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Electrocardiogram interpretation using correlation techniques

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ELECTROCARDIOGRAM INTERPRETATION USING CORRELATION TECHNIQUES

by

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INTRODUCTION

Concepts of Electrocardiography

<u>History</u>

The inherent ability of muscle tissue to produce and
transmit electric current has been known for many years.
Kölliker and Müller, in 1856, demonstrated the presence of
action currents associated with heart beat by placing a frog's
nerve-muscle preparation in contact with a beating heart and
observing that the frog's muscle twitched with each ventri-
cular contraction of the heart. Later in 1887, Ludwig and
Waller first demonstrated a measurable amount of current in
the human body associated with heart contraction by recording
the electromotive force from the precordium on a capillary
electrometer. Then in 1901 the current from the human heart
was measured in an accurate and quantitative manner with the
introduction of the string galvonometer by Willem Einthoven.
Since that time, mechanical improvements (such as vacuum tube
and transistor amplification, and development of better gal-
vanometric instruments of which the oscillograph and heated-
stylus strip-chart recorder are examples) and improvements in
technique (such as better lead shielding and grounding, lead
placement, and development of diagnostic criteria) have
brought electrocardiography to its present state.

The heart and circulatory system

The circulatory system is a closed-loop system in which the blood is circulated to all parts of the body distributing oxygen and nutrition and collecting carbon dioxide and other waste material. The heart is the "pump" for the circulatory system. It has four chambers. The two smaller chambers at the top of the heart are called the right and left auricles, and the two larger chambers below the auricles are the right and left ventricles. The inner wall of the heart separating the right and left pairs of chambers is called the septum.

The general flow path of the circulatory system is as follows. Starting at the right auricle, the blood is forced by auricular contraction down into the right ventricle. When ventricular contraction occurs, the blood is pumped out through the pulmonary artery to the lungs. It returns from the lungs via the pulmonary veins to the left auricle of the heart where auricular contraction forces it into the left ventricle. It is then pumped, by ventricular contraction, from the left ventricle out through the aorta and arteries to the capillary system throughout the body. The blood is col-lected from the capillary system by the veins and is returned to the right auricle via the venae cavae. It may be noted that the left ventricle is 2-1/2 to 3 times thicker than the right ventricle because it is more muscular due to the fact that it must pump harder to distribute blood to the capillary systems in the brain, the organs, and throughout the entire

body. As a consequence, overwork or strain of the left
ventricle, called left ventricular hypertrophy, is a fairly
common malady of the heart and is much more frequently found
than right ventricular hypertrophy.

Physiology of the heart

Polarization and dipole theory   Muscular activity,
such as contraction and relaxation of the heart, is associated
with electrical activity. The electrocardiogram is merely a
representation of this electrical activity associated with the
heart. Cell polarization and basic dipole theory are dis-
cussed in some detail in Burch and Winsor (1) and in Lipman
and Massie (2).

Living resting cells have a series of electrical dipoles
(a positive and negative charge of equal magnitude in close
proximity), or doublets, along their walls. This was demon-
strated by Curtis and Cole by placing electrodes on a single
resting cell (giant axon of the squid and the large cell of
the plant Nitella) and measuring on a galvanometer a potential
on the outer surface of about 50mv positive with respect to
the inner surface. This potential difference across the
membrane surface is called the "membrane resting potential"
and shows that an electromotive force exists across the rest-
ing membrane. Metabolic (life) processes maintain these
electrical dipoles probably by means of chemical processes.

A resting cell is said to be in a polarized state meaning

that an equal number of ionic charges of opposite polarity exist on the inner and outer surface of the membrane. No current is seen unless an injury occurs (changing polarity of the surface) or an electrical stimulus of some kind appears causing contraction. Both have an associated change in permeability of the membrane surface. Since chemical and electrical energy are both potentially reversible, an electrical stimulus causes the membrane to become depolarized (causing contraction), but the chemical reversibility causes a repolarization (relaxation) to follow. Repolarization generally occurs more slowly than depolarization. Injury, however, may cause a permanent partial depolarization in the injured tissues giving rise to so-called "currents of injury". Temperature and some chemicals also affect depolarization and repolarization processes.

The heart itself is made up of thousands of these polarized cells formed into muscle elements. The electrical activity of each element may be represented by a vector and the vector sum of these individual electrical vectors represent the electrical activity of the heart.

Electrical action of the heart   An electrical stimulus pulse which initiates each normal heart cycle originates in a collection of specialized tissue called the sinoauricular (SA) node found in the right auricle (see Figure 1). This pulse "travels" through both auricles in a wavelike manner causing auricular contraction which forces blood from the auricles
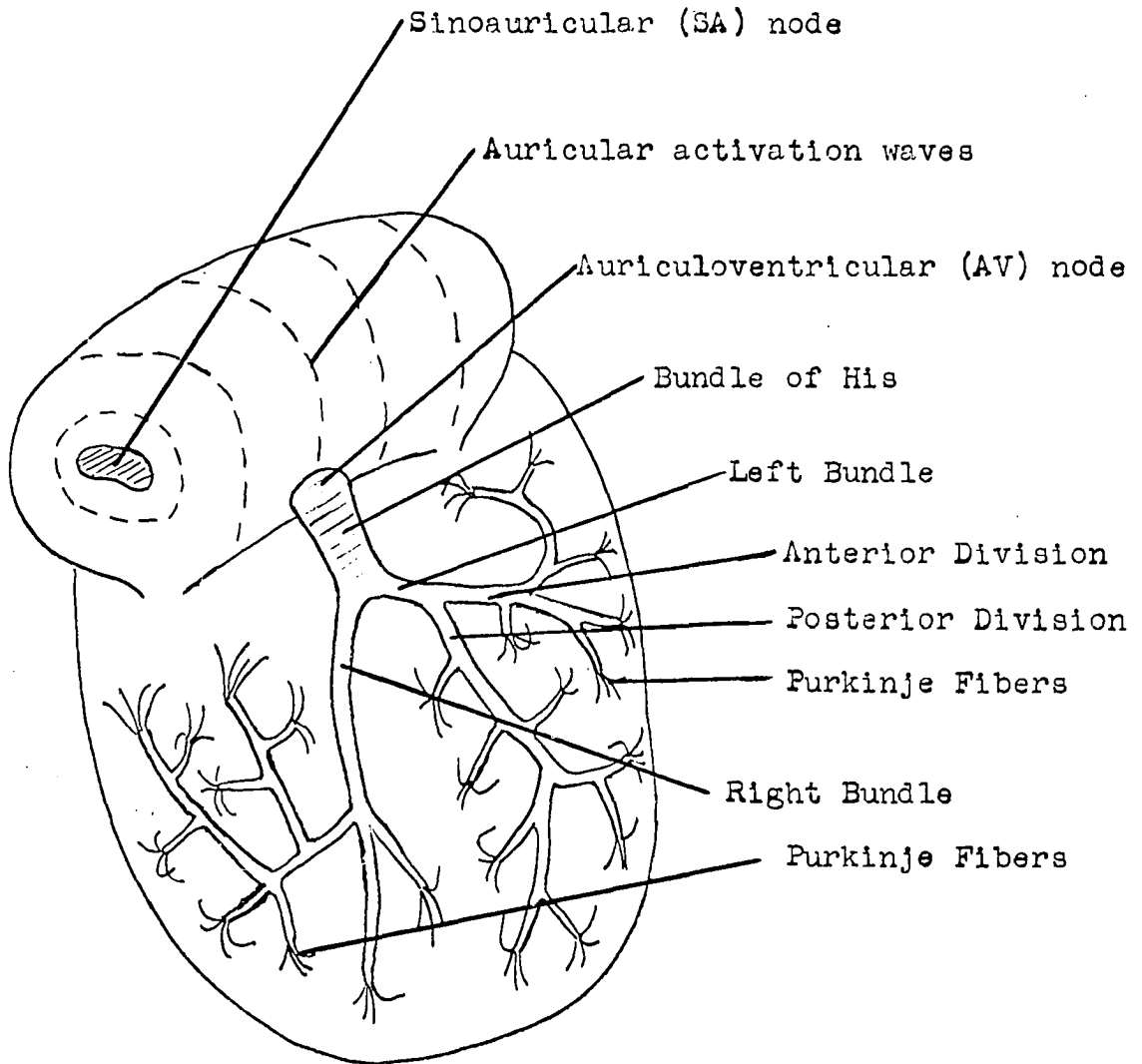
Figure 1. The heart and its conductive tissues

into the ventricles. The electrical pulse then reaches another group of specialized tissue called the auriculoventricular (AV) node located in the septal wall near the auricles. The AV node delays the pulse (during which delay the ventricles are filling with blood), then transmits the excitation pulse into a muscle system called the bundle of His in the interventricular septum, depolarizing it from its left to right side. The pulse then proceeds along the bundle of His which splits into two main branches—a right and a left branch (see Figure 1). The right branch runs along the interventricular septum almost to the apex of the right ventricle. Then it branches into subsidiary twigs which transmit the pulse to a muscle network called the Purkinje system which spreads throughout the right ventricle. Contraction of the right ventricle occurs when the pulse spreads through this Purkinje system. The left branch of the bundle of His almost immediately divides into an anterior and a posterior branch. These branches then subdivide into subsidiary twigs in the anterior and posterior regions respectively of the left ventricle. (Recall it was previously mentioned that the left ventricle is more muscular due to the heavier work load placed on it.) The pulse then travels on through its Purkinje system causing contraction of the left ventricle. Ventricular contraction proceeds from the endocardial (inside) surface to the epicardial (outside) surface.

Thus, in summary, the electrical impulse initiated in the

SA node of the right auricle travels through the auricles to the AV node and then through the bundle of His along its two main branches and subsidiary twigs to the Purkinje system; stimulating the auricular, septal, and ventricular musculatures in a normal cycle. The sequence and timing of the appearance of the stimulus is the key which makes the electrocardiogram useful for diagnosis of some heart injury and malfunction. It is a subtle point but it should be remembered that contraction and relaxation of heart musculature result from the electrical stimulus and not vice versa.

The electrocardiogram

The human body, due to the chemical nature of its fluids, is essentially a volume conductor, that is, a medium which conducts electricity in three dimensions. Thus, a current generated in any part of the body will be conducted to all other parts, being terminated only by the body surface. Various methods may be used to measure the magnitude and direction of the current (produced by potential differences originating in the heart) at various places on the surface of the body keeping in mind that the electric potential is inversely proportional to the square of the distance from the source.

An electrocardiogram (sometimes abbreviated to EKG or ECG) is a graphic representation of the electrical activity of the heart. It is usually recorded by placing electrodes at

various places on the surface of the body and employing some
sort of galvonometer instrument to record the potential varia-
tions in analog form. The ECG then, due to the remote
location of the electrodes from the heart, actually records
potential variations of the electric field of the heart
instead of the actual potential differences at the heart's
surface. It is also important to remember that the electrical
activity displayed at the various electrodes is a result of
the potential variations of the whole heart and not just the
activity of some isolated zone of the heart. These potential
variations recorded are then a superposition of the electrical
activity of all the muscle fibers of the heart. The signal
recorded is, however, greatly affected by the electrical
activity of the surface of the heart nearest the electrode.
Also, the interventricular septum, because of its near sym-
metry and approximately equal and opposite electrical activity,
normally exerts negligible influence on the electrodes except
for a septal Q wave which may appear at electrodes placed in
certain areas of the body surface.

The conventional clinical electrocardiogram in common
clinical use today is the so-called scalar electrocardiogram.
It consists of twelve leads: three bipolar extremity leads,
three unipolar extremity leads, and six unipolar chest leads.
The actual placement of these leads is shown in both Burch and
Winsor (1) and in Lipman and Massie (2). The 12-lead ECG has
as its basis the empirical criteria which have been derived

through its use and have been found to be useful in its interpretation. Recently, more scientific methods to record the electrical activity of the heart have been advanced but they are still experimental and have not yet been accepted into common usage in clinical medicine due to the difficulty in their recording and also because of their lack of associated diagnostic criteria. Two of these experimental methods are so-called three-orthogonal-lead systems proposed by Schmitt (3) and Frank (4) used for vectorcardiography.

