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Signature Douglas W. Minnig
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RELATIONSHIP BETWEEN THE EAR CREASE SIGN AND CORONARY
RISK FACTORS IN A POPULATION OF SECURITY GUARDS

A Thesis
Presented for the
Master of Science
Degree
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Douglas Wayne Minnix
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DEDICATION

This thesis is dedicated to my Lord and Savior

Jesus Christ

Who has been with me from the beginning.

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ABSTRACT

The relationships between the ear crease sign and coronary risk factors in 168 healthy men ranging in age from 23 to 60 years, were studied. The ear crease was examined while the subjects were sitting and was deemed present if it existed as a clear-cut diagonal wrinkle extending across either ear lobe. The waist (Wst) and hip circumferences were measured by a standard tape measure and waist-to-hip ratio (WHR) was determined. The subject's age, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), total cholesterol level (Chol), high-density lipoprotein (HDL), total cholesterol-to-high-density lipoprotein ratio (Chol/HDL), weight (Wt), sum of skinfolds (SSK), body mass index (BMI), height (Ht), and treadmill time to 85% of maximal heart rate (T-mill) were extracted from their medical records. Of the coronary risk factors studied, WHR, Wst, Chol, DBP, MAP, and age were significantly greater in subjects with the ear crease than in subjects without the ear crease. After adjusting for age, only WHR, Wst, DBP, and MAP were significantly greater in subjects with the crease than in subjects without the crease. The prevalence of subjects with and without the crease was not statistically different with regards to a history of smoking or a family history of coronary artery

disease (CAD). The results of this study infer that the crease is related to certain coronary risk factors such as WHR, Wst, DBP, and MAP in a population of healthy males. It is possible that the presence of a crease may reflect an overload of coronary risk factors, rather than any one risk factor alone.

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CHAPTER I

INTRODUCTION

Interest in the early diagnosis and prevention of coronary artery disease (CAD) has stimulated research for simple, noninvasive predictors of the disease (30). A diagonal ear crease, which is a wrinkle extending across the anterior portion of the lobe, has been suggested to be such a predictor (21,22).

At present, there is controversy concerning the relationship between the ear crease and CAD and coronary risk factors. For example, Haft et al. (16) found that the ear crease sign produced a high number of false-negative tests in a population of patients with a high incidence of CAD. Other authors have suggested the ear crease to be related to age but not to CAD (5,25). Also, there is research which demonstrates that individuals with the crease have higher blood pressure, total serum cholesterol (Chol), and are more obese than individuals without the crease. (17,27,32). Conversely, other studies have not demonstrated a difference in Chol, high-density lipoproteins (HDL), blood pressure, smoking, and obesity for individuals with and without the crease (18,19).

Since the presence of an ear crease has been linked to

certain coronary risk factors, additional research seems warranted to define other possible relationships (34). Specifically, associations between the ear crease and waist to hip ratio (WHR) and physical fitness level have not been examined. Both of these traits have been shown to be risk factors for CAD (6,36).

Most of the previous studies have used either pre-mortum or post-mortum patients with or without CAD as subjects. Only a few studies have examined the relationship between coronary risk factors and the ear crease in relatively healthy populations. Of these studies (32,27,18) using a "normal population" none have used a population of subjects that exercised on a regular basis. One should note that inactive individuals have been shown to be at a higher risk of CAD than active individuals (28). Moreover, the relationship between the ear crease and coronary risk factors may be influenced by a population's activity level.

The purpose of this study was to examine the differences between the coronary risk factors among individuals with and without the crease in a "normal population" that exercised on a regular basis. The risk factors that were examined were Chol, total cholesterol/high density lipoprotein cholesterol ratio (Chol/HDL), mean arterial pressure (MAP), body composition (weight, body mass index and sum of skinfolds), smoking, and family history of

CAD. Since age has also been associated with the ear crease, this study examined the influence of age on the relationship between these coronary risk factors and the ear crease.

Another purpose of the present investigation was to observe the differences in WHR and physical fitness levels among individuals with and without the ear crease.

CHAPTER II

REVIEW OF LITERATURE

This review of literature deals with the relationship between CAD, various coronary risk factors and the ear crease. A summary table of these studies can be found in Appendix B.

The original research suggesting an association between the ear lobe crease and coronary artery disease was done by Frank (11) in the early 1970's. Frank noted that 19 patients with a positive ear lobe crease also had one or more of the known risk factors for CAD. Frank's observation led to the suggestion that the ear crease was related to premature CAD.

Lichstein et al. (21) assessed the relationship between CAD, coronary risk factors and the presence of earlobe creases. This study included 531 patients with acute myocardial infarction and 305 randomly selected controls with no clinical evidence of CAD. The patients with acute myocardial infarctions had been under coronary care observation. The controls were patients with no recorded electrocardiographic evidence of abnormal Q waves or ST-segment and T-wave abnormalities. Questionnaires concerning the presence or absence of the ear crease and coronary risk factors, such as hypertension, diabetes

mellitus and smoking habits were completed for both groups. The investigators noted that 47% of the CAD patients exhibited a diagonal ear crease, whereas 30% of the control group had the ear crease. Although the incidence of the ear crease sign was shown to increase from the third to the sixth decade of life, the prevalence of the positive ear crease was higher in the CAD group than in controls for each decade. Of additional interest, Lichstein et al. noted a striking difference in the incidence of the ear crease sign in the CAD subjects than in controls in the 50 to 59 age group. Furthermore, no significant difference was found in the prevalence of diabetes mellitus, hypertension, or smoking in subjects with or without the ear crease. From these results, the authors concluded that the ear crease is not simply a result of common risk factors. Instead, the investigators suggested that the ear crease should be regarded as a sign of CAD, and used to identify patients at risk.

Lichstein and associates' proposition has generated a considerable amount of controversy. Scott (34) noted that the possible relationship between obesity and the ear crease sign was not investigated in Lichstein's study. Scott also mentioned that environmental factors such as patients habitually tugging at their ears were not investigated. According to Scott, one can not infer that the ear crease is

a sign of CAD until possible independent relationships between the ear crease and variables such as coronary risk factors and other environmental factors have been ruled out.

Mehta and Hamby (25) expressed concern that Lichstein's method of examining coronary arteries was lacking. Using arteriography as their technique for determining normal coronary arteries, Meta and Hamby found different results than Lichstein. The investigators found that CAD was present in 159 of 211 patients. Additionally, Meta and Hamby demonstrated that only 16.3% of the patients with CAD had the ear crease whereas 50% of the patients with normal coronary arteries had the ear crease. No statistical significance was found between the two groups. Furthermore, the investigators noted that 65% of the patients over 55 had an ear crease, whereas only 34% below the age of 55 had an ear crease. Thus, Meta and Hamby concluded that the prevalence of the ear crease was not related to CAD, but could be related to the aging process.

Lichstien et al. (23) criticized the size of the control and study groups in Meta and Hamby's research, and suggested that a larger study and control group might produce statistical significance between the groups. Lichstien et al. also suggested that additional information concerning the control group was needed since they were selected, rather than being chosen at random. Furthermore,

Lichstein et al. indicated that the examination of two age groups, those over 55 and those under 55, was not adequate to determine that the crease was related solely to age, independent of CAD. Lichstien et al. found the most striking difference between the study and the control group when the groups were divided into decades.

The controversy between Lichstien et al. (23) and Mehta and Hamby led to more controlled studies. Sternlieb et al. (38) were the first to demonstrate a relationship between the presence of ear creases and coronary arteriographic evidence of the presence or absence of CAD. The investigators noted that out of 133 patients with ear creases, 120 (90%) had CAD, whereas 10 of 11 (91%) patients without ear creases had normal coronary arteries.

Christiansen et al. (7) examined 203 female and 320 male patients admitted to a medical and a surgical department, for the following characteristics: diagonal ear crease, acute myocardial infarction, CAD, arterial hypertension, smoking habits, and diabetes mellitus. The prevalence of the ear crease was shown to increase with age in controls. However, the ear crease was found to be more common in patients with CAD than in controls for each decade above 50 (50-59, 60-69, 70-79, and >79). In the group of patients 50-59 years old a significantly higher frequency of the ear crease was found in patients with CAD than in

controls. No significant difference was reported between the patients with CAD and controls of earlier or latter decades. Additionally, a higher correlation was found between CAD and the ear crease than between coronary artery disease and risk factors such as arterial hypertension, cigarette smoking, and diabetes mellitus. The investigators concluded that the ear crease was independently related to CAD despite its relationship with advancing age and coronary risk factors.

In a later study by Lichstein et al. (22) the status of the ear crease (bilateral, unilateral, or absent) was compared to the degree of coronary artery occlusion in 113 postmortem subjects who were at least 40 years of age. The coronary arteries were examined by one investigator without knowledge of the ear crease findings. The arteries were dissected longitudinally along their entire extramural course and evaluated for the degree of sclerosis of the arterial wall and the degree of occlusion of the arterial lumen. The amount of sclerosis was graded from 0 to 4, with 0 being the least and 4 the greatest involvement. The degree of occlusion of the arterial lumen was also graded from 0 to 4, with 0 designating no occlusion and 4 indicating complete occlusion. An analysis of variance showed a statistically significant difference in the amount of sclerosis of the coronary arteries in the no crease group when compared with the bilateral crease group. Further analysis demonstrated a

significant difference between these two groups after adjusting for age. No significant difference was found when the relationship between the patients' sex and ear crease status was examined. Although non-significant, a trend was noted between the status of the ear crease (bilateral, unilateral, or absent) and the degree of coronary artery occlusion.

Because a relationship between the ear crease and CAD has been demonstrated by several investigators, Sprague (37) assumed that the crease could be used as an indicator of operative cardiovascular complications. In order to test this hypothesis, Sprague examined 222 patients undergoing elective surgery for the presence or absence of the diagonal ear crease, clinical evidence of CAD, intraoperative cardiovascular complications, and postoperative cardiovascular complications. The ear crease was evident in 3 of 120 patients less than 40 years of age and 61 out of 102 patients more than 40 years of age. The prevalence of the ear crease in patients who had CAD was greater than that in patients without clinical evidence of CAD for each decade above the third. Beyond the third decade, 41 of 50 patients (82%) who had CAD were found to have the ear crease. Only 20 of 52 (38.5%) patients without CAD had the ear crease. Above age 40, 42% percent of the patients who had the ear crease developed intraoperative cardiovascular complications,

verses 4.9% of the patients without the ear crease. Twenty-four percent of patients with the crease had postoperative cardiovascular complications, compared with only 4.9% of patients without the crease. These data led the investigator to conclude that the diagonal ear crease is positively related to CAD and not related solely to advancing age, as others have suggested. Furthermore, Sprague suggested that the crease can be used as an indicator of operative risk.

