BLOOD PRESSURE ANALYSIS DURING TREADMILL STRESS TESTING

by

Michael D. Reis

Bioengineering

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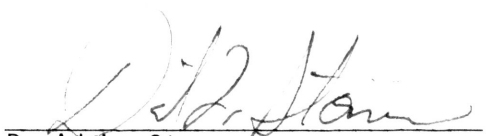
Approved by:



David L. Stoner

April 1980

ABSTRACT

A problem has been identified in that the treadmill stress test is far too inaccurate a test to be used in the diagnosis of coronary heart disease without some method to increase its reliability. An extensive study has been performed concerning exercise work physiology and the response of the body to stress. A theory has been postulated as to the blood pressure response to an increase in heart rate in diseased hearts as opposed to healthy hearts. A method has been proposed to statistically discriminate between patients who are positive and negative for coronary heart disease based solely on blood pressure and heart rate. This method utilizes multivariate discriminant analysis to form discriminant functions for the classification of patients with unknown memberships. Results show that a difference exists in the relationship between systolic blood pressure and heart rate at increased levels of stress for normal patients, compared to those with coronary heart disease.

ACKNOWLEDGMENTS

I would like to express my appreciation to the many people who in one way or another made this research possible. Special thanks go to Dr. David Stoner for his constant support and willingness to give of his time. Also, thanks go to Phil Spector, the graduate student who helped make statistics understandable, and to Dr. Elvin Smith M.D., Assistant Dean of the TAMU medical school, for his sound guidance in my research.

In conclusion, many thanks to all those who helped make the 1979-1980 Fellows program a success.

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INTRODUCTION

Background

In 1977, a cardiologist in Tyler, Texas, Dr. T. H. Alexander, asked the Texas A&M University Bioengineering Department to design and build a system which would enable him to monitor his patient's blood pressure throughout the various stages of treadmill stress testing. Dr. Alexander felt that by closely monitoring the blood pressure of patients during the running of a treadmill test, he would have a better indication as to when a patient might be on the verge of dyspnea and subsequent fainting.

Prior to that time, it had been impossible to take blood pressure readings during the vigorous stress testing due to noise artifact generated by the motion of the subject. The only readings which were obtainable prior to implementation of this device were the blood pressures and heart rates before and after the test; the blood pressure response during the test was thus unobtainable.

After several months of design and experimentation, Dr. David L. Stoner, a biomedical engineering professor at Texas A&M, and an undergraduate assistant designed an appropriate system which underwent testing and was eventually implemented in the cardiology lab at Mother Francis Hospital in Tyler. At that time its staff began to record the blood pressure of all patients throughout the entire stress test.

This system incorporated a continuous wave Doppler crystal and a stereo tuner connected in series so that the flow of blood through the brachial artery could be amplified, filtered, and broadcast throughout

the room via 8 ohm speakers (see Figure 1)(Stoner, 1979).

The Doppler crystal operates on the principle of Doppler, which can be stated as follows: when a target recedes from a fixed source that transmits sound, the frequency of received sound is lowered by an amount such that the fractional change in frequency equals the fractional change in velocity (Webster, 1978), or

$$F_d/F_o = V/C$$

where:

F_d = Doppler frequency shift

F_o = source frequency

V = target velocity

C = velocity of sound

In this application, ultrasound is beemed through the walls of the brachial artery, back scattered by the moving red blood cells, and received by a piezoelectric crystal (Webster, 1978)(see Figure 2). By using a standard sphygmomanometer to measure blood, the Doppler crystal can be used over the brachial artery to detect the Korotkoff sounds which characterize systole and diastole. The results obtained by using this method are experimentally similar to results obtained by the traditional clinical use of a stethoscope over the artery, but much clearer resolution is possible, and noise artifact is not a problem in the use of the Doppler.

Exercise Stress Testing

A common method of exercise stress testing is the treadmill stress test. This and all stress tests function to place the body under

