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Electrocardiographic diagnosis of myocardial infarction and correlation with the pathological findings

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ELECTROCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL INFARCTION AND CORRELATION WITH THE PATHOLOGICAL FINDINGS

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TABLE OF CONTENTS

I. INTRODUCTION

In the diagnosis of myocardial infarction, there are three methods of diagnosis: clinical findings, Electrocardiogram, and pathological findings at post mortem. This paper will deal with the accuracy of the Electrocardiogram in making a diagnosis of myocardial infarction. I will attempt to correlate the Electrocardiographic findings and diagnosis of myocardial infarction with the pathological findings at post mortem.

This correlation is being done to prove or disprove the accuracy of the Electrocardiogram in diagnosis of myocardial infarction. Without establishing the degree of accuracy of Electrocardiogram findings regarding myocardial infarctions, we cannot utilize the full value of the Electrocardiography in Medicine.

The object of this paper is to present a series of cases diagnosed as myocardial infarction by Electrocardiographic and clinical findings, using the criteria which will be outlined later in this paper. These cases will be correlated with the pathological findings in the heart at post mortem.

This paper was accomplished at the University

of Nebraska College of Medicine, and at Bishop Clarkson Memorial Hospital, Omaha, Nebraska, during the School year of 1956-57.

II. REVIEW OF IMPORTANT WORK

In Bayley's[1] article in 1944, he ably presented the knowledge up to that time regarding the production of electrical impulses from the normal and infarcted heart.

In his section on Local Muscle destruction (Infarction) he divided it into two sections: The first that of diffuse damage of fibrosis, which causes the diminishing of the amplitude of the accession deflections without extensively changing their form; the second being localized, severely damaged areas. It is the latter which is being considered in this paper.

A localized, severely damaged area is defined as a new boundary which circumscribes a new and abnormal area, which in turn accounts for a new and abnormal component of QRS. He described the new area which is formed by the infarct as a new shell of electrical forces. "The force arises however in consequence of the now unopposed forces in the diametrically opposite region of the involved shell. As a matter of fact, the new and abnormal force is itself a resultant of the newly unopposed forces of the involved shell." The direction of the new force is stated as being along a line from the center of the

dead region toward the center of the involved ventricles.

In discussing the regions of the heart involved in myocardial infarction he divides them into two main regions, and several lesser divisions. Region Number 1 is supplied by the right coronary artery, and involves the left ventricle adjacent to the diaphragm and neighboring portion of the basal region of the I.V. septum. It is called a "Posterior" infarct. He states that the "infarction involves as a rule at least the subendocardial 1/2 of the wall of the left ventricle. If transmural, the extent as observed from the epicardial surface is less than when observed from the endocardial surface." The infarct vector is usually upward, forward and to the left. Bayley, using six sextants using leads I, II, and III (figure 1) describes the vector as lying in sextant 1 or 2. This distorts the initial QRS loop stroke into the 1st Segment usually. It writes an R1 and Q2 and 3 (figure 2). If vector is in position of figure 3, you will get R1, R2 and Q3. Bayley states, "The magnitude of the abnormal force depends on the circumferential limits of the dead region, and therefore these limits are related proportionally to the amplitude of the abnormal Q2, and Q3 deflections. The duration of the force is primarily proportional to the extent of the

infarct in the direction of the epicardial surface of the subdiaphragmatic wall, and the right ventricular endocardial surface of the septum. Because a majority of posterior infarcts extend halfway or more through the wall, a significant Q3 is taken as 0.04 second or more in duration." Transmural infarcts of posterior type may produce a single downward movement for QRS, or a so called QS deflection, with R1 and Q2 defarctions often also present.

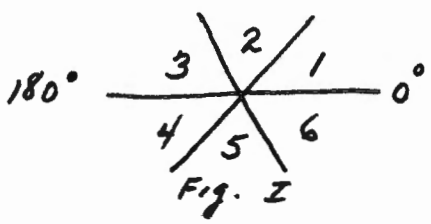


Fig. I

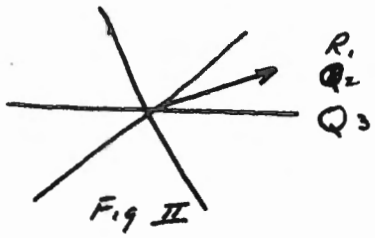


Fig II

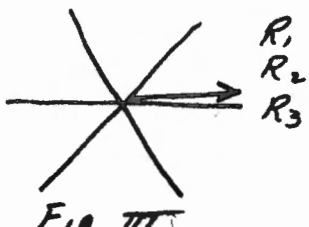


Fig III

Region number 2 considered is ordinarily irrigated by the anterior descending branch of the left coronary artery, and is located in the antero-lateral wall of the left ventricle. This is called an antero-lateral infarction. The positive surface is defined as backward, downward, and somewhat to the right, distorting the initial segment of the QRS into the 4th or adjacent half of the 5th sextant. (figure 4) This is characterized by initial Q1, R2, R3 deflections.

An essentially apical infarct of the region

specified may cause the initial segment of the loop to sweep into that 4th sextant adjacent to the 3rd (figure 5) with a Q1, Q2, R3.

If the infaret is somewhat basal, the initial loop sweeps into the 5th sextant adjacent to the 4th (figure 6).

If in addition to a posterior infarct, there is assumed to be an antero-lateral infarct of similar dimensions, in diametrically opposite position, we will get 2 abnormal forces in equal magnitude and inverse direction. i.e., their sum is zero. It is common to see QRS changes of one infarct annulled by occurrence of another. A staircase Q3 is occasionally produced.

A less common area is strictly anterior region of the ventricular wall. It is composed of the antero-apical position of the I. V. septum and the neighboring portion of the free wall of the left ventricle. This area is irrigated by subdivisions of the anterior descending branch of the left coronary artery. The force is directed posteriorly from the center of the left ventricle. It is perpendicular to the frontal plane, thus there is usually no changes in the extremity leads.

