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# False Positive" electrocardiogram"

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# THE "FALSE POSITIVE" ELECTROCARDIOGRAM

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TABLE OF CONTENTS


| | | Page |
|---|---|---|
| I. | Introduction | 1 |
| II. | Iatrogenic Heart Disease | 4 |
| III. | Sources of Electrocardiographic Error | 7 |
| | A. Technological Sources of Error | 7 |
| | B. Technical Sources of Error | 11 |
| | 1. Improper Preparation of the Patient | 12 |
| | 2. Improper Preparation of the Machine | 14 |
| | 3. Improper Placing of Electrodes | 16 |
| | 4. Improper Lead Length | 19 |
| | 5. Improper Editing, Mounting, or Labeling | 19 |
| | 6. Inadequate Number of Tracings | 21 |
| | 7. Inadequate History | 21 |
| | C. Interpretive Sources of Error | 25 |
| | 1. The P-Wave | 27 |
| | 2. The QRS-Complex | 28 |
| | 3. The RS-T Phenomena | 33 |
| | 4. The Intervals | 44 |
| | 5. The Pressure for Diagnosis | 48 |
| IV. | Summary and Conclusions | 51 |
| V. | Acknowledgement | 55 |
| VI. | Bibliography | |

# INTRODUCTION

Electrocardiographic abnormalities are in themselves not diseases. Ignorance or oversight of this basic fact causes inestimable and unwarranted anxiety among many patients and their families, deprives active citizens of necessary insurance coverage, and contributes to the creation of many "cardiac cripples" among individuals with clinically normal hearts.

The electrocardiograph has become one of the most frequently employed and widely accepted laboratory aids in clinical medicine. An electrocardiogram now is one of the cornerstones in the work-up of nearly every patient suspected of cardiac disease, and in the "routine" evaluation of many active, ostensibly healthy citizens who seek life insurance or various types of employment. Together with its widespread acceptance and use has come an amazingly naive confidence in its infallibility as a diagnostic tool - a credence that reflects the current trend toward reliance on "scientific" diagnosis, but which frequently belies the true nature of the information the electrocardiograph records.

The electrocardiograph simply measures the electrical potentials which are generated by the myocardial cells as they polarize and depolarize during the cardiac cycle and are transmitted through the adjacent heterogenous tissues to the electrodes at the body surface. The electrocardiogram cannot measure directly the thickness of the left ventricular wall, the patency of the coronary arteries,

or the diameter of the cardiac valve orifices, and attempts to quantitate these parameters from the electrical phenomena represented on the clinical electrocardiogram are at best hazardous, frequently inaccurate, and often completely erroneous.

The electrocardiograph does not lie. Within its inherent mechanical limitations, it reflects accurately the electrical potentials of the heart as they are generated during the cardiac cycle, modified by conduction through body tissues, amplified electronically, and recorded graphically. The pathologic electrocardiographic "diagnoses" assigned to clinically normal hearts arise almost exclusively from perverted attempts to translate the specific electrophysiologic data furnished by the electrocardiogram into the anatomical alterations with which they show a high, but not absolute, empirical correlation. Mechanical derangements in the recording apparatus itself and its connections to the patient, together with errors in technique of recording, account for the remainder of aberrant tracings that give rise to erroneous pathologic interpretations.

These technological and technical aberrations have become less frequent sources of interpretive error as simpler, more efficient machines and better standardized techniques have improved the average quality of electrocardiographic tracings. However, with improved tracings has come increased reliance on them in the total evaluation of cardiac status, and an ever increasing pressure on the individual interpreter to make a positive "clinical" interpretation from the electrical data of the tracing. In rare situations

where a suspicious clinical cardiac evaluation may devolve ultimately on the electrocardiogram for "diagnosis," or where an equivocal electrocardiogram represents the only "abnormality," this pressure on the interpreter becomes an obvious and potentially tragic source of misdiagnosis.

To minimize the possibility of overinterpretation of ambiguous electrocardiograms, many electrocardiographers include in their evaluations such statements as "these changes may also occur in patients with normal hearts," and "possibly . . ." or "probably normal electrocardiogram." (22) In exploring more objectively the limits of "possibly (and/or probably) normal electrocardiogram . . .," this paper will classify, describe, and explain several sources of falsely pathologic electrocardiograms recorded from clinically normal hearts in terms of accepted electrocardiographic criteria of normality and current knowledge of the electrophysiologic behavior of the clinically normal heart.

## IATROGENIC HEART DISEASE

The author has observed several patients who presented symptom complexes suggestive of heart disease, and who had been assigned various pathological cardiac diagnoses resulting in partial or complete limitation of physical activity for varying periods of time; e.g., one 39-year-old woman had been virtually bedfast for more than nine years because of reported "pulmonary stenosis, associated right ventricular hypertrophy, and intractable angina pectoris." In each case a pathologic electrocardiographic interpretation had contributed materially to the ultimate diagnosis of cardiac disease; however, upon more objective re-evaluation of the cardiac status of each patient, no clinical evidence of heart disease could be demonstrated. The symptoms manifested by these patients resulted from various pathologic or psychiatric aberrations unrelated to the cardiovascular system, and closer evaluation of the "pathologic" electrocardiograms revealed various artefacts closely simulating the changes consistent with true organic heart disease. In the case cited above, severe costal chondritis was responsible for the "intractable exertional angina pectoris;" a moderate pectus excavatum had displaced the heart leftward and compressed it, causing a functional systolic murmur over the pulmonary valve and "cardiac enlargement" on chest X-ray; and leftward cardiac displacement had caused marked apparent deviation to the right of the mean $\hat{A}QRS$ vector of the electrocardiogram, resulting in a misdiagnosis of "right ventricular hypertrophy."

In each other case, non-cardiac explanations were found for the signs and symptoms of "heart disease" manifested by these patients. Artefacts of extracardiac origin and "over-diagnosis" of equivocal electrocardiograms were responsible for misinterpretation of the tracings in each case where they had contributed to the misdiagnosis of heart disease. In a review of these cases, one obvious question arose: how could a diagnostic tool as widely employed and accepted as the electrocardiogram err so frequently?

Cardiologists recently have expressed much concern for the tragic and unnecessary plight of the falsely diagnosed "cardiac cripple." (6) Frequent articles in the current literature dealing with the accurate clinical evaluation of the heart have emphasized the hazards of translating isolated atypical types of chest pain, certain hemic murmurs, and equivocal subjective symptoms into diagnoses of heart disease. However, in spite of the almost routine use of the electrocardiogram in cardiac evaluation, its role in the misdiagnosis of heart disease only rarely has been stressed in the clinical literature, presumably due to the relative paucity of knowledge concerning the genesis of the components of the electrocardiogram and to uncertainties involving the range of the normal. Printzmetal, and others (1956) reported their experience with patients manifesting subjective symptoms of heart disease and "marked anxiety occasioned or intensified by an inaccurate interpretation of the electrocardiogram." The most important symptoms in their cases were anxiety and chest pain, and "tenderness on firm fingertip

pressure over the anterior chest wall" suggesting costal chondritis was the "most frequent physical finding." They found no clinical evidence of heart disease in this group of patients, and concluded that the initial misdiagnosis could have been avoided by a high index of suspicion of "heart disease of electrocardiographic origin" and awareness of two facts: first, that chest pain does not always represent heart disease and, in fact, "occurs as frequently in patients with normal hearts as in patients with coronary disease," and second, that ST-segment and T-wave changes "do not invariably denote myocardial abnormality" and may "sometimes be caused by the patient's fear or apprehension." (22)

Thus, the relative lack of absolute criteria for the clinical diagnosis of incipient or equivocal signs and symptoms suggestive of cardiac disease becomes obvious, and the clinician may face the unenviable responsibility of deciding black versus white in terms of grey. That he frequently should turn in these equivocal cases to the more "scientific" (ergo trustworthy) information supplied by the electrocardiogram for the ultimate "yes" or "no" decision reflects only justifiable human uncertainty and the slightly more reprehensible blind faith all too frequently vested in the expanding criteria of "scientific" laboratory diagnosis. The following discussion will describe some of the pitfalls inherent in this type of diagnosis with reference to the clinical electrocardiogram.

## SOURCES OF ELECTROCARDIOGRAPHIC ERROR

There are at least three important potential sources of error in the completed clinical electrocardiogram: 1) technological error, due to mechanical failure or maladjustment of the recording apparatus itself; 2) technical error, due to improper preparation of the patient and/or slipshod technique in recording and mounting the finished tracing; 3) interpretive error, based on ill-informed or overzealous clinical diagnoses assigned to equivocal and/or incompletely understood electrical phenomena.

Technological Sources of Error.

Electronic recording devices such as the string galvanometer electrocardiograph and the "direct writer" have inherent mechanical shortcomings, even when they are functioning perfectly, that distort the electrical phenomena they record to a measurable and unavoidable degree. In general, manufacturers of the currently popular, clinically employed electrocardiographs have minimized these mechanical limitations so that they represent clinically insignificant error when the machines are functioning properly. However, the manufacturers cannot control those defects which develop, or are magnified, as a result of normal wear or improper maintenance of the recording apparatus.

