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NON-LINEAR ANALYSIS OF ELECTROCARDIOGRAM TRACINGS IN DETECTION OF OCCULT CORONARY ARTERY DISEASE

BY

JOSEPH R. LEVITT

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NON-LINEAR ANALYSIS OF ELECTROCARDIOGRAM TRACINGS IN DETECTION OF OCCULT CORONARY ARTERY DISEASE

BY

JOSEPH R. LEVITT

FOR

DEPARTMENT OF MECHANICAL ENGINEERING

BY

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Newark, New Jersey
May 1970

ABSTRACT

The general problem of identifying significant characteristics of a system by analyzing the properties of a signal emitted by that system is common to many disciplines, biological as well as physical. In general, achievement of a satisfactory solution depends on the capability of assigning membership of candidate signals to one of a number of mutually exclusive categories.

In this study, a new technique of processing the standard electrocardiograms from human subjects has been developed. This technique employs a set of non-linear transformations which enable the assignment of electrocardiograms into one of two mutually exclusive categories. The first category is that of patients with occult coronary artery disease; the second category is that of subjects free from coronary artery disease.

An introduction to the conventional means for assessing the standard electrocardiogram is presented, and the limitations noted. The new method is then presented which consists in large part of combining non-linear signal processing with a topological re-orientation of conventional cartesian coordinates to yield a multi-vector space which offers an optimum degree of visual perceptibility. Moreover, in this new domain, the transformed EKG tracings taken from subjects free from coronary artery disease

inhabit a closed area, represented by the annular space bounded by two tangent circles. It is then shown that EKG tracings taken from subjects with occult coronary artery disease exhibit patterns which protrude beyond the circular boundaries.

The mathematical transformations are shown to be non-linear and non-analytic in terms of satisfying the Cauchy-Riemann conditions. The assumptions and constraints of the transformations are explored and practical considerations are examined.

Preliminary results obtained to date from over 100 medical cases are presented and analyzed. The tentative findings reveal a high degree of promise for the detection of asymptomatic coronary candidates well in advance of myocardial infarction. Finally, further applications to other medical areas are explored including a discussion of potential applicability to the fields of pharmacology and neurology.

ACKNOWLEDGMENTS

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1 - INTRODUCTION

1.1 Classifying Complex Data

That a body of complex data may contain elements with significance for the human observer represents an age-old problem, extending from man's objective intellectual reconstruction of nature to his subjective and instinctive capability in pattern recognition.

Examples one might mention include recognition of a spoken word independent of the speaker who utters it, recognition of a familiar face within a crowd, recognizing a person from his handwriting, and of distinguishing the sound of one musical instrument from that of another. Clearly, the mind possesses some mechanism for reducing a great deal of data to the barest of essentials.[1]

In the work which follows we shall be concerned principally with the classification of data within the medical field, specifically in cardiology.

1.2 Diagnosing Heart Disease

Application of various data classification methods within the medical field has increased extensively within the past decade. The importance of diagnostic accuracy in heart disease, one of the most gravely prevalent public health problems, cannot be overstated.

Over one eighth of the total American population suffer from some sort of heart disease. It has been established that nearly 50 per cent of deaths in the United States are directly attributable to clinical or covert heart disease or cardiovascular involvement secondary to rheumatic fever, diabetes, and other diseases.[3]

Techniques for accurate diagnosis and prediction of heart disease are clearly of extreme importance. The deductive process applied by a physician to diagnose cardiac anomalies is contingent upon careful obobservation, experience, and some degree of intuition. He is aided in his work by countless procedures and equipment. The electrocardiograph, currently employed even in routine cases, constitutes one of the principal diagnostic tools. Ideally, the electrocardiogram would depict an integrated electrical cardiac function, wherein all active components such as mechanical pumping action and other seemingly minor elements would be equally and exhaustively represented. Actually, it offers only an incomplete recording of the electrical activity of the heart.

1.3 Cardiac Cineangiography

Patients have suffered coronary episodes despite negative physical examination which included a normal

electrocardiogram. Consequently, new techniques for detection of occult coronary artery disease have been developed, particularly cineangiography with cardiac catheterization.

This fluoroscopic procedure involves insertion of a small teflon catheter through a superficial blood vessel in the arm or thigh which is then guided into the heart. If introduced through a ventral anconal vein, the catheter enters through the venous system into the superior vena cava, right atrium, right ventricle, and pulmonary artery. The catheter may be used for extraction of cardiac blood samples, blood pressure measurement, and introduction of certain chemicals into the heart for various purposes. The methodology directing the cardiac insertion of the catheter into the left side of the heart concerns the present study due to its relevance to coronary artery visualization.[4]

Currently, two general approaches to coronary catheterization are available. In one technique, the catheter is inserted into the femoral artery percutaneously and advanced to the ascending aorta, where the tip is placed in the immediate supravalvular region. In the second approach, the catheter is inserted into the brachial artery and directed into

the coronary arterial bed. Catheterization of the right coronary artery is easier from the brachial approach, but the left coronary artery is more easily catheterized from the femoral artery.[4]

Cineangiography consists of the introduction of roentgenopaque liquids into the blood stream, thus permitting serial X-ray demonstration of fluid passage through the heart and its major vessels. The function and condition of these vessels may be visualized directly. Thus considerable information regarding the existence of coronary artery lesions is readily obtained. Selective coronary arteriography enables precise visualization regarding presence, location, and extent of occlusive disease.[4] For example, Figure 1.1 shows the cineangiogram of a normal right coronary artery, with smooth-walled vessels and almost no variation in lumen diameter. A cineangiogram of a diseased right coronary artery is exhibited in Figure 1.2. Here one notes the severe narrowing of the lumen diameter at a distal point indicative of a lesion. Additional comments relative to cineangiography and coronary artery disease will be found in section 6.6.

1.4 Electrocardiographic Pattern Recognition

The cardiographer is trained to identify specific heart anomalies by certain patterns appearing on the

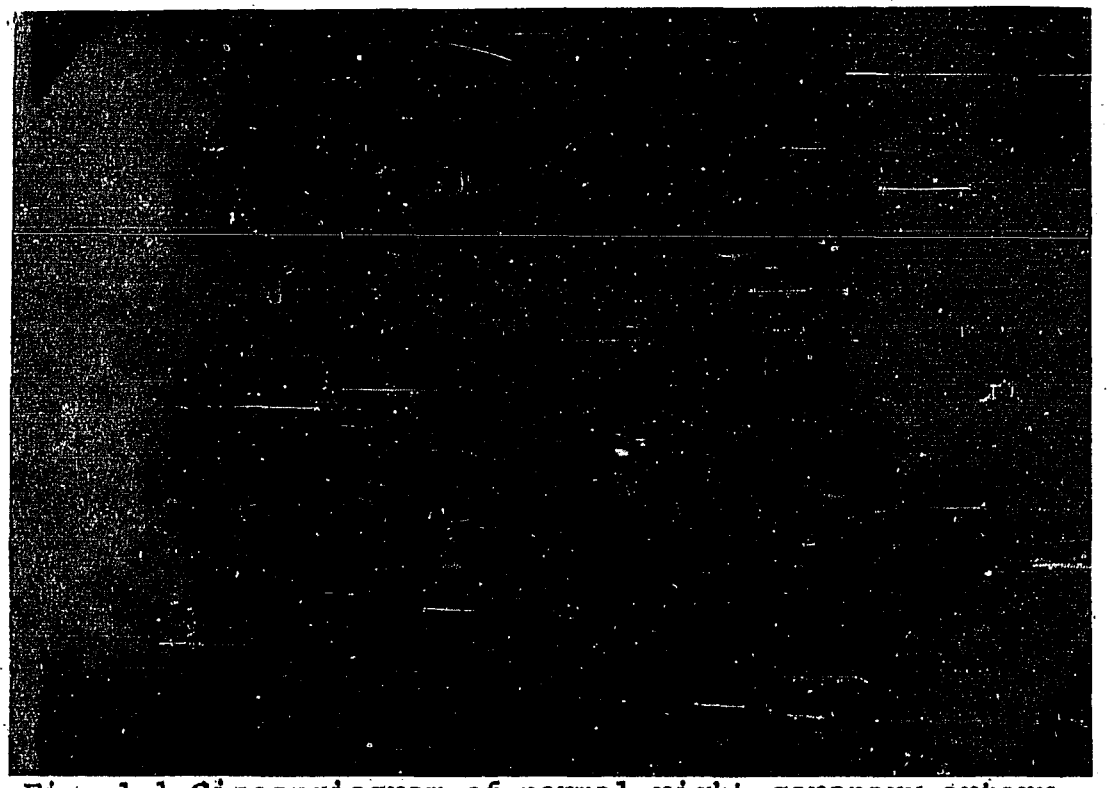


Fig. 1.1 Cineangiogram of normal right coronary artery.

Fig. 1.2 Cineangiogram of diseased right coronary artery.

electrocardiogram. If physical diagnosis and medical history indicate presence of a particular disorder, such as ventricular hypertrophy, a definite electrocardiogram pattern may thus be anticipated.

In this sense, the electrocardiograph performs essentially as an analog computer, assimilating, processing, and visually displaying complex bodies of data.[5] A typical EKG pattern and associated nomenclature is shown in Figure 1.3. Certain signals, such as prolongation of the time interval between the P-wave and QRS complex, usually an indication of delayed conduction at the A-V node, are "translated" into the electrocardiogram language, represented by a series of wave-patterns of varying shape and magnitude. Rhythm and frequency of these patterns are also significant. Since the EKG is essentially an amplitude-time history of cardiac potentials one may correctly assume that these potentials give rise to a process of electrical conduction within the heart.

The "normal" process of conduction of impulses may be interrupted in the following three ways:

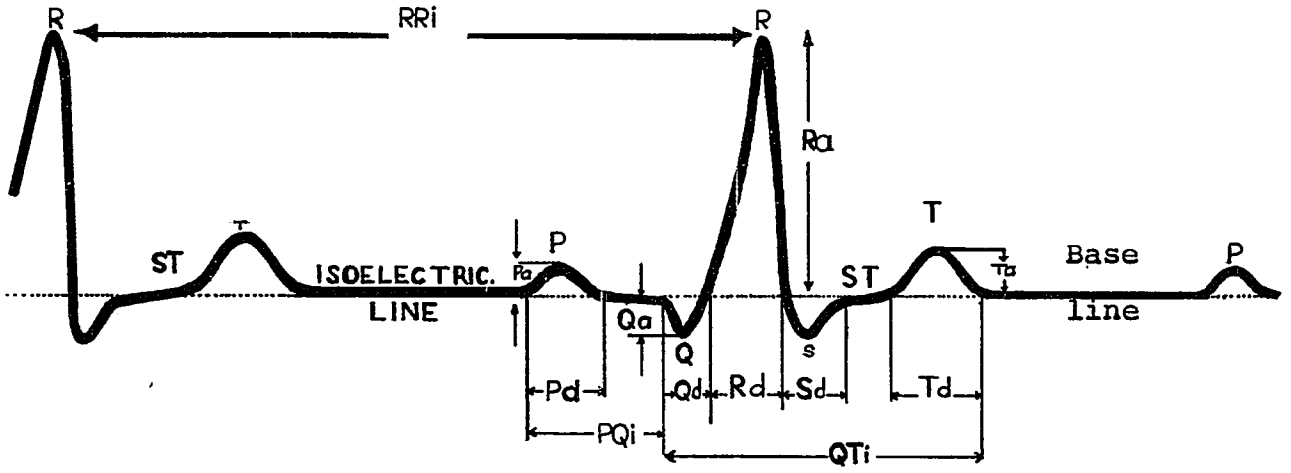
(a) By conduction delay at a given point.

(b) By total disturbance of the impulse at a specific point along its course.

(c) By nonresponsiveness of some portion of the conducting pathway.

Subscript Legend:

a = amplitude
i = interval
d = duration

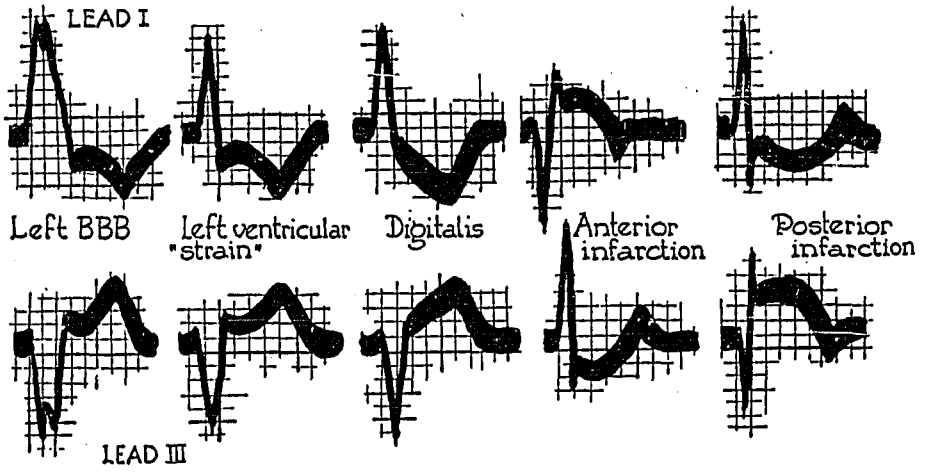


Typical EKG Pattern and Nomenclature
Fig. 1.3

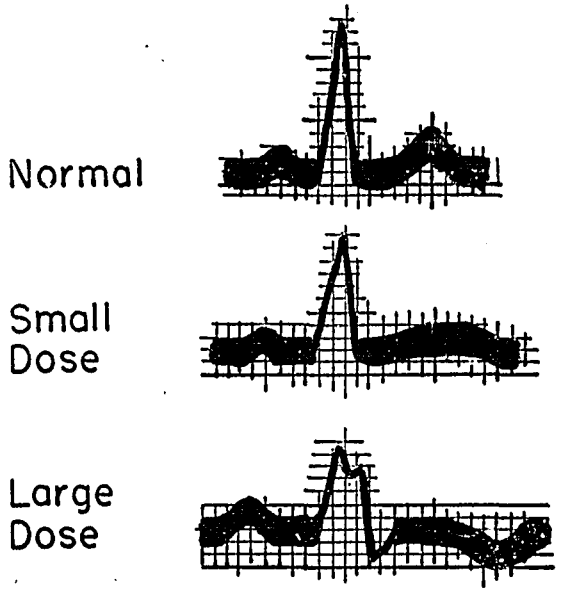
In Figure 1.4 an example of the first instance would be delayed atrioventricular conduction, or abnormally long interval between contraction of atria and ventricles.[8] This is a form of first-degree heart block, often resulting from ingestion of certain drugs such as quinidine. Electrocardiogram interpretation of such a condition would consist of alteration of P and T waves and QRS complex, visually represented by a tendency toward shortening and duplication of the vertical wave.

The second situation might be exemplified by the case of myocardial infarction, either anterior, represented by greatly shortened and rounded waves on the electrocardiogram visual format, or posterior, where the waves often assume a tall, pointed shape. Infarction usually arises from sclerosis or other conditions creating both anatomic and physiologic impediment within the organ, and generally constitutes either second degree or complete heart block, in terms of severity or reversibility.

The third instance may be illustrated by disorders such as the relatively rare <u>atrioventricular dissociation</u>, in which the atrioventricular node ejects impulses at an abnormally high speed, exceeding that at the sinoatrial node, and therefore primarily activates only the ventricles. Nonresponsiveness of



.—Diagrammatic tracings showing various types of S–T segment shifts and T waves.



.—Changes in the P wave, QRS complex and T wave produced by increasing doses of quinidine.

Fig. 1.4 Variations in EKG Patterns

conducting pathways may isolate these supranormal impulses from the atria, which respond to the normal sino-atrial node stimulation. Such condition, generally temporary and reversible and often drug-influenced, may be classed as first-degree heart block.

Most types of coronary artery disease fall within the second category, where the impulse is totally interrupted at some point along its course.[7] The arteries, thickened sclerotically or by hypermegaly, distend the atria and/or ventricles, thereby obstructing normal conduction and transmission, or "input" and "output", of electric impulses. Bundle branch block, a common disorder of this type, involves an interruption of the conduction pathway in the lower, single-branched portion of the bundles of His. The impulse is detoured to the ventricle opposite that normally receiving it, and a grossly abnormal pattern appears on the electrocardiogram. The waves widen, become backwardly diagonal, and exhibit a tendency toward duplicity or separation at the peak.

The term "left ventricular strain" also within this category, represents those electrocardiographic patterns manifesting left ventricular hypertrophy with ischemia, or extreme left axis deviation with late intrinsicoid deflection. The resulting wide QRS complex and T-wave disappearance are translated into a series

of sharpened, slightly shortened, and less frequently-appearing waves on the electrocardiogram image.

The electrocardiogram thus executes a sort of analog processing of all unassorted data received by breakdown and transformation of this data into a linear graphic visual field.

Admittedly, in many cardiac disorders the EKG patterns which emerge are difficult to perceive. However, in occult coronary artery disease, these patterns are so nearly identical with "normal", that subtle changes are almost impossible to perceive in many if not most cases. The main thrust of this research is in the area of assessing this class of EKG's. This is done by transforming the EKG pattern from its conventional form to a new domain which is non-linear but offers a much higher degree of visual perceptibility. This transformed display is denoted as the omnicardiogram (OCG) and will be developed fully in later sections.

1.5 Human Variability in Classifying Electrocardiograms

Caceres[9] has noted that the significance of observer variation in relation to clinical practice can be examined by comparing reports of a series of randomly selected hospital electrocardiograms in order to determine the extent and nature of disagreement on diag-

nostic findings. He considered 561 electrocardiograms of adult hospital patients routinely processed through a hospital heart station. During this period a group of electrocardiographers gave separate interpretations of their EKG findings in accordance with their background and experience. The same electrocardiograms were later analyzed independently by another set of readers. Agreement on interpretation was said to occur when diagnostic findings reported by both groups were the same, and disagreement when one or more diagnoses were reported by only one of the two groups. There was agreement in 59.7 per cent of the total 561 electrocardiograms and disagreement in 40.3 per cent. This report is one of several[10] which appear to suggest that classification of EKG diagnostic findings by experienced electrocardiographers tends to be somewhat subjective. Presumably, variations in methods of measuring and in diagnostic criteria are responsible for divergence of opinion on interpretation. How frequently such problems may arise is suggested by the finding that pathological conditions were reported by only one reading group in thirty nine cases of myocardial infarction. Such discrepancies in EKG interpretation are a matter of concern and tend to support the suggestion that a computer processed electrocardiogram may be

of significant aid to the physician interpreting an electrocardiogram for patient diagnosis, care, or clinical study purposes.

2 -PHYSIOLOGY OF THE HEART

2.1 Anatomy

Thorough comprehension of heart physiology is not necessary for this study; however, a brief description (Fig. 2.1) is helpful. The organ consists of four hollow chambers: two atria and two ventricles. The atria are thin-walled and act as receptacles for afferent arterial blood. The ventricles are thick-walled and consist of several muscular layers constituting the pump section. The two atria and ventricles are separated by partitions called septa. The heart is actually a twin pump: the right side conducting venous blood and the left side arterial blood. They function quite independently and are often termed the "right heart" and the "left heart."

Venous blood enters the right atrium through the superior vena cava and inferior vena cava, channeling blood from superior and inferior anatomic regions, respectively. The third channel delivering efferent venous blood is referred to as the coronary sinus. The left atrium contains orifices of four pulmonary veins, two of which drain blood from each lung. The left ventricular muscle is three or four times heavier than that of the right ventricle, a relationship preserved in the pressure difference between the two sides of

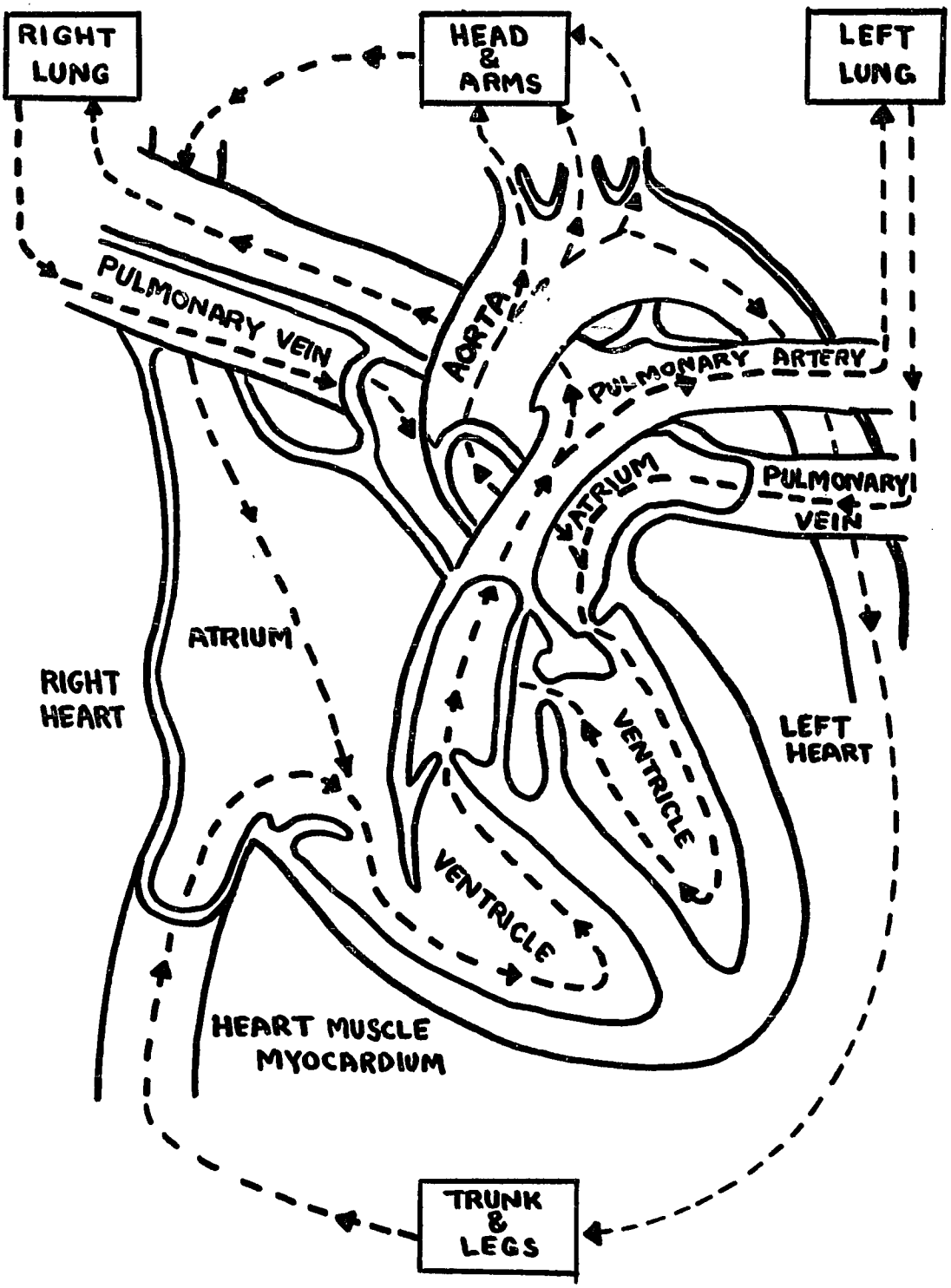


Fig. 2.1 Structure of the Heart

the heart.

As mentioned above, the two sides of the heart are separated by partitions called septa. The ventricular septum contains thick muscle continuous with the "free" walls of the left ventricle.

The four valves consist of the two inflow or atrioventricular valves, preventing atrial reentry of blood during ventricular systole and permitting ejection into the arterial truncks, and the two outflow or semilunar valves, preventing reverse suction of blood from the aorta and pulmonary artery during ventricular diastole.[1]

2.2 Electrical Function

Cardiac rhythmicity is contingent upon activity within the neural conduction system of the heart. The equivalent circuit of this system is a low-frequency capacity-resistence relaxation oscillator. This system functions independently, although it connects with the central nervous system through the autonomic nervous system. These nerve connections affect velocity of impulse formation, especially during physical or mental exertion and sleep. Two nodes originate the neutral electrical potential causing heart muscle contraction during pumping action. The upper sinoatrial node, located at the junction of the superior vena

cava and right atrium, is the principal generator of electrical current in the heart, and it is often referred to as the primary pacemaker. It generates a strong electrical impulse, then recharges for subsequent impulses at the average rate of 70 times per minute. This impulse discharges into the resistance of the atria and causes them to contract. This impulse is simultaneously conducted along the inner layer of the atrial musculature to the secondary source of electrical activity, the secondary pacemaker, or atrioventricular node, located in the lower septal right atrial wall. The pulse is rapid within the atria but decreases as it traverses the atrioventricular node. The atrioventricular node usually discharges at the rate of 50 times per minute, and its lower conduction system has further safeguard components, constituting the tertiary pacemaker: the bundle of His with its branches, and the Purkinje network with the weakest and slowest impulse-producing properties. The rate of impulse formation from the tertiary pacemaker varies from 30 to 50 times per minute (Figure 2.2).[2]

2.3 The Heart-Body System

As with most living systems the problem of the heart-body system is complicated by the initial difficulty of localization and isolation of its compon-

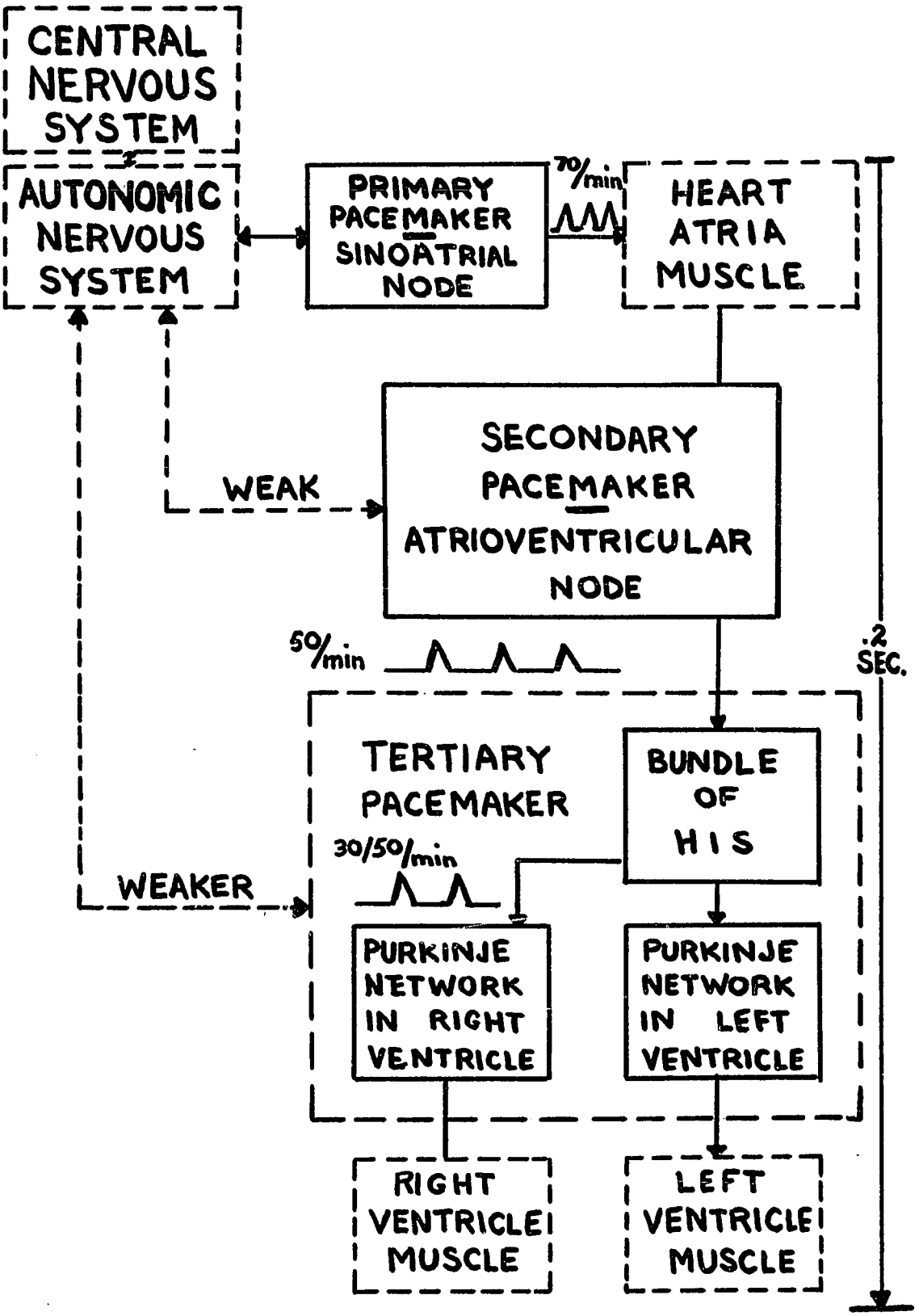


Fig. 2.2 Electrical Conduction System Flow Chart

ents. Isolating the electrical or muscular activity of the heart is insufficient for understanding its overall nervous activity. The extent of autonomic nervous system involvement is obscure, since otherwise normal hearts can be arrested by sufficient mental influence arising from hypertensive situations.[3]

The problem becomes one of identifying pertinent characteristics of the heart-body system by analyzing properties of an active or passive signal emitted by that system. The system is being characterized by a mass of complex data, and requires a method for assimilating this data. In the heart, for example, electrical activity of the brain can be differentiated from action at the sinoatrial node, although interaction may be present under certain conditions. The basis for differentiation is the set of normal actions; autonomic nervous alterations arise from stated systems or environmental influence such as exercise, sleep, or excitement. The system may therefore be subdivided: Primary, secondary, and tertiary heart function (as with the pacemaker series of sinoatrial node, atrioventricular node and bundle of His and Purkinje network) and motor, muscular, chemical, circulatory, and nervous interactions comprise interacting subsystems which emit various electrical signals.[4]

The communicative outputs, principally summarized

by the electrocardiogram, contain signals that can be classified or somehow reduced to significant information about the proper functioning of that system. Patients with clinically normal electrocardiograms have subsequently had coronary episodes, sometimes fatal. Is this a limitation of the electrocardiogram, or a limitation imposed on the diagnostician in recognizing elements because they are so subtle or covert as to escape attention? The impetus of the present work assumes the latter notion, and attempts to determine whether additional information is potentially contained in the electrocardiogram. This implies that current methods of interpretation should be augmented by computer-processed assessment of EKG patterns.

3 - OTHER CARDIOGRAPHIC ANALYSES

3.1 Standard Electrocardiogram Analysis

Significance of heart sounds were realized many years before Rene Laennec's invention of the stethoscope. Auscultation by this instrument has traditionally been the precursory heart examination, and is probably the most important routine analysis available to the general internist. If any abnormalities are detected in these sounds or other symptoms such as pain or dyspnea are present, he immediately orders an electrocardiogram, already a "routine" procedure with many clinicians. The electrocardiogram amplifies the electrical impulses originating within the heart, but only those of sufficient intensity to reach the surface of the skin.

Heart muscle consists of two types of cells, one forming contracting muscle fibers and another comprising the conducting system. Both act as small batteries, discharging a minute electrical potential during contraction and recharging automatically during relaxation, termed depolarization and repolarization, respectively. The electrical impulses from the heart are recorded from external dermal areas and emit three consecutive signals: The weak atrial signal, the intense ventricular depolarization signal, and the moderate ventricular repolarization signal. Weaker signals traver-

sing the conduction system from pacemaker to pacemaker cannot be detected externally, and at the present stage of scientific achievement, it is not feasible to insert electrodes into tissue to make more accurate measurements of these and other signals.

