M	60	48.4	0.49	1.48	2.37	2.93	1.65	1.83	10.84	15.0
	F	60	47.7	0.45	1.18	1.79	1.87	0.88	0.67	6.89	6.7
2 0.1 ppm Serpasil	M	60	48.2	0.45	1.42	2.33	2.79	1.59	2.05	10.74	6.7
	F	60	47.4	0.45	1.24	1.81	1.83	0.79	0.89	6.96	10.0
3 0.2 ppm Serpasil	M	60	50.0	0.48	1.45	2.37	2.84	1.53	1.80	10.54	5.0
	F	60	47.7	0.46	1.25	1.80	1.73	0.72	0.80	6.80	5.0
F value 2/	M		2.00	5.92**	2.02	0.35	2.50	1.56	4.59*	1.37	--
	F		0.10	0.94	3.04	0.14	2.45	6.82**	10.34**	0.91	--
S.E.M.	M		± 0.70	± 0.01	± 0.04	± 0.04	± 0.06	± 0.05	± 0.06	± 0.13	--
	F		± 0.50	± 0.01	± 0.02	± 0.02	± 0.03	± 0.03	± 0.03	± 0.08	--
Non-Significance 3/	M		--	(1-3)	--	--	--	--	(1-3)	--	--
	F		--	--	--	--	--	(1-2) (2-3)	(2-3)	--	--

1/ Reserpine - CIBA.
2/ Numbers with one asterisk indicate significance at the P ≤ 0.05 level while those numbers with two asterisks indicate significance at the P ≤ 0.01 level.
3/ Numbers joined by a dash are non-significantly different at the P 0.01 level.

Table 1. (Cont'd). Determinations of initial and final body weights, the monthly weight gains and the feed efficiency of BBB turkeys fed Serpasil 1/ from 1 day to 24 weeks of age

Treatments	Sex	No. birds	Feed efficiency (lbs feed/lb gain)			
			4-12 wks	12-16 wks	16-20 wks	20-24 wks
1 Untreated	M	60	2.6	3.8	6.6	5.8
	F	60	2.8	4.2	9.4	9.3
2 0.1 ppm Serpasil	M	60	2.6	3.8	6.0	4.8
	F	60	3.0	4.4	10.0	8.2
3 0.2 ppm Serpasil	M	60	2.7	5.0	6.9	5.9
	F	60	2.8	5.1	11.0	9.7

1/ Reserpine - CIBA

Table 2. Mean monthly plasma cholesterol levels (mg percent) of BBB turkeys from 8-24 weeks of age with Serpasil 1/ administration from 1 day through 24 weeks of age

Treatments	Sex	No. birds	Age 8 wks	12 wks	16 wks	20 wks	24 wks
1 Untreated	M	16	148	180	249	249	248
	F	16	150	167	243	247	240
2 0.1 ppm Serpasil	M	16	156	175	245	247	226
	F	16	164	156	236	234	244
3 0.2 ppm Serpasil	M	16	149	167	250	258	232
	F	16	155	170	233	236	239
F value 2/	M		0.49	0.47	0.09	2.17	2.03
	F		1.53	0.69	0.67	1.75	0.19
S.E.M.	M		± 6.21	± 9.35	± 9.05	± 8.31	± 7.99
	F		± 5.98	± 9.00	± 5.74	± 5.08	± 6.75

1/ Reserpine - CIBA
2/ All values were non-significantly different at the $P < 0.05$ level.

Table 3. Common carotid hemodynamic parameters and mean scores of abdominal aortic atherosclerotic plaques of BBB turkeys at 27 weeks of age with Serpasil 1/ administration from 1 day through 24 weeks of age

Treatments	Sex	No. birds	SBP (mm Hg)	DBP (mm Hg)	PP (mm Hg)	MP (mm Hg)	HR (BPM)	Aor. 2/ Sc.
Untreated	M	16	204	144	59	174	163	1.25
	F	16	215	156	59	186	197	0.40
0.1 ppm	M	16	211	148	64	180	170	1.30
Serpasil	F	16	207	149	58	178	183	0.60
0.2 ppm	M	16	194	136	60	165	162	1.55
Serpasil	F	16	201	146	55	173	194	0.40
F value 3/	M	16	0.54	0.77	0.13	0.64	0.46	0.21
	F	16	1.26	1.56	0.24	1.56	1.82	0.39
S.E.M.	M		±11.56	± 6.79	± 6.52	± 8.95	± 6.48	± 0.35
	F		± 6.61	± 4.34	± 4.45	± 5.13	± 5.65	± 0.19

1/ Reserpine - CIBA.
2/ 0 = no incidence
4 = high incidence.
3/ All values were non-significantly different at the $P < 0.05$ level.

Experiment II: Age, Plasma Cholesterol, Hemodynamic Parameters and Atherosclerosis

In this experiment, BBB turkeys hatched in M.S.U. Poultry Science Department incubators were utilized. They were raised in the conventional manner, indoors, receiving feed and water ad libitum. At bi-weekly intervals beginning at 4 weeks of age and extending through 24 weeks of age, 6 male and 6 female untreated turkeys were removed from the group. Blood pressures were determined and 2 ml of blood was removed for cholesterol determination. The birds were then sacrificed and the abdominal aortas removed and scored to ascertain the onset and severity of atherosclerosis.

The bi-weekly measurements of hemodynamic parameters, plasma cholesterol, and abdominal atherosclerotic severity of untreated BBB turkeys from 4-24 weeks of age are presented in Table 4. Hemodynamic values of male and female turkeys rose from 4 weeks to 24 weeks of age reaching a peak at approximately 20-22 weeks of age. The blood pressures of the males were significantly higher than those of the females after 16 weeks of age.

Plasma cholesterol levels again rose significantly in both males and females with advancing age and again there was no significant difference in plasma cholesterol level between males and females at any age. The plasma cholesterol levels were, in general, higher than those reported for male and female turkeys in Experiment I but the turkeys were of a different strain.

At 8 weeks of age, all aortas had either microscopic or macroscopic atherosclerotic lesions. Most of the lesions were protruding into the blood vessel lumen and were thus partially obstructive to the

flow of blood. The plaques occurred as streaks of fatty material on the ventral aspect of the aorta. Very rarely were the streaks accompanied with carotinoid pigment as is frequently reported in chickens. The tom turkeys were affected with a more severe incidence of atherosclerosis which increased with advancing age. Sporadically, certain individuals showed evidence of severe atherosclerosis, especially the older males.

Table 4. Bi-weekly measurement of hemodynamic parameters, plasma cholesterol and abdominal atherosclerotic severity of BBB turkeys from 4-24 weeks of age

	Sex	No. birds	Mean parameters ± S.E.M. 4 wk 1/	8 wk	10 wk	12 wk	14 wk
SBP	M	6	128 ± 5.27	179 ± 7.15	174 ± 5.98	194 ± 5.58	192 ± 8.15
(mm Hg)	F	6		172 ± 10.51	170 ± 3.78	186 ± 8.98	191 ± 9.02
DBP	M	6	94 ± 6.15	131 ± 9.36	133 ± 7.58	152 ± 3.50	152 ± 7.53
(mm Hg)	F	6		141 ± 8.60	142 ± 2.63	150 ± 7.21	153 ± 6.61
PP	M	6	35 ± 2.80	47 ± 4.04*	41 ± 5.34	42 ± 3.70	40 ± 2.96
(mm Hg)	F	6		32 ± 2.27	28 ± 1.29	36 ± 3.20	38 ± 3.22
MP	M	6	111 ± 5.61	155 ± 8.07	154 ± 6.22	173 ± 4.29	172 ± 7.75
(mm Hg)	F	6		157 ± 9.44	156 ± 3.19	168 ± 8.00	173 ± 7.75
HR	M	6	359 ± 10.93	246 ± 7.44	228 ± 7.48	229 ± 10.06	204 ± 4.77
(BPM)	F	6		274 ± 10.47	248 ± 16.52	253 ± 8.73	232 ± 5.06
Chol.	M	6	207 ± 5.03	275 ± 12.96	244 ± 8.34	239 ± 8.40	243 ± 12.56
(mg %)	F	6		251 ± 12.80	243 ± 6.18	244 ± 8.67	250 ± 10.40
Aor.	M	6		0.81 ± 0.28	1.58 ± 0.45	1.50 ± 0.56	0.75 ± 0.21
Sc. 2/	F	6		0.33 ± 0.25	0.63 ± 0.13	0.75 ± 0.28	0.25 ± 0.11
BW	M	6		2.06 ± 0.09*	2.94 ± 0.10**	4.05 ± 0.06**	5.09 ± 0.13**
(Kg)	F	6		1.61 ± 0.09	2.08 ± 0.04	2.73 ± 0.10	3.44 ± 0.16

1/ Sexes pooled.
2/ 0 = no incidence; 4 = high incidence.
* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

Table 4 (Cont'd). Bi-weekly measurement of hemodynamic parameters, plasma cholesterol and abdominal atherosclerotic severity of BBB turkeys from 4-24 weeks of age

	Sex	No. birds	Mean parameters ± S.E.M. 16 wk	18 wk	20 wk	22 wk	24 wk
SBP	M	6	208 ± 10.86	224 ± 5.20*	244 ± 10.68**	233 ± 11.59	231 ± 11.06
(mm Hg)	F	6	219 ± 9.99	192 ± 7.60	198 ± 5.28	223 ± 9.31	216 ± 3.51
DBP	M	6	158 ± 7.89	167 ± 2.83*	175 ± 7.19*	165 ± 7.19	165 ± 6.83
(mm Hg)	F	6	164 ± 5.91	145 ± 6.06	146 ± 3.51	162 ± 4.62	153 ± 6.80
PP	M	6	50 ± 4.17	55 ± 2.96	69 ± 5.07*	68 ± 6.66	66 ± 4.90
(mm Hg)	F	6	54 ± 5.16	47 ± 2.08	52 ± 3.07	61 ± 5.44	63 ± 5.42
MP	M	6	183 ± 9.23	196 ± 3.96*	210 ± 8.70**	199 ± 8.79	198 ± 8.91
(mm Hg)	F	6	192 ± 7.77	169 ± 6.78	172 ± 4.21	193 ± 6.76	184 ± 4.60
HR	M	6	224 ± 8.04	179 ± 4.21	184 ± 9.45	191 ± 6.04	193 ± 7.60
(BPM)	F	6	248 ± 16.60	207 ± 7.82*	203 ± 8.92	205 ± 14.72	241 ± 19.25
Chol.	M	6	344 ± 12.85*	299 ± 15.76	330 ± 26.74	321 ± 7.94	308 ± 11.99
(mg %)	F	6	295 ± 11.25	288 ± 20.65	302 ± 15.63	323 ± 7.32	324 ± 9.03
Aor.	M	6	1.75 ± 0.57**	1.25 ± 0.11	1.30 ± 0.24	1.67 ± 0.28*	1.50 ± 0.33
Sc. 1/	F		0.33 ± 0.17	0.58 ± 0.24	0.50 ± 0.06	0.50 ± 0.06	0.83 ± 0.28
BW	M	6	5.52 ± 0.17**	6.66 ± 0.14**	7.45 ± 0.17**	8.86 ± 0.16**	9.44 ± 0.25**
(Kg)	F		4.20 ± 0.14	4.73 ± 0.08	5.28 ± 0.07	6.27 ± 0.07	6.54 ± 0.05

1/ 0 = no incidence
4 = high incidence.

* Indicates significance at the $P < 0.05$ level.

** Indicates significance at the $P < 0.01$ level.

Experiment III: Effects of Intravenous Injection of Epinephrine on Hemodynamic Parameters of Untreated and Reserpine-Treated Male BBB Turkeys

This experiment was essentially an exploratory trial to determine the effects of an intravenous injection of 0.5 ml of 1/25,000 epinephrine on the hemodynamic parameters of untreated and reserpine-treated male BBB turkeys. This experiment was conducted in two phases, the second phase being a refinement of the first phase. The two phases will therefore be discussed together.

The turkeys were hatched and raised by conventional means by members of the M.S.U. Poultry Science Department. At nine weeks of age the turkeys were separated into 3 lots (untreated, 0.5 ppm and 1.0 ppm reserpine) in the first phase and 4 lots (untreated, 0.1 ppm, 0.5 ppm, and 1.0 ppm reserpine) in the second phase. Each lot consisted of 10 toms. After subsistence on this regime for 12 weeks (age 21 weeks) common carotid hemodynamic parameters were recorded pre- and post-epinephrine injection.

The hemodynamic parameters of the turkeys in the first phase are presented in Table 5 whereas the hemodynamic parameters of those in the second phase are presented in Table 6. Reserpine administration at the level of 0.5 ppm and 1.0 ppm of the ration significantly decreased all hemodynamic parameters. Reserpine at the level of 0.1 ppm of the ration did not alter hemodynamic parameters.

Following the injection of epinephrine, the percentage change values were indexed by using 1.00 percent as the base value. Thus a percent change of 174 indicates the value as 1.74 percent of the original value or a net increase of 74 percent whereas a percent change of

0.89 indicates the value is only 89 percent of the original value or a net decrease of 11 percent. This method of indexing will be used for the remainder of the tables concerning percent change in cardiovascular parameters following the injection of epinephrine.

In all lots, epinephrine injection significantly elevated all hemodynamic parameters. Following epinephrine injection heart rate fluctuated, being elevated in some birds, but decreased in others; the net result being no change in the heart rate of untreated lots, but a slight decrease in heart rate of the reserpine-treated lots. The reserpine-fed turkeys demonstrated a greater percentage change in systolic blood pressure, and especially pulse pressure, than did the untreated turkeys following the intravenous injection of epinephrine. Although the percentage increase in systolic blood pressure and pulse pressure was considerably greater for the reserpine-fed turkeys than for the untreated turkeys, the initial and final mean systolic blood pressures and pulse pressures were significantly lower than those of the untreated lot.

Theoretically, if this amount of epinephrine injected corresponds to the amount of epinephrine released in the body when the tom experiences a "stress," then following a "stress," depending on its nature and severity, systolic blood pressure may be elevated in the neighborhood of 30-35 percent. Individual birds have been observed to reach 500 mm Hg and above following the same 0.5 ml injection of epinephrine. Reserpine therefore does exert some protection against stress by virtue of its hypotensive action.

Table 5. Common carotid hemodynamic parameters pre- and post-epinephrine[1/] of adult male BBB turkeys (age 21 wks) following Serpasil[2/] administration for 12 weeks

Lot	1	2	3			
Treatments (ppm reserpine)	None	0.5	1.0			
No. birds	10	10	10	S.E.M.	F value[3/]	Non-significance[4/]
Pre-epinephrine:						
SBP (mm Hg)	311	205	251	± 18.41	9.13**	(1-3)(2-3)
DBP (mm Hg)	222	169	192	± 10.11	7.55**	(1-3)(2-3)
PP (mm Hg)	89	36	59	± 8.82	10.01**	(1-3)(2-3)
MP (mm Hg)	267	188	222	± 14.14	8.66**	(1-3)(2-3)
HR (BPM)	228	201	199	± 7.46	5.31*	(2-3)
Post-epinephrine:						
SBP (mm Hg)	411	284	365	± 18.41	12.61**	(1-3)
DBP (mm Hg)	269	200	226	± 12.73	8.56**	(1-3)(2-3)
PP (mm Hg)	142	84	138	± 10.35	9.10**	(1-3)
MP (mm Hg)	340	242	296	± 14.51	11.92**	(1-3)(2-3)
HR (BPM)	231	177	180	± 8.97	13.94**	(2-3)
Percent change[5/]						
SBP	1.32	1.39	1.45	± 0.05	1.50	
DBP	1.21	1.18	1.18	± 0.02	1.27	
PP	1.60	2.33	2.34	± 0.23	4.10*	(2-3)
MP	1.27	1.29	1.33	± 0.04	0.48	
HR	1.01	0.88	0.90	± 0.04	1.98	

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Reserpine - CIBA.
3/ Numbers with one asterisk indicate significance at P 0.05 while those numbers with two asterisks indicate significance at P ~ 0.01.
4/ Numbers joined by a dash are non-significantly different at P . 0.01 level.
5/ Percentage values indexed to base 1.00.

Table 6. Common carotid hemodynamic parameters pre- and post-epinephrine[1] of adult male BBB turkeys (age 21 wks) following Serpasil [2] administration for 12 weeks

Treatments (ppm reserpine)	None	0.1	0.5	1.0			
No. birds	10	10	10	10	S.E.M.	F value [3]	Non-significance [4]
Pre-epinephrine:							
SBP (mm Hg)	295	290	223	229	± 13.38	8.18**	(1-2)(3-4)
DBP (mm Hg)	215	213	175	175	± 8.06	3.70*	(1-2)(3-4)
PP (mm Hg)	81	77	48	54	± 2.69	2.91*	(1-2)(3-4)
MP (mm Hg)	255	252	200	202	± 9.59	3.42*	(1-2)(3-4)
HR (BPM)	225	220	203	201	± 8.88	1.01	
Post-epinephrine:							
SBP (mm Hg)	396	387	314	343	± 17.41	9.14**	(1-2)(3-4)
DBP (mm Hg)	258	254	207	217	± 11.63	7.35**	(1-2)(3-4)
PP (mm Hg)	137	133	107	130	± 9.26	3.84*	(1-2-4)
MP (mm Hg)	327	321	261	280	± 13.94	4.47**	(1-2)(3-4)
HR (BPM)	220	209	181	171	± 7.88	2.88	(1-2)(2-3)(3-4)
Percent change: [5]							
SBP	1.36	1.34	1.40	1.52	± 0.04	3.68*	(1-2-3)(3-4)
DBP	1.21	1.19	1.18	1.26	± 0.03	1.45	
PP	1.83	1.78	2.19	2.78	± 0.25	3.59*	(1-2-3)(3-4)
MP	1.28	1.27	1.31	1.39	± 0.03	1.55	
HR	1.01	0.97	0.93	0.86	± 0.02	3.06	(1-2-3)(3-4)

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Reserpine - CIBA.
3/ Numbers with one asterisk indicate significance at the P < 0.05 level while those numbers with two asterisks indicate significance at the P < 0.01 level.
4/ Numbers joined by a dash are non-significantly different at the P < 0.01 level.
5/ Percentage values indexed to base 1.00.