The direct-writer electrocardiograph employs a mechanical writing arm that transmits movements of the galvanometer mechanism directly to a recording stylus of either the thermal or ink type.

Inherent in this system is an unavoidable weight of the writing arm, resulting in an inertia which smooths out the small deflections that may occur in the QRS complex at a frequency rate of 100 to 200 cycles per second, deflections that are readily apparent in the tracings recorded by the more sensitive photorecording systems. Dimond terms this characteristic of direct-writers a "decrease in the frequency response," and states that the minor deflections smoothed out by this type of recorder "cannot be correlated with clinical findings," and "little, if any, clinical significance should be attached to them." (4)

Although the direct-writer does record accurately the clinically recognized electrical abnormalities of the heart, which occur at about 1.3 cycles to 40 cycles per second, in spite of its inherent inertia and insensitivity to higher frequency oscillations, it is more susceptible to maladjustments and mechanical wear than is the photorecorder. (17) These most often manifest themselves as abnormal widening of the intervals and abnormal displacement of segments, both of which may result from an abnormal degree of drag between the recording stylus and paper. The drag may result from a gradual accumulation of debris on the stylus, and has been reported from "deterioration of the vacuum tubes in the amplifier system." (17) Intervals also may be abnormal if the timer in the photorecorders is defective or if the speed at which the paper moves varies in the direct-recording machines. (17)

To minimize even further the inertia of the writing arm, many direct-writing machines employ an electronic damping mechanism which reduces the overshooting at the peak of each deflection as the weight of the recording arm carries it beyond the true peak of the electronic stimulus. If this damping mechanism becomes overactive, the excursion of the writing arm is inhibited, and the complexes recorded are deformed in the same fashion as by an excessive drag of the stylus. If damping is less than desirable, unopposed inertia of the writing arm causes overshooting, with increased amplitude of each deflection (peaked P-and T-waves, deeper Q-waves, increased amplitude of R-waves) which may give rise to misinterpretations. A loose string in the photorecording machines also may cause this overshooting artefact. Underdamping and overdamping artefacts may be recognized by careful examination of the standardization deflection (Fig. 1).

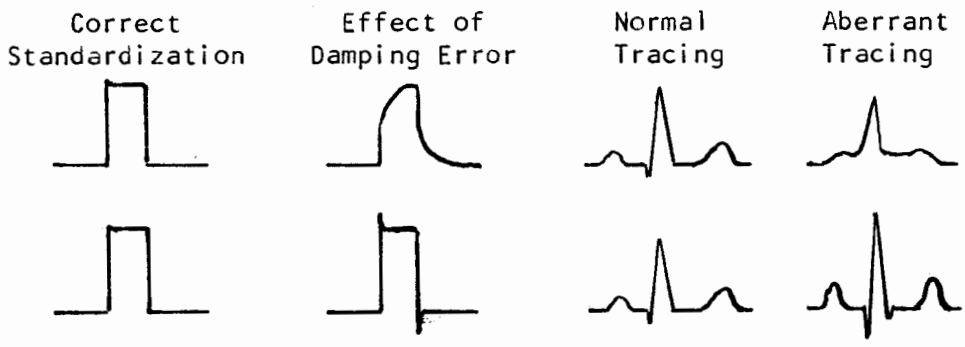


Figure 1. Effect of overdamping and underdamping. Widening of intervals, displacement of segments due to overdamping. Peaked P- and T-wave, deeper Q-wave, higher R-wave due to underdamping.

Defects in the amplification tube system which activates the writing arm may record equal positive and negative voltages as unequal deflections on the tracing, giving rise to interpretive errors in records where amplitude of the deflections may be a key factor. Dimond (1954) refers to this artefact as "asymmetrical response." (4)

Employing electrodes made of different materials may create a relative charge with respect to the two electrodes and cause a corresponding amplification or reduction of the magnitude of the projection of the cardiac vectors on the corresponding lead of the electrocardiogram. (4)

Broken lead wires may explain bizarre artefacts which appear in any two limb leads but not in the third. (4)

After long use, the metal stylus band employed on most direct-writers may become bent, twisted, or worn, resulting in poor contact with the knife edge over which the paper passes and recording a blurred base line of varying width that may hamper interpretations. (4)

An inconstant movement of the base line, unrelated to respirations of the patient, can be caused by varying line voltages supplying the machine, and may be corrected by a voltage regulator placed in the circuit between the wall outlet and the recording apparatus. (4) Base line shifts also may be caused by polarization of electrodes and by swinging of wires conducting electricity close to the lead wires connected to the patient, thereby inducing current in the respective leads. (3) Loose contact any place in the circuit

may produce sudden shifting of the base line. (3)

Kossman (1956) emphasizes that the interpreter may avoid "a grossly erroneous interpretation of a technologically inaccurate record" only by remaining alert constantly for "such variation in performance. . . of the machine," and by "fairly frequent mechanical checks on the precision of the individual instrument." (17)

Technical Sources of Error.

As is true of most laboratory diagnostic aids, the relative value of the clinical electrocardiogram in cardiac evaluation varies directly with the skill of the technician responsible for recording and mounting the tracing. Although inherently less sensitive to small, high-frequency potentials than the photorecording machines, the direct writers in general use comparatively are quite simple to operate, and present only a few pitfalls which an alert technician can avoid by an awareness of their existence and a basic knowledge of their causes and correction.

For purposes of description, the causes of technically unsatisfactory electrocardiograms may be grouped under seven basic errors of omission and commission: 1) improper preparation of the patient; 2) improper preparation of the machine; 3) improper placing of the electrodes; 4) improper lead length; 5) improper editing, mounting, or labeling; 6) inadequate number of tracings; and 7) inadequate history. The first five categories of error result from direct oversight by the technician responsible for preparing the electrocardiogram for interpretation. The last two sources of technically

unsatisfactory tracings do not result directly from primary errors by the technician himself, but are classified here with the technical errors as sins of omission that delete necessary information from the individual electrocardiogram and render clinical correlation of the tracings difficult or impossible.

## Improper Preparation of the Patient

In order to obtain uniform electrocardiograms that are useful for serial comparison of tracings and free of artefacts for accurate interpretation, the technician should prepare each patient for the recording in a manner that insures comfort, freedom from anxiety, and uniform contact with the recording apparatus.

A patient in an uncomfortable position may move to a more comfortable position during the recording, or may develop muscle cramps. Motion during recording may disturb the contact of the electrodes with the body and cause changes in resistance between the skin and the electrodes, thereby initiating movement of the galvanometer mechanism unrelated to cardiac action. In addition, potentials arising from contraction of skeletal muscle may cause changes in current through the galvanometer with small, irregular, sudden deflections of the recording apparatus that may mask critical aspects of the tracing and render interpretation inaccurate or impossible. (2)

Somatic tremors of organic etiology also may cause a confusing electrocardiographic picture. Hollendonner (1957) described a

patient in whom an isolated tremor of the upper extremity produced a "rapid, regular deflection in the electrocardiogram resembling the F-wave of atrial flutter at 280 per minute," associated with a "rapid, regular, clicking sound similar in character and location to that described in atrial flutter with a high degree of A-V block." In this case, a phonocardiogram revealed the simultaneous occurrence of the sound and the electrocardiographic deflection and their disappearance when the patient's arm was restrained. (14) The author has seen similar tracings from a patient with Parkinson's syndrome in whom the rapid, regular electrocardiographic deflections initially proved confusing, resembling atrial flutter, and the literature contains other references to lesions of the basal ganglia causing somatic tremors as a source of electrocardiographic confusion and/or error. (10, 20)

The technician must apply the individual electrodes to each patient prior to recording. Errors can arise here when the skin is not cleaned properly, causing a poor contact of the involved electrode with the skin and resulting in changes in resistance between the skin and electrode that cause sudden, sharp deflections of the base line. (17) A similar error in application of electrodes that may be less obvious upon examination of the finished tracing results from use of copious amounts of electrode jelly on the chest wall, with consequent confluence of the paste applied to adjacent points. Kossman (1956) feels this technical error "may account for the inverted T-wave seen far to the left in some reports of

(otherwise) normal precordial electrocardiograms." (17)

Improper Preparation of the Machine

Although the popular direct writing machines require few precise adjustments prior to operation, clinically significant blurring of complexes, improper amplification, and other artefacts may appear in the finished tracings if the technician fails in any of these initial preparations.