Referring to Figure 1.3, these signals are labeled P-Wave (1); QRS-Complex (2); and T-Wave (3). This total signal is a summation of previous signals and as such, a simplification of actual heart potentials, as the signals are distorted and combined while passing through electrolytes of other body elements. Figure 1.3 is a record of electrical potential plotted against time; the upward deflections representing impulses traveling toward the dermally attached electrode, and the downward deflections representing impulses efferent to the electrode. The P,R and T signals are positive (afferent to the electrode), and the Q and S signals are negative.

A single pair of electrodes may depict a rather unidimensional view of the heart; thus, conventional electrocardiography employs twelve electronic leads affixed to the body surface, six collecting signals from the extremities, and the remainder from various thoracic regions. The size, duration and direction of these signals indicate the extent of electrical activity throughout the cardiac musculature.[1]

Distortions in this pattern indicate heart disease or abnormality. When diseases causing hypertrophy of the left ventricle are present, for example, they alter the amount of electrical signals emitted in different directions, changing the ratio normally existing between the signals in these twelve leads. The presence of "dead muscle" in any area also appears on the electrocardiogram complex of signals. Recent injuries cause "leaks" in the voltage-producing potential of the muscles, and thus are visible in the pattern, usually as shifts in the base line away from normal. Pronounced distortions of the QRS-complexes of signals are indicated by disturbances, termed bundle-branch blocks within lower conduction system regions. Alterations of the P-wave allow diagnoses of hypertrophy or atrial enlargement.

Techniques of electrocardiographic analysis have been sufficiently codified so that a specialist is no longer required to detect many forms of heart disease. There are certain instances, however, in which the experienced diagnostician can detect weaknesses or potential defects escaping the normally-experienced eye.[2] Seven principle areas can be monitored with the EKG; heart rhythm; change in musculature of left or right ventricles, damage to heart muscle due to coronaries; lesser disturbances due to circulatory changes,as after exercise;

damage to the lower conducting system; information regarding the state of the atria; and finally, indication of gross disturbance in water and electrolyte metabolism.

3.2 Vectorcardiography

Vectorcardiography is the closest approximation to the method to be described in this study for transforming the electrocardiogram into a non-linear pattern to facilitate interpretation. The vectorcardiogram converts the linear, pulse-form pattern into a series of loops on the face of a cathode-ray oscilloscope. The generation of a vectorcardiogram is analogous to the mathematical method of handling vectors in which a summary of forces is represented by a loop projected into one plane. It is also analogous to the traditional Lisajou pattern which applies varying voltages to the x and y coordinates of the oscilloscope to produce loop patterns.

Typical vectorcardiograms are shown in Figure 3.1. This figure indicates the QRS electrical forces translated into one plane, interrupted by time signals, shown as dots representing one five-hundredth of a second. This largely depicts the forces of ventricular depolarization; provided by two connecting electrode leads,

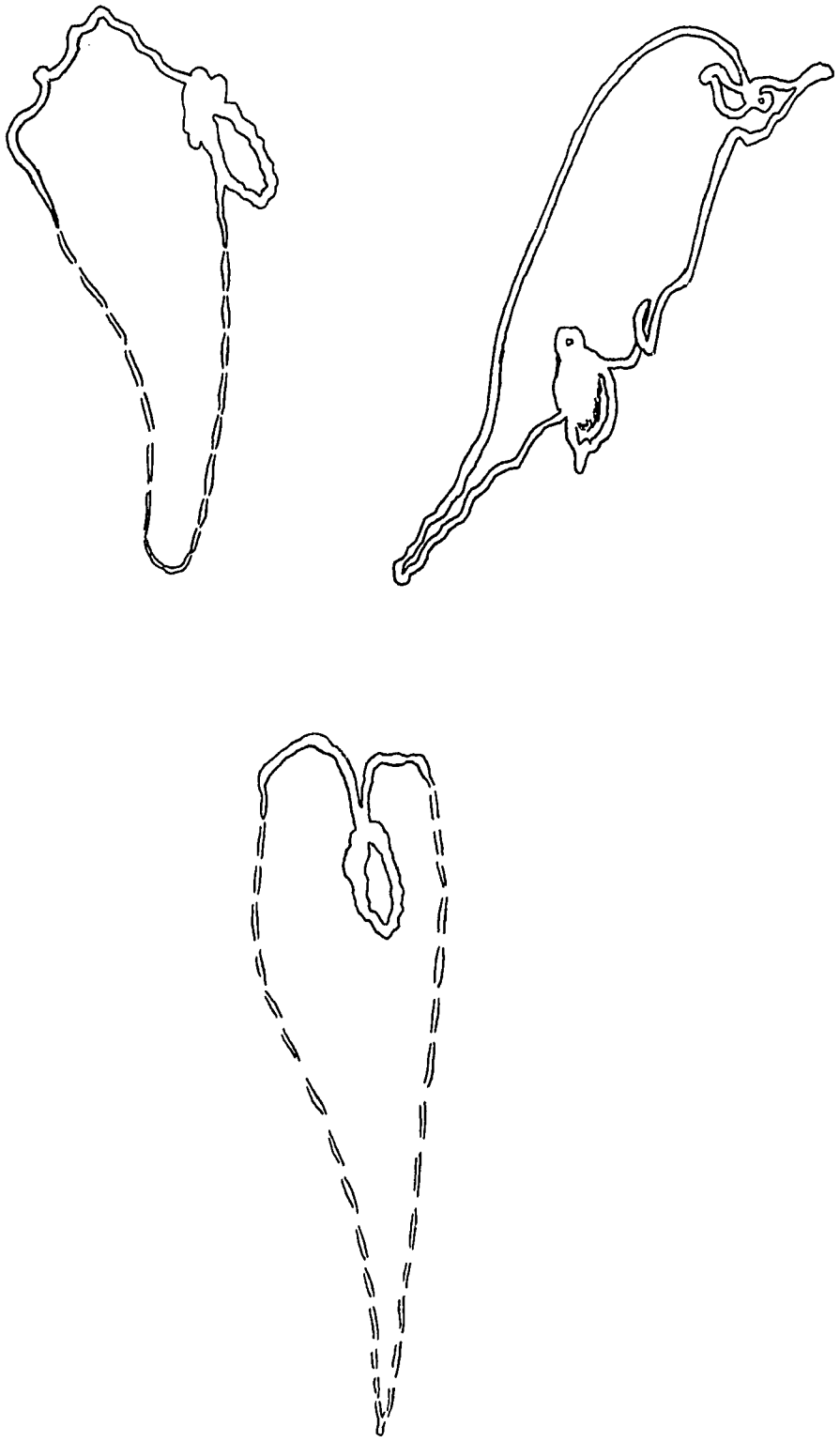


Fig. 3.1 Vectorcardiograms

fed to the x and y coordinates of an oscilloscope. It is possible to obtain three significant patterns by a combination of the leads from the heart, using the signals emitted by the electrodes at the body surface and representing the cranial-caudal, bilateral, and ventral-dorsal axes.[3]

The human body has been described as a "volume conductor" permitting electrical conduction in three dimensions, as for example, a large vessel containing physiologic saline solution or any type of ions in water. By virtue of the chemical nature of its fluids, the current generated by any region of the human body will reach other areas; the only boundary to such conduction of electricity is the body surface.[4] This theory essentially states the principle of vectorcardiography.

The vectorcardiogram (VKG) contains electrocardiographic information, but is a highly complex pattern that only vaguely follows a loop, and which varies from cycle to cycle. Theoretically the vectorcardiogram would present signals in a form more representative of actual cardiac function, as it strives to locate movement in a plane that is roughly realistic. The orthogonal combination should result in a rather comprehensive picture of the heart, summarizing patterns which

are somewhat disconnected in the conventional electrocardiogram, although in practice this is not so, principally due to considerable distortion of the electrical forces when originating at various points on the skin.

Part of the difficulty with vectorcardiography is that the pattern shape has a wide range of variability. The diagnostician must alter his technique with each patient, as each VKG reading (often each pattern of the same heart, taken at different times) presents a radically different pattern. The present method overcomes this difficulty by confining the resulting pattern to a definite form for the normal heart, and therefore providing a ready, visually obvious means for detecting abnormalities.

3.3 Ballistocardiography

Another method for measuring the heart's physiological activity is the ballistocardiograph, invented and systematically developed by Isaac Staar, literally meaning "writing of the heart's missile thrower." It has, however, been found ineffective in detecting preclinical coronary artery disease. Ballistocardiography is a measure of forces transmitted through or accumulated by the springlike fibrils composing the connective tissues of the body. The heart and blood vessels are suspended from the skeleton by this connective tissue, and tug on

the skeleton with each heartbeat. Essentially, the ballistocardiograph is a sensitive measurement of force displacement.

Simultaneous recording of ballistocardiogram and electrocardiogram may reveal marked deviations from normal without mathematical treatment of the curves. Comparison of records taken on the same subject at different times permits detection of even minor variations in function. The normal range of variation is extensive in both electrocardiograms and ballistocardiograms, and transitions to the abnormal occur in the outer margins of this range. Facility of making standardized records, to enable frequent observation, appears to be of greater importance to the clinician than quantitative analysis of the records yielded by either the electrocardiograph or ballistocardiograph.

Applied to detection of coronary artery disease, the ballistocardiogram is not generally utilized in actual diagnosis, but employed to follow changes in contractile function, thus providing valuable data on which to base prognosis. It complements the electrocardiogram, which may localize the site of the lesion. Starr[8,9] accumulated evidence of uniquely abnormal ballistic patterns in case of angina pectoris and asymptomatic healed infarction, by longterm obser-

vation of patients exhibiting moderate or latent forms of these disorders.

The ballistocardiogram usually becomes more abnormal upon slight physical exertion; however, if the injury is small and local, the response is normal.[7] The ballistocardiogram has been found to reveal a number of "false positives" in subjects over 40 years of age presenting no other evidence, current or follow-up, of coronary artery disease.[10,11] The ballistocardiogram has generally been deemed a sensitive instrument but nonspecific for case-finding in latent coronary disease.[6] However, the literature describing this method is quite relevant to the present work in assessing the value of widescale and longterm, follow-up clinical investigation of coronary artery disease.

3.4 Computer Methods of Electrocardiogram Analysis

Attempts to analyze the electrocardiogram by means of computers are best exemplified by programs such as that developed by Caceres for the Public Health Service.[16] To date this and other programs are restricted to diagnosing clinically evident or symptomatic heart disorders which would be apparent to a cardiologist who interpreted the EKG tracings. This is not surprising since the definition of rules and program content were

initially established for the computer by a group of cardiologists. Included in the program are such parameters as the amplitudes and durations of the P, Q, R, S, and T waves. "Normal" and "abnormal" patterns have been established for a large range of clinical conditions. Many pathological conditions have now been catalogued in terms of the basic parameters. In this manner computer measurement of electrocardiograms provide a basis for an automated diagnostic classification with an assessment of the morphology in much the same way as an electrocardiographer. The computer performs the same task more quickly and more consistently. However, predictive analysis, or detection of preclinical cardiac anomalies, is seldom accomplished. This is to be expected since no criteria for such detection has yet been evolved by the electrocardiographers and hence appropriate instructions for detection of pre-clinical disorders has not been furnished to the computer.

Similarly, in vectorcardiography attempts to establish arbitrary "Normal" standards by collection of many VKG's have been only partially successful. What is termed the Frank vectorcardiogram in heterogeneous models of normal men was derived from many

patterns.[12] In 1956, Frank attempted an improvement of the spatial vectorcardiographic system by trial electrocardiograms on a three dimensional homogeneous (inanimate) torso, employing seven electrodes (in contrast to the "conventional" twelve): this lesser number of leads appears to be a significant trend in computer-processed EKG analytic techniques. Frank stated that four electrodes are the minimum number theoretically required in any system of vectorcardiography, as three individual potential differences are necessary to determine the cardiac vector in three dimensions.[13] Thus, a precise representation of the three dimensional heart vector requires simultaneous voltages in three independent leads whose respective vectoral angles and magnitudes are known; the independence of the leads suggests that neither one can be epxressed or derived in terms of a linear combination of the other two.[14] However, the Frank system is linear, does not offer a limited domain of occupancy, and is more expensive and complex than the non-linear display technique described later in this work.

Another approach was recently described by Gamboa[15] et al, in which two groups of patients with normal electrocardiograms were screened for potential biven-

tricular hypertrophy. Group A consisted of controls measured by conventional scalar electrocardiograms; Group B had scalar measurements plus planar vectorcardiographic analysis. Gamboa established a t-test, arbitrarily derived for distinguishing between "normal" and biventricular hypertrophy. It was based on the assumption that "normal" or standard distributions are rarely derived from the conventional electrocardiogram. Gamboa generalized and indicated that this two-dimensional approach could be extended to a multidimensional analysis if N electrocardiogram measurements are made and taken as coordinates in an N-dimensional space. A discriminate function analysis would then be used to select from a wide range of electrocardiograms to establish the standard representative plot of either normal or pathologic hearts.

The approach developed in this dissertation achieves a transformation of the electrocardiogram, derived from only four electrodes, in such a way that it automatically generates a representation within a given pattern transformation of N-dimensional space. The limitations of the dimensions represented are flexibly met as the data presents itself, so that information gained from one set of coordinates will appear as significant only when there is a definite departure from the "normal" pattern in this dimension. The following

sections describe this approach from the mathematical standpoint, indicating how a non-linear transformation achieves the appropriate transfer of N-dimensional information in time into an arbitrary, fixed pattern. This new display is called the "omnicardiogram" and consists of an annular area bounded by two tangent circles. The transformed EKG tracings taken from subjects free from coronary artery disease inhabit the annular area. EKG tracings taken from subjects with occult coronary artery disease exhibit patterns which protrude beyond the circular boundaries.

4 - TRANSFORMATION OF COMPLEX DATA

4.1 Transformation and Natural Form

The method of transformational comparison of related forms has long been recognized as being extremely significant regarding natural forms and the human ability to recognize related systems. The method of coordinate transformation developed by Descartes was generalized by D'Arcy Thompson to apply to natural form[1]. Much as the cartographer performs different uses, taking the spherical map and transferring the information to a flat surface by various means, D'Arcy Thompson's method applies a transform to given biological forms to determine their similarity. Evolutionary shifts in mamalian skulls, for instance, can be accounted for by very specific coordinate transformations. These transformations are far from linear, which perhaps accounts for their disuse in science. Being analytically unwieldy the practical application of the transformation system correlating different species has never surpassed the curiosity stage.

Our present work of codifying functions enabling non-linear transformations similar to D'Arcy Thompson can perhaps serve the biologist in a new way. Given a mathematics which will allow the transform to be un-

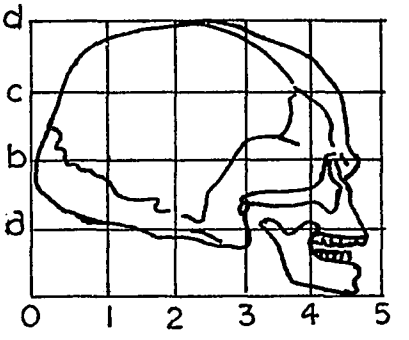
dertaken by a computer, it is conceivable that relationships existing in the evolutionary pattern of human and animal growth can be utilized for biological analysis.

Applied to the present problem, however, the transformation theory graphically illustrates a procedure by which the electrocardiogram may be subjected to non-linear transformation. Consider, for example, the way in which Thompson transforms the human skull into another skull (Fig. 4.1) The shift in relationship of the coordinates indicates how certain regions of the skull have evolved from chimpanzee to human forms.

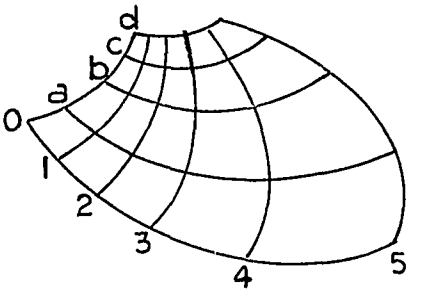
4.2 Mathematics and Form

The study of form nearly escapes the domain of mere description to enter the exact and precisely codified realm of mathematical description. This applies not only to the more regular forms such as circles, ellipses, and various transcendental functions, but also to the highly irregular and elusive forms occuring in nature. The problem is of course to arrive at the transforms and then to determine ways to implement them.[2]

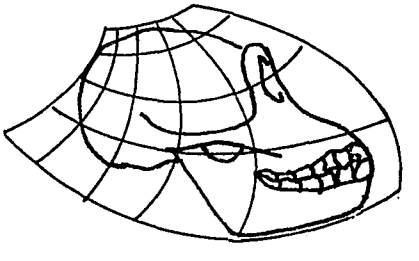
Given such a technique, Thompson's approach not only extends the imagination to provide a convenient way of looking at the evolution of natural form, but introduces an entire new science. A non-linear trans-



Human Skull



Transformation Of
Coordinates



Skull Of Chimpanzee

Fig. 4.1 Evolutionary Transform of Skull Shape

form which is not restricted to a point-by-point correspondence, and which need not be reversible, may lead from an obscure or unusual pattern to one which is more readily grasped by the human brain.

4.3 Class Recognition by Inference

Inference, for example, is carried out quite laboriously but mysteriously by the human mind, and it essentially involves the transformation of seemingly inexplicable and amorphous data into form and theory. That a non-linear relationship exists does not prevent the formal transformation from having meaning. To establish a means for classification we need not preserve all relationships, provided the transformation works. That is, a traditional conformal transformation insists on preserving certain relationships from the one coordinate frame to the other. Angles and their sense must be preserved, and a one-to-one mapping must be invariant. A new approach, to be effective, must allow a complete topological reorientation of related classes without obscuring the prime relationship by which is recognized its uniqueness. The loss of relations partially clarify form, although they may not allow us to regain the original data.[1,3,4]

5 - LINEAR AND NON LINEAR TRANSFORMATIONS

5.1 Pattern Recognition and Coordinate Transformation

Even sophisticated advances in computer technology have not resolved certain aspects of evaluating electrocardiograms. This is particularly true in occult coronary artery disease where the electrocardiogram appears to be within normal bounds even to the experienced cardiologist. Hence, the problem appears to be two-fold. One cannot clearly define it in mathematical parlance nor can one give specific instructions to the computer which states how a solution should be reached.

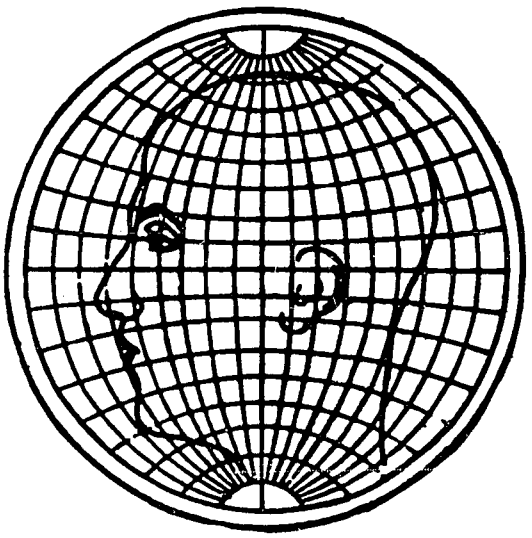
In short this class of problem seems to be an ideal vehicle in which one might employ coordinate transformation in an attempt to optimize pattern recognition between two discrete classes of data.

5.2 Graphical Representation of Data

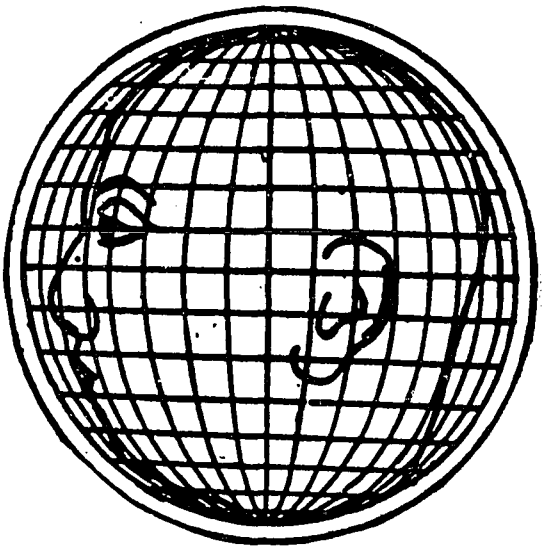
The graphical representation of all forms of data is usually confined to either of two conventional sets of coordinate systems, cartesian and polar. With the exception of the logarithmic type, the coordinates in either of these systems are normally chosen to be linear, although there is no logically compelling reason

for uniformly employing such linear coordinate systems. A given set of functionally related data could be plotted in any of a large variety of coordinate systems, and the resulting patterns would have widely different representations. Some of these representations would be quite complex and difficult to identify. In other suitably chosen coordinate systems,[3] the same set of data might appear as well-behaved patterns and would be correspondingly easier to classify. The strikingly different visual effects often resulting from seemingly minor perturbations in coordinate structure are exhibited in Figure 5.1.

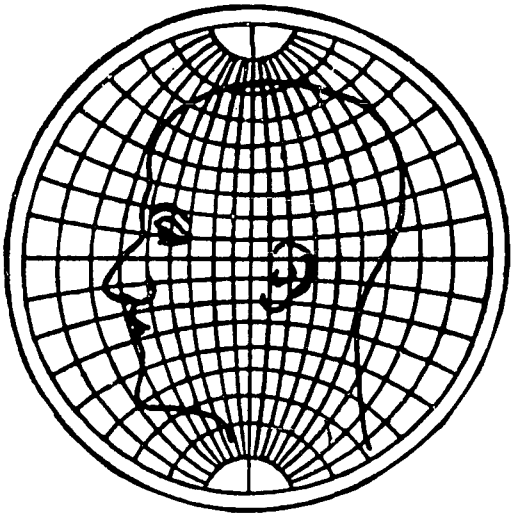
Returning to the electrocardiogram tracing, it is important to recognize that the shape of this signal is not defined until one selects the coordinates system in which this signal is to be displayed. If we use the traditional cartesian set of coordinates to display amplitude versus time, we obtain the pattern as typified in Figure 5.2. Now this conventional amplitude-time tracing can be transformed to the irregular closed curve as in Figure 5.3. Then this curve can be transformed non-linearly into the easily recognizable pattern shown in Figure 5.4. Of course, the coordinates are now highly irregular, but we are only interested in rendering the basic data more accessible to classification.



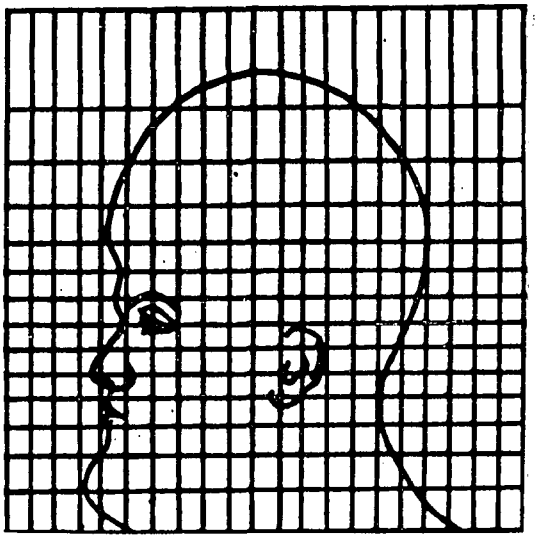
Man's head drawn on globular projection.



Man's head plotted on orthographic projection.



Man's head plotted on stereographic projection.



Man's head plotted on Mercator projection.

Fig. 5.1 Effect of Coordinate Structure Perturbations

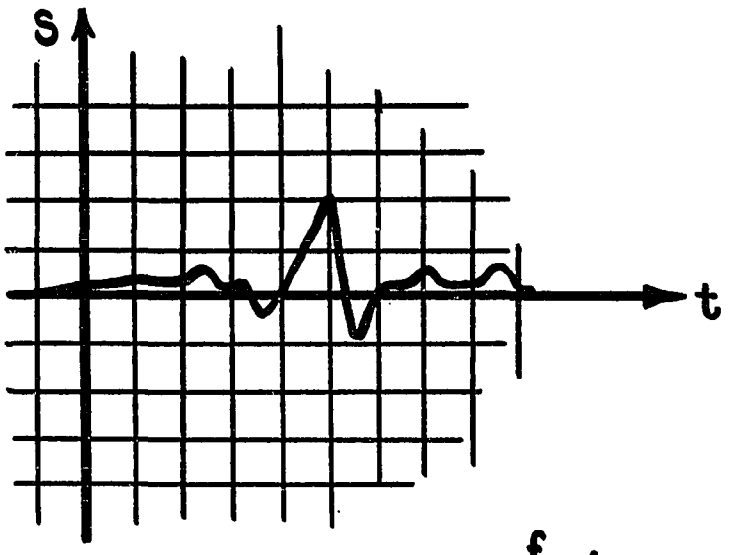


Fig. 5.2

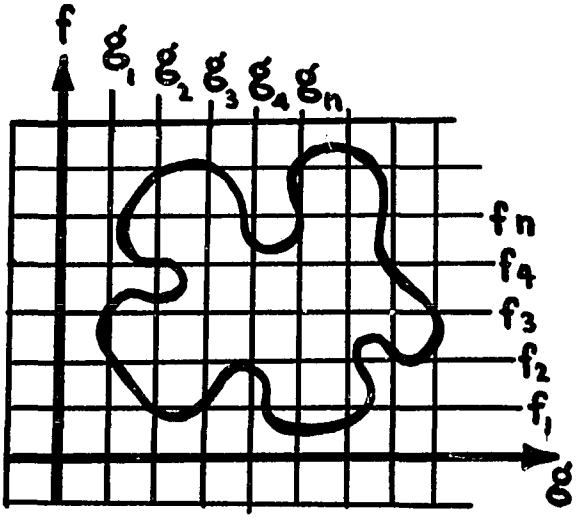


Fig. 5.3

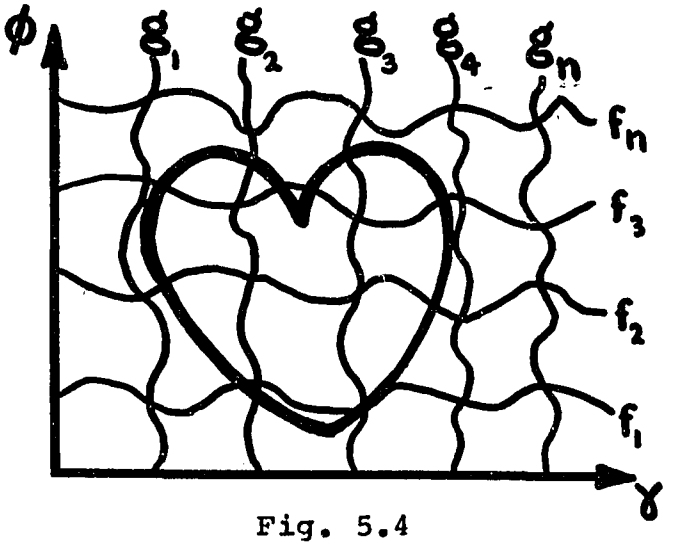


Fig. 5.4

Transform of conventional EKG signal to a non-linear coordinate system

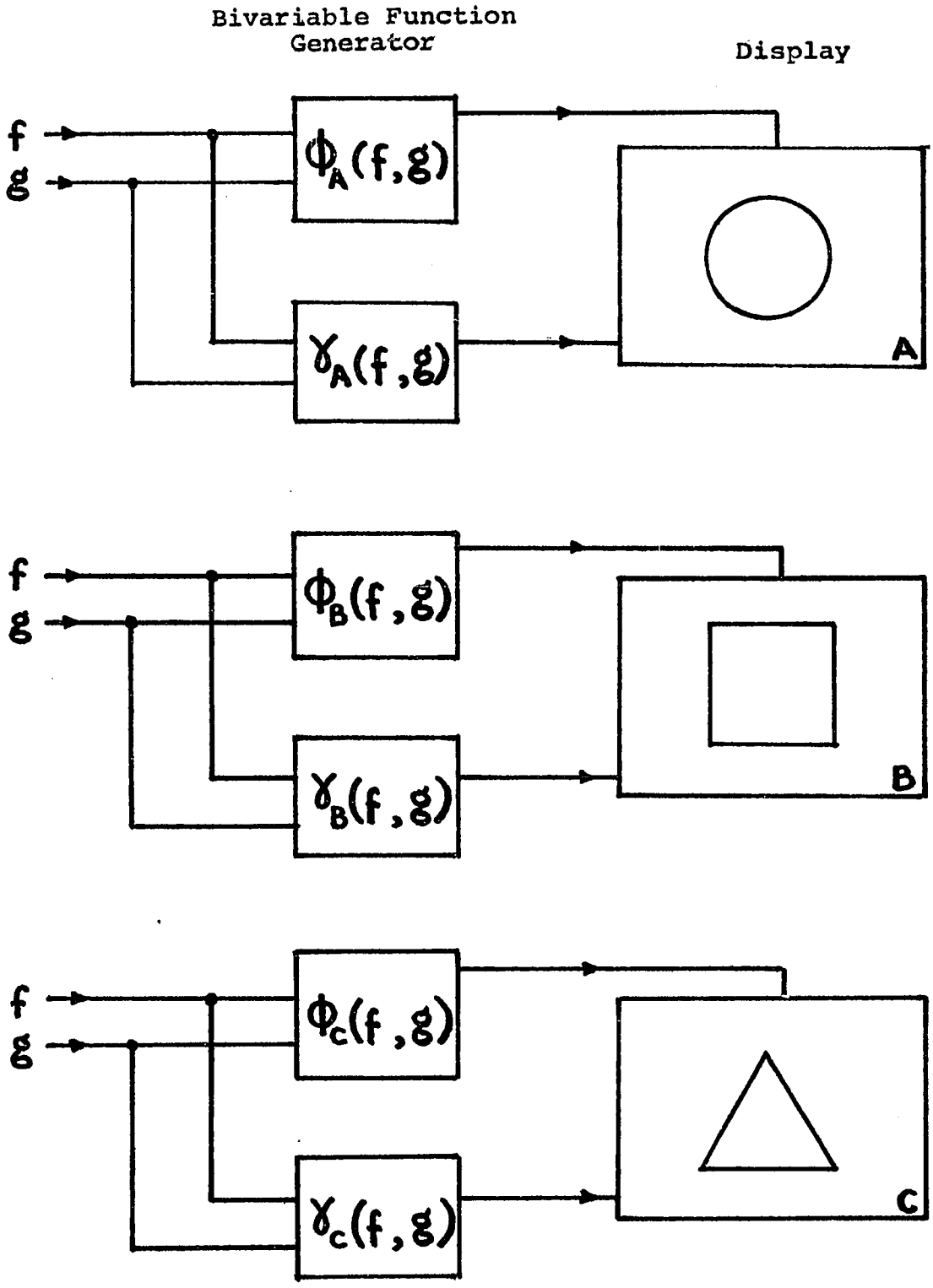


Fig. 5.5 Effects of various transformations on final shape of display

The tracing in Figure 5.2 could have been transformed to any preferential shape, as suggested in Figure 5.5, by choice of a suitable transformation. The proper selection of such transformation function is not always obvious, however.