While the electrocardiogram is a very valuable tool in diagnosing certain heart conditions, it must be kept in mind that it is an indirect picture of the functional and anatomic state of the heart. Some maladies of the heart such as murmurs and other malfunctions of heart valves are not seen (or recognized) in the electrocardiogram at the present state of its development. The electrocardiogram may also be influenced by many things. The heart itself may be affected by such things as drugs, infections, pain, fear, exercise, shock, blood conditions, drinking cold water, excessive amounts of coffee, smoking, and other factors. Also, while the heart may be functioning completely normally, the recorded electrocardiogram may be influenced by skin resistance, heterogeneity of the conducting tissues of the body, body build, heart position within the thoracic cage of the chest, polarization, skeletal muscle tremor, electrical interference, recording techniques, and other factors. However, most of these factors,

whether affecting the heart itself or just the ECG; may be avoided, minimized, or accounted for in interpretation of the ECG. An abnormal tracing almost always implies that the heart is functioning abnormally. Also abnormal heart function causes an abnormal ECG recording if the electrodes are properly positioned on the body with respect to the location of the injury in the heart.

The typical electrocardiogram waveform

The component parts of a typical electrocardiogram have been designated arbitrarily by Einthoven in order of their appearance in the cycle as the P wave, the QRS complex, the T wave, and the U wave (see Figure 2). These pulses or waves that make up the typical ECG cycle are recordings of the electrical contributions of both the auricles and ventricles of the heart.

The auricular contribution   The P wave represents the depolarization wave of the auricles which spreads in a wave-like fashion from the SA node to the AV node. It may be positive going, negative going, diphasic (both positive and negative going), or isoelectric (zero potential).

The PR (PQ) segment represents the delay in transmission of the impulse at the AV node. It is usually zero potential.

The PR (PQ) interval represents the time required to depolarize the auricular musculature plus the delay time encountered at the AV node until the beginning of
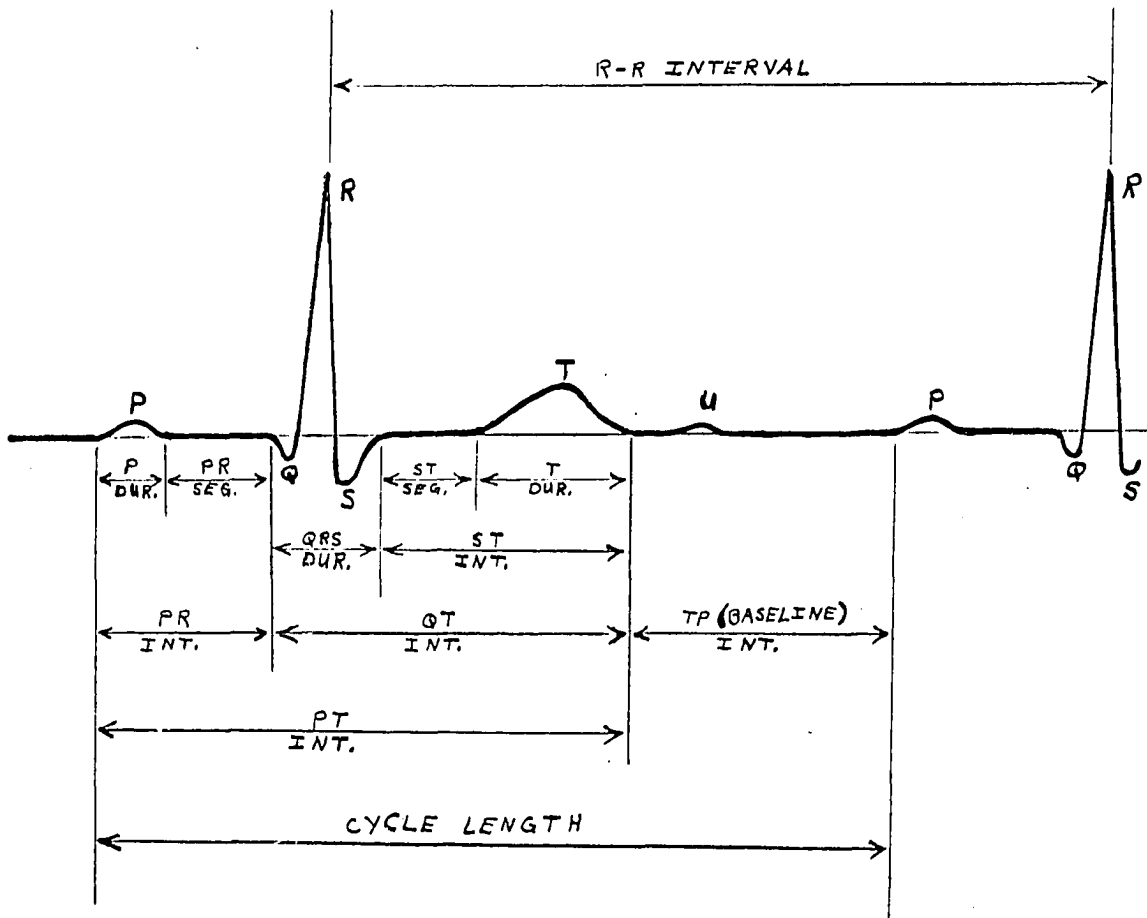
Figure 2. A typical electrocardiogram cycle

depolarization of the ventricles. The repolarization wave of the auricles (called the auricular T wave) is very low in amplitude and is not usually seen. It is included in the PR segment and in the QRS complex. The PR interval, then, is the result of the electrical activity of the auricles.

The ventricular contribution   The QRS complex represents the depolarization wave of the ventricles. It usually consists of an initial downward deflection (Q wave), and initial upward deflection (R wave) and another downward deflection after the R wave (S wave). While the QRS complex is always present in each heart cycle, it may take other forms such as QR, RS, RSR', or others.

The ST segment represents the duration of the depolarized state of the ventricles. It is the time between the completion of depolarization and the start of repolarization while the chemical processes of depolarization are trying to reverse themselves. The ST segment is normally zero potential although it may be either elevated above or depressed below the zero potential baseline.

The T wave represents the repolarization wave of the ventricles. Like the P wave, it may be positive, negative, diphasic, or isoelectric.

The ST interval is the time from completion of depolarization of the ventricles to the completion of their repolarization.

The QT interval represents the entire time needed for

depolarization and repolarization of the ventricles. It is thus the result of the electrical activity of the ventricles and corresponds to the PR (PQ) interval for the auricles.

The "after-potential" The U wave represents an "after-potential" wave of the heart cycle. It is usually of very low amplitude and is generally ignored for diagnostic purposes.

The rest-time The TP interval represents the time between heart cycles when no electrical activity is taking place in the heart. It is therefore considered to be zero potential or the DC level of the heart beat. Its primary use is in determining heart rate.

The entire heart cycle While a heart cycle is usually thought of as beginning with the start of the P wave and lasting until the start of the P wave of the next cycle; it is easier, from a practical standpoint, to find and measure the time between R wave peaks. The period of the heart cycle is therefore often called the R to R interval or simply the RR interval.

Heart disease and the electrocardiogram

As pointed out previously, not every malfunction of the heart can be detected by the electrocardiogram. However, many of the more commonly diagnosed heart ailments do appear in the ECG thus making it an extremely valuable diagnostic tool when properly used and understood. These heart malfunctions detected in the ECG may cause an unusual voltage amplitude,

an unusual time duration for a heart function to occur, or a
combination of these.

Left ventricular hypertrophy previously described as
overwork or strain of the left ventricular musculature, for
instance, appears in standard lead I (a bipolar extremity lead
of left wrist potential with respect to right wrist potential)
as an unusually high amplitude R wave and generally a
depressed ST segment. Right ventricular hypertrophy (the
corresponding malady in the right ventricle), on the other
hand generally appears in standard lead I as a relatively low
amplitude R wave and an abnormally high (or negative going) S
wave. Left bundle branch block, a block in the left branch of
the bundle of His delaying the excitation pulse from continu-
ing on down the septal wall and into the left ventricle,
causes the time duration of the QRS complex to become unusu-
ally long and produces a long, low amplitude R wave in standard
lead I. Right bundle branch block (the corresponding block in
.the right branch of the bundle of His) also causes an abnor-
mally long QRS time duration but with a long, low amplitude S
wave appearing in standard lead I. By knowing lead placement,
the electrical activity of the heart, and its corresponding
affect on the electrocardiogram, one can thus generally deduce
from an abnormal ECG what type of malady produced it and
approximately where it is located.

Purpose and Objectives

The purpose of this dissertation is to present the results of an investigation carried on in cooperation with the United Heart Station at Iowa Methodist Hospital in Des Moines, Iowa. One of the primary studies sponsored by a research grant awarded to Iowa Methodist Hospital is electrocardiogram analysis utilizing digital computers. To date, four different attacks on this problem of electrocardiogram analysis have been undertaken. Balm (5) reports on a study using probabilities from a symptom-disease matrix in the Bayes' rule equation. Gustafson (6) tells of the development of a computer program which inputs amplitude and real-time parameters measured manually from pediatric electrocardiograms and performs an interpretation through the use of sequential decision logic. A similar program has been developed for adult electrocardiograms and is presently being published by Dr. L. F. Staples. Barker (7) describes an investigation of electrocardiograms in the frequency domain applying the Fourier Series expansion for a periodic signal. Mericle (8) and Townsend (9) describe some studies of the application of correlation techniques to electrocardiogram interpretation.

This dissertation presents a study of the use of correlation methods with a single electrocardiogram lead to diagnose certain abnormalities of the heart from "normal" heart functions and to distinguish between several of the more frequently diagnosed abnormalities. It is, thus, an extension and more

detailed study of the work proposed by Mericle and by Townsend.

Mericle proposed correlation as a useful technique for the electrocardiogram analysis problem and showed results from correlating P waves and T waves (using sinusoidal wave approximations) and QRS complexes (using triangular wave approximations). Townsend later used some actual ECG data and correlated entire heart-cycle waveforms to see if he could screen abnormal waveforms from "normals". This dissertation extends these investigations by improving upon the time normalization procedures which limited the range of heart rates used, by investigating whether or not the abnormal waveforms can be subclassified into their own respective disease classifications, and by further studying the use of both a simple highest-correlation comparison technique and a sequential decision-making threshold-logic comparison technique for use with the correlation methods to make the classifications.

It is not the objective of this dissertation to show that any one electrocardiogram lead is sufficient to diagnose all heart malfunctions. Nor is it an objective to describe in detail the hardware which would be required to implement such a method of electrocardiogram interpretation. It is, however, the intent of this study to investigate the usefulness and practicality of the correlation technique in this specialized application of computer interpretation of electrocardiograms.

REVIEW OF LITERATURE

In recent years, the interdisciplinary field of bio-medical engineering has come into prominence. In the area of bionics, biologists are assisting engineers in attempting to model man-made tools after the natural phenomena found in nature; examples being the fly's eye, the bee's homing ability, the heat sensing devices of certain rattlesnakes, the electrical distortionless delay line characteristics of parts of the heart, etc. On the other hand, engineers are cooperating with the medical profession to produce new and improved medical equipment and to apply engineering methods and tools to medical applications. Some examples of this are medical information retrieval applications, the development of new and better medical tools and instruments, development of artificial organs, diagnosis aids, etc.

Ledley (10) discusses using electronic computers in the area of medical diagnosis in 1960 stressing a need for a sound mathematical foundation especially in the fields of logic, probability, and some form of value theory. Later, Gustafson and associates (11,12) discuss this topic further giving examples of some advantages found in using computers as diagnostic tools and showing some results obtained using conditional probabilities. Bendat (13) talks of using statistical methods to analyze random physical phenomena (of which there are numerous examples found in bio-medical research)

referring specifically to probability density functions, correlation functions, and power-spectral density functions.