Failure to eliminate sources of electrical interference from the immediate environment probably is the most frequent source of confusing and unsatisfactory tracings due to inadequate preparation of the machine. If the recorder is not "grounded" properly to the plumbing or other suitable appliance, alternating current may produce regular, symmetrical deflections (60 per second with 60 cycle AC) that notch the baseline and complexes and render interpretation difficult or impossible. This alternating current interference may arise from obvious sources such as metallic objects in contact with the patient and electric blankets over the patient, or from more obscure sources such as synthetic coverings on the examination table, electric lamps in adjacent rooms, wall paint running into the AC outlet and acting as a conductor with the entire wall as a source of 60 cycle interference, and a simple increased humidity in the room causing accumulation of moisture on the patient and equipment and providing a pathway for leakage of interference. (4)

Electric interference at other frequencies may be caused by ringing telephones (20 cycles per second), by dialing telephones

(5 cycles per second), by X-rays producing $\frac{1}{2}$-sine wave artefacts, and by fluorescent lights producing high frequency interference. (4)

Uncleanliness and corrosion of electrodes and ends of lead wires, if neglected by the technician, may cause poor contacts between the patient and the recorder with resultant sharp deflections of the base line. (17) Ironically, overzealous attempts at cleanliness through polishing of electrodes with steel wool or other soft metal abrasives also may cause base line deviations by polarization of the electrodes. (4)

The technician may cause errors in interpretation of ventricular hypertrophy or dilatation, emphysema, myxedema, and other interpretations in which amplitude of the complexes is an important criterion, by failing to adjust the amplification of the recorder to the accepted full standardization of a ten millimeter deflection in response to a stimulus of one millivolt, so that those interpretations consistent with high or low voltage may be suspected erroneously. When very high voltage stimuli truly do exist, the corresponding deflections of the stylus may extend beyond the limits of the recording paper and render interpretation inaccurate unless the technician centers these particular leads individually on the paper and/or reduces the amplification of the recorder until the deflections will trace on the graph paper.

## Improper Placing of Electrodes

In the standard clinical electrocardiogram, the four electrodes that usually are attached to each wrist and ankle, together with the unipolar suction-cup electrode that is moved to each anatomical precordial lead site, provide the only connection between the patient and the recording apparatus. The skill and uniformity with which the technician applies these electrodes to each patient is a significant factor in the reliability of the individual electrocardiogram, and in the interpretation of serial tracings to evaluate changes over a period of time.

An electrode attached to a limb more tightly than the other electrodes are attached will be more positive electrically in relation to the remaining electrodes, and the magnitude of the projection of the cardiac vector on the leads served by this electrode may be altered accordingly. (4)

The conventional location of the limb leads on the distal extremities, and the chest leads in the fourth and fifth intercostal spaces from the right sternal border to the left midaxillary line, provides uniformity of serial electrocardiograms within the limits imposed by normal anatomical variation of chest configuration, position of the heart in the thorax, and the ability of the heterogenous body tissues to act as an ideal volume conductor. However, when deformities of the chest wall or errors in locating the precordial electrodes alter markedly the anatomical sites of the

leads in relation to the position of the heart within the chest, the magnitude of the projection of the cardiac vector on the leads changes accordingly, and apparent deviations of the mean $\bar{A}$QRS suggesting ventricular hypertrophy or "abnormal" Q-waves suggesting myocardial infarction may result.

Paradoxically, as in the case of pectus excavatum described above, either true displacement of the heart within the chest (pectus excavatum) or relative displacement of the heart (misplaced electrodes) shifts the apparent direction of any cardiac vector opposite to the direction of the displacement of the heart. A change in the accepted geometric and spatial reference systems of the leads, caused by the true or relative shift of the center of electrical activity of the heart, explains this paradoxical artefact (Fig. 2). The principle that a true or relative displacement of the heart causes an apparent displacement of the cardiac vector in the opposite direction may be applied in the evaluation of any electrocardiographic "abnormality" in which thoracic deformity, misplaced electrodes, or true cardiac displacement could be involved.

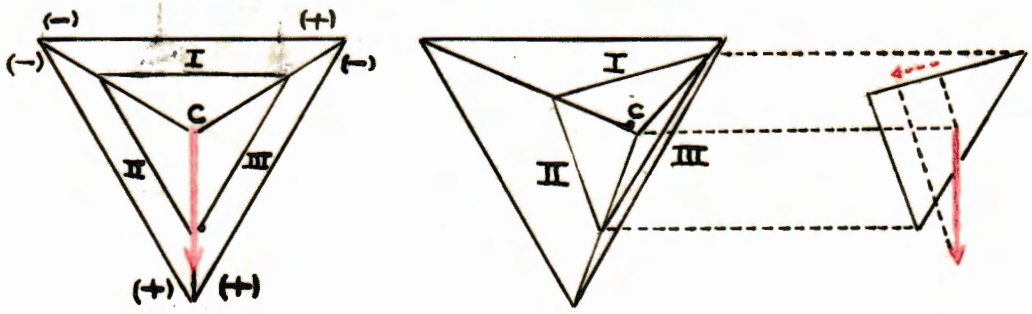


Figure 2. Effect of true or relative displacement of the center of electrical activity of the heart to the left. (A) Einthoven reference system employed in interpretation of clinical electrocardiograms. Geometric orientation of leads I, II, III arbitrarily designated as an equilateral triangle. Center of electrical activity of the heart at point "c". Cardiac vector at + 90° (in red) projects as neither negative nor positive on lead I. (B) Displacement of point "c" to the left alters the geometric orientation of leads I, II, III. Cardiac vector at + 90° now projects to the right as a negative vector quantity in lead I.

A similar error occurs when any two limb lead wires are transposed as the technician connects them to the electrodes. Grant (1957) points out that the limb lead electrocardiogram may be identical to that of primary dextrocardia if the technician reverses the right and left arm lead wires, and emphasizes that this diagnosis should not be made from the limb leads alone. (11) Provided no change in rhythm occurs between leads, mixed lead wires usually can be detected by application of Einthoven's Law, which states that a deflection in lead II has a magnitude which is the algebraic sum of

the simultaneous deflections in leads I and III. (17) Another error related to lead orientation may occur if the technician fails to turn the lead selector switch from the last of the limb leads, aVF, to the central terminal position before he records the precordial leads; thus, all the "precordial" leads will appear identical and may confuse interpretation unless the similarity of each to lead aVF is noted.

## Improper Lead Length

In theory, only one complex from each standard electrocardiographic lead should be required to analyze the electrical activity of the heart as it polarizes and depolarizes during the cardiac cycle. However, in clinical electrocardiography, the technician often records the tracing at the bedside of seriously ill patients, under conditions where artefacts due to electrical interference and movements of the patient may be difficult to eliminate. Consequently, artefacts may distort many complexes. If the technician fails to record a generous number of complexes from each lead, the finished tracing may show only aberrant or distorted complexes in any given lead or leads, or may have an inadequate number of consecutive complexes for diagnosis of arrhythmias.

## Improper Editing, Mounting, or Labeling

The electrocardiographic technician usually is responsible for editing the several lead tracings, labeling them correctly, and mounting them in a designated order on a card or folder prior to

final interpretation by the electrocardiographer. Important errors that may confuse the interpreter can occur during each of these steps.

The technician, who frequently is untrained in evaluation of electrocardiograms, may trim off important abnormalities in cutting the leads to size for mounting. He may label the leads incorrectly and/or arrange them in improper sequence on those folders that provide specific labeled slots for a given length of tracing from each lead. He may also mount leads upside down, which Kossman (1956) has described as an occasional cause of mistaken diagnosis of myocardial infarction. (17) Like mixed lead wires, tracings mounted upside down usually may be detected by the application of Einthoven's Law. (17)

After the tracing is prepared for interpretation, a final error may occur if the record is assigned to the wrong patient. This occurs more frequently in hospitals where many electrocardiograms are taken daily, and particularly if electrocardiograms are transmitted to the laboratory through a permanent wiring system with outlets near the bedside. The source of the electrocardiogram in question may be clarified by comparing the tracing with those recorded from other patients on previous days until similar tracings belonging to another patient are found, or by noting from the record all the other tracings taken on the same day. (17)

## Inadequate Number of Tracings

Certain electrocardiographic interpretations and observations depend upon comparison of serial tracings recorded over varying periods of time for adequate evaluation. True progression of changes can be shown conclusively only by three serial tracings, to confirm a change that only may be suspected from two consecutive electrocardiograms. The diagnosis of acute myocardial infarction from electrocardiographic changes on a single tracing may be hazardous because residual myocardial scars from previous infarctions, or persistent ventricular aneurysms, may cause QRS complex alterations or ST-segment deviations that may suggest acute myocardial damage upon initial examination. Persistence of the abnormalities without demonstrable change in subsequent tracings may confirm the diagnosis of old infarction or ventricular aneurysm and rule out the presence of an acute myocardial infarction. (2, 4, 11, 26)

Obviously, accurate long-term evaluation of the electrocardiographic behavior of the heart is not possible without an adequate number of serial tracings to demonstrate comparative alterations as they may develop during the period of time in question.

## Inadequate History

The electrocardiographer often is expected to interpret the electrical phenomena represented on the electrocardiogram in terms of their possible clinical causes. In order to arrive at these etiologic conclusions with any reasonable degree of certainty, he

should have exact information stating why the tracing was desired and full knowledge of any or all clinical factors which may influence the final tracing before he begins his evaluation.