5.3 Linear Transformations and Conformal Mapping

Real functions of real variables, $y = f(x)$, form the general class of experimentally derived data. As we have seen, the relationship can be exhibited graphically by plotting corresponding values as rectangular coordinates of points in the xy plane. When the variables are complex, the graphical representation of functions is somewhat more involved. For example, if $w = f(z)$ where $z = x+iy$, and $w = u+iv$, then each of the complex variables w and z are represented geometrically by points in the complex plane. Simultaneous representation would require four-dimensional space with orthogonal axes along which the four real variables, x,y,u, and v could be measured. Instead, the information is displayed in two separate planes referred to as the z plane and the w plane. Hence, in a domain defined by the function f, there corresponds to each point (x,y) in the z plane, a point (u,v) in the w plane. That is, the function f(z) maps points in the z plane into the w plane so that

$u = u(x,y)$ and $v = v(x,y)$. This is commonly referred to as mapping or transformation of points between two planes by a suitable function.

In many areas of engineering and physics, an effective but limited form of the general mapping technique is employed, known as conformal mapping. The conditions which limit its use require that the transformation be analytic, i.e., satisfy the Cauchy-Riemann conditions, namely

$$\frac{\partial u}{\partial x} = \frac{\partial v}{\partial y} \qquad \text{and} \qquad \frac{\partial v}{\partial x} = -\frac{\partial u}{\partial y}$$

The mapping of corresponding curves and regions in the two planes usually provides more information about the function than the mapping of individual points. As an example, consider the transformation $w = f(z) = e^z$. As shown in figures 5.6 and 5.7

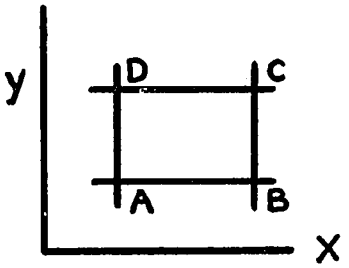


Fig. 5.6

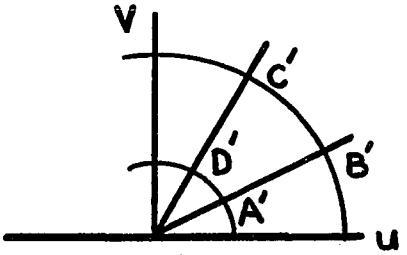


Fig. 5.7

This transformation maps the lines $x = c$ into the circles $\rho = \exp c$, and the lines $y = c$ into the rays $\phi = c$. The rectangular region ABCD in the z plane maps into the region A'B'C'C' bounded by circles and

rays in the w plane.

It appears that a given curve plotted in the z plane may be transformed into an entirely different shape in the w plane. Indeed, proper choice of the conformal transformation is frequently employed to effect a significant change in the original curve so that a simplification of either the geometry or mathematical treatment may be achieved. As mentioned, however, there are several serious restrictions which limit the use of conformal mapping. An essential characteristic of conformal mapping is the fact that the angles and their sense between curves in the z plane are preserved when they appear in the w plane, and in general, a one-to-one correspondence between mapped points is also preserved.

Another useful tool is the linear matrix transformation, such as $[x']=[R][x]$, which accomplishes a rotation and possible rescaling of $[x]$. The properties which make such transformation desirable in certain cases are homogeneity

$$[R][\alpha x]=\alpha[R][x]$$

and additivity

$$[R][x_1+x_2]=[R][x_1]+[R][x_2]$$

In a linear transformation, the inverse of $[R]$, that is $[R]^{-1}$, exists such that $[R]^{-1}[R]=I$. Hence, the reverse transformation $[x]=[R]^{-1}[x']$ may always be

achieved. The elements of the transformation matrix R are of course independent of the vector undergoing transformation.

While the preceding types of transformations are useful in certain cases, there are problems which exhibit such complicated representations in their original domain, or which have sharp discontinuities, that it is difficult to devise a suitably useful transformation if the Cauchy-Riemann conditions are to be obeyed.

Yet, many problems of processing complex signals are solved with ease by humans in their daily lives. We will therefore examine a more general class of transformation which may be visually more meaningful, such as

$$X_i = f_i(s_o, \ldots s_n, t_o, \ldots, t_n) \qquad (1)$$

$$Y_i = g_i(s_o, \ldots s_n, t_o, \ldots, t_n) \qquad (2)$$

where the new coordinates X_i, Y_i, each depend on the entire set of original data points s_i, t_i. In terms of electrocardiograms, s represents amplitude and t represents time. A useful property would be such that the functions f_i and g_i behave in such a way that a selective amplification is achieved for important portions of the original data set while less useful information is selectively attenuated. A method to achieve these properties will be explored in the sections which

follow.

We will allow these transformations to be non-linear, non-unique, non-reversible, and free from restrictions imposed by the Cauchy-Reimann conditions.

A transformation of the type we are seeking might also be expressed as a non-linear matrix where the elements of the matrix R are functions of the s and t coordinates.

5.4 Enlarging the Class of Transformations

Achieving success with the linear methods explored thus far is not readily attainable. The degree of success achieved by linear methods depends on the choice of the transformation and the nature of the classes to be recognized. At present there is no simple unifying theory in non-linear mathematics analagous to vector spaces and operatives for linear mathematics.[2] The classical methods of mathematical physics have often used linearization and we have become accustomed to idealizations of natural phenomena which are frequently made for the sake of arriving at explicit solutions. One type of non-linear transformation which has been considered may be likened to the operation of stretching and compressing a rubber sheet to bring members of a set closer together. In this way N-dimensional space may be locally compressed or ex-

panded. The transformation may take the form of a polynomial expression and if the coefficients of the polynomial are not restricted, the space may "fold" and a multivariate transformation may result.[1]

The problem is to find a non-linear transformation which somehow constrains a set of points from a given class to a new domain which is visually different from some other class even though both classes were imperceptibly different from each other in the initial domain.

5.5 Data Classification and Visual Perceptibility

The central problem of data classification is now defined as the problem of developing functions from sets of finite samples of their respective classes so that these functions will re-structure the original samples into separate regions, each containing the sample points belonging to the respective class. Although the system may be readily generalized to the multi-dimensional case, to facilitate demonstration of the usefulness of the method, the general case of ordered triplets is chosen say $(x_i, y_{ij}, \emptyset_j)$, $i = o,\ldots,q$ $j = o,\ldots,p$, such that they represent $(q+1)$ $(p+1)$ values of x, y, and $\emptyset$, where $y = f(x,\emptyset)$ is known only at discrete points stated. This unknown function may be represented by a polynomial of the following form:

$$Z(x,\phi_j) = \sum_{i=0}^{q} \alpha_i(x) f(x_i, \phi_j) \qquad (3)$$

where the α_i's are polynomials of the q^{th} degree in x, chosen such that the conditions

$$Z(x_i, \phi_j) = f(x_i, \phi_j), \quad \begin{matrix} i=0,\ldots,q \\ j=0,\ldots,p \end{matrix} \qquad (4)$$

are satisfied. Notice that if $\alpha_i(x) = \delta_{\ell i}$, where δ is the Kroneker delta,

$$\alpha_i(x) = \frac{\prod_{k=0}^{q} (x-x_k)}{(x - x_i) \prod_{k=0}^{q} (x_i - x_k)} \qquad (5)$$

where

$$\delta_{\ell i} = \begin{matrix} 0 \text{ if } \ell \neq i \\ 1 \text{ if } \ell = i \end{matrix} \qquad (6)$$

Applying the same procedure to the variable ϕ, we obtain

$$Z(x, \phi) = \sum_{j=0}^{p} \sum_{i=0}^{q} \alpha_i(x)\ \beta_j(\phi) f(x_i, \phi_j) \qquad (7)$$

where $\beta_j(\phi)$ is defined in the same manner as (6). Evaluating the summation over i, we obtain

$$Z(x, \phi) = \sum_{j=0}^{p} \beta_j(\phi) F(x, \phi_j) \qquad (8)$$

Now suppose there are given n relations in the new domain X,Y.

$$Y = f(y, \phi_j), \quad j = 1,\ldots,n \qquad (9)$$

It follows that the value of Y at any point:

$$Y = f\ (y,\ \phi_j)\Big|_{y=c} \qquad \phi_1 \leq \phi_j \leq \phi_n \tag{10}$$

may be represented by a power series of the form

$$Y\ (c,\ \phi_j) = \sum_{r=1}^{m} K_r \phi_j^{(r)} \tag{11}$$

Suppose that there are also n given relations,

$$X = g\ (x,\ \phi_j), \qquad j = 1, \cdots, n \tag{12}$$

By the same method

$$X(c',\ \phi_j) = \sum_{n=1}^{m} K_n'\ \phi_j^{(r)} \tag{13}$$

Thus, given the coordinate transformations

$$\left.\begin{aligned} Y &= f\ (y,\ \phi_r) \qquad (14)\\ X &= g\ (x,\ \phi_r) \qquad (15)\end{aligned}\right\} \quad r = 1, \cdots, m$$

The required reorientation mapping is defined and complete.

5.6 A Clustering Transformation

Let us assume that each coordinate of the original data set undergoes a continuous transformation given by the generalized mapping equations of (14) and (15) which maps the origin into itself and which also obeys the following constraints (see Figure 5.8).

X = 0 starts at zero and increases monotonically to 2π.

Y = ρ starts at zero, ends at zero and is constrained between the limits of zero and

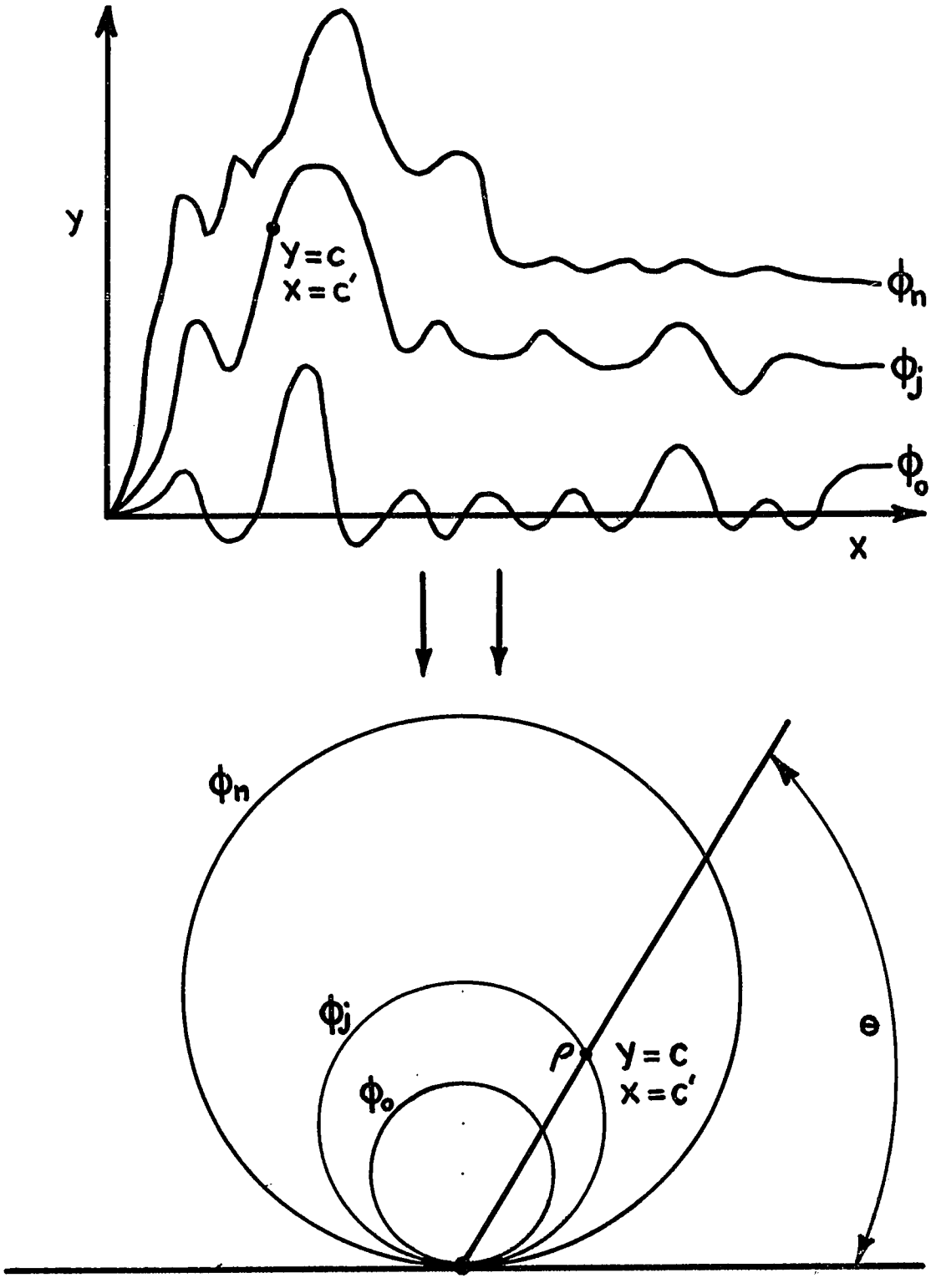


Fig. 5.8 A Clustering Separation

some arbitrary constant.

Now in (8) let $F(x, \phi_j)$ be a set of continuous functions of the variable x, with $F_j(x)$ corresponding to the p values of ϕ_j.

For simplicity, we will choose the β_j terms to be defined as follows

$$\begin{aligned} \beta_0(\phi) &= 1 \\ \beta_1(\phi) &= (\phi - \phi_0) \\ \beta_2(\phi) &= (\phi - \phi_0)\ (\phi - \phi_1) \\ \beta_p(\phi) &= \prod_{r=0}^{p-1} (\phi - \phi_r) \end{aligned} \tag{16}$$

Hence $Z(x,\phi) = F_0(x) + (\phi - \phi_0)F_1(x) + \ldots$

$$\prod_{r=0}^{p-1} (\phi - \phi_r)F_p(x) \tag{17}$$

If the surface for Z can be adequately indicated by three reference curves, we would obtain

$$\begin{aligned} Z(x,\phi) = F_0(x) &+ (\phi - \phi_0)F_1(x) + \\ &(\phi - \phi_0)(\phi - \phi_1)F_2(x) \end{aligned} \tag{18}$$

The weighting functions F(x), are easily shown to be

$$F_0(x) = y(x,\phi_0) \tag{19}$$

$$F_1(x) = \frac{y(x,\phi_1) - F_0(x)}{(\phi_1-\phi_0)} \tag{20}$$

$$F_2(x) = \frac{y(x,\phi_2) - F_0(x)}{(\phi_2-\phi_0)(\phi_2-\phi_1)} - \frac{F_1(x)}{(\phi_2-\phi_1)} \tag{21}$$

Thus any three specified curves in the family $y(x,\phi)$ can be represented precisely.

In practice $y(x,\phi_0)$ and $y(x,\phi_2)$ are selected to be two extremes of the envelope of curves and $y(x,\phi)$ is some intermediate curve.

For values of ϕ different from ϕ_0, ϕ_1, or ϕ_2, a parabolic interpolation is obtained. This result is seen from substitution of equations (19), (20), and (21) into (18). Evaluating the result at x=c, gives

$$[Z(x,\phi)]_{x=c} = k_1\phi^2 + k_2\phi + k_3$$

5.7 Application To Additional Reference Curves

If more than three reference curves are employed, the number of terms in equation (18) may be extended. However, the degree of the polynomial expression representing the interpolated function also increases, thereby causing the function Z to behave erratically in the regions situated away from the reference curves.

For a five-curve approximation to the function it is advantageous to write:

$$\left.\begin{aligned} &\text{for } \phi \leq \phi_2 \\ &\quad Z(x,\phi) = F_2(x) + F_1(x)(\phi - \phi_2) + F_0(x)(\phi - \phi_2)(\phi - \phi_1) \\ &\text{and for } \phi \geq \phi_2 \\ &\quad Z(x,\phi) = F_2(x) + F_3(x)(\phi - \phi_2) + F_4(x)(\phi - \phi_2\ (\phi - \phi_3) \end{aligned}\right\} \quad (22)$$

This insures that each of the five reference curves $y(x,\phi_0)$, $y(x,\phi_1)$,..., $y(x,\phi_4)$ will be generated precis-

ely, and for intermediate values of ϕ a satisfactory second degree interpolation will be obtained.

The values of F(x) which are required are:

$$F_2(x) = y(x,\phi_2) \tag{23}$$

$$F_1(x) = \frac{y(x,\phi_1) - F_2(x)}{(\phi_1 - \phi_2)} \tag{24}$$

$$F_0(x) = \frac{y(x,\phi_0) - F_2(x)}{(\phi_0 - \phi_2)(\phi_0 - \phi_1)} - \frac{F_2(x)}{(\phi_0 - \phi_1)} \tag{25}$$

$$F_3(x) = \frac{y(x,\phi_3) - F_2(x)}{(\phi_3 - \phi_2)} \tag{26}$$

$$F_4(x) = \frac{y(x,\phi_4) - F_2(x)}{(\phi_4 - \phi_2)(\phi_4 - \phi_3)} - \frac{F_2(x)}{(\phi_4 - \phi_3)} \tag{27}$$

In practice the procedure is repeated piecewise until all reference curves are exhausted.

5.8 The Case of Ordered Quadruplets

We have already developed the method for generating a function of two arguments based on a set of ordered triplets. We seek now to extend the method to include a function of three variables. Although this is more difficult, it is not a formidable task.

This is equivalent to generating a function $y=y(x,\phi,\gamma)$ where only a discrete set of ordered quadruplets are defined.

The preceding equation may be represented as

$$y(x,\emptyset)_{\gamma=\gamma_0},\ y(x,\emptyset)_{\gamma=\gamma_1},\ \ldots,\ y(x,\emptyset)_{\gamma=\gamma_m} \tag{28}$$

We define a new function $Z(x,\emptyset,\gamma)$ as

$$\begin{aligned} Z(x,\emptyset,\gamma) = {} & \psi_0(x,\emptyset) + \psi_1(x,\emptyset)(\gamma-\gamma_0) \\ & + \psi_2(x,\emptyset)(\gamma-\gamma_0)(\gamma-\gamma_1) + \ldots \\ & + \prod_{r=0}^{n-1} \psi_n(x,\emptyset)(\gamma-\gamma_r) \end{aligned} \tag{29}$$

Now each of the terms in equation (29) represents a case of ordered triplets which can be generated in the manner already described.

Hence the problem of generating an arbitrary function of three variables may be transformed to that of generating it by a series of bi-variable functions. Again the question arises as to the number of terms to be retained in (29). The choice depends on such factors as how well the function behaves in the area of interest and on the degree of precision which must be attained in the separation of classes.

In general three terms will prove to be adequate so that we have

$$\begin{aligned} Z(x,\emptyset,\gamma) = {} & \psi_0(x,\emptyset) + \psi_1(x,\emptyset)(\gamma-\gamma_1) \\ & + \psi_2(x,\emptyset)(\gamma-\gamma_0)(\gamma-\gamma_1) \end{aligned} \tag{30}$$

The ordered triplets or bivariable functions to be generated are:

$$\Psi_0(x,\emptyset) = y(x,\emptyset)_{\gamma=\gamma_0} \tag{31}$$

$$\Psi_1(x,\emptyset) = \frac{y(x,\emptyset)_{\gamma=\gamma_1} - \Psi_0(x,\emptyset)}{(\gamma_1 - \gamma_0)} \tag{32}$$

$$\Psi_2(x,\emptyset) = \frac{y(x,\emptyset)_{\gamma=\gamma_2} - \Psi_0(x,\emptyset)}{(\gamma_2 - \gamma_0)\ (\gamma_2 - \gamma_1)} - \frac{\Psi_1(x,\emptyset)}{(\gamma_2 - \gamma_1)} \tag{33}$$

The flow chart for the computer implementation is presented in appendix A.

6 - PRESENTATION OF RESULTS

6.1 Groups Studied and Methods of Study

The non-linear transformation for the interpretation of EKG tracings has been tested on a group of patients referred to Dr. R.W. Brancato, Director of Cardiac Catheterization Laboratories at St. Michael's Medical Center, Newark, N.J. These patients had been referred by their attending physicians because of certain findings suggestive of coronary artery disease. Of these findings, some of the more acute symptoms were characterized as atypical chest pain or discomfort, cardiomegally of unknown etiology, angina pectoris, and dyspnea on physical exertion.

As summarized in Table I, a total of 228 patient histories were examined. In most of the cases studied, the resting EKG was entirely within normal limits, and the presence or absence of coronary artery disease was established by cardiac catheterization with opaque visualization cineangiography.

Following cineangiography, a patient was classified by the examining physician as "normal" if no significant lesions in the coronary arteries were detected. If cineangiography revealed the presence of one or more significant lesions, the patient was classified in the category of coronary artery disease (CAD).

We intentionally restricted the major portion of the study to those patients displaying normal conventional EKG

TABLE I

SUMMARY OF DATA FROM ST. MICHAEL'S MEDICAL CENTER

Cine Diagnosis	Assessment of EKG	Number of Cases	Digitized	
			1 Cycle	2 Cycles
Normal	Normal	59	21	33
CAD	Normal	38	24	14
Normal	Not. Avail.	14	1	1
CAD	Not. Avail.	10	0	0
Normal	Abnormal	19	7	2
CAD	Abnormal	56	6	1
Incomplete	-	32	0	0
		228	59	51

Note: CAD = Coronary Artery Disease

tracings throughout all twelve standard leads to ascertain the value of our non-linear transformation in detecting occult coronary artery disease. However, instead of employing all twelve standard leads we transformed only two limb and two precordial leads. Initially only one cycle or "complex" of each of these four leads was digitized and processed on the computer. Later in the study two cycles for each of the four leads were digitized rather than one. Employing the non-linear transformations previously described, composite templates for "normal" subjects were constructed by the computer separately for EKG leads I, II, V_4 and V_6. The "normal" template was synthesized in the manner described in section 5.6, from EKG tracings of 24 candidates who were randomly chosen from a group of 59 patients with normal cineangiograms. These are identified in Table III by a double asterisk (**) next to the case number. In structuring the composite template (OCG) for each of the four leads, several of the transformed leads exhibited bizarre behavior and were excluded from use in forming the template boundaries. These leads are identified by "O" the "out-of-bound"symbol in Table III. This procedure is accounted for in the statistical treatment in section 6.4.

In every case the cycles in any EKG lead were randomly selected. Digitization of EKG tracings to computer punched cards was accomplished by employing a semiautomatic Gerber analog to digital converter.

6.2 Results and Discussion

Table II summarizes our results based on 38 cases displaying normal EKG's but with coronary artery disease as established by cineangiography. In this table, the letter "I" signified that the transformed lead remained within the established bounds of its template or omnicardiogram. The letter "O" signifies that the transformed lead protruded beyond the annular space of its template. If two or more leads transgressed the bounds for a given case, this was scored as a "success", or positive finding, in detection of coronary artery disease, and is designated by the letter "S" in Table II. If less than two leads deviated outside, this was regarded as a "failure" and designated "F". Where two entries appear in a given column for a given case, they represent the respective results of separate cycles. These double entries will be considered later.

There were 24 CAD cases where one cycle per lead had been digitized. Among this group there were 3 false negatives giving a detection rate for this sample of 21/24 = 88%. Table II also lists 14 CAD cases where two cycles were digitized for each lead. The rationale for processing two cycles rather than one is based on the physiological presumption that subtle pathological conditions may reveal themselves sporadically from cycle to cycle.

TABLE II

RESULTS OF NON-LINEAR TRANSFORMATION OF NORMAL EKG TRACINGS OBTAINED FROM CAD PATIENTS

Case No.	EKG Lead I	II	V4	V6	OCG Diagnosis	Score
142	I	O	O	O	CAD	S
148	I	O	O	I	CAD	S
157	O	I	O	I	CAD	S
164	O	O	O	O	CAD	S
173	O	O	I	O	CAD	S
174	O	O	O	O	CAD	S
175	O	O	O	O	CAD	S
204	O	O	I	O	CAD	S
205	O	O	O	O	CAD	S
239	O	O	O	O	CAD	S
245	I	I	I	I	Normal	F
392	O	I	I	I	Normal	F
448	O	O	O	I	CAD	S
449	I	O	I	O	CAD	S
457	O	O	I	I	CAD	S
468	O	O	O	I	CAD	S
469	O	O	I	O	CAD	S
473	I	I	I	I	Normal	F
478	O	O	I	I	CAD	S
483	O	O	O	O	CAD	S
497	I	O	O	O	CAD	S
500	I	O	O	O	CAD	S
503	I	O	I	O	CAD	S
509	O	I	O	I	CAD	S
308	OI	OO	OO	OO*	CAD	S
479	OO*	OO*	II*	OI*	CAD	S
519	II	OO*	OO	OI	CAD	S
529	OO	OO	OO	OI	CAD	S

*Single Cycle Digitized Twice

TABLE II Continued

Case No.	EKG Lead I	II	V4	V6	OCG Diagnosis	Score
538	OO	OO	IO	II	CAD	S
547	OO	II	II	OI	CAD	S
555	II	OO	II	OI	CAD	S
556	OO	IO	OO	OO	CAD	S
563	OO	II	II	OO	CAD	S
579	II	II	II	II	Normal	F
582	II	II	OO	OO	CAD	S
583	OO	OO	II	II*	CAD	S
610	II	II	IO	II	Normal	F
629	OO	OO	OI	II	CAD	S

* Single Cycle Digitized Twice

Where two cycles per lead were digitized, that case has eight entries (two per lead). If three or more cycles exceeded the bounds of their template, that case was classified as CAD and scored as a success "S". Based on two false negatives in a group of 14 CAD cases, the detection rate for this small sample is 12/14 = 86%. As a composite group of 38 CAD cases made up of EKG's digitized on either a single cycle basis or a double cycle basis, the detection rate is 33/38 = 87%.

Table III presents the results of the non-linear transformation of EKG's derived from patients with normal appearing cineangiogram as interpreted by the examining physician.

It should be emphasized at this point that the use of the word "normal" is perhaps misleading from a clinical standpoint. In general, truly "normal" subjects are not referred to the cardiac catheterization laboratory by their attending physicians. As previously mentioned, all of these patients had exhibited certain symptoms suggestive of coronary artery disease. While the presence of significant coronary artery disease was denied on the basis of cineangiography, we cannot exclude the presence of other systemic diseases without an exhaustive study of the entire medical history. This will be discussed further.

TABLE III

RESULTS OF NON-LINEAR TRANSFORMATION OF NORMAL EKG TRACINGS OBTAINED FROM NORMAL SUBJECTS

Case No.	EKG Lead I	II	V4	V6	OCG Diagnosis	Score
144**	O	O	O	O	CAD	F
150**	I	I	I	I	Normal	S
155**	I	O	I	I	Normal	S
163**	I	I	I	I	Normal	S
165**	I	I	I	I	Normal	S
172**	I	I	I	I	Normal	S
177**	O	O	O	O	CAD	F
179	I	I	I	I	Normal	S
187	O	O	O	O	CAD	F
193	I	O	I	I	Normal	S
196**	O	O	I	I	CAD	F
246**	I	I	I	I	Normal	S
408**	I	I	I	I	Normal	S
433**	I	I	I	O	Normal	S
435	I	I	I	I	Normal	S
436	I	I	I	I	Normal	S
444	I	I	O	I	Normal	S
470**	O	I	I	I	Normal	S
488**	I	I	I	I	Normal	S
495**	I	I	I	I	Normal	S
512	O	O	I	O	CAD	F
185	OO	OO	II*	IO*	CAD	F
197	OO	II	II	II*	Normal	S
215	ØØ	IO	IO	OO	CAD	F
237**	II	II*	II	II	Normal	S
238**	OI	OO	OO*	OI	CAD	F

* Single Cycle Digitized Twice

** Member of Group Used to Generate "Normal" Template

TABLE III CONT'D

RESULTS OF NON-LINEAR TRANSFORMATION OF NORMAL EKG TRACINGS OBTAINED FROM NORMAL SUBJECTS

Case No.	EKG Lead I	II	V4	V6	OCG Diagnosis	Score
312	OI	II	II	II	Normal	S
313	II	OO	II*	II*	Normal	S
412**	II	OO*	II*	II*	Normal	S
414**	II	II	II	II	Normal	S
415**	IO	OO	OI	II	CAD	F
417**	II	II	II	II*	Normal	S
421	II	II*	II	II*	Normal	S
482**	II	OO	II	II	Normal	S
485**	II	II	II	II	Normal	S
491**	II	OO	OO	II	CAD	F
517**	OO	II	II	II	Normal	S
530	II*	II	II	II	Normal	S
533	II	II	II	II	Normal	S
534	II	OO	II	II	Normal	S
552	II	II	II	II	Normal	S
558	OO	OO	OO	OO	CAD	F
567	II	OO	II	II	Normal	S
570	II	OI	II	II	Normal	S
571	II	II	IO	OI	Normal	S
573	II	OO	II	II*	Normal	S
584	OI	OI	II	II	Normal	S
587	II	II	II	II	Normal	S
589	OO	OI	OO	IO	CAD	F
592	II	II	OI	II	Normal	S
599	II	OI	II	II	Normal	S
622	IO	II	II	II	Normal	S
625	II	OO	II	II	Normal	S
630	II	IO	OI	II	Normal	S

* Single Cycle Digitized Twice

** Member of group used to generate "Normal" template

Of the 54 "normal" cases presented in Table III, 24 of them have already been used to synthesize the "normal" template. These cases are designated by the double asterisk (**) and will not be employed to test the validity of the template. Of the remaining 30 "normal" cases, six were classified as CAD based on the same criteria applied to their transformed leads as previously discussed.

These results appear to yield a false positive rate of 6/30 = 20%.

6.3 Discussion of False Positives

Table IV presents some interesting findings on 11 cineangiogram "normal" cases which were classified as CAD on the basis of the "omnicardiogram" findings and are therefore regarded as false positives. All of these medical records were subsequently diligently studied. From the records it was learned that case #144 was suffering from jaundice and gallbladder disease. Case #177 had an active duodenal ulcer, cholecystitis, and had a myocardial infarction four years ago.

Indeed, out of the 11 cases which could be researched and presented in Table IV, only two were found to be free from evidence of pathology. These results, while far from conclusive, begin to suggest the possibility that subtle changes may be present in "normal"

TABLE IV

SUMMARY OF ELEVEN CASES WITH NEGATIVE CINEANGEOGRAPHIC FINDINGS YIELDING FALSE OCG POSITIVES

Case No.	Medical Findings
144	jaundice; gall bladder disease
177	active duodenal ulcer; cholecystitis; myocardial infarction 4 years ago
185	considered borderline normal
187	on digitalis; anemia; incipient diabetes
196	myocardial infarction 2 years ago
215	chronic cholecystitis with cholelithiasis
238	no evidence of pathology
415	on nitroglycerine for last 6 months; high cholesteral (312); previous EKG shows first degree AV block
491	no evidence of pathology
512	pronounced systolic narrowing of mid anterior descending artery
558	active duodenal ulcer

looking electrocardiograms which are somehow coupled to various types of pathology, and not merely coronary artery disease alone. Further research-work in this area may prove to be rewarding.

6.4 Testing of Hypothesis

Several statistical tests may be employed in order to examine the validity of the results. Specifically, because of its special applicability here, we propose to use the χ^2 test for goodness of fit to test if our experimental results are compatible with those obtained from cineangiography. For the case histories examined, OCG classifications of "normal" EKG tracings yielded 29 normals and 39 CAD's. Cineangiography yielded a distribution of 30 normals and 38 CAD's. In accordance with good practice, the original 24 EKG's utilized to synthesize the OCG were not employed again.

This is a problem for testing the hypothesis that the frequencies observed for CAD and normal patients by means of the omnicardiogram are compatible with those predicted by coronary cineangiography.

	CAD	Normal
Omnicardiogram	39	29
Cineangiogram	38	30

Calculations give: $\chi^2 = \frac{(38-39)^2}{38} + \frac{(30-29)^2}{30} = 0.06$

From a standard table of χ^2 distribution, the 5 per cent critical value of χ^2 for one degree of freedom is $\chi_o^2 = 3.84$. Since $\chi^2 < \chi_o^2$ the hypothesis is acceptable here. Hence there is no reason on the basis of this test for doubting that the frequencies obtained with the omnicardiogram are compatible with those obtained from coronary cineangiography.