Until 1976, much of the research suggested that the ear crease was related to age, coronary risk factors, or CAD. In 1976, Andresen and associates (3) proposed yet another possible explanation of the ear crease. The investigators examined 101 patients greater than 30 years of age who were suffering from diabetes mellitus for the diagonal ear crease and presence or absence of diabetic retinopathy. Andresen et al showed that patients with retinal angiopathy had a significantly higher frequency of ear creases than the patients with normal retinal vessels. The mean age in the two groups did not differ significantly. The authors concluded that the diagonal ear crease was positively related to diabetic retinopathy. In addition, Andresen and colleagues stated that earlier research, which suggest an association between the diagonal ear crease and coronary angiopathy, in conjunction with the results of their study

indicate a correlation between the ear crease and generalized vascular disease.

In 1977, Frank (12) provided additional evidence that suggested a relationship between the ear crease and generalized vascular disease. Frank observed 2 men, 53 and 51 years of age, with classic angina, positive stress tests and an ear crease. Neither were hypertensive or diabetic and both had normal coronary arteriograms. Although Frank suggested that his observation may reflect a relationship between cardiac small vessel disease and the ear crease, he indicated that such a hypothesis warrants further study before any conclusions can be made.

Doering et al. (9) carried out a study intended to resolve the controversies of the ear crease. During a 10-week period, Doering and associates evaluated 88 patients who were 30 to 80 years of age for Chol, blood pressure, smoking histories, diabetes, CAD, height (Ht), weight (Wt), and the ear crease sign. The ear crease was defined as a deep wrinkle in the lobular portion of the auricle extending for a distance greater than or equal to one-third the length of the ear lobe. The ear crease was determined to be present if it was unilateral or bilateral. A patient was considered to have CAD if he or she were diagnosed with CAD or had a history of angina pectoris, previous myocardial infarction or electrocardiographic evidence of myocardial ischemia.

Statistical analysis revealed a significant difference between the controls and patients with CAD with regard to age, Wt, having ever been a smoker, and the incidence of diabetes and ear creases. After controlling for age and gender, statistical analysis showed that patients with CAD and controls differed with respect to Wt and the presence of the ear crease. The authors concluded that although the ear crease seems to be related to age, age alone does not account for the relationship between CAD and ear creases. Additionally, the investigators suggested that since both the incidence of creases and the incidence of CAD are related to age, both could be functions of changes occurring in the vascular system. Doering et al. believed that since the ear lobe is a highly vascularized structure, it may show these changes by starting to crease and fold.

Pasternac et al. (29) investigated the relationship between degenerative skin changes and coronary atherosclerosis. Twenty-six patients undergoing aortocoronary bypass or valve replacement were observed for cutaneous degenerative changes and coronary atherosclerosis. The severity of cutaneous changes were determined by dermal biopsies. The severity of atherosclerosis was determined by arteriography. Although the workers found an overall correlation between the severity of degenerative skin changes and coronary atherosclerosis, severe coronary

atherosclerosis was found to occur independently of skin changes. Pasternac et al. concluded that the predictive value of degenerative skin changes are limited.

Haft et al. (16) examined 445 patients for the arteriographic evidence of CAD and the presence or absence of an ear crease. An ear crease was considered to be present if there was a diagonal or a longitudinal line across the earlobe connecting the tragus to the edge of the lobe. CAD was considered present if coronary arteriography illustrated a stenosis of 50% or more. The crease was found in 132 of 243 (54.3%) men with CAD compared with 19 of 66 (28.8%) men without CAD. It was found in 27 of 63 (42.9%) women with CAD compared with 14 of 73 (19.2%) women without CAD. To determine whether age influenced the relationship between CAD and the ear crease sign the data were statistically adjusted for age. Patients were divided into the following age groups: greater than or equal to 39, 40 to 45, and 60 and older. The age-adjusted data revealed no significant differences between men and women with CAD and men and women with normal coronary arteries with respect to the incidence of the ear crease sign. Further analysis showed that among 151 men with an ear crease 132 (87.4%) also had CAD. Among 41 women with an ear crease, 27 (65.9%) had CAD. The investigators concluded that although the specificity of the sign was high (87.4% for men and 65.9% for women) the

sensitivity was low (54.3% for men and 42.9% for women). The presence of a crease increased the chances that CAD was present by factors of only 1.25 in men and 1.74 in women. According to the authors, if their data were extrapolated to a hypothetical asymptomatic population with a hypothetical 10% incidence of CAD, only 1 in 5.77 asymptomatic men with the ear crease sign would be expected to have CAD.

Kaukola et al. (19) studied 286 randomly selected patients who had undergone coronary angiography because of typical symptoms or signs of CAD or atypical chest pain. Out of 286 patients, 200 were diagnosed with at least 50% stenosis in one of the main epicardial arteries. One-hundred and forty-four (72%) of the patients diagnosed with CAD had the ear crease whereas only 18 (21%) of the controls had the ear crease. The prevalence of the ear crease was shown to increase with age. However, within each age group (30-39, 40-49, 50-59) the prevalence of the ear crease among patients diagnosed with triple-vessel disease was greater than in controls. Furthermore, multivariate analysis showed no association between the ear crease and coronary risk factors such as increased Cholesterol and triglycerides, low HDL, hypertension, smoking, diabetes, and obesity. The investigators concluded that a relationship between the ear crease and CAD was not simply the result of a pseudo-correlation.

Shoenfeld et al. (35) investigated correlations between the ear crease and coronary risk factors (hypertension, diabetes, smoking, hyperlipidemia and hyperuricemia), myocardial infarction, and ethnic origin (Ashkenazi Jews versus non-Ashkenazi Jews) as well as with diabetic and hypertensive retinopathies. Subjects were diagnosed with a myocardial infarction on the basis of the clinical picture, electrocardiographic changes, and enzyme elevations. The presence of an ear crease was determined on both sides while the subject was supine and seated. The ear crease was defined as a deep wrinkle in the lobular portion of one or both auricles, extending for a distance greater than or equal to one-third of the ear lobe length. In addition, the investigators performed an earlobe biopsy on 12 of the subjects. A higher prevalence of the ear crease was found among myocardial infarcted patients (77%) than controls (40%). In addition, statistical analysis revealed a significant association between subjects with a myocardial infarction and subjects with an ear crease in all age groups (30-50, 51-60, 61-70, 71-80, 81-90). However, only one of the coronary risk factors, hypertension, was significantly related to the ear crease in subjects thirty to fifty and sixty-one to seventy years of age. No risk factor was found to be related to the ear crease in all age groups. Furthermore, a higher prevalence of the ear crease was found

in patients of Ashkenazi origin than patients of non-Ashkenazi origin. Additionally, a higher prevalence of the ear crease was found among patients in whom myocardial infarction was combined with diabetic retinopathy or hypertension. The earlobe biopsies revealed torn elastic fibers in all subjects with myocardial infarction and/or ear lobe crease. The investigators concluded that these data show a relationship between the ear crease and myocardial regardless of age or the presence or absence of coronary risk factors. The authors also inferred that the ear crease could be a marker for patients at risk for myocardial infarction in which coronary risk factors have been masked by genetic influences. Since the ear crease was more prevalent among myocardial infarction patients of Ashkenazi origin with hypertension and/or diabetic retinopathy, the authors suggested that the ear crease could be an indication of coronary risk factors which may be genetically inherited.

Kristensen (20) examined the prevalence of the bilateral, diagonal ear crease in normotensive subjects and hypertensive subjects with indications of vascular problems. The relationship between the ear crease, hypertension, and atherosclerotic C3-F gene was also studied. The ear crease sign was found in 19 out of 74 (25%) hypertensive males and in only 1 out of 29 (3%) normotensive control males. Corresponding figures for females were 5 out of 51 (8%) and

2 out of 26 (7%). Comparison of age-matched hypertensive males with and without the crease demonstrated that 13 out of 19 (68%) males with the crease had either angina, myocardial infarction, congestive heart failure, stroke, or retinal thrombosis. In males without the crease 2 out of 15 had claudication while the remainder had no vascular complications. The relative risk for vascular complications among hypertensive subjects with the crease was 10 times higher than the relative risk for hypertensive subjects without the crease. The atherosclerotic C3-F gene was found in 7 out of 19 (37%) hypertensive males with the crease, in 4 out of 14 (29%) without the crease, and in 7 out of 31 (22%) normotensive males. No association was found between the ear crease and serum lipids or a positive family history of hypertension. Kristensen suggested that these results support an association between the diagonal ear crease and CAD, and hint at a possible relationship to the atherosclerotic C3-F gene.

Fisher and Sievers (13) examined 200 consecutive full-heritage Southwestern American Indian patients for the presence of an ear crease, CAD, and coronary risk factors. A bilateral or unilateral ear crease was sought while each patient was in a sitting position. CAD was deemed present if there was a manifestation of angina pectoris, myocardial ischemia. Statistical analysis of age and sex adjusted data

revealed no significant association between the ear crease and CAD. Furthermore, there was no significant relation between the presence of a crease and coronary risk factors such as diabetes mellitus, hypertension, body mass index (BMI), smoking history, or tribal affiliation. The investigators concluded that the presence or absence of the ear crease was not influenced by underlying pathological conditions in patients of this ethnic origin. However, there appeared to be a relationship between age and the ear crease.

Merlob et al. (26) sought to determine the incidence of the ear crease in newborn infants and in older school children. In addition, correlations between the ear crease and CAD as well as coronary risk factors were examined. Of 1,039 infants examined, the ear crease was found in 48 (4.6%) newborns during the initial examination. None of the infants had additional congenital anomalies. Only 1 mother of the newborn infants had an ear crease. One mother suffered from juvenile diabetes, 2 had hypertension during their pregnancy, while the remaining 45 were healthy. A family history of the infants revealed that 2 grandparents had CAD, 7 had diabetes, and 4 had hypertension. At the time of discharge, which was usually 2 or 3 days after birth, the ear crease was present in only 6 infants. There was no family history indicative of CAD or any other risk factor in

any of the 6 infants. After 4 to 7 months, the ear crease had disappeared in all but 2 of the 48 infants. Of the 685 school children examined, only 1 had the ear crease. Merlob and colleagues implied that the ear crease found at birth is due to pressure and folding of the ear lobule in utero and is less likely to be associated with premature CAD. The investigators suggested that the ear crease, as a coronary risk factor, develops in later life.