The specific application of electrocardiogram interpretation was chosen by several groups. Some logical reasons for this choice are the following. Data waveforms are fairly easy to obtain. The diagnosis of heart disease from electrocardiograms is reasonably well developed at the present "state of the art" of electrocardiogram interpretation. Also, from an engineering standpoint, it is helpful that the electrocardiogram gives a cyclic or repetitive signal which, when properly recorded, has a relatively high signal to noise ratio. For these reasons, then, several groups have decided to try various techniques to interpret electrocardiograms.

Caceres and associates (14) at the U.S. Public Health Service record clinical 12-lead electrocardiograms in analog form at several remote stations, then bring the tapes to a central processing unit, sample the analog signal 625 times per second, and use the signal and its derivative to pattern recognize various amplitude and time duration parameters. These parameters, in turn, are used to perform an interpretation. Pipberger and colleagues (15) at Georgetown University record corrected three-orthogonal-lead system electrocardiograms in analog form on magnetic tape and then sample the signal at 1000 samples per second. They use this lead system to compute heart electrical vectors and diagnose heart ailments using these time-varying vectors and their associated

"loops" in a vectorcardiography technique. Cady and associates (16) at New York University also use a three-orthogonal-lead system, but extract certain parameters from the three leads sampled at 1000 samples per second. They have developed coefficients from Fourier analysis which are used in discriminant function equations to diagnose heart disease.

Stark and colleagues (17) at M.I.T. have studied the use of a multiple adaptive-matched-filter technique using a three-orthogonal-lead system sampled with a base sampling rate of 600 samples per second. They separate the heart cycle into three distinct segments: the PR interval, the QRS interval, and the ST interval. After a time-normalization and an amplitude-normalization procedure, a series of waveform segments are presented to a large number of initially randomized memory filters. The memory filter which is most similar to each of the segments presented is modified by that segment and thus adapts. Once the basic patterns have been "learned" by presenting a large number of cases of that basic pattern to the filter, large scale ECG processing may utilize these fully formed filters.

Warner and associates (18) at Salt Lake City explored the use of conditional probabilities and Bayes' rule from probability theory in heart disease diagnosis. They developed a matrix of conditional probabilities (for a set of thirty-three congenital heart diseases and a set of fifty diagnostic symptoms) from a population of 1035 patients. They tested the

matrix on 36 cases. Their conclusion was that the computer diagnosis was right as often as was the first diagnosis of each of three experienced cardiologists, with their diagnoses being based on the same clinical information.

METHOD OF INVESTIGATION

Theoretical Considerations

Correlation as a time average or an ensemble average

A few definitions will be helpful in the following dis-
cussions and are thus in order at this point. An "ensemble"
is a set or collection of data records. A "random process"
is a set of records which can be described only in terms of
its statistical properties. A "stationary random process" is
a random process whose ensemble statistical properties do not
change with translations in time. An "ergodic stationary
random process" is a process whose ensemble statistical proper-
ties are the same as the statistical properties obtained from
time averages on an arbitrary record from the ensemble. If
only a single record is available instead of an ensemble of
records, the concept of stationarity as defined above does not
apply. As a consequence, a concept of "self-stationarity"
has evolved in which the single record is broken up into a
number of shorter subrecords and it is verified that the
statistical properties from one subrecord to another are
equivalent.

For stationary random processes, correlation may be per-
formed as either a time average or an ensemble average.
Lee (19) gives equations for these averages as follows.

For a time average, the equation is

$$(1) \qquad \phi_{xx}(\tau) = \lim_{T \to \infty} \frac{1}{T} \int_{-\frac{T}{2}}^{\frac{T}{2}} f_x(t) f_x(t+\tau) \, dt$$

(This equation points out that the autocorrelation function is merely a shape comparison of the waveform with a time shifted version of itself.)

For an ensemble average, the equation is

$$(2) \qquad \phi_{xx}(\tau) = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} x_1 x_2 P_{(x_1, x_2; \tau)} \, dx_1 \, dx_2$$

(In Equation 2, $P_{(x_1, x_2; \tau)} = P_{\left(x_1 = x_{(t_1)}, x_2 = x_{(t_2)}; t_2 - t_1 = \tau\right)}$ where x_1 and x_2 are values $x_{(t)}$ can take at times t_1 and t_2.)

Lee (19) also discusses the necessary conditions for equivalence of these two averages at some length. In general, the ergodic property is sufficient. However, for the purposes of this investigation, it is not deemed necessary nor desirable to engage in a rigorous discussion of these conditions. The reason for this is that the correlations involved in this study are a time average but cannot be tied to random process theory because these correlations involve crosscorrelating an

unknown signal corrupted by noise against a known, noiseless, signal. This will be discussed in more detail later.

Autocorrelation and crosscorrelation function equations

For a general aperiodic time function whose values are specified for all time, an autocorrelation function may be defined as a time average by

$$(3) \qquad \phi_{xx}(\tau) = \int_{-\infty}^{\infty} f_x(t) f_x(t+\tau) dt$$

A periodic time function whose values are defined at all values of the time argument over a complete period T may be defined as a time average by the equation

$$(4) \qquad \phi_{xx}(\tau) = \frac{1}{T} \int_{-\frac{T}{2}}^{\frac{T}{2}} f_x(t) f_x(t+\tau) dt$$

This is the equation which applies to the signals used in this investigation. For machine calculation, it is frequently desirable to convert these continuous signals into discrete values. If $f_x(t)$ is divided into N equally spaced intervals and the discrete values obtained at the ends of these intervals, an approximate autocorrelation function suitable for computation by computers is given by

$$(5) \qquad \phi_{xx}(\tau) \simeq \frac{1}{N+1} \sum_{k=1}^{N} x_k x_{k+\tau}$$

The corresponding crosscorrelation equation is

$$(6) \qquad \phi_{xy}(\tau) \simeq \frac{1}{N+1} \sum_{k=1}^{N} x_k y_{k+\tau}$$

when both $f_x(t)$ and $f_y(t)$ are divided into N intervals.

Some properties of these correlation functions follow. The autocorrelation function is an even function of the time shift τ whose magnitude is maximum at zero time shift. This maximum value at $\tau=0$ is the mean-square or variance of the time function. Also, any time function has but one auto-correlation function, but any autocorrelation function may have many corresponding time functions. On the other hand, the crosscorrelation function is not necessarily an even function of time shift nor does it necessarily have its maximum magnitude at zero time shift.

Relation to matched-filter theory

The autocorrelation function has been previously approached in this dissertation from the standpoint that it is a measure of the similarity of the shape of a waveform with a time shifted version of itself. A second way of looking at this is that the autocorrelation of a given signal is the same as the time reverse of the convolution of that signal. This is shown by comparing the autocorrelation integral

$$\left(\frac{1}{T}\int_{\frac{-T}{2}}^{\frac{T}{2}} f_{(t)} f_{(t+\tau)} dt\right)$$ with the convolution integral

$$\left(\frac{1}{T}\int_{\frac{-T}{2}}^{\frac{T}{2}} f_{(t)} f_{(\tau-t)} dt\right).$$ These equations show that while both

involve operations of displacement, multiplication, and
integration; convolution also involves an operation of
"folding back" the displaced waveform (or taking its time
negative) as is evidenced by the $f_{(\tau-t)}$ term.

Still another approach to the autocorrelation function
is through the power spectrum and the matched filter. It may
be observed that the autocorrelation function is the same for
any sample of noise which has the same frequency content as
another noise sample but may take on a variety of different
waveform configurations. It then becomes evident that the
autocorrelation function of a waveform does not depend upon
the waveform itself, but instead it is dependent upon the
waveform's frequency content. In order to consider this in
terms of Fourier analysis, some definitions are again in
order.

The frequency content of a waveform can be defined by an
amplitude spectrum where this amplitude spectrum is a plot of
the magnitude of the Fourier components versus frequency. We
may also obtain a phase spectrum for a waveform which is a

plot of the phase of the Fourier components versus frequency.
A power spectrum of the waveform can be obtained by plotting
the square of the absolute magnitude of the amplitude of the
Fourier components versus frequency. This power spectrum
accentuates variations from "flatness" of the amplitude spec-
trum but like the amplitude spectrum, contains no phase
information.

The autocorrelation function contains the same informa-
tion as the power spectrum of the waveform, but this
information is given in the form of a function of time (or
time shift) instead of a function of frequency. This is
pointed out by the relationship that the power spectral
density function is the Fourier transform of the autocorrela-
tion function as shown by Brown (21). Thus, the auto-
correlation function may be formed by squaring the amplitudes
of the Fourier components, setting them into phase with each
other at the origin of a new time scale, and then adding them
together as pointed out by Anstey (22). Another way of saying
this is that if an input signal (of known amplitude and fre-
quency spectra) is input into a system (whose amplitude and
phase versus frequency spectra are known), there appears an
output signal whose amplitude spectrum is the product of the
input signal's amplitude spectrum times the system's amplitude-
frequency response, and whose phase spectrum is the sum of the
input signal's phase spectrum plus the system's phase-
frequency response. Therefore, for any signal, a system

called a "matched filter" may be defined whose amplitude-frequency response has the same form as the signal's amplitude spectrum and whose phase-frequency response is exactly opposite to (or the negative of) the signal's phase spectrum. The output from this system then gives an amplitude spectrum proportional to the power spectrum of the input, but the phase spectrum is zero. The matched filter corresponding to a given signal then has an impulse response which is proportional to the signal reversed in time but with a time lag. Then for any physical waveform $x_{(t)}$, a filter which is matched to $x_{(t)}$ is, by definition, one with an impulse response of the form $g(\tau) = kx_{(\Delta-\tau)}$ where k and Δ are arbitrary gain and time lag constants. It may then be said that the autocorrelation function of a waveform is equivalent to the output obtained by passing the waveform through its matched filter.

From an intuitive standpoint, this relationship between the autocorrelation function of a signal and the frequency spectrum of that signal may be seen by noting that if the autocorrelation function falls off rapidly as a function of time shift τ, it would be expected that the frequency spectrum of the signal is predominately high frequency components. Alternatively, if the autocorrelation function falls off slowly, it would be expected that the signal would contain a predominance of low frequency components.

Matched filter theory and its relationship to correlation are discussed in more detail by Turin (23), Mericle (8),

and Townsend (9).

Correlation and mean-square-error criterion

In using electrocardiograms to diagnose heart disease, no matter what method is used or what ECG parameters are extracted, the diagnosis itself boils down to comparing these parameters via the method used against standard values for the parameters derived through theory and through their use in a large number of cases. By means of some criterion, the difference (or error) between the desired or standard parameters and the actual parameters must be considered to determine how well the heart is functioning. The choice of this error criterion determines which of the parameters of the signal are considered to be important.

In electrocardiogram interpretation, it would seem that a small error may easily be caused by factors such as age, body build, orientation of the heart, fear, etc., which have no direct bearing on the actual health of the heart. Therefore, an error criterion should be chosen which gives less relative weighting to small errors and more weighting to large errors which are more likely to be caused by heart disease. Although there are several error criteria which meet this qualification, the mean-square-error criterion fills it well, and has additional advantages of generally being relatively easy to manipulate mathematically and is fairly well understood and commonly used in general practice.

In the mean-square-error criterion, the average value of the square of the difference between the actual signal and the desired signal is the important characteristic considered to determine the state or quality of the system. If we denote $f_x(t)$ as the actual output of the system and $f_s(t)$ as the desired output (or standard), then the mean-square error, regardless of the nature of the system, may be defined by the

expression $\overline{e^2} = \lim_{T \to \infty} \frac{1}{2T} \int_{-T}^{T} [f_x(t) - f_s(t)]^2 dt$. For most problems

then we wish to minimize this mean-square error in order to have the actual signal most closely match the desired signal.