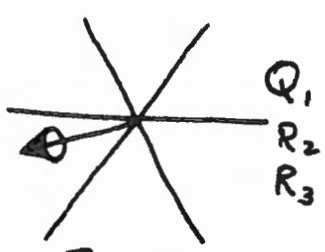


Fig. IV

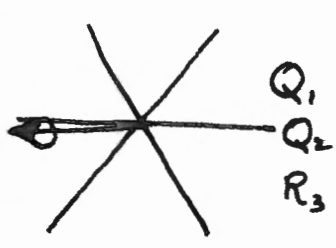


Fig. V

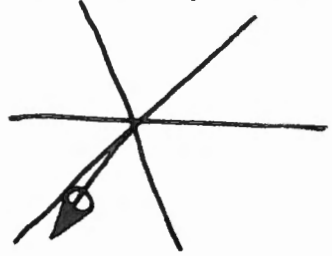


Fig. VI

Strictly posterior infarcts are stated as rare, the region being near the ventricular base, and irrigated by subdivisions of the circumflex branch of the left coronary artery. The 0.04 vector is pointed anteriorly. No changes are observed on the Limb leads. One may distinguish between the strictly anterior or posterior infarcts by the precordial leads.

If the entire apex of the left ventricle is infarcted, and this does occasionally occur, it tends to nullify effects of the mean spatial QRS, thus the QRS loop and deflections are very small.

Bayley states that in general there is failure to infarct the wall of the right ventricle, and gives the following reasons: Thinness of the ventricular wall and the presence of many thebesian veins. It is his opinion that if there is decreased intra arterial pressure with coronary failure, there is back flow via the veins, thus combating the anoxid effect of decreased coronary flow.

It was stated that in right bundle branch block, with a QRS prolongation, there is usually a deep and wide S in lead I. The left ventricle undergoes accession first, and the order is normal. The left ventricle thus writes the initial accession. Thus a posterior wall infarct will give a Q2 and Q3. The initial

QRS goes into the 1st and 2nd sextant.

In left bundle branch block, the initial QRS deflections are of little help. The onset of the QRS is of the R1, R2 and R3 type. If a small Q1 is present, it may be a complete left bundle branch block. The presence of Q1, R2 and R3 may be with transmural infarct of septum and left bundle branch block.

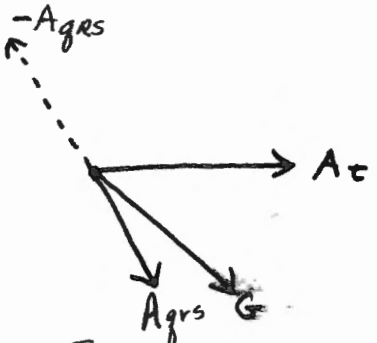


Fig. VII

In Bayley's[1] article in section IV, he discussed the Regression Process and the Mean electrical axis. There are certain definitions and statements to be made prior to the summary of his discussion.

T is defined as the regression process.

Aqrs is defined as the mean spation QRS vector.

At is the mean spatial T vector (or regression vector).

The sum of the above is equal to a new vector - Gz or Aqrst (Aqrs plus At = G)

Thus derived from the above equations At = -Aqrs + G (See figure 7).

Following the derivations above, thus changes in T caused by QRS changes, are called secondary (2°) T wave changes, and changes in T caused by alterations of the Gradient G are called primary (1°) T wave changes.

If Aqrs moves about a pivot point with a constant G, then At moves correspondingly, thus a secondary T wave change.

If G is made to move around pivot point in an arbitrary path, with a constant Aqrs, then At moves in a similar path, thus the primary T wave changes.

Bayley has shown that within relatively normal limits G = H where H equals the Base-Apex axis of the ventricles.

In two examples, A and B as follows, figures 8 and 9 respectively, secondary T wave changes will be shown. In A which is arbitrarily stated as a verticle heart, G is in the 5th sextant adjacent to the 6th. In B, which is picked as a horizontal heart, the G is located parallel to the positive arm of Lead I.

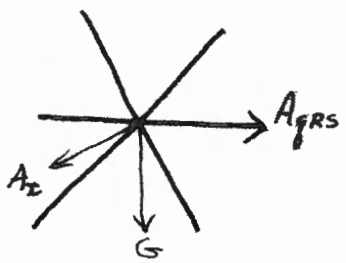


Fig. VIII

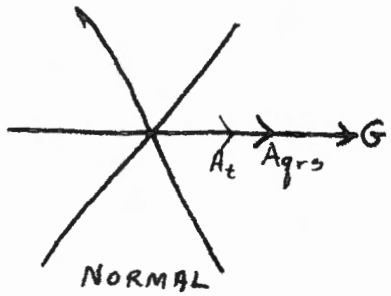


Fig IX

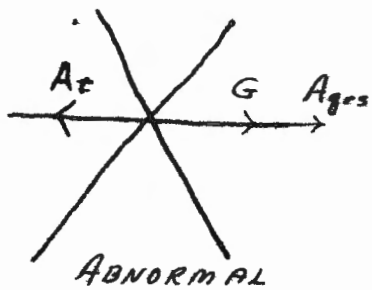


Fig. X

In figure 8 we have a vertical heart, and assume the Aqrs is on the positive limb of lead I, and At is on the positive limb of lead III. Bayley states that this is abnormal because the angle between Aqrs and G is greater than 60°. He claims that the maximal angle possible to still be normal is 30°. You will also have an inverted T1. T2 will be low or diphasic. Thus this is a secondary T wave change.

In B (figure 9) we have a horizontal heart, G and Aqrs is parallel to the positive limb of lead I. In this case, the entire situation is within normal limits. T1 is assumed to be positive unless Aqrs exceeds G in magnitude deflection. Since G was assumed to be normal, then for Aqrs to be abnormal with an inverted T1, A qrs would have to show an abnormal growth without rotation.

A growth of Aqrs may result from ventricular hypertrophy (figure 10). Therefore the T wave changes in both cases are secondary in kind and add nothing further in the diagnosis of ventricular hypertrophy.

If in Example A (figure 11) using the vertical heart with the Aqrs and G both changed as shown, but with the H still in the vertical position, and assuming Aqrs and G lie on positive arm of lead I, G is greater than Aqrs by 1/3 T1 and T2 are positive. T3 is inverted. In this case, T waves appear normal, but are actually abnormal, because:

1. Aqrs show same rotation abnormality as previous, i.e., ventricular (left) hypertrophy.

2. The apparently normal T waves are abnormal because of the presence of secondary changes which have been annulled by the primary changes. The latter is represented by a growth and positive rotation of G with respect to H.