Pasternac and Sami (30) have questioned the relationship between coronary atherosclerosis and the ear crease independent of age. To assess this relationship, the investigators examined the predictive value of the ear crease sign and its relation to age and other coronary risk factors. Pasternac and Sami examined 340 patients consecutively admitted to the Montreal Heart Institute for coronary arteriography. Each patient was studied for the ear crease, a personal history of myocardial infarction, a family history of myocardial infarction or sudden death before the age of 55, cigarette smoking, corneal arcus, age, systolic and diastolic blood pressures, and Chol, triglycerides, and glucose. The presence or absence of CAD was assessed by angiography. The ear crease was determined to be present if it was a unilateral or bilateral diagonal crease of the lobular portion of the auricle(s). CAD was demonstrated in 91.1% of the patients with the ear crease

and in 60.5% of those without the sign. The predictive value of the absence of the sign was 39.5% and the positive predictive value of the sign was 95.7% for men and 66.7% for women. The ear crease was significantly more common among patients with severe CAD (50% stenosis or greater). The only risk factor which was significantly more common among patients with the crease than in patients without the crease was corneal arcus (33). Although there may be an association between corneal arcus and age, the presence of both corneal arcus and age further increased the probability of CAD. A linear correlation between age and the ear crease was observed both in the patients with and without CAD. However, the ear crease was also independently associated with CAD. The authors concluded that the ear crease could identify symptomatic patients with extensive CAD. In an asymptomatic population with a low prevalence of CAD the crease appeared to be of limited value in predicting CAD. However, Pasternac and Sami suggested that the sign may point out individuals who are aging more rapidly than is usual and are thus prematurely at risk of CAD.

Elliott (10) was the first to examine the relationship between CAD and the ear crease utilizing a large sample size from an American hospital. Elliott observed 1,000 patients in the supine position for the ear crease. The presence or absence of CAD was diagnosed by clinical and angiographic

criteria. As seen in many of the previous studies, Elliott's research indicated an increased frequency of the ear crease with advancing age. Furthermore, an association between the ear crease and CAD was also demonstrated. This relationship was present even after adjusting the data for age. Data for patients who underwent coronary angiography were separately examined. One-hundred and twenty-one patients of 205 who underwent coronary angiography had an ear crease and CAD. Thirty-nine of the 205 patients had neither CAD nor the ear crease and 27 had CAD but no ear crease. Statistical analysis indicated a highly significant relation between the ear crease and CAD. Finally, the risk factors belonging to the patients who underwent coronary angiography were analyzed independently. Only the ear crease and previous acute myocardial were found to correlate with the presence of angiographically demonstrable CAD. The ear creases did not correlate in a statistically significant manner with any of the other risk factors for CAD, nor with age or sex of the patients. Elliott concluded that the ear crease was a strong risk factor for CAD in a general medical patient population and a stronger prognostic factor in patients undergoing cardiac catheterization. Elliott indicated that it should be regarded as a risk factor rather than a diagnostic test since it has a sensitivity of only 60% and a selectivity of 65%.

Gral and Thornburg (15) investigated the frequency of ear creases in 234 veterans 24 to 99 years of age (mean 66). The investigators also examined the association of ear creases in this population with certain diseases as well as with a number of physiologic and laboratory determinations. The crease was considered to be present if it was either a unilateral or bilateral, diagonal wrinkle cut across at least 50% of the earlobes' surface. Clinical diagnoses were obtained from the patient's medical records. The laboratory data consisted of Chol, glucose, urea nitrogen, creatinine, uric acid, calcium, phosphorus, albumin, and hemoglobin. Other patient data included clinical diagnosis, age, sex, Wt, Ht, and blood pressure as well as history of smoking. Gral and Thornburg found the ear crease in 119 (51%) of 234 patients. Seventy-three patients (51%) with some form of cardiovascular disease (coronary artery disease, cerebrovascular disease, peripheral vascular disease, atherosclerotic heart disease, hypertension) also had an ear crease. The rest, 92 patients, had no clinically apparent cardiovascular disease. The incidence of earlobe creases in this group was 50%. Additionally, the ear crease was not statistically related to clinical diagnoses, laboratory data, or any other physiologic parameter. The creases were found to be present in about half the study group regardless of the diagnosis. Thus, it was concluded by the authors that

the ear creases were not specifically related to any cardiovascular diseases. However, Gral and Thornburg indicated that the significance of the ear crease in the diagnosis of CAD may be greater in a younger age group, because in the elderly these creases may occur as part of aging unaccompanied by cardiovascular diseases.

Jorde and associates (18) sought to determine if the presence of an ear crease was related to coronary risk factors. The sample consisted of 686 subjects who were relatively free from cardiovascular disease, but prone to hypertension, stroke or early CAD. Hypertension was present in 68, diabetes in 20 and CAD manifestations in 10. Data collected from each subject included diastolic and systolic blood pressures, cigarette smoking, Wt, Ht, scapular skinfold thickness, Chol, HDL, intracellular sodium, sodium-lithium countertransport, plasma renin level, and the presence of diabetes and hypertension. The ear crease was observed as a diagonal wrinkle extending across a third or more of the earlobe's surface while each subject resumed a standing position. In addition, familial aggregation of earlobe creases was tested using a subset of 82 adults older than age 30 in 29 sibships. No significant differences were noted between patients with and patients without an ear crease with regards to coronary risk factors. In addition, no familial aggregation was found for earlobe creases. The

investigators concluded that the ear crease was not associated with any other risk factor and may therefore be an independent diagnostic sign for CAD.

Gibson and Ashikaga (14) examined the relationship between the ear crease, CAD, and aortic valve stenosis. One-hundred adult Caucasian patients who were diagnosed with aortic stenosis (50% reduction in orifice size) were tested for hemodynamic severity of the obstructed valve and the degree of CAD involvement via cardiac catheterization and coronary arteriography. The minimum criterion for valve stenosis was a peak aortic valve gradient of 15 mmHg. Significant CAD was defined as narrowing greater than 50%. Each of these patients was also observed for the ear crease. The ear creases were classified as mild (Grade 1), moderate (Grade 2), or severe (Grade 3). A mild crease was defined by the authors as a superficial crease measuring less than a third of the distance from the tragus to the ear lobe edge, and severe as a deep indentation connecting the tragus to the lobe edge. A moderate crease was any finding intermediate to these. No useful statistical correlations were found. Gibson and Ashikaga concluded that the ear crease sign is of little practical value in influencing any particular clinical decision.

Brady et al. (5) evaluated 261 men undergoing coronary arteriography for the presence or absence of the ear crease.

Other parameters assessed by Brady and colleagues included age, medical and cardiac history, and smoking habits. Classification of CAD included normal coronary arteries (no lesions detected), minor lesions (less than 50% occlusion), and single-vessel (greater than 50% occlusion), double-vessel, and triple-vessel disease. The ear crease was deemed present if it was either a bilateral or a unilateral, diagonal crease of the lobular portion of the auricle. The investigators noted an ear crease in 85% of patients with some degree of CAD. However, 85% of patients without the ear crease also had some degree of occlusion. Furthermore, no statistical differences were noted among patients with minor lesions, single, double, or triple-vessel involvement and the proportion of corresponding ear creases. Patients with the ear crease had a greater prevalence of hypertension and were significantly older. No significant difference was observed between the occurrence of CAD in patients with and without the ear crease with increasing age. Multivariate analysis was carried out to simultaneously control for the influence of age and hypertension. The results from this analysis showed no relationship between the ear crease and the presence of CAD. The authors concluded that the association between the ear crease and CAD was due to the fact that the prevalence of both the ear crease and CAD increase with age.

Cumberland et al. (8) examined the relationship between the degree of coronary atherosclerosis and the presence of a diagonal ear crease in 800 consecutive autopsies performed for medico-legal reasons. Coronary arteries were considered significantly stenosed if one or more of the three main coronary arteries was narrowed greater than 75% by atheromatous lesions. Out of 593 patients with no ear creases, 62 were found to have significant stenotic coronary atherosclerosis. Out of 184 patients with the ear crease sign, 77 were diagnosed with significant stenotic coronary atherosclerosis. Statistical analysis by use of the chi-square test demonstrated a positive correlation between the presence of the ear crease and significant stenotic coronary atherosclerosis. The sensitivity of the ear crease sign was shown to be 55% and its specificity calculated at 83%. The positive predictive value of the ear crease was demonstrated to be 42% while the negative predictive value was determined to be 90%. The researchers concluded that there was a statistically significant correlation between the presence of the diagonal ear crease and the presence of severe stenotic coronary atherosclerosis. In addition, Cumberland and associates inferred that the absence of the ear crease may be indicative of the absence of significant coronary atherosclerosis. This suggestion was based on the ear crease sign's high specificity.

Ishii, et al. (17) studied the relationship between the ear crease and the extent of coronary and aortic atherosclerosis in conjunction with the age, blood pressure, and Chol of 100 autopsied men. Ear creases were graded and defined as groups 1, 2, and 3 according to the depth and length in both ears. The extent of atherosclerosis was estimated visually as the percentage of intimal area with macroscopically identifiable raised atherosclerotic lesions, fibrous plaques, calcified lesions, and complicated lesions in the total intimal surface area. As found in most studies, Ishii et al. observed an increase in the prevalence of ear creases with age. The extent of atherosclerosis was also found to increase with advancing age. However, the extent of the disease was greatest in the group where the ear crease sign was most evident (group 3) regardless of age. There were no statistical differences between groups 1 and 2 in relation to the disease. Aortic atherosclerosis was significantly greater in group 3 than in group 1 in all decades examined, and was greater in group 3 than in group 2 in the seventh and eighth decades. No significant differences were found in systolic or diastolic blood pressures between any of the groups. Chol was significantly higher in group 3 than in groups 1 or 2 except in the sixth decade. Multivariate analyses demonstrated that the degree of the ear crease was dependent on the extent of coronary

atherosclerosis, but was independent of age, Chol, and blood pressure. Conversely, the extent of coronary atherosclerosis was significantly and independently correlated with the degree of the ear crease, but not with age, systolic blood pressure, or Chol. As for the extent of aortic atherosclerosis, the factors of age, total ear crease score, and systolic blood pressure were all statistically significant predictors. The investigators concluded that the ear crease provides a significant external marker for atherosclerosis and may reflect a persistent overload of atherosclerosis risk factors, such as Chol, hypertension, and age.

Summary

The lack of rigorous study design and potential biases in research assessing the relationship between the ear crease, CAD, and coronary risk factors has been noted throughout the literature. Diversity in study designs included an inconsistency in criteria for determining CAD. One potential bias relates to the types of populations which have been used.

Many investigators believe that diversity in study design and potential biases has yielded conflicting results throughout the literature. Contrasting results have left substantial questions regarding the real association between

the crease and CAD. While many studies suggested that the ear crease is related to CAD and not age or coronary risk factors, others have suggested that the relationship is influenced by age and other coronary risk factors.