Experiment IV: Determination of Cardiac Output and Simultaneous Measurement of Cardiovascular and Hemodynamic Parameters Pre- and Post-Epinephrine Injection in Adult Male BBB Turkeys

The purpose of this experiment was to determine the cardiac output and to ascertain the effect of an intravenous injection (0.5 ml of 1/25,000 dilution) of epinephrine on cardiovascular and hemodynamic parameters of adult male BBB turkeys. Carotid and popliteal arterial blood pressures were recorded simultaneously with the cardiac output determinations. The turkeys were hatched and raised by conventional means by members of the M. S. U. Department of Poultry Science. At 30 weeks of age the turkeys were divided into 3 strains; the M.S.U. inbred strain, a commercial strain, and a cross between the M.S.U. and commercial strain. Each strain consisted of 10 male BBB turkeys. At this time, cardiovascular parameters were recorded pre- and post-epinephrine injection.

The cardiovascular parameters and atherosclerotic scores, and the common carotid blood pressures pre- and post-epinephrine and the percent change in these parameters are presented in Tables 7, 8, and 9 respectively. Analyses of the hemodynamic data indicated a statistical difference between strains, therefore, the data for each strain are listed separately in each table.

There was no strain difference in cardiovascular parameters or atherosclerotic scores or in the cardiovascular response to epinephrine. An average of the three strains established the cardiac output for the untreated mature male BBB turkey as 231 ml per $Kg^{0.734}$/min. In general, epinephrine injection caused a decreased cardiovascular response. Minute volume decreased due to a lowered heart rate. Stroke volume

Table 7. Cardiovascular parameters pre- and post-epinephrine[1/] of adult male BBB turkeys

	Strains			F value[2/]	S.E.M.
	Comm	Comm X MSU	MSU		
Pre-epinephrine:					
MV (ml)	1502	1739	1587	0.55	± 160.08
HR (BPM)	145	148	153	0.19	± 9.18
SV (ml)	10.63	12.08	10.38	0.58	± 1.22
$CO/Kg^{0.734}$ (ml)	216	248	227	0.81	± 17.90
CO/Kg (ml)	105	118	108	0.54	± 9.07
BW (Kg)	14.23	14.63	14.65	0.36	± 0.39
Aor. Sc.	2.13	2.24	1.82	0.81	± 0.25
Ht wt (gms)	57.65	55.97	57.03	0.23	± 1.74
Post-epinephrine:					
MV (ml)	1351	1468	1610	2.75	± 79.70
HR (BPM)	120	127	137	0.42	± 9.49
SV (ml)	11.81	11.55	11.78	0.01	± 0.57
$CO/Kg^{0.734}$ (ml)	217	252	252	1.26	± 19.14
CO/Kg (ml)	96	98	110	1.31	± 6.58
Percent change:[2/]					
MV	0.85	0.78	0.83	0.20	± 0.08
HR	0.84	0.85	0.82	0.06	± 0.04
SV	1.11	0.93	1.14	0.21	± 0.11
$CO/Kg^{0.734}$	0.93	0.96	0.94	0.02	± 0.10
CO/Kg	0.85	0.78	0.83	0.21	± 0.08

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Percentage values indexed to base 1.00.

Table 8. Common carotid hemodynamic parameters pre- and post-epinephrine[1/] of adult male BBB turkeys

	Strains					
	A Comm	B Comm X MSU	C MSU	F value[2/]	S.E.M.	Non-significance[3/]
Pre-epinephrine:						
SBP (mm Hg)	281	300	322	4.69*	± 9.44	(A-B)(B-C)
DBP (mm Hg)	193	204	213	1.49	± 8.20	
PP (mm Hg)	88	96	109	4.21*	± 5.16	(A-B)(B-C)
MP (mm Hg)	237	252	268	3.17	± 8.50	
HR (BPM)	145	148	153	0.19	± 9.18	
SRU/$Kg^{0.734}$	1.12	1.12	1.15	0.03	± 0.09	
SRU/bird	0.16	0.15	0.17	0.20	± 0.02	
Post-epinephrine:						
SBP (mm Hg)	379	393	419	3.61	± 10.40	
DBP (mm Hg)	238	248	257	0.46	± 13.29	
PP (mm Hg)	141	145	163	1.35	± 9.59	
MP (mm Hg)	309	321	338	1.69	± 10.93	
HR (BPM)	120	127	137	0.42	± 9.49	
SRU/$Kg^{0.734}$	1.78	1.40	1.38	1.79	± 0.16	
SRU/bird	0.27	0.23	0.21	0.71	± 0.06	
Percent change:[4/]						
SBP	1.33	1.30	1.27	0.68	± 0.04	
DBP	1.25	1.22	1.14	2.89	± 0.03	
PP	1.64	1.48	1.86	2.09	± 0.13	
MP	1.29	1.27	1.21	1.49	± 0.03	
HR	0.84	0.85	0.82	0.06	± 0.04	
SRU/$Kg^{0.734}$	1.53	1.20	1.44	0.40	± 0.28	
SRU/bird	1.51	1.25	1.58	0.44	± 0.28	

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Numbers with one asterisk indicate significance at the $P < 0.05$ level.
3/ Letters joined by a dash are non-significantly different at the P 0.05 level.
4/ Percentage values indexed to base 1.00.

Table 9. Popliteal hemodynamic parameters pre- and post-epinephrine[1] of adult male BBB turkeys

	Strains					
	A Comm	B Comm X MSU	C MSU	F value[2]	S.E.M.	Non-significance[3]
Pre-epinephrine:						
SBP (mm Hg)	266	285	304	4.03*	± 9.33	(A-B)(B-C)
DBP (mm Hg)	188	196	213	1.63	± 10.00	
PP (mm Hg)	78	86	91	0.65	± 7.96	
MP (mm Hg)	241	241	248	0.79	± 4.52	
HR (BPM)	145	148	153	0.19	± 9.18	
SRU/$Kg^{0.734}$	1.07	1.00	1.17	1.57	± 0.07	
SRU/bird	0.16	0.15	0.17	2.89	± 0.01	
Post-epinephrine:						
SBP (mm Hg)	359	380	390	1.53	± 11.01	
DBP (mm Hg)	232	233	231	0.02	± 8.03	
PP (mm Hg)	127	148	159	4.03*	± 7.02	
MP (mm Hg)	298	306	309	0.60	± 6.77	
HR (BPM)	120	127	137	0.42	± 9.49	
SRU/$Kg^{0.734}$	1.34	1.25	1.33	0.15	± 0.13	
SRU/bird	0.25	0.21	0.20	1.29	± 0.02	
Percent change:[4]						
SBP	1.36	1.33	1.25	0.82	± 0.06	
DBP	1.30	1.15	1.09	2.22	± 0.06	
PP	1.48	1.74	1.64	1.58	± 0.10	
MP	1.27	1.25	1.26	0.05	± 0.04	
HR	0.84	0.85	0.82	0.06	± 0.04	
SRU/$Kg^{0.734}$	1.21	1.17	1.14	0.12	± 0.10	
SRU/bird	1.30	1.28	1.22	0.20	± 0.96	

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Numbers with one asterisk indicate significance at the $P < 0.05$ level.
3/ Letters joined by a dash are non-significantly different at the $P > 0.05$ level.
4/ Percentage values indexed to base 1.00.

increased slightly but not sufficiently to maintain the pre-epinephrine cardiac output/$Kg^{0.734}$.

The M.S.U. strain had a significantly higher carotid and popliteal blood pressure than did the commercial strain. The systolic blood pressure of the commercial-M.S.U. cross was intermediate between the two strains alone. The pulse pressures were high especially in the M.S.U. strain; however, this is apparently normal for the adult tom. It was also observed that the common carotid arterial systolic blood pressure was consistently higher than the systolic blood pressure in the popliteal artery. Since the birds were in a supine position during the blood pressure determinations, the difference between carotid and popliteal arterial blood pressures was attributed to the greater peripheral resistance encountered by the blood passing from the heart to the popliteal artery. Perhaps this increased resistance is encountered in the abdominal aorta, a major site for obstructive atherosclerotic lesions. Following the injection of epinephrine, systolic and diastolic blood pressures increased about the same in the common carotid and popliteal arteries. Pulse pressure increased most in the group with the highest pulse pressure (MSU group). Common carotid pulse pressures also exhibited a greater percentage increase following epinephrine injection than did the popliteal pulse pressures. This further indicates an area of reduced distensibility between the heart and the popliteal artery.

The marked difference in aortic atherosclerotic severity may have been influential in the cardiovascular and hemodynamic response to epinephrine injection. Hence, the results were divided into two groups, namely, those birds with an aorta score less than 2.00 and those birds with an aorta score of 2.00 or greater.

The cardiovascular parameters of adult male BBB turkeys with an aorta score above or below 2.00 and their response to epinephrine injection are presented in Table 10. The group with the higher aortic score had a significantly higher minute volume and a significantly higher cardiac output than did the low incidence group. Since the heart rates of the high and low incidence groups were essentially the same, the stroke volume was higher in the group with the higher incidence of atherosclerosis. Following the injection of epinephrine, the group with the higher aortic score demonstrated the greater cardiovascular response. This response was noted only in minute volume and cardiac output which decreased 25 and 27 percent respectively as compared to the low incidence group which decreased only 17 percent for both minute volume and cardiac output.

The common carotid and popliteal hemodynamic endpoints of adult male BBB turkeys with an aorta score above or below 2.00 and their response to epinephrine injection are presented in Table 11. The group with the higher aorta score exhibited a higher mean common carotid blood pressure which was due mainly to an increased systolic blood pressure. Popliteal blood pressures did not exhibit as noticeable an increase as did the common carotid blood pressures. The group with the least severity of atherosclerosis demonstrated a significantly greater percentage increase in common carotid and popliteal mean blood pressure following the injection of epinephrine than did the high incidence group. The greatest percentage increase was in systolic blood pressure which was reflected by an elevated pulse pressure. It was also noted that the injection of epinephrine increased the difference between common carotid and popliteal hemodynamic parameters whether in the high or low incidence group.

Table 10. Cardiovascular parameters pre- and post-epinephrine[1/] of adult male BBB turkeys

	Aorta score < 2.00	Aorta score > 2.00	S.E.M.
No. birds	8	12	
Pre-epinephrine:			
MV (ml)	1322	1723**	± 142.22
HR (BPM)	151	152	± 9.68
SV (ml)	9.05	13.12*	± 1.32
$CO/Kg^{0.734}$ (ml)	196	241**	± 17.30
BWT (Kg)	14.23	14.80	± 1.82
Aor.Sc.[2/]	1.69	2.57**	± 0.35
Post-epinephrine:			
MV (ml)	1097	1292	± 62.44
HR (BPM)	133	137	± 7.73
SV (ml)	7.51	10.89**	± 0.69
$CO/Kg^{0.734}$ (ml)	163	176	± 10.21
Percent change:[3/]			
MV	0.83	0.75	± 0.08
HR	0.88	0.90	± 0.09
SV	0.83	0.83	± 0.05
$CO/Kg^{0.734}$	0.83	0.73	± 0.04

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ 0 = no incidence; 4 = high incidence.
3/ Percentage values indexed to base 1.00.
* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

Table 11. Common carotid and popliteal hemodynamic parameters pre- and post-epinephrine[1/] of adult male BBB turkeys

	Carotid			Popliteal		
	Aor Sc <2.00	Aor Sc >2.00	S.E.M.	Aor Sc <2.00	Aor Sc >2.00	S.E.M.
No. birds	8	8		12	12	
Pre-epinephrine:						
SBP (mm Hg)	288	312	± 8.84	288	286	± 7.72
DBP (mm Hg)	195	210	± 7.60	213	196	± 6.70
PP (mm Hg)	94	101	± 4.56	75	87	± 4.01
MP (mm Hg)	242	261	± 7.90	250	241	± 7.66
HR (BPM)	151	152	± 8.58	151	152	± 8.58
Post-epinephrine:						
SBP (mm Hg)	412	390	± 9.80	395	358**	± 8.89
DBP (mm Hg)	238	248	± 12.69	275	233**	± 8.14
PP (mm Hg)	161	154	± 8.99	129	119	± 8.22
MP (mm Hg)	317	318	± 10.33	335	292*	± 10.07
HR (BPM)	169	167	± 8.89	169	167	± 8.89
Percent change:[2/]						
SBP	1.43	1.25*	± 0.05	1.37	1.25*	± 0.05
DBP	1.22	1.18	± 0.03	1.29	1.19	± 0.03
PP	1.71	1.52*	± 0.07	1.72	1.37**	± 0.06
MP	1.31	1.22	± 0.03	1.34	1.21*	± 0.03
HR	1.12	1.10	± 0.02	1.17	1.10	± 0.02

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Percentage values indexed to base 1.00.
* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

Experiment V: Determination of Thoracic and Abdominal Aortic Elastance at Pressures Below, Equal to and Above the Physiological Blood Pressure Range

For this study, 120 mature male BBB turkeys were divided into 6 groups and placed on experiment. They were reared in conventional housing and they received feed and water ad libitum. The study was designed to accomplish several objectives at the same time. Group 1 was a heat treatment group. Untreated toms were taken from the outside during the winter (temperature range -10° F to 20° F) and subjected to a temperature of 100° F and a relative humidity of 65-75 percent for four hours. Circulatory dynamics were recorded immediately after the heat treatment. In chickens an increase in body temperature has been reported to decrease blood pressure (Rodbard and Tolpin, 1947) and cardiac output, but increase systemic resistance (Sturkie and Vogel, 1961). The turkeys in Group 2 were placed on a 0.05 percent BAPN supplemented ration for four weeks. BAPN interferes with normal collagen metabolism and has been demonstrated to produce aortic rupture in turkeys. Group 3 was actually a subdivision of Group 2. After 4 weeks on a BAPN supplemented ration one-half the turkeys in Group 2 were placed on a 0.05 percent BAPN plus 1 ppm reserpine supplemented ration for one week. Reserpine, a commercial tranquilizer, at 1 ppm in the ration, has been shown effective against aortic rupture in the field. The turkeys in Group 4 were placed on a 1 ppm reserpine-supplemented ration for one week. The turkeys in Group 5 were placed on 100 mg of ascorbic acid (Vitamin C) per pound of feed for four weeks. Ascorbic acid is essential for normal collagen metabolism. Group 6 consisted of untreated toms which were used only for elastance studies.

At the end of the treatment period, the toms were individually placed on a wooden restraining holder and the cardiac output and common carotid and popliteal arterial blood pressures recorded simultaneously. Following these measurements 0.5 ml of a 1/25,000 dilution of epinephrine was injected into the bird and the same cardiovascular parameters were measured. Each bird therefore served as its own control. When these measurements were completed, the turkey was sacrificed and the entire aorta removed. Thoracic and abdominal aortic segments were removed, cleaned, their branches tied and their elasticity measured. The remaining portion of the aorta was cut open and visually scored for atherosclerotic severity. Following the determination of elastance, the abdominal aorta was also scored.

The results are presented in Tables 12-30. Cardiovascular and elasticity parameters will be discussed separately. The correlation tables will also be discussed separately to relate these parameters to each other. Since there was no interaction between strains and treatments, in any of the evaluations, it is assumed that all the strains reacted similarly to the treatments. BAPN does not influence hemodynamic parameters (Ringer, 1961) therefore the BAPN group could be used as a control for the cardiovascular and hemodynamic determinations.

The cardiovascular parameters of adult male BBB turkeys pre- and post-epinephrine injection and their percent change are presented in Tables 12, 13 and 14, respectively. Before the injection of epinephrine the only strain difference that existed was in heart rate and stroke volume. In this respect, the M.S.U. strain had a higher heart rate and therefore a lower stroke volume than did the commercial strain. The heart rate of the heat and BAPN groups was highest; however, reserpine

alone or when added to the BAPN group significantly decreased the heart rate. Although reserpine lowered the heart rate, stroke volume was increased sufficiently to produce a significantly higher cardiac output per $Kg^{0.734}$ than was found in the other treatments. Heart weight was lowest in the BAPN group and the weight was not altered by reserpine administration.

Following the injection of epinephrine there was no strain difference in cardiovascular parameters. In general, all cardiovascular parameters decreased, probably due to the increased peripheral resistance. Minute volume was significantly decreased in the BAPN group which was not mediated as much by a reduction in heart rate as it was by a reduction in stroke volume. The BAPN group, therefore, had the lowest cardiac output per $Kg^{0.734}$. Reserpine and vitamin C did prevent minute volume and hence cardiac output per $Kg^{0.734}$ from dropping as much as that in the BAPN and heat groups following the injection of epinephrine.

The common carotid and popliteal hemodynamic parameters of adult male BBB turkeys pre- and post-epinephrine and their percent change are reported in Tables 15-17 and 18-20, respectively. Prior to the injection of epinephrine there were strain and treatment differences in all of the carotid and popliteal hemodynamic parameters. The M.S.U. strain had the highest hemodynamic parameters and the commercial strain the lowest with the cross between the two strains falling somewhere in between.