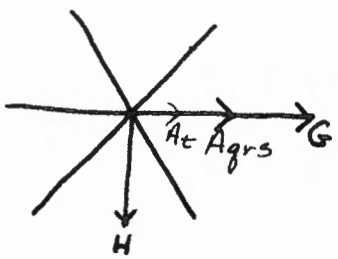


Fig. XI

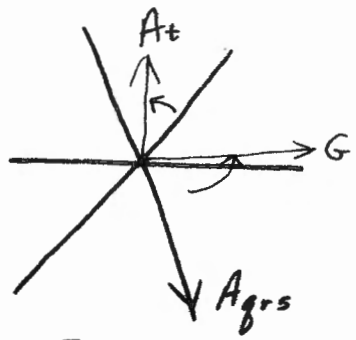
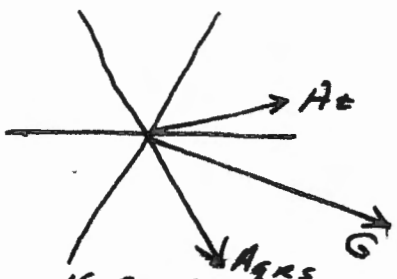


Fig. XII



~~Fig XIII~~

Bayley states that when the area under T deflection is small, the deflection is either low or diphasic. He also feels that we should avoid usage of the term Axis deviation since it is confusing when using the

former type of reading of Electrocardiograms.

It is his opinion that the axes Aqrs, At, and G are of equally great importance, and must be described in both direction and magnitude, both with respect to triaxial reference system and with respect to Base Apex axis H.

Some definition of terms used by Bayley are:

diversion - change away from normal in magnitude and direction;

reversion - change toward normal in magnitude and direction.

He feels that we must state the specific axis with respect to which the diversion or reversion has taken place. "Polar angles" are positive or negative, depending upon whether the direction of measurement appears counterclockwise or clockwise respectively. "Rotation" of a mean axis implies a change of direction only. (The direction is positive or negative depending whether its motion is counterclockwise or clockwise.)

SECTION V ON PRIMARY T WAVE CHANGES

All primary T wave changes are caused by a local alteration in the normal lack and uniformity of duration of the excited state in the epicardial and subendocardial muscle layers of the ventricles.

LOCAL VENTRICULAR ISCHEMIA is either acute or chronic. Either may be present without pain, however pain is usually present with acute ischemia. Chronic ischemia usually has history of paroxysmal dyspnea. The differences between Acute and Chronic ischemia in Electrocardiogram readings are few, and present in the rate of appearance, duration, and rate of disappearance of T wave changes.

	Appearance	Duration	Disappearance
ACUTE	Minutes to Days	Same	Comparable rate
CHRONIC	Weeks to Months	Months-Years	Often interrupted by death

In both varieties, associated abnormalities are hypertensive, syphillitic and arteriosclerotic. In Acute ischemia, it is most commonly found in the arteriosclerotic type of heart disease, with spasm often found as the etiology. In sub-Acute ischemia, it may be caused by a transitory increase in blood pressure as in Acute Nephritis.

If the ischemic area is supplied by the Right Coronary artery (figure 12,) the G has positive rotation to sextant 1 or neighboring part of 2. We must assume Aqrs remain normal in the 6th sextant.

Since Aqrs is constant, G only moves and takes At with it, thus this is a primary T Wave change present. T1 is flat or small positive and T2 and T3 are negative. With development of local ischemia, the gradient undergoes a diversion which tends to bring G into that of a line drawn from the center of the local ischemia to the center of the involved ventricle.

In ischemia of the left coronary artery areas, (figure 13) G is rotated negatively, into the 5th or 6th sextant. Aqrs is constant and normal in the 6th sextant. At is rotated along with G, giving a large, negative T1, equally large positive T3, and a zero or diphasic T2.

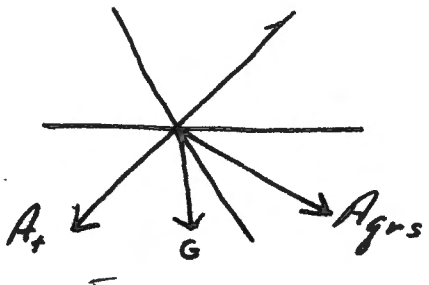


Fig. XIII

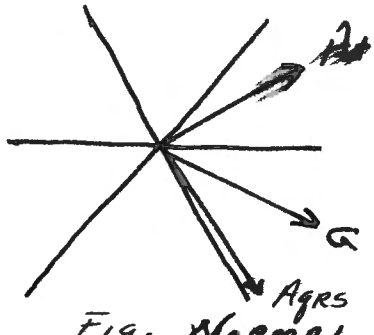


Fig. NORMAL

When the areas under T waves are large (negative or positive), the effective events or regression may begin before those of accession are complete, and the RS-T junction is displaced from the base, but in the direction of the regression effect. RS-T displacements of this kind are not to be confused with injury.

Regarding the T waves, there are no important differences between those changes ascribed to uncomplicated local ischemia and those ascribed to perifocal infarct.

The acute variety of the local ischemia may herald the oncoming infarct, and the primary T Wave changes disappear temporarily as a result of development of injury effects. Then the same T wave changes appear subtotally as evidence of perifocal ischemia of infarct.

The injury effects the RS-T displacements, which disappear with recurrence of ischemia effects.

On unipolar leads, the delay of regression at the epicardial surface adjacent to exploring electrode causes sharp T inversion. Anterolateral ischemia gets V4, 5, 6 inversion.

Strict apical ischemia shortens the gradient, and may reverse it to sextant 3. The former may diminish positive T's, the latter giving negative T1, 2, 3.

If Aqrs and G are in 6th sextant, At appears in 3rd sextant as G becomes less than Aqrs. Diffuse ischemia localized to the base of the heart may increase gradient G in normal direction and increase positive T's in V1 and 2.

SECTION VI ON THE INJURY EFFECT AND ITS RELATIONSHIP TO MYOCARDIAL INFARCTION.

In man the RS-T junction may be elevated 1-2 mm from base line (as seen in Precordials) indicating a regression effect already starting at the time the accession ends. Usually regression effect in the limb leads are not seen until a short interval of time has elapsed between the end of accession and start of recession - called the RS-T segment.