The purpose of this study is not to answer all the questions presented in the literature. However, we do intend to determine whether or not the crease is related to coronary risk factors. By using a population of subjects relatively free from CAD, we hope to eliminate a potential bias in the study.

CHAPTER III

METHODOLOGY

One-hundred and sixty-eight male and 36 female volunteers, 23 to 60 years of age, participated in the study. Each subject was previously determined to be free of coronary artery disease by his/her physician. The subjects routinely exercised for 20-30 minutes three times per week while maintaining an intensity of 70%-85% of their maximal heart rate.

Data Collection

Each participant was questioned about medication he/she had been receiving, advised of any risk associated with the study, and signed an informed consent form. Next, the subjects were evaluated for an ear crease. Following the ear crease screening, the participants were measured for waist (Wst) and hip (Hip) circumferences and WHR. After the subjects were evaluated for the ear crease and WHR, medical records were used to determine the presence of coronary risk factors. These coronary risk factors included Chol, HDL, Chol/HDL, systolic blood pressure (SBP), diastolic blood pressure (DBP), history of smoking, family history of CAD, age, Wt, Ht, BMI, sum of skinfolds (SSK), and time to 85% of

maximal heart rate on treadmill (T-mill).

Determination of the Ear Crease

The ear crease was examined while subjects were sitting. A crease was considered present if it existed as a clear-cut diagonal wrinkle extending across either ear lobe using criterion described by Lichstein and associates (21).

Determination of Waist-to-Hip Ratio

WHR was determined by the method described by Bray (6). Each subject was measured for Wst and Hip circumferences by a standard, non-flexible tape measure. The Hip circumference was measured in centimeters at the maximal gluteal protrusion (unclothed) while the subject remained standing with heels touching. The Wst circumference was measured in centimeters at the smallest circumference below the rib cage and above the umbilicus (unclothed). The ratio of Wst circumference to Hip circumference was computed by dividing the circumference of the Wst by that of the Hip.

Data

Out of the initial 204 participants, the data for 168 males was compiled for statistical analysis. Thirty-six female subjects were excluded from the study because only one had an ear crease.

Statistical Analyses

Statistical analysis via chi-square was computed to determine the relationship of the ear crease (yes or no), with various coronary risk factors. The coronary risk factors examined included a family history of CAD (yes or no), and smoking history (yes or no). A one-way analysis of variance analysis was used to determine the relationship of the ear crease (yes or no) with Chol, HDL, Chol/HDL, SBP, DBP, age, Wt, Ht, BMI, SSK, Wst, Hip, WHR, and T-mill. Because age may influence the relationship between the crease and coronary risk factors, the mean values were adjusted by an analysis of covariance using age as a covariable.

CHAPTER IV

RESULTS

Frequency of Ear Creases

Out of 168 male subjects evaluated for a crease, 63 (37.5%) had the crease and 105 (62.5%) did not. As seen in most studies, the frequency of the ear crease increased with age. Figure 1 demonstrates an increase in the prevalence of the ear crease in 168 subjects with each age group.

Relationship between the Ear Crease and Coronary Risk Factors

The relationships between the ear crease and coronary risk factors for 168 male subjects are shown in Table 1. A significant difference was observed ($P < 0.05$) in age, WHR, MAP, Chol/HDL, Chol, Wst, and DBP between groups (Table 1). An analysis of covariance demonstrated a significant difference ($P < 0.05$) in MAP, Chol/HDL, Wst, DBP, and WHR between groups even after the mean scores had been adjusted for age (Table 2). Chi-square analysis demonstrated a lack of association ($P > 0.05$) between individuals with a crease and without a crease with regards to a family history of CAD and history of smoking (Table 3).

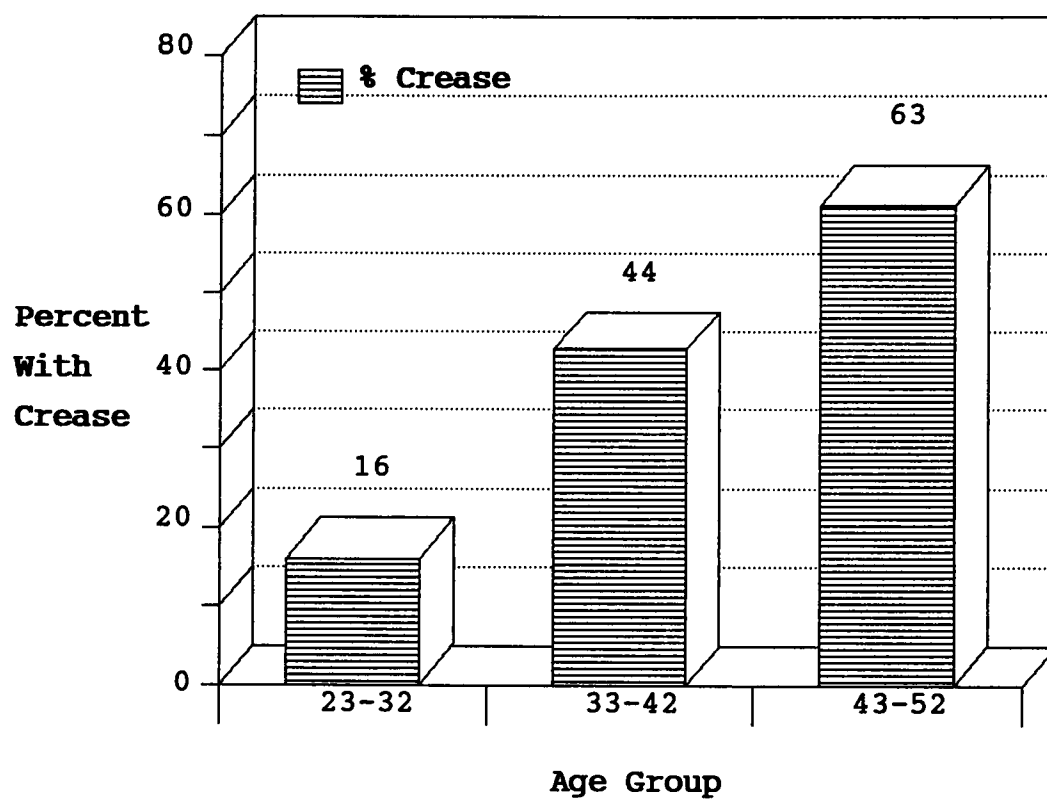


Figure 1. Frequency of Creases with Age Group.

Table 1. Means $\pm$ SD for Coronary Risk Factors in Subjects With and Without a Crease.

Coronary Risk Factors	No Ear Crease	n	Ear Crease	n	
Age (yrs)	32.5 $\pm$ 7.2	105	40.5 $\pm$ 7.2	63	*
BMI (kg/m ²)	26.5 $\pm$ 3.3	96	26.8 $\pm$ 3.3	58	
WHR	.91 $\pm$.05	105	.95 $\pm$.05	63	*
MAP (mmHg)	89.5 $\pm$ 7.9	92	93.8 $\pm$ 7.9	57	*
SSK (mm)	47.1 $\pm$ 16.9	94	51.9 $\pm$ 16.9	59	
T-mill (min)	9.2 $\pm$ 1.1	88	8.9 $\pm$ 1.1	54	
Hip (cm)	102.0 $\pm$ 7.0	105	102.6 $\pm$ 7.0	63	
Wst (cm)	92.7 $\pm$ 9.1	105	97.5 $\pm$ 9.1	63	*
SBP (mmHg)	118.8 $\pm$ 11.2	92	121.2 $\pm$ 11.2	57	
DBP (mmHg)	75.1 $\pm$ 8.1	92	80.3 $\pm$ 8.1	57	*
Ht (in)	70.8 $\pm$ 3.2	96	71.1 $\pm$ 3.2	58	
Wt (lbs)	188.1 $\pm$ 28.2	98	193.7 $\pm$ 28.2	62	
Chol (mg/dl)	185.9 $\pm$ 36.8	68	203.1 $\pm$ 36.8	46	*
Chol/HDL	4.1 $\pm$ 1.2	19	5.0 $\pm$ 1.2	11	*
HDL (mg/dl)	48.5 $\pm$ 10.1	19	42.5 $\pm$ 10.1	11	

* P<0.05

Notes: n = number of subjects

Table 2. Age adjusted Means $\pm$ SD for Coronary Risk Factors in Subjects With and Without a Crease.

Coronary Risk Factors	No Ear Crease	n	Ear Crease	n
WHR	.91 $\pm$.05	105	.94 $\pm$.05	63 *
MAP (mmHg)	89.8 $\pm$ 8.3	92	93.3 $\pm$ 8.5	57 *
Wst (cm)	93.2 $\pm$ 9.4	105	96.7 $\pm$ 9.7	63 *
DBP (mmHg)	75.4 $\pm$ 8.5	92	79.9 $\pm$ 8.7	57 *
Chol (mg/dl)	186.6 $\pm$ 38.9	68	202.0 $\pm$ 39.8	46
Chol/HDL	4.1 $\pm$ 1.2	19	5.1 $\pm$ 1.3	11 *

* P<0.05

Notes: n = number of subjects

Table 3. Prevalence of Creases in Subjects with a History of Smoking and a Family History of CAD.

Groups of Subjects	No Crease	Crease
<u>Smoking History</u>		
Yes	28 (45%)	34 (55%)
No	20 (30%)	46 (70%)
<u>Family History of CAD</u>		
Yes	20 (42%)	28 (58%)
No	43 (37%)	74 (63%)

Notes: CAD = coronary artery disease.

CHAPTER V

DISCUSSION

As observed in most studies (5,9,10,15,16,19,21,25,30,32,37), the ear crease was related to age, and the prevalence of the crease increased with each decade. Research has suggested that the relationship between age and the ear crease could influence other relationships with the crease (5,25). For example, Brady et al. (5) implied that the relationship between CAD and the ear crease was due to the fact that the prevalence of the crease and CAD each increase with age. However, the results from the present study suggest that the crease is related to coronary risk factors such as MAP, DBP, WHR, and Wst independent of age.

Individuals with an ear crease had higher Chol levels than those without a crease ($P < 0.05$). However, after adjusting for age, the mean Chol levels were not significantly different. With the exception of one study, this finding has been consistent throughout the literature (Table B-2, Appendix B).

HDL was slightly higher for subjects with the crease than subjects without the crease. However, no significant difference was noted between subjects with and without the crease with regards to HDL ($P < 0.05$). One should note that

the population of subjects who were evaluated for HDL was quite small. According to Lichstein et al. (23), a small control population could affect relationships with the crease. Therefore, it is possible that HDL levels could have been significantly different between subjects with and without a crease if the population in the present study was larger. However, the crease's lack of association with HDL has been demonstrated in studies which used larger populations (18,19,20).