In general, before the injection of epinephrine, the popliteal hemodynamic parameters were lower than those of their carotid artery counterparts. Again, this may well reflect a pressure reduction over an area of increased resistance such as the atherosclerotic lesions

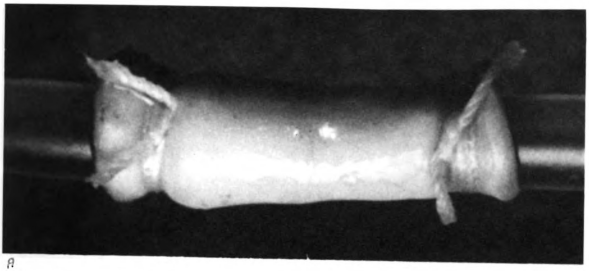
which develop spontaneously in the abdominal aorta during aging. In both the carotid and popliteal blood pressures, the BAPN and heat treated group had a normal mean blood pressure; whereas, reserpine alone or in combination with BAPN, significantly reduced the mean blood pressure. Reserpine accomplished this decrease in mean blood pressure principally through a reduction in diastolic, and to a lesser extent, systolic blood pressure. Reserpine also significantly lowered the systemic resistance. Pulse pressures were significantly lowered in the vitamin C group. This was accomplished through a reduction in systolic blood pressure and an increase in diastolic blood pressure. Since diastolic blood pressure is dependent upon the elastic recoil of the aorta and since vitamin C promotes collagen anabolism, the increased diastolic pressure was most likely brought about by the increased number of collagen fibers in the thoracic aorta.

Following the injection of epinephrine, significant strain differences were found only in systolic, diastolic and mean pressures. Again in both carotid and popliteal arteries, the M.S.U. strain reached higher pressures following epinephrine injection than did the commercial strain, probably because they were higher initially. When epinephrine was injected, the hemodynamic parameters of all treatments increased. Although the reserpine treated group had one of the greater percentage increases in mean pressure following the injection of epinephrine, it began at a lower level and the absolute value attained still tended to be lower than the mean pressures of the other treatments. Heat for some reason significantly elevated the epinephrine-induced mean pressure above all other treatments. This was accomplished mainly through an increased systolic blood pressure. Heat, however,

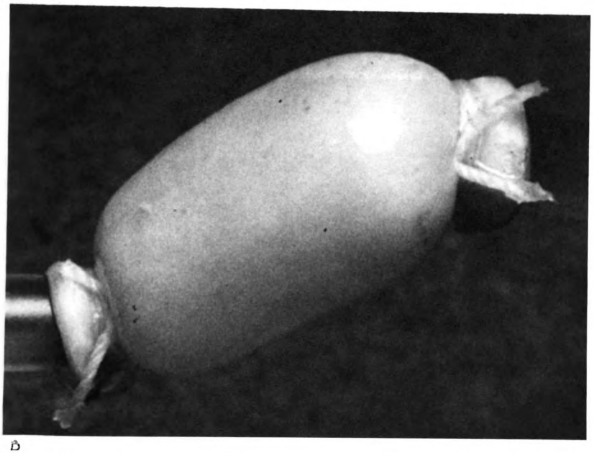
did not significantly alter the heart output (minute volume) in relation to the other treatments and, therefore, did not lower the calculated systemic resistance. Since epinephrine is a peripheral vasoconstrictor, carotid and popliteal systemic resistance units increased as expected, but less so in the BAPN and vitamin C groups. This increased systemic resistance had a negative affect on cardiovascular parameters.

In reporting the static elastic properties of the arterial wall, the values were divided into three pressure ranges. Elastances were calculated at 25 mm Hg intervals at pressures below the normal physiological systolic blood pressure range (25-150 mm Hg), at the normal physiological systolic blood pressure range (175-350 mm Hg), and above the normal physiological systolic blood pressure range (375-500 mm Hg). The mean elastance values under the various treatments at pressures below, at and above the physiological systolic blood pressure range are presented in Tables 21-23, respectively for the thoracic aortas and in Tables 24-26, respectively for the abdominal aortas.

In general, throughout all the pressures the thoracic aortas were more distensible than the abdominal aortas and this became more pronounced at higher pressure values (Figs. VI, VII). These results were expected as collagen has a much higher elastance value when compared to elastin (Bergel, 1961) and collagen comprises the majority of the abdominal aorta whereas elastin predominates in the thoracic aorta (Hass, 1942). All values reported herein are for a 20 mm length of aorta. Increasing the length to 30 mm lowers, whereas decreasing the length to 10 mm increases, the elastance value (personal observation). A piece of pure rubber tubing 20 mm in length and of the same approximate diameter and thickness as that of the average aorta was subjected



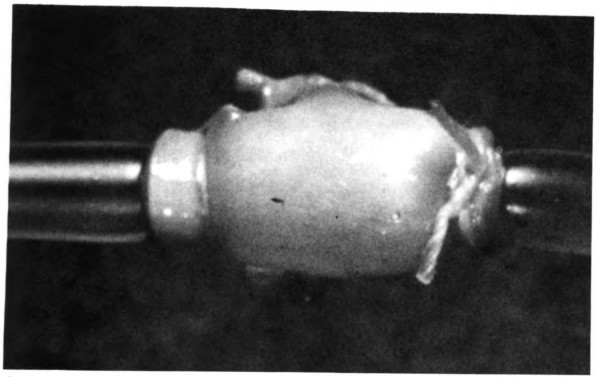
A



B

Figure VI. Photograph illustrating the distensibility of the thoracic aorta at 0 mm Hg (A) and at 500 m. Hg (B).

A



B

Figure VII. Photograph illustrating the distensibility of the abdominal aorta at 0 mm Hg (A) and at 500 mm Hg (B).

to pressures and the elastances calculated. The rubber tubing was much more elastic than either the thoracic or abdominal aortas, especially at higher pressures where it was 2.5 times more elastic than the abdominal aorta and over 10 times more elastic than the thoracic aorta.

The data agree with elastances calculated for thoracic aortas of rabbits (Balkrishna et al., 1961), and dogs (Peterson, et al. 1960) but not with elastance values reported by Bergel (1961) for dogs. Bergel (1961) used 60 mm lengths of aorta, which were much longer than those used in this experiment and may account for the differences. Bergel also reported no difference between elastances of the thoracic and abdominal aorta whether below, at or above the systolic blood pressure of the dog. The data of this experiment demonstrates a slight difference in elastances between thoracic and abdominal aortas at low pressure ranges. This difference becomes magnified as the pressure is increased until at 500 mm Hg the abdominal aorta is 5-6 times more elastic than is the thoracic aorta.

At pressures below the normal physiological systolic blood pressure range of the adult male BBB turkey the untreated and vitamin C group thoracic aortas had the highest elasticity. The vitamin C group had significantly higher elastances at pressures above 100 mm Hg than did the other groups. BAPN decreased elasticity and reserpine added to BAPN or reserpine alone improved the elastance values, especially at the higher pressures, but they did not approach the untreated elastance values. In the abdominal aorta, reserpine alone maintained elastance at the untreated and vitamin C level but it did not overcome the lowering of elastance by BAPN. In the abdominal aorta, however,

heat treatment tended to improve elastance whereby it decreased elastance in the thoracic aorta.

At pressures within the normal physiological systolic blood pressure range both vitamin C and heat treated thoracic aortas had improved elastances over the untreated values. BAPN or reserpine at this pressure range apparently did not influence elastance values although reserpine tended to decrease elastance. In the abdominal aorta BAPN again lowered elastance values and reserpine in combination with BAPN offered no protection. Reserpine alone, however, maintained elastance values at the untreated level which differed from its effect on the thoracic aorta. Vitamin C and heat treatment again significantly improved elastances.

Tables 23 and 26 may be termed stress tables for these present the pressures attained by tom turkeys during stress or excitement. In our laboratory, temporary pressures up to and in excess of 500 mm Hg have been produced in mature male BBB turkeys by injection of 0.5 ml of 1/25,000 epinephrine. At pressures above the normal physiological systolic blood pressure range the beneficial effect of vitamin C on the thoracic aorta was most noted. Heat treated groups still remained higher than the untreated lots, but did not reach the vitamin C values. BAPN did not seem to decrease elasticity whereas reserpine did tend to reduce elasticity. When the thoracic aortas were subjected to pressures above 400 mm Hg, however, 30 percent of the aortas ruptured in the BAPN lot, 25 percent in the BAPN plus reserpine lot, 10 percent in the reserpine lot and only 5 percent in the untreated and vitamin C lot. No aortas were ruptured in the heat treated lots. In the abdominal aorta the vitamin C lot had the highest elastance value. The aortas of the

birds in the untreated and heat treatments had the same elastances. BAPN alone and in combination with reserpine continued to significantly lower elastance values. The reserpine group had elastance values between the BAPN and the untreated groups. Also of interest was the fact that it was not possible to rupture any abdominal aorta under any treatment, even when pressures of 500 mm Hg were exceeded.

The results of three successive pressure volume (Hysteresis) curves obtained from the same segment of thoracic and abdominal aorta are presented in Figures VIII and IX, respectively. During successive inflations, all vessels became progressively larger. In the thoracic aorta the first cycle of inflation and deflation resulted in a wide loop, and at the end, the zero-pressure volume had increased considerably. The width between the initial inflation and deflation was not as wide as the initial cycle reported by Bergel (1961), however, the size of the aortic segment was also smaller (20 mm) than that reported in 1961 by Bergel (60 mm) for dogs. The inflation curve of the initial cycle demonstrates that the thoracic aorta of the male BBB turkey has a linear distensibility up to a high pressure range and that the thoracic aorta was most distensible in the physiological systolic blood pressure range. As the hysteresis loops were repeated they became closer together until after the third cycle they were almost identical.

Analysis of the hysteresis curve for the abdominal aorta shows that it is much more elastic than is the thoracic aorta, probably due to its greater amount of collagen. Again the loops became narrower and closer together with successive cycles. The abdominal aorta is quite distensible up to a pressure of 100 mm Hg after which the pressure volume curve begins to plateau as more and more connective tissue elements are brought into play.

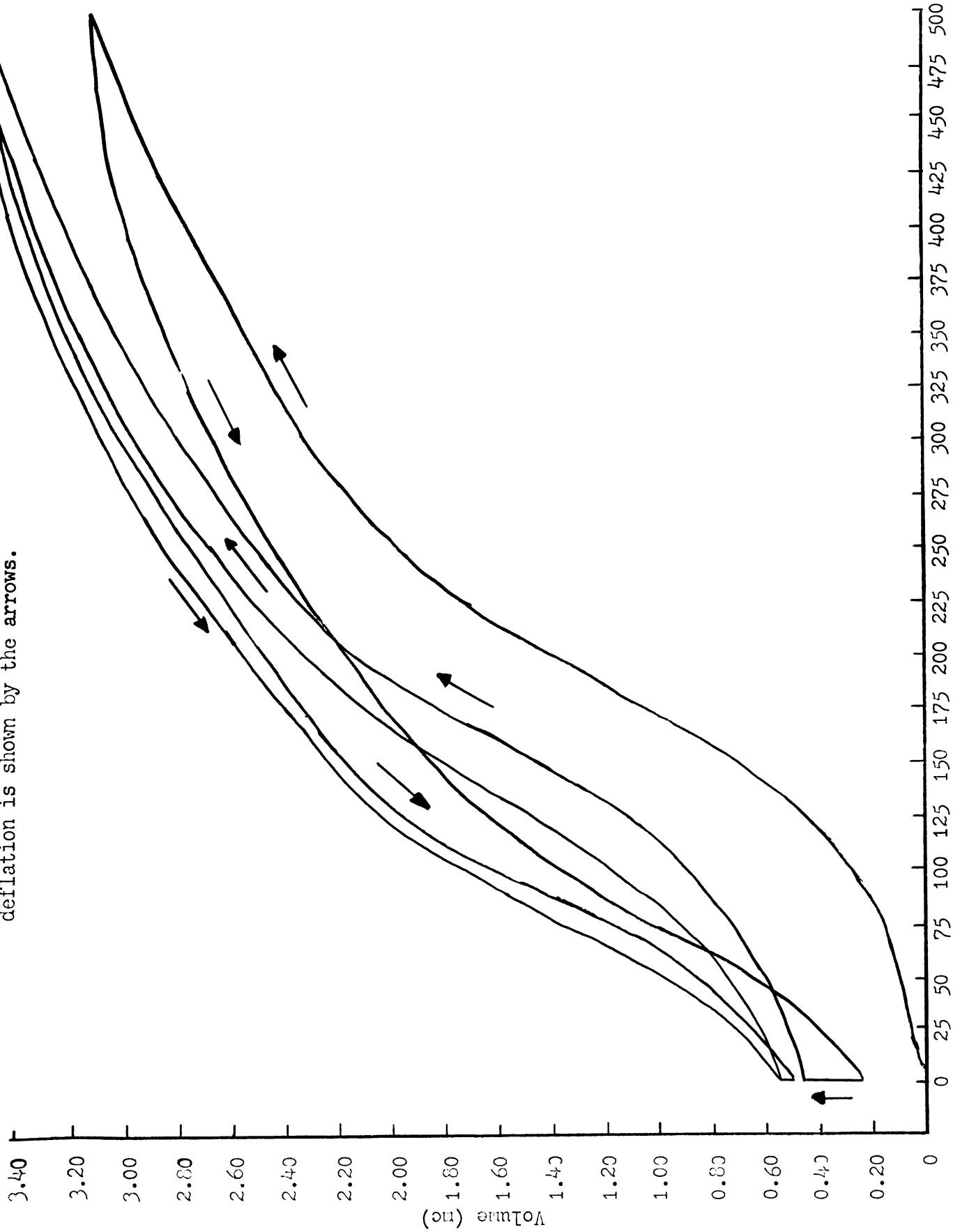


Figure VIII. Three consecutive pressure-volume curves of a thoracic aorta. The sequence of inflation and deflation is shown by the **arrows.**

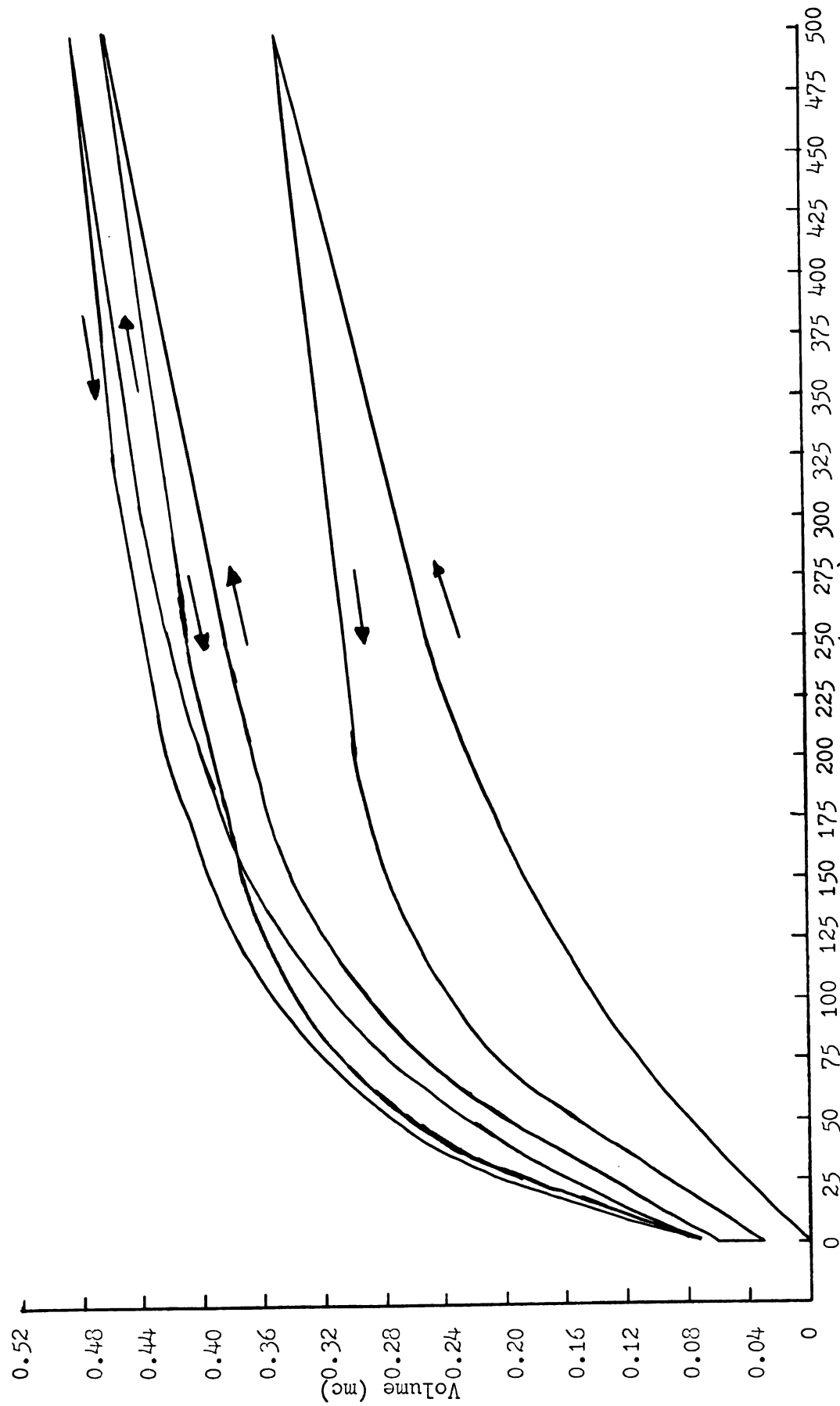


Figure IX. Three consecutive pressure-volume curves of an abdominal aorta. The sequence of inflation and deflation is shown by the arrows.