6.5 Discussion of False Negatives

As previously stated, the OCG failed to detect five cases which are classified as CAD by coronary cineangiography. These are designated as false negatives and their complete medical records were diligently examined to gain some insight which might explain these discrepancies. Two of the five cases are worth mentioning. Cineangiogram findings in case #245 were not regarded as sufficiently serious to require surgery. Instead, the physician recommended that the patient be reexamined one year later. The implication here appears to be that if coronary artery disease is present, it is not of grave significance. Case #392 is also interesting, but for a different reason. While severe coronary artery disease is implicated, the arteriogram findings also indicate

"a remarkable degree of collateralization around the crux." These findings are suggestive that new avenues are being developed by the heart to serve as alternate routes to replace the blocked branches of the major coronary arteries. In other words, functionally, the heart is attempting to compensate for the blockage of blood caused by the formation of coronary lesions. The remaining cases have been designated as candidates for surgery and must therefore be regarded as frank failures since they were not detected by the omnicardiogram.

6.6 Coronary Cineangiography and Coronary Artery Disease

To assess the validity of our results it is important at this time to consider that each of the tests has been compared with a common reference, coronary cineangiography. It is of some importance to review this "yardstick" and its role in diagnosing coronary artery disease.

Of greatest importance is the fact that coronary symptoms are often vague and subclinical. The physical findings, at rest or during exercise, may render adequate information only in late stages of disease, when arterial and myocardial alterations are far advanced.[3] Thus, the natural history of coronary artery disease and its sequelae often remain largely unknown.[1] In the absence of definitive EKG evidence of myocardial infarction, clinical

diagnosis of coronary disease has depended primarily on proper evaluation of the patient's history and routine physical findings, and accuracy has thus varied with the ability of the physician. In 1966, Proudfit et al[4] undertook a clinical study of 1000 patients suspected of coronary artery disease. Diagnosis, by Proudfit, based on appraisal of the clinical records without a knowledge of the arteriographic findings yielded an average correlation of 83% with cineangiogram findings. One of the considerations examined by Proudfit in the correlation of clinical and arteriographic findings is the necessarily arbitrary separation of degree of estimated arterial obstruction. It appears from his results that mild degrees of obstruction rarely cause myocardial infarction. His work also appears to indicate that correlation between clinical appraisal and arteriographic findings increases with the severity of coronary artery obstruction.

In 1967, Kemp[2] reported the findings of a three-year observation study of terminal coronary artery disease, confirmed by pathologic diagnosis at autopsy. A total of 29 postmortem arteriograms were undertaken on these patients and studied "blindly" (without knowledge of necropsy or autopsy findings). Only three functional errors resulted. The author concluded that "selective coronary cineangiography is a highly accurate means of evaluating morphology of both normal and diseased coronary

vessels." In Kemp's study, coronary cineangiography appears to be quite accurate as a diagnostic procedure.

However, except for severe coronary artery disease (and we must assume that all the subjects in Kemp's study were in this category since they came to autopsy) the evaluation of the degree of occlusion by cineangiography is somewhat subjective on the part of the examining physician. Robbins and Rodriguez[5] have emphasized the difficulty of quantification of coronary atherosclerosis even at post mortem examination. Using three different techniques in the same hearts, these authors found a surprising variance in estimates of intraluminal stenosis assessed by different observers.

Then how does one correlate the decrease in lumen size of a coronary artery with the severity or the prognosis of the disease? To a degree, the evaluation of the coronary arteriogram leaves these questions unsettled.

We have merely attempted to list some of the factors which should be considered in any discussion of coronary artery disease as well as the chief diagnostic tool, coronary cineangiography.

These factors may be of interest in evaluating the statistical comparison between OCG assessment and cineangiography in establishing the degree of confidence for detecting occult coronary artery disease.

6.7 Study of Electrocardiograms with Abnormal Tracings

Although the main thrust of the research has been directed toward detecting coronary artery disease from normal electrocardiogram tracings it was felt that the study could also profit from an examination of abnormal electrocardiogram tracings. Table V presents a summary of results obtained from the OCG assessment of abnormal EKG's. Nine cases came from patients having normal coronary arteries and seven cases from patients suffering from coronary artery disease.

The results indicate that abnormal EKG's produce, in general, abnormal OCG's. This would indicate that pre-screening of EKG's is important.

6.8 Experimental Procedures and Sources of Error in Processing the Electrocardiogram

Randomly selected electrocardiograms were evaluated by at least one cardiologist (and generally by two) without knowledge of the arteriographic findings. This procedure assured an unbiased interpretation of each EKG. Except for a small group of EKG's, all those selected for study were free from any definitive evidence of cardio-vascular impairment. Following the selection of these normal, conventional EKG's, they were documented by case

TABLE V

SUMMARY OF NON-LINEAR TRANSFORMATION OF CASES WITH ABNORMAL EKG FINDINGS

	Case	Lead I	Lead II	Lead V4	Lead V6	OCG Diagnosis	Score
	221	I	O	O	I	CAD	F
Normal	232	I	O	I	I	Normal	S
Coronary	234	I	O	I	O	CAD	F
Arteries	402	I	O	O	O	CAD	F
	428	O	O	O	O	CAD	F
	496	I	O	O	I	CAD	F
	516	I	I	I	I	Normal	S
	562	OO	OO	OO	OO	CAD	F
	623	OO	OO	OO	II	CAD	F
	407	O	O	O	O	CAD	S
Coronary	425	I	O	I	O	CAD	S
	427	II	OO	II	II	Normal	F
Artery	450	O	O	O	O	CAD	S
Disease	454	O	I	I	O	CAD	S
	471	I	I	I	I	Normal	F
	474	O	O	I	I	CAD	S

number and enlarged photographically to double size. Leads I, II, V_4 and V_6 were then digitized by a Gerber analog-to-digital converter having a precision of 500 counts to the inch. The digitized information was converted to punched cards in a format suitable for the RCA Spectra 70 digital computer. The choice of the two limb leads (I, II) and two precordial leads (V_4, V_6) was intended to optimize the likelihood of obtaining three orthogonal views of the electrical vector of the heart.

Initially only one cycle was chosen from each of the four stated leads. Later in the study, two cycles from each of the four leads were processed. In all cases, choice of cycle was made on a purely random basis.

It was soon discovered that a source of error could be introduced by the process of digitization. Also, proper choice of base line was thought to be a line drawn through the isolectric portion of the EKG tracing but this was not always possible. Even slight variation in the choice of a base line sometimes led to some gross errors in diagnosis as evidenced by the bizarre changes in omnicardiogram patterns. As experience was gained in the drawing of base lines and in the actual digitization of the raw data, the incidence of discrepancies was markedly reduced.

A template or omnicardiogram for each lead was

constructed from groups of tracings designated as normal based on arteriographic findings.

As a check on digitization error, the same cycle of several leads were digitized twice. These data are marked by an asterisk in Table II and Table III.

In particular 22 leads derived from 14 separate cases were digitized twice. In only 2 cases was the second digitization of a cycle in disagreement with the first. It is interesting to note that in neither of these cases would the OCG diagnosis have been changed.

As a final note, one should mention that results obtained from typical cases appear in Appendix B. To have included all of the raw data in the dissertation would have provided an unsurmountable logistics problem. However, all the results in the form of original EKG's and computer print-outs are available with the Dissertation Advisor at the Newark College of Engineering for further research.

7 - CONCLUSIONS

The standard electrocardiogram is an extremely useful tool for the diagnosis of certain types of heart diseases. However, it generally fails to detect the existence of occult or preclinical coronary artery disease as demonstrated by selective coronary artery cineangiography.

In this work, a new method of processing four standard leads of the conventional electrocardiogram has been developed. This consists of a non-linear processing of the EKG leads into a new domain offering a high degree of visual perceptibility. The new display, termed the omnicardiogram (OCG), consists of a domain characterized by an annular area bounded by two circles tangent at one point. In the OCG display, tracings deviating outside of the prescribed annular region are assumed to be indicative of possible pathology or abnormality.

In the clinical study described in this work, much of the OCG evidence of coronary artery disease was subsequently confirmed by cardiac cineangiography. Yet the standard EKG tracings gave no evidence of abnormality.

Using the results of cardiac cineangiography as a standard, the OCG correctly detected the presence of occult coronary artery disease in 87% of the CAD patients.

The OCG also gave false positives in 20% of the cases that had been judged free from CAD by means of cardiac cineangiography. However, most of these patients were found to be suffering from other major pathological disorders. More work is indicated to establish if there is a connection here.

This dissertation has primarily tried to elucidate the capabilities of non-linear transformation by a single approach -- demonstration of the feasibility of detecting occult coronary artery disease from ordinary EKG tracings.

We would like to see our work extended by additional investigations to enable expansion and perfection of what appears to be a most fruitful area in bioengineering.

8 - RECOMMENDATIONS

8.1 Omnicardiogram Applied to Other Cardiac Disorders

The results of our study have provided evidence which strongly suggests a correlation between omnicardiogram (OCG) findings and coronary artery disease.

Smaller and less complex than the vectorcardiogram, the OCG potentially offers much simpler visual patterns which may depict many other cardiovascular conditions in addition to coronary artery disease. The accumulation of data will indicate how these correlations can be made. New departures might have to be emphasized, in which case it might be necessary to alter the original pattern to aid recognition. At this point it is clear that all such alterations could be effected quite simply by a change in the computer transformational program.

It is even conceivable that this pattern could be applied to diagnosis of other systemic disorders possibly influenced by or coupled to heart action. It is not unreasonable to assume that the action and proper functioning of the heart may reflect directly, albeit subtly, the physiologic state of other elements such as the lungs, veins, blood constitution, and so on. This reasoning can be assessed only after considerable clinical investigation and research.

8.2 Possible Application to Pharmacology and Neurology

A survey of the literature reveals rather definitive evidence for a correlation between pharmacologic and enzymologic characteristics of laboratory animals afflicted with certain diseases[2,3,4,5,9] or exposed to particular drugs[6,10,13] for prolonged periods, electrocardiographic changes[1,7,13,14] and time[1,2]. These correlates appear most prominent and accessible to analysis in wide scale[8,11] prolonged experiments[12].

Thus according to the theory of the greatest quantity and most variable quality yielding most statistically accurate and conclusive data, application of the nonlinear display technique might be recommended for electrocardiographic investigation in the laboratory as well as in clinical situations. Specific application to the testing of new drugs on experimental laboratory animals appears most intriguing. It may well result that the adverse effects of an experimental drug may be assessed by analysis of the omnicardiogram during the first weeks or months of drug ingestion, rather than the five year period presently prescribed by the F.D.A.

Another fruitful area, unrelated to the cardiovascular system, might be the study of electro-encephalograms to determine existence of specific neurological disorders.

It is hoped that our present work will arouse sufficient interest for the exploration of these and other new domains.

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APPENDIX A

Computer Flow Diagram for Non-Linear Transformation of EKG Tracings.

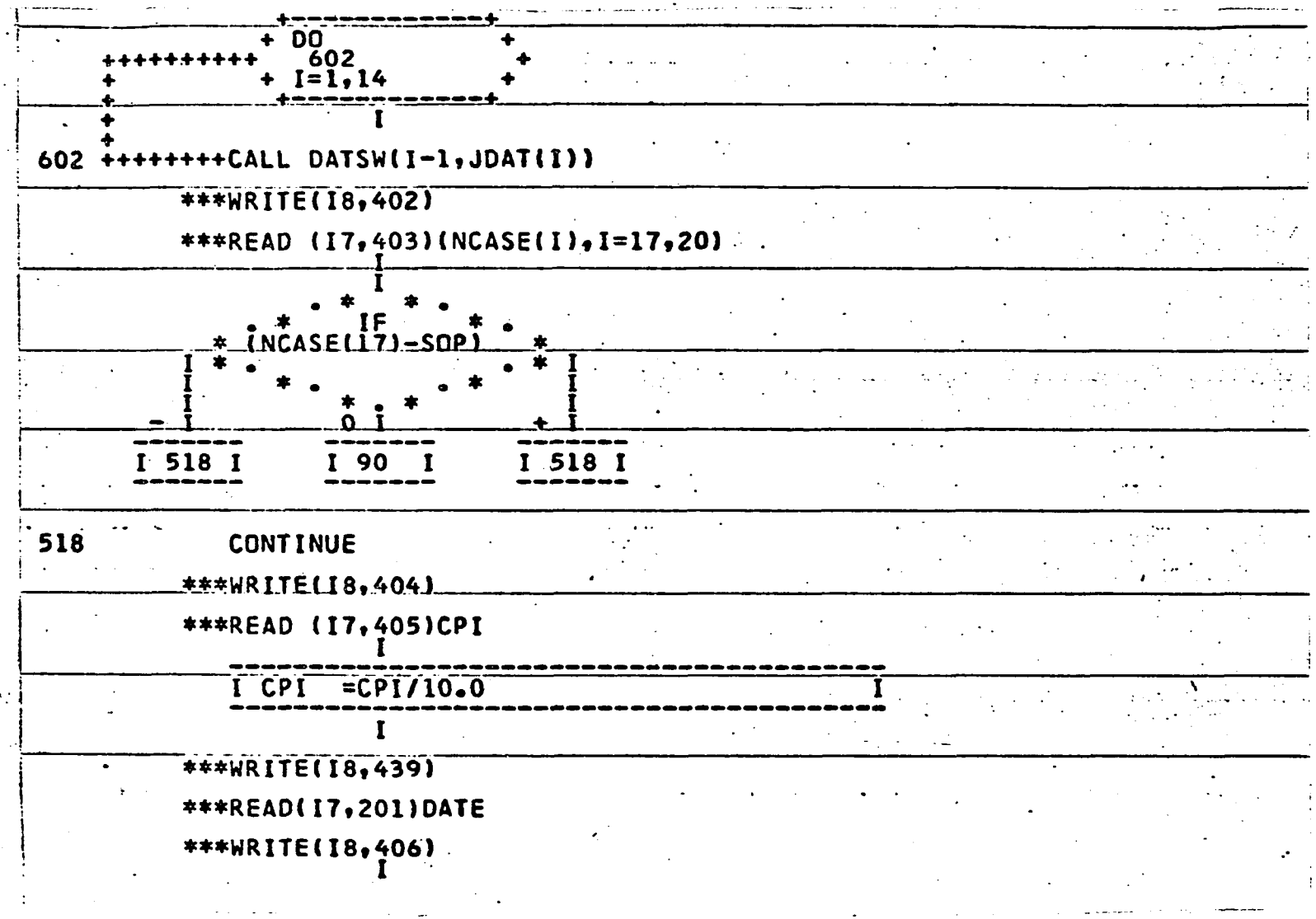
COMPUTER FLOW DIAGRAM
FOR NON-LINEAR
TRANSFORMATION
OF EKG TRACINGS

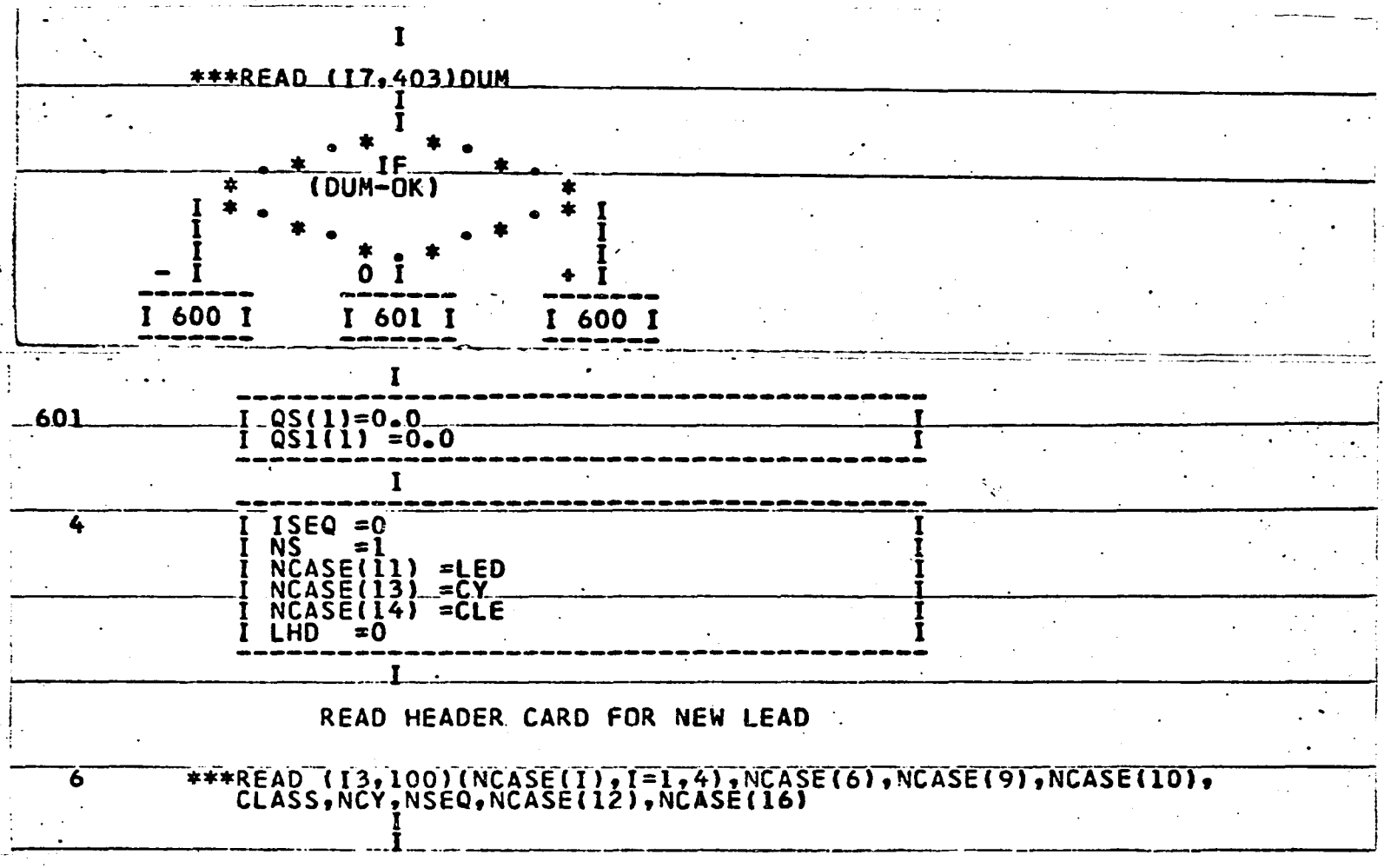
S = EKG Amplitude
T = Time

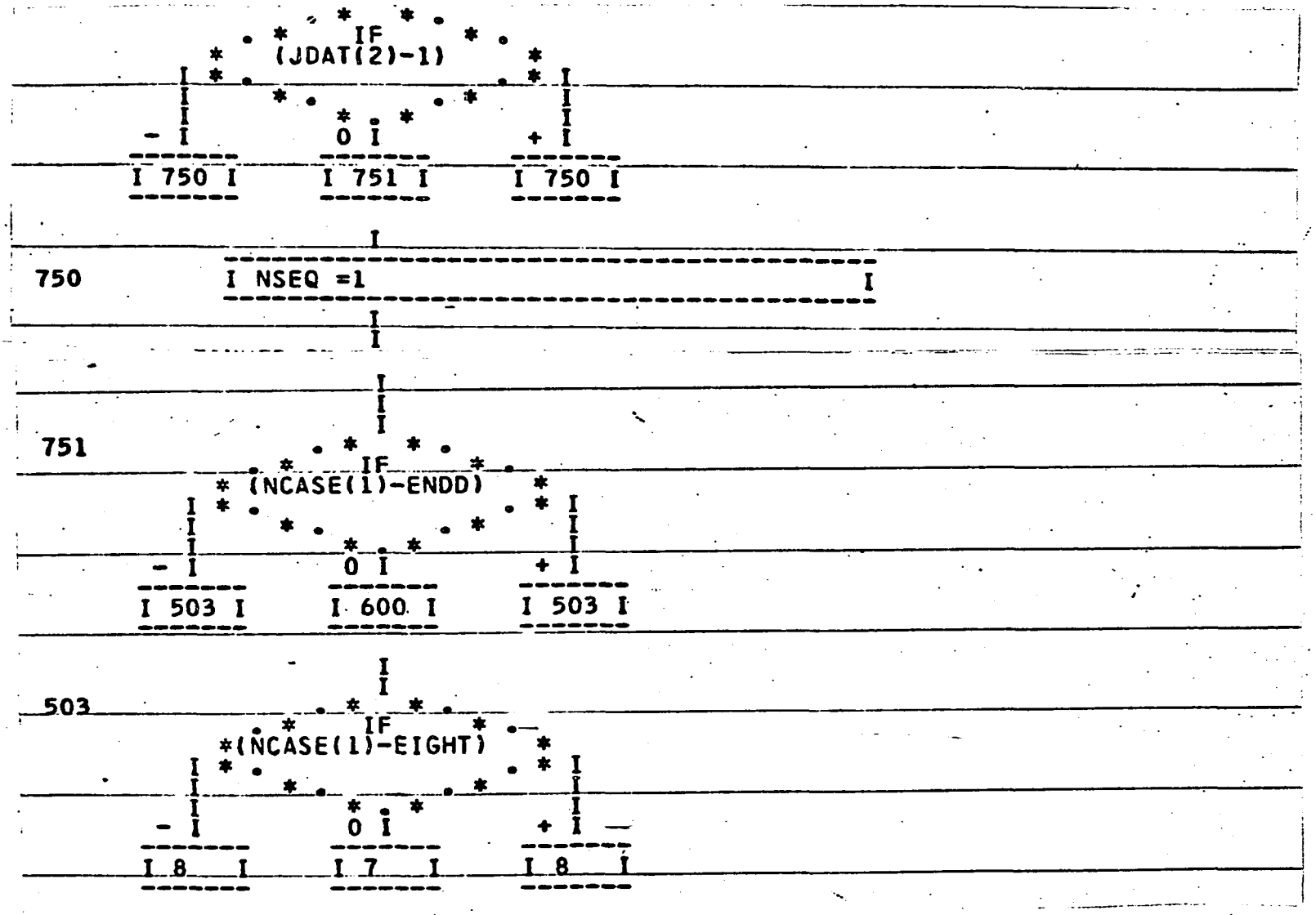
```
                I
      SUBROUTINE 3
     ***REAL NCASE(20)
     ***REAL LED
        DIMENSION DATE(4),LTEST(8)
        DIMENSION ALPHA(4),DUMMY(4),TP(9),SP(9)
        DIMENSION AX(252),AY(252)
        DIMENSION IX(126),IY(126)
        DIMENSION S(126),T(126),S1(126)
        DIMENSION QS(252),QT(252),QS1(252)
        DIMENSION  JDAT(14),AI(4),ROT(4),BSL(4)
        COMMON NCASE,I1,I2,I3,I4,I5,I6,I7,I8
        EQUIVALENCE(QS(130),S(1  )),(QT(130),T(1)),(QS1(130),S1(1))
        DATA ENDD/'9999'/,LE D/'LEAD'/,SEQ/' SEQ'/,BLANK/'    '/
        DATA SOP/'STOP'/
        DATA AGE/'AGE '/,DA,TE/'  DA','TE  '/
            ,ALPHA/' A  ',' B  ',' C  ',' D  '/,CY,CLE/'  CY','CLE '/
            ,EIGHT/'8888'/
        DATA OK/'OK'/,AI/'   I','  II','  V4','  V6'/
100     FORMAT(8A4, 8X,I1,9X,I1,22X,A4,A3)
201     FORMAT(10A4)
203     FORMAT(1H ,55X,'BASELINE IS   S  = ',
               F7.2,' T  + ',F7.2)
```

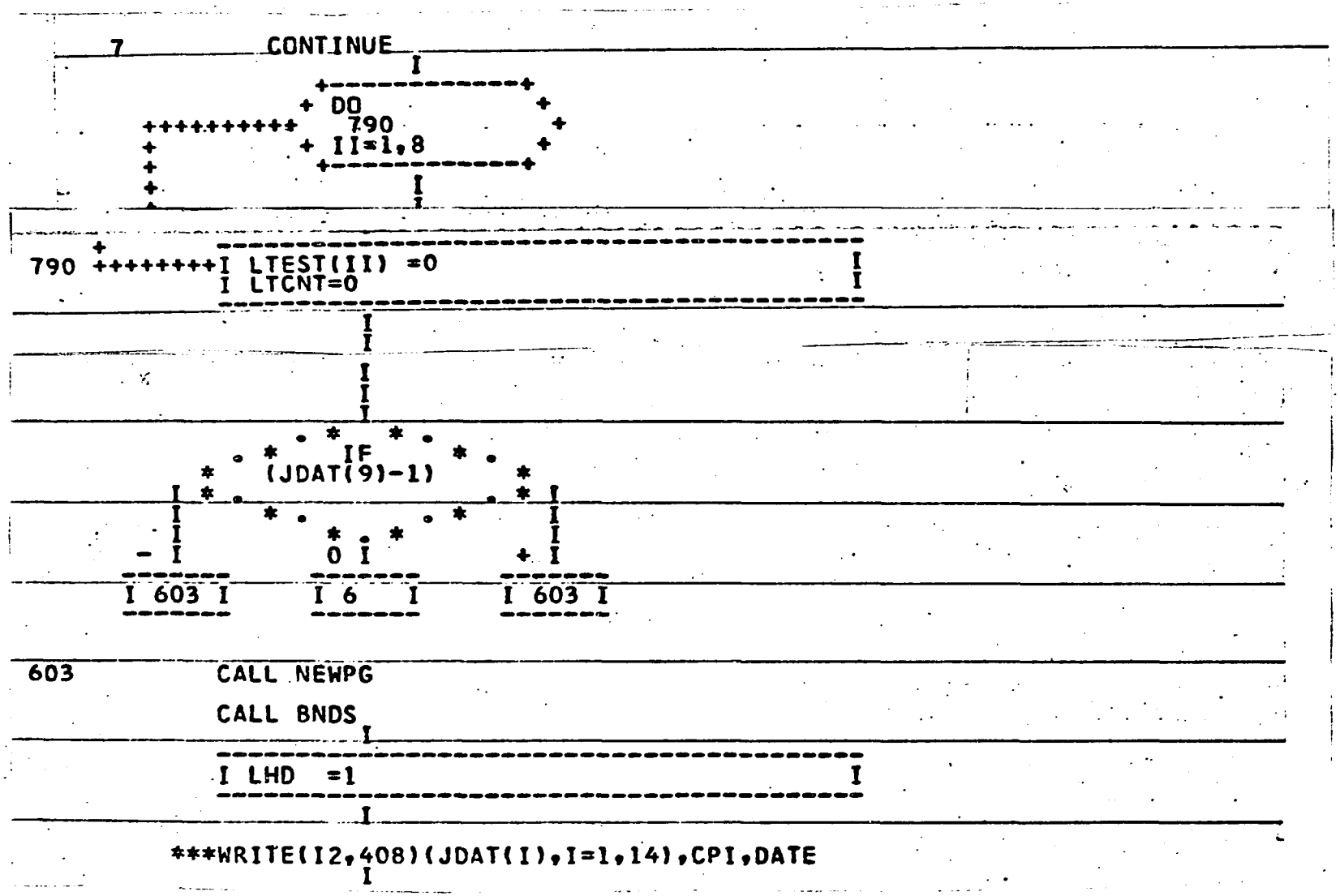
```
401         FORMAT(' SWITCH POS RESULT'/,
                   '  SET. THEN PRESS START'//)
402         FORMAT('ENTER 16 CHAR.ID ,ENTER STOP TO CALL EXIT',/)
403         FORMAT(4A4)
404         FORMAT('ENTER CPI'/)
405         FORMAT(F20.0)
406         FORMAT('CHECK EVERYTHING. THEN ENTER ''OK'' OR ''BLAHT''',/)
407         FORMAT(8(2F4.1,1X))

                    I
408         FORMAT(1H+,14I1,F10.3,'  X  10  CPI     DATA DISPLAY SYSTEMS EKG
            ANALYSIS',5X,4A4)
409         FORMAT('HEADER ERROR ON LEAD ID. FIX + PRESS START')
410         FORMAT('DATA  ERROR .FIX + PRESS START TO REREAD HEADER')
419         FORMAT(/,' ************************************************************
            ************************************************',/,  ' *****',20A4)
418         FORMAT(' ROT8 PTS=',2(2F9.1,3X),'BL PTS=' , 2(2F9.1,3X))
417         FORMAT(8(2F4.1,1X),2A4)
411         FORMAT(18F4.1,2A4)
412         FORMAT( 8(F6.1,    F6.1    ),4A4)
413         FORMAT(1X,18F6.1,4A4)
440         FORMAT(1H+,T75,'NSS')
437         FORMAT(1H1)
430         FORMAT(   ' THETA=',F10.4)
431         FORMAT(' INVALID,REQ TO PLOT EKG ON Q WITH COMPOSITE NOT DISABLD')
```

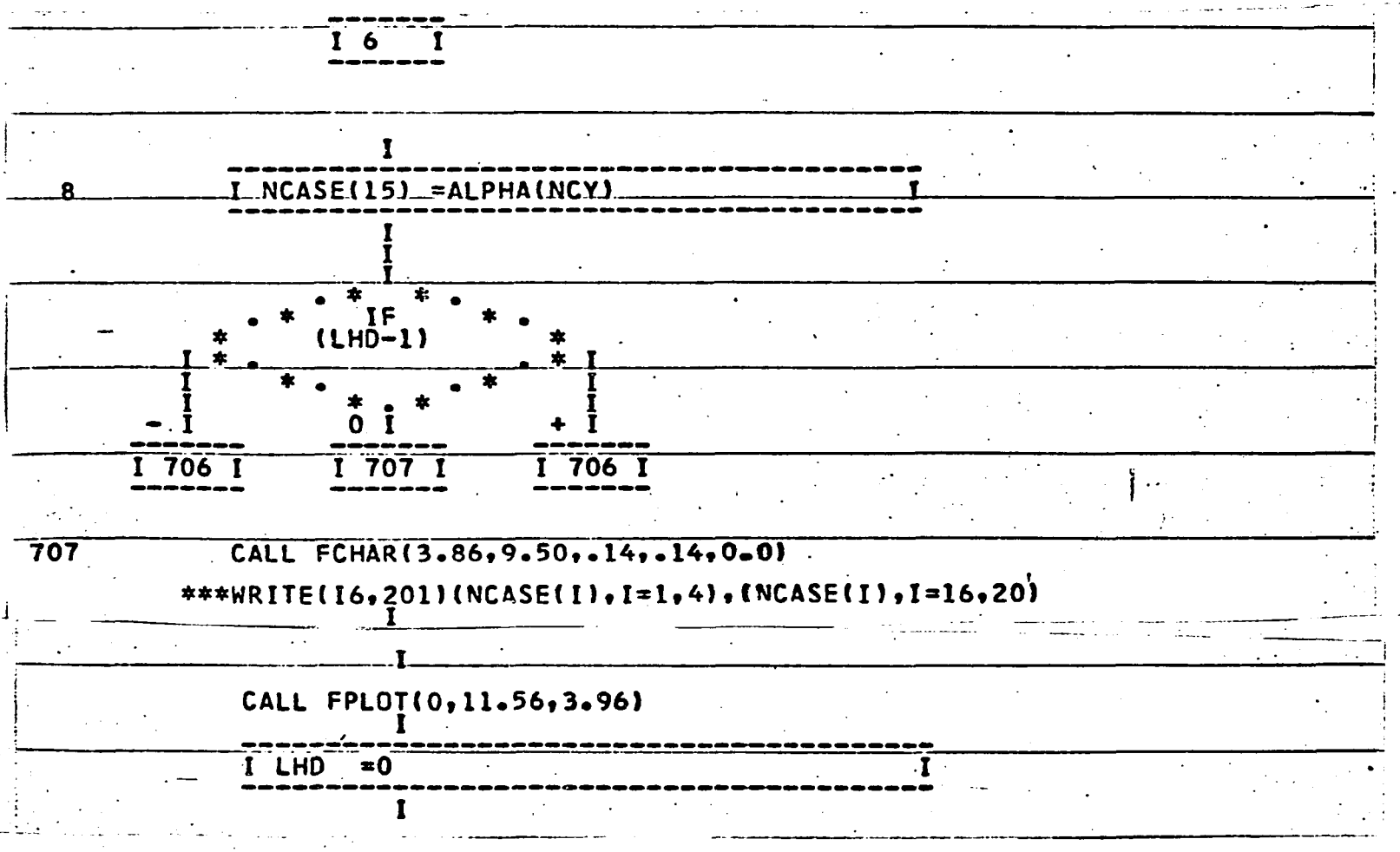
```
 432          FORMAT(1H+,T62,'L GT',E4.1)
 433          FORMAT(' INVALID,REQ TO PLOT XFM ON Q WITH COMPOSITE NOT DISABLD')
 434          FORMAT(14A4,3I4,I1,2I4,4I1)
 435          FORMAT(/1X,14A4,3I4,I1,2I4,I1)
 436          FORMAT(1H1,20A4)
 438          FORMAT(' NSS')
 439          FORMAT(' ENTER DATE')
1000          FORMAT(2F10.5)
 441          FORMAT(1H+,T82,'STO')
              AAXVL(AINI,HITE,AAGN,CPID)=AINI+HITE/AAGN*CPID
                    I
              -----------------------------------------
              I NCASE(5) =AGE                         I
              I NCASE(7) =DA                          I
              I NCASE(8) =TE                          I
              I J1   =1                               I
              I J2   =2                               I
              I J3   =3                               I
                    I
              J4=4
                    I
              -----------------------------------------
              I J5   =5                               I
              I BX   =5.                              I
              I AL1MX=3.9                             I
              -----------------------------------------
                    I
 517      ***WRITE(I8,401)
 600      ***PAUSE 0001
                    I
```