Since low HDL levels and high Chol levels have been associated with CAD and these variables have not been satisfactorily linked with the crease, one may question the crease's ability to predict coronary risk factors, especially blood lipids. However, previous studies have neglected to investigate or report the relationship of the crease with Chol/HDL. When the Chol/HDL ratio was examined in the present study, the ratio appeared to be significantly higher in the ear crease group when compared to the no ear crease group ($P < 0.05$). Additionally, Chol/HDL was higher in the ear crease group after adjusting for age. One should note that a small population was used to assess Chol/HDL for individuals with and without a crease. Because of the small population we do not conclude that the crease predicts Chol/HDL. However, research suggests that the partitioning of the Chol value into Chol/HDL is important in determining

the risk of CAD (2). We believe that a confirmed association of the crease with higher Chol/HDL ratios would support its use as a predictor of coronary risk factors, namely blood lipids. Hence, it appears necessary that more studies evaluate this relationship before dismissing the crease as a predictor of blood lipid measures.

DBP, and MAP were also demonstrated to be higher in subjects with the crease than in subjects without the crease. These relationships existed even after the data were adjusted for age. Out of 14 other studies, 9 found no significant difference in blood pressure for those with and without the crease. However, the majority of studies which found no significant difference in blood pressure with regards to the crease used a population of individuals who were patients exhibiting symptoms of CAD (See Appendix). Jorde (18) suggested that the utilization of a symptomatic population could introduce biases and confounding effects in an evaluation of the association between blood pressure and ear creases. Using a population of healthy individuals, we found MAP and DBP to be significantly higher for those with the crease. It should be noted that other studies which used an asymptomatic population have examined the relationship between the crease and blood pressure. Out of 4 of these studies, 2 found the crease to be related to blood pressure (Table B-2, Appendix B). Therefore, the true relation of the

crease with blood pressure appears unresolved. However, none of the studies which used an asymptomatic population defined blood pressure in terms of MAP (17,18,27,32). Perhaps future analysis of the relationship between the crease and MAP will aid in determining the creases ability to predict hypertension or elevated blood pressures.

In the present study, SSK, BMI, and WT were not found to be significantly different between individuals with and without the crease. Four out of 5 other studies found that there was no significant difference in adiposity between subjects with and without the crease (Table B-3, Appendix B). Thus, the majority of findings seem to infer that the crease is not related to adiposity.

Until the present study, no other researcher has examined the relationship between the crease and WHR. However, fat distribution has been shown to be a more important coronary risk factor than overweight or total body fatness (6). A high WHR has been associated with a 2-fold or greater risk of heart attack, stroke, hypertension, diabetes mellitus, and gallbladder disease (6). A WHR >1.0 for men and >0.8 for women has been associated with an increased risk of CAD (31). The results from the present study suggest that WHR is significantly higher in subjects with the crease than subjects without the crease. Thus, it appears that the ear crease may not be related to total adiposity, but the

crease appears to be related to WHR which is a more important risk factor than total adiposity.

Additional evidence linking the crease with body fat distribution is the fact that the waist circumference was significantly greater in individuals with the crease than in those without the crease ($P < 0.05$). However, no differences in hip circumferences were found between individuals with and without the crease ($P > 0.05$). Interestingly, an increase in abdominal fat has been found to increase the risk of diabetes and CAD (6). Conversely, gluteal obesity appears to have a much lower risk (6). Since the crease was found to be related to abdominal adiposity and not gluteal adiposity, we suggest that the crease may be more closely related to the distribution of body fat than body fatness, per se. However, we suggest that future investigations include an analysis of the distribution of body fat in their studies. Consistent findings which conclude a positive relationship between the crease and WHR would prove the crease to be valuable in predicting patients at risk of CAD.

No difference was noted in the prevalence of the crease between individuals with and without a history of smoking ($P > 0.05$). These findings are consistent throughout the literature (Table B-2, Appendix B). Apparently, the presence of the crease does not seem to be associated with smoking.

The prevalence of a family history of CAD was not

significantly different between the crease and no crease groups ($P>0.05$). Although a small number of studies have looked at the relationship between the crease and a family history of CAD, their findings were not different from the present study's (Table B-3, Appendix B).

No significant difference was found between individuals with and without the crease regarding time to 85% of the maximal heart rate (MHR). However, Slattery and Jacobs (36) found that at a given submaximal workload individuals with an exercise heart rate of greater than 135 beats per minute had a 40% increase in the risk of CVD mortality compared to subjects with a lower heart rate less than 105 beats per minute. The current study did not quantify submaximal exercise test values in terms of heart rate. However, one would expect the individuals with greater treadmill times to have lower submaximal heart rates at a given work load (1). Since individuals with lower treadmill times would have a greater risk of CVD, it seems logical to imply that that individuals with the crease would have lower treadmill times than individuals without the crease. However, it appears that physical fitness levels as indicated by time to 85% MHR are not significantly different between subjects with and without the crease.

In conclusion, this is the only study to suggest that MAP, DBP, WHR, and Wst are related to the crease in a

population of healthy individuals. However, MAP, WHR, and Wst have not been examined before and future reference to these risk factors for study design may serve to illuminate the value of these risk factor's relationships with the crease. Furthermore, conflicting findings concerning the crease's relationship with blood pressure and/or hypertension remain. We believe that on-going investigations of the association between the crease, blood pressure and other risk factors should take precautions to select an ample sample size and an appropriate population (asymptomatic) for study. A lack of association between the crease and important risk factors such as smoking and a family history of CAD has been confirmed in this study. However, it has been suggested that the crease may not be related directly to any certain risk factor but may reflect a persistent overload of risk factors (17). Thus, an important direction for future studies would be to examine the relationship of the crease with each subject's overall risk profile, in which the individual risk factors are numerically weighted according to their importance. This approach would help to establish whether the ear crease is a simple and valuable predictor of persons at risk of CAD.

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APPENDICES

APPENDIX A

INFORMED CONSENT FORM

You are invited to participate in a research study, the purpose of which is to examine the relationship between the earlobe crease and coronary artery disease risk factors.

You will be examined for an ear crease. The ear crease examination will be conducted by simple observation of the ear lobe for a unilateral or bilateral fold in the skin. Furthermore, you will give the investigator permission to use current risk factor information regarding total cholesterol, resting blood pressure, body fat percentage, weight, age, family history of coronary artery disease, smoking, diabetes mellitus, and waist to hip ratio. If the data for your waist to hip ratio is not available, then it will be measured after the ear crease has been examined. The waist to hip ratio is determined by measuring the circumference of the waist (around the natural waist or at the level of the umbilicus) and the hip (at the level of the maximum protrusion of the buttocks) by a tape measure.

There are no risks involved with the ear crease examination or the waist to hip ratio measurements.

The results from this study will be made available to you upon request. Data from this study may be used in

research reports or presentations; however, your name and other identity will not be disclosed.

You are free to decide whether or not to participate in this study and are free to withdraw from the study at any time, without penalty or prejudice, nor will your withdrawing have any effect on the services normally provided to you.

Before you sign this form please ask questions about any aspects of the study which are unclear to you.

AUTHORIZATION:

I, _____, have read the above and decided to participate in the research project described above. My signature also indicates that I have received a copy of this consent form.

Participant's signature

Date

Witness

Date

Investigator: Douglas W. Minnix

Address:

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APPENDIX B

Table B-1 Summary of Relationships between Creases and CAD from Other Studies.

Study	n		%Subjects/Crease		Criteria for CAD	
	<u>CAD</u>	<u>Controls</u>	<u>CAD</u>	<u>Controls</u>		
Lichstein, et al.(21)	531	305	47%	30%	Clinical Evidence	*
Mehta& Hamby.(25)	159	52	60%	50%	Arterio-graphy	
Sternlieb, et al.(38)	121	10	90%	9%	Angio-graphy	*
Christiansen, et al.(7)	32	66	47%	14%	O	*
Sprague (37)	50	52	82%	38%	Clinical Evidence	*
Doering, et al.(9)	50	38	88%	37%	Clinical Evidence	*
Haft, et al.(16)	243m 63w	66m 73w	54%m 43%w	29%m 14%w	Arterio-graphy	*
Kaukola, et al.(19)	200	86	72%	21%	Angio-graphy	*
Fisher& Sievers(13)	9m 8w	47m 50w	55%m 75%w	75%m 44%w	Clinical Evidence	
Pasternac& Sami(30)	217m 40w	35m 48w	62%m 45%w	17%m 19%w	Arterio-graphy	*
Elliott (10)	148	57	82%	32%	Angio-graphy	*
Gral& Thornburg(15)	284	92	51%	50%	Clinical Evidence	
Lichstein, et al.(22)	0	0	0	0	Autopsy	*
Gibson& Ashikaga(14)	0	0	0	0	Arterio-graphy	
Brady, et al.(5)	222	39	67%	67%	Arterio-graphy	
Cumberland, et al.(8)	139	638	55%	17%	Autopsy	*

Table B-1 (continued)

Study	n		%Subjects/Crease		Criteria for CAD
	<u>CAD</u>	<u>Controls</u>	<u>CAD</u>	<u>Controls</u>	
Ishii, et al.(17)	0		0	0	Autopsy *

* $P < 0.05$

Notes: 0 = no information; m = men; w = women; n = number of subjects; / = with; CAD = coronary artery disease.

Table B-2 Summary of the Relationships between the Crease and Blood Glucose, Smoking, Blood Pressure, Chol, and HDL as demonstrated by Other Studies.

Study	Blood Glucose	Smok- ing	BP	Chol	HDL	Pop- ulation	n
Lichstein, et al.(21)	-	-	-	00	00	Sympto- matic	836
Lichstein, et al.(22)	-	-	-	00	00	Sympto- matic	113
Pasternac& Sami(30)	-	-	-	-	00	Sympto- matic	340
Kaukola, et al.(19)	-	-	-	-	-	Sympto- matic	286
Shoenfeld, et al.(35)	-	-	74%I	-	00	Sympto- matic	842
Kristensen (20)	0	00	91%I	-	-	Sympto- matic	180
Fisher& Sievers(13)	-	-	-	00	00	Sympto- matic	200
Elliott (10)	-	-	-	-	00	Sympto- matic	1,000
Gral& Thornburg(15)	-	-	-	-	00	Sympto- matic	234
Jorde et al.(18)	-	-	-	-	-	Asympto- matic	134
Brady, et al.(5)	00	16%D	13%I	00	00	Sympto- matic	261
Ishii, et al.(17)	00	00	-	15x	00	Asympto- matic	100
Moncada, et al.(27)	-	00	17%I	-	00	Asympto- matic	300
Rhoads& Yano(32)	00	00	0x	00	00	Asympto- matic	00

Notes: %I = significant percent increase in the prevalence of the crease with specified coronary risk factor over the prevalence of the crease without specified coronary risk factor; %x = significant percent increase in mean coronary risk factor value for individuals with the crease over the mean coronary risk factor value for those without the crease; - = no significant difference in either prevalence of crease or mean values; 00 = no information; 0x = significant increase in mean values for individuals with the

Table B-2 Notes (continued)

crease but no information was given regarding the percent increase; %D = significant percent decrease in the prevalence of the crease with specified coronary risk factor over the prevalence of the crease without specified coronary risk factor; Chol = cholesterol; HDL = high density lipoproteins.