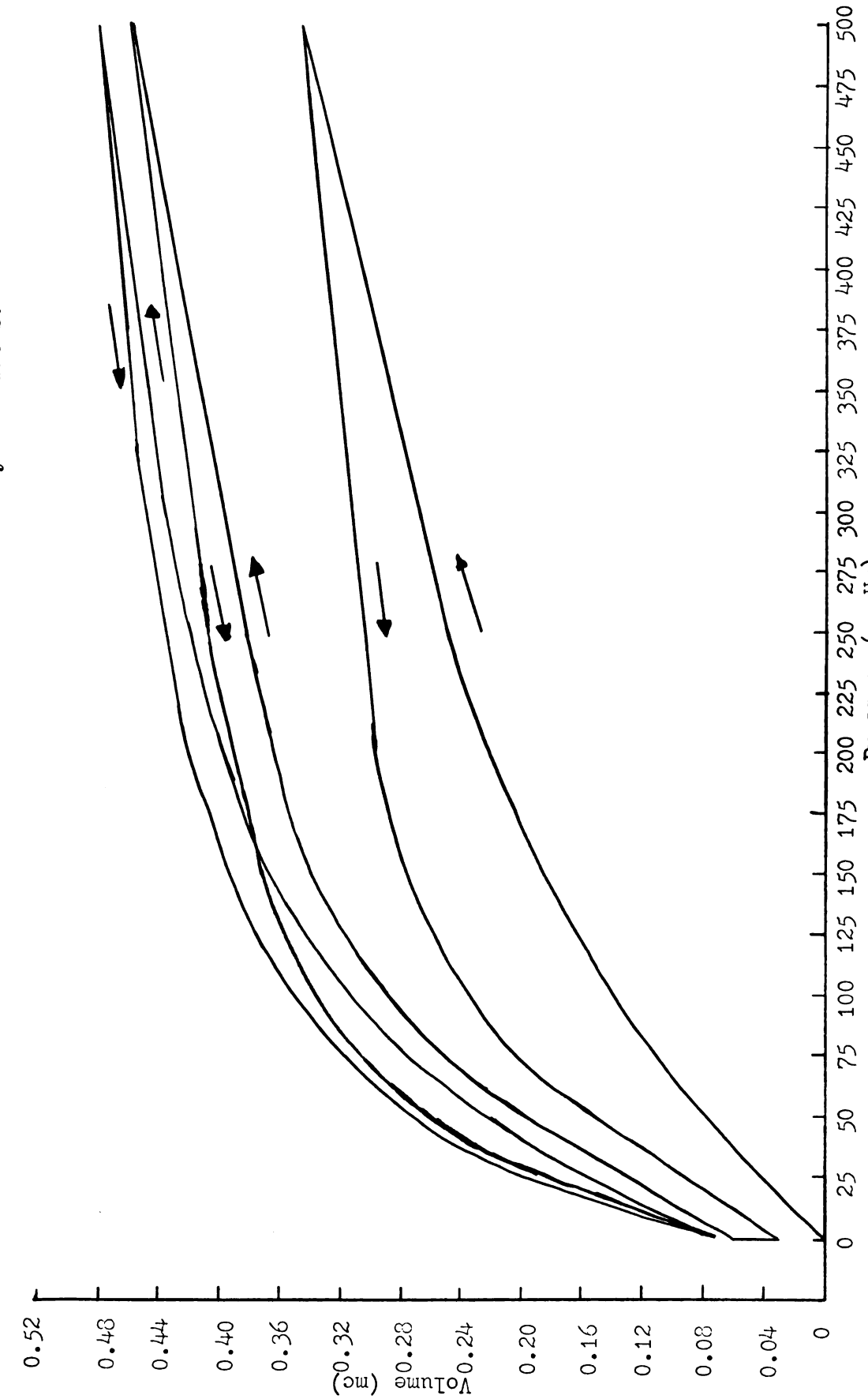
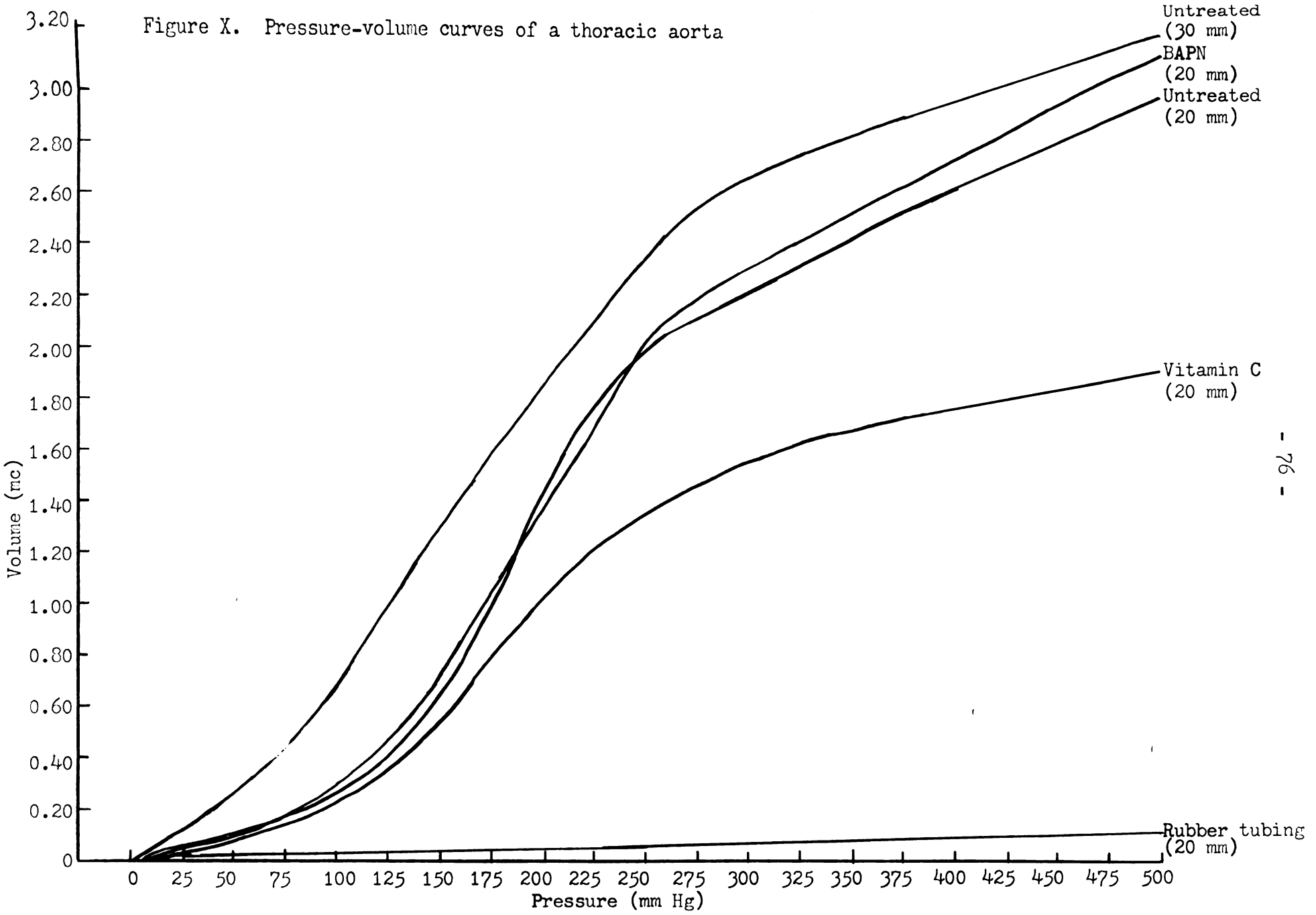


Figure IX. Three consecutive pressure-volume curves of an abdominal aorta. The sequence of inflation and deflation is shown by the arrows.

Typical pressure-volume curves of untreated, BAPN, and vitamin C treated thoracic and abdominal aortas are presented in Figures X and XI, respectively. A piece of pure rubber tubing, identical in size to the aortic segment used is shown for comparison. Ascorbic acid appears to have increased the elasticity of the thoracic and abdominal aortas whereas BAPN treatment had no effect in the thoracic aorta but did, however, decrease the elasticity of the abdominal aorta. Increasing the length of either thoracic or abdominal aortic segments from 20 to 30 mm markedly increased their distensibility. Both aortic segments were considerably more distensible than a 20 mm length of pure rubber tubing with a thickness comparable to the vessels used in this experiment.

Partial correlation coefficients of three variates of cardiovascular parameters are presented in Tables 27 and 28. Systolic blood pressure was positively correlated with diastolic blood pressure, heart rate and blood vessel thickness. Williams et al. (1962) also found a significant positive correlation between systolic blood pressure and total aortic thickness or the ratio of intimal to total thickness. Fregly (1962) found a positive relationship between systolic blood pressure and heart weight in rats. Diastolic blood pressure was positively correlated with heart rate and blood vessel thickness but demonstrated a negative correlation with heart weight. Blood vessel thickness was positively correlated with pulse pressure but negatively correlated with heart rate. Pulse pressure, heart weight and percent change in systolic blood pressure following the injection of epinephrine were all positively correlated with body weight whereas systolic blood pressure, heart rate and thoracic elastance at 250 mm Hg were negatively correlated

Figure X. Pressure-volume curves of a thoracic aorta



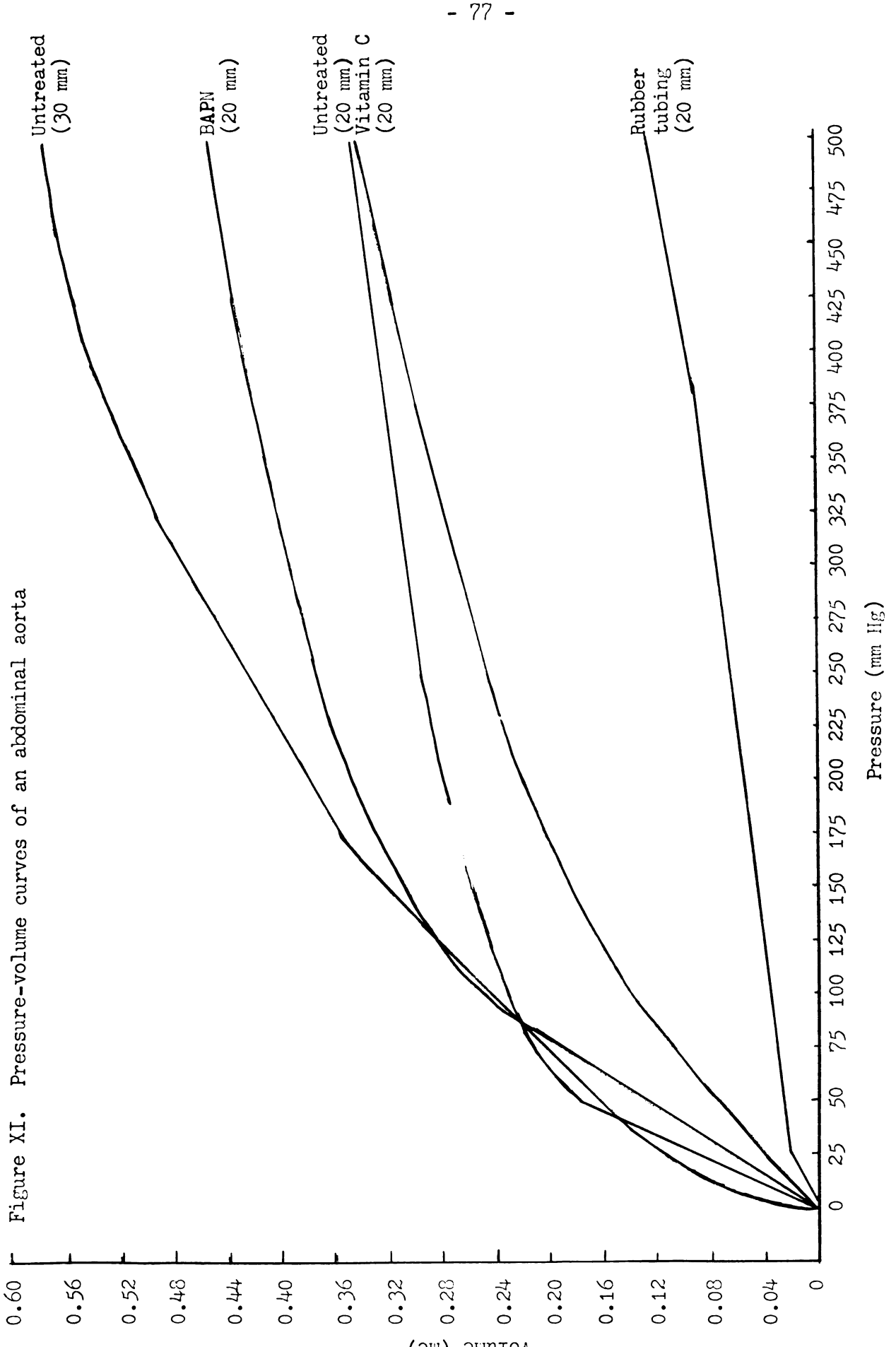


Figure XI. Pressure-volume curves of an abdominal aorta

with body weight. Minute volume was positively correlated with stroke volume and negatively correlated with heart rate whereas stroke volume was positively correlated with heart rate but negatively correlated with systolic blood pressure and blood vessel thickness. The percent change in systolic blood pressure was negatively correlated with diastolic blood pressure, pulse pressure, blood vessel thickness and thoracic elastance at 250 mm Hg.

Correlation coefficients of two variates are presented in Tables 29 and 30. In general, it was found that pulse pressure was positively correlated with diastolic blood pressure and thoracic elastance at 250 mm Hg but negatively correlated with the percent change in systolic blood pressure subsequent to the injection of epinephrine. The percent change in the systolic blood pressure of the popliteal artery following epinephrine injection was positively correlated with minute volume and negatively correlated with diastolic blood pressure. Abdominal elastance or atherosclerotic severity could not be correlated with any cardiovascular or hemodynamic parameters.

Table 12. Cardiovascular parameters of adult male BBB turkeys

Strains	Treatments	No. birds	MV (ml)	HR (BPM)	SV (ml)	CO/Kg (ml)	$CO/Kg^{0.734}$ (ml)	BW (Kgm)	Aor Sc.[1]	Ht.Wt. (gms)
A Comm										
	1. Heat	7	1877	176	10.68	258	127	14.80	2.33	58.07
	2. BAPN	7	1647	171	9.68	247	124	13.29	1.37	50.11
	3. BAPN + Reserpine	7	1695	120	14.39	250	125	13.49	1.28	55.10
	4. Reserpine	6	1534	141	10.96	232	120	12.72	1.43	51.67
	5. Vitamin C	6	1755	150	11.92	249	123	14.34	1.88	61.25
B Comm X MSU										
	1. Heat	7	1735	161	11.00	251	125	13.89	1.35	58.90
	2. BAPN	6	1753	165	10.80	256	129	13.65	1.99	52.07
	3. BAPN + Reserpine	6	1715	126	13.72	260	132	13.02	0.99	54.33
	4. Reserpine	7	1724	145	12.09	244	125	13.75	1.03	56.49
	5. Vitamin C	6	1628	143	11.56	246	124	13.03	1.33	51.90
C MSU										
	1. Heat	6	1779	193	9.42	252	123	14.49	2.01	58.83
	2. BAPN	7	1508	191	7.97	230	117	12.92	1.07	49.93
	3. BAPN + Reserpine	7	1770	124	14.52	259	127	13.91	1.41	56.11
	4. Reserpine	7	1607	148	11.43	237	119	13.38	1.29	57.14
	5. Vitamin C	6	1748	170	10.30	262	132	13.30	1.50	55.45
F value[2]										
Strains			0.68	4.83*	2.09*	1.06	1.46	0.01	1.09	0.07
Treatments			1.54	20.90**	8.79**	2.44*	3.67**	5.08**	4.41**	4.96**
S.E.M.										
Strains			±13.61	±3.25	±0.40	±4.62	±2.34	±0.18	±0.12	±0.88
Treatments			±61.08	±4.61	±0.57	±6.56	±3.32	±0.25	±0.17	±1.25
Non-significance[3]										
Strains			--	(A-B)	(A-B)(B-C)	--	--	--	--	--
Treatments			--	(1-2)(4-5)	(1-2-4-5)	(1-2-3-5)	(2-4) (1-3-5)	(1-3-5) (2-3-4-5)	(2-3-4-5)	(2-3-4) (1-3-4-5)

1/ Aortic score 0 = no incidence; 4 = high incidence.

2/ Numbers with one asterisk indicate significance at the $P < 0.05$ level while those numbers with two asterisks indicate significance at the $P < 0.01$ level.

3/ Numbers and letters joined by a dash are non-significantly different at the $P < 0.01$ level.