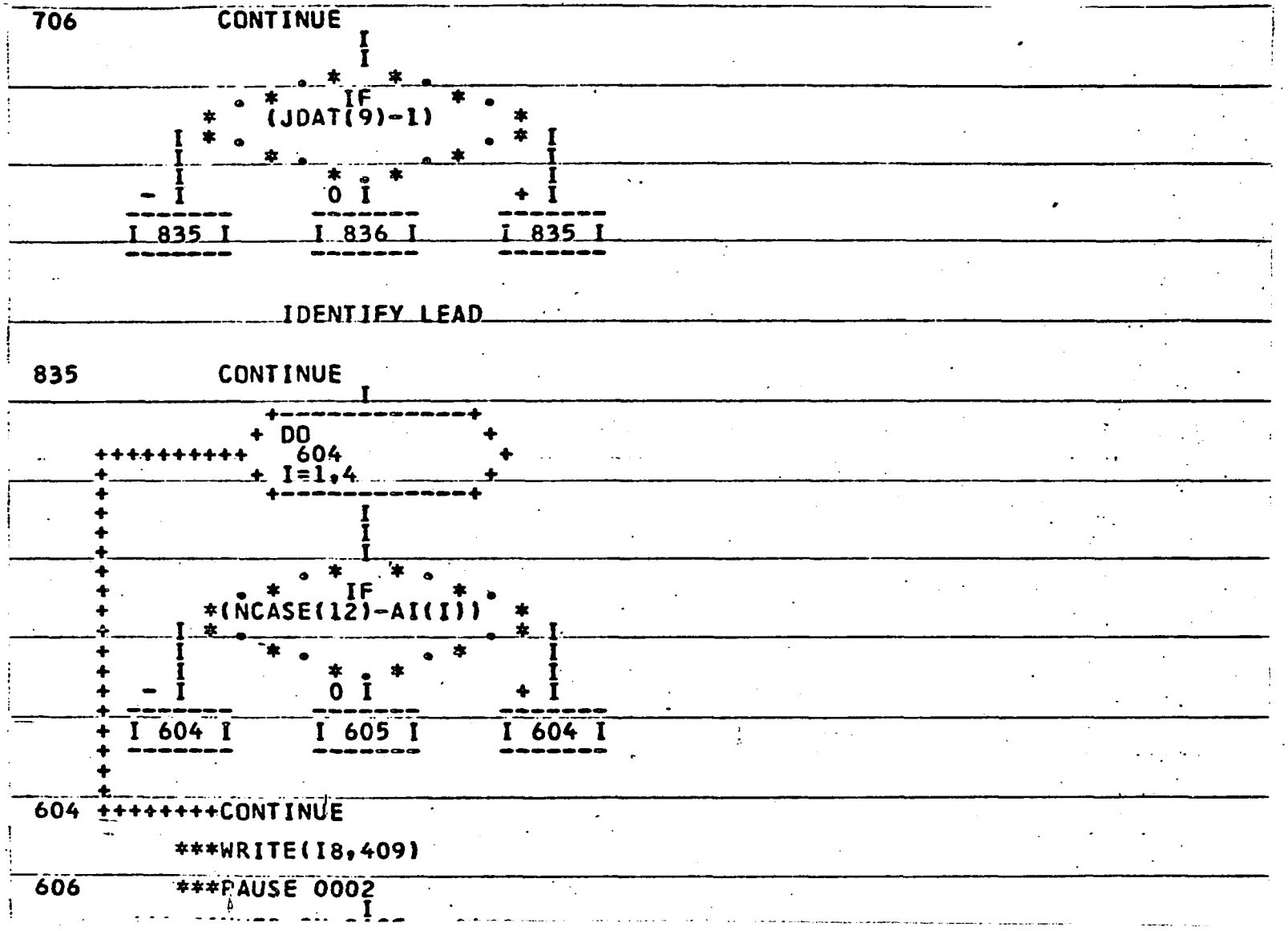

DO
602
I=1,14
602 CALL DATSW(I-1,JDAT(I))
***WRITE(I8,402)
***READ (I7,403)(NCASE(I),I=17,20)
IF
(NCASE(17)-SOP)
-
0
+
I 518 I
I 90 I
I 518 I
518 CONTINUE
***WRITE(I8,404)
***READ (I7,405)CPI
I CPI =CPI/10.0 I
***WRITE(I8,439)
***READ(I7,201)DATE
***WRITE(I8,406)

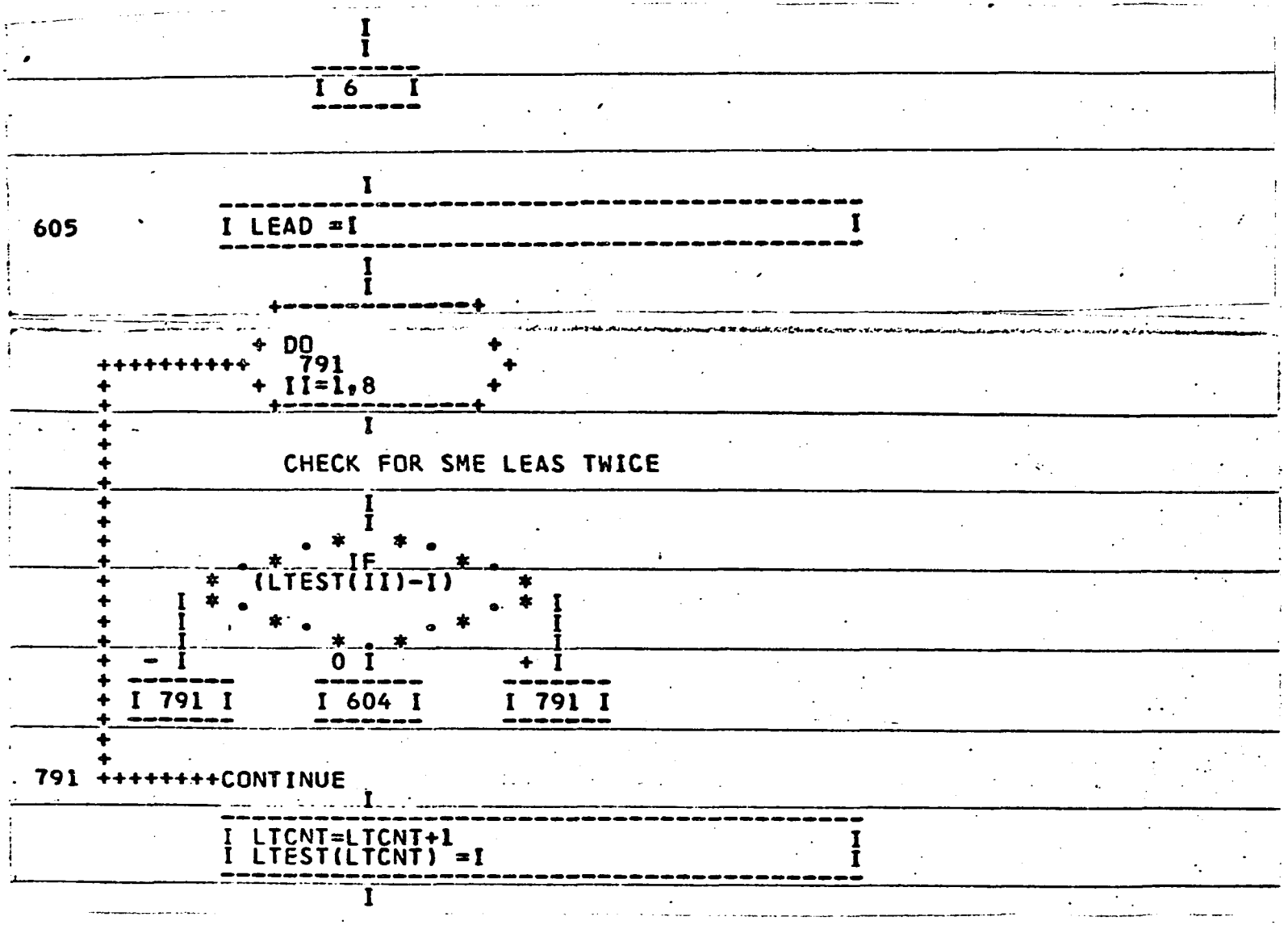

***READ (I7,403)DUM
IF (DUM-OK)
- 0 +
600
601
600
601
QS(1)=0.0
QS1(1) =0.0
4
ISEQ =0
NS =1
NCASE(11) =LED
NCASE(13) =CY
NCASE(14) =CLE
LHD =0
READ HEADER CARD FOR NEW LEAD
6
***READ (I3,100)(NCASE(I),I=1,4),NCASE(6),NCASE(9),NCASE(10),
CLASS,NCY,NSEQ,NCASE(12),NCASE(16)

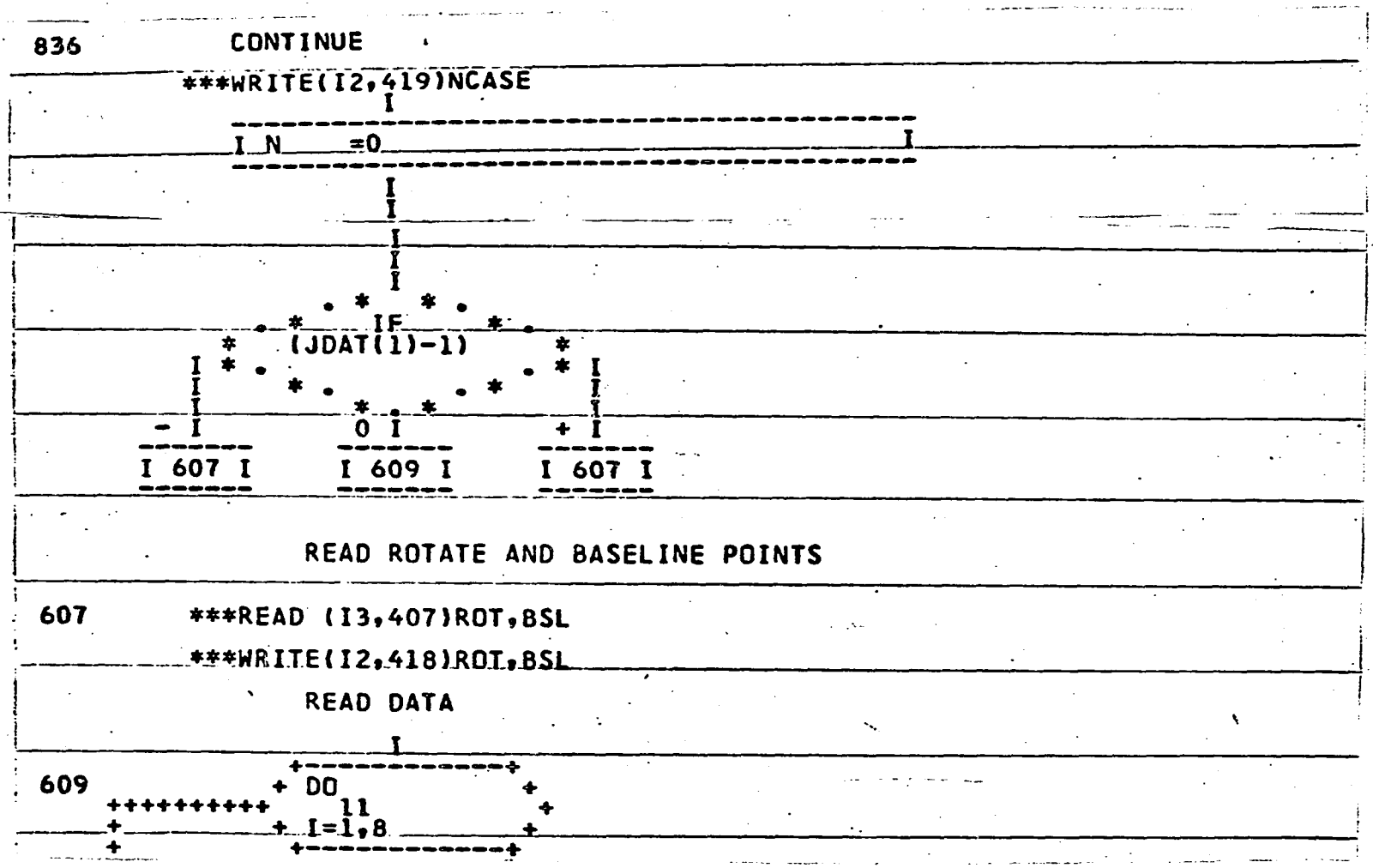

IF
(JDAT(2)-1)
- I
0 I
+ I
I 750 I
I 751 I
I 750 I
750
I NSEQ =1 I
751
IF
(NCASE(1)-ENDD)
- I
0 I
+ I
I 503 I
I 600 I
I 503 I
503
IF
(NCASE(1)-EIGHT)
- I
0 I
+ I
I 8 I
I 7 I
I 8 I


7 CONTINUE
DO 790 II=1,8
790 LTEST(II) =0
LTCNT=0
IF (JDAT(9)-1)
- 0 +
603
6
603
603 CALL NEWPG
CALL BNDS
LHD =1
***WRITE(I2,408)(JDAT(I),I=1,14),CPI,DATE

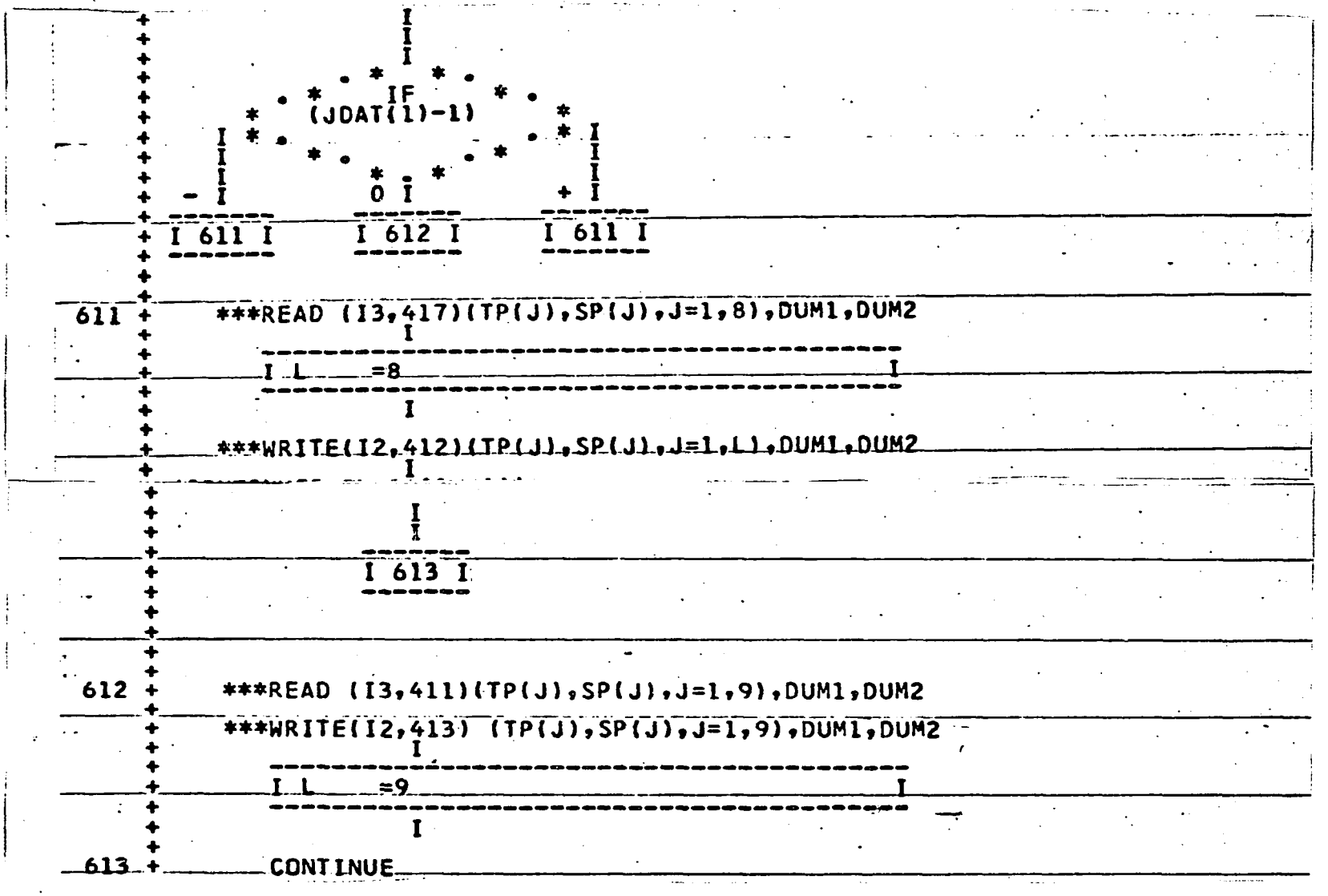

I 6 I
8
I NCASE(15) =ALPHA(NCY)
IF
(LHD-1)
- I
0 I
+ I
I 706 I
I 707 I
I 706 I
707
CALL FCHAR(3.86,9.50,.14,.14,0.0)
***WRITE(I6,201)(NCASE(I),I=1,4),(NCASE(I),I=16,20)
CALL FPLOT(0,11.56,3.96)
I LHD =0

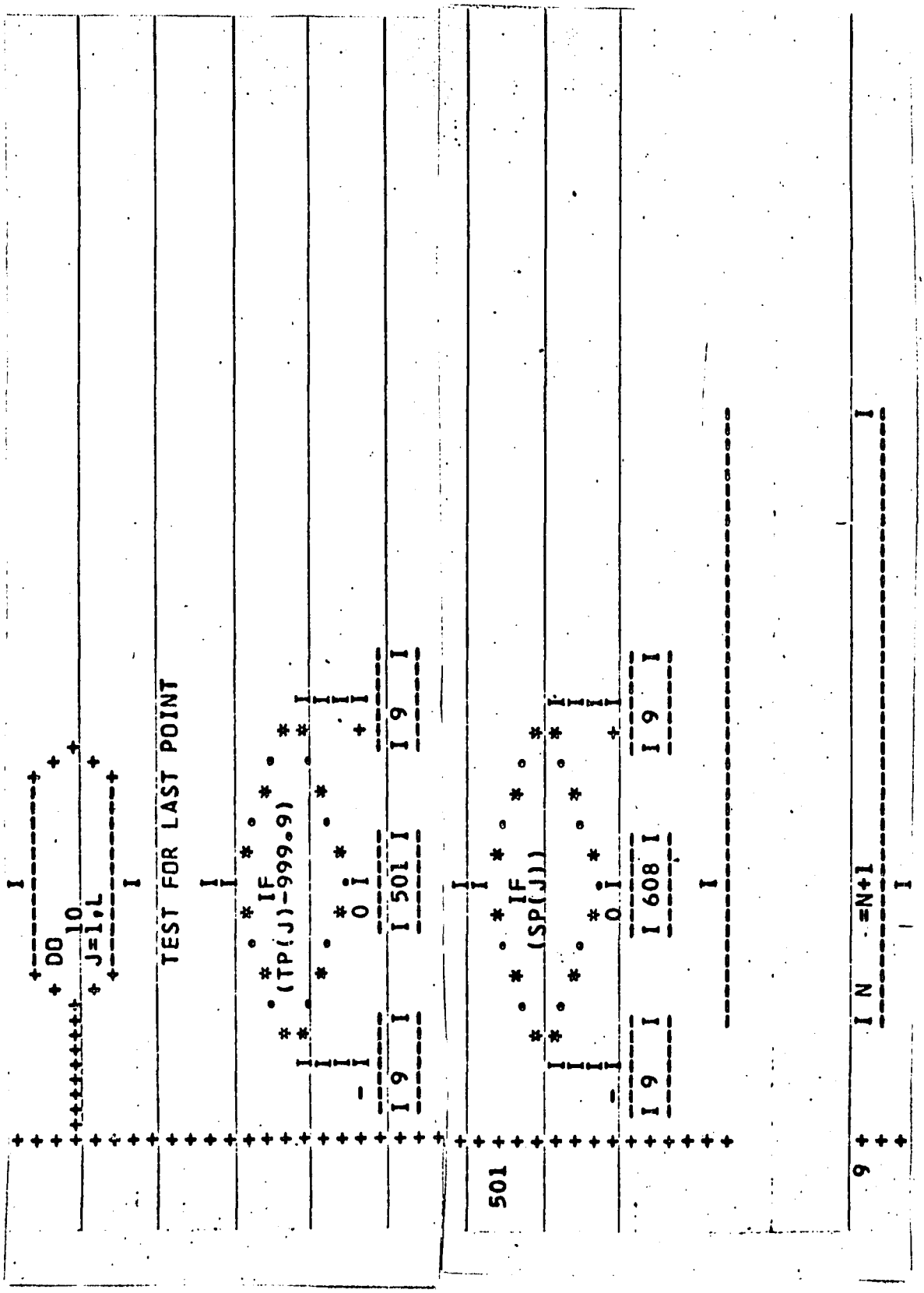

706 CONTINUE
IF
(JDAT(9)-1)
- 0 +
835 836 835
IDENTIFY LEAD
835 CONTINUE
DO
604
I=1,4
IF
*(NCASE(12)-AI(I))
- 0 +
604 605 604
604 +++++++CONTINUE
***WRITE(I8,409)
606 ***PAUSE 0002

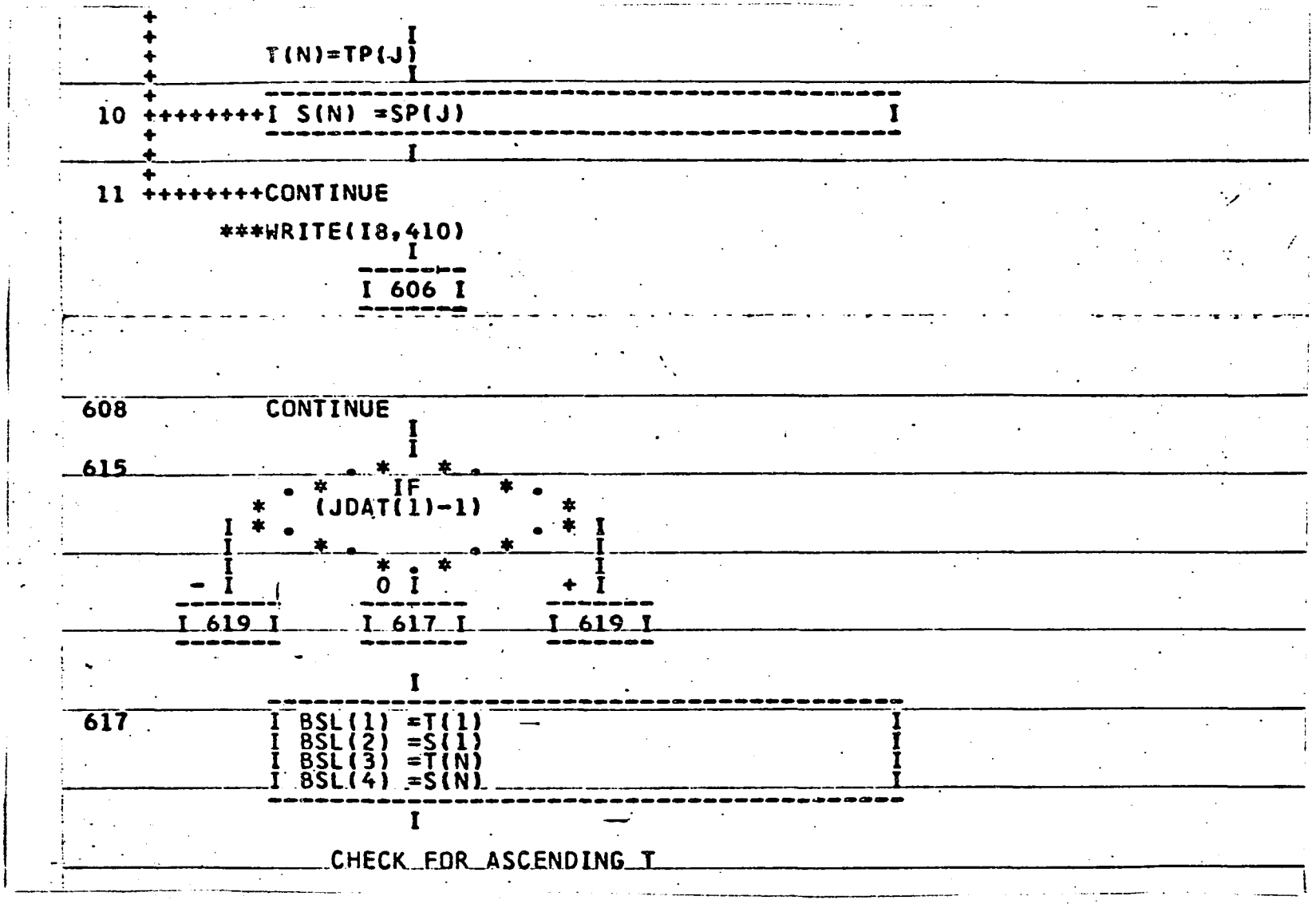

6
605 LEAD =I
DO 791 II=1,8
CHECK FOR SME LEAS TWICE
IF (LTEST(II)-I)
- 0 +
791 604 791
791 CONTINUE
LTCNT=LTCNT+1
LTEST(LTCNT) =I


836 CONTINUE
***WRITE(I2,419)NCASE
I N =0
IF
(JDAT(1)-1)
- 0 +
607
609
607
READ ROTATE AND BASELINE POINTS
607 ***READ (I3,407)ROT,BSL
***WRITE(I2,418)ROT,BSL
READ DATA
609 DO
11
I=1,8

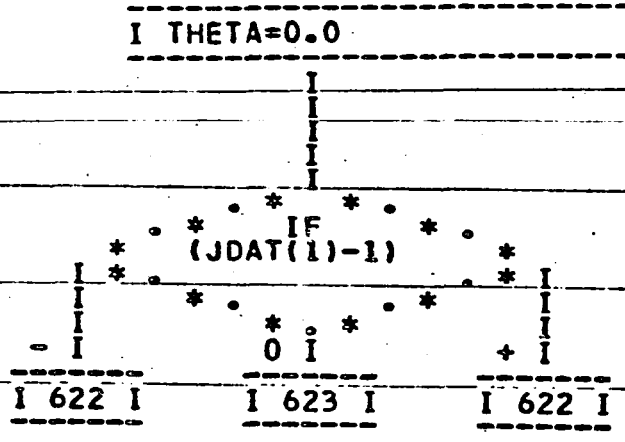
12A


IF
(JDAT(1)-1)
- 0 +
611
612
611
611 ***READ (I3,417)(TP(J),SP(J),J=1,8),DUM1,DUM2
I L =8
***WRITE(I2,412)(TP(J),SP(J),J=1,L),DUM1,DUM2
613
612 ***READ (I3,411)(TP(J),SP(J),J=1,9),DUM1,DUM2
***WRITE(I2,413) (TP(J),SP(J),J=1,9),DUM1,DUM2
I L =9
613 CONTINUE