Table B-3 Summary of the Relationships between the Crease and Triglycerides, Adiposity, Uric Acid, and Family History of CAD as demonstrated by Other Studies.

Study	Trigly- ceride	Adip- osity	Uric Acid	Family History of CAD	Pop- ulation	n
Lichstein, et al.(21)	00	00	00	00	Sympto- matic	836
Lichstein, et al.(22)	00	00	00	00	Sympto- matic	113
Pasternac& Sami(30)	-	00	00	-	Sympto- matic	340
Kaukola, et al.(19)	-	-	00	00	Sympto- matic	286
Shoenfeld, et al.(35)	-	00	-	00	Sympto- matic	842
Kristensen (20)	-	00	00	00	Sympto- matic	180
Fisher& Sievers(13)	00	-	00	00	Sympto- matic	200
Elliott (10)	00	00	00	-	Sympto- matic	1,000
Gral& Thornburg(15)	00	-	-	00	Sympto- matic	234
Jorde et al.(18)	00	-	00	00	Asympto- matic	134
Brady, et al.(5)	00	00	00	00	Sympto- matic	261
Ishii, et al.(17)	00	00	00	00	Asympto- matic	100
Moncada, et al.(27)	-	00	00	00	Asympto- matic	300
Rhoads& Yano(32)	00	*CO	00	00	Asympto- matic	00

Notes: CO = significant correlation between prevalence of crease with specified coronary risk factor but no values were given; - = no significant difference in either the prevalence of the crease or mean values; 00 = no information; n = number of subjects; CAD = coronary artery disease.

APPENDIX C

Table C-1 Data for Chol, HDL, Age, Wt, Ht, and DBP for Subjects With and Without a Crease.

Subject	Crease	Chol (mg/dl)	HDL (mg/dl)	Age	Wt (lbs)	Ht (in)	DBP (mmHg)
1	NO			43	258.0	77.0	78
2	YES			39	186.0	68.0	88
3	NO	140		26	169.0	65.0	70
4	YES	260		27	224.0	74.5	62
5	YES	160		33	158.0	71.0	70
6	NO			36	159.0	69.0	66
7	NO			31	180.0	69.0	80
8	NO	212	55	29	150.0	67.0	72
9	YES	194	53	54	152.0	70.5	
10	NO			47	175.0	73.0	70
11	YES			44	247.0	79.0	78
12	NO	218		24	212.0	71.0	88
13	YES	194		39	195.0	69.5	70
14	NO			43	185.5	71.0	80
15	NO	150		27	161.5	70.0	78
16	NO			27	156.5	67.0	66
17	NO	172		30	200.0	74.0	70
18	YES	206		38	194.0	67.0	84
19	NO	215		37	226.0	67.0	
20	YES			38	191.5	69.0	80
21	NO	164		37	139.5	65.0	60
22	YES	260		44	214.0	77.0	78
23	NO	160		28	210.0	79.0	90
24	YES			33	183.0	67.0	86
25	NO	166		31	166.0	67.0	82
26	YES	170	33	27	208.0	74.0	80
27	YES	187		46	180.0	75.0	76
28	NO			30	215.0	73.0	80
29	NO	163		32	210.0	76.0	80
30	NO	184		29	140.0	77.5	82
31	NO	165		39	145.5	67.0	70
32	NO			37	193.0	70.0	66
33	YES			41	185.0	69.5	
34	YES			46	202.0	72.0	60
35	YES	252	59	40	206.0	73.0	78

Table C-1 (continued)

Subject	Crease	Chol (mg/dl)	HDL (mg/dl)	Age	Wt (lbs)	Ht (in)	DBP (mmHg)
36	NO	235		47	193.0	70.0	60
37	NO	140		28	205.0	70.0	68
38	NO	140		27	190.0	72.0	78
39	NO			30	171.0	72.0	70
40	YES	187		33	170.0	74.0	80
41	NO	187	35	42	210.0	74.0	82
42	YES	170		35	183.0	72.0	86
43	NO	290		28	165.0	66.0	70
44	NO			44	169.0	67.0	70
45	NO	188		35	221.0	73.0	80
46	NO			36	193.0	72.0	80
47	YES	224		52	150.0	68.0	88
48	NO	237		40	171.0	68.0	68
49	NO	191		37	184.0	69.0	82
50	NO	285		37	198.0	75.0	76
51	YES	175		34	199.0	69.3	82
52	YES	235		44	166.3	70.8	82
53	YES	183		45	153.0	68.0	80
54	YES	216		38	184.5	70.0	90
55	NO	150	56	39	165.5	71.3	68
56	NO	191	47	26	210.5	70.0	74
57	NO			23	217.0	68.0	80
58	YES	149		44	199.0	72.0	74
59	NO	153		29	175.0	70.0	72
60	NO			31	181.0	72.0	68
61	NO			38	206.0	71.0	80
62	NO	190		27	194.0	72.0	68
63	YES	204		43	169.0	67.0	90
64	YES	178		43	180.0	71.0	82
65	NO	192	52	43	165.0	71.0	70
66	YES			38	156.0	67.0	74
67	YES	180		29	195.0	71.0	72
68	YES			31	198.5	76.0	86
69	NO	190		27	218.0	72.0	82
70	YES	196		30	223.0	71.0	70
71	NO	185		32	198.0	70.0	84
72	NO	160	47	28	190.0	72.0	82
73	YES			26	205.0	72.0	68
74	NO			24	190.0	71.5	
75	NO			24	170.0	71.0	76

Table C-1 (continued)

Subject	Crease	Chol (mg/dl)	HDL (mg/dl)	Age	Wt (lbs)	Ht (in)	DBP (mmHg)
76	YES			41	177.5	68.0	74
77	NO			35	217.5	76.5	68
78	NO	170		24	168.5	72.0	66
79	NO	165		26	155.0	68.0	68
80	NO	180		32	194.5	71.0	72
81	NO	174	52	48	163.3	67.0	68
82	NO			33	195.0	74.0	78
83	NO			24	184.0	68.5	70
84	YES			43	233.5	75.0	82
85	NO	220	31	31	199.5	73.0	74
86	NO	177	42	60	164.0	65.0	88
87	NO	185		38	192.0	72.0	84
88	NO	252		28	247.8	76.0	74
89	NO			33	284.5	76.0	88
90	YES	200		33	187.8	69.0	70
91	NO	184		30	236.0	72.0	60
92	NO	145	44	28	183.5	72.0	70
93	YES	132		27	180.5	68.0	78
94	NO	145	72	30	143.0	68.0	70
95	YES	166	32	58	206.3	73.0	
96	NO	184	67	32	242.0	72.0	78
97	YES	225		45	195.5	73.5	78
98	NO	142		33	187.0	67.5	80
99	YES	237	37	42	228.0	73.0	86
100	YES	209		43	210.0	69.0	80
101	YES			57	181.0	72.0	82
102	NO	211	51	36	199.0	74.0	82
103	YES	232		40	308.0	73.0	88
104	YES	256	53	42	210.0	72.0	86
105	NO	190		37	193.0	70.5	72
106	NO	163		50	214.5	77.0	84
107	NO			25	210.0	73.0	70
108	NO			33	154.0	73.0	86
109	NO			24	126.5	68.0	76
110	YES	215		42	216.0		96
111	YES	232		39	266.5		96
112	YES			39	216.5	69.0	88
113	NO			24	183.0	72.0	
114	YES	249		43	191.8	71.0	84
115	NO	170		24	176.0	65.0	84

Table C-1 (continued)

Subject	Crease	Chol (mg/dl)	HDL (mg/dl)	Age	Wt (lbs)	Ht (in)	DBP (mmHg)
116	NO	275	44	29	192.0	67.0	72
117	NO			26	188.0	70.5	82
118	NO	180		38	159.0		58
119	YES	180		32	183.0	66.0	78
120	YES	240		35	200.0	72.0	84
121	YES	164	30	55	179.0		80
122	NO	200		43	254.5	73.0	76
123	NO	250		34	163.0	65.0	70
124	NO	146		24	184.0	72.3	60
125	NO	301	47	29	184.7	72.0	78
126	YES			29			
127	NO	197		29	176.5	73.5	78
128	YES	238		43	159.0	72.0	78
129	YES	270	48	35	202.5	77.0	78
130	YES	153		41	153.7	68.5	88
131	NO	170		29	165.0		80
132	YES			38	192.0	70.0	78
133	NO	185	57	37			
134	NO	182		32	145.0	67.0	78
135	NO			39			
136	NO			40			
137	NO			38	175.0	70.0	100
138	NO	224		27	221.0	72.5	70
139	NO			27	171.0	72.0	70
140	YES	170		42	218.0	74.5	112
141	YES	170		50	176.0		80
142	NO	120		30	159.8	71.0	70
143	NO	230		31			
144	NO	200		45			
145	NO			41	205.5	68.0	72
146	NO	130		27	163.0	58.5	80
147	NO	129		30			
148	NO	181		27	221.5	75.0	82
149	NO			32	159.4	71.0	72
150	YES	224	43	29	183.0	66.0	78
151	NO	161		30			
152	YES	200		39	194.0	73.5	72
153	YES	194	35	60	193.0	74.0	70
154	YES	177	45	46	215.0	68.0	
155	NO			29	165.0	71.0	68

Table C-1 (continued)

Subject	Crease	Chol (mg/dl)	HDL (mg/dl)	Age	Wt (lbs)	Ht (in)	DBP (mmHg)
156	NO			32	230.0	74.0	82
157	NO	187	44	28	190.0	68.0	76
158	YES			49	142.0	71.0	64
159	NO	210		26	185.0	71.0	80
160	NO			36	207.0	71.0	80
161	NO			38	217.0	75.0	
162	NO			29	188.0	72.0	
163	NO	135	38	34	183.0	72.0	84
164	YES			5	164.0	72.0	80
165	YES	205		40	168.0	66.0	
166	NO	180	41	36	179.0	66.0	
167	YES	223		47	224.0	73.0	94
168	NO			36	185.0	68.5	68

Notes: Chol = cholesterol; HDL = high density lipoproteins; Wt = weight; Ht = height; DBP = diastolic blood pressure.

