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CRITERIA FOR THE ELECTROCARDIOGRAPHIC DIAGNOSIS
OF MYOCARDIAL INFARCTION IN THE PRESENCE OF LEFT
BUNDLE BRANCH BLOCK

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I. Introduction

The electrocardiographic identification of myocardial infarction in the presence of left bundle branch block has been reported in the literature to be difficult. Yet this electrocardiographic diagnosis is of importance inasmuch as left bundle branch block is often a complication of myocardial infarction; it was present in 20 (7.7 per cent) of 375 cases of acute occlusion of the coronary artery reviewed by Master and associates.¹ The scope of this paper will be a review of selected literature in chronological sequence from experimental infarcts produced in dogs to actual case studies attempting to differentiate myocardial infarction in the presence of left bundle branch block. Although the vector approach is more scientific in diagnosing this distinction, the various pattern changes in the mechanical events of the cardiac cycle with respect to left bundle branch block and infarction as presented by the various studies will be compared and correlated. The term "infarction" will be limited to necrosis in any part of the heart without regard to site of the infarct, i.e. apical, anterior, or posterior in comparing it with left bundle branch block.
II. Normal Cardiac Activation

The cardiac impulse arises at the sino-auricular node which is the normal pacemaker of the heart, spreading from here through the auricular musculature, producing the P wave in the electrocardiogram. Following this is an isoelectric segment which represents the slowing down of the impulse thru the auricular-ventricular node with the PR interval being that amount of time it takes for the impulse from the sino-auricular node to spread through the entire auricle in addition to the time it takes to travel through the auricular-ventricular node. Normally this interval is .2 seconds or less. After breaking through the AV node the impulse travels rapidly through the bundle of His, the right and left bundle, the Purkinje's fibers and through the ventricular myocardium from endocardium to epicardium with ventricular activation represented by the QRS complex, normally .1 second or less. The ST segment follows this representing complete depolarization of the entire heart, and because no electric forces are normally apparent during this period, the ST segment is iso-electric. With the beginning of repolarization from epicardium to endocardium the T wave is written and after complete
repolarization electrical forces are again absent and diastole commences with an iso-electric line appearing after the T wave.

Utilizing the precordial leads $V_1$ and $V_6$ from a pattern point of view, in an adult normal ventricular activation commences with depolarization of the interventricular septum and depolarization of the right ventricle both from left to right traveling in the general direction of $V_1$ and therefore producing a positive deflection (r wave) in that lead. These same forces face away from $V_6$ producing a negative deflection (q wave) in this lead called a septal q. Finally left ventricular depolarization is directed to the left in the general direction of $V_6$ and away from $V_1$ therefore producing a strong positive deflection (r wave) in $V_6$ and a deep negative deflection (S wave) in $V_1$. The intrinsicoid deflection in $V_1$ representing the arrival of the depolarization wave in the epicardial surface of the right ventricle occurs normally within .02 seconds after the beginning of the QRS complex. The intrinsicoid deflection in $V_6$ (peak of r) represents the arrival of the depolarization wave in the epicardial surface of the left ventricle normally occurring within .05 seconds.
From a vector point of view the instantaneous vectors with respect to time may be considered in the transverse plane. The initial vector is anteriorly directed and the terminal vector posteriorly directed, thus during a single QRS cycle the instantaneous QRS vector normally rotates from pointing anteriorly to pointing posteriorly. Thus at V₁ the QRS complex starts with a small R wave of .02 to .03 seconds and the remainder of the deflection is a deep S wave. V₆, which lies more closely in the frontal plane of the body, is predominantly a positive deflection, starting often with a tiny Q wave of no more than .02 seconds and the remainder of the deflection a tall R wave.
III. Changes in the Electrocardiogram in Left Bundle Branch Block

From a pattern point of view, as a result of blocking the left bundle the interventricular septum is activated by the right bundle in the direction from right to left producing an upright deflection in $V_6$. The forces representing right ventricular activation traveling away from the $V_6$ position may produce a notching on the upstroke of the R wave. Activation of the left ventricle is delayed because it receives the impulse slowly through the musculature of the interventricular septum rather than through the rapid left bundle. As a result of the aberrant depolarization wave the QRS complex is prolonged to .12 seconds in $V_1$ and $V_6$ but the intrinsicoid deflection r wave in $V_1$ arrives .02 seconds after the beginning of the QRS complex. Thus septum depolarization from right to left produces an initial positivity in the left ventricular cavity, doing away with the septal q waves usually seen in leads facing the left ventricle.