Many factors unrelated to the immediate indications for the electrocardiogram may influence profoundly the final tracing submitted for interpretation. For example, the orientation of the anatomical and electrical axes of the heart within the thoracic cavity vary from a vertical tendency in the tall, thin individual with a low diaphragm to a more horizontal orientation in the short, plump, or pregnant individual with a higher diaphragm. (26) Very obese individuals, whose excess tissues have increased electrical resistance, may show a tendency to lower voltage complexes than slender individuals with less resistant tissue interposed between the heart and electrodes on the body surface. Thus, the electrocardiographer must know the body configuration of the individual whose tracing he intends to interpret before he can evaluate adequately possible abnormalities of the mean $\hat{A}$QRS axis, apparent high or low voltage complexes, and possible repolarization abnormalities of the RS-T interval.

Evans and Lloyd-Thomas (1957) emphasized how the position of the heart in the thoracic cavity could influence the projection of the cardiac vector on the standard electrocardiographic leads. They described thirteen patients who presented with nonspecific chest pain and/or palpitation with no other demonstrable evidence of cardiovascular disease on "routine clinical examination," but who all

showed depression of the S-T segment in leads II and III accompanied by occasional Q-waves in the same leads. Exercise dic not exaggerate these changes, thus "separating them from those due to. . . infarction." In each case, X-ray of the chest revealed wide separation of the cardiac and diaphragmatic shadows, especially during inspiration. They postulated that separation of the heart from close contiguity with its surrounding structures, especially the diaphragm, "disturbs the anatomical medium that customarily transmits the varying electrical potential generated by cardiac contraction. . ." so that the tissues do not act as an ideal volume conductor and the spatial orientation of the cardiac vectors is altered accordingly. (7) Thus, when the heart is separated from the diaphragm, due to the decreased conductivity of lung tissue the cardiac potentials follow a new path of least resistance via the more conductive vertebral structures and orient themselves more posteriorly, changing the magnitude and/or direction of their projection on the standard leads of the electrocardiogram (Fig. 3).

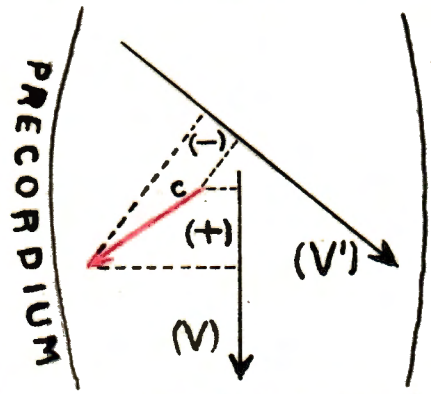


Figure 3. Alteration of orientation of the lead vector by separation of heart from diaphragm. Center of electrical activity of the heart at point "c". Cardiac vector in red. (V) Lead vector when the heart contacts the diaphragmatic structures; projection of the cardiac vector on the lead vector is a positive vector quantity. (V') More posterior orientation of lead vector, due to decreased conductivity of lung tissue, when the heart and diaphragm are separated; projection of the cardiac vector on the lead vector becomes a negative vector quantity.

The direction of the mean QRS vector also varies somewhat with the age of the patient. Grant (1957) states that right ventricular predominance orients the mean QRS axis "nearly horizontally rightward" in direction at birth, and that it tends to become directed more inferiorly and slightly leftward during infancy, continues leftward to become nearly parallel with the lead II axis in later childhood, and shifts further leftward with age, corresponding to the "gradual development of left ventricular predominance anatomically and physiologically in the adult." (11) The mean T-wave axis in infancy points leftward and posteriorly, away from the predominant right ventricle, and gradually swings more rightward and anteriorly with age as "right ventricular predominance regresses and left ventricular predominance gradually appears." (11)

The QRS-T angle varies accordingly, "not often exceeding 45° in the frontal plane or 60° in the anterior-posterior plane" in the normal adult electrocardiogram. (11)

The interpreter also must be informed of the use of cardiac drugs such as digitalis and quinidine, which affect the electrical behavior of the heart in a specific manner, before he can evaluate accurately changes in the electrocardiogram resembling these effects.

The relative necessity of certain historical information -- especially indications for the electrocardiogram, age of the patient, body type, and drugs in use -- to orient the electrocardiographer in his evaluation of the electrocardiogram is clear; the contribution this information makes toward accurate correlation of the electrical data of the tracing with the clinical impression desired must be emphasized.

Interpretive Sources of Error.

Assuming that the electrocardiograph functions properly and that the finished record contains no technical flaws -- assumptions which will be more or less justified by the condition of the apparatus and the skill and reliability of the technician -- still another source of falsely pathologic electrocardiographic interpretations may contribute to misdiagnosis. This category of errors is based on the interpretation of the electrical data of the record; its incidence necessarily varies inversely with the skill and clinical experience of the individual electrocardiographer; its source lies in perverted attempts to correlate the anatomic malformations and

lesions noted clinically or pathologically with the modifications of the electrical behavior of the heart noted in the electrocardiographic leads. The correlation between the two, though often good, never can be perfect.

Empirical criteria delineating acceptable limits of normal for the electrical behavior of the human heart have evolved from observation of a very large number of clinically normal and diseased hearts. As more observations become available, the empirical limits of normal electrical behavior of the heart will be revised accordingly to include the greater fund of information, and a more accurate correlation between these empirical electrical criteria of normality and true absence of cardiac pathology presumably will result. However, until the electrical behavior of the heart can be described and understood in its entirety, occasional healthy hearts will continue to behave beyond the best known empirical limits of electrical normality and erroneously will be considered diseased.

For purposes of description, various non-cardiac factors that may cause the electrical behavior of the heart to approach or exceed the empirical limits of electrical normality will be discussed in relation to the segments of the electrocardiographic complex which they alter, recognizing that some factors may influence more than one segment of the complex.

## The P-Wave

Because the sinoatrial node where normal atrial activation begins lies in the right atrium, the atrial depolarization wave normally passes across the atria from right to left; thus, the normal P-wave is an upright deflection in leads I and II, and may be either upright or inverted in lead III. It normally persists for about 0.08 second and rarely has an amplitude greater than 2.0 mm. in the limb leads. (11)

Tall, peaked P-waves associated with right auricular dilation and broad, notched P-waves associated with left auricular dilation are the most common P-wave changes correlated with organic heart disease. (11)

In his study of emotional influences on the electrocardiograph, Weiss (1956) described increased amplitude of the P-wave in response to emotional stresses evoking responses of the sympathetic nervous system, and attributed the peaked P-waves to increased conduction velocity in the atrial musculature. (31) Gardberg and Rosen (1957) noted an increased amplitude of the P-wave in response to exercise, ingestion of food, smoking, and hyperventilation, and felt that the increased heart rate noted in each case may have been a common etiologic factor. (9, 25)

Increased amplitude of the P-wave secondary to these factors may suggest right auricular dilatation and confuse interpretation in hearts where this effect is marked.

## The QRS Complex

The QRS complex of the electrocardiogram is recorded coincident with the passage of the activation wave through the ventricular myocardium and represents the potentials generated by the depolarization process as they pass from the heart via the tissues to the electrodes at the body surface and are recorded. Although the individual instantaneous vectors contributing to the QRS complex may vary markedly in normal hearts, the mean spatial AQRS vector shows relatively much less variation, being directed rightward and anteriorly at birth and swinging gradually leftward and posteriorly as left ventricular predominance develops with age. (11) The accepted limits of normal variation in the direction of the frontal plane mean $\hat{A}QRS$ vector in the adult are + 110° and - 30°. (4) Arbitrary right and left axis deviation exist beyond these limits. The QRS complex rarely exceeds 0.08 second in duration in the normal heart, and does not show initial negative deflections, Q-waves, greater than 0.03 second in duration. (4) In general, deviations of the QRS complex from these criteria usually are assumed to indicate organic heart disease. However, several exceptions to the above criteria have been described in the absence of primary cardiac disease.

In their description of the "syndrome of the suspended heart", illustrated above under Inadequate History, Evans and Lloyd-Thomas (1957) described "abnormal" Q-waves in leads II and III, and attributed them to a more posterior orientation of the lead vector

secondary to lung tissue inter posed between the heart and the diaphragm. (7)

Peñaloza and Echevarria (1957) studied the electrocardiograms of ten patients taken from sea level to an altitude of 15,000 feet for one year, and noted that the SÂQRS vector shifted markedly to the right and posteriorly. Although they suspected the development of incipient right ventricular hypertrophy later in the year, they attributed the early shift to a "clockwise rotation around the long axis of the heart, with backward rotation of the apex, determined by hyperventilation at high altitudes." (21)

Grant (1957) described a condition "commonly seen in perfectly normal young people" in which the terminal QRS vectors are directed markedly rightward and superiorly with no prolongation of the QRS interval in an "$S_1S_2S_3$ syndrome." (4) The "abnormal" direction of these terminal vectors may confuse interpretation of an otherwise normal tracing, suggesting right ventricular hypertrophy.