Normally, regression isn't strong enough to be recorded immediately, thus the flat segment of no recording.

If regression is strong enough to record immediately after accession, the RS-T segment is displaced either up or down. In this case the RS-T is absent and the onset of T Wave appears to take off from the final ascending or descending limb of QRS.

If QRS duration is prolonged, the RS-T is absent and the RS-T junction is displaced.

Thus early regression and late accession equals displacement of the RS-T segment.

These are all displacements of permanent type in contrast to displacement of temporary type as in injury of muscle. The permanent type is associated with QRS complexes which have big areas under the curve, and that

the direction of displacements is like in sign to that of the area under T - the adjacent displacements.

THE TERM INJURED MUSCLE implies an altered physiologic rather than a structural change. It resembles a zone of local ischemia except for the degree of insufficiency and the electrical effect produced. The injury zone is regarded as the 2nd and more severe type of ischemia.

A zone of local ischemia is characterized by local prolongation in the effective duration of excited state, or by local, tardy onset of regression. The next grade appears to be inability to complete locally the delayed regression process.

ON THE ECG EVOLUTION OF MYOCARDIAL INFARCT

I. EARLY IMPAIRED CORONARY FLOW:
   1. The region of decreased flow may pass into 1st grade ischemia.
   2. The only change is in striking primary T wave changes.

II. After variable period of time, more central regions of 1st grade ischemia passes into second grade ischemia and to the injured state.
   1. If zone extends to epicardium, RS-T signs of

injury appear.

2. Potentially transient QRS changes appear.

3. Early primary T wave changes disappear.

If fortunate, the injury zone reverts to ischemic zone with return to primary T wave changes. Usually clinical signs of heart damage (elevated WBC, ESR and Temperature) are absent.

III. If unfortunate, infarct occurs.

1. Extension of injured area extends till only a small perifocal area of ischemia remains.

   a. Striking RS-T displacements

   b. More definite QRS changes

   c. Initial primary T wave changes absent except in precordial leads adjacent to area involved.

Limited endocardial infarction occurs. At some critical stage of extension of injury, death of central portion occurs. Here the clinical picture is complete. The dead zone may progress or be stationary. In the former, the QRS changes accordingly.

IV. HEALING - early, 2-4 weeks after infarction.

1. Injured zone diminishes with reversal to ischemic state. RS-T displacements disappear when injury area no longer is at the epicardial surface.

2. Primary T wave changes of ischemia at epicardium appears.

V. HEALING - months later

1. Ischemia zone diminished with return to normal. Primary T wave changes disappear.
2. Permanent QRS changes associated with dead zone remain.

In summary of myocardial infarction and its evolution, there are 5 stages in the evolution and they are as follows:

1. Initial diversion of gradient G, which is a measurement of primary T wave changes.
2. Development of the axis of injury
   a. concurrent revision of G
   b. concurrent diversion of Aqrs, which measure respectively
   c. the appearance of RS-T junction displacements, the subtotal disappearance of primary T Wave changes, and appearance

of QRS changes

3. The decay of the axis of injury, second concurrent diversion of G, and concurrent subtotal revision of Aqrs, i.e., RS-T disappearances, primary T wave changes appearances, QRS changes disappear.
4. The final reversion of G, and final disappear-- ance of primary T wave changes.
5. Permanent persistence of a diversion of Aqrs and At which measure permanent QRS changes and the permanent secondary T Wave changes.

Stage 1 is traversed quickly. Infarcts usually show in stage 2 or 3. Stage 4 is usually well prolonged, and referred to as healing.

In a single curve, stages 2 and 3 resemble each other closely, and clinical criteria must be used to ascertain presence of infarct. Stage 5 is known as the healed infarct.

Grant and Estes[2] felt that there are three main Electrocardiogram abnormalities in Acute Myocardial infarctions. 1) "The instantaneous vectors for the first 0.03-0.04 second of the QRS loop point away from the region of the heart where the infarction lies. The cause of this change in direction of the initial QRS vectors is the 'Dead Zone' effect." (This effect is

described by Bayley and by Grant and are essentially the same) "For descriptive convenience, the resultant of the instantaneous vectors during this initial part of the QRS loop is called the 0.04 vector". 2) "The mean spatial T vector is changed in direction and points away from the infarcted region. It is believed this is caused by the ischemia surrounding the infarct (the dead zone vector and ischemia T vector point in the same direction, also have the same electrical sign, but the two vectors are not necessarily parallel)". 3) "An ST vector develops as manifested by a shift of the ST segment in the limb and precordial leads. The vector tends to point toward the infarcted region of the heart."

Grant and Estes[2] discussed the electrical significance of the QRS and T vectors as follows. "The QRS complex is written when the membrane of the cells loses its relative cation impermeability. The ions are free to move across the membrane and the alignment of cations and anions on the opposite sides of the membrane are lost. The electrical force behind this movement of charges at each membrane in the heart writes the QRS complex. "The T wave is manifested when myocardial cell metabolism causes the membranes to become relatively impermeable to cations, and the cations and anions are forced to align themselves on opposite sides

of the membrane. It is the movement of the ions into this orientation on myocardial membranes which writes the T Wave." The T process is called repolarization or regression of the activated state. The QRS process is called depolarization or accession of the activated state. The T process is more persistent, thus a longer duration of T wave than of QRS wave.

The mechanism of ST abnormalities are "believed due to 'leaking' of charges across the membranes of injured cells, such is called the 'Injury Current'. The electrical force or pressure is called the Injury Force."