A decision must also be made as to a set of parameters to be used with this error criterion to perform a system analysis (or diagnosis). As described previously, Barker (7) investigated Fourier components in his M.S. thesis. Balm (5) used conditional probabilities in his M.S. thesis. Gustafson (6) used pulse amplitudes and time durations. For this dissertation, it was decided to use the correlation function in an extension of work done by Mericle (8) and Townsend (9).

It is shown by Truxal (20) that if the input is a stationary random process, the signals are adequately described by the correlation function when the selected error criterion is the minimization of the mean-square error. Bendat (13) points out that the correlation of stationary random records brings about an improvement in signal-to-noise ratio of the output

signal over the input signal. However, for the application in the investigation performed for this dissertation, it must be noted that the correlations performed are not tied directly to random process theory.

In this investigation, the criterion used is a mean-square-error criterion. This error is the difference between the actual system output (the crosscorrelation function of the input signal against a standard waveform) and the desired system output (the autocorrelation function of the standard waveform). It is noted that this error measures the difference between the actual unknown input signal and the known standard waveform as reflected through the matched filter (or correlation process). The standard waveform is a known signal, uncorrupted by noise, which represents a waveform disease category or class. A set of these standard waveforms are stored in the computer in a normalized discrete form. An unknown waveform which is to be classified via the correlation techniques is then put into a normalized discrete form. The difference in shape between this unknown waveform and the standard waveform against which it is to be crosscorrelated may be considered as error. It is the mean-square of the correlation function difference representation of this error then which is to be minimized for classification by cross-correlating the unknown waveform against several standard waveforms, or the actual value of this mean-square error representation may be used by setting a threshold value of

acceptable error for classification by a single crosscorrelation. A perfect pattern match would then indicate zero mean-square error as indicated by crosscorrelation which reduces to autocorrelation.

From another viewpoint, the unknown waveform may be considered to be the standard waveform corrupted by "noise" where this "noise" is the error or actual shape difference. The crosscorrelation then is a correlation of a known standard waveform, $s(t)$, with the unknown waveform $x(t)$, which is now the standard waveform plus noise, $s(t) + n(t)$. The time-average crosscorrelation then is

$$\phi_{sy}(\tau) = \overline{s_{(t)}\left[s_{(t+\tau)} + n_{(t+\tau)}\right]} = \overline{s_{(t)}s_{(t+\tau)} + s_{(t)}n_{(t+\tau)}}$$

$$= \overline{s_{(t)}s_{(t+\tau)}} + \overline{s_{(t)}n_{(t+\tau)}}$$

or

$$\phi_{sy}(\tau) = \phi_{ss}(\tau) + \phi_{sn}(\tau)$$

It is the second term of the last equality (the crosscorrelation of the standard waveform with the noise waveform) which is to be minimized for classification and approaches zero with a perfect pattern match (or zero-noise waveform).

It is from this viewpoint that the correlations in this study parallel the matched-filter technique. Turin (23)

discusses the signal detection case where the input to a linear filter is a signal plus noise or noise alone. The linear filter is matched to the signal to be detected. The output of the matched filter is a component due to noise alone plus a component due to the signal (if present) since the filter is linear. The component due to the signal is a relatively narrow, but high amplitude, pulse which can be detected by using a threshold level. Turin discusses mean-square criteria by showing that matched filters maximize the output signal-to-noise power ratio.

The correlations used in this investigation are a slight extension of the signal-detection problem described in the preceeding paragraph. By matching a filter to a desired signal, a matched filter could be used to select that signal from a set of input signals corrupted by noise by properly setting the detection threshold level. By recalling that, when a signal is input into its matched filter, the output of the matched filter is the autocorrelation function of the input signal; it becomes apparent that the computer-run correlations are the digital equivalent of analog matched filters.

From a second viewpoint, the crosscorrelations used in this investigation are a straight-forward minimization of mean-square-error between the crosscorrelation of the input waveform with the standard waveform and the autocorrelation of the desired standard waveform. By correlating the unknown

waveform against several filters which are matched to several possible desired input waveforms, classification can be made on a basis of which desired input waveform the unknown waveform correlates highest with, thus minimizing the mean-square error.

When a minimization of mean-square error between an actual output and the desired output is discussed, certain considerations must be made to insure that this minimization is indeed a practical one. This error is often described as noise corrupting the output when the desired output is a faithful reproduction of the input (with a gain factor and a time shift possible). Thus a minimization of the mean-square of this noise (or error) could certainly be accomplished by a filter which is merely an open circuit (or has a zero gain factor). This filter is not practical, however, since it also reduces the signal to zero. Thus, some constraints on this minization of mean-square error may be necessary from a practical standpoint.

If a signal corrupted by noise $\left(s_{(t)} + n_{(t)}\right)$ is input into any linear filter, the output signal $\left(s'_{(t)} + n'_{(t)}\right)$ will be composed of a component due to $s_{(t)}$ only and a component due to $n_{(t)}$ only. If this output signal is considered at some time t_0, Turin (23) points out that the linear filter matched to $s_{(t)}$ will maximize the output signal-to-noise power ratio $[s'_{(t_0)}]^2 \Big/ [n'_{(t_0)}]^2$. If the class of all filters which give

the same signal power at time $t_0 \left([s'_{(t_0)}]^2 \right)$ is considered, then the matched filter by maximizing the above power ratio effectively minimizes the mean-square error (or noise) described by $\overline{[n'_{(t_0)}]^2}$. In summary, correlation (or a matched filter) operating on a known signal corrupted by noise may be considered to be a minimization of mean-square error criterion under the constraint that the signal power is held constant.

Experimental Procedures

General considerations

Having settled on the correlation technique for this application to electrocardiograms, several specific problems must be considered. The problems of determination of sampling rate, time normalization, amplitude normalization, and exactly what should be correlated involve a fair amount of thought and justification. These will be discussed in more detail later. However, two other considerations are of a more general nature and depend upon what the research is intended to show or accomplish.

First is the consideration of what lead or lead-system should be used in the correlation technique. The first thought that comes to mind to a researcher who was educated in the field of science is that, since the electric potential field in a three dimensional volume conductor is of interest, one of the proposed three-orthogonal-lead systems would

theoretically contain maximum information. However, this thought is soon tempered by the realization that discrete time-correlation is a sum of products of ordinates of the waveforms to be correlated against each other, and thus a minimization of the number of waveforms (or leads) is highly desirable. It is also found after investigating these three-orthogonal-lead systems that part of the reason that they are not accepted in routine clinical practice is that the lead placement on individual patients is relatively difficult to standardize and also that these lead systems deviate slightly from orthogonality in the present "state of the art". It is not the intent of this dissertation to find an optimal set of leads upon which correlation methods may be used, but instead to investigate whether or not the correlation technique is a feasible and practical method of screening abnormal electro-cardiograms from normal electrocardiograms and also to differentiate between abnormalities. It was decided to attempt to perform this investigation using a single lead if possible.

After discussions with Dr. L. F. Staples, M.D., a specialist in adult electrocardiography at Iowa Methodist Hospital in Des Moines, standard lead I was chosen for two reasons. It is his opinion, and also the opinion of many others, that standard lead I is the best overall lead for diagnosis of abnormal electrocardiograms. It is noted, how-ever, that each individual abnormality is most readily

detected in the lead placed nearest to the diseased portion of the heart and thus standard lead I will not necessarily be the best lead to display some heart abnormalities. However, this particular lead is a practical one since it is the potential at the right wrist with respect to the potential at the left wrist. It may thus be readily recorded without the necessity of the patient partially disrobing and it may be recorded in either a sitting or supine position. This point is very important if the motivation for this technique is a quick, easy, and inexpensive method for mass interpretation of electrocardiograms from a large population.

A second general consideration is what abnormalities to correlate in order to best demonstrate the feasibility of this technique (in essence, to see if correlation methods will divide abnormals from normals, and classify individual abnormalities). It is soon apparent that it is not practical to try to demonstrate all maladies of the heart which appear in electrocardiograms. It was therefore thought to be desirable and reasonable to demonstrate feasibility using several regions of the heart. Two fairly common maladies found in the left ventricle of the heart are left ventricular hypertrophy (LVH) and left bundle branch block (LBB). Right ventricular hypertrophy (RVH) and right bundle branch block (RBB) are the corresponding maladies of the right ventricle and were thought to be desirable for demonstration even though RVH is diagnosed relatively infrequently.

Sampling rate

The rate at which each waveform is to be sampled must also be given consideration. Since discrete time-correlation is carried out by a sum of products of the ordinates (values of the individual samples) through several values of time shift, it is desirable to use the lowest possible sampling rate to minimize the number of samples and thus minimize the computation time required by all the multiplications. However, a well-known sampling theorem given by Truxal (20) and others states that the sampling frequency must be at least twice the highest frequency component of interest contained in the input signal. Thus, it must be decided what is the highest frequency component necessary for diagnosis.

Several investigators have performed Fourier analyses of electrocardiograms to determine the highest significant frequencies contained therein. Thompson (24), in studying normal electrocardiograms, concluded that an upper limit of 51.6 cycles per second is all that is needed to completely pass normal electrocardiograms. Langner and Geselowitz (25) concluded from their investigations that frequencies at least as high as 500 cycles per second and possibly as high as 1000 cycles per second are needed to faithfully reproduce electrocardiograms which contain "notches" caused by some maladies. Scher (26) concluded from his studies that frequencies above 100 cycles per second are insignificant although noting that the higher frequency components described by Langner were

present. Barker (7) reached a conclusion from the cases he studied that frequencies over 40 cycles per second are insignificant. It may also be noted that most electrocardio-gram recording equipment used routinely in hospitals today pass only frequencies below about 40 cycles per second.

Thus, it becomes evident from reading the literature that one may find as many opinions of "highest frequencies needed" as the number of articles one reads. These variations are caused by differences in the case-populations studied and by differences of definition of what is "significant" for diagnosis. Again, the investigator must be tempered by recall-ing that correlation techniques, data handling problems, and computer time require that the number of data samples be minimized. Also, from a practical standpoint, it is desirable to be able to filter out 60 cycle noise. It was thus decided to allow frequencies up to 50 cycles per second so that a low pass filter with a cut-off frequency of 50 c.p.s. could be incorporated in hardware. This, by the sampling theorem, requires a sampling rate of 100 samples per second.

What is to be correlated

Another consideration is an answer to the question, "Should the entire ECG cycle be correlated or should it be separated into parts and these parts correlated independently?" Townsend (9) used the entire ECG cycle in his correlation study but noted that the time of no heart activity (the TP

baseline interval) contributed nothing to the correlation coefficient since it is zero potential, and that the P wave contributes only in a small way because its area is usually relatively small. Mericle (8) thought that each pulse (the P wave, the QRS complex, and the T wave) of the ECG cycle is caused by a different physiological heart function and should be correlated against its counterparts independently of each other. Stark and colleagues (17) in a similar line of reasoning considered the PR (PQ) interval, the QRS interval, and the ST interval to be independent; so they were treated as such with the advantage that the PR and ST intervals contain lower frequencies only and could thus be sampled at a lower rate than could the QRS interval.

Since disease is thought to occur independently in the auricles or in the ventricles, for the purposes of this dissertation the PR (PQ) interval (resulting from the electrical action of the auricles) may be considered independently of the QT interval (resulting from the electrical action of the ventricles). However, since the QRS complex and the ST interval result from the depolarization and repolarization of the same ventricular musculature, it is thought that the real-time relationship of these two portions of the ECG may be of some importance and that these two time intervals should not be considered to be independent but, instead, should be considered as a single entity (namely, the QT interval). The remainder of the ECG cycle, the so-called TP baseline of zero

heart activity, gives no contribution to correlation and is of importance only to calculate heart rate (or cycle length) and to establish the dc level of the waveform.