Some electrocardiographers maintain that the anatomic modification of ventricular hypertrophy is more evident from the electrocardiographic changes it causes than from any other clinical or laboratory examination. (17) QRS complex criteria for the diagnosis of ventricular hypertrophy may vary with individual electrocardiographers, but usually do include a swing of the mean SAQRS axis toward the ventricle predominantly involved, a variable increased amplitude of QRS deflections, and a delay of the intrinsicoid

deflection with a variable increase in the total duration of the QRS complex; a rotation of the RS-T vectors, tending toward 180° opposition to the mean SÂQRS axis, is usually included with the QRS complex criteria. (4) It is not the purpose of this discussion to debate the varying criteria for the diagnosis of ventricular hypertrophy, but rather to point out the hazards of making the diagnosis by any of the above QRS criteria. Dimond (1954) emphasizes that the rotation of the mean SÂQRS axis left and posteriorly in left ventricular hypertrophy can also be caused by a "high diaphragm with the heart in a horizontal postion;" that. . . "a tall, thin man with a normal heart may have taller complexes at $V_5$ than a thick-chested man with considerable ventricular hypertrophy," casting doubt on the amplitude of the QRS complexes as an arbitrary criterion; and that "tangential or lateral spread. . . of the depolarization wave through the ventricular myocardium. . . may obviate the supposed value of the intrinsicoid deflection." (4) Clearly, the diagnosis of ventricular hypertrophy from the electrocardiograph alone may be hazardous, especially where the changes involved may be minimal.

Several primary cardiac conditions may present electrocardiographic pictures similar to those of myocardial infarction. Although most of these occur in individuals with pre-existing heart disease, they are included here because an erroneous electrocardiographic diagnosis of infarction is a serious mistake in any individual case. Sodi-Pallares (1956) divided these errors into four groups, according to the location of the infarct they suggested. (26)

Anteroseptal infarction may be imitated by conditions resulting in right auricular dilatation associated with qR or QR patterns in the right precordial leads. Tricuspid stenosis or regurgitation, chronic cor pulmonale, acute cor pulmonale, and interatrial septal defect were implicated as causes of the right auricular dilatation. Left bundle branch block with QS complexes in the right precordial leads also may suggest anteroseptal infarction. (26)

Conditions causing left auricular dilatation may show QS tracings in leads I and VL and thus may mimic lateral infarcts of the left ventricle. Mitral lesions are the primary offenders. (26)

Tracings erroneously suggesting inferoseptal infarction, with Q-waves in leads III and VF, may be due to "numerous normal circumstances, notably those with a deep $Q_3$ in the horizontal hearts of obese people," and also to acute and chronic cor pulmonale and Wolff-Parkinson-White syndrome of the B type. (13, 26)

Extensive anterior infarction may be suggested by tracings showing QS complexes in leads I and VL, as well as in all the precordial leads, in patients with chronic cor pulmonale. These complexes "probably correspond to intracavitary potentials" which appear in the leads mentioned as a result of marked descent of the diaphragm with backward displacement of the apex, allowing ". . . the electrode to explore the base of the heart and, therefore, its cavities." (26)

The special electrocardiographic variations described above inherently may be more susceptible to misdiagnosis of myocardial infarction because many are taken from individuals with known cardiac disease who may complain of chest pain and other suggestive symptoms. The hazards in the diagnosis of myocardial infarction from isolated QRS complex changes are clear from these examples. However, Sodi-Pallares points out that in each case careful attempts to correlate associated P-wave changes, RS-T interval alterations, and the clinical history with the QRS abnormalities should reveal their true origin. (26)

Apparent low voltage of the QRS complexes may suggest myocardial disease where none exists. Lipman (1956) emphasizes that an excessively thick or obese chest wall, or a decrease in the conductivity of the tissues around the heart due to emphysema or to pleural or pericardial effusion, may cause low voltage tracings in the absence of primary heart disease. (18)

Wasserman, and others (1956) reported electrocardiographic observations from twelve patients with cerebrovascular accidents. In several of these tracings "electrical systole. . . occupied the entire interval up to the initiation of the next succeeding atrial impulse." These "QRS changes" were associated with marked prolongations of the S-T interval and deep and wide T-wave inversions. No primary myocardial disease was demonstrated, and they felt that "indirect effects of the profound disturbances which involved directly or indirectly various portions of the brain involving the

cardiac centers" may have caused the changes. (30)

Although the majority of alterations of the QRS complex beyond the empirical limits of electrical normality probably do represent primary myocardial disease, the QRS complex changes described above, in each case suggesting myocardial abnormalities that did not exist, show that apparent abnormalities of ventricular depolarization may have several sources, and certainly all do not represent specific types of organic heart disease.

## The RS-T Phenomena

The RS-T interval is measured from the end of the QRS complex to the end of the T-wave, and represents the duration of the depolarized state plus the duration of repolarization. (3) The early and late T-vectors normally differ ± 10° in direction, and the normal position of the mean spatial T-wave is related to the position of the mean spatial QRS so that the QRS-T angle seldom exceeds 45° in the frontal plane or 60° in the anterior-posterior plane in the normal adult heart. (11) Although the T-waves normally may vary with the position of the QRS, Burch (1955) states that "a negative T-wave in lead I, for all practical purposes, is definite evidence of myocardial disease," as is deviation of the S-T segment from the base line "more than plus or minus one millimeter in the standard leads." (3)

Myocardial infarction, pericarditis, trauma, ventricular hypertrophy, or exercise in patients with coronary disease cause recognized pathologic RS-T abnormalities, frequently associated with characteristic QRS complex alterations. (2, 4, 11, 26)

The effects of several non-pathologic and/or extracardiac factors implicated in RS-T changes are less well defined, and many authors have emphasized the similarity of these innocuous RS-T changes to those caused by true myocardial or coronary artery disease. (2, 4, 6, 8, 9, 14, 17, 19, 21, 23, 24, 25, 28) These factors include changes in heart rate, (, 25), exercise (9, 25), eating of food (9, 25), smoking (9, 25), changes in the respiratory position (9, 10, 23, 25), use of digitalis (2, 4, 11, 26, and others), hiatus hernia and gall bladder disease (2), cerebrovascular accidents (30), chronic anoxia (high altitudes) (12), emotions (22), electrolyte disturbances and use of mineralocorticoids (5, 12), diabetes mellitus (16), surgical sepsis (22), drinking iced water (4, 26), Wolff-Parkinson-White syndrome (13), persistence of the 'juvenile T' pattern (4), influence of the T-a wave (4, 9), and normal S-T segment elevations in young people (2, 4, 11, 26). Attempts to establish empirical criteria which will differentiate the RS-T changes due to myocardial disease from those due to various extracardiac factors have reached no really satisfactory answer. (17)

Since electrocardiograms frequently are taken under uncontrolled conditions and interpreted without regard to, or knowledge of, the possible effects of many of these extracardiac factors, they constitute an obvious and potentially tragic source of electrocardiographic misdiagnosis. Fischbach (1956) feels that interpretation of recent myocardial injury, damage, or infarction on the basis of

minor fluctuations in the T-waves caused by extracardiac factors is "probably the most frequent source of error in electrocardiographic interpretation." (8)

Even experienced cardiologists can not agree whether several of the above factors truly do influence the electrocardiograph or not. Bloom and Gubbay (1957) studied 15 patients with hiatus hernia under fluoroscopic observation in the left lateral position during a barium swallow with simultaneous recording of leads I, VF, $V_6$, and $V_8$. They demonstrated the hernia fluoroscopically in 11 of the 15 patients, but could demonstrate none of the T-wave changes attributed to hiatus hernia by many other authors; nor were they able to elicit any vagal effects that "could be ascribed to reflex or mechanical effects arising in the hernia," as judged by observation of the heart rate, rhythm, and measurement of the P-wave and P-R intervals. (2)

Similarly, Kalliomaki and others (1956) studied the electrocardiograms of a large number of diabetics in varying states of control, but with "clinically normal hearts," and failed to corroborate inverted T-waves and depressed S-T segments described in several previous studies, concluding that "diabetes mellitus in cases of pure metabolic disease without late diabetic vascular complications exerts very little or no influence upon the standard electrocardiogram; neither does mild diabetic acidosis." (16)

Gardberg and Rosen (1957) felt that the empirical criteria for normal behavior of the RS-T vectors were inadequate and too frequently erroneous, so they undertook a "controlled study. . .

of the more common nonpathologic factors. . . operative in everyday life. . . 1) exercise, 2) the taking of food, 3) combinations of 1 and 2, 4) smoking, 5) change in posture, and 6) changes in the respiratory level," believing that only a "fundamentally physiologic approach to the study of the recovery (T) potentials" could furnish ultimately satisfactory criteria to correlate the confusion of empirical data. (9) They recorded electrocardiograms in the lying and sitting positions from "normal young men and women (18 to 30 years of age)" in the "fasting (state), after 20 deep knee bends and every three minutes for fifteen minutes, after breakfast and every fifteen minutes for one hour, after a repetition of the exercise, and after smoking one cigarette." (9)