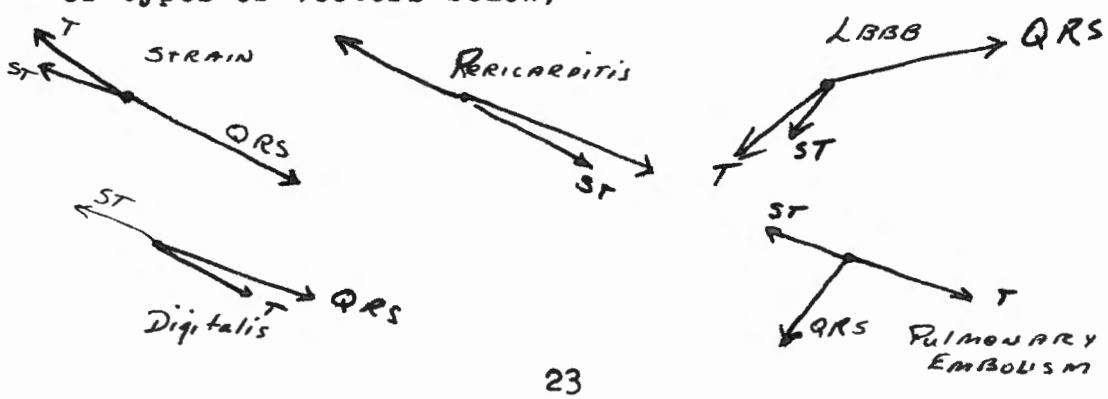
"The ST segment deviation seen in Electrocardiogram is due not to the appearance of the injury force, during the ST interval, but to the absence of the Injury Force during this interval. (Between the end of QRS and beginning of T, there is no repolarization or no charges on cell to cause the leak, which appears when repolarization starts.) The vector obtained by measuring deviations of the ST segment in the various leads is then exactly opposite to the actual injury forces. Since it is easier to measure deviation of the baseline in ST segments, than in the T-QRS segment (using P-R interval for baseline), the vector calculated from ST segments is used in clinical Electrocardiogram interpretation to represent the injury force vector."

Other causes of St segment shifts are:

1. Digitalis - ST vector is opposite the mean QRS
2. Ventricular strain - here the forces are T forces, not injury forces. The ST and T are the same.
3. Uncomplicated Bundle Branch Block - ST is parallel to the T, and 180° from the QRS vector.
4. Tachycardia - the QT is shortened, the T forces appear in the ST interval, with deviation of ST.
5. Occasionally in normal persons, early and large T, with displacement of ST.

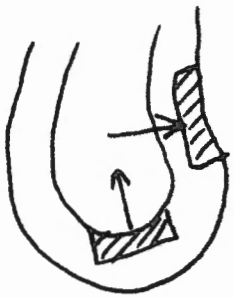
"In all 5, the ST deviation is due to normal repolarization forces generated during the ST segment, and not due to injury forces."

"The injury ST vector indicates damage is greater than ischemia." Other causes other than infarction are pericarditis, pulmonary embolism, myocardial abscess, angina pectoris, etc." (See Diagram of types of vectors below)



"The injury ST effect is usually transitory. T wave changes due to ischemia may persist for months. Other causes of ST changes are rarely so transient."

"Acute myocardial infarction principally involves the epicardial layers of the heart in the vast majority of cases. Under the circumstances, the ST vector points toward the infarcted region of the heart. Thus it is opposite in direction to the dead zone 0.04 vector and ischemic T vector."



In subendocardial injury, the forces behind the lead are opposite in direction to epicardial injuries, i.e., ST points away from in the injured area. Examples are angina pectoris, acute coronary insufficiency, momentary anxiety or fear. "The disturbance is usually diffuse in the left ventricle, therefore the ST vector points away from the apex of the heart. The QRS and T vectors are usually unaltered in such cases." Occasionally there is endocardial or epicardial ischemia with T wave abnormalities. If the Electrocardiogram evidence of subendocardial injury persists for more than 1-2 days, suspect subendocardial infarct.

"Electrocardiogram changes of subendocardial

injury are seen in exercise tolerance and anoxia tests used for Electrocardiogram confirmation of the clinical diagnosis of angina pectoris. A test is positive when the Electrocardiogram shows ST segment changes of left subendocardial injury during the experiment." (One may find similar changes in tachycardia, and with digitalis.)

In Grant and Estes[2] under the unequivocal diagnosis of acute myocardial infarction, there are four criteria. All four must be present to have a diagnosis.

1. The three Electrocardiogram abnormalities, all of which must be present
   a. 0.04 vector abnormality
   b. Injury ST vector
   c. Ischemia T vector
2. The three vector abnormalities must be shown to have directions characteristic of infarction. The 0.04 vector and T (mean spatial) should be approximately 180° from the ST mean spatial vector. (In normal persons, there is area where Q wave and negative T can be found - on the back in younger persons, and on the anterior right chest in older persons. This can be distinguished by the vectors.)

3. The three vectors must have directions which identify an anatomically reasonable region of the heart for the location of the infarction.
4. Finally, the Electrocardiogram changes must fit the clinical picture.

Regarding the age of an infarct as seen in Electrocardiogram, the vector abnormality varies directly with the age of the infarct.

In a few hours after onset, the QRS and ST are usually fully developed. The characteristic T waves may not be evident. A subendocardial ischemic T vector may be present, but is usually gone in 12 hours. Then the T vector soon is classic for an epicardial ischemia pattern.

In the course of healing the ST is the first to vanish. If a small infarct is present, it is lost in 2-3 days. Usually it will last 2-3 weeks. At the end of the latter period, the ST-T portion shows what is known as coving, a convex appearance of the descending limb of the inverted T wave. As months pass, the ischemic T wave becomes smaller and smaller in the ischemic direction, and in time it may be normal in direction. Usually some degree of ischemic effects persist (Wide RS-T angle) and will become permanent.

The dead zone 0.04 vector becomes smaller in magnitude and the loop deformity shorter in duration. Occasionally the deformity will disappear entirely.

In the case of an old infarct, plus an acute infarct in another area, the 0.04 vector is midway between the two. The ST-T is governed by the recent infarct. If several infarcts are present, the ST-T is dominated by the most recent infarct, with a reduction in the total generating myocardium, with a very small QRS. With multiple infarcts, the conductive pathways may be interrupted with tortuous loop and a prolongation in duration.

In their discussion regarding the "healed infarct", Grant and Estes state that the injury vector has disappeared, the T vector may have returned to normal, and that the 0.04 vector is of the utmost importance. In the normal patient, the 0.04 vector is usually tangential to the septum and is relatively parallel to the mean spatial QRS, i.e., the normal loop is narrow and elongate. In an infarct, the 0.04 vector is pointing away from one or another region of the left ventricle. It is usually directed 90° or more from the direction of the mean spatial QRS vector, producing a deformity in the initial part of the loop.