Since the ventricles constitute the major portion of the muscular mass of the heart, they are correspondingly responsible for the major part of the cardiac waveform. The auricular activity does play an important part in determining the basic rhythm of the heart beat, but since auricular abnormality generally occurs less frequently than disease of the ventricles, it was decided to investigate only four ventricular abnormalities for this dissertation. Thus the part of the ECG cycle to be correlated is the QT interval. It is noted, however, that from a practical standpoint, much of the time saved by correlating only the QT interval instead of an entire heart cycle would be lost because it necessitates some sort of a pattern recognition of the electrocardiogram to separate the PR interval, the QT interval, and the TP baseline interval from each other.

Time normalization

Since correlation may be thought of as a shape comparison and since the waveforms under consideration vary both in magnitude and in time duration from causes other than heart disease, it is soon apparent that some sort of time and amplitude normalization must be performed in order to make the correlation meaningful. For example, if a test waveform had

the exact same shape as the standard waveform against which
it is to be crosscorrelated but the test waveform had only
half the amplitude and half the time duration as the standard
waveform, crosscorrelation would have little significance
until the test waveform is time normalized to twice its
original time duration and is amplitude normalized to twice
its original amplitude. After these normalizations have been
performed, the crosscorrelation reduces to autocorrelation and
gives a perfect match as was desired for identical shapes.

Time normalization will be considered first. The simpl-
est time normalization is merely linearly expanding or
contracting the time duration of the test waveform until it
equals the time duration of the standard waveform. This
corresponds to merely varying the sampling rate. However,
when the time durations of the various portions of the electro-
cardiogram (the PR interval, the QRS interval, the ST interval,
and the TP baseline interval) are studied, it is found that
they do not each vary linearly as a function of heart cycle
length (RR interval).

To study the time duration variation of these intervals
more closely, the following investigation was undertaken. A
statistical population of 3,641 adult electrocardiograms was
obtained from the Heart Station of Iowa Methodist Hospital in
Des Moines. The following intervals were measured in these
cases: RR, PR, QRS, and QT (see Figure 2). From these
measurements, the following parameters can be readily computed:

$$\text{Heart rate (beats/min)} = \frac{60 \text{ sec/min}}{RR \text{ sec/beat}}$$

$$\text{ST interval} = QT - QRS$$

$$\text{PT interval} = PR + QT$$

$$\text{TP baseline interval} = RR - PT$$

The population of cases was arranged in order by the value of the heart rate for the individual cases. The range of heart rates was from 40 beats per minute to 170 beats per minute with the mode of the population occurring at 75 beats per minute. The mean value of each of the time interval parameters was found at each heart rate level represented and the results are plotted in Figure 3. The reason that heart rate (in beats per minute) was chosen as the abscissa of the plot was that it was desired to have each of the time intervals under study vary over a wide range of heart rate. As is seen from Figure 3, QRS does not vary appreciably with heart rate and can be considered a function of heart disease only. PR interval decreases slightly with increasing heart rate but its variation is quite linear. QT interval decreases a little more than PR interval as heart rate increases but a linear-variation approximation is still reasonable. RR interval decreases rapidly and non-linearly with increasing heart rate since it is, in fact, proprotional to the inverse of heart rate. PT interval and ST interval vary reasonably linearly with heart rate since they depend upon QT, PR, and QRS in combination. TP baseline interval (not actually plotted on the
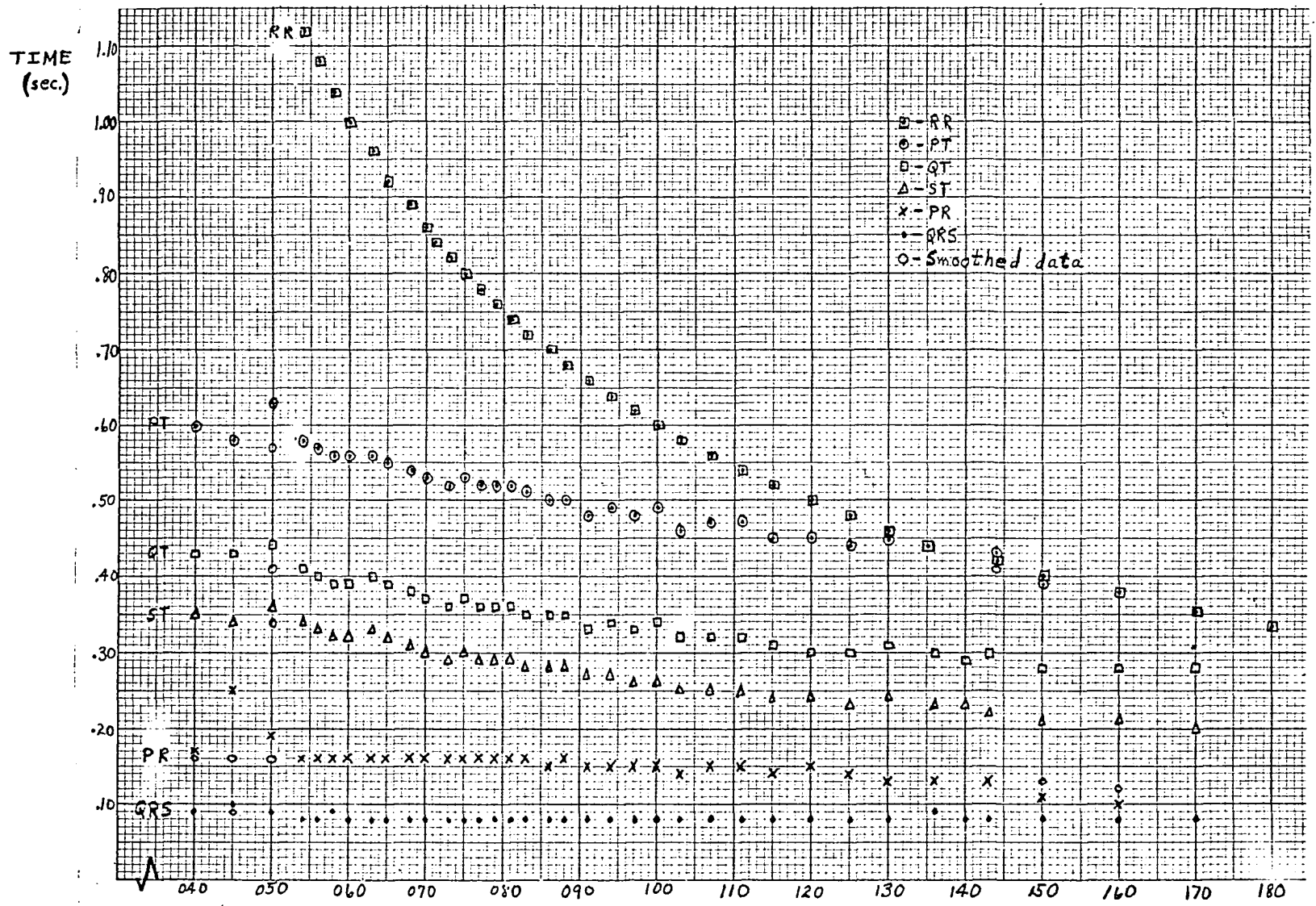
Figure 3. ECG interval relationships

graph but readily seen as the distance from the PT curve up to the RR curve) varies non-linearly due to its dependence upon RR.

Since the population used to derive Figure 3 did include some cases which had abnormally long or short PR intervals and abnormally long QT intervals (where abnormality in these cases is decided upon by unproven and debatable empirical criteria), these abnormal cases were removed and the remaining cases were run again. The results were that all the curves were essentially the same but somewhat smoothed out due to the removal of extreme cases.

It is also apparent from Figure 3 that if PR interval, QT interval, and TP baseline interval were plotted as functions of RR interval; they would not vary linearly.

Townsend (9) in his correlation studies first linearly time-normalized the entire RR interval but found that when the heart rate of the test waveform deviated very far from the heart rate of his standard waveform, troubles developed. He later corrected for these non-linearities in test waveform heart rates varying appreciably from his standard waveform heart rate of 75 beats per minute. It is apparent from Figure 3 (recalling that TP baseline interval is represented by the distance from the plot of PT up to the plot of RR) that most of the non-linearity in the variation of RR interval with heart rate lies in the TP baseline interval which is zero electrical potential and contributes nothing to the correlation

coefficient anyway. This is further justification for discarding this TP baseline interval when using correlation methods.

Since it was decided to be concerned only with the QT interval for the purposes of this dissertation, examination of Figure 3 justifies approximating as linear the variation of the mean QT interval with heart rate. It is noted that, for each given heart rate, there is some variation of QT interval from this mean value plotted in Figure 3. However, at the present "state of the art" of electrocardiogram interpretation, variation of QT interval from a mean value for the given heart rate is usually noted but not given much diagnostic significance. This variation may be an effect of age, sex, heart rate, medication, temperature, and other factors aside from heart disease. Thus, for this dissertation, all QT intervals regardless of heart rate are time-normalized by linearly expanding or contracting their time base to 0.35 seconds which is approximately the value of the QT interval for the modal heart rate value of 75 beats per minute. This is accomplished by effectively varying the sampling rate slightly from the base sampling rate of 100 samples per second to insure that each QT interval is represented by 36 evenly spaced samples (100 samples per second times 0.35 seconds gives 35 samples and then add a final end-point sample giving 36).

If it is desired to give consideration to the variation of QT interval for each given heart rate, the sampling rate

variation could be determined from the plot of mean QT

interval versus heart rate (Figure 3) for the given heart

rate and then sample the QT interval at this sampling rate

regardless of its real time duration. By doing this, the

nicety of having all QT intervals containing the same number

of samples would be lost. This time-normalization procedure

should be considered if the intervals of interest were the PR

interval or the entire PT interval, because variation of the

PR interval for each heart rate is considered to be of some

importance in diagnosing auricular disease.

Amplitude normalization

After time normalization has been accomplished, some sort

of amplitude normalization must be considered. Expansion or

contraction of the time base without a compensating adjustment

of amplitude values would cause the waveform to lose its

original shape. The first thought that comes to mind is that,

in order to keep the exact shape that the waveform had prior

to time normalization, the amplitude normalization should be

performed by multiplying the amplitude of the waveform by the

same factor as was used in multiplying the time base in the

time normalization. However, again upon investigating proper-

ties of the electrocardiogram, this method of amplitude

normalization gives rise to difficulties. It is found that

extremely rapid heart rates (with corresponding short time

bases) often have pulses whose amplitudes are on the same

order as the pulse amplitudes in electrocardiograms with
average heart rates. Thus when the factor that is used to
expand the time base for time normalization is used in
amplitude normalization, the result is a normalized electro-
cardiogram containing huge pulses but, nevertheless, pulses
of the same shape as they were before normalization. The same
is found to be true for extremely slow heart rates with
corresponding long time bases but with average pulse ampli-
tudes. This results in very small pulses of the same shape
as they were originally. With these examples at hand, it
becomes evident that a simple straight-forward shape-retention
criterion is probably not what is wanted for amplitude
normalization to solve the diagnosis problem. Possibly a
peak-amplitude-equalization criterion or an area-equalization
criterion would be better suited for this problem.

The peak-amplitude-equalization criterion was considered
next because of its simplicity and ease of performance.
However, it was noted that in some cases where the test wave-
form is abnormal and the standard waveform is normal (or vice
versa), it is possible that it would be attempted to match the
amplitude of a negative going Q wave pulse to that of a posi-
tive going R wave pulse. The next thought then is to match
the magnitude of the peak of largest pulse in the QRS complex
(or of the largest pulse in the entire ECG waveform) or the
magnitude squared of these pulses. This is a possibility but
in considering the area-type equalization explained in the

next paragraphs, it was decided that that criterion had certain advantages which merited its use.

The next criterion considered was an area-equalization criterion. Again, if the total value of the areas are made to be equal under this criterion, the diffi-ulty of possibly trying to equate a negative area to a positive area is encountered (negative area being area of a negative going pulse). Thus a magnitude of total area or total-area-squared criterion is considered. It is granted that a magnitude of total area is fairly easily computed by trapezoidal approximations or other methods, but the amplitude-normalization procedure used by Townsend (9) is considered to have advantages over equating magnitudes of the area of the waveforms.