Table 13. Cardiovascular parameters following the injection of epinephrine[1] to adult male BBB turkeys

Strains	Treatments	No. birds	MV (ml)	HR (BPM)	SV (ml)	CO/Kg (ml)	$CO/Kg^{0.734}$ (ml)
A Comm							
	1. Heat	7	1252	144	8.82	172	84
	2. BAPN	6	1107	152	7.55	168	84
	3. BAPN + Reserpine	6	1209	141	8.49	179	87
	4. Reserpine	3	1310	143	9.27	177	99
	5. Vitamin C	4	1335	142	9.94	189	93
B Comm X MSU							
	1. Heat	7	1168	141	8.52	169	84
	2. BAPN	3	1104	150	7.27	164	83
	3. BAPN + Reserpine	6	1193	146	8.20	181	92
	4. Reserpine	7	1300	157	8.50	179	95
	5. Vitamin C	3	1208	121	10.98	183	92
C MSU							
	1. Heat	4	1211	136	9.50	173	84
	2. BAPN	2	1109	162	5.97	163	82
	3. BAPN + Reserpine	5	1263	150	8.56	186	91
	4. Reserpine	5	1341	147	8.62	192	95
	5. Vitamin C	4	1353	130	10.64	196	102
F value[2]							
Strains			2.73	0.22	0.38	2.59	1.94
Treatments			8.49**	2.07	9.04**	17.74**	6.80**
S.E.M.							
Strains			± 26.65	± 5.14	± 0.34	± 3.86	± 1.83
Treatments			± 37.77	± 7.30	± 0.48	± 5.47	± 2.59
Non-significance[3]							
Strains			--	--	--	--	--
Treatments			(1-2-3) (1-3-4-5)	--	(1-2-3-4) (1-4-5)	(1-2-3-4) (1-3-4-5)	(1-2-3) (3-4-5)

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
3/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 14. Percent change[1] in cardiovascular parameters following the injection of epinephrine[2] to adult male BBB turkeys

Strains	Treatments	No. birds	MV	HR	SV	0.734 CO/Kg	CO/Kg
A Comm							
	1. Heat	7	0.67	0.82	0.83	0.67	0.66
	2. BAPN	6	0.67	0.88	0.77	0.67	0.67
	3. BAPN + Reserpine	6	0.71	1.19	0.61	0.71	0.69
	4. Reserpine	3	0.81	1.02	0.79	0.76	0.81
	5. Vitamin C	4	0.76	0.93	0.82	1.25	0.76
B Comm X MSU							
	1. Heat	7	0.68	0.89	0.77	0.68	0.68
	2. BAPN	3	0.68	0.91	0.80	0.68	0.67
	3. BAPN + Reserpine	6	0.70	1.17	0.60	0.70	0.69
	4. Reserpine	7	0.76	1.10	0.73	0.74	0.76
	5. Vitamin C	3	0.76	0.84	0.93	0.76	0.76
C MSU							
	1. Heat	4	0.67	0.52	0.97	0.68	0.67
	2. BAPN	2	0.67	0.84	0.73	0.67	0.67
	3. BAPN + Reserpine	5	0.71	1.22	0.59	0.71	0.70
	4. Reserpine	5	0.76	1.03	0.64	0.76	0.76
	5. Vitamin C	4	0.79	0.77	1.05	0.77	0.79
F value[3]							
	Strains		0.38	1.40	1.08	0.33	0.41
	Treatments		7.06**	12.75**	12.31**	13.75**	7.38**
S.E.M.							
	Strains		± 0.02	± 0.03	± 0.03	± 0.02	± 0.02
	Treatments		± 0.02	± 0.05	± 0.04	± 0.02	± 0.02
Non-significance[4]							
	Strains		--	--	--	--	--
	Treatments		(1-2-3) (3-4-5)	(1-2-5) (2-4)(3-4)	(1-2-4) (1-5)(3-4)	(1-2-3-4)	(1-2-3) (3-4-5)

1/ Percentage values indexed to base 1.00.
2/ Injection of 1/25,000 epinephrine.
3/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
4/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 15. Common carotid hemodynamic parameters of adult male BBB turkeys

Strains	Treatments	No. birds	SBP (mm Hg)	DBP (mm Hg)	PP (mm Hg)	MP (mm Hg)	HR (BPM)	SRU/ $Kg^{0.734}$	SRU/ bird
A Comm									
	1. Heat	7	243	171	72	207	176	0.81	0.11
	2. BAPN	7	254	169	85	212	171	0.85	0.13
	3. BAPN + Reserpine	7	204	131	73	168	120	0.68	0.10
	4. Reserpine	6	191	131	60	161	141	0.70	0.11
	5. Vitamin C	6	213	169	44	191	150	0.77	0.11
B Comm X MSU									
	1. Heat	7	256	180	76	217	161	0.87	0.13
	2. BAPN	6	270	180	90	225	165	0.89	0.13
	3. BAPN + Reserpine	6	218	128	90	173	126	0.67	0.10
	4. Reserpine	7	228	147	81	187	145	0.77	0.11
	5. Vitamin C	6	235	177	58	206	143	0.87	0.14
C MSU									
	1. Heat	6	286	198	88	242	193	0.97	0.14
	2. BAPN	7	284	185	99	235	191	1.02	0.16
	3. BAPN + Reserpine	7	245	142	88	194	124	0.81	0.12
	4. Reserpine	7	243	164	79	204	148	0.88	0.13
	5. Vitamin C	6	268	209	59	238	170	0.95	0.14
F value[1]									
	Strains		25.51**	17.23**	13.81**	25.18**	4.83*	8.13**	10.25**
	Treatments		28.40**	43.92**	17.67**	36.61**	20.90**	16.67**	21.75**
S.E.M.									
	Strains		± 4.26	± 2.84	± 2.34	± 3.34	± 3.25	± 0.03	± 0.005
	Treatments		± 6.04	± 4.03	± 3.32	± 4.73	± 4.61	± 0.04	± 0.003
Non-significance[2]									
	Strains			(A-B)	(B-C)	--	(A-B)	(A-B)(B-C)	(A-B)(B-C)
	Treatments		(1-2)(1-5) (3-4-5)	(3-4) (1-2-5)	(2-3) (1-3-4)	(3-4) (1-2-5)	(1-2) (4-5)	(3-4) (1-2-4-5)	(3-4) (1-2-5) (1-4-5)

1/ Numbers with one asterisk indicate significance at the $P < 0.05$ level while those numbers with two asterisks indicate significance at the $P < 0.01$ level.

2/ Numbers and letters joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 16. Common carotid hemodynamic parameters following the injection of epinephrine[1] to adult male BBB turkeys

Strains	Treatments	No. birds	SBP (mm Hg)	DBP (mm Hg)	PP (mm Hg)	MP (mm Hg)	HR (BPM)	SRU/ $Kg^{0.734}$	SRU/ Bird
A Comm									
	1. Heat	7	433	268	165	351	144	2.06	0.29
	2. BAPN	7	403	249	155	326	152	1.97	0.30
	3. BAPN + Reserpine	7	407	249	158	328	141	1.86	0.28
	4. Reserpine	6	362	242	120	302	143	1.82	0.25
	5. Vitamin C	6	379	238	141	309	120	1.78	0.27
B Comm X MSU									
	1. Heat	7	445	280	165	363	141	2.14	0.31
	2. BAPN	6	409	257	152	333	150	2.04	0.30
	3. BAPN + Reserpine	6	408	249	160	327	146	1.81	0.27
	4. Reserpine	7	410	249	161	330	157	1.85	0.26
	5. Vitamin C	6	393	248	145	321	127	1.40	0.23
C MSU									
	1. Heat	5	443	283	160	363	136	2.05	0.29
	2. BAPN	7	426	270	157	348	162	2.17	0.32
	3. BAPN + Reserpine	7	429	257	172	343	150	1.84	0.27
	4. Reserpine	6	440	275	165	358	147	1.98	0.28
	5. Vitamin C	6	419	257	163	338	137	1.38	0.21
F value[2]									
	Strains		7.47**	6.35**	1.71	8.71**	0.22	0.34	0.19
	Treatments		4.39**	4.95**	1.87	5.41**	2.07	9.63**	8.31**
S.E.M.									
	Strains		± 5.07	± 3.61	± 4.29	± 3.85	± 5.14	± 0.04	± 0.006
	Treatments		± 7.21	± 5.13	± 6.11	± 5.47	± 7.29	± 0.06	± 0.008
Non-significance[3]									
	Strains		(A-B)(B-C)	(A-B)(B-C)	--	(A-B)(B-C)	--	--	--
	Treatments		(1-2-3-5) (2-3-4-5)	(1-2-5) (2-3-4) (2-4-5)	--	(1-5) (2-3-4-5)	--	(1-2-4) (2-3-4-5)	(1-2) (3-4-5)

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
3/ Numbers and letters joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 17. Percent change[1] in common carotid hemodynamic parameters following the injection of epinephrine[2] to adult male BBB turkeys

Strains	Treatments	No. birds	SBP	DBP	PP	MP	HR	SRU/ $Kg^{0.734}$	SRU/ bird
A Comm									
1.	Heat	7	1.79	1.57	2.31	1.70	0.82	2.56	2.54
2.	BAPN	7	1.59	1.48	1.82	1.54	0.88	2.30	2.28
3.	BAPN + Reserpine	7	2.03	1.94	2.27	1.99	1.19	2.97	2.95
4.	Reserpine	6	1.90	1.86	1.97	1.89	1.02	2.38	2.24
5.	Vitamin C	6	1.98	1.63	3.37	1.82	0.93	2.42	2.41
B Comm X MSU									
1.	Heat	7	1.75	1.54	2.23	1.68	0.89	2.49	2.48
2.	BAPN	6	1.52	1.43	1.70	1.48	0.91	2.15	2.15
3.	BAPN + Reserpine	6	1.89	1.97	1.83	1.91	1.17	2.74	2.70
4.	Reserpine	7	1.83	1.70	2.09	1.78	1.10	2.43	2.39
5.	Vitamin C	6	1.75	1.45	2.71	1.62	0.84	2.17	2.06
C MSU									
1.	Heat	5	1.55	1.44	1.80	1.50	0.52	2.24	2.23
2.	BAPN	7	1.50	1.47	1.60	1.49	0.84	2.15	2.12
3.	BAPN + Reserpine	7	1.80	1.82	1.99	1.72	1.22	2.30	2.38
4.	Reserpine	6	1.81	1.68	2.14	1.75	1.03	2.36	2.35
5.	Vitamin C	6	1.55	1.37	2.23	1.47	0.77	1.98	1.94
F value[3]									
	Strains		7.19**	4.35*	3.60*	8.04**	1.40	6.51**	4.59*
	Treatments		22.50**	41.30**	10.69**	25.14**	12.75**	18.20**	16.15**
S.E.M.									
	Strains		± 0.03	± 0.03	± 0.08	± 0.03	± 0.03	± 0.06	± 0.07
	Treatments		± 0.04	± 0.04	± 0.11	± 0.04	± 0.05	± 0.09	± 0.09
Non-significance[4]									
	Strains		(A-B)(B-C)	(A-B)(B-C)	(A-B)(B-C)	(A-B)(B-C)	--	(A-B)(B-C)	(A-B)(B-C)
	Treatments		(1-4-5) (3-4-5)	(1-2-5)	(1-3-4) (2-3-4)	(1-2-5) (3-4)	(1-2-5) (2-4)(3-4)	(1-2-4-5) (1-3-4)	(1-2-4-5) (1-3-4)

1/ Percentage values indexed to base 1.00.
2/ Injection of 1/25,000 epinephrine.
3/ Numbers with one asterisk indicate significance at the $P < 0.05$ level while those numbers with two asterisks indicate significance at the $P < 0.01$ level.
4/ Numbers and letters joined by a dash are non-significantly different at the $P < 0.01$ level.

Table 18. Popliteal hemodynamic parameters of adult male BBB turkeys

Strains Treatments	No. birds	SBP (mm Hg)	DBP (mm Hg)	PP (mm Hg)	MP (mm Hg)	SRU/ $Kg^{0.734}$	SRU/ bird
A Comm							
1. Heat	7	222	169	54	196	0.77	0.11
2. BAPN	7	236	162	74	198	0.80	0.12
3. BAPN + Reserpine	7	194	128	65	161	0.65	0.10
4. Reserpine	6	203	150	50	175	0.76	0.12
5. Vitamin C	6	205	171	35	188	0.76	0.11
B Comm X MSU							
1. Heat	7	235	171	64	203	0.81	0.12
2. BAPN	6	250	171	79	211	0.83	0.12
3. BAPN + Reserpine	6	198	132	66	166	0.64	0.10
4. Reserpine	7	218	152	67	185	0.76	0.11
5. Vitamin C	6	205	165	40	185	0.79	0.12
C MSU							
1. Heat	6	253	180	73	217	0.87	0.12
2. BAPN	7	259	179	80	220	0.95	0.15
3. BAPN + Reserpine	7	208	138	70	173	0.67	0.10
4. Reserpine	7	225	161	64	193	0.84	0.13
5. Vitamin C	6	257	213	43	235	0.99	0.15
F value[1]							
Strains		16.00**	8.60**	6.20**	12.24**	8.13**	9.33**
Treatments		38.04**	30.56**	15.98**	47.73**	23.58**	26.67**
S.E.M.							
Strains		± 3.59	± 3.03	± 2.47	± 2.75	± 0.02	± 0.003
Treatments		± 5.10	± 4.30	± 3.51	± 3.91	± 0.03	± 0.004
Non-significance[2]							
Strains		--	(A-B)	(A-B)(B-C)	(A-B)	(A-B)	(A-B)
Treatments		(1-2)(1-5) (3-4)(4-5)	(1-2-5)	(1-3-4) (2-3)	(1-2-5)	(1-2-4-5)	(1-2-4-5)

1/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
2/ Numbers and letters joined by a dash are non-significantly different at the P 0.01 level.

Table 19. *Popliteal hemodynamic parameters following the injection of epinephrine[1] to adult male BBB turkeys*

Strains Treatments	No. birds	SBP (mm Hg)	DBP (mm Hg)	PP (mm Hg)	MP (mm Hg)	SRU/ $Kg^{0.734}$	SRU/ bird
A Comm							
1. Heat	7	392	257	135	324	1.91	0.26
2. BAPN	7	367	242	125	305	1.82	0.28
3. BAPN + Reserpine	7	384	234	150	309	1.77	0.26
4. Reserpine	6	335	246	89	291	1.75	0.24
5. Vitamin C	6	388	277	111	333	1.79	0.25
B Comm X MSU							
1. Heat	7	406	272	132	338	2.00	0.29
2. BAPN	6	371	249	123	310	1.90	0.28
3. BAPN + Reserpine	6	372	232	141	302	1.67	0.25
4. Reserpine	5	389	257	132	324	1.84	0.26
5. Vitamin C	6	365	253	112	309	1.70	0.26
C MSU							
1. Heat	5	419	282	137	351	2.00	0.23
2. BAPN	7	379	253	126	316	1.96	0.29
3. BAPN + Reserpine	7	391	238	153	314	1.68	0.25
4. Reserpine	7	407	266	141	337	1.78	0.26
5. Vitamin C	6	386	293	93	340	1.78	0.26
F value[2]							
Strains		4.66**	2.61	1.88	4.78**	0.40	0.21
Treatments		2.39*	10.31**	5.07**	4.87**	11.40**	4.98**
S.E.M.							
Strains		± 5.42	± 3.59	± 4.60	± 3.91	± 0.04	± 0.007
Treatments		± 7.71	± 5.12	± 6.56	± 5.56	± 0.06	± 0.010
Non-significance[3]							
Strains		(A-B)(B-C)	--	--	(A-B)	--	--
Treatments		(1-3) (2-3-4-5)	(1-4-5) (2-3)(2-4)	(1-2-3) (1-2-4) (2-4-5)	(1-4-5) (2-3-4-5)	(1-2-4-5) (2-3-4-5)	(1-2) (3-4-5)

1/ Injection of 0.5 ml of 1/25,000 epinephrine.
2/ Numbers with one asterisk indicate significance at the P 0.05 level while those numbers with two asterisks indicate significance at the P 0.01 level.
3/ Numbers and letters joined by a dash are non-significantly different at the P 0.01 level.

Table 20. Percent change[1] in popliteal hemodynamic parameters following the injection of epinephrine[2] to adult male BBB turkeys

Strains	Treatments	No. birds	SBP	DBP	PP	MP	SRU/ $Kg^{0.734}$	SRU/ bird
A Comm								
	1. Heat	7	1.77	1.54	2.56	1.71	2.52	2.47
	2. BAPN	7	1.56	1.50	1.69	1.46	2.26	2.25
	3. BAPN + Reserpine	7	2.01	1.86	2.43	1.95	2.91	2.93
	4. Reserpine	6	1.69	1.65	1.77	1.67	2.29	2.15
	5. Vitamin C	6	1.90	1.63	3.26	1.78	2.37	2.38
B Comm X MSU								
	1. Heat	7	1.73	1.60	2.08	1.67	2.46	2.42
	2. BAPN	6	1.49	1.46	1.57	1.47	2.14	2.10
	3. BAPN + Reserpine	6	1.89	1.76	2.14	1.84	2.64	2.55
	4. Reserpine	5	1.68	1.65	1.77	1.66	2.34	2.29
	5. Vitamin C	6	1.79	1.55	2.78	1.68	2.27	2.26
C MSU								
	1. Heat	5	1.68	1.62	1.87	1.65	2.48	1.99
	2. BAPN	7	1.46	1.43	1.59	1.45	1.99	1.99
	3. BAPN + Reserpine	7	1.89	1.74	2.29	1.83	2.54	2.50
	4. Reserpine	7	1.81	1.66	2.23	1.74	2.30	2.26
	5. Vitamin C	6	1.51	1.38	2.18	1.45	1.89	1.84
F value[3]								
	Strains		3.79*	2.38	4.08*	2.44	7.88**	5.75**
	Treatments		22.39**	21.55**	12.89**	25.00**	34.93**	22.22**
S.E.M.								
	Strains		± 0.03	± 0.03	± 0.08	± 0.03	± 0.05	± 0.06
	Treatments		± 0.04	± 0.04	± 0.11	± 0.04	± 0.08	± 0.09
Non-significance[4]								
	Strains		(B-C)	--	(B-C)	--	(A-B)(B-C)	(A-B)(B-C)
	Treatments		(1-4-5)	(1-2-5) (1-4-5) (3-4)	(1-3-4) (2-4)	(1-4-5)	(1-3) (1-4-5) (2-4-5)	(1-3) (1-2-4-5)

1/ Percentage values indexed to base 1.00.
2/ Injection of 1/25,000 epinephrine.
3/ Numbers with one asterisk indicate significance at the $P < 0.05$ level while those numbers with two asterisks indicate significance at the $P < 0.01$ level.
4/ Numbers and letters joined by a dash are non-significantly different at the $P < 0.01$ level.