"The 0.04 deformity in infarctions explains why Q waves have been such reliable indicators of myocardial

infarction in the past." Normally there is no Q in the Limb leads. This is because usually the mean vector is within the area where no Q will be registered on the Electrocardiogram.

Grant and Estes[2] used the following criteria for Electrocardiogram diagnosis of anterior and posterior infarcts. An infarct is stated as anterior if:

1. There is a Q wave 0.03 or 0.04 second duration in Lead I. (Actually points away from the lateral or apical wall) (Left coronary artery supplies the anterior and lateral heart.)
2. Since the 0.04 vector in this location points posteriorly, the QRS complexes at the Precordial positions V1-V4 almost always have initial negative deflections (or Q waves).

An Infarct is posterior if:

1. A Q wave of 0.03 to 0.04 second duration is present in Lead III.
2. The 0.04 vector points away from the diaphragmatic and posterior surface (supplied by right coronary artery.)
3. The 0.04 vector points left and anterior. "Therefore in these cases, there is a Q3

and the initial R waves in precordial leads are taller than normal."

An aVL deformity (Q wave) is often helpful in the diagnosis of an anterior infarct, and a Q wave in aVF is often helpful in the diagnosis of a posterior infarct.

It is Grant and Estes' opinion that it is not necessary to memorize patterns when it is realized that the diagnostic change in QRS electrical field produced by infarction is the deformity of the initial part of the QRS loop.

Often non conventional leads are used in the diagnosis of infarction, because it is suspected, and not proven, and also because of the dependence upon presence of Q waves for the diagnosis. Using the deformity of the initial QRS loop to diagnose a dead zone, the diagnosis is made without the presence of a Q wave, and the area on the chest can be predicted where a Q wave may be obtained.

It is possible for Q waves to be present in normal persons. The difference between normal and the old infarct is in the difference in the location of the Q area in the two subjects. Normally the location of the Q area depends upon the age group, and electrical position of the heart. In the young subject,

with a vertical heart, the 0.04 points forward, and the Q area is on the back. With increasing age, the mean spatial QRS rotates left and posteriorly, accordingly the 0.04 tends to follow it, thus the Q area moves to the right, around the right side of the back, around the side and to the right chest. (Q area is defined as the negative deflection of the 0.04 second, and lies in an area away from the 0.04 vector.) Because the 0.04 vector and QRS is more anterior, they have higher R waves in V1 and V2 in younger people. In older subjects with the vector to the left and posteriorly, V1 and V2 may have been small R waves. In marked Left Ventricular hypertrophy the QRS is even more posterior, with occasional Q in V1, 2 and 3.

In myocardial infarction, the 0.04 is abnormal in direction. The Q area faces the left ventricle, and lies to the opposite side than it would normally. The polarity and electrode positions for the conventional leads are such that whenever a Q wave of 0.03-0.04 second duration occurs in one or another of these leads, the Q area must lie somewhere near, or on the left side of the chest. Therefore myocardial infarction must always be considered whenever Q waves

of this duration are encountered in any of the conventional leads, and this will include the vast majority of cases of myocardial infarction.

Hearts in unusual electrical positions may have Q areas lying normally on the left side of the chest, and produce Q waves resembling those of myocardial infarction. If in the vertical electrical position, the 0.04 vector is relatively vertical, and the Q area may lie high on left side of the back or mid back, facing the ventricular cavities. V1 may record 0.03-0.04 second duration Q waves not due to infarct. If the heart is horizontal electrically, the Q wave has another meaning. It would (Q area) be facing lateral wall of left ventricle. In an infarct at the AV orifices, there is little 0.04 change in direction, thus difficult to differentiate from normal.

The Q wave must have a duration of at least 0.03 second of the QRS loop before Electrocardiogram Diagnosis of myocardial infarction can be made. Actually if one has the directions of the 0.04 vector and the mean spatial QRS vector, one has all the information needed for clinical interpretation.

With the presence of Q3, one must remember that myocardial infarction isn't the only cause of Q waves

of 0.04 or over. A change in position of the heart with a resultant change in position of the QRS loop, plus horizontal hearts in normal persons gives a Q3. With a Q3 in a horizontal heart, one must always distinguish between infarction and positional change. To do this have the patient take a deep breath, and do Lead III over. If the Q wave is because of position, it will disappear on deep inspiration. In an infarct at the AV orifices, there is little 0.04 change in direction, thus difficult to differentiate from normal. If there is present Q3 and a Q in aVF, the possibility of infarction is greater than if Q3 alone were present. Also if a Q2 is present, the possibility is greater than if Q3 alone, and the diagnosis is almost certain for infarct.

In an uncomplicated Bundle Branch Block, the 0.04 vector is generated from the septum, the wave travelling from the normal to the blocked side in 0.04 second. In certain anatomical conditions, this change in 0.04 vector may be sufficient to cause it to point toward a negative pole in a conventional lead. For example, in left ventricular hypertrophy with tiny R waves in V1, 2, 3, if left bundle branch block occurs, the Q waves may appear in V1, 2, 3. Since the 0.04 vector points away from the anterior surface

of the heart, it may be confused with anterior infarct.

Since excitation of the subendocardium of left ventricle is delayed in left bundle branch block, the dead zone effect of myocardial infarction in the free wall of the left ventricular infarction is not likely to be manifested during the first 0.04 second of the QRS cycle in a subject with left bundle branch block.

With infarct of septum plus left bundle branch block, the septal contribution to the 0.04 vector is eliminated and excitation of the right ventricle dominates initial part of the loop. The 0.04 is right and anterior with a Q in lead I. Thus in left bundle branch block, a Q in lead I is suggestive of infarction of septum.

Grant and Estes in concluding their section on infarction state that vectors are most useful in old or healed infarcts or when tracing is not typical, and warn to be wary of diagnosis of infarction from Electrocardiogram alone.

Grant and Murray[3] in their article on QRS Deformity of Myocardial Infarction, which included 190 cases of myocardial infarction, noted an important and most unexpected finding in their study of the cases. They noted that in over a third of the cases, the terminal vectors of the QRS cycle were altered in

addition to the early vectors.

In the strictly anterior infarct, terminal vector alteration was infrequent, occurring in only eight of 51 cases. The terminal vector was caused to point superiorly and rightward in the frontal plane so that S waves appeared in all three limb leads.