They observed that all of the above factors caused qualitatively similar effects. The mean $\hat{SAT}$ swung either to the left or right or remained unchanged, and usually diminished in magnitude. In general, the T-waves and RS-T segment tended opposite to the main deflection of the QRS, showing flat T-waves in leads 1, $V_4$, $V_5$, and $V_6$, with occasional inversion; RS-T segment shifts were common, with alterations greater than one millimeter "not rare." The magnitude of RS-T shifts was proportional to the amplitude of the main deflection of the QRS complex and to the increase in heart rate. Although occasionally they observed RS-T changes with no increase in heart rate after a meal, the ingestion of food usually did cause an increased heart rate, and they felt this may have been the etiologic common denominator in the observed changes. (9) Further, they

concluded that the nonpathologic factors they studied, and/or the increased heart rate secondary to these factors, caused the RS-T alterations by a primary diminution of the ventricular gradient, which Wilson originally described as the net electrical effect of differences in the rates of repolarization in the various regions of the ventricles. (26)

Since repolarization requires much more time than depolarization, most of the areas of the myocardium are undergoing repolarization simultaneously, except at the very beginning and end of the process; thus, many of the instantaneous dipoles throughout the myocardium cancel each other electrically during repolarization, and the net effect equals the "algebraic sum of the dipole effects of the endocardial and epicardial surfaces." (26) These surface effects may be represented by two electrically opposed monophasic curves illustrating depolarization and repolarization at the endocardium and epicardium (Fig. 4). If depolarization and repolarization occurred at a uniform rate at both the endocardial and epicardial surfaces (Fig. 4a), the T-wave (repolarization) would be equal in area and opposite in polarity to the QRS complex (depolarization). However, in the human heart, the T-wave normally records as a deflection in the same direction (polarity) as the QRS complex and of a somewhat different area (magnitude) than the QRS complex. The simplest explanation for this phenomenon is that repolarization occurs more rapidly at the epicardium than at the endocardium for reasons explained only by theory (Fig. 4b).

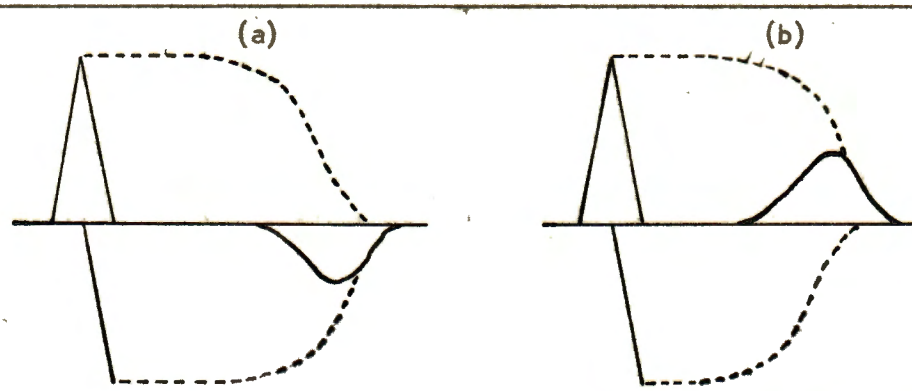


Figure 4. Effect of rates of repolarization at the endocardium and epicardium. (a) Rates of depolarization and repolarization at endocardium (upper monophasic curve) and epicardium (lower monophasic curve) equal. Deflections of QRS complex and T-wave equal in area and opposite in polarity. (b) Rate of repolarization more rapid at the epicardium. QRS complex and T-wave show same polarity and different areas (magnitude).

Where repolarization at the endocardium and epicardium are not uniform, depolarization (QRS complex) and repolarization (T-wave), added algebraically, do not cancel each other electrically, but rather have a finite algebraic sum which is a measure of the net electrical effect of differences in rates of repolarization at the two surfaces, the ventricular gradient of Wilson. Since the T-wave theoretically would record equal and opposite to the QRS complex if endocardial and epicardial repolarization proceeded at equal rates, the gradient potentials represent the individual instantaneous potential differences between this theoretical T-wave and the observed T-wave (Fig. 5). (26)

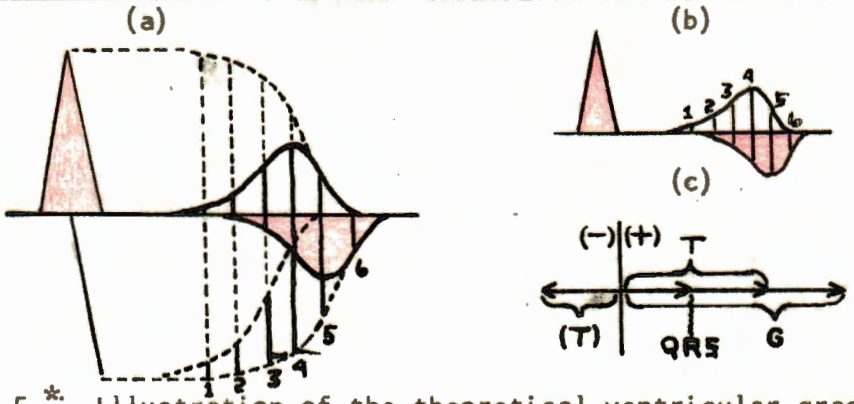


Figure 5.* Illustration of the theoretical ventricular gradient potentials. (a) Monophasic curves resulting in the theoretical and observed T-waves superimposed on each other (see Figs. 4a and 4b). Instantaneous negative potentials 1-6 are equal to the simultaneous instantaneous potential differences between the observed and theoretical T-waves (the gradient potentials). (b) Area of QRS complex equal to area of theoretical T-wave. Gradient potentials from (a) shown as instantaneous potential differences between theoretical and observed T-waves. (c) Vectorial representation of $\hat{A}QRS$, $\hat{A}T$, $\hat{A}(T)$, G. $\hat{A}QRS = \hat{A}(T)$. $\hat{A}T + \hat{A}(T) = G$. $G = \hat{A}QRS + \hat{A}T$.
* After Gardberg and Rosen, 1957.

Gardberg and Rosen (1957) pointed out that the algebraic sum of the depolarization and repolarization potentials is constant regardless of the orientation in time of the endocardial and epicardial monophasic curves; thus, the gradient potentials remain constant regardless of the time relation of endocardial activation to epicardial activation, and are independent of the area of the myocardium excited first. (26)

Gardberg and Rosen (1957) cited Ashman's work with the effect of heart rate on the ventricular gradient in the turtle heart, in which he recorded simultaneous monophasic curves from a cooled and uncooled region of the same heart and found that the longer monophasic curve from the cooled area was shortened more than the

shorter monophasic curve from the uncooled area, the curves approaching the same form and duration, with a simultaneous diminution of the gradient between them, with very rapid heart rates. (26) From this experimental evidence, they reasoned that, if the same phenomenon applied to the human heart whose monophasic curves (rates of repolarization) are not uniform, then as the gradient diminished with increased heart rate the T-wave should become lower and finally become inverted (Fig. 6). (26) Their clinical observations on the effect of exercise, ingestion of food, smoking, and change in respiratory position, coincident with an increased heart rate, on the RS-T phenomena in electrocardiograms from clinically normal hearts agreed with this postulate. (9)