Schematic of Blood Pressure Measurement Device

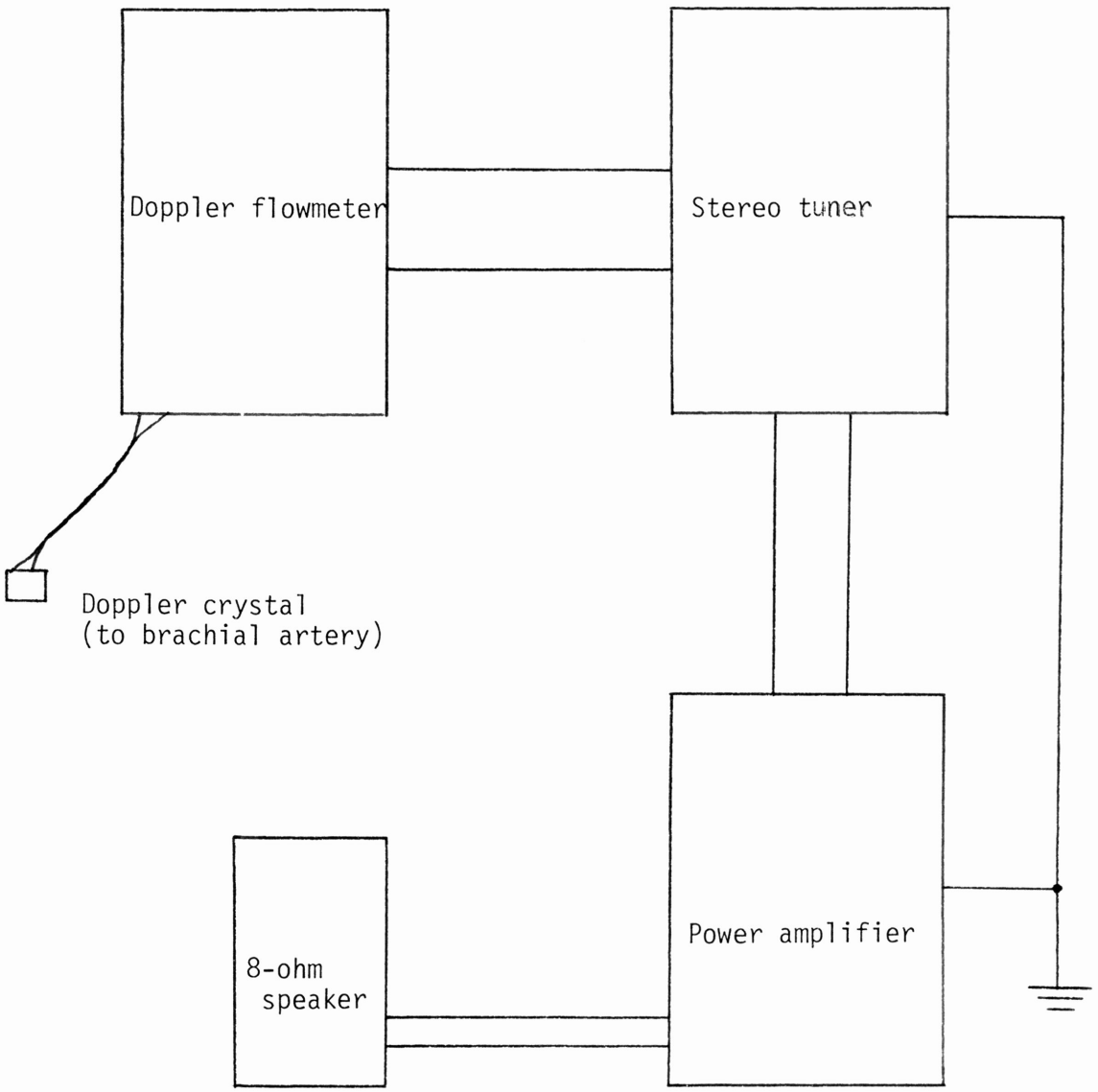

Figure 1

Doppler Ultrasonic Blood Flowmeter

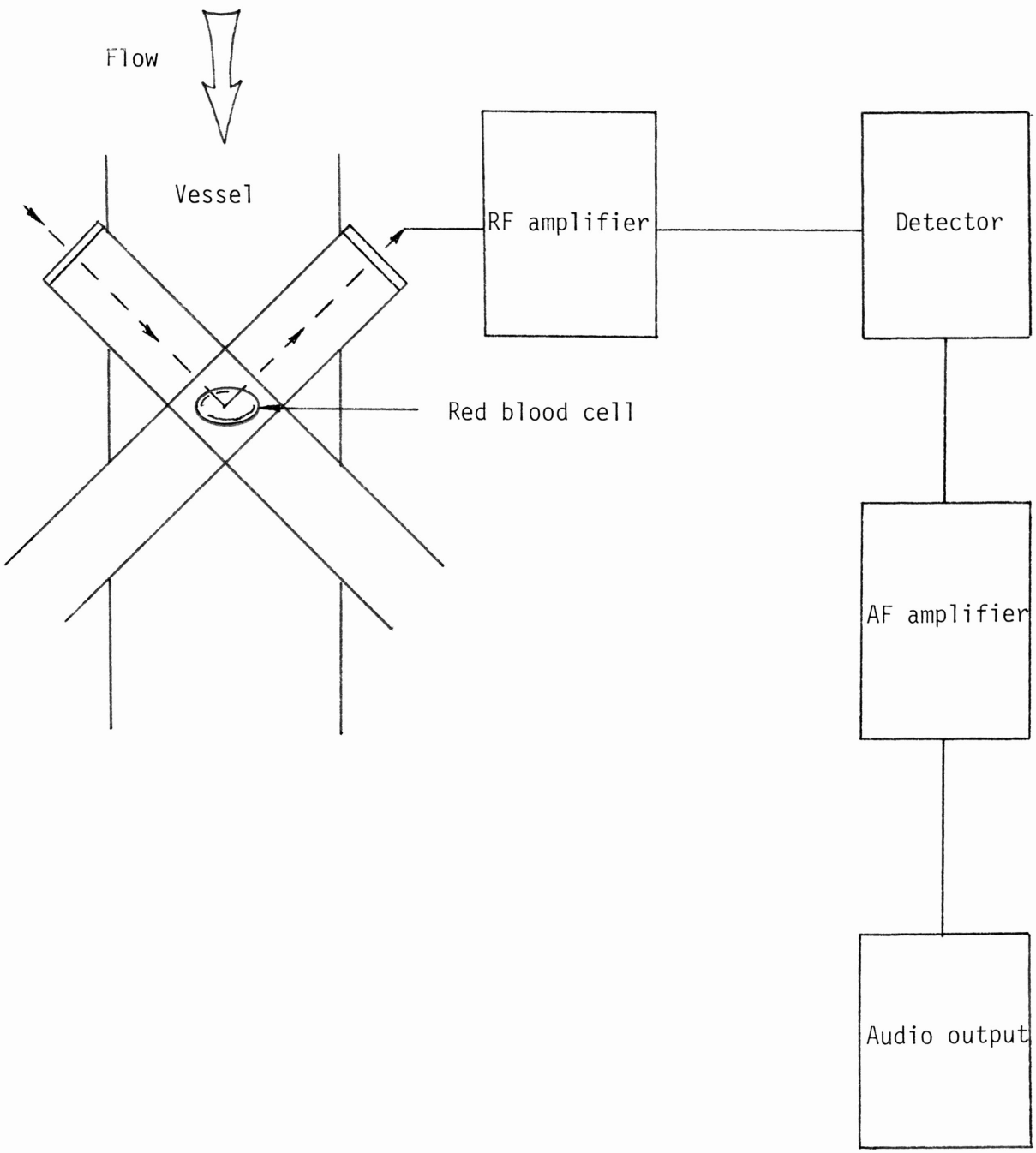

Figure 2

stress, so that the response of body to stress can be observed and analyzed. As the body is stressed, the metabolism of the body is greatly increased and the metabolic functions of the heart are also increased. This is evidenced by an increase in the O_2 consumption of the myocardium, the heart rate, and the cardiac output. The physician can therefore observe the electrical functioning of the heart under stress via an electrocardiogram.

Dr. Alexander's patients ran a treadmill test under a modified Bruce protocol. This differs from the standard Bruce protocol in that the times between increases in treadmill speed and gradient are shortened. This serves to make the test more intense, raising the patient's heart rate to its maximum value much more quickly. This is important because many patients cannot finish the test due to exhaustion, the modified protocol enabled more patients to finish the test.

The treadmill stress test has undergone quite a bit of criticism in recent years regarding its effectiveness in diagnosing coronary heart disease (CHD). Usually, the test is used as a screening device with patients who are diagnosed as positive being referred on for an arteriogram. Interestingly enough, the complaint with treadmill stress testing was not that it resulted in too many false negatives (patients with CHD who are told they are healthy). The problem lies in the fact that the test resulted in too many false positives. This caused healthy people to be sent to the hospital to have arteriograms run and thus undergo the risk, pain, and expense of an unnecessary surgical procedure.