Townsend (9) used, for his amplitude normalization, equalization of the autocorrelation functions of the two waveforms for a zero time shift. This autocorrelation function with zero argument may be viewed from several standpoints. As pointed out previously, it is the mean-square value of the time function. Since it is simply the average value of the time function squared, it may also be viewed as an average power with the interpretation that the function is considered to be the voltage across (or the current through) a one ohm resistance. Forcing equivalence of the autocorrelation function with zero time shift also may be viewed as being proportional to the equivalence of the area squared in that, by squaring the time function and summing or integrating this

squared function over the time base, it yields a result which
may be viewed as area squared divided by time base.

All of these amplitude-normalization criteria mentioned
give a crosscorrelation between test waveform and standard
waveform which reduces to an autocorrelation of the standard
waveform in the case of a perfect shape match. However, the
criterion in which the autocorrelation functions with zero
argument are made to be equal for the two waveforms has the
added advantage that the maximum value of the crosscorrelation
function for the two waveforms can never exceed the auto-
correlation function value of the standard waveform at zero
time shift, and only approaches this value as the two wave-
forms become closer to a perfect match.

The practical performance of this criterion of setting
the autocorrelation functions of the two waveforms equal is
done as follows. Let the standard waveform be noted by $f_s(t)$
and the test waveform (or unknown waveform) be denoted by
$f_x(t)$. Each waveform is represented by N samples. The auto-
correlation function with zero argument for the standard
waveform may be computed by the equation

$$(4) \quad \phi_{ss}(0) = \sum_{i=1}^{N} \left[f_{s_i}(t) \right] \left[f_{s_i}(t+0) \right] = \sum_{i=1}^{N} \left[f_{s_i}(t) \right]^2 = K$$

By the amplitude normalization procedure, the amplitude of
each of the N samples of the test waveform is multiplied by
some constant factor "a" which makes the autocorrelation

function of the test waveform at zero time shift equal to the value "K" as defined in Equation 4. Thus denoting the zero time-shift normalized autocorrelation function of the test waveform by $\phi_{xx}^n(0)$, we have

(5)
$$\phi_{xx}^n(0) = \sum_{i=1}^{N}\left[(a)f_{x_i}(t)\right]\left[(a)f_{x_i}(t+0)\right]$$

$$= \sum_{i=1}^{N}\left[(a)f_{x_i}(t)\right]^2 = K$$

Equation 5 may be factored as follows:

(6)
$$\phi_{xx}^n(0) = \sum_{i=1}^{N}\left[(a)^2 f_{x_i}^2(t)\right] = (a)^2 \sum_{i=1}^{N}\left[f_{x_i}(t)\right]^2 = K$$

This is, of course, merely the factor "a^2" times the auto-correlation function with zero argument of the unnormalized test waveform or $(a^2)\phi_{xx}(0)$. Equating Equation 6 to Equation 4 yields

(7)
$$K = \phi_{xx}^n(0) = (a^2)\phi_{xx}(0) = \phi_{ss}(0)$$

The amplitude normalization factor "a" may then be found from the equality of the last two factors of Equation 7 as being

the square root of ratio of the autocorrelation of the standard waveform to the autocorrelation of the test waveform (both of these autocorrelation functions being computed with zero time shift). Thus the solution easily computed on a digital computer is

$$(8) \qquad a = \sqrt{\frac{\sum\limits_{i=1}^{N} \left[f_{s_i}(t) \right]^2}{\sum\limits_{i=1}^{N} \left[f_{x_i}(t) \right]^2}}$$

The factor "a" is then multiplied times the value of each sample in the test waveform to accomplish the amplitude normalization.

EXPERIMENTAL RESULTS

Actual Experimental Method Used

In the investigation undertaken for this dissertation
which was to study the feasibility of the correlation tech-
nique in its application to the electrocardiogram interpreta-
tion problem, a total of 87 actual case waveforms were used.
The object of the study was to see if each of several disease
waveform types could be screened from "normal" waveforms and
also if they could be correctly classified into their own
respective disease categories. The term "normal" is in itself
a somewhat vague term since it encompasses a fairly wide range
of waveform shapes. Thus "normal" as used in this disserta-
tion may be defined as "not having abnormal or disease
characteristics". To accomplish this object, four representa-
tive ventricular heart diseases were chosen for study in
addition to normal waveforms. These were right and left
ventricular hypertrophy, and right and left bundle branch
block. Also, representative waveforms of five other maladies
were included with these test waveforms to see if they would
be diagnosed as normal or abnormal although no standard wave-
form was developed for these cases and thus no attempt was
made to classify them into their correct disease category.
The number of cases in each group are given in Table 1. The
cases for each of the first four groups listed in Table 1 were
picked at random from a larger population of each group except

Table 1. Data cases used

Number of cases	Range of heart rate (beats/min)	Group abbreviation	Group name
35	51-110	NOR	Normal
14	44-111	LBB	Left bundle branch block
13	57-110	LVH	Left ventricular hypertrophy
10	38-100	RBB	Right bundle branch block
8	64-175	RVH	Right ventricular hypertrophy
3	81-92	PMI	Posterior myo-cardial infraction
1	115	ICD	Intraventricular conduction defect
1	68	ABT	Abnormal T waves
1	83	AVB	Atrioventricular block
1	43	NSD	Non-specific myocardial disease

that a couple cases in each group may have been especially selected to insure a fairly wide range of heart rates. For the RVH group, the total population available at the time of this study consisted of about 12 cases and several of these were discarded because of poor quality tracings or because they had extremely atypical waveshapes. The last seven cases

listed in Table 1 were picked at random.

An attempt was then made to find typical waveshapes for each group to serve as standard waveforms against which the test waveforms could be crosscorrelated. Two methods of finding these standard waveforms were tried. In one method, mean values of various parameters in the QT interval were found (considering large populations for each group). A pseudo-waveform was drawn and sampled for each group based upon these parameter mean-values. A second method was to examine all the selected cases in each group and to pick an actual case (or slightly modified version of an actual case) which typically represented that entire group or even slightly accentuated the characteristics common to that group but differentiating it from cases in the other groups. While the standard waveforms resulting from this second method were selected in a somewhat arbitrary manner, they were found to give better results than the standard waveforms found by the first method. It is also noted that the variation in the shapes of waveforms considered to be normal made it desirable to use two normal standard waveforms of slightly different shapes whereas each of the four disease groups required only one standard waveform.
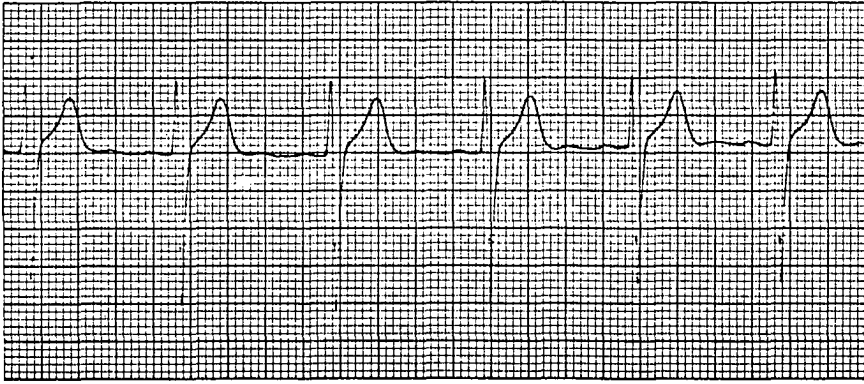
A typical electrocardiogram analog recording is shown in Figure 4. At the time this study was started, hardware was not available to record this time function in analog form on magnetic tape and to sample this analog signal at a variable sampling rate to perform time normalization. As a result,
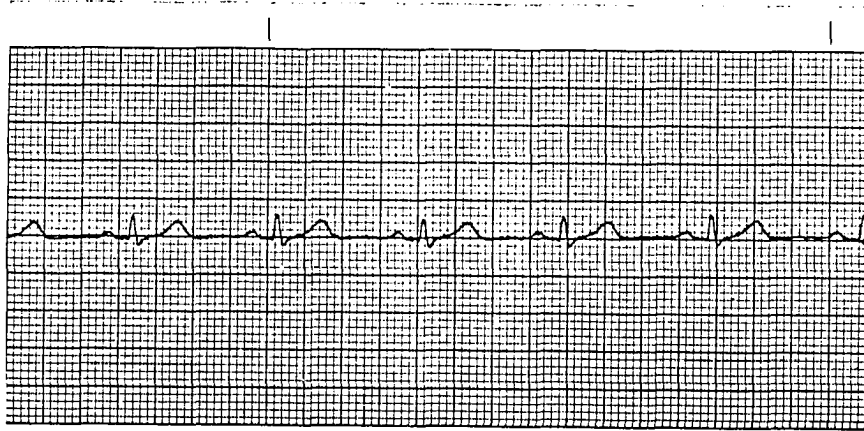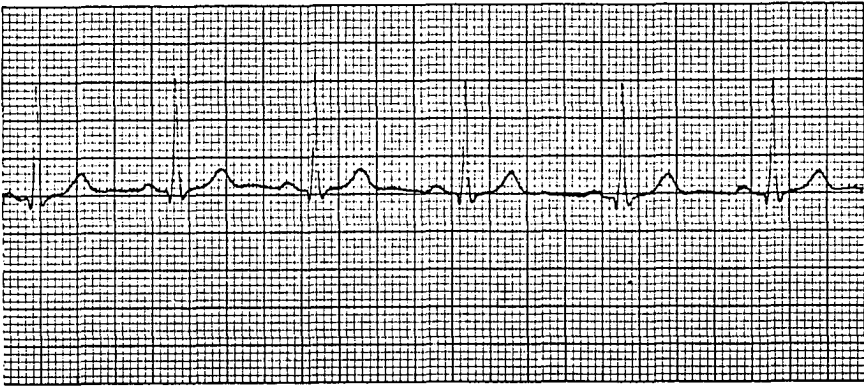
Figure 4. Typical analog ECG recordings

the analog signal for each of the 87 cases used was manually

redrawn in a "blown-up" form onto 8-1/2 × 11 grid paper and

then was hand sampled to insure each QT interval was repre-

sented by exactly 36 evenly spaced samples. These 36 three-

digit samples for each test case were then keypunched onto two

IBM cards per case along with case identification and heart

rate.

Computer Run Correlations

The punched cards containing the data samples for each

of the 87 cases was then run on a correlation program written

for the IBM 1440 computer system which was made available for

these studies by the Computer Center in the Heart Station at

Iowa Methodist Hospital in Des Moines. A general logic-flow

diagram for this correlation computer program is given in

Figure 5. Only the maximum positive correlation coefficient

was to be considered so, to reduce computer time for these

crosscorrelations, the time shift was limited to shifting five

samples left to ten samples right and all intermediate time

shift values. Each crosscorrelation requires about six

seconds on the 1440 computer.