Table 21. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the thoracic aorta of adult male BBB turkeys below the normal physiological systolic blood pressure range (25-150 mm Hg)

Strains Treatments	No. birds	Pressures (mm Hg) 25	50	75	100	125	150
A Comm							
1. Heat	7	8.16	6.68	5.91	4.84	4.19	3.92
2. BAPN	7	5.14	5.47	5.25	4.56	3.92	3.29
3. BAPN + Reserpine	7	6.96	5.99	5.36	4.15	3.43	2.58
4. Reserpine	6	7.69	6.35	5.44	4.04	3.13	2.37
5. Untreated	6	10.59	9.28	8.40	6.41	4.71	3.55
6. Vitamin C	6	10.52	9.47	8.75	7.04	5.39	4.08
B Comm X MSU							
1. Heat	7	8.98	7.41	6.41	5.19	4.39	3.65
2. BAPN	6	6.00	7.32	6.81	5.61	4.73	3.55
3. BAPN + Reserpine	6	6.84	6.27	5.67	4.43	3.58	2.68
4. Reserpine	7	7.89	6.57	5.65	4.24	3.37	2.59
5. Untreated	7	10.73	8.97	8.05	6.19	4.77	3.60
6. Vitamin C	6	11.53	10.09	9.32	7.54	5.93	4.60
C MSU							
1. Heat	6	5.95	5.50	6.04	4.19	3.57	3.24
2. BAPN	7	7.01	7.14	6.75	5.48	4.72	3.63
3. BAPN + Reserpine	7	8.81	7.58	6.49	4.79	3.73	2.58
4. Reserpine	7	7.76	6.40	5.51	4.09	3.17	2.42
5. Untreated	7	11.87	9.75	8.80	6.82	5.23	4.06
6. Vitamin C	6	12.32	10.47	9.65	7.80	6.22	4.89
F value[1]							
Strains		1.77	2.31	1.43	1.71	1.56	0.28
Treatments		20.64**	19.38**	16.61**	28.90**	21.89**	21.56**
S.E.M.							
Strains		± 0.32	± 0.29	± 0.25	± 0.15	± 0.13	± 0.11
Treatments		± 0.45	± 0.41	± 0.35	± 0.22	± 0.19	± 0.15
Non-significance[2]							
Strains		--	--	--	--	--	--
Treatments		(1-2-3-4) (5-6)	(1-2-3-4) (5-6)	(1-2-3-4) (5-6)	(1-2-3) (1-3-4)	(1-2)(1-3) (2-5)(3-4)	(1-2-5) (3-4)

1/ Numbers with two asterisks indicate significance at the P < 0.01 level.
2/ Numbers joined by a dash are non-significantly different at the P > 0.01 level.

Table 22. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the thoracic aorta of adult male BBB turkeys at the normal physiological systolic blood pressure range (175-350 mm Hg)

Strains	Treatments	No. birds	Pressures (mm Hg) 175	200	225	250	275	300	325	350
A Comm										
	1. Heat	7	3.87	3.76	3.77	3.85	3.99	4.12	4.26	4.45
	2. BAPN	7	2.84	2.46	2.37	2.32	2.38	2.40	2.46	2.53
	3. BAPN + Reserpine	7	2.28	2.01	2.02	2.05	2.13	2.20	2.30	2.38
	4. Reserpine	6	2.01	1.81	1.78	1.79	1.85	1.94	2.03	2.11
	5. Untreated	6	2.98	2.43	2.14	2.01	2.07	2.15	2.28	2.37
	6. Vitamin C	6	3.29	2.79	2.68	2.65	2.74	2.84	2.96	3.08
B Comm X MSU										
	1. Heat	7	3.27	3.03	2.92	2.94	3.04	3.13	3.24	3.37
	2. BAPN	6	2.87	2.45	2.24	2.31	2.37	2.43	2.49	2.56
	3. BAPN + Reserpine	6	2.29	2.09	2.12	2.15	2.24	2.32	2.42	2.51
	4. Reserpine	7	2.18	1.94	1.91	1.91	1.98	2.06	2.15	2.23
	5. Untreated	7	2.98	2.53	2.27	2.18	2.25	2.33	2.44	2.54
	6. Vitamin C	6	3.74	2.17	3.04	2.99	3.07	3.17	3.30	3.43
C MSU										
	1. Heat	6	3.16	3.11	3.16	3.28	3.43	3.56	3.71	3.86
	2. BAPN	7	2.93	2.42	2.31	2.25	2.32	2.35	2.41	2.48
	3. BAPN + Reserpine	7	2.10	1.88	1.87	1.88	1.98	2.07	2.17	2.27
	4. Reserpine	7	2.12	1.95	1.93	1.94	2.00	2.09	2.18	2.27
	5. Untreated	7	3.41	2.94	2.65	2.53	2.61	2.68	2.80	2.89
	6. Vitamin C	6	3.95	3.31	3.16	3.08	3.16	3.27	3.40	3.54
F value[1]										
	Strains		0.10	0.17	0.50	0.41	0.46	0.42	0.50	0.50
	Treatments		20.41**	22.77**	22.52**	26.04**	28.13**	28.38**	29.00**	33.27**
S.E.M.										
	Strains		± 0.10	± 0.08	± 0.08	± 0.08	± 0.08	± 0.08	± 0.08	± 0.08
	Treatments		± 0.14	± 0.12	± 0.12	± 0.11	± 0.11	± 0.11	± 0.12	± 0.12
Non-significance[2]										
	Strains		--	--	--	--	--	--	--	--
	Treatments		(1-5-6) (2-5) (3-4)	(1-6)(2-3) (2-5)(3-4) (5-6)	(1-6)(2-3-4-5)	(2-3-4-5)	(2-3-4-5)	(2-3-4-5)	(2-3-4-5)	(2-3-4-5)

1/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
2/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 23. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the thoracic aorta of adult male BBB turkeys above the normal physiological systolic blood pressure range (375-500 mm Hg)

Strains	Treatments	No. birds	Pressures (mm Hg) 375	400	425	450	475	500
A Comm								
	1. Heat	6	3.70	3.83	3.66	3.78	3.95	4.06
	2. BAPN	7	2.62	2.72	2.83	3.10	3.18	3.21
	3. BAPN + Reserpine	7	2.48	2.57	2.66	2.74	2.92	3.00
	4. Reserpine	6	2.21	2.29	2.37	2.43	2.50	2.56
	5. Untreated	6	2.48	2.58	2.69	2.78	2.84	2.93
	6. Vitamin C	6	3.21	3.33	3.86	3.99	4.13	4.28
B Comm X MSU								
	1. Heat	7	3.30	3.42	3.47	3.57	3.73	3.82
	2. BAPN	6	2.65	2.73	3.02	3.14	3.21	3.34
	3. BAPN + Reserpine	6	2.60	2.69	2.78	2.86	3.00	3.08
	4. Reserpine	7	2.33	2.41	2.49	2.56	2.63	2.71
	5. Untreated	7	2.65	2.75	2.86	2.96	3.11	3.21
	6. Vitamin C	6	3.57	3.70	4.05	4.18	4.32	4.44
C MSU								
	1. Heat	6	3.78	3.92	4.00	4.12	4.28	4.38
	2. BAPN	7	2.57	2.65	2.74	2.80	2.87	2.99
	3. BAPN + Reserpine	7	2.36	2.44	2.53	2.60	2.77	2.86
	4. Reserpine	7	2.36	2.45	2.53	2.60	2.67	2.74
	5. Untreated	7	3.00	3.10	3.20	3.30	3.34	3.43
	6. Vitamin C	6	3.69	3.83	3.97	4.10	4.24	4.36
F value[1]								
	Strains		1.31	1.38	2.55	1.55	1.18	2.38
	Treatments		37.69**	40.77**	48.55**	53.90**	52.27**	48.58**
S.E.M.								
	Strains		± 0.06	± 0.06	± 0.06	± 0.05	± 0.06	± 0.06
	Treatments		± 0.08	± 0.08	± 0.08	± 0.08	± 0.08	± 0.09
Non-significance[2]								
	Strains		--	--	--	--	--	--
	Treatments		(1-6) (2-3-4) (2-3-5)	(1-6) (2-3-4) (2-3-5)	(1-6)(3-4) (2-3-5)	(1-6)(3-4) (2-3-5)	(1-6)(3-4) (2-3-5)	(1-6)(3-4) (2-3-5)

1/ Numbers with two asterisks indicate significance at the P ≤ 0.01 level.
2/ Numbers joined by a dash are non-significantly different at the P ≤ 0.01 level.

Table 24. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the abdominal aorta of adult male BBB turkeys below the normal physiological systolic blood pressure range (25-150 mm Hg)

Strains	Treatments	No. birds	Pressures (mm Hg) 25	50	75	100	125	150
A Comm								
	1. Heat	6	7.73	10.02	11.69	12.03	12.82	13.24
	2. BAPN	7	6.29	6.73	8.17	9.07	10.07	10.42
	3. BAPN + Reserpine	6	4.94	5.75	6.96	7.84	8.70	9.12
	4. Reserpine	6	9.05	9.27	10.64	10.92	11.24	11.51
	5. Untreated	6	9.36	8.54	9.51	9.93	10.47	11.09
	6. Vitamin C	6	6.61	7.66	8.80	9.50	10.27	10.95
B Comm X MSU								
	1. Heat	7	9.25	9.98	11.16	11.40	12.19	12.41
	2. BAPN	6	5.06	5.79	6.84	7.61	8.51	9.10
	3. BAPN + Reserpine	6	5.31	6.11	7.27	8.14	9.04	9.54
	4. Reserpine	7	8.67	9.10	10.73	11.01	11.37	11.59
	5. Untreated	7	8.67	7.93	9.19	9.77	10.42	11.03
	6. Vitamin C	6	6.68	7.73	8.88	9.57	10.34	11.02
C MSU								
	1. Heat	6	7.80	9.76	11.41	12.19	12.86	13.31
	2. BAPN	7	4.31	4.92	5.82	6.54	7.40	8.11
	3. BAPN + Reserpine	7	6.23	5.93	6.89	7.81	8.79	9.37
	4. Reserpine	7	10.40	10.24	11.27	11.57	11.82	12.15
	5. Untreated	7	9.79	8.66	9.96	10.40	10.95	11.56
	6. Vitamin C	6	6.67	7.72	8.85	9.56	10.33	11.00
F value[1]								
	Strains		2.90	0.12	0.54	0.39	0.47	0.29
	Treatments		26.71**	37.63**	43.84**	32.77**	24.51**	24.72**
S.E.M.								
	Strains		± 0.26	± 0.21	± 0.21	± 0.21	± 0.22	± 0.21
	Treatments		± 0.37	± 0.30	± 0.30	± 0.30	± 0.32	± 0.30
Non-significance[2]								
	Strains		--	--	--	--	--	--
	Treatments		(1-4-5) (2-3-6)	(1-4)(2-3) (5-6)	(1-4)(2-3) (5-6)	(1-4)(2-3) (5-6)	(1-4)(2-3) (4-5)(5-6)	(2-3) (4-5-6)

1/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
2/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 25. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the abdominal aorta of adult male BBB turkeys at the normal physiological systolic blood pressure range (175-350 mm Hg)

Strains	Treatments	No. birds	Pressures (mm Hg) 175	200	225	250	275	300	325	350
A Comm										
	1. Heat	6	13.61	14.04	14.55	14.90	15.47	15.85	16.42	17.12
	2. BAPN	7	11.03	11.48	12.10	12.62	13.30	13.83	14.41	14.79
	3. BAPN + Reserpine	6	9.74	10.26	10.90	11.45	12.18	12.77	13.46	14.11
	4. Reserpine	6	11.90	12.15	12.67	13.18	13.80	14.37	14.88	15.45
	5. Untreated	6	11.76	12.21	12.92	13.45	14.21	14.78	15.44	16.02
	6. Vitamin C	6	11.79	12.50	13.33	14.01	14.82	15.54	16.27	17.04
B Comm X MSU										
	1. Heat	7	12.91	13.33	13.94	14.49	15.26	15.76	16.42	17.63
	2. BAPN	6	9.79	10.44	11.13	11.76	12.41	13.04	13.65	14.21
	3. BAPN + Reserpine	6	10.17	10.70	11.26	11.80	12.50	13.13	13.77	14.35
	4. Reserpine	7	11.98	12.18	12.73	13.23	13.87	14.46	14.97	15.54
	5. Untreated	7	11.74	12.28	13.04	13.65	14.42	15.03	15.71	16.31
	6. Vitamin C	6	11.86	12.57	13.40	14.08	14.89	15.60	16.34	17.11
C MSU										
	1. Heat	6	13.63	14.13	14.79	15.38	16.08	16.68	17.34	17.99
	2. BAPN	7	8.97	9.63	10.30	10.93	11.59	12.24	12.87	13.46
	3. BAPN + Reserpine	7	9.92	10.52	10.93	11.42	12.04	12.65	13.28	13.82
	4. Reserpine	7	12.51	12.80	13.23	13.70	14.27	14.80	15.31	15.85
	5. Untreated	7	12.24	12.69	13.41	13.95	14.71	15.28	15.95	16.52
	6. Vitamin C	6	11.85	12.56	13.38	14.07	14.87	15.59	16.33	17.09
F value[1]										
	Strains		0.24	0.17	0.19	0.10	0.12	0.05	0.01	0.03
	Treatments		23.61**	25.80**	31.10**	37.34**	42.51**	45.87**	49.85**	75.90**
S.E.M.										
	Strains		± 0.20	± 0.18	± 0.17	± 0.15	± 0.15	± 0.14	± 0.13	± 0.12
	Treatments		± 0.29	± 0.26	± 0.24	± 0.22	± 0.21	± 0.20	± 0.19	± 0.17
Non-significance[2]										
	Strains		--	--	--	--	--	--	--	--
	Treatments		(2-3) (4-5-6)	(2-3) (4-5-6)	(2-3) (4-5-6)	(2-3) (4-5-6)	(1-6)(2-3) (4-5)(5-6)	(1-6)(2-3) (4-5)(5-6)	(1-6)(2-3) (4-5)(5-6)	(1-6) (2-3)

1/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
2/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 26. Mean values for static modulus of elasticity (Dynes/cm^2 X 10^5) for the abdominal aorta of adult male BBB turkeys above the normal physiological systolic blood pressure range (375-500 mm Hg)

Strains	Treatments	No. birds	Pressures (mm Hg) 375	400	425	450	475	500
A Comm								
	1. Heat	6	17.60	17.94	18.30	18.55	18.87	19.14
	2. BAPN	7	15.40	15.87	16.36	16.83	17.15	17.51
	3. BAPN + Reserpine	6	14.70	15.20	15.72	16.25	16.79	17.27
	4. Reserpine	6	16.09	16.58	17.09	17.58	18.00	18.47
	5. Untreated	6	16.57	17.15	17.74	18.22	18.70	19.14
	6. Vitamin C	6	17.79	18.50	19.18	19.84	20.43	21.07
B Comm X MSU								
	1. Heat	6	18.09	18.55	18.96	19.28	19.73	20.05
	2. BAPN	6	14.82	15.36	15.94	16.49	16.97	17.43
	3. BAPN + Reserpine	6	14.93	15.42	15.97	16.47	16.94	17.36
	4. Reserpine	7	16.19	16.69	17.22	17.72	18.13	18.57
	5. Untreated	7	16.94	17.47	18.08	18.57	19.07	19.54
	6. Vitamin C	6	17.86	18.57	19.25	19.91	20.50	21.14
C MSU								
	1. Heat	6	18.47	18.93	19.35	19.65	20.09	20.40
	2. BAPN	7	14.04	14.60	15.18	15.72	16.23	16.69
	3. BAPN + Reserpine	7	14.42	14.91	15.46	15.99	16.49	16.98
	4. Reserpine	7	16.46	16.91	17.37	17.84	18.25	18.72
	5. Untreated	7	17.09	17.66	18.25	18.73	19.21	19.65
	6. Vitamin C	6	17.84	18.55	19.24	19.90	20.49	21.13
F value[1]								
	Strains		0.04	0.39	0.54	0.32	0.49	0.56
	Treatments		73.12**	72.90**	69.67**	61.76**	60.77**	60.21**
S.E.M.								
	Strains		± 0.12	± 0.12	± 0.12	± 0.13	± 0.13	± 0.13
	Treatments		± 0.17	± 0.27	± 0.17	± 0.18	± 0.18	± 0.19
Non-significance[2]								
	Strains		--	--	--	--	--	--
	Treatments		(1-6)(2-3) (4-5)	(1-6) (2-3)	(1-6) (2-3)	(1-5) (2-3)	(1-5) (2-3)	(1-5) (2-3)

1/ Numbers with two asterisks indicate significance at the $P < 0.01$ level.
2/ Numbers joined by a dash are non-significantly different at the $P > 0.01$ level.