Coronary Arteriography

An arteriogram (or angiogram) is composed of the following steps: a large bolus of radiopaque dye is injected rapidly into a vessel leading directly to the coronary arteries, and the hemodynamics of the vessels are recorded on X-ray film. The dye is injected via a catheter inserted through a vessel in the arm and threaded into the heart. Heart catheterization and arteriography can lead to ectopic beats and/or ventricular fibrillation due to stimulation from the catheter or the contrast material. For this reason, a ventricular defibrillator must be readily available during heart catheterization.

It is clear that the treadmill stress test alone is at best a crude approximation to a patient's status with respect to CHD. If the test could be made more reliable, thus assisting the doctor in the proper diagnosis of CHD, many unnecessary arteriograms could be avoided. Thus in this research, I have attempted to use the blood pressure readings taken during the stress test as a method of increased reliability in the diagnosis of CHD.

In order to obtain meaningful results, the solution to this problem had to be approached from two directions, both of which hopefully would lead to the same conclusion.

First of all, a theory had to be formulated which predicted the effect of stress on a healthy heart, and on a diseased heart. It was necessary to determine the blood pressure response to an increased heart rate in both healthy and diseased patients. The solution to this problem was not as simple as one might expect. Very little research has been done in the field of blood pressure response to stress in relation

to CHD. Consequently, extensive research had to be conducted, beginning with basic discussions of physiology of the heart and the pathologic conditions of CHD, and extending to recently published complex discussions of exercise physiology.

Secondly, if the groups of positive and negative patients were postulated to be sufficiently dissimilar to allow discrimination, a suitable method had to be selected in order to statistically analyze the blood pressure data and answer the following questions: (1) Which variables were significant in discriminating between patients with and without CHD? (2) How sure can one be of results obtained using various statistical methods?

The pursuit of these answers resulted in research on various statistical methods and their applications, and in consultation with several statisticians concerning possible options.

LITERATURE REVIEW

Basic Physiology of the Heart

Basically, the heart is a pump which not only does extraordinary amounts of external work, but also supplies itself with the energy to operate. Since the heart is a constantly working muscle, it requires large quantities of nutrients, and rapid and efficient removal of waste products of metabolism in order to remain functional. These requirements are met by the coronary arteries, branching blood vessels which cover the surface of the heart and extend into the musculature, supplying every cardiac muscle cell with oxygen.

The flow in the coronary arteries is approximately 225 ml/minute, or four to five percent of the total cardiac output (Guyton, 1976). As blood passes through the coronary circuit, an unusually large portion of its oxygen is given up. Arterial blood with an oxygen content of 20 volume percent is reduced to about 5 volume percent, in other words the extraction of oxygen is high (Burton, 1965). The coronary arteries supply an unusually rich capillary network in which practically every muscle cell is adjacent to a capillary. The structure of the larger vessels is such that on the outer surface of the heart lie the large epicardial coronary arteries which are the primary suppliers of blood to the heart (see Figure 3). Below these arteries is a layer of smaller intramuscular arteries which penetrate the musculature and supply nutrients to the surrounding tissue on their way to the endocardium. Finally, below this bed of vessels lies the subendocardial arterial plexus, the innermost layer of blood vessels (Gray, 1977). Here, in

Diagram of Coronary Vasculature

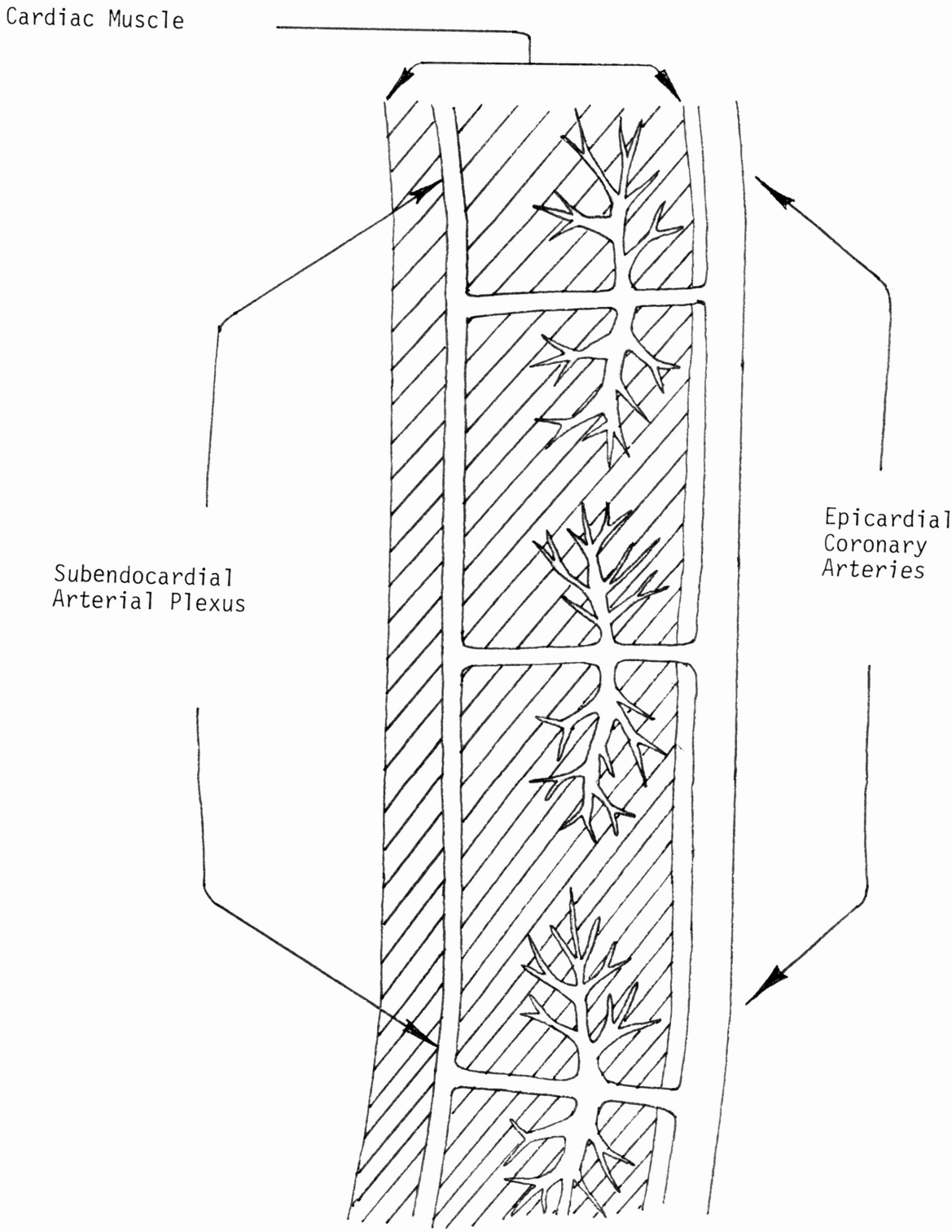

Figure 3

the depths of the ventricular musculature, the flow of blood drops to zero during the contraction of the heart (systole) due to the intraventricular pressure and the force exerted by the overlying layers of muscle during systole. Similarly when the heart is at rest (diastole) the blood flow through the subendocardial plexus is several times greater than flow in the outermost arteries. In order to pass such a large amount of blood the subendocardial arteries have to be much larger than the nutrient arteries in the middle and outer layers of the heart muscle.