From a vector point of view in bundle branch block, the sequence of depolarization and repolarization is altered. Basically there are three defects
which account for all QRS vector abnormalities encountered clinically.

1. When there is death of the myocardium, as in infarction the cells can neither be repolarized or depolarized, therefore no instantaneous QRS vectors are found in this region of the heart and if the patient survives, the abnormality in the QRS forces is often restricted to a small portion of the QRS loop.

2. In complete bundle branch block the sequence in which the various regions of the ventricles are depolarized is altered, with a change of the contour of the QRS loop as a result and excitation must spread through the myocardial syncytium. This prolongs the QRS interval as conduction through this system is one-tenth as fast as by conduction pathways.

3. If there is an increase in size of one or another ventricle the QRS vectors generated from that ventricle become larger in magnitude causing the QRS complexes on the various leads to be larger than normal in amplitude. There is no prolongation of the QRS interval as in left bundle branch block.
IV. Changes in the Electrocardiogram in Infarction

From a pattern point of view and for practical purposes, most infarcts occur over the left ventricle either anteriorly or posteriorly. An electrocardiogram manifesting typical findings of infarction will show QS or QR complexes in leads normally displaying RS or QR complexes. The Q wave will be more than 2mm deep and .04 seconds wide in infarction with the normal septal q of not more than 2 mm deep and .02 second wide. If the infarction is acute these same leads will show ST segment elevations signifying current of injury, the T waves will be inverted as the result of ischemia. The ST segment elevation is the most transitory because the myocardium, after infarction, will quickly die or recover, and in either case the current of injury will disappear. The Q wave, once established and representing dead muscle, is usually a permanent finding after an infarction has occurred. The Q wave is the best indicator of infarction as many conditions from ventricular hypertrophy to ischemia to electrolyte imbalance may alter the T wave. From a vector point of view, there are four abnormalities present in acute myocardial infarction and if all four abnormalities are present, this gives greater
significance to the fact that an infarction actually happened.

The normal QRS interval is less than .1 second. In an acute infarction the initial .04 second of the QRS vector tends to point away from the site of the infarct.

Because of ischemia in the tissues surrounding the infarct the mean T vector tends to point away from the site of the infarct and is more or less parallel with the initial .04 vector. Thus deep Q waves with inverted T waves in lead I, III, or a precordial lead have a high degree of reliability in the diagnosis of an old infarct.

An S-T vector due to injury current is produced which points toward the site of the infarction and in turn is opposite in direction to the initial .04 vector and the mean T vector. Thus in acute infarction the S-T segment is elevated in those leads which have Q waves and inverted T waves and depressed in leads with initial R waves and upright T waves, but the previous S-T vector abnormality is only transitory and returns to normal usually in a few weeks.

In many cases of infarction the terminal .04 vector has an abnormal direction because of peri-
infarction block with little or no prolongation of the QRS interval when this takes place.
V. Comparison of Criteria for Differentiating Left Bundle Branch Block in the Presence of Infarction

In this experiment, the effects of anterior infarction complicated by bundle branch block upon the form of the QRS complex of the canine electrocardiogram was experimentally demonstrated.

In an experimental study seventy dogs were used. The heart was exposed, the right or left branch of the bundle of His was cut and the anterior descending coronary artery was ligated in its midportion. The chest was then carefully restored and after a period of seven to forty days, when the animal had recovered completely from the operation, the electrocardiographic observations were made. The standard limb leads and unipolar precordial leads were taken with the chest intact.

In dogs with normal intraventricular conduction, infarcts similar in size and location to those induced in these experiments usually give rise to large Q deflections in Lead I. The significance of Q leads in Lead I in humans is later discussed by Bodeman.

In a comparison of animals number 64 with left branch block only and animals 68 and 70 with both left branch block and anterior infarction, the limb
leads displayed no changes in the ventricular complex that suggested the presence of infarcted cardiac muscle.

In conclusion then myocardial infarcts in dogs induced by ligating the anterior descending coronary artery in its middle third do not usually modify the QRS complexes of the standard limb leads in a characteristic manner when bundle branch block is present. When left branch block is present, infarction of the kind in question does not give rise to characteristic changes in the QRS complexes of the precordial leads because the potential of the left ventricular cavity and, therefore, of the epicardial surface of the surface of the infarcted region is positive during the earliest part of the QRS interval.