Table 27. Partial correlation coefficients ("r") of cardiovascular parameters

	Variates				
	I. x = SBP(C) y = BVT(T) z = HR a = BW	II. x = MV y = DBP(C) z = SBP(C) a = BW	III. x = HR y = DBP(C) z = %ΔSBP a = BW	IV. x = SV y = MV z = HR a = SBP(C)	V. x = BVT (T) y = SV z = DBP a = BW
$r_{xy.z}$	+ 0.8144**	- 0.2551	+ 0.5391*	+ 0.8585**	- 0.2639
$r_{xy.a}$	+ 0.7507**	- 0.1771	+ 0.4590*	+ 0.8139**	- 0.4624*
$r_{xz.y}$	+ 0.5589*	- 0.1710	+ 0.3208	+ 0.4909*	+ 0.5834**
$r_{xz.a}$	+ 0.5379*	- 0.0917	- 0.0816	+ 0.2314	+ 0.6288**
$r_{xa.y}$	- 0.4839*	+ 0.2692	- 0.4893*	- 0.4597*	- 0.1645
$r_{xa.z}$	- 0.1241	+ 0.2925	- 0.5097*	- 0.4568*	+ 0.0162
$r_{yz.x}$	- 0.4564*	+ 0.9342**	- 0.8497**	- 0.5487*	- 0.1400
$r_{yz.a}$	- 0.1238	+ 0.9264**	- 0.8270**	- 0.2332	- 0.3497
$r_{ya.z}$	- 0.2415	- 0.0701	+ 0.0598	- 0.1012	+ 0.0211
$r_{ya.x}$	+ 0.2802	- 0.2756	- 0.0524	+ 0.2471	+ 0.0635
$r_{za.x}$	- 0.5084*	- 0.2968	+ 0.3320	+ 0.4556*	- 0.2755
$r_{za.y}$	- 0.5797**	- 0.0626	+ 0.2940	+ 0.3341	- 0.3121

* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

Table 28. Partial correlation coefficients ("r") of cardiovascular parameters

	Variates			
	VI. x = % △SBP(C) y = E @ 250 mm Hg(T) z = DBP (C) a = BW	VII. x = PP (C) y = BVT (T) z = % △ SBP a = BW	VIII. x = Aor Sc y = MV z = % △ SBP a = DBP	IX. x = BW y = DBP z = Ht Wt a = SV
$r_{xy.z}$	- 0.5878**	+ 0.2088	+ 0.2807	- 0.0928
$r_{xy.a}$	- 0.4983*	+ 0.6033**	+ 0.2588	- 0.3121
$r_{xz.y}$	- 0.8579**	- 0.4939*	+ 0.2689	+ 0.5034*
$r_{xz.a}$	- 0.8270**	- 0.7063**	+ 0.2751	+ 0.5986**
$r_{xa.y}$	+ 0.1355	+ 0.2797	- 0.0858	+ 0.0211
$r_{xa.z}$	+ 0.2944	+ 0.6101**	+ 0.1530	+ 0.2371
$r_{yz.x}$	- 0.3786	- 0.4593*	- 0.0096	- 0.3616
$r_{yz.a}$	+ 0.1194	- 0.6475**	- 0.3121	- 0.5464*
$r_{ya.z}$	- 0.5877*	+ 0.1160	- 0.3996	- 0.4711*
$r_{ya.x}$	- 0.5182*	- 0.3295	- 0.2391	- 0.3497
$r_{za.x}$	+ 0.0598	+ 0.6967*	- 0.8482**	- 0.2045
$r_{za.y}$	- 0.2039	+ 0.4649*	- 0.8635**	- 0.3167

* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

Table 29. Correlation coefficients ("r") of two variates

Variates	"r"
Carotid Artery	
SBP vs Ht Wt	+ 0.1040
DBP vs PP	+ 0.5054*
PP vs E @ 250 (T)	+ 0.5598*
PP vs Aor Sc	- 0.1500
PP vs MV	+ 0.0949
PP vs HR	- 0.0441
PP vs % — SBP after epinephrine	- 0.6789**
E @ 250 (T) vs Aor Sc	- 0.0477
E @ 250 (T) vs MV	+ 0.0753
BVT (T) vs Ht Wt	- 0.0444
BVT (T) vs MV	- 0.1822
Ht Wt vs MV	+ 0.0987

* Indicates significance at the P 0.05 level.
** Indicates significance at the P 0.01 level.

Table 30. Correlation coefficients ("r") of two variates

Variates	"r" value
Popliteal Artery	
E @ 250 mm Hg (A) vs Aor Sc	- 0.0660
E @ 250 mm Hg (A) vs % Δ SBP after epinephrine	- 0.2854
E @ 250 mm Hg (A) vs DBP	+ 0.3436
E @ 250 mm Hg (A) vs PP	+ 0.3543
E @ 250 mm Hg (A) vs MV	- 0.2950
Aor Sc vs % Δ SBP after epinephrine	+ 0.3075
Aor Sc vs SBP	+ 0.2220
Aor Sc vs DBP	+ 0.0960
Aor Sc vs PP	- 0.2127
Aor Sc vs MV	+ 0.2888
% Δ SBP after epinephrine vs DBP	- 0.5873**
% Δ SBP after epinephrine vs PP	- 0.2678
% $\angle$ SBP after epinephrine vs MV	+ 0.5099*
DBP vs PP	+ 0.1269
DBP vs MV	- 0.4245
PP vs MV	- 0.0890

* Indicates significance at the $P < 0.05$ level.
** Indicates significance at the $P < 0.01$ level.

DISCUSSION

These findings reveal that the blood pressure of the turkey increases with age, being higher in the male than in the female. This confirmed work of Ringer and Rood (1959) and Speckmann and Ringer (1961) who, with Gibson and deGruchy (1955) suggested that hemodynamic parameters were associated with the aortic rupture syndrome. In one experiment, however, there was no sex difference in blood pressure or cholesterol at 27 weeks of age, yet the male turkeys exhibited a higher incidence of atherosclerotic plaques in the abdominal aorta than did the female turkeys. A plausible explanation for this as compared to other blood pressure measurements may involve environmental temperature as similar effects of temperature on blood pressure were observed in experiments conducted during summer months. It was during this warm weather that the blood pressures were recorded. A similar effect of temperature (Weiss and Borbely, 1957) and season (Weiss et al., 1957) on blood pressure has also been reported for the chicken. It may well be that the high temperatures cause extreme peripheral vasodilation thus reducing the blood pressure. The fact that reserpine at low levels did not significantly reduce the blood pressure is not surprising as the pressure levels were already at a low point. A strain difference in blood pressure has been shown to exist in turkeys and this may be associated with a possible strain susceptibility to aortic rupture.

Blood pressure measurements in the turkey consistently show common carotid systolic blood pressures higher than those of the popliteal

artery. In chickens, Kaupp, (1923, 1924) reported femoral mean pressures lower than carotid mean pressures reported by Stubel (1910). In considering the influence of resistance on pressure one must keep in mind the point of resistance and the point of pressure measurement. If the resistance is peripheral to the point of measurement, then increased resistance results in increased blood pressures. On the other hand, consider a length of tube with fluid flowing through it. Pressure at the upstream end is always greater than at the other end because of the dissipation of energy in overcoming the resistance offered by the entire length of tube to flow. Perhaps the lower blood pressures toward the periphery were caused by the spontaneous obstructive atherosclerotic plaques found in the abdominal aorta of chickens (Katz and Stamler, 1953) and turkeys (Ringer, 1960). These lesions possibly increased vascular resistance between the heart and point of blood pressure measurement.

The work of Kroeker and Wood (1955) compares simultaneously recorded central and peripheral arterial pressure pulses during rest and in a supine position. These workers recorded pressures directly by intra-arterial needles and catheters. In general they reported that peripheral systolic and pulse pressures, i.e., arm and leg, were the same at rest and uniformly exceeded central systolic blood pressures. These workers, and recently Tjong and Verheugt (1960), found that diastolic and mean blood pressures in humans at rest showed a smaller but uniform decrease toward the periphery. This confirmed previous work by Wood et al. (1951). Other work by Hamilton et al. (1936) and Schnabel et al. (1952) also showed that there was an increase in systolic blood pressure peripherally, but that diastolic and mean blood pressures remained relatively constant.

A more recent report on comparative indirect blood pressures of humans by Spittel et al. (1961) showed that in a resting supine position, thigh systolic blood pressures were considerably higher than systolic blood pressures measured in the arm. These researchers found that in the majority of patients with abdominal aortic aneurysms, both thighs had low systolic pressures as compared to those in the arms; in fact, more than half of these patients had a lower systolic blood pressure in the thigh than in the arm. Perhaps such low thigh pressures were also due to obstructive atherosclerotic lesions in the aorta. Spittel et al. (1961), however, did not measure the atherosclerotic severity of their patients.

Normal systolic blood pressures in the neighborhood of 300 mm Hg for male turkeys seem phenomenal when compared to human systolic blood pressures of 120-140 mm Hg; in fact, so far as the author knows, male BBB turkeys have the highest normal blood pressure reported for any animal. It is possible that blood pressure may be the initiating and final cause of aortic rupture. Atherosclerosis in advanced stages is characterized by lipid infiltration into the media causing vascular weakness (Best and Taylor, 1956) and coupled with an increased pulse pressure (as occurs in the male BBB turkey) and the erratic hemodynamic fluctuations, as might occur during stress (epinephrine injection), the vascular layers may become separated resulting in a dissecting aneurysm. Blood under high pressure forces its way between the layers causing a bulging of the wall. Since the tension on the wall of any fluid-containing cylinder is proportional to the radius (law of Laplace) a vicious cycle is established progressively weakening the wall until a sharp elevation of pressure causes the final rupture.

In view of the increased blood pressure of the BBB male turkey above other animals, and the higher pressure attained during stress, it is highly probable that blood pressure may be an important factor in the aortic rupture syndrome. Work reported by Speckmann (1959) demonstrated that reserpine, a hypotensive tranquilizer, significantly decreased blood pressure in the adult male BBB turkey. The birds exhibited no tranquilization. Later, the finding that reserpine affords protection against aortic rupture in turkeys (Carlson, 1959; Barnett, 1960; Morrison, 1960; and Waibel, 1960) certainly suggests that hemodynamic aspects are important in this condition.

Since systolic blood pressure was primarily influenced by reserpine, it was thought that reserpine may have had some effect on the heart itself, possibly altering cardiac output to absorb the greater work load during stress conditions. A method was devised for continuously recording the primary dilution curve of radiophosphorus. This eliminated arterial sampling and allowed a more accurate evaluation of the primary curve. The normal cardiac output of the male BBB turkey was determined as 231 ml per $Kg^{0.734}$ per min. This is slightly higher than cardiac outputs reported for the chicken (Sapirstein and Hartmann, 1959; Sturkie and Vogel, 1959), but slightly lower than that reported in rats, (Sapirstein and Hartmann, 1959).

In order to evaluate the effects of stress on cardiovascular dynamics, epinephrine was injected and the parameters measured. According to Wiggers (1955) epinephrine injection in mammals caused peripheral vasoconstriction but central vasodilation resulting in a net vasodilation. Epinephrine excites the heart causing an increased heart rate, cardiac output and force of contraction and it is these

cardiovascular parameters which increase blood pressure in view of a decreasing vascular resistance. In the turkey epinephrine injection increased heart rate initially by direct stimulation of the heart but also caused peripheral vasoconstriction. As heart rate increased, blood pressures increased quickly causing the homeostatic blood pressure mechanisms to decrease heart rate. Apparently the aortic arch and carotid sinus bodies (assuming these receptors are present in the turkey) are very sensitive to blood pressure fluctuations as heart rate was decreased almost instantaneously. Blood pressure continued to rise, however, due to constriction of peripheral vessels caused by epinephrine. It was during this period of decreasing heart rate and increasing blood pressure that cardiac output was determined. With a decreased heart rate and increased venous return, stroke volume must increase if normal cardiac output was to be maintained. Following the injection of epinephrine, however, stroke volume decreased 20 percent and consequently so did cardiac output. Apparently an injection of 0.5 ml of 1/25,000 epinephrine was too high for the turkey. The increased venous return to the heart, concomitant with an increased arterial pressure so that the left ventricle can put out only a small stroke volume, caused blood to be impounded in the lungs (Johnson et al., 1937). This probably resulted in such a diastolic enlargement that the reserve power of the heart was surpassed causing the heart to decompensate. Such cardiac decompensation decreased both systolic discharge and minute volume; yet blood pressure was still increased due to the intense peripheral vasoconstriction. The net effect of epinephrine injection in the turkey was a negative cardiovascular response, although blood pressure was increased.

When epinephrine was injected into reserpine-treated turkeys blood pressure increased significantly showing a greater percentage increase than in untreated birds injected with epinephrine; however, the absolute blood pressure attained was significantly lower in the reserpine-treated group than in the untreated group. Reserpine did not alter cardiac output per $Kg^{0.734}$ before or after the injection of epinephrine but it did tend to lower systemic resistance. Systemic resistance in the reserpine-treated group demonstrated a smaller percentage increase following the injection of epinephrine than it did in the other groups. Considering hemodynamic parameters to be of prime importance in the aortic rupture syndrome, the prophylactic action of reserpine on aortic rupture in the turkey appears to be due to its hypotensive action both in normal and stress conditions. Since it is reported that reserpine indirectly depletes bound serotonin in the chicken (Huber and Link, 1962) thus causing vasodilation and hypotension it is postulated that this relationship of reserpine and serotonin also exists in the turkey and is the mechanism by which the blood pressure is reduced and prevented from rising very much following the injection of epinephrine.

Although the injection of 0.5 ml of 1/25,000 epinephrine was undoubtedly a considerable strain on the cardiovascular system, the turkey apparently was able to adjust successfully to the pressure fluctuations as there were no apparent adverse effects. The successful adjustment to hypertension produced by epinephrine plus the beneficial effect of non-hypotensive tranquilizers on aortic rupture (Burger et al. 1961) coupled with the failure to rupture abdominal aortas in the elastance determinations, even though pressures of 500 mm Hg were

exceeded, suggests that hemodynamics alone are not the cause of aortic rupture.

Work in other laboratories on chickens and humans has demonstrated an association between hypertension, hypercholesterolemia and atherosclerosis (Katz and Stamler, 1953). Very little work has been reported concerning plasma cholesterol level in turkeys. Waibel et al. (1960) reported that the plasma cholesterol level of BBB turkeys was 129 mg percent at 5 weeks of age.

The plasma cholesterol level of the BBB turkey was found to increase from 4 to 16-18 weeks of age after which it plateaued. These values correspond to those reported by Waibel et al. (1960) and they continued to rise with advancing age. The plasma cholesterol level of commercial turkeys was lower than that of the M.S.U. stock. The aortic atherosclerotic scores and hemodynamic values were also higher in the group with the higher plasma cholesterol level. It is a common thought that the differences between the plasma cholesterol levels of groups of turkeys may result from different dietary regimes; however, in this experiment the same commercial turkey feed was fed ad libitum to both groups and blood samples were taken at the same time of day for both groups. It is therefore suggested that in addition to blood pressure there may also exist strain differences in plasma cholesterol and susceptibility to atherosclerosis.

There are no sex or strain differences in blood pressures (Ringer et al. 1955) or plasma cholesterol level in pigeons yet there is a marked strain difference in susceptibility to atherosclerosis (Clarkson et al., 1959; Lofland and Clarkson, 1960; Prichard et al., 1962). These authors believe that atherosclerosis in the pigeon is genetically

determined. Ringer (1962) believes that a vascular weakness may be bred into turkeys susceptible to aortic rupture.

Perhaps in certain strains of turkeys the significantly higher blood pressure of the male above the female accelerates atherogenesis to the point where the aortic vessel is significantly weakened and the additional hypertension experienced during stress is sufficient to cause rupture of the aorta.

There was no sexual difference in plasma cholesterol; however, the male BBB turkey after 16 weeks of age has been reported to have a significantly higher blood pressure than the female BBB turkey (Ringer and Rood, 1959; Speckmann and Ringer, 1961). The severity of aortic atherosclerotic plaques increased with age, being more severe in males than in females. The greatest increase in plasma cholesterol level was between 12-16 weeks of age, the age during which blood pressure has been reported to be increasing rapidly in BBB turkeys (Ringer and Rood, 1959) and the age during which aortic rupture occurs most frequently (Ringer, 1959).

The importance of plasma cholesterol in atherogenesis is stressed in the following example. If a cholesterol fed cockerel has a plasma cholesterol concentration of 100 mg percent, chances are 1 in 2 that it will have atherosclerotic lesions. If, however, the plasma cholesterol concentration is 300 mg percent, there is still 1 chance in 10 that it has no lesions at all (Tennent et al., 1957). If the theory of Tennent et al., (1957) applies to turkeys, the mature BBB turkey, with a plasma cholesterol level of 249 mg percent would have a very good chance of getting atherosclerosis. Yet, in this work, there was no sex difference in plasma cholesterol but the males had a significantly

higher incidence of atherosclerosis than did the females. Also in pigeons, there is no difference in the plasma cholesterol level between birds susceptible and resistant to spontaneous atherosclerosis, yet there is a marked difference in incidence and severity (Clarkson and Lofland, 1961). The aortic cholesterol but not plasma cholesterol was found to parallel the incidence and severity of atherosclerosis in chickens (Fisher et al., 1959). Also in man there is a marked sex differential in death rates from arteriosclerotic heart disease in middle age, yet there is no apparent sex difference for blood pressure or cholesterol level (Stamler, 1962).

Reserpine administration at the level of 0.1 ppm and 0.2 ppm in the feed of BBB turkeys did not offer any protection against the increase in plasma cholesterol level or the severity of atherosclerosis with advancing age. Reserpine at 0.2 ppm and above in the feed did reduce blood pressure.