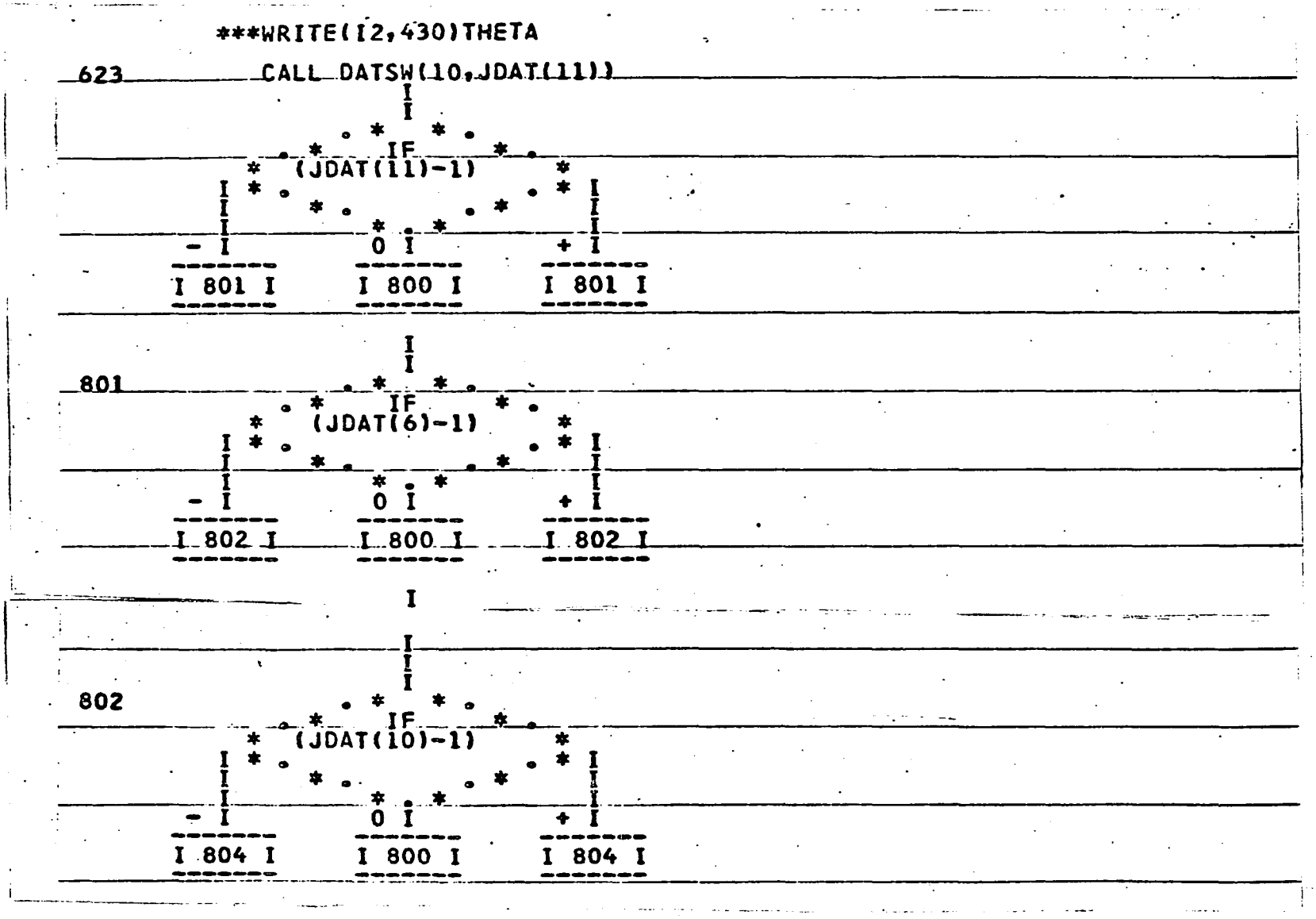

DO 10 J=1,L
TEST FOR LAST POINT
IF (TP(J)-999.9)
501
IF (SP(J))
9
N =N+1

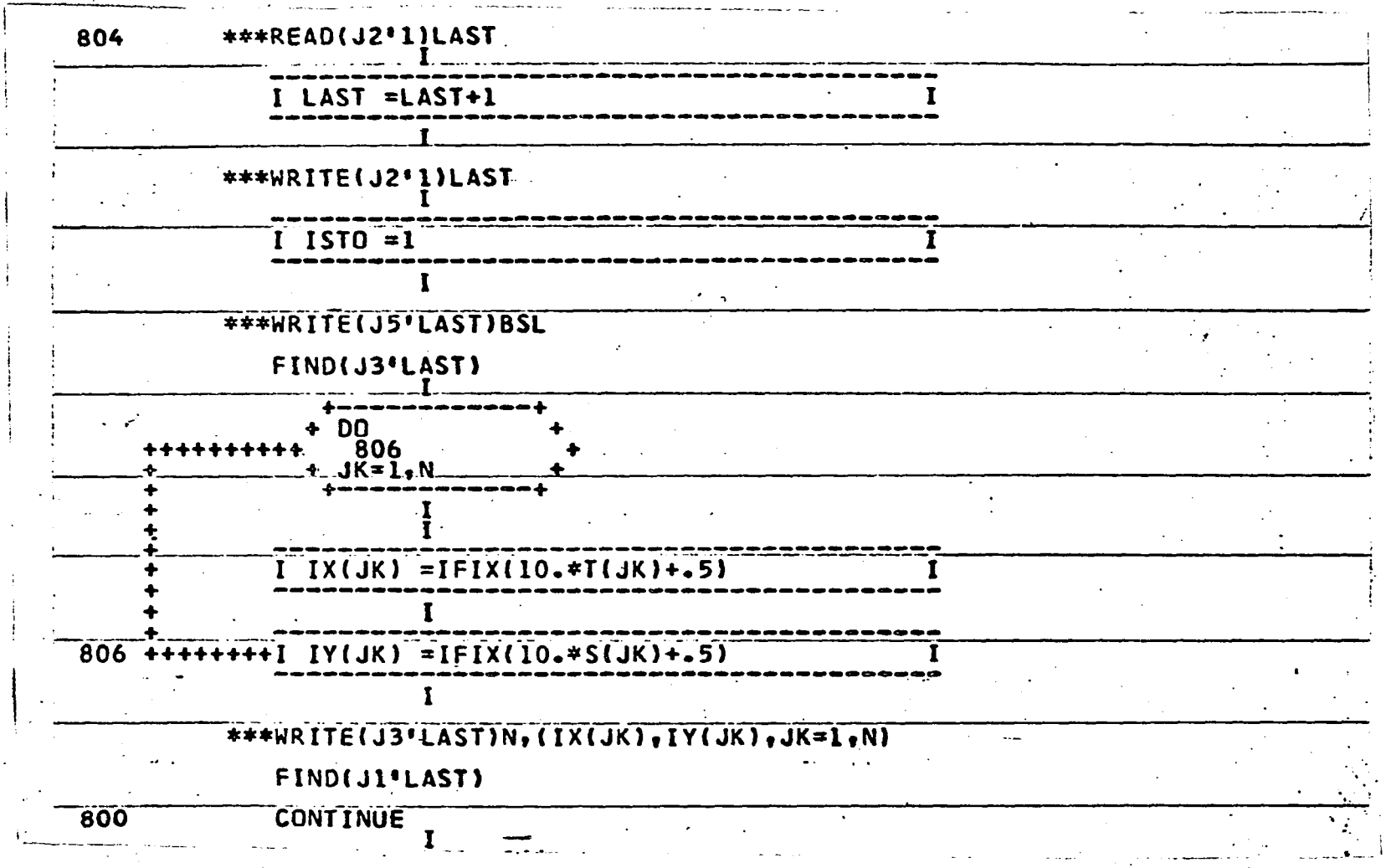

T(N)=TP(J)
10 S(N) =SP(J)
11 CONTINUE
***WRITE(I8,410)
606
608 CONTINUE
615
IF
(JDAT(1)-1)
-
0
+
619
617
619
BSL(1) =T(1)
BSL(2) =S(1)
BSL(3) =T(N)
BSL(4) =S(N)
CHECK FOR ASCENDING T

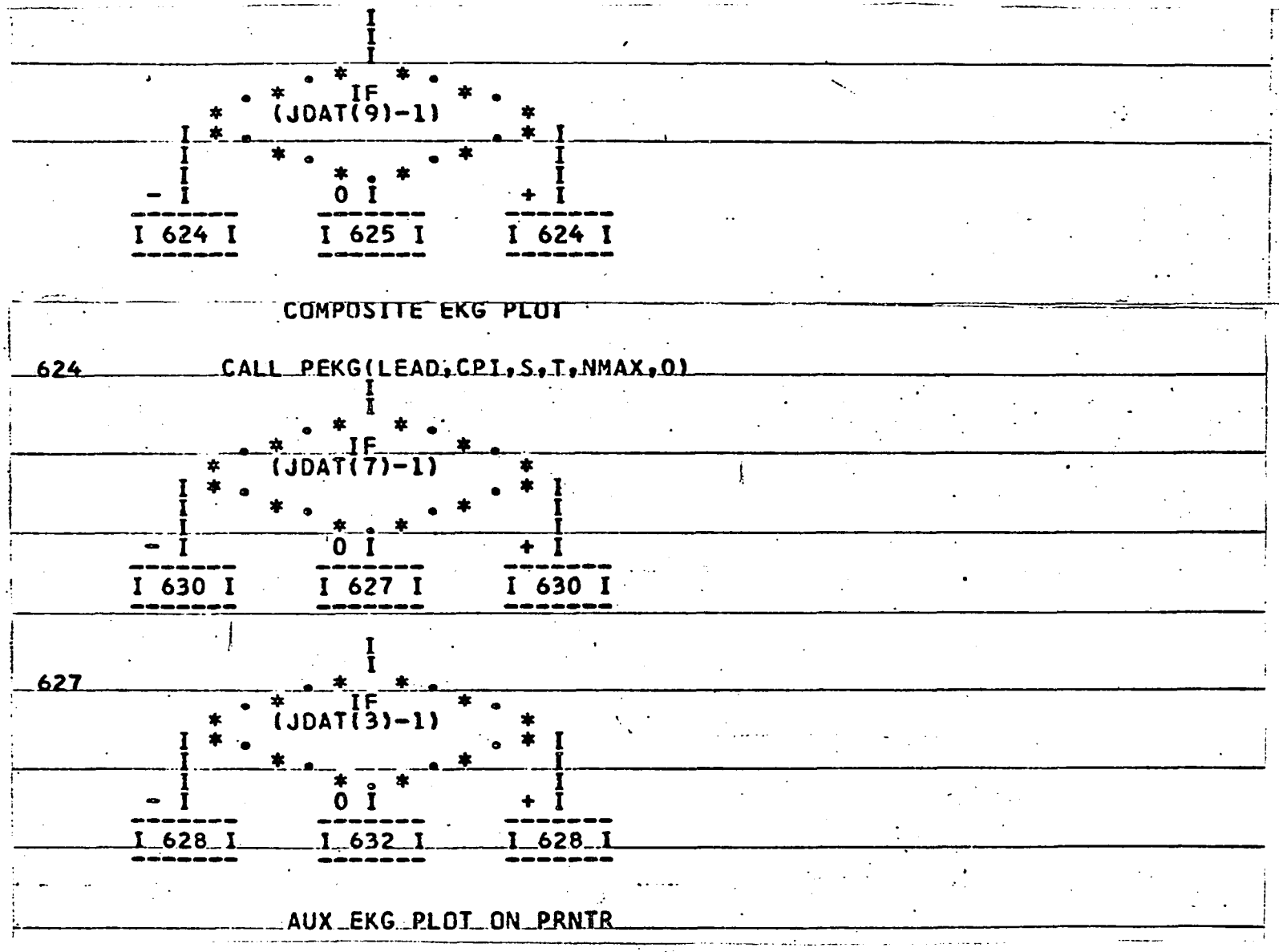
```
619          CALL SORT(T,S,N)
                       I
             ------------------------------------------
             I NMAX =N                                I
             ------------------------------------------
                       I
                  CHECK T=T
620          CALL TEQT(T,S,NMAX)
                       I
```

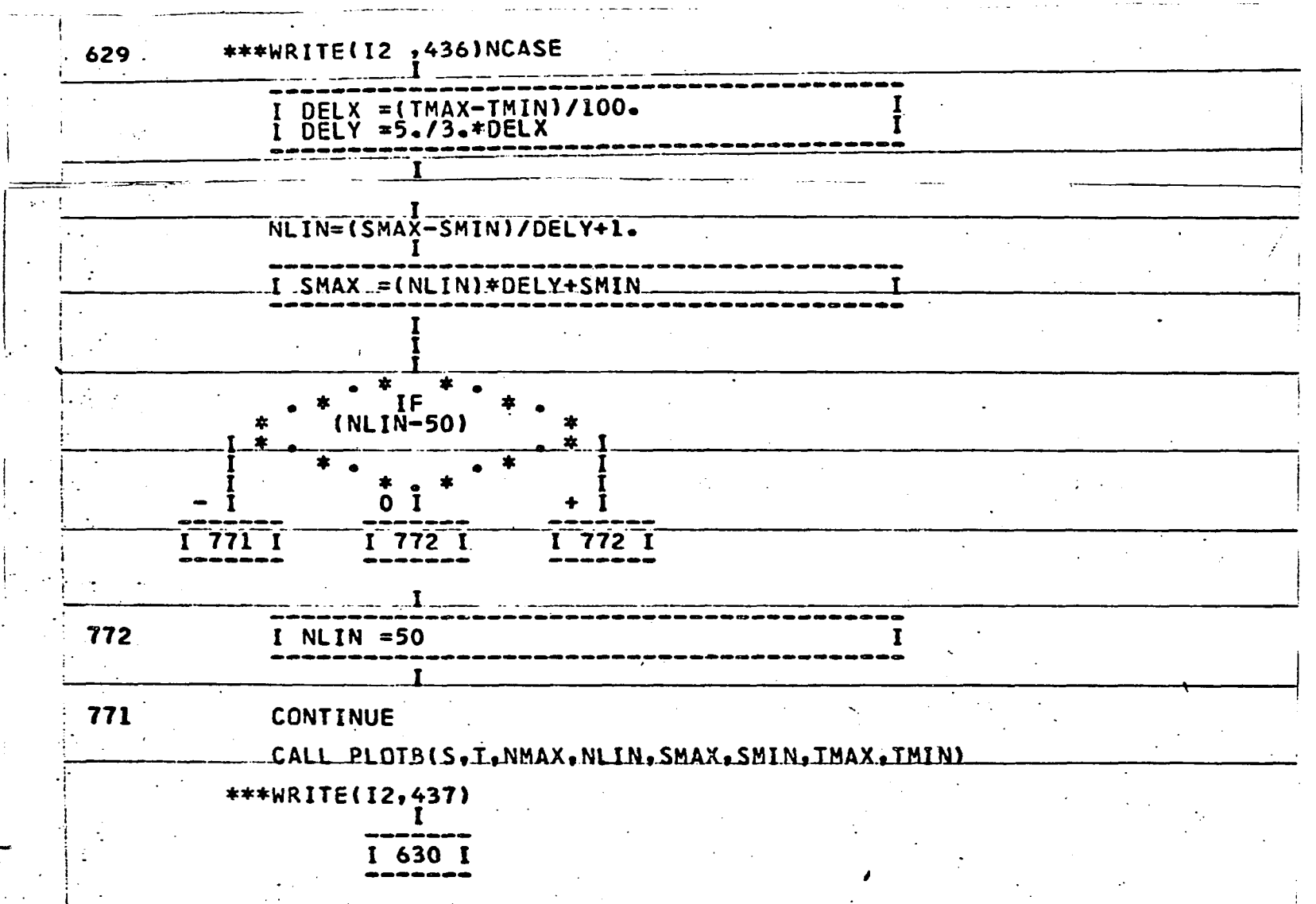


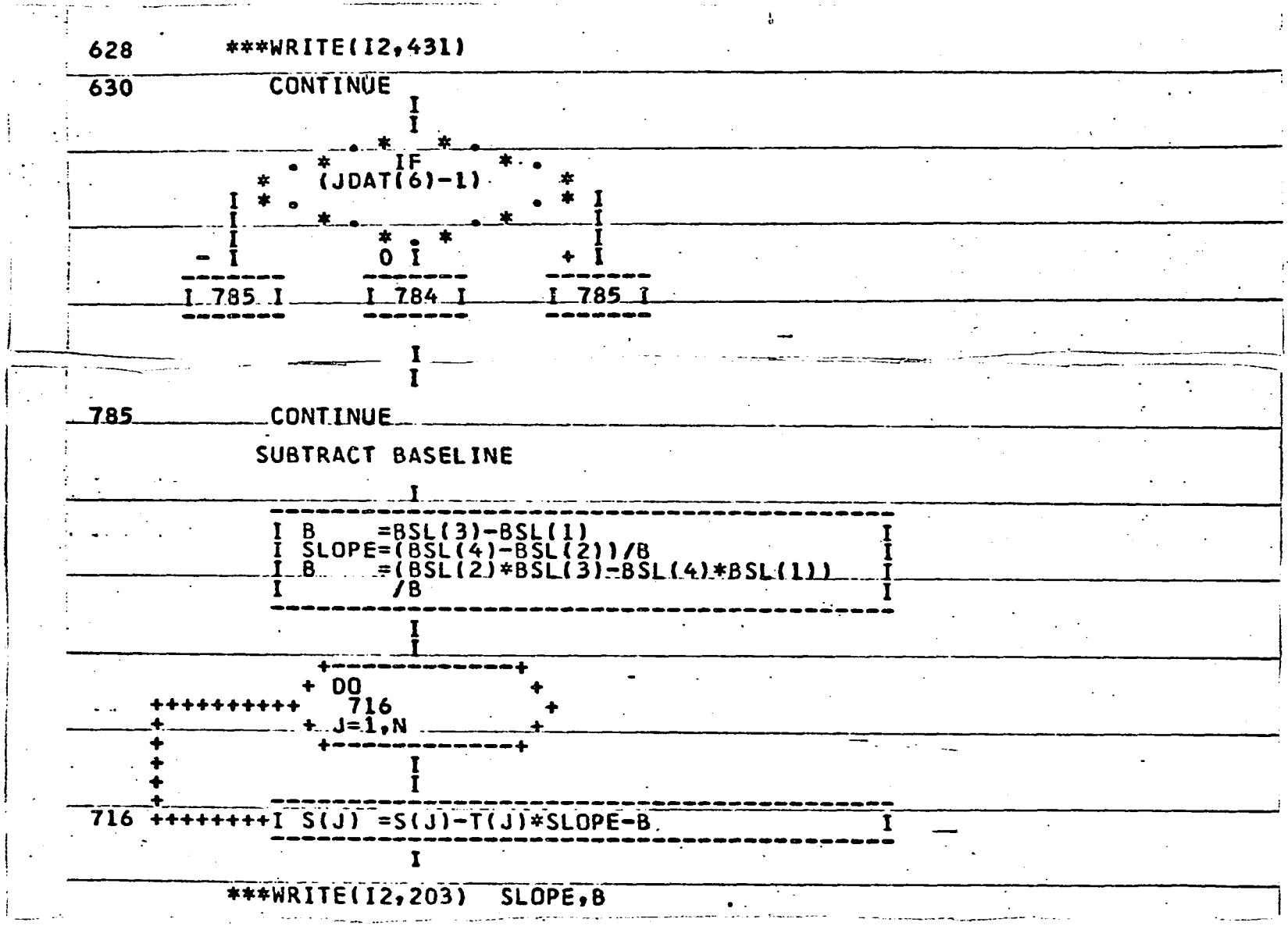

```
                  ROTATE EKG
622          CALL ROT8(ROT,T,S,NMAX,THETA)
             CALL ROT8(ROT,BSL(1),BSL(2) ,1,THETA)
             CALL ROT8(ROT,BSL(3),BSL(4) ,1,THETA)
```

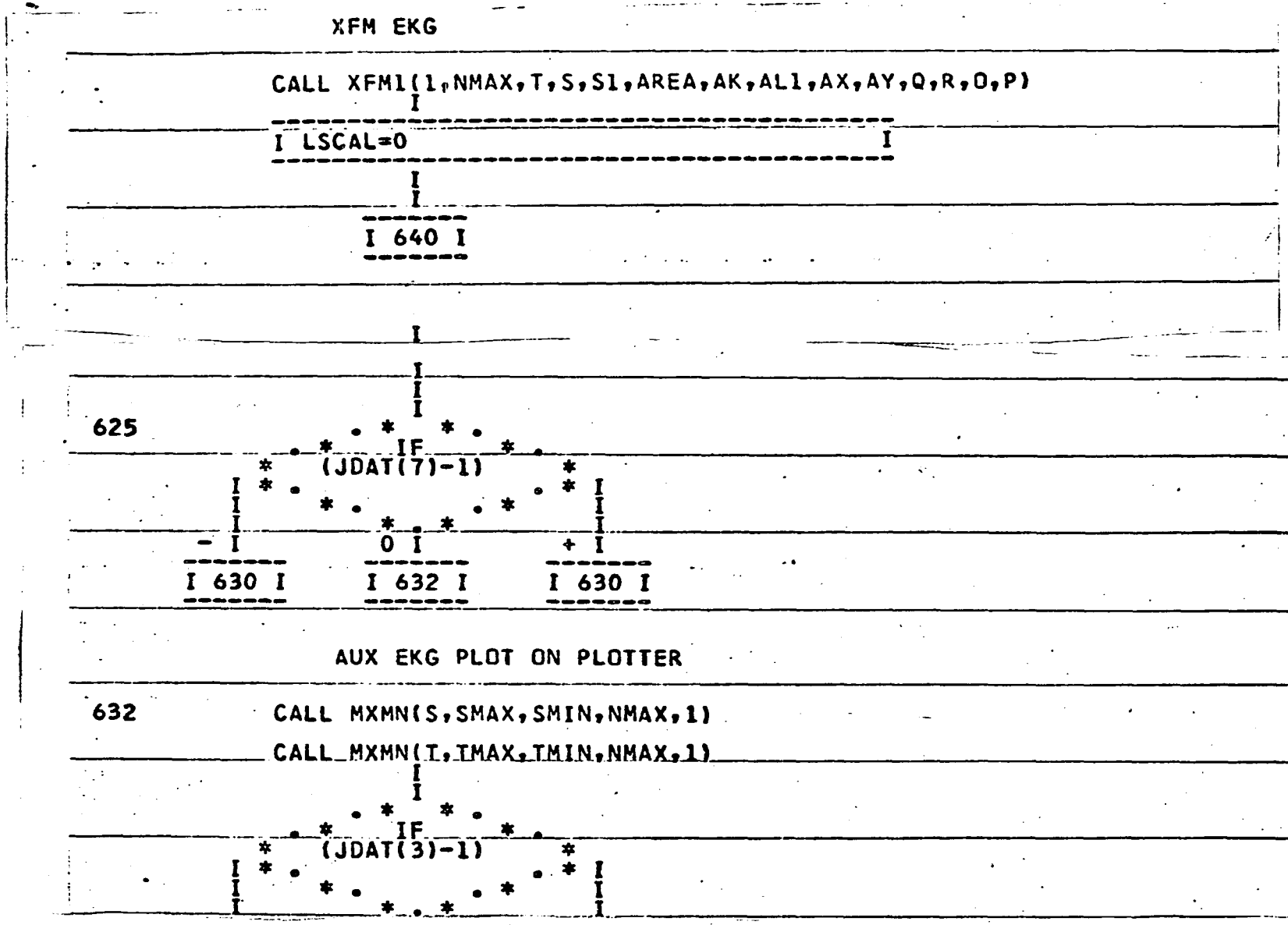

***WRITE(12,430)THETA
623
CALL DATSW(10,JDAT(11))
IF
(JDAT(11)-1)
- 0 +
801 800 801
801
IF
(JDAT(6)-1)
- 0 +
802 800 802
802
IF
(JDAT(10)-1)
- 0 +
804 800 804

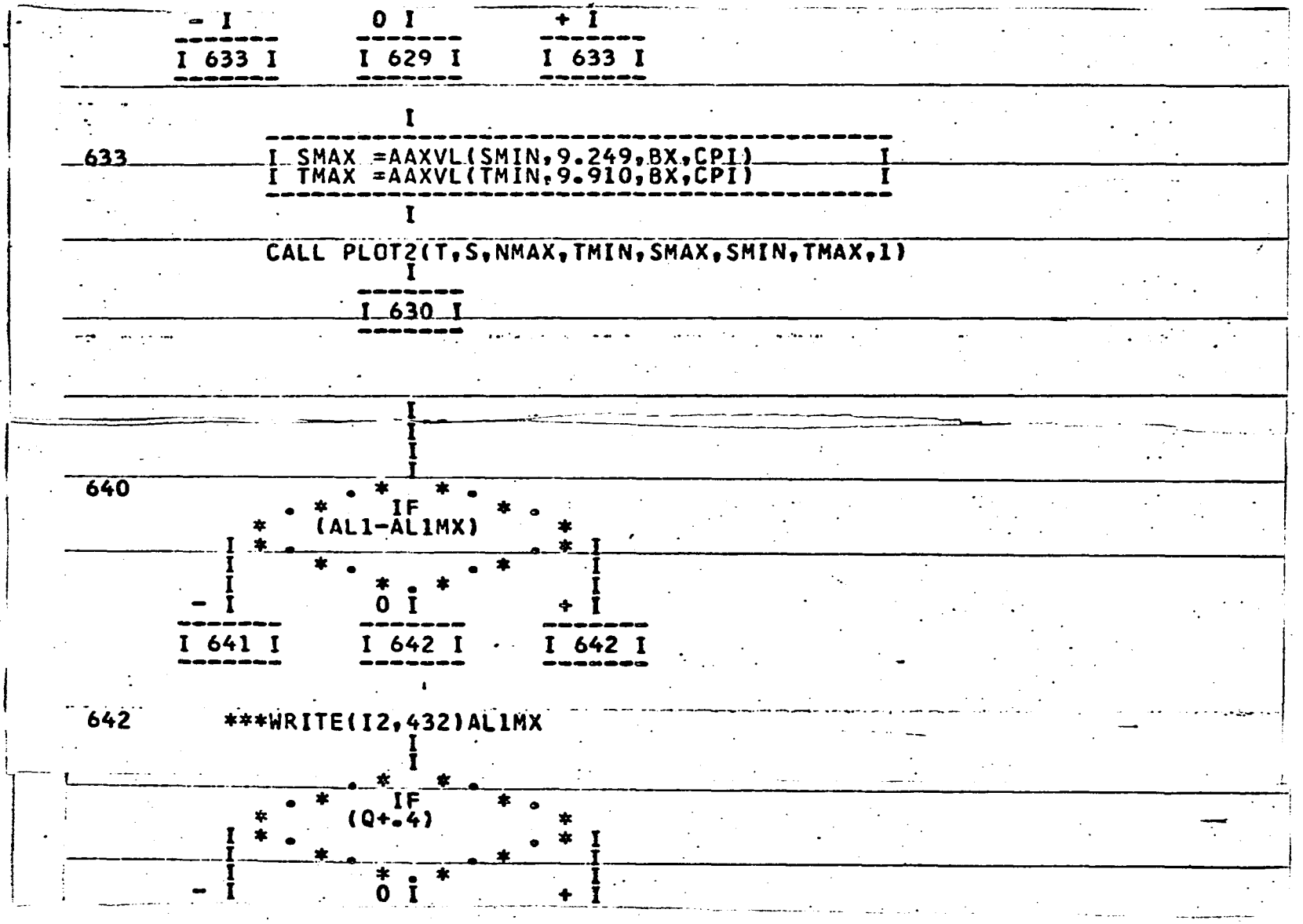

804 ***READ(J2'1)LAST
LAST =LAST+1
***WRITE(J2'1)LAST
ISTO =1
***WRITE(J5'LAST)BSL
FIND(J3'LAST)
DO 806 JK=1,N
IX(JK) =IFIX(10.*T(JK)+.5)
806 IY(JK) =IFIX(10.*S(JK)+.5)
***WRITE(J3'LAST)N,(IX(JK),IY(JK),JK=1,N)
FIND(J1'LAST)
800 CONTINUE

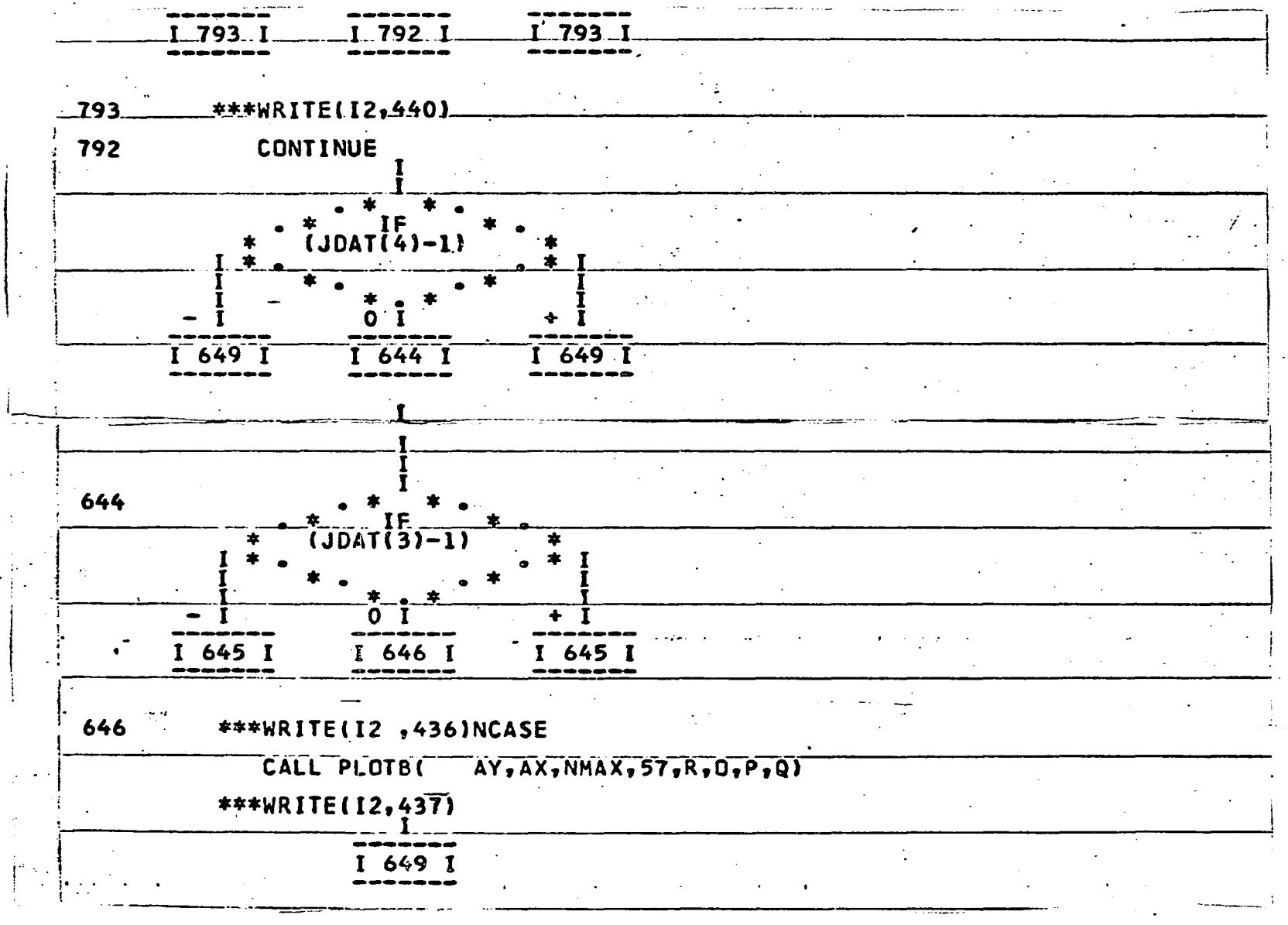

IF
(JDAT(9)-1)
- I
0 I
+ I
I 624 I
I 625 I
I 624 I
COMPOSITE EKG PLOT
624
CALL PEKG(LEAD,CPI,S,T,NMAX,0)
IF
(JDAT(7)-1)
- I
0 I
+ I
I 630 I
I 627 I
I 630 I
627
IF
(JDAT(3)-1)
- I
0 I
+ I
I 628 I
I 632 I
I 628 I
AUX EKG PLOT ON PRNTR

