1954

The Significance of Q Wave in posterior myocardial infarction

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Submitted in Partial Fulfillment for the Degree of Doctor of Medicine

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May 1, 1954

Omaha, Nebraska

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INTRODUCTION

In 1930, Pardee made a distinct contribution to electrocardiography when he directed attention to the deep Q wave appearing in Lead III in some electrocardiograms. He observed that many of the patients exhibiting the deep Q_3 suffered from the anginal syndrome, and he suggested that the large Q wave was strongly suggestive of coronary disease (1).

Soon afterwards, clinicians everywhere began to find deep Q_3's in the electrocardiograms of many persons who apparently had no heart disease. Naturally, this led to efforts to find other clues or more exacting criteria so that distinction could be made between pathologic and normal Q waves. The Q waves in Lead II were than considered, and after Wilson's introduction of unipolar extremity leads, clinicians had the use of the left leg lead to aid in diagnosis of posterior infarction. More recently, the value of esophageal, deep epigastric, and back leads have been investigated. The theory of the use of these leads is that they should give much the same information in posterior infarction, that precordial leads do in anterior infarction.

The purpose of this paper is to present the diag-

nostic value of the Q wave changes encountered in the various leads in posterior infarction. Since the precordial, left arm and right arm unipolar leads, and Lead I do not exhibit Q wave changes in posterior infarction, they will be omitted in this paper.

PROPOSED CRITERIA FOR A PATHOLOGIC Q WAVE

Pardee's initial criteria for differentiating a pathologic from a normal Q_3 were as follows (1):

1. The Q_3 must be at least 25% of the largest excursion of the QRS in any lead.
2. There must be no right axis deviation.
3. R_3 must be present and S_3 absent.
4. The QRS complex must not be M- or W-shaped.

In 1934, Durant (2) studied the electrocardiograms of 96 patients with a deep Q_3. Later correlation of the records with autopsy findings led him to suggest that a Q_3 could be followed by an S_3, and a QRS complex could be W-shaped and still be pathologic. He proposed the following criteria for electrocardiographic diagnosis of posterior infarction:

1. Q_3 should equal at least one-half of the largest QRS complex in any lead.
2. Q_2 should be present and at least one-fourth as large as R_2.

3. Left axis deviation or normal electric axis should be present.

4. An inverted T wave in Leads II and III help confirm the diagnosis.

The same year Wallace (3) confirmed Durant's observation that the presence of a Q_2 along with a Q_3 was more likely to represent myocardial infarction. Several years later, Bayley (4) studied 19,000 electrocardiograms along with the associated clinical data. He concluded that a Q_3 of less than 0.03 second duration was of little importance with respect to coronary disease unless there were also typical changes in the R-S segment and T waves. He then set his criteria that a Q_3 must be 0.04 second or more in duration with a Q_2 of 1.0 mm. or more to be indicative of posterior infarction.

In 1944, Lyle (5) studied the standard and unipolar extremity leads of the electrocardiograms of 29 patients with a deep Q_3 conforming to Pardee's criteria. She noted that 10 of these patients had a deep Q wave in lead VF, and these patients all had a typical history of coronary occlusion. She concluded that if the Q (in VF) is 25% or more than the R (in VF), there is evidence of a posterior infarction. This diagnosis

is enhanced if the Q is 0.04 second or more in duration, and the QRS complex in VF is followed by an inverted T wave. Using this criteria she observed that an R_3 need not be present, and a Q_3 may be less than 25% of the largest R wave and still be significant of posterior infarction.

The following year, Goldberger (6), using his augmented unipolar extremity leads, analyzed the electrocardiograms of 50 cases of posterior infarction and 100 cases of normal and hypertrophied hearts. All of these cases exhibited a Q wave in Lead aVf of varying amplitude and duration. Thirty of the 50 with posterior infarction showed a QaVf of 40% or more of the QRS complex in Lead aVf, and the T waves were coved in all but three of these, and these three cases had old healed posterior infarcts. In all but one of the 30, the QaVf was 0.04 second or more in duration. In the 100 cases without infarction, only two had a Q:QRS ratio of 40%, and the duration of the Q waves in these cases was less than 0.04 second. Therefore, he concluded that when the QaVf has a duration of 0.04 second or more and has an amplitude of 40% or more of the entire QRS complex, that record indicates posterior infarction. An inverted T wave in Lead aVf enhances

the diagnosis.