In a study of 169 cases of bundle branch block by Sodeman in human beings defined by the QRS interval measuring .12 seconds or more, and pronounced slurring or notching of the QRS complex as being present. In 92 cases, there was no S wave in Lead I and these were classified as left branch block.

The purpose of this article was to present and discuss observations on the evidence, in standard Lead I, of QRS complexes which display an initial
downward deflection, or \( Q \) wave.

On the theoretical grounds, one might expect that in left bundle branch block, damage to the ventricular septum would lead to the appearance of a \( Q \) deflection in Lead I. In uncomplicated left branch block, the cavity of the right ventricle is negative throughout the QRS interval, but the cavity of the left is initially positive because of the direction of the electrical forces produced by activation of the septal muscle from right to left. This initial positivity is transmitted through the still inactive free wall of the left ventricle to the outer surface of the chamber and to the adjacent parts of the body, including the left side of the precordium, the left axilla, and, when the heart is in a relatively horizontal position, as in most patients with left branch block, to the left arm. Under these circumstances the QRS complex of leads from the left side of the precordium display no deflection, and those of Lead I are of the same form. However when the septum is extensively damaged the electrical forces produced by its activation are reduced or abolished, and the initial negativity of the right ventricular cavity is transmitted to the left, and, consequently, to
those regions on the left side of the body that are initially positive in left branch block when the septal muscle is healthy. When this happens, Q deflections occur in leads from the left side of the precordium. They may be expected in lead I also.

The article goes on to state that bundle branch block in man is not an isolated event, but is almost always complicated by other cardiac abnormalities. Thus the form of the electrocardiogram is determined not only by the failure of one bundle branch to conduct, but by extensive lesions of the ordinary ventricular muscle, as in infarction, and by involvement of other conducting tracts or the Purkinje networks. Therefore the possibility that the presence of Q wave deflections in left branch block may be due to a combination of conduction defects must be borne in mind.

Of the 92 cases of left bundle branch block defined by the criteria previously given the frequency of Q deflections in lead I were such that eight cases had them present whereas 84 cases had Q waves absent. In conclusion, an initial downward or Q deflection is very uncommon in human left branch block and when it does occur in an electrocardiogram, other-
wise characteristic of this conduction defect, a lesion of the ordinary muscle of the ventricular septum should be suspected.

Myers et al reviewed a series of EGG and pathologic findings in infarction. Infarction of the interventricular septum was demonstrated pathologically in 102 cases, which represented an incidence of 63 per cent in a series of 161 cases.

Of these 102 cases, a QRS interval of .12 or more, an initial upstroke in all leads facing the left ventricle, and an abnormally delayed intrinsicoid deflection in left axillary leads were found in four cases and were attributed to left bundle branch block independent of the septal infarct in three of these. In the remaining case, autopsy revealed an acute infarct limited to the left side of the apical two-thirds of the septum, and the subendocardial layer of the anterior and posterior walls of the left ventricle, and the pattern was attributed to septal activation by impulses distributed through the right Purkinje plexus.

At necrotopsy in all four cases infarcts involved approximately one half of the septum. Since half of the septum was spared in each of these four
cases, initial R, instead of Q waves would have been expected in left ventricular leads if left bundle branch block had been present. Because of the previously mentioned evidence against left bundle branch block, it was concluded that the conduction defect in these cases was in the free wall, but that a conduction defect in the septum could not be excluded positively.

Thus in left bundle branch block, irrespective of the presence or absence of septal infarction, right ventricular Leads VI and V2 may display a Q wave, representing initial negativity of the right ventricular cavity due to reversal in the vector associated with septal activation. The right-to-left activation of the septum may produce a greater negative force over the right ventricle that the positive force produced by the free wall of the right ventricle. They concluded "Thus, in cases of left bundle branch block, the presence or absence of septal infarction cannot be determined from the contour of the QRS complex in right ventricular leads."