Response of the Body to Stress

During strenuous exercise, the energy requirements of the heart are greatly increased due to the increased metabolism rate of the cardiac muscle cells. Since, as previously stated, a very high extraction of O_2 from the blood occurs normally in the heart, this demand for more oxygen cannot be met by more efficient extraction of O_2 from the blood, rather it must be met by increased coronary blood flow (Goldschlager, 1976). Although coronary flow is also affected by nervous control, it is generally agreed that the major factor in local blood flow regulation is the demand of the heart for oxygen (Guyton, 1976). It is believed that a lack of oxygen causes dilation of coronary arterioles. Several mechanisms by which this dilation is accomplished have been suggested but the most promising theory at present is as follows: a decrease in oxygen tension in the cardiac tissues directly reduces the amount of oxygen available to the coronary vessels which causes the arterial walls to weaken. As the walls weaken, the vessels dilate spontaneously

(Guyton, 1976).

This dilation of the coronary vessels is very important, in fact, it is essential to life itself. Were it not for this dilation a pathologic condition would exist, characterized by insufficient coronary blood flow. This condition is called coronary heart disease.

Atherosclerosis

The most frequent cause of this disease is atherosclerosis. Atherosclerosis is the deposition of lipids and cholesterol beneath the tunica intima at many points in the arteries. These areas of deposition frequently become calcified or invaded by fibrous tissue. This results in the development of atherosclerotic plaques and relatively hardened arterial walls which can no longer dilate in response to increased oxygen demand (Guyton, 1976).

Since the first few centimeters of the coronary arteries are a common site for formation of these plaques, ischemic heart disease is a common result. Although the heart is equipped to combat this build-up of plaques by developing collateral circulation in the areas of occluded arteries, the sclerotic process will usually develop beyond the limits of collateral development (Morris, 1978). Once this has occurred, the working capacity of the heart is severely limited, and there is a high probability of myocardial infarction which is cell death caused by an insufficient supply of oxygen to a specific area of cardiac muscle.

The purpose of treadmill stress testing is to recognize the onset of coronary atherosclerosis, before it has reached deadly proportions,

so that measures can be taken to prolong the patient's life. These measures may be as simple as a change in diet and a daily exercise program, or as drastic as coronary bypass surgery. By making the treadmill stress test a more dependable tool of the doctor, coronary heart disease could be made much easier to diagnose, and lives could no doubt be saved.

METHODS

Formulation of a Theory

The data collected by Dr. Alexander and his associates consisted of three measurements: systolic blood pressure, diastolic blood pressure, and heart rate, measured at eleven time intervals. One reading was taken before the stress test began; five readings were taken at two minute intervals during the test; and five readings were taken after the test at two minute intervals.

In order to determine the proper method for analysis of this data, a theory had to be formed concerning the expected results. In other words; it remained to be determined if the blood pressure response to an increased state of stress differed in healthy and diseased patients. If this difference existed and could be determined, general trends in the data could be identified, and various groups of patients could be classified.

This question of blood pressure response is not trivial. Many factors are involved in the regulation of blood pressure and they are all designed to keep the blood pressure within a relatively small, physiologically acceptable range. In determining the response of pressure to a stressed heart, it was helpful to consider the two pressure measurements independently. Systolic blood pressure is the higher of the two readings, and represents the maximum pressure generated by the heart during contraction. Diastolic pressure on the other hand, represents the minimum pressure within the blood vessels as the heart relaxes.

Significance of Systolic Blood Pressure

Since systolic pressure represents the maximum force exerted by the heart during the cardiac cycle, it is proportional to the ability of the heart to perform work. Therefore, it might be expected that in a diseased heart, the systolic blood pressure would drop. What actually occurs is that the heart works harder to maintain the desired pressure. But by increasing its work at a resting state, the heart sacrifices its ability to increase its output as drastically as a healthy heart. Therefore, a person with coronary heart disease will tire after performing a seemingly menial task. This is because his maximum cardiac output has been exceeded by even mild exercise, and the tissues of his body are not receiving sufficient oxygen.

Significance of Diastolic Blood Pressure

Diastolic pressure represents the pressure in the blood vessels while the heart is at rest. Naturally in order to assure constant profusion of the periphery and to prevent retrograde flow, the diastolic blood pressure must never fall below the values of pressure downstream. During diastole, the valves between atria and ventricles are opened, and blood flows from the atria and fills the ventricles. At the same time, blood flow in the coronary arteries reaches a maximum value. This is because during systole, the ventricular musculature is in total contraction and all of the blood vessels within the muscle are flattened and totally occluded by this pressure. As the heart relaxes in diastole, the natural rigidity of the coronary vessels causes them to open spontaneously and to carry blood throughout the heart. If these

vessels are occluded to the point that blood cannot pass through them, cardiac tissue can become ischemic.

The Regulation of Blood Pressure

In order to make a reasonable prediction concerning the effect of stress on the blood pressure response of a diseased heart, it was necessary to understand the various factors involved in blood pressure regulation. There are many effector and feedback mechanisms for the control of blood pressure. Therefore, it usually cannot be said that a change in blood pressure is the result of one distinct phenomenon.

One of the effector components for regulation of blood pressure is local vasodilation of the working musculature. Blood flow to an active muscle can increase by fifteen times during strenuous exercise and this increase is more than can be accounted for by sympathetic stimulation alone (Smith, 1976). Therefore, there must be some intrinsic decrease of vascular resistance in the working muscle during exercise. This decrease in resistance is brought about by vasodilation. As exercise is initiated, and the muscle's vessels dilate, sympathetic stimulation causes an increase in arterial pressure which greatly increases the blood flow through the working muscle. Were it not for sympathetic action, the blood pressure would drop drastically as a result of vasodilation. But the two mechanisms working in conjunction act to supply working muscle with more blood while decreasing blood flow in resting muscle.