As was stated previously, the amplitude-normalization

procedure used forced this maximum cross correlation coeffi-

cient to be less than or equal to (in the case of perfect

match) the standard waveform autocorrelation function value

with zero time shift. If this maximum crosscorrelation
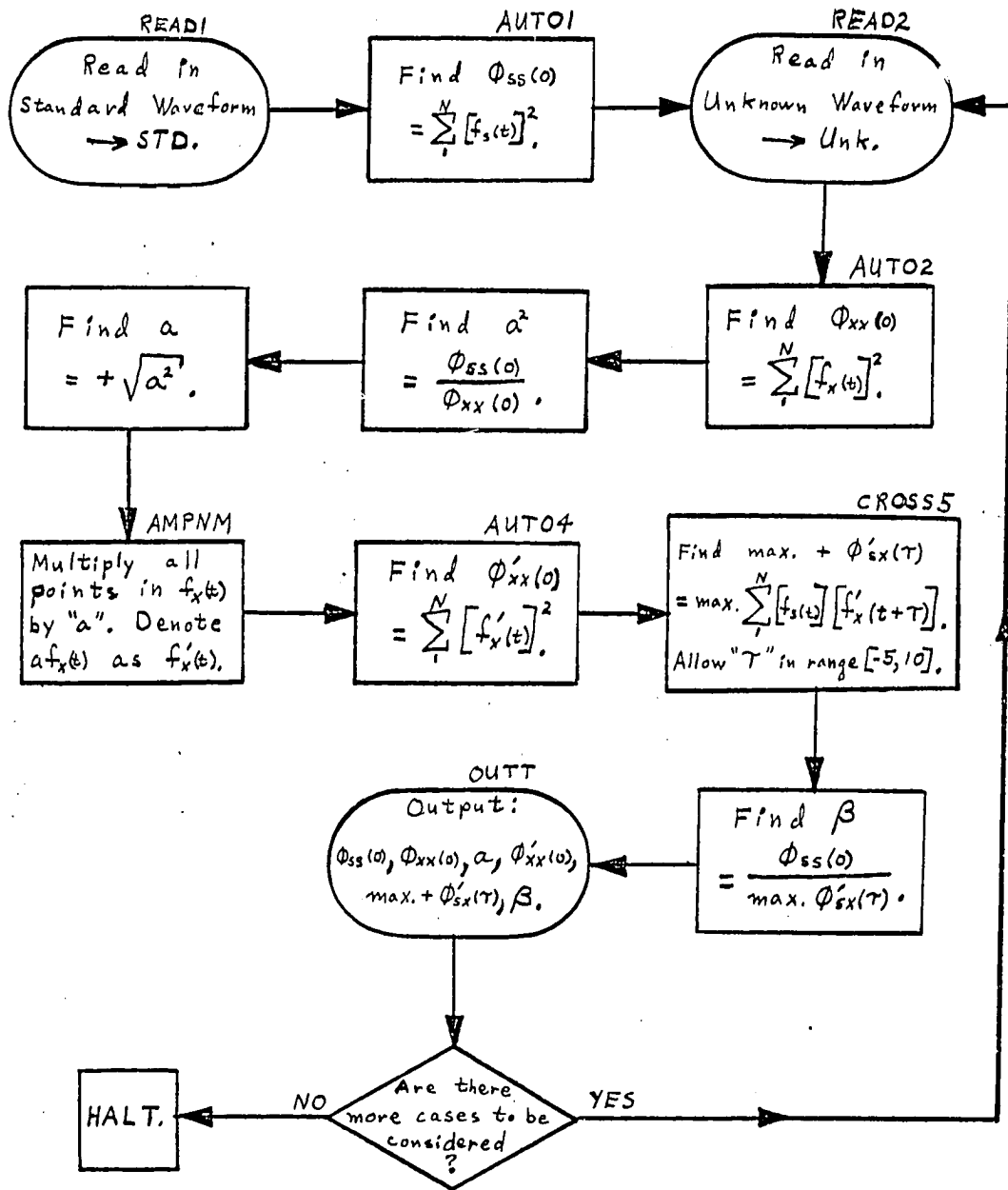
Figure 5. A logic diagram for the correlation computer program

coefficient is then divided by the autocorrelation function value for zero time shift, an index results which must lie between plus and minus one including the end points. The correlation computer program was programmed to carry out this division and the result then of the computer computations was a diagnostic index (arbitrarily designated β) which was constrained to fall in the closed interval [0,1]. Negative values are eliminated since negative crosscorrelation values are not retained, but are instead considered to be zero.

Table 2 shows the results of the computer crosscorrelations. The values in the body of the table are the diagnostic index, β, discussed in the preceeding paragraph. The heart rate and real-time length of the QT interval are also shown in Table 2 to demonstrate the effectiveness of the time-normalization procedure. This will be discussed in more detail later.

Discussion of Results

Several conclusions may be drawn from the computer cross-correlation results given in Table 2. Two general methods of interpreting these figures into a diagnosis scheme were tried. One method was sequentially crosscorrelating the test waveform against each standard waveform one at a time, comparing the diagnosis index β against a threshold value for that standard waveform, and making a decision as to whether the test waveform would be classified as belonging to that group or if

Table 2. Computer correlation results

Test waveform	QT int.	Heart rate	Standard waveforms					
			NOR17	NOR98	LBB11	LVH04	RBB04	RVH11
NOR01	39	51	95	84	37	44	56	53
NOR02	36	51	94	81	36	49	61	70
NOR03	44	52	87	93	61	86	78	45
NOR04	38	56	92	80	41	40	53	54
NOR05	41	58	89	91	68	80	77	52
NOR06	40	64	91	94	66	85	79	49
NOR07	43	65	94	93	57	78	75	53
NOR08	44	65	99	86	46	63	66	55
NOR09	41	65	99	85	40	63	69	62
NOR10	40	65	83	91	77	84	76	46
NOR11	39	67	86	94	68	89	80	47
NOR12	36	68	85	93	67	90	81	47
NOR13	38	68	89	90	58	87	84	56
NOR14	37	68	88	59	16	19	39	62
NOR15	36	70	92	87	54	77	82	66
NOR16	36	71	83	94	68	90	80	44
NOR17	38	71	100	86	44	62	66	59
NOR18	33	75	90	94	68	85	81	52
NOR19	32	75	87	90	68	86	77	47
NOR20	38	75	96	88	52	69	72	55
NOR21	36	75	91	93	58	82	76	46
NOR22	38	75	86	72	62	69	81	78
NOR23	32	75	89	94	68	83	78	51
NOR24	33	75	72	87	84	83	75	44
NOR25	31	79	95	76	34	55	66	75
NOR26	36	79	95	79	53	57	67	67
NOR27	36	81	89	76	44	64	78	83
NOR28	36	83	91	68	42	39	58	70
NOR29	40	88	95	89	51	74	75	65
NOR30	34	88	86	93	68	80	74	44
NOR31	37	90	94	92	61	80	76	53
NOR32	29	90	85	92	64	83	76	43
NOR33	34	100	89	75	39	65	69	81
NOR34	32	100	87	84	63	72	72	64
NOR35	32	110	90	84	62	81	87	70

Table 2. (continued)

Test waveform	QT int.	Heart rate	Standard waveforms					
			NOR17	NOR98	LBB11	LVH04	RBB04	RVH11
LBB01	44	45	38	54	96	63	45	30
LBB02	60	61	38	35	87	78	59	38
LBB03	39	63	55	64	97	80	67	38
LBB04	44	68	31	57	92	68	48	18
LBB05	44	68	52	63	96	76	63	41
LBB06	48	71	36	48	97	67	55	35
LBB07	43	71	48	58	95	64	57	38
LBB08	52	75	38	54	97	70	47	23
LBB09	46	77	63	76	97	78	67	38
LBB10	48	81	52	61	93	75	62	41
LBB11	52	83	44	54	100	71	52	33
LBB12	48	94	26	35	91	56	48	35
LBB13	46	100	34	46	97	66	45	25
LBB14	41	111	28	36	93	68	43	27
LVH01	50	57	78	86	65	94	81	44
LVH02	50	63	45	54	85	87	67	32
LVH03	52	64	64	75	79	96	81	41
LVH04	48	65	61	75	71	100	82	37
LVH05	34	65	59	76	80	94	77	39
LVH06	42	66	66	71	69	91	75	48
LVH07	33	71	64	80	67	97	80	32
LVH08	46	71	53	69	66	93	76	34
LVH09	50	75	67	65	79	90	81	53
LVH10	48	81	65	80	75	97	78	38
LVH11	52	88	74	84	68	97	80	44
LVH12	44	94	56	70	69	98	79	35
LVH13	38	110	73	81	81	91	79	44
RBB01	55	38	57	53	57	85	90	67
RBB02	46	50	70	43	30	47	75	72
RBB03	42	65	75	55	36	58	81	80
RBB04	42	73	67	68	56	83	100	73
RBB05	42	73	83	65	42	60	85	79
RBB06	36	75	76	49	37	45	79	87
RBB07	32	83	80	75	61	82	91	68
RBB08	44	94	59	65	95	80	72	45
RBB09	34	96	56	26	27	27	58	75
RBB10	38	100	78	80	72	86	91	64

Table 2. (continued)

Test waveform	QT int.	Heart rate	Standard waveforms					
			NOR17	NOR98	LBB11	LVH04	RBB04	RVH11
RVH01	34	64	38	15	14	24	54	81
RVH02	32	65	61	38	23	37	69	90
RVH03	54	70	50	34	19	41	51	71
RVH04	30	83	84	62	38	45	73	88
RVH05	36	83	83	70	40	67	77	87
RVH06	30	103	64	81	83	65	53	30
RVH07	28	115	51	34	35	43	76	97
RVH08	30	175	48	23	26	27	64	75
PMI01	44	81	71	75	81	88	77	42
PMI02	40	88	66	62	73	86	74	51
PMI03	32	92	54	63	79	97	81	41
NSD01	56	43	60	66	73	97	81	45
AVB01	40	83	41	66	91	79	60	26
ABT01	41	68	74	87	71	93	79	43
ICD01	40	115	23	34	90	70	59	16

further crosscorrelations would be required. Table 3 gives the results of this sequential-crosscorrelation and decision-making method. The order in which standard waveforms are crosscorrelated against as shown in Tables 2 and 3 was arbitrarily chosen on the basis of the expected relative frequency of occurrence of the respective disease groups in an effort to minimize the number of crosscorrelations performed on a large population of unknown waveforms.

Table 3. Results of the sequential-correlation method

Step No.	Standard waveform	Threshold value of β[a]	Waveforms missed[b]	Waveforms falsely classified[c]	Highest β value below threshold[d]
1.	NOR17	$\beta \geq .86$	Normal cases 10,12,16,24, 32	None	RVH05 $\beta = .84$
2.	NOR98	$\beta \geq .90$	NOR24	None	ATB01 $\beta = .87$
3.	LBB11	$\beta \geq .90$	LBB02	AVB01,ICD01 RBB07[e]	LBB02 $\beta = .87$
4.	LVH04	$\beta \geq .90$	LVH02	NDS01,PMI03 ABT01	PMI01 $\beta = .88$
5.	RBB03	$\beta \geq .90$	RBB cases 02,04,05,06, 08,10	None	RBB04 $\beta = .85$
6.	RVH11	$\beta \geq .90$	RVH cases 02,04,05,06, 08,09	None	RVH05 $\beta = .88$
7.	Classify as highest β[f]	--	RBB cases 06,09 RVH case 06	RBB cases 06[e],09[e] RVH case 06[e]	--

[a]That value of β for which classification occurs and further crosscorrelations are not required if the β under consideration is greater than or equal to this threshold value.

[b]Waveforms which belong to the classification or category represented by the standard waveform but not classified.

[c]Waveforms which do not belong to the classification or category represented by the standard waveform but classified falsely.

[d]The waveform with the highest diagnosis index β below the threshold value.

[e]Cases whose classification was wrong.

[f]If classification has not occurred prior to Step 7, classify the waveform into the category represented by the standard waveform with which the highest diagnosis index β was computed.

The results are that all normal waveforms are classified as normal and all abnormal waveforms are classified as abnormal. However, of the 52 abnormal waveforms, four are classified in the wrong abnormal group as denoted by footnote (e) in Table 3. It is also noted that all seven abnormal cases for which there was no standard waveform representing their specific disease category were classified as being abnormal.

The second method of pattern classification was to cross-correlate every test waveform against all standard waveforms and then classify the test waveform into the group of the standard waveform which it crosscorrelates with best (or gave the highest diagnosis index). This method gives the exact same overall results as the first method described although standard NOR98 classified ten normal waveforms by this method which were classified by NOR17 in the first method. This second method, while requiring less logic than the first method, has the disadvantage of requiring a maximum number of crosscorrelations and thereby using more computer time.