Dressler and associates undertook a study in an effort to differentiate the features of "pure left bundle branch block from superimposed changes due to myocardial infarction with special emphasis given to
changes of the RS-T segment and the T wave." The study included twenty-eight patients with what appeared to be "uncomplicated" left bundle branch block and fifteen patients for whom the diagnosis was made of myocardial infarction in addition to block in the left division of the bundle. The authors were well aware that in the majority of patients bundle branch block is due to coronary arteriosclerosis and is the result of an ischemic lesion interrupting the continuity of the intraventricular conduction pathways. However the number of patients with "uncomplicated" left bundle branch block available in this study was small because an effort was made to exclude not only patients with a history suggestive of myocardial infarction but also those which there was complaint at any time of anginal pain in conjunction with either effort or rest. Since it had been repeatedly stressed that myocardial infarction sometimes occurs in the absence of a history of a classical coronary attack, and that it may be indicated merely by short spells of angina of effort or of rest the central "uncomplicated" left bundle branch block group was screened in the sense that there was neither a suspicious history nor any other evidence suggestive of myocardial infarction.
In the patients with "complicated" left bundle branch block, the diagnosis of myocardial infarction was based on post-mortem findings, evidence of infarction shown by tracings which either preceded the development of bundle branch block or were taken after disappearance of bundle branch block, and the classical clinical picture and confirmatory laboratory evidence of myocardial infarction.

1. QRS Changes

Of the twenty eight patients with uncomplicated left bundle branch block, certain features of the QRS complex are of significance in the differential diagnosis of superimposed myocardial infarction. Q waves were not observed in Lead I but were present three times in Lead III associated with an R deflection, and once in chest lead five. The amplitude of the Q waves was less than 1.0 mm and less than one-fourth the size of the following R deflection and their duration was no longer than 0.03 seconds. No notching of the Q wave was observed in "pure" left bundle branch block. Thus in this group of twenty eight patients, a Q wave was never present in Lead I, whereas Sodeman and associates have observed a Q₁ in eight out of ninety-two patients classified as having left bundle branch block according to his criteria as previously stated.
2. ST Segment Changes

Attention was now focused on the features of the final ventricular complex because abnormalities of the RS-T segment and the T waves are of paramount importance in the diagnosis of myocardial infarction complicating bundle branch block, especially when changes of the QRS complex are absent of of equivocal meaning. In bundle branch block the initial and final ventricular complexes tend to move in opposite direction, the more as the amplitude of the QRS complex becomes large. Of the uncomplicated 28 cases of left bundle branch block prominent R deflections were invariably present in Lead I and in the precordial lead from the C₆ position. They were usually associated with depressed RS-T junctions, and, in general, depression of the RS-T junction increased with the amplitude of the R wave.

3. Changes in the T Wave

Inversion of the T wave was less regularly associated with prominent R deflections than was depression of the RS-T junction. Inverted and mainly inverted T waves were present in Lead I in seventeen instances and in the precordial lead from position C₆ in fourteen instances of the total of 28. The inverted or mainly inverted T waves were always of asymmetrical shape,
their ascending limb pursuing a steeper course than
the descending limb. Of the twenty-eight patients
the RS-T junction was depressed in all but three inst-
ances, and in the latter right was at the isoelectric
line.

4. Pattern Changes Associated with Infarction

Of the fifteen patients in this series the diag-
nosis of anterior infarction in association with left
bundle branch block was made for ten patients.

Q Waves were observed both in Lead I and in leads
from positions on the extreme left side of the chest
in six instances; in Lead I only in one, and in leads
from the left side of the precordium only in one inst-
ance. Dependable evidence of myocardial infarction
in the presence of left bundle branch block is the
combination of diagnostic changes of the qRS complex
with significant features in the final ventricular com-
plex. In the group of ten patients with anterior wall
infarction complicating left bundle branch block, the
most frequently encountered significant change of the
final ventricular complex was an inverted T wave with
symmetrical limbs and either isoelectric or elevated
RS-T junction. This sign was observed in eight inst-
ances; in three it was present in Lead I and pre-
cordial leads from the left side simultaneously; in
four it was observed in Lead I only; and in one, only in leads from the left side of the precordium. Next in frequency ranged significant elevation of the RS-T junction in the chest leads. It was present in seven. In three instances of infarction with left bundle branch block, prominent S waves in the chest leads were associated with plus-minus T waves.

In the presence of left bundle branch block, posterior infarction manifests itself more often by significant features in the final ventricular complex that by changes of the QRS complex.

Sodeman and associates had previously observed a QS deflection in Lead III in one-third of a group of "unselected" patients with left bundle branch blocks. In Dressler's series of twenty-eight patients with uncomplicated left bundle branch block, a QS deflection was present in Lead III in 25 percent. They concluded that the finding of QS deflection or Q waves in Lead III has no significance for the diagnosis of posterior infarction unless it is associated with Q waves in Lead II. "We have never observed a Q wave in Lead II when left bundle branch block was uncomplicated."