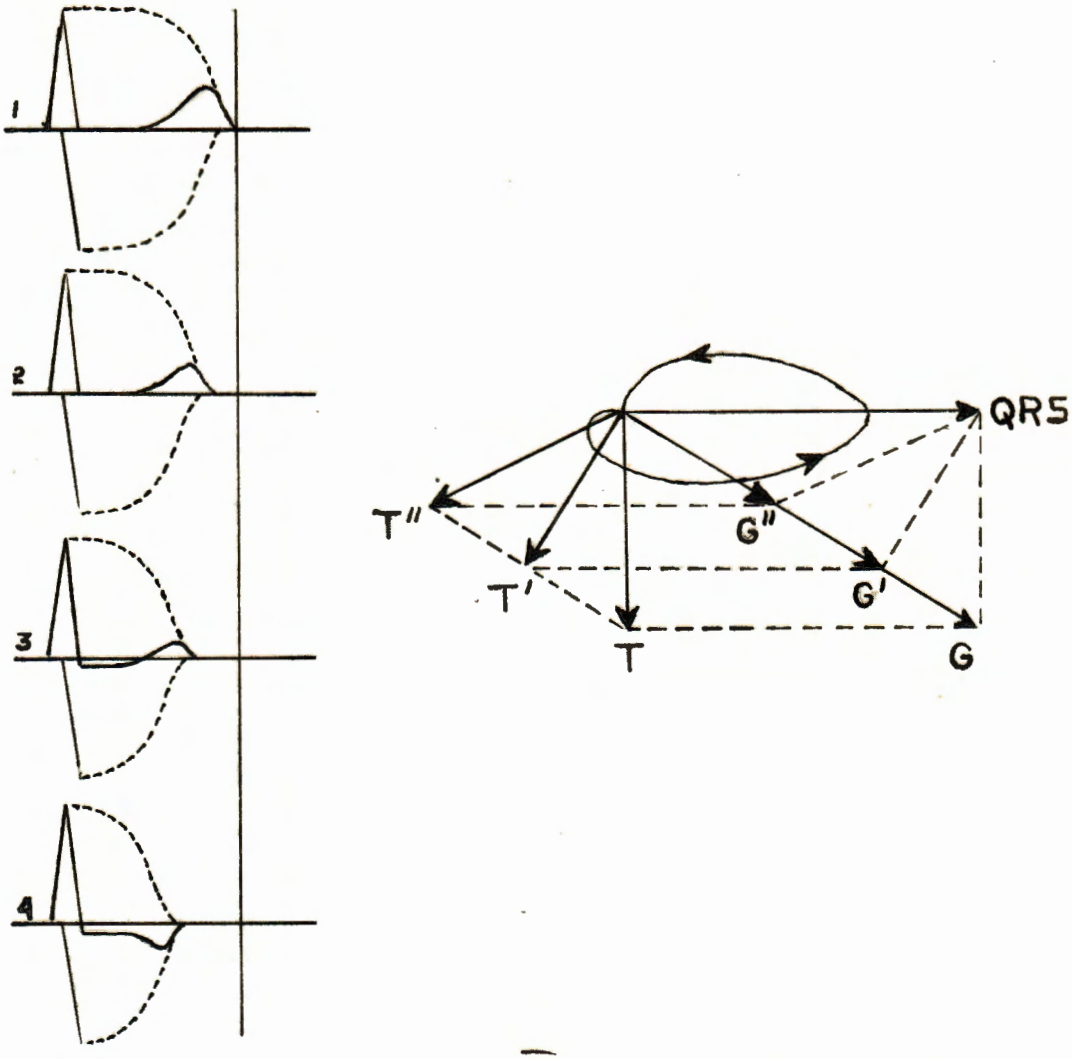


Figure 6.* At left, effect of increasing heart rate upon two monophasic curves of different durations. Discussion in text. At right, effect on the mean T vectors of a primary diminution in the ventricular gradient with no change in its direction. Spatial angle between the mean QRS and mean T vectors increases.
* After Gardberg and Rosen (1957).

Further, Gardberg and Rosen (1957) pointed out Ashman, Gardberg, and Byer's conclusion that the orientation of the mean spatial QRS vector was at a spatial angle of approximately 90° to the longitudinal axis of rotation of the heart, with the spatial gradient vector oriented between the two at a spatial angle of about 30° with the QRS, because the depolarization wave passes more or less diagnonally across the ventricular wall while the postulated repolarization forces of the gradient act more perpendicularly across the ventricular wall (Fig. 7). (26)

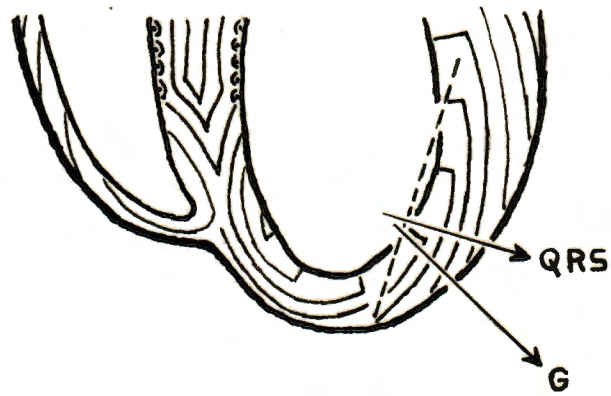


Figure 7.* Relation of the mean spatial QRS vector and spatial gradient to the longitudinal axis of the heart. $S\hat{A}QRS$ lies approximately 90° from longitudinal axis. $S\hat{A}G$ between $S\hat{A}QRS$ and longitudinal axis about 30° from $S\hat{A}QRS$ in nearly the same plane.
* After Gardberg and Rosen (1957).

Consequently, rotation of the heart counterclockwise locates the frontal plane gradient to the right of the mean QRS, and clockwise rotation of the heart places the gradient to the left of the mean QRS; thus, in response to a primary diminution of the ventricular gradient, the T-wave should deviate to the right in a clockwise-

rotated heart, to the left in a counterclockwise-rotated heart, and should appear smaller but should not change its direction in a non-rotated heart. This postulate also agreed with their electrocardiographic observations of the effects on repolarization of the nonpathologic factors they studied, and explained the apparently random T-wave deviations described above. (26)

From this observation and reasoning, Gardberg and Rosen (1957) concluded that any of the nonpathologic factors which they studied could cause "RS-T segment shifts and T-wave inversions. . . in almost any lead of the electrocardiogram of normal persons" as a result of a primary diminution of the ventricular gradient, and that accurate "interpretation of the effects of these factors without . . . the concept of the ventricular gradient. . . (would be). . . impossible." (26)

Although this concept of the ventricular gradient indeed may explain, in time, some of the RS-T changes associated with other poorly understood extracardiac influences on the electrocardiogram, the greatest security in the evaluation of isolated RS-T deviations probably still lies in the admonition of Kossman (1956) to "differentiate these. . . by recourse to. . . a careful history. . . a thorough physical examination, and (adequate) laboratory survey." (17)

## The Intervals

The P-R interval and the Q-T interval concern the clinical electrocardiographer most. The P-R interval begins with the P-wave and extends to the first deflection of the QRS complex, representing the time excitation requires to depolarize the atria, the A-V node, the bundle of His, and the first portion of the left bundle branch to the musculature of the interventricular septum; empirically, most healthy hearts show P-R intervals of 0.10 to 0.12 second in childhood, 0.12 to 0.16 second in adolescents, and 0.14 to 0.22 second in the adult. (11) The Q-T interval begins with the first deflection of the QRS complex and extends to the last observed deflection of the T-wave; healthy hearts show an individual Q-T interval variation with the heart rate, but empirically the Q-T interval exceeds 0.38 second only at rates less than 60 beats per minute in the normal heart. (11)

Grant (1957) states that "the commonest A-V arrhythmia is prolongation of the P-R interval." (11) Both organic cardiac pathology and reversible, extracardiac metabolic and neurogenic factors may cause this first degree heart block. Of the common organic cardiac causes, toxic myocarditis, and especially the myocarditis of rheumatic fever, is the most frequent cause of an increased P-R interval in young people; however, the degree of lengthening of the P-R interval does not correlate with the severity of the myocarditis in rheumatic fever. (11) In the older age groups, arteriosclerotic heart disease compromising the circulation to the septal musculature in the area of the conduction tissue frequently impairs A-V

conduction and prolongs the P-R interval. (11, 26) Myocardial infarction adjacent to the area of the A-V conduction tissue also may cause a prolonged P-R interval, or may predispose to the Wenckebach period, a progressive increase in the P-R interval followed by a dropped beat and repetition of the cycle which also is seen occasionally with rheumatic myocarditis. (11)

In view of the known pathologic implications of retarded A-V conduction, the extracardiac metabolic and neurogenic factors that increase the P-R interval may suggest even more strongly organic cardiac disease where none exists, because they occur most frequently in the chronically ill, the debilitated, and those with other pre-existing cardiac disease. (1, 11, 26, 27)

Experimentally, Hall and others (1955) produced first degree heart block in rabbits by administering large amounts of desoxycorticosterone acetate, a mineralocorticoid, both with and without saline, and found that the block was reversed by cortisone, a glucocorticoid, via an obscure mechanism. This effect as yet has not been described in human hearts. (12)

However, metabolic electrolyte disturbances in humans frequently do affect A-V conduction. The electrocardiogram of the patient with chronic renal disease, progressive uremia, and potassium retention may show a prolonged P-R interval, associated with characteristic QRS-T effects, that progresses to complete A-V block as the hyperkalemia which eventually produces cardiac asystole and death develops. (26) Conversely, hypokalemia due to diuretic

therapy with potassium-excreting agents also may predispose the heart to A-V dissociation. (11)

Emotional disturbances and other neurogenic factors also may exert a similar, but indirect, depressant effect on the A-V conduction tissue through an increased vagal tone. Weiss (1956) described a prolonged P-R interval in response to emotional stimulation of the parasympathetic autonomic system in otherwise clinically normal adults and children. (31) Benedict and Evans (1952) reported several cases of second-degree heart block with the Wenckebach pnenomenon in otherwise normal young men; anxiety associated with draft examinations, autonomic imbalance, or emotional instability precipitated the blocks and marked sweats, nausea, syncope, and dizziness accompanied them. (1) Clearly, an observed A-V block associated with such distressing symptoms might be interpreted as organic heart disease, erroneously.