In the anterolateral infarcts nearly half of the cases showed terminal deviation of the QRS. In all cases the vector was pointed superiorly, leftward and posteriorly.

In the diaphragmatic infarction, one third of the cases (total of ninety-nine) showed terminal deviation rightward, inferiorly and slightly posteriorly.

In the majority of these cases, there was no prolongation of the QRS interval, but in a very few, there was prolongation from 0.02 to 0.04 second.

The exact mechanism is not known exactly. First, Bayley and Bedford in 1940 first described a conduction defect called the "peri-infarction block" and suggested that with a subendocardial location of the infarct, the normal spread of excitation from endocardium to the uninfarcted epicardium overlying the infarct was prevented, and excitation could reach this

region only by circuitous spread around the infarct with resultant delay of 0.04 second or more. The terminal vector was found to be approximately exactly opposite to the primary QRS vector, but Grant and Murray noted a difference in that there was not present any great amount of prolongation of QRS, while Bayley first and Bedford noted prolongation beyond .12 second in all their cases.

Because of the lack of prolongation of the QRS, Grant and Murray feel that the area where excitation was delayed was probably normally excited during the first 0.04 second of QRS. They feel that the Antero-lateral infarcts probably tend to involve the anterior division of the left bundle branch, and the Posterior or diaphragmatic infarcts tend to involve the posterior division of the left bundle branch. An infarct in any of the two regions, if large enough, would tend to cause the excitation to spread first through the other division, so that excitation of the myocardium adjacent to the infarct would be delayed by 0.04 second, but with little delay of the total QRS interval.

III. METHODS AND MATERIAL

In selection of material, all cases of myocardial infarction, either acute or chronic, were obtained from the post mortem file in the Electrocardiographic department at the Bishop Clarkson Memorial Hospital, Omaha, Nebraska. These cases were selected during the period between July 1, 1953, and March 1, 1957. These cases were consecutive, and every case had at least one or more Electrocardiographs recorded before death. Only cases with complete post mortem reports, including gross and microscopic examination, plus a complete clinical summary, were selected.

The Electrocardiographs of the cases selected were obtained, the tracings were read and recorded by vector methods, and the vectors drawn in the frontal and oblique horizontal planes. The presence of the infarct, and the position of the infarct were determined by the criteria of Bayley and Grant, plus slight modifications by Angle. The post mortem report was then obtained, and the result of the report was correlated with the diagnosis and position of the infarct as previously recorded. (See Chart II)

Also, certain clinical data was recorded, such as the post mortem file number, age, sex, history of chest pain and related symptoms, pertinent complicating

factors, and the supposed date of the infarct in respect to the dates of the separate recordings. (See Chart I)

IV. RESULTS AND INTERPRETATION

CHART I

CASE NO.	AGE	SEX	HISTORY	COMPLICATIONS	DATE OF INFARCT
1 (18)	71	M	Dyspnea, cough 1 year	Congestive failure one year	Undetermined
2 (19)	59	M	Angina 7 yrs	Hypercholesterolemia 15 yrs duration	Old infarct date unknown
3 (23)	80	M	None	Hypertension, CVA	1 day before ECG
4 (41)	65	M	Chest pain for 7 mos, with increase in frequency and intensity until could only be relieved by Morphine	None	Just prior to ECG Tracing
5 (43)	65	M	Admitted Jan 54, heart attack 5 yrs ago, followed by continual substernal pain which became refractory to rest and medication	None	Just prior to ECG Tracing
6 (60)	50	F	No history or physical. Severe chest pain on admission.	Severe diabetic	Unknown
7 (63)	68	F	Dyspnea 2 yrs. Sudden onset of chest pain day of admission	Emphysema Hydrothorax	Prior to admission by 1-2 days
8 (66)	85	F	Admitted 6-28-55. Dyspnea since morning. Marked shock on admission.	Pulmonary edema Hydrothorax Atelectasia	7-1-55. Just prior to ECG tracing

CHART I (Contd)

CASE NO.	AGE	SEX	HISTORY	COMPLICATIONS	DATE OF INFARCT
9 (67)	81	M	Admitted 6-8-55 for 4th time with chill, dever, temperature and diarrhea. One mo before had episode of dyspnea on 3rd admission.	Arrythmias, aberrant	Unknown
10 (73)	61	M	Admitted 9-8-55 with abdominal pain. A presumed diagnosis of carcinoma of prostate, and was on estrogen therapy	Lt ventricular hypertrophy. Premature auricular and ventricular beats	Unknown
11 (99)	70	M	Admitted 5-12-56. No history or phycial	Rt BBB Paroxysmal Auricular tachycardia	5-12-56
12 (101)	53	F	Admitted 4-22-56 with epigastric fullness, pain in rt scapula. Aortic Graft done on 5-25-56. On 6-4-56 severe chest pain radiating down both arms.	Aneurysm of abdominal aorta	6-4-56
13 (106)	71	M	Admitted 7-2-56 comatose, dyspneic, cyanotic.	Hypertension. Arteriosclerotic disease of heart	7-2-55
14 (114)	66	M	Admitted 9-3-56 with chest pain 3 wks duration	Rt BBB 1:2 AV Block	Just Prior to Admis.
15 (115)	58	M	Admitted 9-15-56. Sudden onset chest pain, radiating to lt shoulder that day. Infarct 4 yrs before. Moderate angina since	Obese	Old Infarct in 1952. New Infarct just prior to admis.