Sympathetic stimulation has been called the "emergency" or "fight or flight" reflex. It has the effect of increasing the efficiency of

the heart as a pump by, (1) increasing heart rate, (2) increasing myocardial contractility, and (3) by decreasing both the diastolic and systolic ventricular volumes (this causes an infusion of blood into the periphery) (Smith, 1976). Sympathetic stimulation also tends to increase the mean circulatory pressure and the venous return to the heart.

There are many other control mechanisms which are known to regulate blood pressure. Some are documented and some are merely postulated to explain observed phenomena. These mechanisms include a hydrodynamic feedback system, cortical radiation (activating impulses to working muscle from the cortex), humoral feedback, baroreceptor feedback, muscle afferent feedback, and others (Smith, 1976). These are presented solely to indicate exactly how complicated the control mechanisms for cardiovascular response and blood pressure regulation are.

Still the question remains to be answered; how does a heart with partially occluded coronary arteries affect peripheral blood pressure response in muscle? In the case of a treadmill stress test the working muscles are in the legs while the blood pressure readings are taken at the brachial artery in the arm. Thus, by the previous model of sympathetic stimulation/vasodilation, the blood flow in the arm should be greatly reduced and the pressure elevated. In a diseased heart the coronary arteries cannot provide sufficient blood to the working cardiac musculature, and therefore the heart does not work efficiently as a pump. As a result, it would be expected that the blood perfusion pressure would drop somewhat when this point of cardiac insufficiency was reached. The systolic blood pressure should drop more drastically than

diastolic, because it is the systolic pressure which represents the maximum force exerted by the contracting heart. Diastolic pressure might be expected to drop as well, but again not as drastically as the systolic pressure.

Having made these basic assumptions, it became possible to select an appropriate analytical method for the collected data and begin classification.

Statistical Analysis

The analytical power desired in this situation was an ability to statistically discriminate between two groups, namely patients who were positive and negative for CHD. The specific test which performs this function, called discriminant analysis, was the method of analysis chosen in this case.

In order to distinguish between groups, a set of discriminating variables was selected, namely systolic and diastolic blood pressure and heart rate. To enable the statistical procedure to discriminate, these discriminating variables had to measure characteristics which were expected to differ. The procedure then performed a mathematical weighting and linear combination of the discriminating variables so that the two groups were forced to be as statistically distinct as possible. This was accomplished by performing one or more linear combinations of the discriminating variables. These "discriminant functions" were of the form:

$$D_i = d_{i1}Z_1 + d_{i2}Z_2 + \ldots + d_{ip}Z_p$$

where D_i = score on discriminant function "i"

d_{in} = weighting coefficients

Z_i = standardized values of the p discriminating variables used in the analysis (Zar, 1974)

These functions were formed so as to maximize the separation of the groups.

Once the functions were formed it was possible to pursue the two research objectives of this tool: analysis and classification.

The analysis aspects provided several tools for the interpretation of data. One of these was a test for measuring the success with which the discriminating variables actually discriminated when combined into the discriminant functions. Another was a test for homogeneity of the variance within a given group of variables.

After the initial computation and analysis, the classification techniques were applied. This involved the derivation of a set of classification functions which would permit the classification of patients with unknown memberships. This classification of unknown memberships was accomplished by the use of separate linear combinations of the discriminating variables for positive and negative patients. This produced a probability of membership in each group, and the patient was assigned to the group with the correspondingly higher probability.

The entire set of data was also analyzed in another similar but distinct method. The data was combined and reduced by the use of factor analysis. Factor analysis, characterized by its data reduction capabilities, assisted in the detection of underlying patterns of relationships in the data. If such a relationship existed, the data was

rearranged or reduced to a smaller set of factors which were taken as source variables that accounted for observed interrelations in the data.

RESULTS

SAS Subroutine Procedures

The discriminant analysis performed on the data involved the use of Statistical Analysis System. Statistical Analysis System (SAS) is a system consisting of various tools for all types of computer analysis of data. It includes the capacities for information storage and retrieval, data modification and programming, statistical analysis, and others.

The SAS subroutine which handles discriminant analysis is designated PROC DISCRIM. The output of DISCRIM contains the following information taken directly from the SAS manual (Barr and Goodnight, 1979).

(1) Values of the classification variable, frequencies, and prior probabilities for each group.

(2) Simple descriptive statistics including the number of observations, sum, mean, variance, and standard deviation for each group.

(3) The within-group covariance matrix S_t for each group.

(4) The within-group correlation matrix for each group.

(5) If the pooled covariance matrix is used, the linearized discriminant function includes the pooled variables.

(6) The results of a chi-square test of homogeneity of the within-group covariance matrices.

(7) The generalized squared distance between groups.

(8) The classification results for each observation, including the observation number (if an ID statement is included, the values of the identification variable are printed instead

of the observation number), the actual group for the observation, the group into which the developed criterion would classify it, and the posterior probability of its membership in each group.

(9) A summary of the performance of the classification criterion.

In addition to the utilization of PROC DISCRIM, a factor analysis procedure, PROC FACTOR was used. The discriminant analysis was performed on a combination of a merged pool of the raw data and the four factors, and also on the factors alone. This was done twice using two different data sets. One set included systolic blood pressure and heart rate, and the other contained diastolic blood pressure and heart rate. A program was also run using all three variables combined but the combination seemed to mask differences between pools and thus lower discriminating capacity drastically.

The discriminating function was obtained from DISCRIM by using a data set consisting of 45 patients. Then, in order to test the discriminating capacity of the functions, a special option called TESTDATA was utilized. This option used data for 50 different patients and attempted to classify them by using the discriminating function obtained previously. This classification of patients with unknown membership is exactly the type of test which could be done in a cardiac clinic as an aid in the diagnosis of CHD. It involves nothing more than summing the products of discriminant functions and variables and can be performed easily with the use of a hand held calculator with a continuous memory. It is this application which would be the ultimate practically applicable result of my project.

Initial Output

When this program was run initially, it failed to perform the analysis needed. This was due to the fact that out of 48 patients studied, only 6 had complete data present. That is to say, that the other 42 patients had one or more missing values in the data describing their stress tests. The DISCRIM procedure takes all the values of variables for a particular patient and stores them in an array or matrix. Since all of the analysis was done using this matrix, no missing value could be permitted, and any patient with a missing value was eliminated from the analysis. Therefore, the output from this program consisted of discriminant analysis performed on 6 patients, and it provided no useful information.