In the material of this study, posterior infarction was diagnosed in six patients, and all of them
but one presented QS deflections or Q waves in Lead III. A Q wave in Lead II was absent in four out of six tracings.

Besides the finding of a QS deflection or Q wave in Lead III, uncomplicated left bundle branch block had in common with posterior infarction some features of the final ventricular complex including elevation of the RS-T junction in Leads II and III, depression of the RS-T junction in Lead I and in leads from the left side of the precordium, and inversion of the T wave in Leads II and III.

In a series of experimental infarcts produced in dogs and comparing these with actual clinical patients, Kennamer and Frinzel proposed changes in the QRS complex and S-T segment which may be useful in the diagnosis of myocardial infarction in the presence of left bundle branch block, despite the absence of the coronary Q wave.

1. QRS Changes

The experimental study on the change in the QRS complex was performed by cutting the left branch of the bundle of His in nine dogs from four days to four weeks after ligation of the anterior descending artery. Results produced three kinds of infarction
patterns consistent with findings indicating that strong positive potentials recorded on the epicardial surface of ventricles with bundle branch block are generated primarily by depolarization of the epicardial and subepicardial layers directly beneath the electrode.

A. Subendocardial infarction left the outer layers of the ventricle intact causing the surface potential during bundle branch block to be the same as in uninfarcted ventricles.

B. Patchy infarction which inactivated some, but not all, subepicardial muscle caused a diminution of the surface positivity, presumably because the amount of subepicardial tissue undergoing depolarization was abnormally small.

C. Through and through infarction of the ventricle with bundle branch block eliminated all electrical activity between the epicardial electrode and the subjacent cavity with the dead tissue serving as a conductor through which the small positive cavity potential is transmitted to the surface. The conclusion is that experimental infarcts confined to the deeper layers of the myocardium do not alter the normal surface complex. In ventricles with normal conduction
or with bundle branch block, therefore, the surface electrocardiogram appears to reflect primarily the status of subepicardial muscle and is relatively uninfluenced by deeper layers of the wall. Among the animals studied, the epicardial R wave recorded over ventricles with bundle branch block was considerably smaller when extensive transmural infarcts were present beneath the electrode than when the underlying subepicardial muscle remained intact. This would indicate that in patients with left bundle branch block, the development of a "hole" through the anterior wall of the ventricle should be manifested by an abrupt decline of the R wave in precordial leads from overlying sites.

This group has observed several patients with left bundle branch who developed marked reductions in the size of the left ventricular R wave in association with myocardial damage. One patient had a persistent left bundle branch block following two earlier myocardial infarctions with R waves 7.5 mm tall in V5 and 7 mm tall in V6. The patient then endured another myocardial infarction and the height of the R waves was 3 mm and 2 mm in V5 and V6 respectively. The authors explain this reduction by the probable development of a more
through-and-through infarction, or more extensive patchy infarction with epicardial involvement. Emphasis is also placed on the taking of a control electrocardiograms following suspected myocardial damage to show a marked reduction in the height of the R wave as compared with preinfarction control tracings, then the diagnosis of infarction is confirmed.

In conclusion "In patients with bundle branch block, the development of myocardial infarcts involving large amounts of subepicardial muscle may be manifest by a decrease in the magnitude of the R wave in precordial leads overlying the left ventricle."

2. S-T Segment Changes

It had been previously stated that in acute myocardial infarction significant changes are usually limited to the S-T segment without bundle branch block, the S-T segment and T wave usually move in a direction opposite to the QRS complex. In those leads where there is a tall broad R deflection, the onset of the S-T segment is usually depressed and the T wave inverted. In those leads where there is a prominent wide S wave there is usually upward displacement of R-ST junction together with an upright T wave. Dressler had previously found that in chest leads V2 and V3 this elevation may be as high as 7 mm and the upright T wave as high as 19 mm in amplitude.
In order to understand more fully the S-T segment changes in the presence of left bundle branch block, a series of experiments were performed in which simultaneous epicardial and subendocardial electrocardiograms were recorded when acute myocardial infarctions was superimposed on left bundle branch block in five dogs. Control electrocardiograms were taken from the epicardial, subendocardial, cavity, and limb leads, and after the control tracings, a large branch of the anterior descending coronary artery was tied with tracings being taken at 30 seconds, intervals following the occlusion. At 30 seconds, one minute and one and one half minutes after the tie, the surface lead showed progressive S-T segment elevation while in the subendocardial lead the S-T segment remained isoelectric. Thus the charges which occur in dogs in S-T elevation are primarily the same when bundle branch block is present as when normal conduction is present.