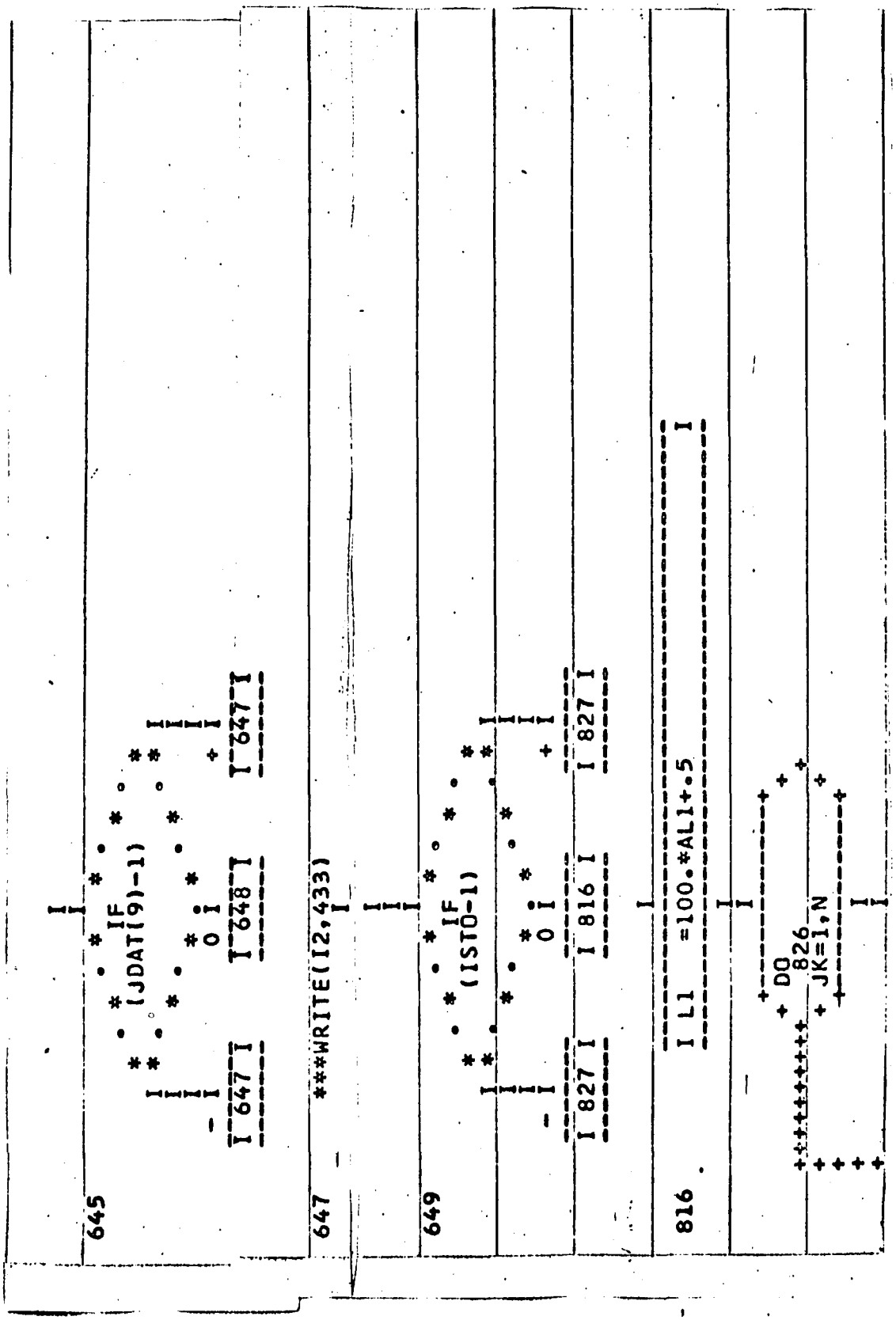

629
***WRITE(I2 ,436)NCASE
DELX =(TMAX-TMIN)/100.
DELY =5./3.*DELX
NLIN=(SMAX-SMIN)/DELY+1.
SMAX =(NLIN)*DELY+SMIN
IF
(NLIN-50)
-
0
+
771
772
772
NLIN =50
772
CONTINUE
771
CALL PLOTB(S,I,NMAX,NLIN,SMAX,SMIN,TMAX,TMIN)
***WRITE(I2,437)
630

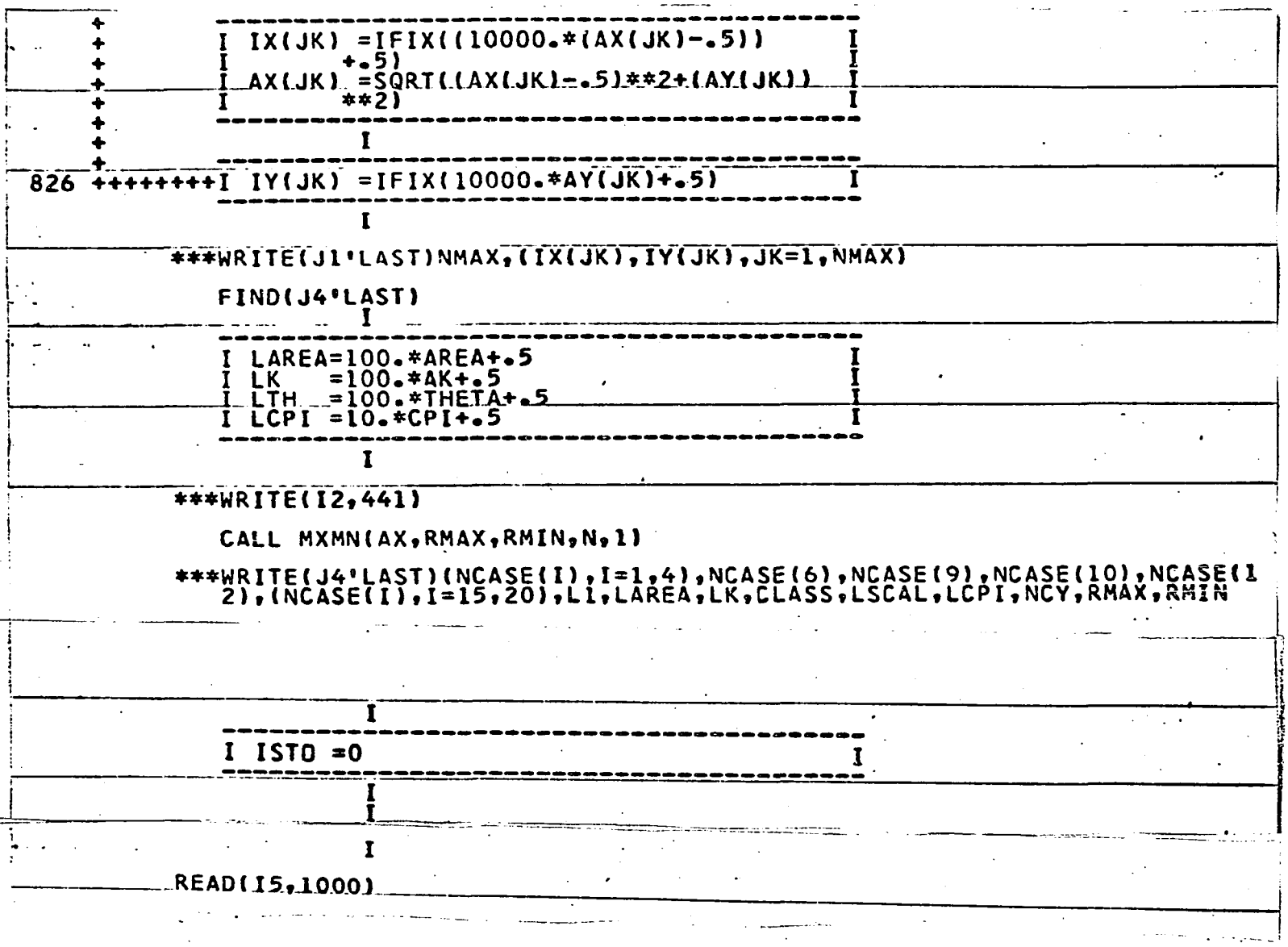

628 ***WRITE(I2,431)
630 CONTINUE
IF (JDAT(6)-1)
- 0 +
785
784
785
785 CONTINUE
SUBTRACT BASELINE
B =BSL(3)-BSL(1)
SLOPE=(BSL(4)-BSL(2))/B
B =(BSL(2)*BSL(3)-BSL(4)*BSL(1))
/B
DO 716
J=1,N
716 S(J) =S(J)-T(J)*SLOPE-B
***WRITE(I2,203) SLOPE,B


XFM EKG
CALL XFM1(1,NMAX,T,S,S1,AREA,AK,AL1,AX,AY,Q,R,O,P)
LSCAL=0
640
625
IF
(JDAT(7)-1)
-
0
+
630
632
630
AUX EKG PLOT ON PLOTTER
632
CALL MXMN(S,SMAX,SMIN,NMAX,1)
CALL MXMN(T,TMAX,TMIN,NMAX,1)
IF
(JDAT(3)-1)

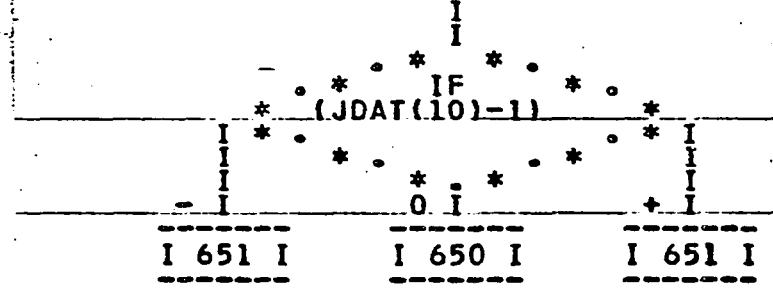

- I
0 I
+ I
I 633 I
I 629 I
I 633 I
633
I SMAX =AAXVL(SMIN,9.249,BX,CPI) I
I TMAX =AAXVL(TMIN,9.910,BX,CPI) I
CALL PLOT2(T,S,NMAX,TMIN,SMAX,SMIN,TMAX,1)
I 630 I
640
IF
(AL1-AL1MX)
- I
0 I
+ I
I 641 I
I 642 I
I 642 I
642
***WRITE(I2,432)AL1MX
IF
(Q+.4)
- I
0 I
+ I


I 793 I
I 792 I
I 793 I
793 ***WRITE(I2,440)
792 CONTINUE
IF
(JDAT(4)-1)
- I
0 I
+ I
I 649 I
I 644 I
I 649 I
644
IF
(JDAT(3)-1)
- I
0 I
+ I
I 645 I
I 646 I
I 645 I
646 ***WRITE(I2 ,436)NCASE
CALL PLOTB(AY,AX,NMAX,57,R,0,P,Q)
***WRITE(I2,437)
I 649 I

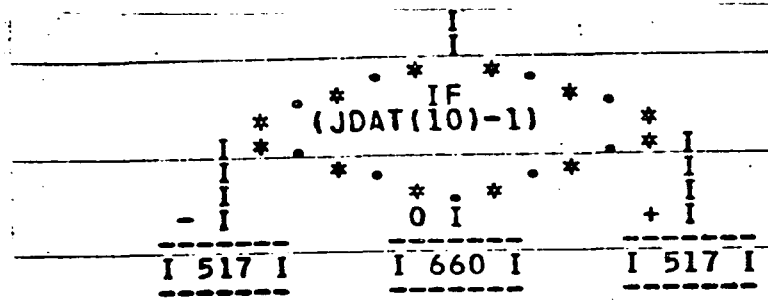

645
IF
(JDAT(9)-1)
0
647
647
647
***WRITE(12,433)
649
IF
(ISTO-1)
0
827
816
827
827
816
L1 =100.*AL1+.5
DO
826
JK=1,N


IX(JK) =IFIX((10000.*(AX(JK)-.5))
+.5)
AX(JK) =SQRT((AX(JK)-.5)**2+(AY(JK))
**2)
826 IY(JK) =IFIX(10000.*AY(JK)+.5)
***WRITE(J1'LAST)NMAX,(IX(JK),IY(JK),JK=1,NMAX)
FIND(J4'LAST)
LAREA=100.*AREA+.5
LK =100.*AK+.5
LTH =100.*THETA+.5
LCPI =10.*CPI+.5
***WRITE(I2,441)
CALL MXMN(AX,RMAX,RMIN,N,1)
***WRITE(J4'LAST)(NCASE(I),I=1,4),NCASE(6),NCASE(9),NCASE(10),NCASE(1
2),(NCASE(I),I=15,20),L1,LAREA,LK,CLASS,LSCAL,LCPI,NCY,RMAX,RMIN
ISTO =0
READ(I5,1000)

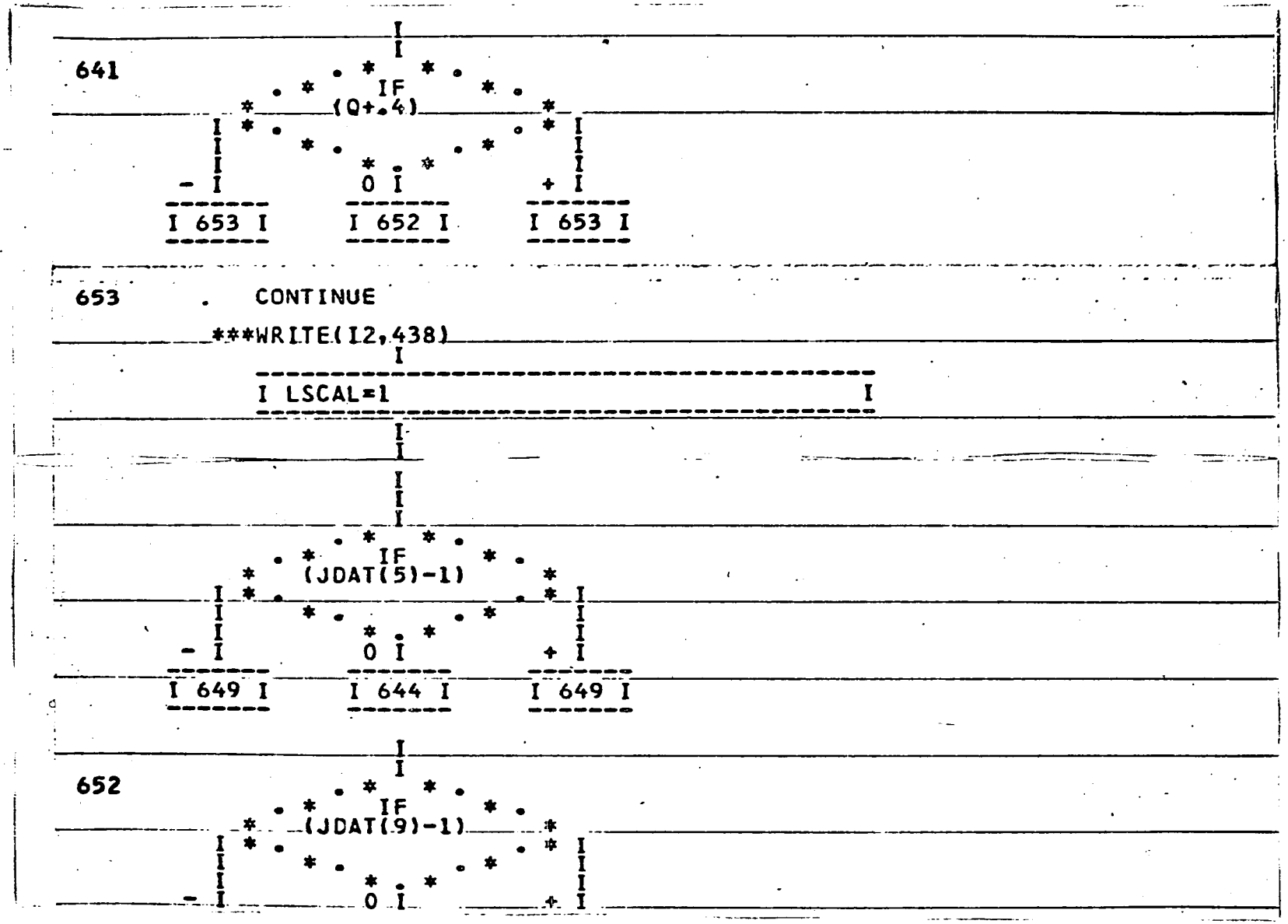
```
      WRITE(I5,434)(NCASE(I),I=1,4),NCASE(6),NCASE(9),NCASE(10),NCASE(12
     ),(NCASE(I),I=15,20),L1,LAREA,LK,LSCAL,LTH,LCPI

827   CONTINUE
      CALL DATSW(9,JDAT(10))
```

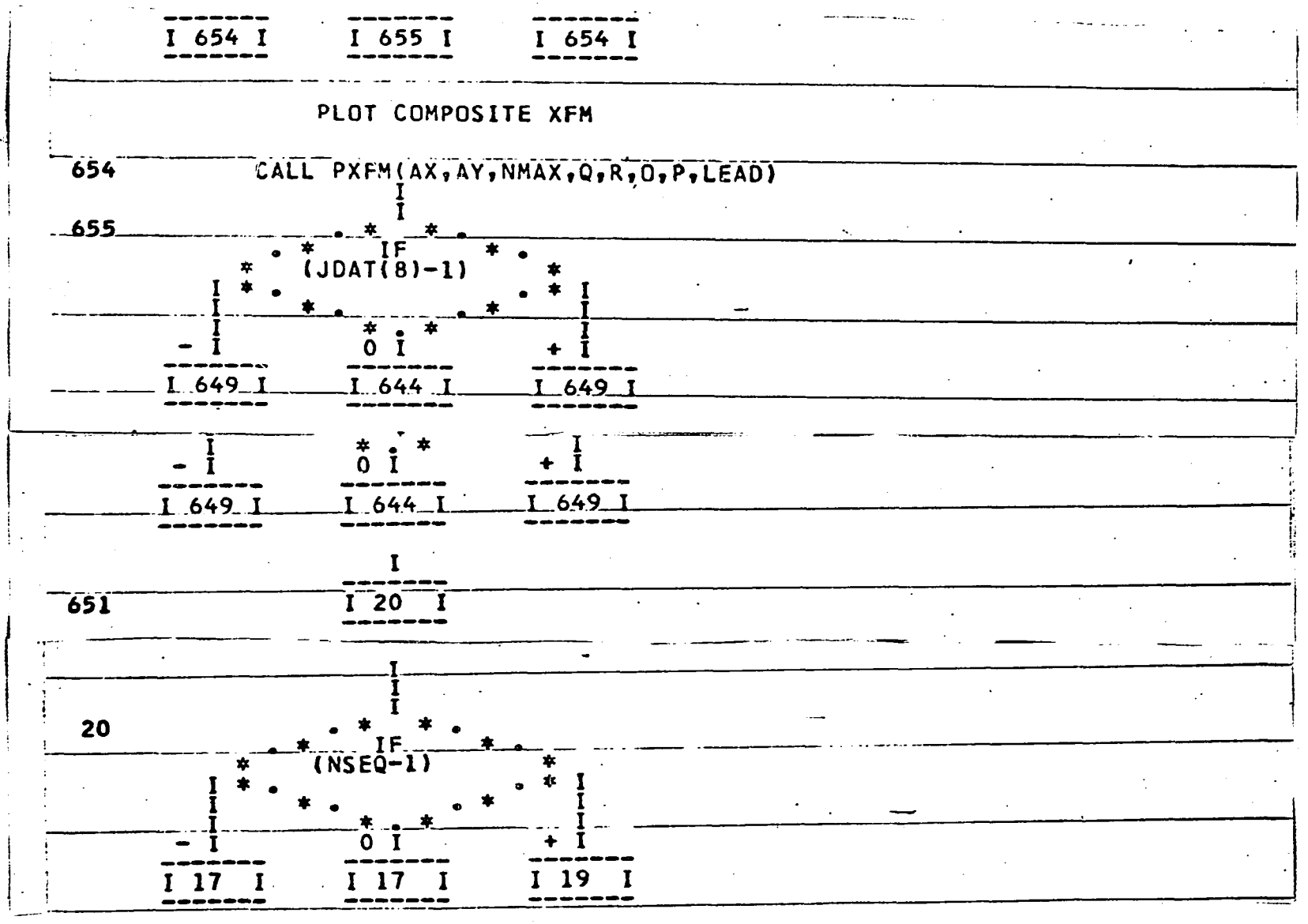


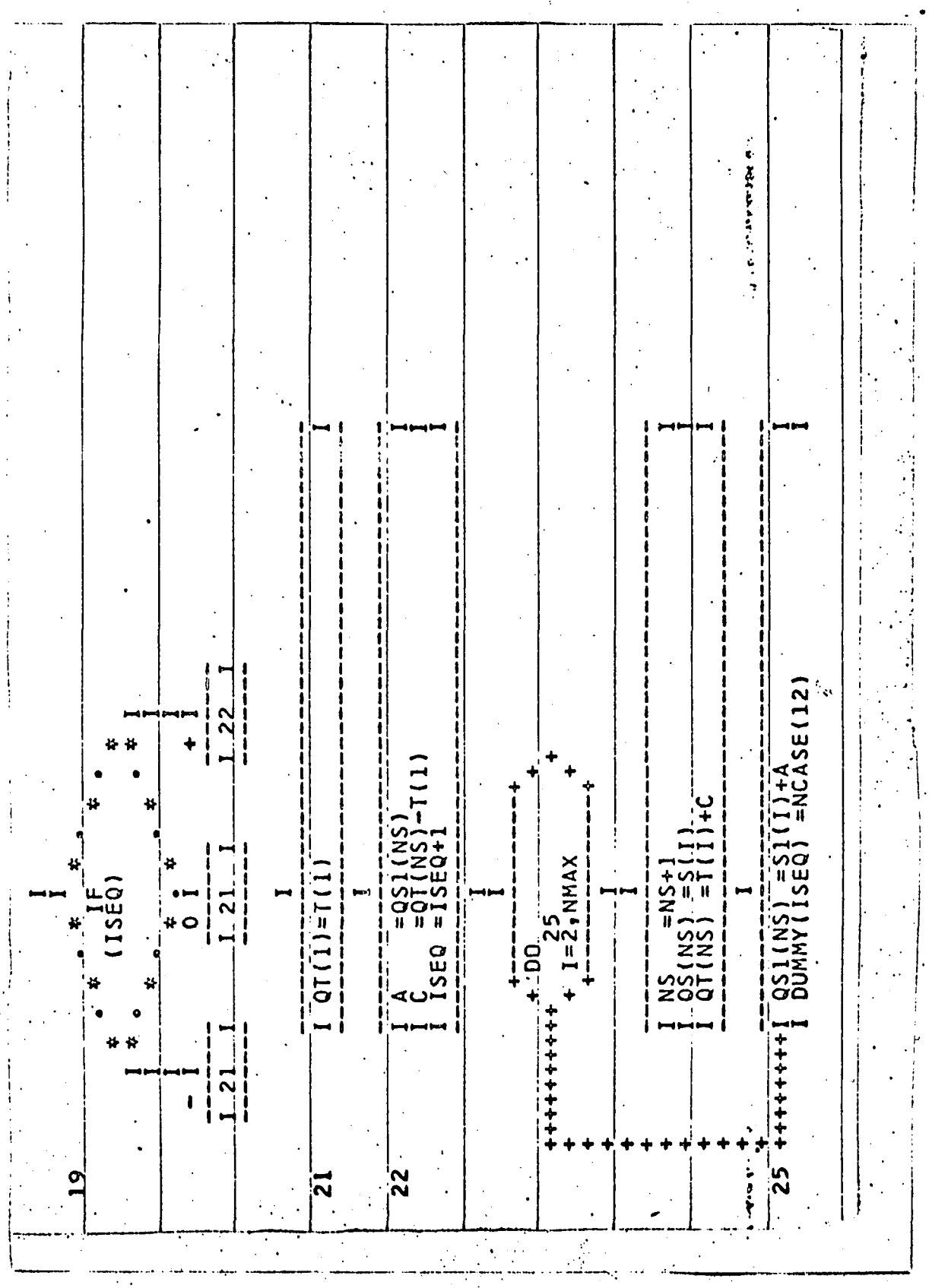

```
650   CONTINUE
     ***PAUSE0003
      CALL DATSW(9,JDAT(10))
```




```
660       CONTINUE
          BL PERT SUBPROG HERE
      ***WRITE(I8,1000)BSL(2),BSL(4)
      ***READ (I7,1000)BSL(2),BSL(4)
                    I
                    I
      ***WRITE(I8,402)
      ***READ (I7,403)(NCASE(I),I=17,20)
                    I
                 -------
                 I 630 I
                 -------

               PLOT XFM ON AUX PLOT

648       CALL PLOT2(AX,AY,NMAX,Q,R,O,P,2)
                    I
                 -------
                 I 649 I
                 -------
```


641
IF
(Q+.4)
-
0
+
653
652
649
644
649
653
652
CONTINUE
***WRITE(12,438)
LSCAL=1
IF
(JDAT(5)-1)
IF
(JDAT(9)-1)


654
655
651
20
PLOT COMPOSITE XFM
CALL PXFM(AX,AY,NMAX,Q,R,O,P,LEAD)
IF (JDAT(8)-1)
IF (NSEQ-1)
- 0 +
649 644 649
649 644 649
20
17 17 19


19
IF
(ISEQ)
0
21
QT(1)=T(1)
22
A =QS1(NS)
C =QT(NS)-T(1)
ISEQ =ISEQ+1
DO 25 I=2,NMAX
NS =NS+1
QS(NS) =S(I)
QT(NS) =T(I)+C
25
QS1(NS) =S1(I)+A
DUMMY(ISEQ) =NCASE(12)

APPENDIX B

Computer Print-out of Omnicardiograms from several cases and exhibits of their respective electrocardiograms.