Upon analysis of the raw data, on the patients of missing values, two separate groups became apparent. One group consisted of patients who had not had their heart rate recorded before the test began. The other group consisted of patients who had not finished the test due to exhaustion, dyspnea, or other reasons specified by the doctor in charge. In order to perform the analysis, some adjustment had to be made if any results were expected.

The problem with the group of patients for which data was lacking on the initial heart rate was solved fairly easily. By examining Dr. Alexander's files, I obtained resting heart rate values (which were recorded at other times) for all the patients who lacked this data. While obtaining this supplementary data, I also checked the files of patients who were not missing data on their initial heart rates, and

looked up their resting heart rates. The values recorded for their initial heart rates in the files and before the test were all quite similar (all were within plus or minus 3 mm Hg). Therefore, I do not feel that any sensitivity of the test was lost in this addition of data.

After the data on initial heart rates had been added, there still remained 29 patients whose missing data was due to the fact that they had failed to finish the stress test. These patients fell into 3 groups, those who failed to finish the last 2, 4, and 6 minutes of the test. Since the majority of the patients who were diagnosed as positive for CHD had failed to complete the test, eliminating them from the analysis would destroy any chance of good results.

Similarly, if analysis were done on the 3 groups separately, (1) the groups would have been too small, and (2) an insufficient number of negative patients would have been included. The only feasible alternative remaining was to eliminate the last 6 minutes during the stress test for everyone, and perform the analysis using the initial four minutes during the test, and the ten minutes following the test. There is no doubt that this did not hurt the analysis and the final results, but it was a necessary adjustment, and was the result of laboratory considerations which were beyond anyone's control.

Finally, after these adjustments, the program ran successfully and printed out statistically correct and physically analyzable results. The program obtained is being submitted with this report, but excerpts of significant sections are included within these pages.

The first results which provided information as to the nature of the data being studied was the factor patterns (see Figure 4). As

can be seen, the factors obtained using both systolic and diastolic blood pressure contain some very interesting and statistically significant patterns. Each of the factors obtained can be seen to statistically represent different physical characteristics of the data. Take as an example factor number 3 derived from systolic blood pressure. The negative values indicate that there is some physical explanation of the systolic blood pressure response to stress by a factor which subtracts values for increasing systolic blood pressures and heart rates and adds values for decreasing systolic blood pressures and heart rates. In other words, this factor could be physically interpreted to represent the slope of the blood pressure and heart rate curves. Other definite patterns are also visible in the factors, although they might not be so easily explained.

These definite patterned factors indicate that the variability of the data is mathematically as well as physically interpretable. In other words, the data makes sense; it is consistent from patient to patient and explains much of the variability of the data. The exact quantity of variability which was described by each factor is shown below in tabular form.

Systolic Blood Pressure and Heart Rate	Factor 1	39.6%
	Factor 2	20.3%
	Factor 3	10.8%
	Factor 4	7.8%
	Total	78.5%
Diastolic Blood Pressure and Heart Rate	Factor 1	31.9%
	Factor 2	29.1%
	Factor 3	9.2%
	Factor 4	8.5%
	Factor 5	9.2%
	Total	85.9%

The next interesting portion of output was the eigen values. These values indicate the portion of the variability of the data which can be explained. The larger these values the better, because the magnitudes of the eigen values indicate the probability that there exists a linear combination of variables which will explain a large proportion of the data. This is a requirement of the data if it is to be successfully discriminated.

Another portion of output which actually provided information on how well the two discriminant functions were separated was the generalized square distance function (see Figures 5 and 7). This indicated the distance between the two groups (which should ideally be very large) and the distance within the two groups (which should be very small). The ideal generalized square distance function would be as follows:

from CL	0	1
0	minimum	maximum
1	maximum	minimum

The above is the case because it is desired that each group be as concentrated as possible, hence low values for distances between 0 and 0, and between 1 and 1. Similarly, in order to discriminate between the two groups, they should be as far apart as possible so as to be distinct, hence, high values for distances between 0 and 1.

The results of the discriminating classification tell the entire story. As can be seen in Figures 5 and 7 the test classified every patient perfectly into the correct group (misclassifications are denoted by an asterisk). This means that when the classification of a

patient is known, there are sufficient differences between positive (1) and negative (0) patients that can be extracted from the data so that the two groups can be discriminated between successfully. As shown, the discrimination is 100% positive, in almost every case. The results are entirely similar whether systolic blood pressure or diastolic pressure as shown by the two columns on the right which are the normalized probabilities for classification.

Unfortunately, the classification of test data did not provide quite as astoundingly accurate results as I had hoped it would. The results of these tests can be seen in Figures 6 and 8.

In the analysis using systolic blood pressure and heart rate (Figure 6) there were 13 correct classifications of negative patients, and 15 correct classifications of positive patients. But there were 17 false positive classifications and 5 false negative classifications. In all, 28 patients were correctly classified while 22 were incorrectly classified. The tendency for misclassifications seems to tend toward false positives which was the characteristic I had hoped to correct by this project. But the five false negatives are totally unacceptable as is the case in most all medical diagnoses.

The test classifications using diastolic blood pressure and heart rate for analysis performed slightly worse than did the previous test. In this analysis (Figure 8) there were 4 correct classifications of negative patients and 16 correct for positive patients. There were 26 misclassifications of false positive and 4 false negatives. In all, 20 patients were correctly classified while 30 were incorrectly classified.

FACTORS DERIVED FROM SYSTOLIC BLOOD PRESSURE AND HEART RATE

4 FACTORS WILL BE RETAINED.

FACTOR PATTERN

	FACTOR1	FACTOR2	FACTOR3	FACTOR4
SBP1	0.62419	0.34678	-0.16875	-0.36974
SBP2	0.64572	0.34025	-0.39800	0.20469
SBP3	0.61549	0.39904	-0.37664	0.24404
SBP7	0.26843	0.40692	-0.11204	0.73631
SBP8	0.64906	0.42293	0.24131	0.36867
SBP9	0.66630	0.43481	0.45336	-0.07966
SBP10	0.49218	0.35677	0.65635	-0.22398
SBP11	0.33708	0.59607	0.51429	-0.03214
HR1	0.61220	0.20738	-0.29703	-0.30592
HR2	0.81844	0.03286	-0.33922	-0.19753
HR3	0.79994	0.01050	-0.36486	-0.28299
HR7	0.49013	-0.71527	0.07939	0.18931
HR8	0.58423	-0.61893	0.09948	0.16188
HR9	0.66689	-0.58356	0.23630	0.01335
HR10	0.78052	-0.51245	0.07544	0.05191
HR11	0.73100	-0.53715	0.14632	0.03306