The authors then site several cases of S-T segment changes which have occurred spontaneously after a two-step exercise test with attacks of chest pain more severe that ordinary angina pectoris, but there were no signs of acute myocardial infarction, and it was believed that these represented episodes of coronary
insufficiency or coronary failure.

One example includes a patient who had left bundle branch block and had recently developed angina pectoris. Before exercise his control electrocardiogram revealed the left bundle branch block with S-T depression of 1 mm. in Lead I, S-T elevation of 4 mm. with upright T wave in V2; and S-T depression of .5 mm and a biphasic T wave in V6. Immediately following a two-step exercise test, the depression in Lead I became 2 mm; V2 showed S-T elevation of 7 mm. with a taller more peaked T wave; and V6 showed a 1.5 mm. depression. This depression of the S-T segment in the leads overlying the left ventricle in combination with S-T elevation in leads overlying the right ventricle has been observed in several other patients with left bundle branch block who had similar episodes of chest pain.

Thus "the diagnosis of acute myocardial infarction can be made from the finding of significant S-T segment elevation following the large, broad, positive complex of left bundle branch block. Other changes in the coronary circulation may also be reflected by alterations in the RS-T segment despite the presence of left bundle branch block" namely, coronary insufficiency as
well as a positive two-step exercise test.

Thus even in the absence of the coronary \( q \) wave in myocardial infarction the authors conclude that the previously discussed changes would aid in making more conclusive the diagnosis of coronary artery disease in the presence of left bundle branch block. Specifically the S-T segment changes may be detected only if the previous tracings are available for comparison.

Rhoads, Edwards, and Pruitt\(^9\) correlated pathologic electrocardiographic, and clinical data in 39 cases in which there was a confluent myocardial infarct in the presence of left bundle branch block. The material consisted of cases in which necropsy was performed at the Mayo Clinic between January, 1947, and April, 1959, in which all of the following characteristics prevailed; 1. a confluent myocardial infarct, either recent or healed, as revealed on examination; 2. availability of electrocardiograms, including a minimum of three standard leads and three precordial leads, taken at a time when the infarct was present as judged retrospectively by pathologic examination correlated with clinical findings. 3. presence of left bundle-branch block when the infarct was present as judged by pathologic exam-
ination and clinical history. The 39 cases of left bundle branch block were included in this study if they met all of the following criteria; 1. QRS complex of .12 seconds or greater. 2. Sinus origin of the QRS complex. 3. P-R interval of .12 seconds or greater. 4. No S wave in Lead I. 5. Broad R wave in left precordial leads, or in Lead AVL if the left precordial leads were transitional in form. 6. Intrinsicord deflection starting .08 seconds or longer after the beginning of the QRS complex in leads over the left ventricle and 7. QS or rS wave forms in Lead V₁ with normal intrinsicord deflection.

They conclude that substantial evidence of transmural anteroseptal myocardial infarction in the presence of left bundle branch block would appear to be afforded by a Q deflection in Lead I or a Q deflection or Q wave equivalent in precordial lead V₅.

Supportive evidence of infarction involving the posterior wall of the left ventricle in the presence of left bundle branch block would appear to be afforded by a Q deflection in Standard Leads II and III as it was 3 of 39 cases in which the diagnosis of posterior infarct was made at necropsy. This compares consistently with Dressler.
In two of the three instances in which electrocardiograms showing left bundle branch block were recorded both before and after myocardial infarction, the height of the R waves in the standard leads decreased markedly. This is consistent with Dressler and associates.

Changes in the S-T segments and T waves of a degree to be considered suggestive of myocardial infarction occurred in a minority of cases in which myocardial infarcts were recent, which is not consistent with Dressler.