Reserpine has been shown to decrease elevated serum cholesterol levels and inhibit the development of aortic atheromatosis in normotensive and hypertensive rats maintained on an atherogenic diet (Smith and Rossi, 1962). These investigators also demonstrated that reserpine decreased blood pressure only in hypertensive rats and that the hypertensive state increased the severity of atherogenesis in cholesterolized rats which were not fed reserpine. In humans with hypertension, aortic cholesterol was significantly higher than in normotensive patients; however, this same relationship was not necessarily true for the blood (Rosenthal, 1934). In the case of hypertension, the accumulation of cholesterol in the human aorta was predictable, and this accumulation was a function of initial concentration (Holmes et al., 1958). Aortic

cholesterol concentration has been reported to parallel plasma cholesterol concentration in the chicken (Weiss and Fisher, 1959). Blood pressure itself was able to influence the metabolism of cholesterol in the human aorta (Holmes et al., 1958) and elevated blood pressure has been reported to accentuate atherogenesis (Daley et al., 1943).

If the theory of Daley et al. (1943) that hypertension accelerates atherogenesis in humans is correct, then even though the plasma cholesterol levels of male and female turkeys are not significantly different, the higher blood pressure of the males will accentuate atherogenesis.

Growing male BBB turkeys are very susceptible to spontaneous atherosclerosis (Gottlieb and Lalich, 1954; Carnaghan, 1955; Gibson and deGruchy, 1955; Speckmann and Ringer, 1962). These areas of vascular degeneration are found in the same general anatomical location of the aorta as that in which the rupture normally occurs. Early reports by Gottlieb and Lalich (1954), Carnaghan (1955) and Gibson and deGruchy (1955) suggested that aortic rupture is not only precipitated by a dissecting aneurysm, but occurs in association with spontaneous atherosclerosis. Vascular degeneration could weaken the vessel sufficiently so that it may hold under normal physiological pressures, but during stresses, when blood pressure is elevated, the vessel ruptures. Ringer (1962) has likened the atherosclerotic blood vessel to tubeless tires with a damaged sidewall. The tire will probably hold up over the smooth highway, but over the rough dirt road, it blows out as the bumps increase the pressure inside.

When the turkeys were divided into two groups, one having a low incidence (≤ 2.00) and one having a high incidence (> 2.00) of atherosclerosis, expected cardiovascular changes occurred with rising systemic

resistance. Turkeys with high aortic atherosclerosis had a higher mean carotid blood pressure, which was due mainly to an increased systolic blood pressure, cardiac output, and minute volume, than did the low incidence group as the heart tends to overcompensate. This means it is working harder. Since heart rates of the two groups were essentially the same, the stroke volume was higher in the group with the higher atherosclerotic severity. Popliteal blood pressures did not exhibit as noticeable a difference as did carotid blood pressures when they were divided into groups based on atherosclerotic severity. There were no apparent differences between popliteal hemodynamics in the two groups. Following the injection of epinephrine, the low incidence group demonstrated a marked increase in carotid mean blood pressure. The greatest percentage increase was in systolic blood pressure which was reflected in an elevated pulse pressure. Systolic blood pressure rose 37 percent as compared to only a 25 percent increase in the high incidence group. Although there were no differences between popliteal blood pressures of low and high incidence groups before epinephrine, there was quite a difference following the administration of epinephrine. Popliteal mean blood pressures demonstrated greater percentage increases following the injection of epinephrine than did carotid mean blood pressures. Again systolic blood pressure was responsible for the tremendous increase in mean blood pressure. The popliteal systolic blood pressure increased 43 percent in the low incidence group as compared to only a 25 percent increase in the high incidence group. This was reflected in a greater percent change in pulse pressure of the low incidence group (71 percent) as compared to the high incidence group (52 percent). Perhaps the higher initial

blood pressure of the high incidence group was responsible for a lower percentage increase following epinephrine injection. The decreased cardiac output, however, was greater in the high incidence group (27 percent) than in the low incidence group (17 percent), possibly because the high incidence group had a greater cardiac output at the beginning.

When vascular degeneration is produced by BAPN, which interferes with connective tissue metabolism, a condition very similar to aortic rupture occurs. Waibel (1960) and Barnett (1960) reported that reserpine retarded the onset of BAPN-induced aortic rupture. Reports from Colorado indicate that vitamin C (Thornton, 1960) also delayed the onset of BAPN toxicity and may possibly afford protection against aortic rupture by its anabolical influence on collagen metabolism. Collagen is responsible for most of the strength of blood vessels, especially during hypertensive conditions. These findings place considerable emphasis on vascular composition, both quantitatively and qualitatively, and on their orientation.

Studies were therefore conducted to determine aortic elasticity and to evaluate the influence of BAPN, ascorbic acid and atherosclerosis on these values. Elasticity is that property of materials which enables them to resist deformation by the development of a resisting force or tension. The term must not be confused with distention. Pressure and tension are the forces which determine the equilibrium in a cylindrical vessel. The law of Laplace states that the pressure and radius of a vessel determine the tension that is developed. The greater the tension, the higher the elastance. Wolf (1952) believes the phenomenon of aneurysmal expansion can be explained by the Laplace equation. Pressure within a cylindrical vessel is the same in the

distended portion as it is in the undistended portion. Tangential tension in the wall, however, varies with the radius, being very high in the distended portion and low in the undistended portion. Rupture therefore, when it occurs, will involve the distended portion. According to Hooke's Law, perfectly elastic or Hookean substances have a linear stress-strain relationship. This proportionality is expressed in familiar elastic moduli such as Young's modulus. The aorta, being a visco-elastic system, does not demonstrate such linearity between stress and strain (Bergel, 1961). Assuming blood vessels to be cylindrical and the evidence that the arterial wall is more extensible longitudinally than circumferentially (Fenn, 1957), if change in length is prevented, then the effective circumferential modulus becomes a function of the radial and circumferential parameters (Lambossy and Müller, 1954).

The Young's modulus of an isotropic tube, which does not change in length on inflation, was given by Love (1927). This equation was rearranged into an incremental modulus by Bergel (1961). These researchers, along with Burton (1951) and Balkrishna et al (1961) have applied the law of Laplace to the aorta as they have used the change in radius with a given pressure change to be indicative of the tension developed. Love's equation has been rearranged so that a change in volume with a given pressure change is indicative of the vessel's elasticity. Although it is more practical to relate the modulus of elasticity to pressure, the parallel arrangement and function of the constituents in the arterial wall (Reuterwall, 1921) implies that the properties of the arterial wall are more directly related to radius than to pressure. Blumenthal (1956c) emphasizes this point as he reports that blood pressure only changes by a factor of four between

the aorta and capillary bed whereas radius changes by a factor of 325. Peterson et al. (1960) also related wall thickness and radius to elasticity.

Reuterwall (1921) was the first to introduce the idea of "accommodation" that later became known as "hysteresis" (Wood, 1954). Remington (1955) and Bergel (1961) observed a gradual increase in vessel size following several inflations and deflations which were accompanied by narrowing of the hysteresis curve. Turkey aortas demonstrated their greatest distensibility at the physiological blood pressure range. Their extensibility was also impaired at pressures above and below the physiological systolic blood pressure range. Roy (1880) studied the relation between internal pressure and volumetric capacity and also concluded that the aortic walls were most extensible at pressures corresponding to the normal blood pressure of the animal and at higher blood pressures the extensibility of the vessel wall was considerably impaired.

Banga and Balo (1961) reported that the data for tensile strength seemed to reflect more truly the severity of atherosclerosis than did age, while the opposite was true for the modulus of elasticity. Burton (1954), Peterson (1960) and Levene (1961) concluded that collagen plays a direct role in the maintenance of tensile strength of the aorta against abnormal stress. Lykke et al. (1960) found that the aortas of BAPN-treated mice had lower breaking strains than did controls. Levene and Gross (1959) had previously reported that the capacity to maintain aortic tensile strength depends not only on the quantity, but also on the state of aggregation of the collagen present.

The distensibility of the aorta, on the other hand, depends upon

the elastic properties of its components. The high degree of distensibility of the aorta has been attributed to its large percentage (30-40) of elastic tissue present (Hass, 1942). Nichol (1955) has shown that the distensibility of the aorta of rabbits was increased in experimental cholesterol atherosclerosis due to the deposition of cholesterol and lipids in the intima of the arteries. Balkrishna et al. (1961), also working with rabbits confirmed the work of Nichol (1955) but in addition found that in the cholesterol fed rabbit, the distensibility of the lateral diameter was increased whereas along the ventral diameter the distensibility was decreased. Further, these researchers found preferential sites for cholesterol deposition and plaque formation along the areas of greater distention. Burton's (1951) theoretical equilibrium diagrams further show how, in the presence of weakened elastic tissue, as occurs in atherosclerosis, a slight increase in pressure may cause the vessel to rupture.

There was a tendency for BAPN to decrease and vitamin C to elevate the elastance values. This became more significant at elevated pressures, when aortic rupture probably occurs. Of significant interest was the fact that the aortas from a high number of turkeys receiving BAPN ruptured during the elastance determination. The rupture occurred only in the thoracic aorta and could not be produced in the abdominal aorta even when pressures of 500 mm Hg were exceeded. This suggests that the tensile strength of the aorta may be more important than its elasticity. This theory was also suggested by Hass (1942) and Banga and Balo (1961).

Further, histological examination of atherosclerotic and non-atherosclerotic aortas by the author (not reported) showed that in

non-atherosclerotic aortas, the elastin and collagen components are arranged in an orderly, laminated manner which is probably necessary for their greatest strength as a visco-elastic system. In the aortas affected with atherosclerosis, the vascular components were very disorganized with considerable lipid infiltration, reaching far into the media. Vitamin C increased and BAPN decreased the number of collagen fibers per unit area but did not correct this vascular disorganization. With vitamin C, elasticity is improved, but maximum tensile strength is not attained.

Also of interest in the elasticity data was the fact that elastances at lower pressures were high; they then decreased to a point and then increased consistently with an increased pressure. The increase in elastances at low pressures has been ascribed to the muscular elements in the arterial walls. These are thought by Remington (1957) to be less distensible than elastic tissues, but also to have a larger viscous "dashpot" in series (Maxwellian element). This would in time give a decrease in the effect of the muscle-restoring force at a given length (Burton, 1954). This explanation, however, for the increase in elastance at lower pressures is not in agreement with the results of Alexander (1947) who found that the rate of distensibility did not greatly alter the pressure-volume curve of an isolated dog aorta, which in essence, tends to obliterate the significant dashpot concept. The increase of elasticity at the higher pressure is thought to be related to the increased stretch put on some of the connective tissue elements in the arterial wall (Burton, 1954; Remington, 1957).

Although atherosclerosis seems to be involved in the aortic rupture syndrome, the high blood pressure of the male BBB turkey may aggrevate this condition by causing abnormal cholesterol metabolism in the aorta, increased plaque formation and arterial degeneration thus reducing distensibility and paving the way for rupture. The hypertension experienced following a stress may be the pressure-head necessary to rupture the weakened aorta. The benefit of reserpine to the aortic rupture syndrome may be due to its hypotensive action and ability to smooth hemodynamic fluctuations which occur during stress rather than its ability to alter cardiac dynamics or vascular components.

SUMMARY

Plasma cholesterol level was found to increase with age in both male and female Broad Breasted Bronze (BBB) turkeys from 4 weeks of age to approximately 16 weeks of age after which age the plasma cholesterol level plateaued. The adult plasma cholesterol level was determined as 249±8 mg percent for commercially purchased turkeys and 344±13 mg percent for M.S.U. turkeys. There was no sexual difference in plasma cholesterol level.

In these experiments atherosclerosis began early in age, for by 8 weeks of age all turkeys had either microscopic or macroscopic atherosclerotic lesions. Atherosclerotic scores of the abdominal aorta indicated a greater severity for the males than for the females. Sporadically, certain individuals showed evidence of severe atherosclerosis. Individual aortas showed gradations in atherosclerotic severity with aging of the birds. Reserpine administration at the level of 0.1 ppm and 0.2 ppm in the feed did not offer any protection against the increase in plasma cholesterol or the severity of atherosclerosis with advancing age.

The cardiac output of untreated mature male BBB turkeys was determined by an isotcpe dilution technique using radioactive-phosphorus (P^{32}) as the indicator. Common carotid and popliteal arterial blood pressures were measured directly and were recorded simultaneously with the cardiac output determinations pre- and post-epinephrine injection by means of two strain guages connected to a recording polygraph.

From the cardiovascular measurements systemic resistance was calculated. The mean cardiac output of mature male BBB turkeys was 231 ± 18 ml per $Kg^{0.734}$ per minute. The cardiac parameters were not affected by strain but were significantly influenced by atherosclerotic severity. Turkeys with a higher incidence of atherosclerosis had a greater minute volume, cardiac output and stroke volume than turkeys with little or no atherosclerosis. The injection of 0.5 ml of 1/25,000 epinephrine caused a negative cardiovascular response; minute volume, cardiac output and stroke volume decreased.

Blood pressures increased with advancing age in both male and female BBB turkeys. Several of the hemodynamic parameters were influenced both by strain and atherosclerotic severity. The M.S.U. strain of turkeys had a significantly higher systolic blood pressure than did the commercial strain of turkeys with the cross between the two strains of turkeys falling in between. The group of turkeys with the highest atherosclerotic score also had the highest systolic blood pressure. The popliteal arterial blood pressures were consistently lower than their common carotid artery counterparts and were not influenced as much by strain and atherosclerotic severity. The injection of epinephrine caused a pronounced elevation of blood pressure. This was accompanied by a negative cardiovascular response and a concomitant increase in systemic resistance which elevated blood pressure. Reserpine significantly reduced blood pressure whereas BAPN and vitamin C had no affect.

Partial correlation coefficients of three variates of cardiovascular and hemodynamic parameters indicated that systolic blood pressure was positively correlated with diastolic blood pressure, heart rate and

blood vessel thickness. Diastolic blood pressure was positively correlated with heart rate and blood vessel thickness but demonstrated a negative correlation with heart weight. Blood vessel thickness was positively correlated with pulse pressure but negatively correlated with heart rate. Pulse pressure, heart weight, and percent change in systolic blood pressure following epinephrine injection were all positively correlated with body weight whereas systolic blood pressure, heart rate and thoracic elastance at 250 mm Hg were negatively correlated with body weight. Minute volume was positively correlated with stroke volume and negatively correlated with heart rate whereas stroke volume was positively correlated with heart rate but negatively correlated with systolic blood pressure and blood vessel thickness. The percent change in systolic blood pressure following the injection of epinephrine was negatively correlated with diastolic blood pressure, pulse pressure, blood vessel thickness and thoracic elastance at 250 mm Hg. Abdominal elastance or atherosclerotic severity could not be correlated with any normal cardiovascular or hemodynamic parameters.

A method for the determination of the static modulus of elasticity for the thoracic and abdominal aorta and an equation rearranged for elastance calculations as a function of change in volume have been developed. The aortic segments were most distensible in the physiological blood pressure range, the pressure-volume curves being sigmoid in nature. The vessel segments also exhibited hysteresis. The thoracic aortas were much more distensible than the abdominal aortas; the elastances of untreated male BBB turkeys being in the neighborhood of 3.28 ± 0.11 and 13.95 ± 0.24 dynes/cm^2 X 10^5 respectively in the physiological pressure

range. This difference becomes more pronounced at higher pressures. At pressures below (25-150 mm Hg), at (175-350 mm Hg) or above (375-500 mm Hg) the physiological blood pressure range, ascorbic acid treatment tended to increase, whereas BAPN treatment tended to decrease, elastance values.

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APPENDIX

I Modified Method for the Direct determination of Serum Cholesterol

REAGENTS

I Standard Cholesterol Solution (1 mg per milliliter): Dissolve 100 mg of pure, dry, ash-free cholesterol in 100 ml of 100% glacial acetic acid (use only Merck glacial acetic acid conforming to the dichromate test).

II Ferric Chloride Solution: Dissolve 10 gms. of ferric chloride, reagent grade (Mallinckrodt ferric chloride) in 100 ml of 100% glacial acetic acid (Merck).

III Color Reagent: Dilute 2.0 ml of the ferric chloride solution to 200 ml with C. P. concentrated sulfuric acid (Baker). To prevent ferric chloride from precipitating, add 100 ml of sulfuric acid into volumetric flask then swirl while slowly adding 2.0 ml of the ferric chloride solution. Then bring entire solution to 200 ml with sulfuric acid.

PROCEDURE:

Coleman Spectrophotometer

pc 14

Wavelength 560 millimicrons

Filter 14-214

Light path 13.5 mm

Amount of Standard (1 mg/ml)	Amount of Glacial Acetic Acid	Distilled Water	Color Reagent
0.1 ml	9.9 ml	0.1 ml	7.0 ml
0.2	9.8	0.1	7.0
0.3	9.7	0.1	7.0
0.4	9.6	0.1	7.0
0.5	9.5	0.1	7.0
Blank	10.0	0.1	7.0
Plasma (0.1 ml)	9.9	--	7.0

To 0.1 ml of plasma in a dry, clean 30 ml test tube add 10.0 ml of Merck glacial acetic acid. To this add 7.0 ml of the color reagent and mix immediately. It is desirable to run each tube in duplicate, also each tube must be carried through separately and mixed immediately by pouring from one tube to another several times to insure proper mixing. Measure the percent transmission of the solution after it has come to room temperature and determine the total cholesterol content from a calibration curve. Cork test tubes while cooling to avoid loss of mixture. The serum or plasma may be frozen once, but it must be shaken after melting for accurate distribution of cholesterol.