Table C-2 Data for Wst, Hip, T-MILL, SSK, MAP, AND Chol/HDL for Subjects With and Without a Crease.

Subject	Crease	Wst (cm)	Hip (cm)	T-MILL (min)	SSK (mm)	MAP (mmHg)	Chol/HDL (mg/dl)
1	NO	103.5	112.5	8.2	66.5	91.9	
2	YES	95.5	105.0	8.5		101.2	
3	NO	83.0	93.0	11.0	41.0	93.1	
4	YES	103.3	109.0	9.2	60.0	79.8	
5	YES	85.3	95.8	8.4	33.0	83.2	
6	NO	80.3	98.0	9.3	31.5	99.0	
7	NO	94.3	97.8	9.0	55.5	93.9	
8	NO	77.0	90.8	10.0	21.3	87.8	3.9
9	YES	88.5	91.3		37.5		3.7
10	NO	85.5	97.5	10.2	46.0	80.6	
11	YES	106.5	110.3	6.5	64.0	95.2	
12	NO	102.3	107.3	8.5	58.5	102.5	
13	YES	100.0	108.5	9.4	79.0	83.9	
14	NO	90.3	96.0	9.1	43.0	89.9	
15	NO	77.8	92.8	10.5	31.2	91.9	
16	NO	82.5	93.5	8.1	38.0	80.5	
17	NO	97.0	105.3	9.3	50.5	85.8	
18	YES	100.3	100.0	7.4	47.2	95.9	
19	NO	115.5	121.0		85.0		
20	YES	103.0	101.0	9.2		93.2	
21	NO	77.0	87.5	10.1	21.5	73.2	
22	YES	112.0	107.0	10.0	79.5	97.8	
23	NO	98.0	106.5	9.6	58.0	100.6	
24	YES	100.0	100.5	10.0	55.0	93.9	
25	NO	89.0	97.3	12.0	37.5	97.2	
26	YES	94.0	103.3	9.4	47.0	93.2	5.2
27	YES	94.5	103.3	9.5	30.5	88.5	
28	NO	100.0	108.5	7.0	75.0	97.8	
29	NO	95.0	98.0		49.0	96.5	
30	NO	84.8	90.0	8.0	44.0	94.5	
31	NO	83.5	94.5	9.1	42.5	83.2	
32	NO	96.0	101.0	9.3	57.0	81.2	
33	YES	98.5	105.0		57.5		
34	YES	101.5	98.0	10.4	51.0	76.5	
35	YES	98.5	110.0	8.4	80.0	86.6	4.3
36	NO	103.5	105.0	9.2	61.0	73.9	
37	NO	96.3	109.5	9.3	45.5	88.5	
38	NO	92.0	101.5	10.0	36.5	90.9	
39	NO	86.3	97.5	9.3	31.5	83.2	

Table C-2 (continued)

Subject	Crease	Wst (cm)	Hip (cm)	T-MILL (min)	SSK (mm)	MAP (mmHg)	Chol/HDL (mg/dl)
40	YES	87.0	97.0	9.0	47.5	92.5	5.3
41	NO	94.5	102.3	6.4	44.0	94.5	
42	YES	89.0	97.8	10.1	31.5	99.2	
43	NO	90.5	97.8	9.0	65.0	82.5	
44	NO	93.0	96.8	9.6	56.0	84.5	
45	NO	108.3	111.5	8.6	88.0	96.5	2.7
46	NO	103.5	104.0	10.0	85.0	90.6	
47	YES	89.0	93.0	8.0	38.5	101.9	
48	NO	91.5	100.0	10.0	60.5	77.9	
49	NO	97.0	105.0	7.2	68.0	91.2	
50	NO	91.0	106.0	9.0	38.4	83.9	4.1
51	YES	99.0	107.0	9.0	42.0	91.2	
52	YES	94.0	97.0	7.4	46.6	96.5	
53	YES	84.5	95.5	9.3	40.0	90.6	
54	YES	95.5	98.3	9.1	33.5	103.2	
55	NO	87.5	93.3	8.1	29.0	81.9	3.7
56	NO	97.3	107.5	9.4	55.5	88.5	
57	NO	96.0	108.0	9.0	57.5	93.9	
58	YES	98.5	105.0	9.2	47.0	89.2	
59	NO	85.8	96.8	10.2	33.0	87.2	
60	NO	89.5	97.5	9.2	52.0	81.9	3.4
61	NO	104.5	106.0	9.1	81.5	93.2	
62	NO	101.0	101.8	9.0	72.0	85.8	
63	YES	95.8	96.0	9.0	43.5	102.5	
64	YES	95.0	101.0	8.0	35.8	96.5	
65	NO	91.5	101.8	10.5	47.0	89.8	3.7
66	YES	88.0	92.5	8.4	31.0	89.2	
67	YES	97.0	102.0	9.2	59.5	88.5	
68	YES	83.0	101.0	11.1	36.0	97.2	
69	NO	100.0	102.5	9.3	46.5	97.8	
70	YES	110.0	113.0	9.0	89.0	83.2	3.4
71	NO	104.0	110.5	7.0	69.0	102.5	
72	NO	93.0	104.5		41.0	91.2	
73	YES	96.3	103.8	9.5	57.0	78.6	
74	NO	91.3	104.3		32.0		
75	NO	83.8	94.5	10.0	27.2	87.2	3.4
76	YES	100.5	99.5	8.1	63.6	89.2	
77	NO	99.0	107.5	9.3	54.4	82.5	
78	NO	90.3	98.0	9.4	52.4	81.2	
79	NO	81.5	92.8	10.0	32.7	82.5	

Table C-2 (continued)

Subject	Crease	Wst (cm)	Hip (cm)	T-MILL (min)	SSK (mm)	MAP (mmHg)	Chol/HDL (mg/dl)
80	NO	86.5	105.3	9.5	30.4	88.5	
81	NO	89.0	99.5	10.3	51.0	87.8	3.3
82	NO	91.0	101.5	9.3	53.5	91.9	
83	NO	92.5	103.5	9.2	40.0	83.2	
84	YES	103.5	110.5	7.6	74.6	94.5	
85	NO	99.5	104.8	8.0	49.8	87.9	7.1
86	NO	89.5	98.0	5.3	18.0	104.5	4.2
87	NO	92.5	102.8	10.2	15.8	95.9	
88	NO	102.5	112.0	9.4	20.4	89.8	
89	NO	122.0	120.5	9.0	31.6	101.9	
90	YES	97.5	97.0	9.4	59.0	83.2	
91	NO	104.8	110.5	12.0	23.0	73.2	
92	NO	84.5	97.8	11.0	27.0	88.5	3.3
93	YES	85.5	99.3	8.1	31.6	97.1	
94	NO	79.5	92.8	9.5	28.3	81.2	2.0
95	YES	97.5	103.5		39.2		5.2
96	NO	90.0	102.5	9.0	82.0	91.9	2.7
97	YES	88.5	106.5	9.0	31.8	87.9	
98	NO	98.3	108.8	7.2	68.5	93.2	
99	YES	107.5	113.0	9.4	62.8	96.6	6.4
100	YES	98.5	112.0	9.3	57.8	92.5	
101	YES	96.5	101.0	7.4	50.2	95.2	
102	NO	88.5	103.5	10.0	26.8	92.6	4.1
103	YES	134.5	132.5	6.4	99.0	102.5	
104	YES	97.8	103.0	9.3	57.0	100.5	4.8
105	NO	86.0	106.5	9.0	66.7	81.2	
106	NO	94.5	106.5	8.0	47.8	99.2	
107	NO	87.8	107.0	9.2	30.5	86.5	
108	NO	83.5	95.8	7.0	27.2	93.9	
109	NO	74.0	90.5	9.3	24.8	89.9	
110	YES	105.3	108.5	9.0	75.4	113.8	
111	YES	115.0	118.0	9.0	56.0	117.2	
112	YES	105.3	101.5	9.4	57.0	97.9	
113	NO	86.5	97.5				
114	YES	102.5	101.8	9.0	54.8	100.5	
115	NO	90.0	98.0	8.5	46.4	95.9	
116	NO	97.0	107.5	10.2	56.5	87.8	6.3
117	NO	78.3	95.5	7.1	29.8	95.2	
118	NO	84.3	100.0			79.1	
119	YES	103.0	100.0	9.0	83.6	87.9	

Table C-2 (continued)

Subject	Crease	Wst (cm)	Hip (cm)	T-MILL (min)	SSK (mm)	MAP (mmHg)	Chol/HDL (mg/dl)
120	YES	93.0	103.5	8.4	46.4	97.2	
121	YES	93.5	97.5	8.3	39.5	97.8	5.5
122	NO	110.0	117.0	7.0	80.0	91.8	
123	NO	90.0	98.5	9.3	55.8	83.2	
124	NO	92.3	98.0			69.9	
125	NO	92.0	101.5	9.2	43.9	91.9	6.4
126	YES	90.0	103.5				
127	NO	93.5	101.5	9.1	44.4	91.9	
128	YES	90.3	94.5	7.1	43.5	85.3	
129	YES	94.5	104.0	10.2	38.4	89.2	5.6
130	YES	87.3	95.3	9.2	38.0	101.9	
131	NO	86.3	96.5	8.0	48.3	89.9	
132	YES	98.5	106.5	8.5	61.6	93.2	
133	NO	91.0	94.5				3.2
134	NO	80.0	94.0	9.3	31.0	90.5	
135	NO	115.5	112.0				
136	NO	87.0	99.5				
137	NO	96.5	113.0	7.3	68.0	111.2	
138	NO	101.3	109.0	.1	57.0	89.8	
139	NO	83.5	97.0	10.1	26.5	83.2	
140	YES	101.8	104.8	10.2	38.4	121.2	
141	YES	100.0	93.8			96.5	
142	NO	79.5	96.8	10.4	29.8	87.2	
143	NO	102.5	113.0				
144	NO	112.5	112.5				
145	NO	107.8	105.5	9.2	74.2	91.1	
146	NO	84.0	91.0	11.0	48.4	91.9	
147	NO	90.0	99.0				
148	NO	94.5	107.0	9.4	30.6	97.8	
149	NO	81.0	92.0	10.1	38.2	85.2	
150	YES	97.5	101.5	9.3	53.6	91.2	5.2
151	NO	89.0	100.0				
152	YES	99.0	102.5		43.8	84.5	
153	YES	100.0	101.5		60.5	82.5	5.5
154	YES	114.3	112.0		64.5		3.9
155	NO	89.8	92.8	10.0	32.5	82.5	
156	NO	106.8	121.3	9.3	64.8	97.2	
157	NO	98.3	104.8	9.4	64.5	89.9	4.3
158	YES	80.5	89.3	9.2	29.0	81.8	
159	NO	89.5	105.0	9.3	32.0	95.2	

Table C-2 (continued)

Subject	Crease	Wst (cm)	Hip (cm)	T-MILL (min)	SSK (mm)	MAP (mmHg)	Chol/HDL (mg/dl)
160	NO	102.5	105.5	9.2		94.5	
161	NO	97.3	108.5		59.5		
162	NO	85.3	95.3		39.5		
163	NO	87.5	98.5	9.3	36.2	99.2	3.6
164	YES	85.5	95.8	7.5	33.5	92.5	
165	YES	88.5	96.5		46.5		
166	NO	91.3	99.8		63.0		4.4
167	YES	109.0	106.5	9.0	69.5	105.0	
168	NO	96.8	104.8	9.2	52.5	85.2	

Notes: Wst = waist circumferences; T-Mill = time on treadmill until 85% of maximal heart rate; SSK = sum of skinfolds; MAP = mean arterial blood pressure; Chol/HDL = cholesterol to high density lipoprotein ratio.