It is also interesting to note that all four of the mis-classified abnormal waveforms were in disease categories affecting the right ventricle. This result was anticipated, however, for the following reason. Any injury or disease of the heart shows up best in an ECG lead in closest proximity to the part of the heart where the injury or disease has occurred. Standard lead I was used for this study because it was desired

to find if a single lead taken on an easily accessible part
of the body is sufficient to diagnose most heart ailments.
Also, standard lead I is generally accepted as the best single
lead (of the twelve clinical scalar ECG leads in routine use)
to diagnose the broadest spectrum of heart diseases. While
LVH and LBB are usually seen quite well in lead I (as is shown
by Table 2), they are generally better seen in chest lead V6
which covers nearly the same area of the heart as lead I. And
while RVH and RBB can often be noted in lead I, they definitely
appear more prominantly in chest lead V1 which covers an area
of the heart quite different from lead I. Recent studies
along these lines at Iowa Methodist Hospital appear to show
that, while standard lead I is probably the best single
routinely used ECG lead, a few heart maladies just do not
generally show up in that lead. Some posterior infarcts and
septal abnormalities are examples. The above mentioned study
seems to indicate that, using pulse-amplitude and time-
duration criteria, two or three leads are necessary to see
essentially all electrocardiographic abnormalities.

Table 2 also shows that the QT interval length ranged
from 0.28 seconds in RVH07 to 0.55 seconds in RBB01 and that
the heart rate ranged from 38 beats per minute in RBB01 to 175
beats per minute in RVH08. The fact that these waveforms were
classified correctly indicates that the normalization pro-
cedures used in this study are probably valid for this problem.
However, the fact that the QT interval must be found prior to

crosscorrelation has the disadvantages of requiring a pre-
liminary pattern recognition of some sort. This will be
discussed in more detail under the topic "Automation of this
Procedure".

Another point of consideration which may be derived from
Table 2 is an answer to the question, "Given a diagnosis from
either of the two methods described, how confident can one be
that it is the correct diagnosis?" After close study of each
disease group, normal, and the seven other abnormal waveforms
listed at the end of Table 2; the range of the diagnosis
index β was noted for each of these groups correlated against
each of the standard waveforms. It is noted that there is a
high correlation between some normal patterns (see NOR11, 12,
and 16) and the LVH04 standard waveform. This high correla-
tion causes no problems in either of the two classification
schemes mentioned. These same patterns correlate even higher
with the normal standard waveform NOR98 which circumvents the
problem in the second scheme (after crosscorrelating the test
waveform against all standard waveforms, classify it into the
category represented by the standard waveform with which it
correlates highest). Also the test waveforms are cross-
correlated against the normal standard waveforms first and
accepted as normal at that time in the first scheme (a
sequential-correlation, threshold-comparison, decision-making
scheme).

Since manual techniques were required to prepare the

data-waveforms for this investigation, the number of actual cases considered was small (as shown in Table 1). Since the total number of cases was 87, this is considered to be too small of a statistical population to rigorously derive meaningful confidence levels. Also, only four common ventricular diseases were considered. It is known from other investigations performed at Iowa Methodist Hospital that some less frequently diagnosed abnormalities, due to the location of the injury in the heart, appear in other electrocardiogram lead waveforms but are not seen in standard lead I at the present "state of the art" of electrocardiogram interpretation. These abnormalities would not be expected to be found using this one-lead system.

The results indicated in Table 2 for the last seven miscellaneous disease cases are encouraging. They correlate quite poorly with the "normal" standard waveforms and quite well with disease standard waveforms—the only exception being ABT01 (abnormal T waves) which correlates fairly well with the NOR98 standard waveform.

The results shown in Table 2 would indicate that, since all 87 cases are correctly screened as abnormal or "normal", one might expect that no more than one case out of 88 would be classified wrong as to whether it is normal or abnormal. This approaches a confidence level of 99 percent. (Where this "confidence level" may be defined by saying that out of a random selection of 100 cases, one would expect no more than

one to be classified in error—or conversely, one would expect

at least 99 out of 100 to be correctly classified.) It must

be granted, however, that the statistical population (the 87

cases shown in Table 1 from which this 99 percent confidence

level is derived) is biased toward "normal" and the four

ventricular diseases considered to the near exclusion of all

other heart disease. It is nevertheless thought that a larger

unbiased population would give results nearly equaling this

confidence level.

In regards to confidence levels pertaining to correct

subclassification of abnormals into their respective disease

classifications, no apparent meaningful levels can be derived

due to the small populations (ranging from 8 to 14 cases) for

these individual disease groups. Table 2 does indicate,

however, that one might expect a fairly high confidence level

for abnormalities of the left side of the heart (represented

by LBB and LVH) when a β threshold value is used to make the

classification. But a lower confidence level would be

expected for right-side diseases (represented by RBB and LBB).

This result is also anticipated when the lead electrode place-

ment of standard lead I is considered recalling that injuries

on the area of the heart nearest the electrodes appear best in

that particular lead.

Another point in order under a "discussion of results" is

a comparison of results with the results obtained by using

other methods. Since this dissertation is an extension of

work done by Mericle (8) and Townsend (9), their results are of particular interest.

Mericle (8) investigated the feasibility of the correlation technique as applied to the electrocardiogram analysis problem. He used some sinusoidal and triangular wave approximations and showed, from a theoretical viewpoint, that correlation would be useful and practical. He did not actually use case data in an attempt to diagnose heart disease.

Townsend (9), however, did use 55 case waveforms in showing correlation to be useful in screening abnormal cases from "normal" cases. For each case, he used an entire cycle of data obtained from chest lead V6 (a chest lead covering an area of the heart very close to the area covered by standard lead I as used in this investigation). His 55 case waveforms consisted of 36 "normals" and 19 abnormals. He set his β threshold value at 0.85—high enough to avoid misclassifying any abnormals as normal. The heart rate for his population ranged from 43 to 110 beats per minute. He found, on a first iteration, that he misclassified five normal waveforms as abnormal but that all five of these cases had heart rates outside the range 60 to 100 beats per minute. He then went back and made a time-normalization correction to account for these extreme heart rates and found he then misclassified only one normal waveform as abnormal. His final results were then that he correctly classified 35 of 36 "normals" and all 19 abnormals—giving an overall figure of 54 out of 55 waveforms

correctly classified. He also noted the good correlation for LBB and LVH seen in this investigation.

The only other results available are from a study conducted at Iowa Methodist Hospital in Des Moines, Iowa, which performed an electrocardiogram interpretation using pulse-amplitude and time-duration criteria rather than a shape criterion as used in this investigation. The study was undertaken to see if a single easily recordable electrocardiogram lead could be found which would screen abnormal cases from "normal" cases. Standard lead I was chosen for that study also. Criteria were developed based upon about 2000 cases and the conclusions were that while the single lead would screen out about 85 percent to 90 percent of all abnormals, some of the missed abnormals were serious misses (such as some posterior infarcts). By adding some criteria from a second electrocardiogram lead, standard lead II, this two-lead system would screen out about 95 percent to 98 percent of all abnormals. By adding criteria from a third lead, chest lead V2, the three-lead system was found to screen out about 99 percent of all abnormals in an overall population of over 5000 cases. It is interesting to note that the three leads derived in this study approximate the three-orthogonal-lead systems under study in some research efforts today.

While no direct comparison of the results of this dissertation with the results of the Iowa Methodist Hospital study can be made, it is the opinion of the investigator that

the correlation technique can do at least as well as (and probably better than) the amplitude-time parameter criteria given the same lead system input from which each must make a classification. This opinion is based upon the ability of the correlation technique to screen out the four ventricular abnormalities studied, and also upon the relative ease with which the seven other disease cases shown at the end of Table 2 were classified as abnormal even though no standard waveform was derived for their specific disease classification.

AUTOMATION OF THIS PROCEDURE

As previously stated, lack of hardware with certain capabilities necessitated some manual procedures for this investigation. Since this study was initiated and these manual procedures were carried out, the Heart Station at Iowa Methodist Hospital in Des Moines has acquired equipment which will record one channel of ECG data in analog form on magnetic tape along with pertinent pulse-coded data on a second channel for patient identification, ECG lead amplification standardization, data-start pulses, and error pulses. There is also now available an analog-to-digital converter which has fixed sampling rates of 375, 750, and 1500 samples per second but also has the capability of being synchronized by some external signal generator to any sampling rate in the range of 0-1500 samples per second. What is still required to make automatic the procedures outlined in this investigation is some pattern recognition hardware which will compute and set the appropriate sampling rate for each individual case to accomplish the time normalization. However, it is granted that a fixed sampling rate could be used, the pattern recognition could be done digitally, and interpolation techniques could be used to accomplish the re-sampling of the waveform at the proper rate for time normalization.

This pattern recognition problem is not trivial whether it is done digitally or by hardware. If the time

normalization is to be carried out based only upon heart rate as done originally by Townsend (9), the problem is only to recognize heart rate or its corresponding RR interval (or cycle length). This problem is not too difficult because the QRS complex occurs in every heart cycle whether the case at hand is diseased or normal; and consecutive QRS complexes may be fairly readily recognized by several methods employing derivatives, high-frequency-peak detection, and other techniques. Once two consecutive QRS complexes have been found, the distance from a point in one of them to the corresponding point in the other is the cycle length and the sampling rate may be set accordingly.

However, for the purposes of this dissertation, it was also desired to time normalize out the variation of the length of the QT interval for each given heart rate. This means that all QT intervals are to be time normalized to a set number of samples regardless of heart rate or QT interval length. To accomplish this then, the QT interval must be recognized for every case and this, in turn, means that the start of the QRS complex and the end of the T wave must be found. The start of the QRS complex is, again, relatively easy to find since the complex is present in every full heart cycle of every case and has characteristic high-frequency peaks and large derivatives. The T wave, on the other hand, is more difficult to recognize. In fact, in certain disease states, the T wave is completely absent from certain ECG lead recordings. This problem may be

overcome in one of three ways: (1) use another ECG lead in which the T wave is present to find QT interval; (2) assume the QT interval is the value of the mean QT interval for that heart rate (see Figure 3) and set the sampling rate accordingly; or (3) develop some criterion for time normalizing the QRS complex alone and use zero-valued conversions to obtain the fixed required number of samples for the QT interval. If the T wave is present, there are certain methods to recognize it (such as searching for maximum deviation from baseline in a certain length sector of the heart cycle following the QRS complex).

A semi-automatic method, but relatively simple one, would be to display the ECG tracing on an oscilloscope with the heart cycle synchronized with the trace sweep, have a technician read the RR interval or QT interval (or any other desired interval) from a calibrated grid on the face of the oscilloscope, consult a nearby chart plotting sampling rate versus interval length to achieve the desired time normalization, and set a vernier knob controlling sampling rate accordingly. While this method may be less accurate than electronic or digital computer methods of accomplishing the same end, it would appear to be less subject to gross error and would require much less electronics or computer time.

SUMMARY AND CONCLUSIONS

A study was undertaken to see if a single, easily-recorded electrocardiogram (ECG) lead could be used, employing correlation techniques, to separate abnormal ECG's from "normal" ECG's and further to classify the abnormal ECG's into their respective disease categories. Only the portion of the ECG cycle resulting from the electrical action of the ventricles was considered and four ventricular diseases along with several cases of "normal" ECG's were studied. The results indicate that correlation is a valid technique for diagnosis or pattern classification but they also indicate that, while certain diseases could be seen on the lead studied with a good degree of confidence, more than one ECG lead would probably be required to be able to diagnose a broad spectrum of known heart ailments located in diverse areas of the heart.

In comparison with results of others, the results of this investigation indicate an improvement over the results obtained by Townsend (9) in both the range of heart rates applicable and also in the overall accuracy of screening abnormal waveforms from normal waveforms. The results of the correlation technique described in this investigation also appear to be (in the opinion of the investigator) as good as—or better than—the results of a study conducted at Iowa Methodist Hospital in Des Moines (which performed an electrocardiogram interpretation from pulse-amplitude and time-duration parameters) if both techniques are given the same input lead system.

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