FACTORS DERIVED FROM DIASTOLIC BLOOD PRESSURE AND HEART RATE

5 FACTORS WILL BE RETAINED.

FACTOR PATTERN

	FACTOR1	FACTOR2	FACTOR3	FACTOR4	FACTOR5
DBP1	-0.24728	0.48370	-0.49076	0.53696	0.06197
DBP2	-0.18895	0.62164	-0.16292	0.39841	-0.49484
DBP3	-0.26928	0.76677	-0.01251	0.20667	-0.48723
DBP7	-0.44664	0.59732	0.35938	-0.23861	-0.28089
DBP8	-0.38087	0.71139	0.45870	-0.19858	-0.01641
DBP9	-0.50958	0.66063	0.42028	-0.15108	0.11932
DBP10	-0.45979	0.66429	0.04609	0.20494	0.47855
DBP11	-0.23406	0.66052	-0.14414	0.22206	0.54866
HR1	0.22253	0.67574	-0.27312	-0.47357	0.08741
HR2	0.56181	0.56128	-0.40372	-0.32614	-0.07343
HR3	0.56795	0.50046	-0.35489	-0.43314	0.01864
HR7	0.77718	0.08584	0.33288	0.25434	-0.01335
HR8	0.78116	0.17501	0.32641	0.13497	0.08592
HR9	0.83614	0.21570	0.26164	0.10009	0.13994
HR10	0.83478	0.29765	0.07502	0.10285	0.04228
HR11	0.86136	0.27825	0.08605	0.19202	-0.04021

Figure 4

DISCRIMINANT ANALYSIS OF INITIAL DATA

USING SYSTOLIC BLOOD PRESSURE AND HEART RATE

GENERALIZED SQUARED DISTANCE TO CL

FROM CL	0	1
0	66.88040784	75.58889280
1	77.97855091	68.82636653

PH	FROM CL	CLASSIFIED INTO CL	0	1
284	0	0	1.0000	0.0000
851	0	0	1.0000	0.0000
717	0	0	1.0000	0.0000
121	0	0	1.0000	0.0000
906	0	0	1.0000	0.0000
226	0	0	1.0000	0.0000
138	0	0	1.0000	0.0000
868	0	0	1.0000	0.0000
103	0	0	1.0000	0.0000
779	0	0	1.0000	0.0000
261	0	0	1.0000	0.0000
86	0	0	0.9999	0.0001
933	0	0	0.9979	0.0021
765	0	0	1.0000	0.0000
852	0	0	0.9947	0.0053
196	0	0	1.0000	0.0000
441	0	0	1.0000	0.0000
956	0	0	0.9178	0.0822
515	0	0	1.0000	0.0000
1007	0	0	1.0000	0.0000
320	0	0	1.0000	0.0000
74	0	0	1.0000	0.0000
949	1	1	0.0000	1.0000
643	1	1	0.0000	1.0000
596	1	1	0.0000	1.0000
161	1	1	0.0000	1.0000
236	1	1	0.0000	1.0000
701	1	1	0.0000	1.0000
234	1	1	0.0000	1.0000
529	1	1	0.0000	1.0000
363	1	1	0.0000	1.0000
134	1	1	0.0000	1.0000
984	1	1	0.0002	0.9998
733	1	1	0.0000	1.0000
941	1	1	0.0000	1.0000
775	1	1	0.0000	1.0000
475	1	1	0.0000	1.0000
715	1	1	0.0000	1.0000
277	1	1	0.0000	1.0000
224	1	1	0.0000	1.0000
872	1	1	0.0000	1.0000
687	1	1	0.0000	1.0000
543	1	1	0.0001	0.9999
862	1	1	0.0000	1.0000
670	1	1	0.0000	1.0000

Figure 5

RESULTS OF DISCRIMINATING TEST ON CHECK DATA

USING SYSTOLIC BLOOD PRESSURE AND HEART RATE

FROM CL	0	1	TOTAL
0	13	17	30
	43.33	56.67	100.00
1	5	15	20
	25.00	75.00	100.00
TOTAL	18	32	50
PERCENT	36.00	64.00	100.00
PRIORS	0.5000	0.5000	

PN	FROM CL	CLASSIFIED INTO CL		0	1
119	1	1		0.0000	1.0000
933	1	0	*	0.9979	0.0021
395	0	0		1.0000	0.0000
819	0	1	*	0.1041	0.8959
737	0	1	*	0.0000	1.0000
552	1	0	*	1.0000	0.0000
856	0	1	*	0.0000	1.0000
688	0	1	*	0.0000	1.0000
449	0	1	*	0.0000	1.0000
178	0	0		1.0000	0.0000
334	0	1	*	0.0000	1.0000
783	0	1	*	0.0000	1.0000
289	0	1	*	0.0007	0.9993
999	0	0		0.9964	0.0036
>286	0	1	*	0.0000	1.0000
774	0	1	*	0.0000	1.0000
455	0	0		0.9806	0.0194
222	0	0		0.9686	0.0314
440	0	0		1.0000	0.0000
256	0	0		1.0000	0.0000
>286	0	1	*	0.0000	1.0000
655	0	0		0.9769	0.0231
271	0	0		1.0000	0.0000
546	0	0		0.9999	0.0001
736	0	1	*	0.0000	1.0000
630	0	1	*	0.0000	1.0000
545	0	0		1.0000	0.0000
912	0	1	*	0.0000	1.0000
530	0	1	*	0.0000	1.0000
223	0	1	*	0.0000	1.0000
603	0	0		0.8787	0.1213
561	0	0		1.0000	0.0000
303	0	1	*	0.0013	0.9987
111	1	1		0.0000	1.0000
414	1	1		0.0000	1.0000
179	1	1		0.0001	0.9999
432	1	1		0.0000	1.0000
841	1	0	*	1.0000	0.0000
977	1	1		0.0000	1.0000
957	1	1		0.0000	1.0000
711	1	1		0.0000	1.0000
198	1	1		0.0003	0.9997
345	1	1		0.0000	1.0000
883	1	1		0.0000	1.0000
805	1	0	*	1.0000	0.0000
986	1	1		0.0000	1.0000
101	1	1		0.0000	1.0000
274	1	1		0.0000	1.0000
167	1	1		0.0000	1.0000
650	1	0	*	1.0000	0.0000