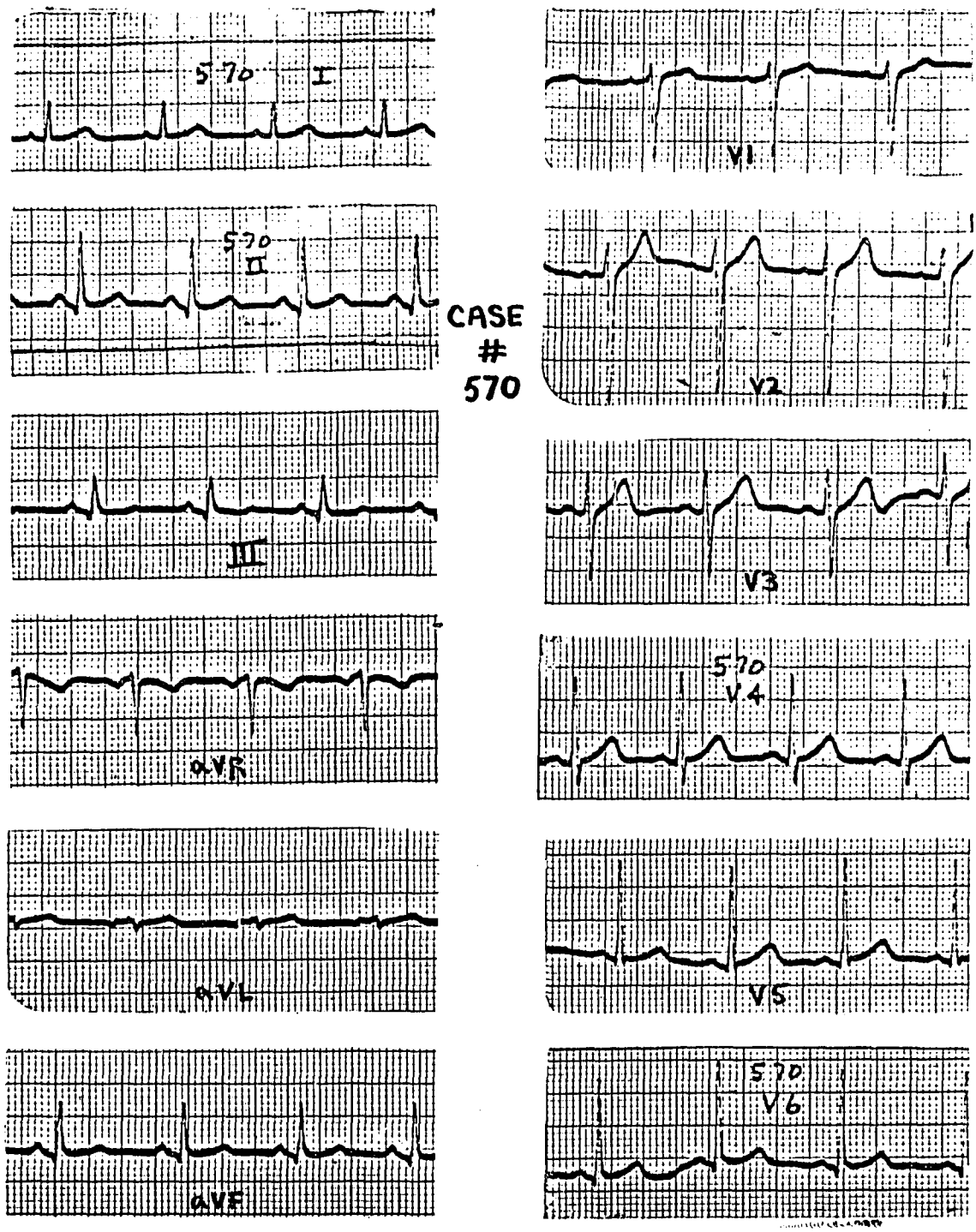


Fig. 9.1 Case #570. Normal Coronary Arteries
Normal EKG

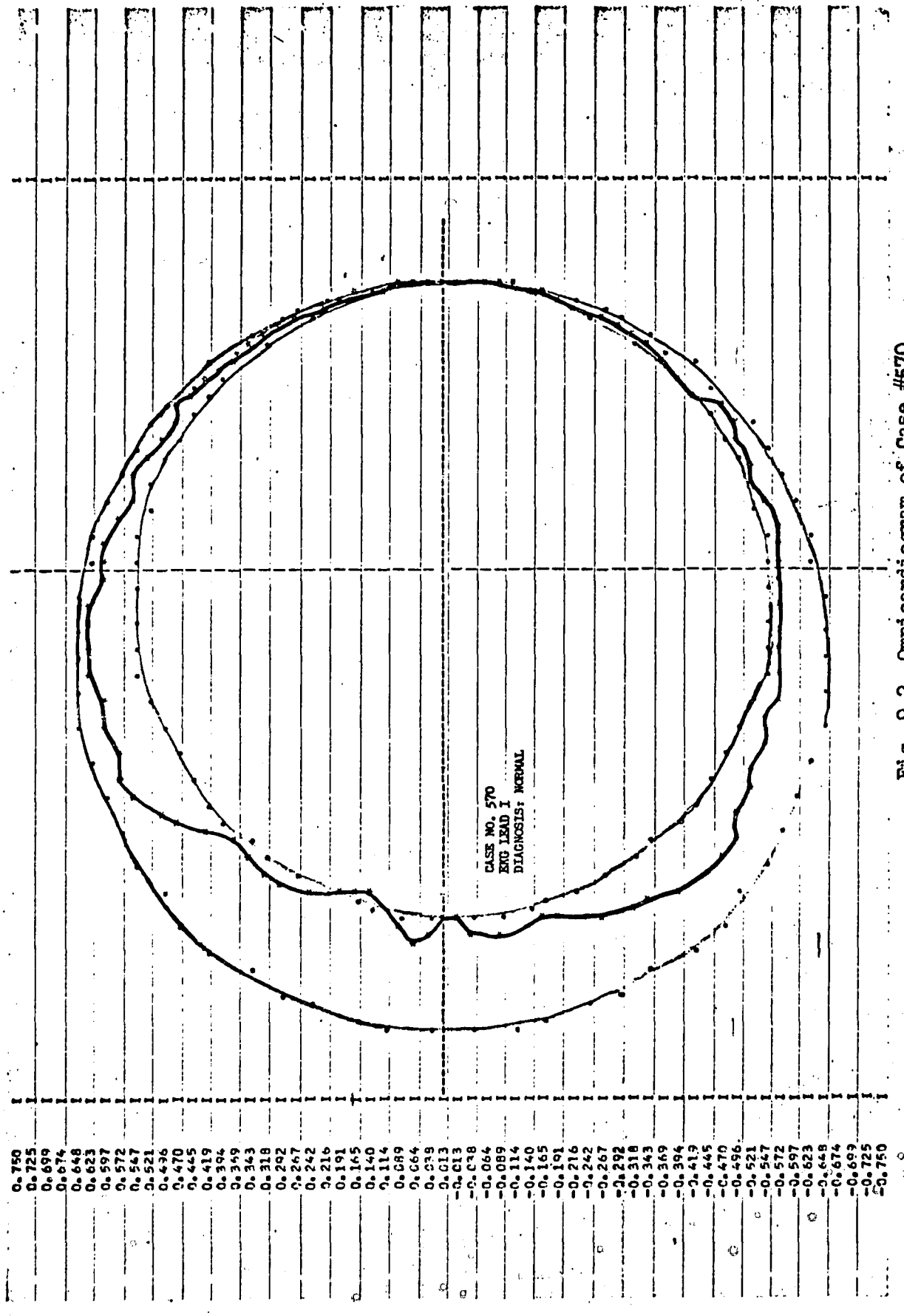


Fig. 9.2 Omnicardiogram of Case #570

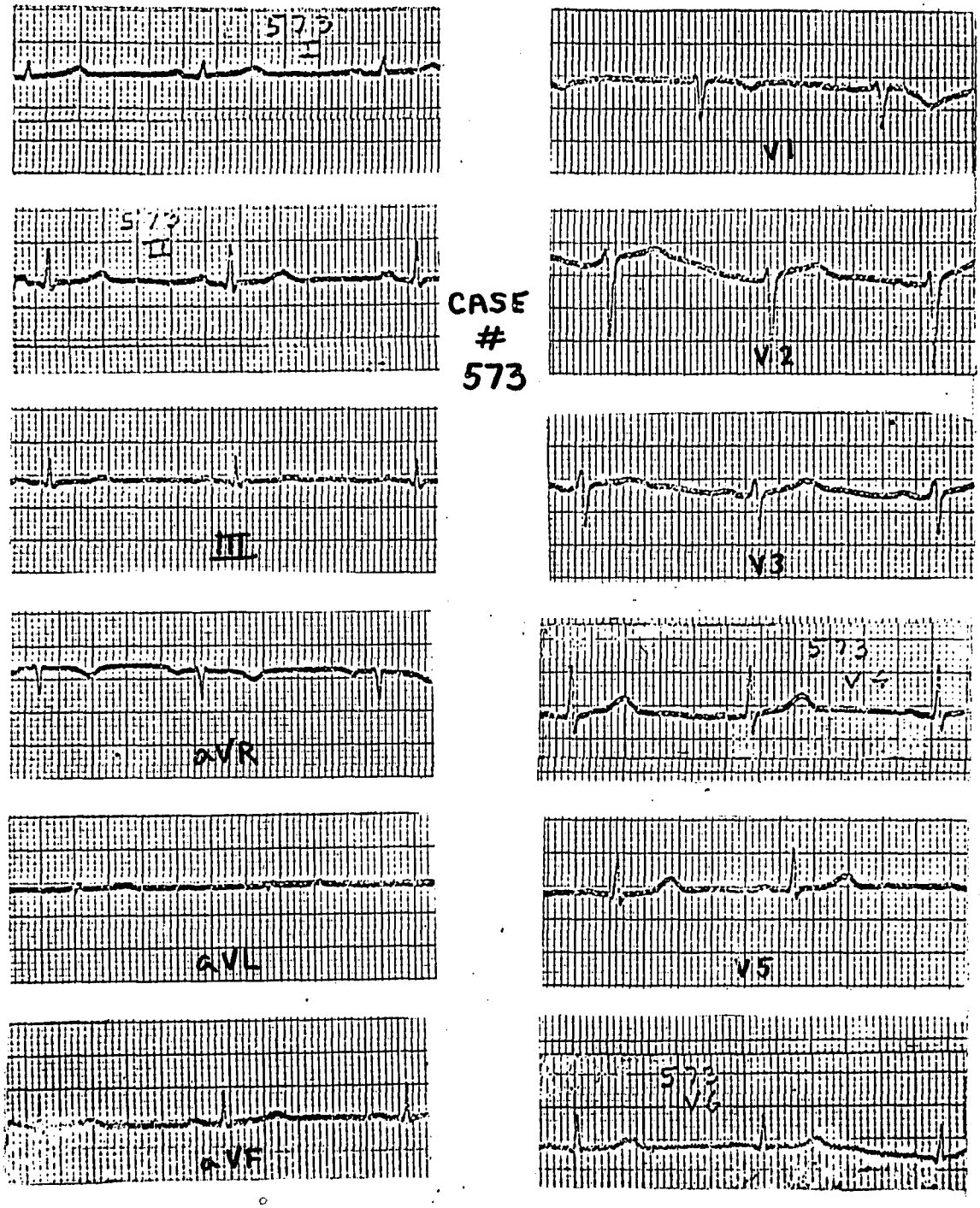


Fig. 9.3 Case #573. Normal Coronary Artery
Normal EKG

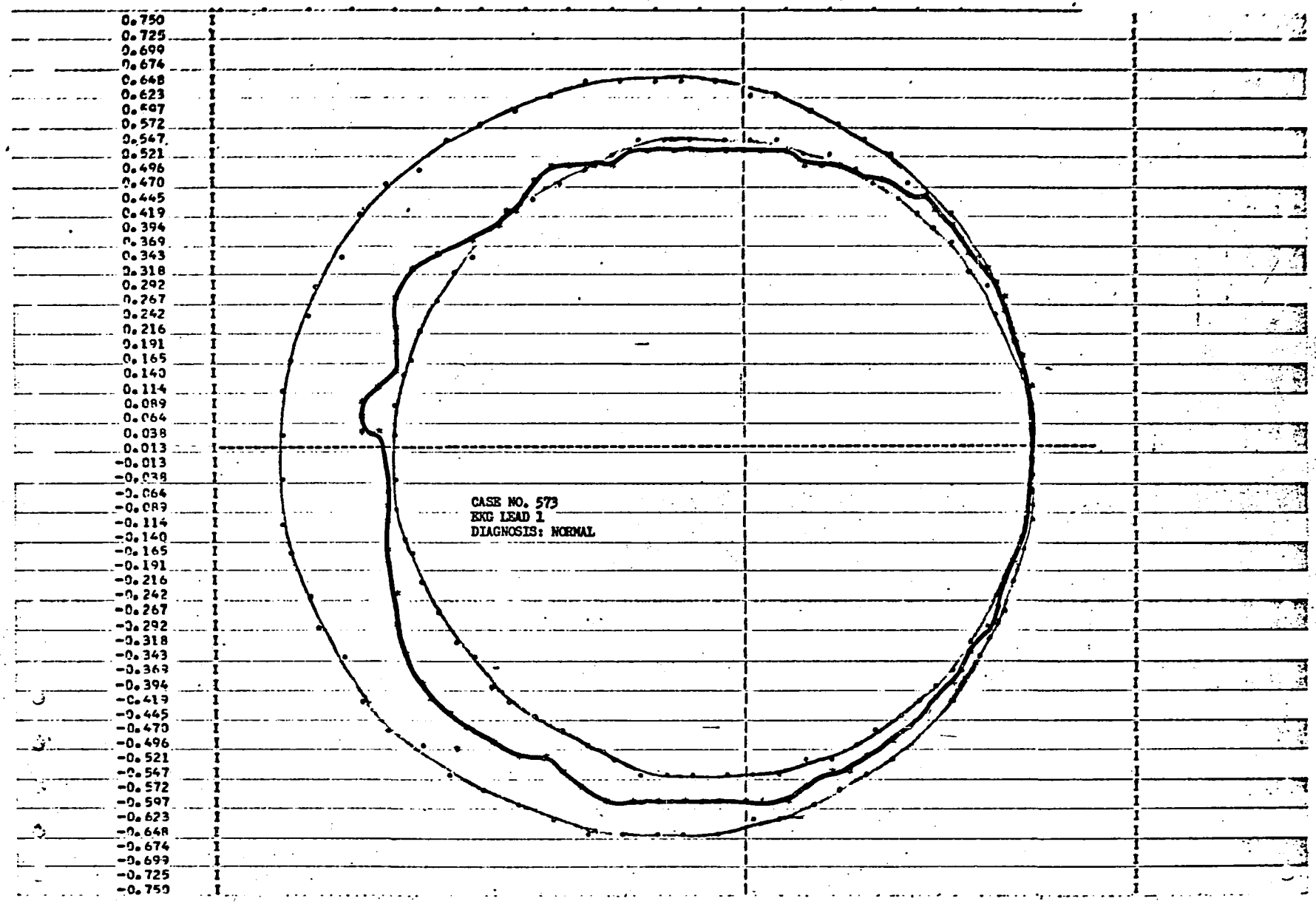


Fig. 9.4 Omnicardiogram of Case #573

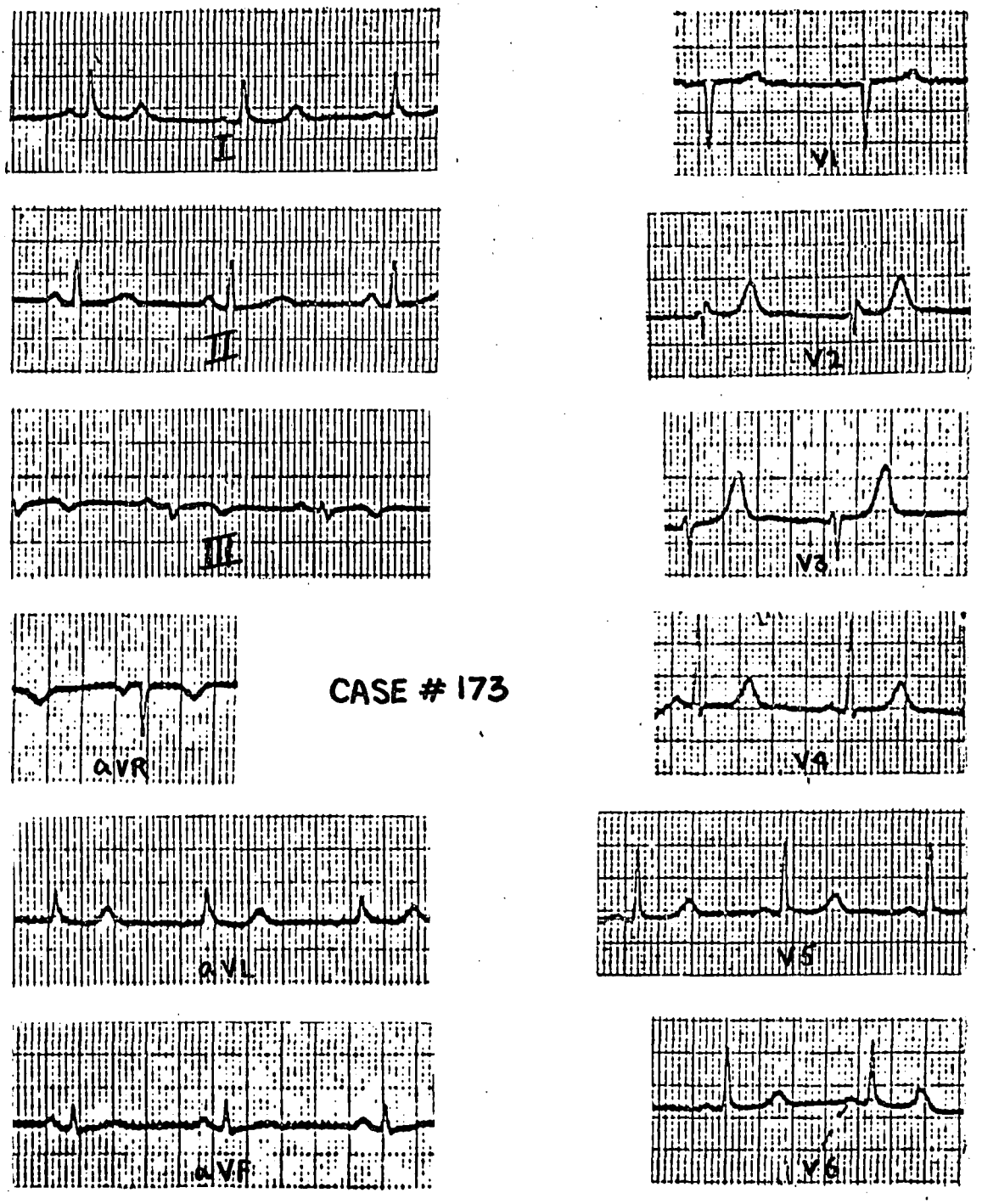


Fig. 9.5 Case #173. Coronary Artery Disease
Normal EKG

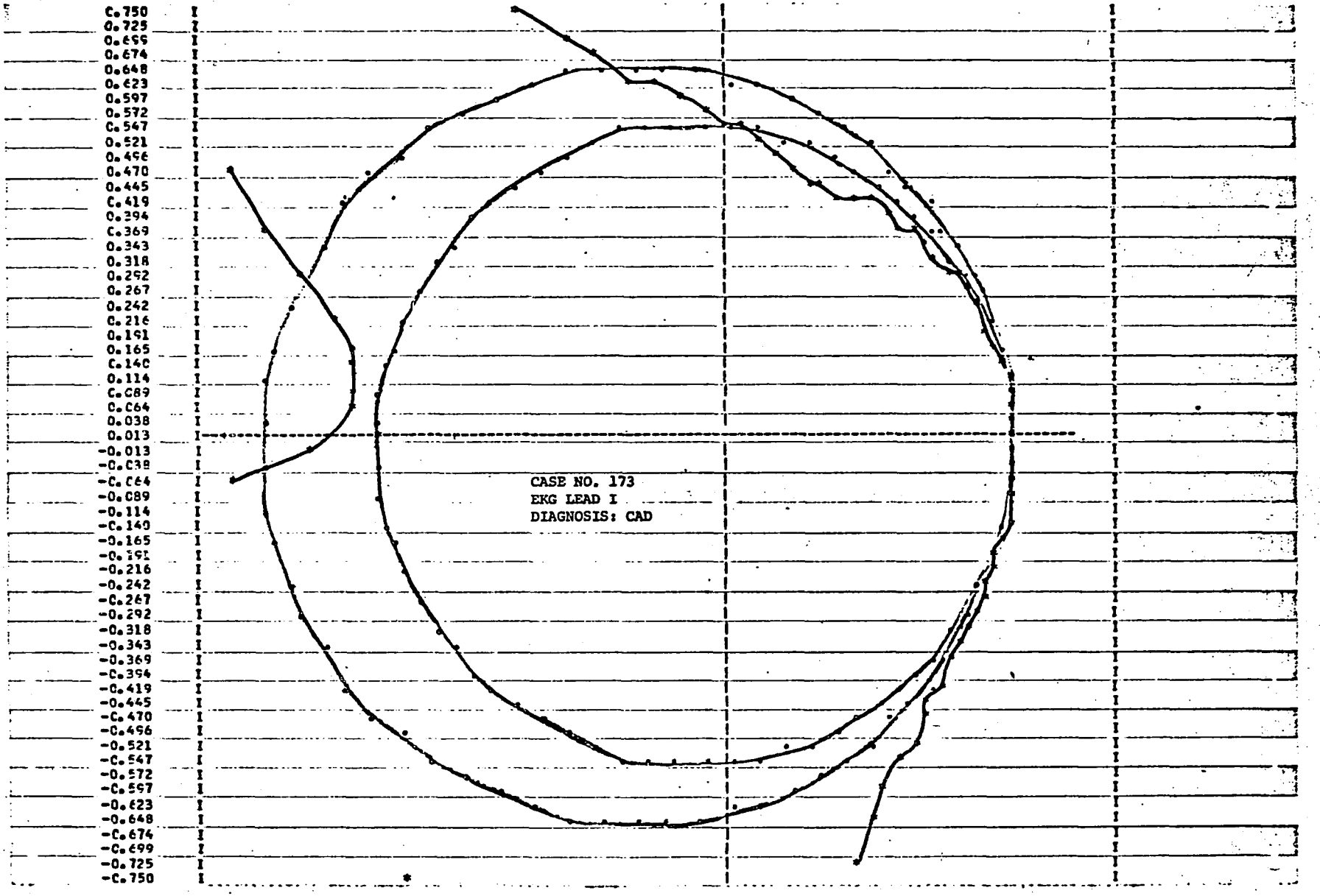


Fig. 9.6 Omnicardiogram of Case #173

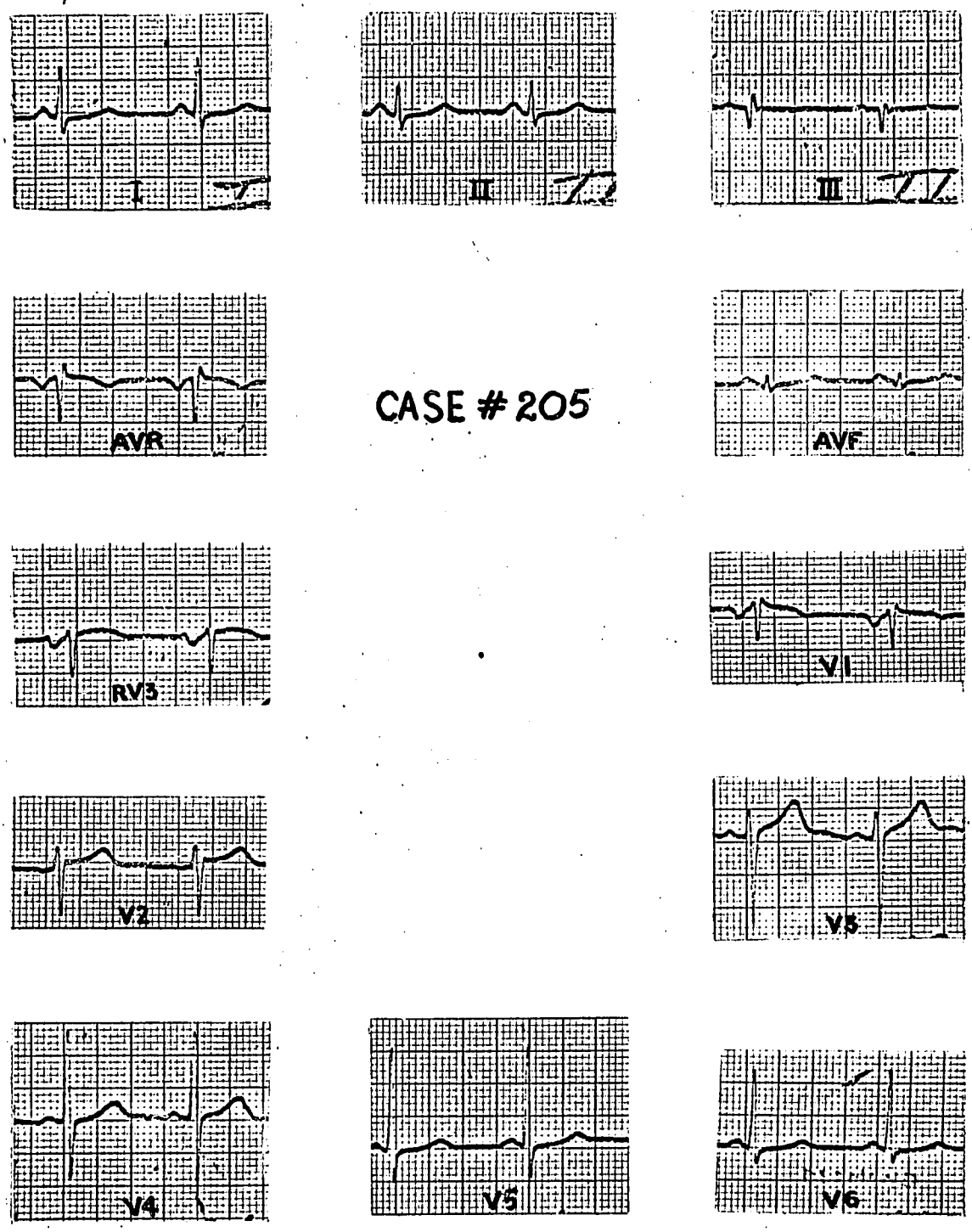


Fig. 9.7 Case #205. Coronary Artery Disease
Normal EKG

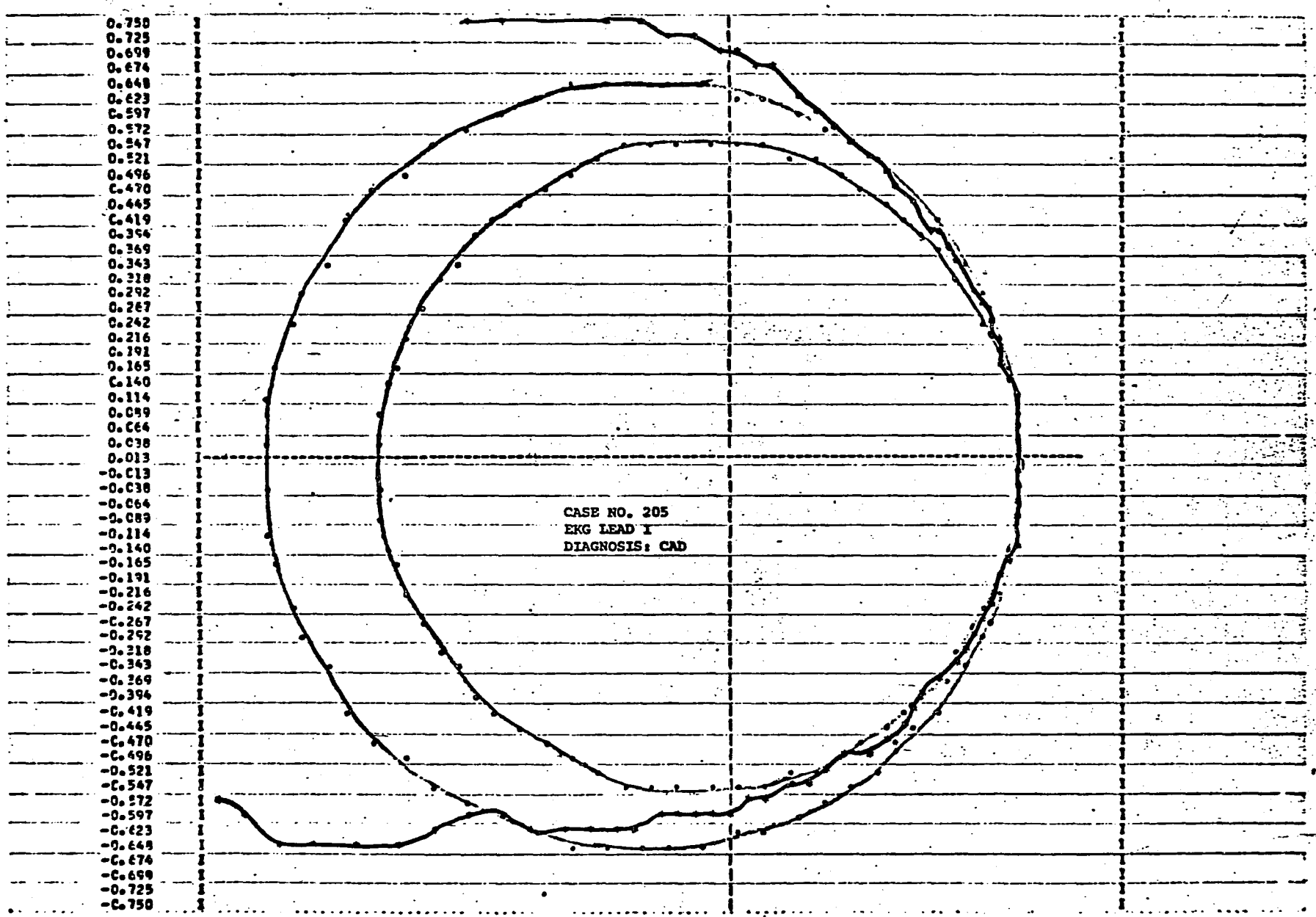


Fig. 9.8 Omnicardiogram of Case #205

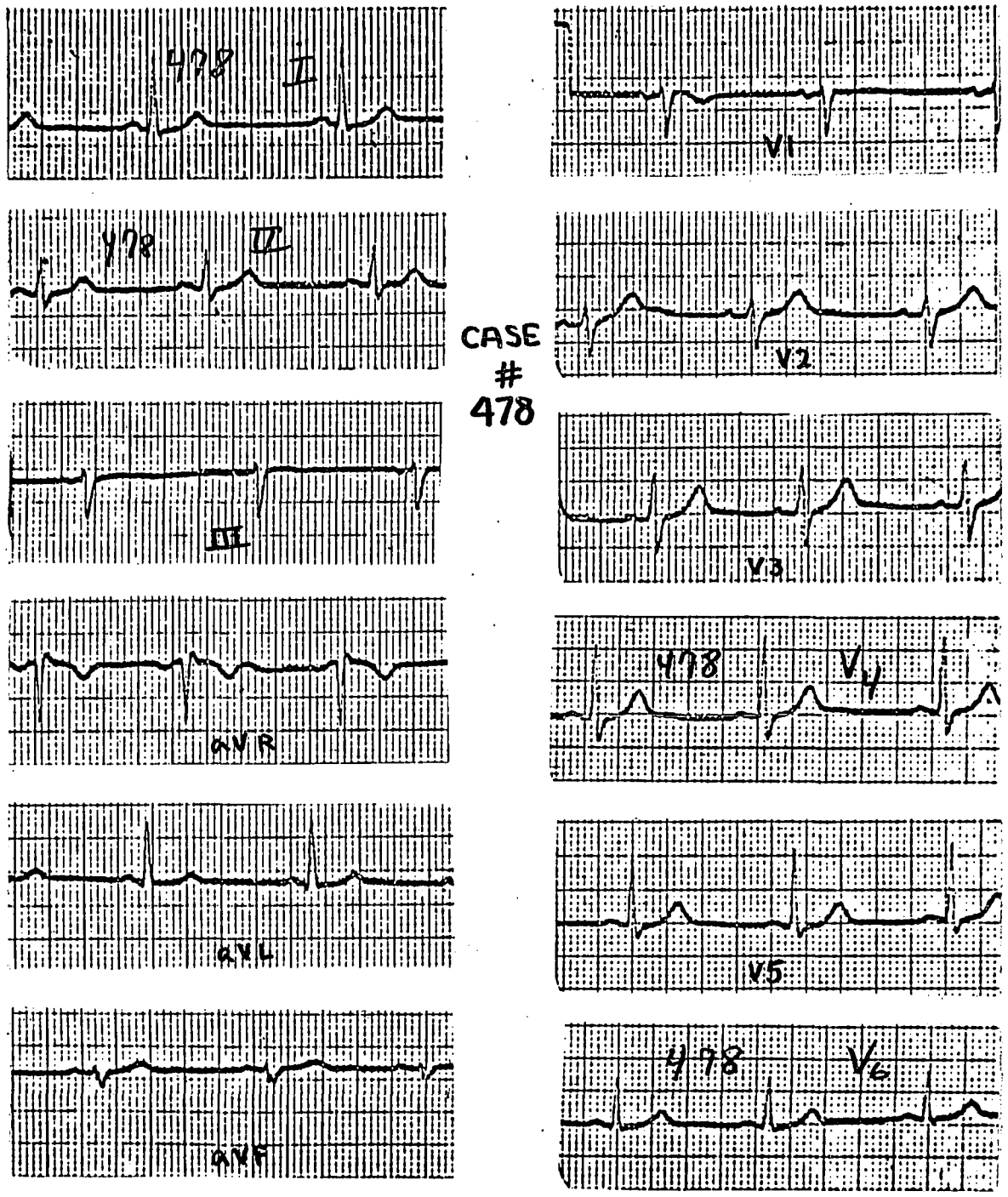


Fig. 9.9 Case #478. Coronary Artery Disease
Normal EKG

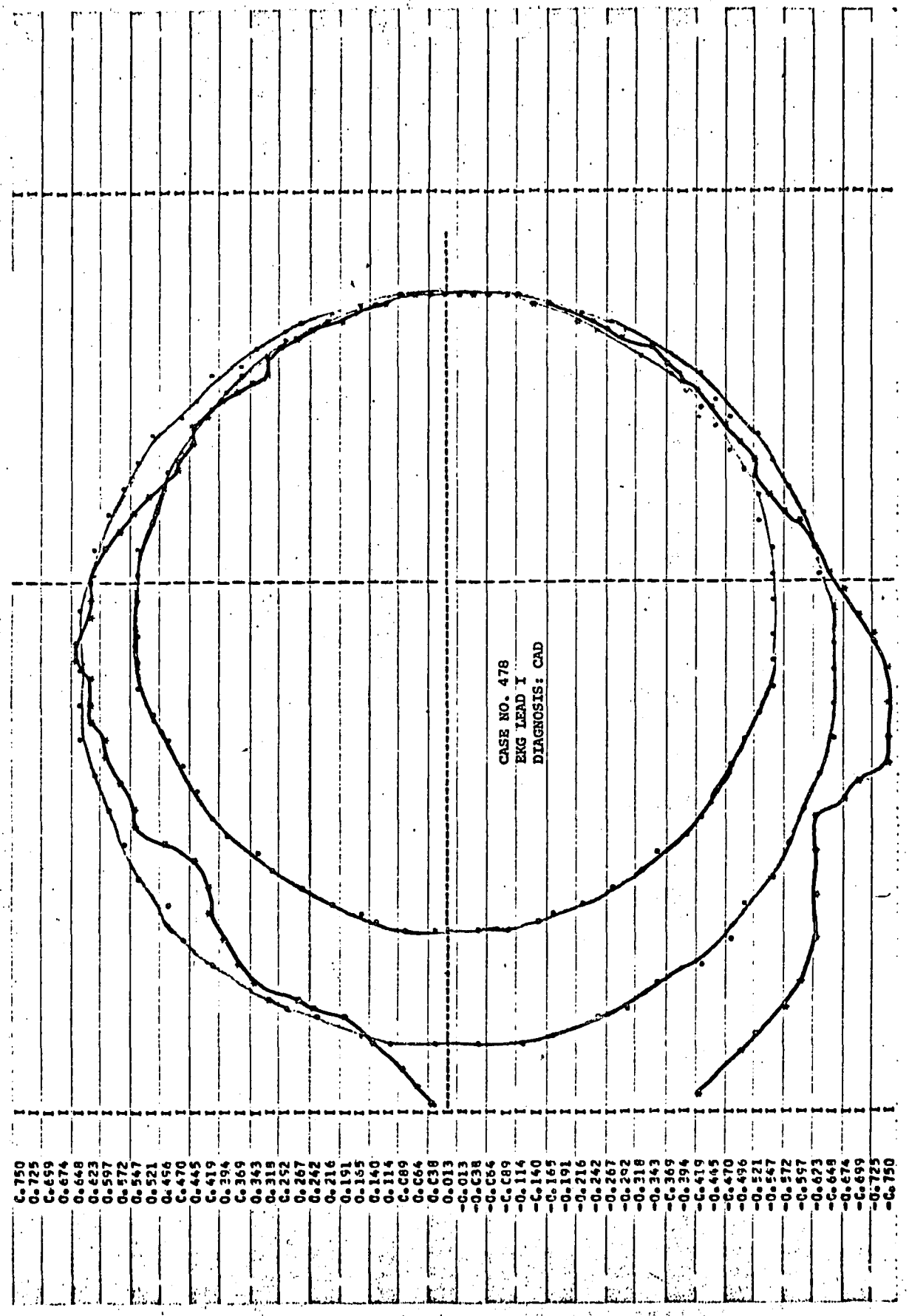


Fig. 9.10 Omnicardiogram of Case #478

VITA

Mr. Joseph R. Levitt was born in in

. He received a Bachelor's Degree in Mechanical Engineering from the Cooper Union School of Engineering, and his Master's Degree in Mechanical Engineering from the Polytechnic Institute of Brooklyn. His professional technical experience spans 17 years involving diverse facets of the aerospace industry in the areas of design engineering, data processing and simulation. Five years of his experience include the teaching of physics and mechanical engineering subjects. He is a licensed professional engineer in the State of New York and is a member of the New York Academy of Sciences and the Institute of Electrical and Electronics Engineers. Mr. Levitt is listed in "American Men of Science" and has presented numerous technical papers in many fields of engineering.