B. Vectors Changes

"In myocardial infarction the first forces of the QRS interval are changed in direction. What happens to this deformity of first forces when left bundle branch block alters the way in which excitation enters the left ventricle, one might guess that left bundle branch block will further alter these initial QRS vector abnormalities of infarction. This is exactly the case. No matter what direction the initial forces have been caused to take by infarction left bundle branch block will cause them to point leftward and posteriorly. This is completely obscures the initial QRS force deformity of infarction, and with our present knowledge, there is no way to make the diagnosis of infarction from the
QRS complexes when left bundle branch block is present.

This means that there is no way to detect infarction in the presence of left bundle branch block from the direction of the initial QRS vector. Nor has any abnormality of later QRS forces been so far identified in left bundle branch block which would indicate the presence of infarction. To be sure, if the infarction is acute there may be S-T and T vector abnormalities present characteristic of infarction, but these and the direction of the ventricular gradient are the only electrocardiographic clues of infarction to be seen in the tracing with left bundle branch block.

Some of the pattern St-T changes have already been discussed. If it is due to a conduction defect, it will be directed from 150 to 180 degrees away from the mean spatial QRS vector. If it is not more or less opposite in direction to the mean QRS vector, the presence of current of injury may be suspected.

Primary T-vector abnormalities are due to alterations of myocardial cellular metabolism, and repolarization is delayed at the affected region of the heart which take place when repolarization in impaired but not prevented at one or another region of the heart.

A secondary T-vector abnormality is seen in
ventricular conduction disturbances in which, the T-vector alteration is specific for the alteration in QRS vectors which the conduction defect has produced.

The ventricular gradient is a measurement of the extent to which the mean spatial T-vector in a given patient has been prevented from having its hypothetical direction opposite to the QRS vector, thus with left bundle branch block the ventricular gradient is unchanged in magnitude or direction, indicating that only secondary T-vector changes are present, but if the ventricular gradient has a different and abnormal direction, indicating that the T waves and T vector are affected by both primary and secondary alterations.

DePasquale and Burch presented the spatial vectorcardiograms of fifteen patients with left bundle branch block complicated by myocardial infarction proved by autopsy and describe distortions in the QRS-SE-loops not usually found in patients without myocardial infarction. They suggest continued correlation of the spatial vectorcardiogram in left bundle branch block with autopsy studies should be useful in making this distinction.
Using vector analysis angle postulates the electrocardiographic recognition of myocardial infarction from QRS changes and in the presence of left bundle branch block based on the demonstration of either of the two vectors or loops resulting from infarction, namely, the QRS-death and peri-infarction block vectors.

"If an electrocardiogram prior to infarction is not available, the analysis can still be made if there are serial changes on two or more electrocardiograms taken after infarction."
VI. Summary

Various studies from experimental infarcts produced in dogs to actual clinical studies based on pattern and vector changes have been presented attempting to differentiate myocardial infarction in the presence of left bundle branch block.

In chronological sequence the criteria of left bundle branch block and changes in the Q wave, QRS changes, alterations of the ST segment and T wave changes are compared in the presence of myocardial infarction mainly by patterns, and by vectors to a lesser extent.

Most of the authors agree that with the prolongation of the QRS interval in left bundle branch block, the septal Q of myocardial infarction is obliterated, transitory alterations of the S-T segment may occur in acute myocardial infarction, whereas changes in the T wave may be caused by a variety of conditions including coronary insufficiency and exercise. The value of a control electrocardiogram with left bundle branch block taken before the new infarct is discussed.

However from a vector point of view without regard to the site of infarct, the alteration of
the ventricular gradient in primary and secondary T wave changes is discussed. Even in the absence of a previous tracing, the possibility of diagnosis of myocardial infarction is postulated with any one of two infarction vectors being present.
VII. Conclusion

In complete left bundle branch block the QRS complex is prolonged to .12 seconds usually altering the septal Q wave normally seen in leads facing the left ventricle. Left bundle branch block may be manifest after infarction making the diagnosis of a new infarct superimposed upon the old infarct difficult. The Q wave will be more than 2 mm. deep and .04 second wide in infarction with the normal septal Q not more than this. If the infarction is acute, ST segment elevations are transitory as the myocardium, and the current of injury will disappear.

Criteria for pattern differentiation is discussed by various authors each giving his own criteria defining bundle branch block in the presence of infarction of various parts of the ventricle. No conclusion can be drawn from these studies as various changes in the Q wave, QRS complex, and RS-T segment are not conclusive and sometimes contradictory. However the value of a suspected post-infarction electrocardiogram is agreed upon as a valuable adjunct.
BIBLIOGRAPHY