CHART I (Contd)

CASE NO.	AGE	SEX	HISTORY	COMPLICATIONS	DATE OF INFARCT
16 (117)	67	F	Admitted 9-19-56 with chest pain, duration 1½ yr. Severe onset on day of admission Elevated WBC, ESR and Trans-aminase	Left BBB	Just prior to admission
17 (119)	79	F	Admitted 10-2-56 with upper abdominal pain. Nausea and vomiting for 4 days before admission. Coronary occlusion 6 mo prior	None	Just prior to admission
18 (130)	89	M	Admitted 8-13-56 with progres-sive dyspnea, cardiomegaly, hepatomegaly. On 8-21-56 began fibrillating, in acute deep shock; dyspnea and cyanosis developed. Chest pain on 9-6-56. Died 10-8-56.	None	Probably during terminal stage
19 (143)	77	M	Admitted 10-30-56. Infarct 8 yrs prior. Two weeks history of intractable mild failure at time of admission.	Old Rheumatic fever. LBBB	Unknown
20 (154)	95	F	Admitted 1-17-57 with no history or physical. Had dyspnea, cyanosis, and died of convulsion on 1-19-57.	None	Unknown

CHART II

NUMBER	POSITION OF INFARCT	POST MORTEM FINDINGS
1 (8)	Antero-Septal (acute)	Necrosis along lt ventricle and anterior I.V. Septum
2 (19)	Lateral (old)	Fibrosis of anterior wall of lt ventricle extending to apex.
3 (23)	Anterior (acute)	Thrombosis of anterior descending branch of lt coronary artery. Rupture of the anterior descending branch of lt coronary artery.
4 (41)	High anterolateral (acute)	Fibrous area in curve of lt ventricle. Old thrombus in lt circumflex artery. Recent rt coronary thrombus.
5 (43)	Questionable old antero septal infarct. Massive antero lateral acute infarct	Recent thrombus in anterior descending branch of lt coronary and lt circumflex of the lt coronary. Infarct of the anterior and apical portion of the I.V. septum. Infarction of anterior lateral wall of lt ventricle approximately 2 wks old. Marked Fibrosis.
6 (60)	Antero septal (acute) Peri Infarction block.	Occlusion of anterior descending branch of lt coronary. Infarct of the anterior wall of the lt ventricle and anterior portion of I.V. septum.

CHART II (Contd)

NUMBER	POSITION OF INFARCT	POST MORTEM FINDINGS
7 (63)	Questionable hi posterior (acute)	Thrombosis of lt coronary proximal to the bifurcation. Infarct of anterior wall of lt ventricle.
8 (66)	Anterior Inferior (acute)	Thrombus of anterior descending branch of lt coronary. Infarct of anterior wall of lt ventricle and I.V. septum.
9 (67)	No definite diagnosis of either old or new infarct.	Marked fibrosis of anterior wall of lt ventricle and anterior I.V. septum. Recent occlusion of rt coronary and old occlusion of anterior descending branch of lt coronary. Recent and old infarct of the anterior wall of lt ventricle and of I.V. septum.
10 (73)	No diagnosis of either old or new infarct.	Infarct of apex of lt ventricle.
11 (99)	Infarct of antero lateral aspect of lt ventricle (acute)	Recent infarct of anterior lt ventricle and I.V. septum. Occlusion of the descending branch of the lt coronary artery
(12) (101)	Large anterior inferior infarct with peri infarction block	Healed infarct of anterior I.V. xeptum and anterior aspect of lt ventricle

CHART II (Contd)

NUMBER	POSITION OF INFARCT	POST MORTEM FINDINGS
13 (106)	Anterior Superior with peri-infarction block. (acute)	Recent thrombus in the lt coronary artery. Infarct of the anterior wall of lt ventricle and the I.V. septum.
14 (114)	Lateral, questionable anterior	Recent thrombus in anterior descending branch of lt coronary and in rt coronary. Infarct of anterior lt ventricle and I.V. septum. Rupture of infarct in lt Ventricle.
15 (115)	Massive anterior inferior wall infarct (acute)	Infarct in the anterior wall of the lt ventricle and I.V. septum. Occlusion of the anterior descending branch of the lt coronary artery.
16 (117)	Anterior Inferior infarct (Acute)	Occlusion of main rt and lt coronary arteries. Recent infarct of myocardium involving the wall of the lt ventricular apex and I.V. septum.
17 (119)	Anterior apical (acute)	Infarct of anterior lateral and posterior wall of lt ventricle, also of the apex. Occlusion of lt main coronary. Healed infarct of Myocardium involving the I.V. septum and the anterior wall of the lt ventricle.

CHART II (Contd)

NUMBER	POSITION OF INFARCT	POST MORTEM FINDINGS
18 (130)	Anterior	Infarct of anterior infero I.V. septum and apex. Thrombus in anterior descending branch of the lt coronary artery.
19 (143)	Posterior Inferior (acute)	Recent infarct of the posterior wall of the lt ventricle.
20 (154)	Posterior (Acute)	Infarct of posterior wall of lt ventricle. Recent thrombus in rt coronary artery.

Among the twenty cases reviewed, eighteen diagnoses of myocardial infarction were made correctly. These cases showed excellent correlation with the pathological report. Two of the cases reviewed showed infarction at post mortem, but diagnosis of myocardial infarction could not be made using the criteria stated.

In the series of cases, the ages were between 50 and 95 years of age, and the sexes involved were 14 men and 6 women.

Three cases of peri-infarction block were diagnosed from the Electrocardiogram tracings, using as criteria the abnormality of the terminal 0.04 second QRS, plus prolongation of the QRS interval.

V. CONCLUSION

We have attempted to show in a series of 20 cases, selected from the post mortem files by their gross and microscopic diagnosis of myocardial infarction, that there is correlation between the diagnosis of Acute and Chronic myocardial infarction using the clinical findings and Electrocardiogram tracings. It is to be noted however, that there was no attempt to first make the diagnosis of myocardial infarction from the clinical records and Electrocardiogram recordings, but that the presence of the infarct was first ascertained by reading the post mortem reports. If the former method had been used, there might have been entirely different results, and I would assume possibly less correlation between the clinical and pathological findings.

VI. SUMMARY

The major works of Bayley and Grant were reviewed regarding vector Electrocardiography in the diagnosis of myocardial infarction. Also reviewed was Grant's article on Peri-Infarction block. The criteria proposed by the two authors, plus slight modifications by Angle, was selected. Twenty cases of myocardial infarction were selected from the post mortem files by their pathological diagnoses. The clinical history and Electrocardiograms were reviewed using the criteria as stated. There were 18 diagnoses of myocardial infarction made during this review. Two cases were not diagnosed as myocardial infarction. Three cases of peri-infarction block were diagnosed by their Electrocardiogram findings. A correlation was made between the clinical, Electrocardiogram, and the pathological diagnoses, and interpreted as an excellent correlation using this method of selection of cases and this criteria for the diagnoses of myocardial infarction.

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