Table C-3 Data for SBP, Smoking History, Family History of CAD, WHR, and BMI for Subjects With and Without a Crease.

Subject	Crease	SBP (mmHg)	Smoking History	Family History of CAD	WHR	BMI (kg/m ²)
1	NO	120	YES	NO	.92	30.7
2	YES	128	YES	NO	.91	28.3
3	NO	140	NO	YES	.89	28.2
4	YES	116	YES	NO	.95	28.4
5	YES	110	NO	NO	.89	22.1
6	NO	166	YES	NO	.82	23.5
7	NO	122	YES	YES	.96	26.6
8	NO	120	YES	NO	.85	23.5
9	YES		YES	NO	.97	21.5
10	NO	102	YES	NO	.88	23.1
11	YES	130	YES	NO	.97	27.9
12	NO	132	NO	YES	.95	29.6
13	YES	112	NO	NO	.92	28.4
14	NO	110	NO	NO	.94	25.9
15	NO	120	YES	NO	.84	23.2
16	NO	110	NO	NO	.88	24.5
17	NO	118	YES	YES	.92	25.7
18	YES	120	YES	NO	1.0	30.4
19	NO		NO	YES	.96	35.5
20	YES	120	NO	NO	1.0	28.3
21	NO	100	YES	NO	.88	23.3
22	YES	138	YES	YES	1.0	25.4
23	NO	122	YES	NO	.92	23.7
24	YES	110	YES	NO	1.0	28.7
25	NO	128	NO	YES	.92	26.1
26	YES	120	NO	YES	.91	26.8
27	YES	114	YES	NO	.92	22.5
28	NO	134	NO	NO	.92	28.4
29	NO	130	YES	NO	.97	25.6
30	NO	120	YES	YES	.94	16.4
31	NO	110	YES	NO	.88	22.8
32	NO	112	NO	YES	.95	27.8
33	YES		YES	NO	.94	27.0
34	YES	110	YES	NO	1.0	27.5
35	YES	104	YES	NO	.90	27.2
36	NO	102	YES	NO	.99	27.8
37	NO	130	NO	YES	.88	29.5

Table C-3 (continued)

Subject	Crease	SBP (mmHg)	Smoking History	Family History of CAD	WHR	BMI (kg/m ²)
38	NO	117	NO	NO	.91	25.8
39	NO	110	YES	YES	.89	23.0
40	YES	118	YES	YES	.90	21.9
41	NO	120	NO	NO	.92	27.0
42	YES	126	YES	NO	.91	24.9
43	NO	108	NO	NO	.93	26.7
44	NO	114	YES	YES	.96	26.5
45	NO	130	NO	NO	.97	29.2
46	NO	112	YES	NO	1.0	26.2
47	YES	130	NO	YES	.96	22.9
48	NO	98	NO	NO	.92	26.1
49	NO	110	YES	NO	.92	27.2
50	NO	100	YES	NO	.86	24.8
51	YES	110	NO	NO	.93	29.2
52	YES	126	NO	NO	.97	23.4
53	YES	112	YES	NO	.89	23.3
54	YES	130	YES	NO	.97	26.5
55	NO	110	YES	YES	.92	23.0
56	NO	118	NO		.91	30.2
57	NO	122	NO	NO	.89	33.1
58	YES	120	YES	NO	.94	27.0
59	NO	118	NO	NO	.89	25.2
60	NO	110	YES	NO	.92	24.6
61	NO	120	NO	NO	.99	28.8
62	NO	122	NO	YES	.99	26.4
63	YES	128	YES	YES	1.0	26.5
64	YES	126	YES	YES	.94	25.2
65	NO	130	NO	NO	.90	23.1
66	YES	120	YES	YES	.95	24.5
67	YES	122	NO	YES	.95	27.3
68	YES	120	NO	NO	.82	24.2
69	NO	130	NO	NO	.98	29.6
70	YES	110	NO	NO	.97	31.2
71	NO	140	YES	NO	.94	28.5
72	NO	110	NO	NO	.89	25.8
73	YES	100	NO	YES	.93	27.9
74	NO		NO	YES	.88	26.2
75	NO	110	NO	NO	.89	23.8
76	YES	120	NO	NO	1.0	27.0

Table C-3 (continued)

Subject	Crease	SBP (mmHg)	Smoking History	Family History of CAD	WHR	BMI (kg/m ²)
77	NO	112	NO	NO	.92	26.2
78	NO	112	NO	NO	.92	22.9
79	NO	112	NO	NO	.88	23.6
80	NO	122	NO	YES	.82	27.2
81	NO	128		NO	.89	25.6
82	NO	120		YES	.90	25.1
83	NO	110		NO	.89	27.6
84	YES	120		NO	.94	29.2
85	NO	116		NO		26.4
86	NO	138		NO	.91	27.3
87	NO	120		NO	.90	26.1
88	NO	122		NO	.92	30.2
89	NO	130		NO	1.0	34.7
90	YES	110	YES	NO	1.0	27.8
91	NO	100		NO	.95	32.1
92	NO	126	NO	YES	.86	24.9
93	YES	136	NO	NO	.86	27.5
94	NO	104	YES	NO	.86	21.8
95	YES			YES	.94	27.3
96	NO	120		NO	.88	32.9
97	YES	108		YES	.83	25.5
98	NO	120		NO	.90	28.9
99	YES	118		YES	.95	30.1
100	YES	118		NO	.88	31.1
101	YES	122		NO	.96	24.6
102	NO	114		YES	.86	25.6
103	YES	132		YES	1.0	40.7
104	YES	130		NO	.95	28.5
105	NO	100		YES	.81	27.4
106	NO	130		NO	.89	25.5
107	NO	120		NO	.82	27.8
108	NO	110		NO	.87	20.4
109	NO	118		YES	.82	19.3
110	YES	150		YES	.97	
111	YES	160		YES	.98	
112	YES	118		NO	1.0	32.0
113	NO			NO	.89	24.9
114	YES	134		YES	1.0	26.8
115	NO	120		NO	.92	29.3

Table C-3 (continued)

Subject	Crease	SBP (mmHg)	Smoking History	Family History of CAD	WHR	BMI (kg/m ²)
116	NO	120		NO	.90	30.1
117	NO	122		NO	.82	26.7
118	NO	122		NO	.84	
119	YES	108		NO	1.0	29.6
120	YES	124		NO	.90	27.2
121	YES	134		NO	.96	
122	NO	124		NO	.94	33.6
123	NO	110		NO	.91	27.2
124	NO	90	NO	NO	.94	24.8
125	NO	120	NO	YES	.91	25.1
126	YES		YES	YES	.87	
127	NO	120	NO	NO	.92	23.0
128	YES	100	YES	YES	.96	21.6
129	YES	112	NO	NO	.91	24.1
130	YES	130	NO	YES	.92	23.1
131	NO	110	NO	NO	.89	
132	YES	124	YES	NO	.93	27.6
133	NO		YES	NO	.96	
134	NO	116	YES	NO	.85	22.8
135	NO		YES	NO	1.0	
136	NO		NO	NO	.87	
137	NO	134	YES	NO	.85	25.1
138	NO	130	YES	YES	.93	29.6
139	NO	110	NO	YES	.86	23.2
140	YES	140	NO	NO	.97	27.7
141	YES	130	NO	NO	1.1	
142	NO	122	NO	NO	.82	22.3
143	NO		YES	YES	.91	
144	NO		NO	NO	1.0	
145	NO	130	NO	YES	1.0	31.3
146	NO	116	NO	NO	.92	33.6
147	NO		NO	NO	.91	
148	NO	130	YES	YES	.88	27.7
149	NO	112	NO	NO	.88	22.3
150	YES	118	NO	NO	.96	29.6
151	NO		NO	NO	.89	
152	YES	110	YES	NO	.97	25.3
153	YES	108	NO	NO	.99	24.8
154	YES		NO	NO	1.0	32.8

Table C-3 (continued)

Subject	Crease	SBP (mmHg)	Smoking History	Family History of CAD	WHR	BMI (kg/m ²)
155	NO	112	NO	NO	.97	23.1
156	NO	12	NO	NO	.88	29.6
157	NO	118	YES	YES	.94	29.0
158	YES	118	YES	NO	.90	19.8
159	NO	126	NO	NO	.85	25.9
160	NO	124	YES		.97	28.9
161	NO		NO	NO	.90	27.2
162	NO		NO	YES	.90	25.6
163	NO	130	YES	NO	.89	24.9
164	YES	118	YES	NO	.89	22.3
165	YES		YES	YES	.92	27.2
166	NO		YES	NO	.92	29.0
167	YES	128	YES	NO	1.0	29.6
168	NO	120	YES	NO	.92	27.8

Notes: SBP = systolic blood pressure; CAD = coronary artery disease; WHR = waist to hip ratio; BMI = body mass index.

VITA

Douglas Wayne Minnix was born on August 7, 1967 in Bluefield, West Virginia. He graduated from Bluefield High School in May of 1985 and was enrolled at Bluefield College in Bluefield, Virginia the following August. Having been introduced to Karate, Japanese Culture, and Oriental Medicine at age 11, he traveled to Japan in 1988 to broaden his education in these areas. After 6 months of study abroad, he returned to Bluefield College where he completed his Bachelor of Science degree in Physical Education in May of 1990.

In August of 1990, he entered the University of Tennessee at Knoxville and began work on his Master of Science degree in Exercise Physiology.

After his graduation in August of 1992, he plans to work in the field of Exercise Science and Preventive Medicine. Furthermore, he plans on continuing his education in the field of Exercise Science, Preventive Medicine, and Karate with the intention of teaching these disciplines at a Christian College.