* MISCLASSIFIED OBSERVATION

Figure 6

DISCRIMINANT ANALYSIS OF INITIAL DATA

USING DIASTOLIC BLOOD PRESSURE AND HEART RATE

GENERALIZED SQUARED DISTANCE TO CL

FROM CL	0	1
0	53.57611291	69.70835036
1	73.08336832	60.08568657

PN	FROM CL	CLASSIFIED INTO CL	0	1
284	0	0	1.0000	0.0000
851	0	0	1.0000	0.0000
717	0	0	1.0000	0.0000
121	0	0	1.0000	0.0000
906	0	0	1.0000	0.0000
226	0	0	1.0000	0.0000
138	0	0	1.0000	0.0000
869	0	0	1.0000	0.0000
103	0	0	1.0000	0.0000
779	0	0	1.0000	0.0000
261	0	0	1.0000	0.0000
86	0	0	0.9772	0.0228
933	0	0	1.0000	0.0000
765	0	0	1.0000	0.0000
850	0	0	0.9994	0.0006
196	0	0	1.0000	0.0000
443	0	0	1.0000	0.0000
956	0	0	0.9985	0.0015
515	0	0	1.0000	0.0000
107	0	0	1.0000	0.0000
329	0	0	1.0000	0.0000
74	0	0	0.9993	0.0007
949	1	1	0.0000	1.0000
647	1	1	0.0000	1.0000
596	1	1	0.0000	1.0000
161	1	1	0.0000	1.0000
236	1	1	0.0000	1.0000
701	1	1	0.0000	1.0000
234	1	1	0.0000	1.0000
629	1	1	0.0000	1.0000
363	1	1	0.0000	1.0000
134	1	1	0.0000	1.0000
984	1	1	0.0000	1.0000
733	1	1	0.0000	1.0000
941	1	1	0.0000	1.0000
775	1	1	0.0000	1.0000
479	1	1	0.0000	1.0000
715	1	1	0.0000	1.0000
277	1	1	0.0000	1.0000
224	1	1	0.0000	1.0000
872	1	1	0.0000	1.0000
687	1	1	0.0000	1.0000
543	1	1	0.0903	0.9097
862	1	1	0.0000	1.0000
682	1	1	0.0000	1.0000

Figure 7

RESULTS OF DISCRIMINATING TEST ON CHECK DATA

USING DIASTOLIC BLOOD PRESSURE AND HEART RATE

FROM CL	0	1	TOTAL
0	4	26	30
	13.33	86.67	100.00
1	4	16	20
	20.00	80.00	100.00
TOTAL	8	42	50
PERCENT	16.00	84.00	100.00
PRIORS	0.5000	0.5000	

OBS	FROM CL	CLASSIFIED INTO CL		0	1
119	1	0	*	0.9989	0.0011
937	1	0	*	1.0000	0.0000
395	0	1	*	0.0000	1.0000
815	0	1	*	0.0000	1.0000
737	0	1	*	0.0000	1.0000
552	1	1		0.0000	1.0000
856	0	1	*	0.0000	1.0000
678	0	1	*	0.0001	0.9999
449	0	1	*	0.0000	1.0000
179	0	0		1.0000	0.0000
334	0	1	*	0.0000	1.0000
783	0	1	*	0.0000	1.0000
289	0	1	*	0.0000	1.0000
999	0	1	*	0.0000	1.0000
296	0	1	*	0.0000	1.0000
374	0	1	*	0.0000	1.0000
455	0	1	*	0.0000	1.0000
200	0	1	*	0.0000	1.0000
442	0	0		0.9819	0.0181
256	0	1	*	0.0000	1.0000
286	0	1	*	0.0000	1.0000
655	0	1	*	0.0000	1.0000
271	0	1	*	0.0000	1.0000
546	0	1	*	0.0000	1.0000
736	0	0		1.0000	0.0000
630	0	1	*	0.0000	1.0000
545	0	1	*	0.0000	1.0000
910	0	1	*	0.0000	1.0000
532	0	1	*	0.0000	1.0000
203	0	1	*	0.0000	1.0000
603	0	1	*	0.0000	1.0000
561	0	1	*	0.0000	1.0000
303	0	0		1.0000	0.0000
111	1	1		0.0000	1.0000
414	1	1		0.0000	1.0000
179	1	1		0.0000	1.0000
432	1	1		0.0000	1.0000
840	1	1		0.0000	1.0000
977	1	1		0.0004	0.9996
957	1	1		0.0000	1.0000
711	1	1		0.0000	1.0000
196	1	0	*	0.9990	0.0010
346	1	1		0.0000	1.0000
883	1	1		0.0000	1.0000
805	1	1		0.0000	1.0000
986	1	1		0.0000	1.0000
121	1	0	*	0.6210	0.3790
274	1	1		0.0000	1.0000
167	1	1		0.0000	1.0000
650	1	1		0.0000	1.0000

* MISCLASSIFIED OBSERVATION (50)

Figure 8

DISCUSSION OF RESULTS AND CONCLUSION

The results obtained from this project are not nearly as far reaching as I had hoped they would be. The real purpose of the project, the correct classification of patients on the basis of blood pressure and heart rate has not been obtained to any practical degree. Nevertheless, I do feel that significant progress has been made toward that goal, and that significant results have been obtained in the process.

To the best of my knoweldge this is the first time that anyone has ever tried to formulate a procedure for the computer based diagnosis of coronary heart disease on the basis of blood pressure and heart rate alone. A successful accomplishment of this task would be a great advancement in the field of cardiology, and would also provide interesting and even revolutionary insights into the pathological functioning of the heart with CHD. Just because the desired results were not attained does not indicate that this research has failed. Rather, a method has been proposed for the diagnosis of CHD and the study of this method has shown that this diagnosis could definitely be possible.

The fact that definite factor patterns and high eigen values were obtained combines to indicate that the data is both mathematically and physically interpretable. In addition, the discriminant functions which were formed have perfect records of classifications. This indicates that the blood pressure and heart rate do indeed measure quantities which are significantly different for positive and negative patients, and that this difference can be used to discriminate.

It is my feeling, that a continuation of this research could yield very interesting and applicable results. There are many other statistical methods which could be applied to the data. One of these is factor rotation which could well lead to the results expected. In addition, there are many things which could be done to the data before it is analyzed which could cause a better yield of results. If the groups were larger or were divided up into more exclusive populations, the results would no doubt improve. The only criteria for consideration in this study was that the patient be a male, aged 40 to 65. If this group was further subsdivided on the basis of whether the patient smokes, his dietary habits, whether his lifestyle is sedentary or active, or any other variable known to be linked to CHD, results would again improve. If physical and family history could somehow be integrated into the analysis, there is really no predicting as to how accurate the diagnosis might be. The possibilities are virtually limitless, and the information learned from a continuation of the research in this direction could well provide a new understanding of coronary heart disease in the medical and social communities.

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