Of the pharmacologic agents which may affect A-V conduction, the digitalis derivatives rank by themselves; the progressively prolonged P-R interval, accompanied by a shortened Q-T interval, cupped RS-T segment, and lowered T-wave, usually identifies digitalis clearly on the electrocardiogram. (11) However, overdosage with digitalis also may cause the Wenckebach phenomenon mentioned above; and occasionally the changes seen with digitalis may resemble very closely those due to primary myocardial disease, especially coronary sclerosis or occlusion, causing diagnostic confusion and/or error. (26)

The Wolff-Parkinson-White syndrome, with its diagnostic delta-wave, causes the only pathologically shortened P-R interval that oc-

curs with any frequency (13); thus, exercise (9), eating of food (9), smoking (9), change in the respiratory level (9), and other extracardiac factors associated with an increased heart rate and shortened P-R interval seldom confuse evaluation of the electrocardiogram.

Changes in the duration of electrical ventricular systole, the Q-T interval of the electrocardiogram, may follow either the development of primary myocardial disease or the effects of several extracardiac factors.

Sodi-Pallares (1956) described several pathologic entities that characteristically may prolong the Q-T interval: 1) mechanisms that overload either ventricle and/or increase the intraventricular pressure, such as hypertension, acute cor pulmonale, aortic insufficiency or stenosis, pulmonary stenosis, and A-V block; 2) myocardial anoxia, as in the ischemic stage of myocardial infarction, or myocardial inflammation, as in rheumatic fever, diphtheria, pneumonia, and scarlet fever; 3) the ventricular hypertrophies and bundle branch blocks; 4) pellegra and/or beri-beri heart disease; and 5) heart failure (myocardial decompensation), all causing other characteristic electrocardiographic changes associated with the increased Q-T interval. (26)

Several extracardiac factors that are reversible clinically also may increase the Q-T interval: 1) hypokalemia, classically showing an associated negative RS-T segment shift resembling subendocardial injury; 2) hypocalcemia, with its characteristically prolonged RS-T segment and normal T-wave configuration; 3) hypothy-

roidism with myxedema, also classically associated with a slow rate, low QRS voltage, and low or flat T-waves; 4) quinidine toxicity, with associated characteristic RS-T changes suggesting subendocardial injury; and 5) barbiturate toxicity. (5, 11, 12, 26)

Clearly, evaluation of electrocardiographic interval changes caused by the reversible metabolic and toxic factors above, without adequate clinical information about the nutritional, electrolyte, and acid-base balances of the patient, may suggest erroneously that organic cardiac pathology has caused the changes when no intrinsic heart disease exists at all.

## The Pressure for Diagnosis

Several authors have emphasized that the electrocardiographer should comment only on the specific electrophysiological data the tracing shows to avoid overenthusiastic and frequently erroneous correlation of the electrical phenomena with proposed anatomic cardiac pathology that may not exist. (6, 8, 15, 17, 18, 22) Especially tenuous empiricism bridges the interpretive hiatus between the electrical record and its anatomic correlation with several of the diseases affecting myocardial repolarization, as described above. Yet, in spite of the calculated risk, frequently the clinician pressures the interpreter to venture across that gap and reach a clinical or anatomical interpretation of the electrocardiogram that may save time in cardiac "diagnosis" by giving a "push-button" evaluation for the busy physician and thus justifying the cost of the tracing. (18)

The diagnosis of right ventricular hypertrophy while the normal right ventricular predominance of infancy prevails can be one of these calculated risks, because the normal finding here differs from the abnormal finding only in degree. (11) Empirically, an R/S ratio in $V_1$ greater than one after the age of four months, or an initial R deflection in $V_1$ greater than 20 millimeters at any age have been considered valid criteria for the diagnosis; however, merely the wide range of normal variation observed in the amplitude of the precordial deflections, reflecting changes of body build or of electrode placement, should obligate the interpreter to use extreme caution in making this diagnosis by any such empirical criteria. (11, 18)

The diagnosis of biventricular hypertrophy likewise lacks the confidence of many cardiologists, and remains controversial. Lipman (1957) terms biventricular hypertrophy a "twilight" (borderline) interpretation (18), and Grant (1957) questions its validity both because he feels that the free right ventricle always enlarges and thickens somewhat as the septum hypertrophies with the left ventricle, and because he believes that the QRS forces from even marked right ventricular hypertrophy probably could not affect the QRS forces of marked left ventricular hypertrophy significantly, since the very thickest right ventricle never attains even the thickness of the normal left ventricular wall. (11)

The possible hazards and consequences of pressure toward overdiagnosis in differentiating benign, atypical chest pain with innocuous RS-T changes from those due to coronary disease, together with

the ambiguities inherent in the interpretation of incipient ventricular hypertrophy, have been discussed above under The RS-T Segment and The QRS Complex, respectively.

The electrocardiographer may create still another anxious cardiac invalid if he overemphasizes a meaningless arrhythmia which may have no true clinical or pathologic significance, as in the usual patient with paroxysmal atrial tachycardia recurring at long intervals. (18)

Johnson (1957) concedes that the "physician. . . who expects . . . the electrocardiograph. . . to write out the correct diagnosis . . . in all patients. . . may become a danger to his community . . . (through) patients. . . wrongly condemned to the life of cardiac cripples. . .", and points out ". . . how a basically accurate and reliable technic may go astray." (15) With complete accord, Lipman (1956) deplores misuse of the electrocardiogram for etiologic diagnosis, for prognosis, or to "check the cardiac status" of the patient hastily prior to surgery, emphasizing with Fischbach (1956) that no direct correlation of any sort exists between the activation currents of electrical systole and the contractile strength of muscular systole. (8, 18)

The lesson is clear. . . "The more one learns about the fundamentals of electrocardiography the more conservative he becomes in the interpretation of tracings. . ." (Frank N. Wilson) (18)

## SUMMARY AND CONCLUSIONS

An abnormal electrocardiogram is not a disease. The electrocardiograph records only the electrophysiologic manifestations of cardiac action, and can never measure directly the structural anatomy of the heart or the dynamics of cardiac contraction.

The clinical reliability of the electrocardiogram varies directly with the degree of correlation the interpreter may achieve between the electrical behavior of the heart and its functional parameters; this correlation will be less than perfect because it must rely on variable human techniques and a basic understanding of cardiac electrophysiology that also is less than perfect.

In its contribution to the total evaluation of the heart, the final interpretation of the electrocardiogram may err to a degree which varies directly with three factors that control both its final form and clinical significance:

1. Technology: a precisely accurate record requires a finely sensitive machine.

   The manufacturers of modern clinical electrocardiographs have minimized mechanical error beyond the limits of clinical significance, when the machines function properly; however, the manufacturers can not control errors in the finished tracing due to wear and poor care of the machines.

2. Technique: uniformity of performance is the essence of

every ultimately reliable interpretive criterion; yet the most precise machine can perform only as consistently as the hand that guides it.

Lack of uniformity, and technically unsatisfactory records, result from: (a) improper preparation of the patient; (b) improper preparation of the machine; (c) improper placing of the electrodes; (d) improper editing, mounting, or labeling of the finished electrocardiogram; (e) improper lead length; (f) an inadequate number of tracings; and (g) an inadequate clinical history accompanying the tracing.

3. Human Decision. The correlation of any cardiac disease with its manifest electrical behavior relies absolutely on the specific knowledge available and the clinical insight applied; the human element in both is fallible.

Because the cardiac activation wave passes through the normal heart quite rapidly and follows the anatomically defined conduction tissue rather closely, deviations from established criteria for normality of the QRS complex usually do represent alterations of the depolarization wave by organic cardiac lesions; however, confusing exceptions may follow changes in cardiac orientation within the thorax.

In contrast, many deviations from established criteria for normality of the RS-T phenomena may confuse

the electrocardiographer, because numerous neurogenic, anatomic, biochemical, and pharmacologic factors that influence cardiac position and/or physiology may alter the relatively slow, diffuse metabolic repair of myocardial membrane potentials (repolarization) that the electrocardiograph records during the RS-T interval.

The electrocardiographer also may overinterpret tracings in response to pressure from busy clinicians who ask for evaluations of the patency of the coronary arteries, the status of the cardiac valves, or the thickness of the ventricular myocardium. Inevitably, misdiagnosis of heart disease, with all its consequences, must result from this practice.

Until medical science can describe and understand completely the electrophysiologic behavior of the human heart, clinically normal hearts occasionally will behave beyond the best known criteria of electrical normality, and will appear pathologic. That these hearts can mislead even the most competent electrocardiographers, and thereby suggest misdiagnoses to the clinician, is clear.

Thus, the physician may avoid electrocardiographic diagnosis of heart disease where none exists anatomically only by 1) acquiring a sound knowledge of the electrical phenomena of the heart and their relation to the electrocardiogram, 2) deliberately avoiding all pressure to interpret tracings more specifically than the electrical

information clearly warrants, and 3) considering the clinical signs and symptoms of the patient in the interpretation of every electrocardiogram.

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