The same year Myers and Oren (7) studied the esophageal leads, history, laboratory and clinical data of 50 patients, and diagnosed 24 as having posterior infarction. Forty-five of these patients conformed to Pardee's criteria for posterior infarction. The standard leads in some of the 21 patients excluded from the infarction group (but included in Pardee's group), were indistinguishable from some of the proved cases of infarction. Lead aVf revealed a Q wave of 25% or more of the corresponding R wave in 22 of the 24 with infarction, and in only 3 of the 21 without (2 of these 3 had a QRS voltage of less than 0.5 millivolt, and the third was a horizontally positioned heart). Thus, a much closer correlation existed with the interpretation of the aVf lead than with the standard leads based on Pardee's criteria.

In 1949, Lowen and Pardee (8) analyzed 59 records from patients diagnosed as having posterior infarction and compared the results using Pardee's criteria with that of Lyle, Goldberger, Myers and Oren. They found that 58 of the 59 fulfilled Pardee's criteria, 55 cases fulfilled the criteria of Lyle, Myers, and Oren, and only 13 Goldberger's. They concluded that Pardee's

criteria, using the standard leads, was most dependable.

Believing that in the previous study made by Myers and Oren, the cases may have been a little "too select" because they had chosen only records with a deep Q_3, Myers, Klein, et al (9) chose 110 cases which had posterior infarction proven at autopsy and studied the electrocardiograms of these cases. Thirty-five of the records showed horizontal or semi-horizontal position of the heart. Of the remaining 75 cases, 42 were diagnosed as having posterior infarction, 17 as borderline to suggestive, and 16 as negative. They used the following criteria:

1. QRS voltage in Lead aVf must be 0.5 millivolt or more.
2. Duration of QaVf must be 0.03 second or more as measured from onset to nadir.
3. QaVf must be 25% or more of the corresponding R as measured from the bottom of the base line to nadir.

Cases were borderline to suggestive if:

1. They fulfilled 2 of the above 3 requirements.
2. They fulfilled number 1, and had a distinct Q wave followed by a prolonged notched or coarsely slurred R wave in Lead aVf.

They observed that Leads II and III failed to provide diagnostic evidence of posterior infarction when aVf was negative. Also application of the Pardee criteria to the interpretation of findings in the standard leads led to error in a number of cases where correct diagnosis could have been made from the findings in Lead aVf.

Yu and Blake (10) analyzed the records on 109 patients which had a Q_3 of 25% or more of R_3. Fifty-four of these patients were considered to have posterior infarction on the basis of typical electrocardiogram changes, i.e.; Q waves in Lead III at least 25% of R_3 and often a Q_2, plus, either an elevated RS-T segment in Lead III, or a T_3 (and often a T_2) showing progressive lowering and finally sharp inversion. Also, 50 of these 54 had supportive evidence in the form of clinical and laboratory data. The other 4 were proven at autopsy to have posterior infarction. Their findings are listed in Table I. Yu and Blake conclude that in an adult, the presence of a QaVf which is 25% or more of the corresponding R and has a duration of 0.04 second or more is quite diagnostic of posterior infarction. Also, they conclude that normal persons may have a Q_3 conforming to Pardee's criteria.

PATIENTS	WITH INFARCTION		WITHOUT INFARCTION	
Incidence of:	No. of cases	%	No. of cases	%
Q_3	54	100	55	100
QaVf	53	98	21	38
Q_3 at least 25% of R_3	54	100	55	100
QaVf at least 25% of RaVf	49	91	3*	6
Duration of Q_3 of 0.04 second or more	31	54	7	12
Duration of QaVf of 0.04 second or more	18	34	0	0
Duration of QaVf of 0.03 second or more	32	59	1	2

*Two of these 3 were children who may normally have a deep QaVf.

TABLE I

Lack of adequate diagnostic criteria with the use of standard and unipolar leads has led to study of other leads in the diagnosis of posterior infarction. Esophageal leads were first used by Waller in 1889, but it remained for Hamilton and Nyboer (11) to focus medical attention on their applicability in myocardial infarction. Nyboer (12) noted that the esophageal ventricular electrocardiograms which were characteristic of posterior infarction closely resembled pre-

cordial electrocardiograms which were characteristic of anterior infarction and vice versa. In some cases in which standard leads were equivocal, a definite diagnosis of posterior infarction was established with esophageal leads. Burchell (13) concluded, after a study of 50 cases with esophageal leads, that cases of posterior infarction which could be diagnosed with esophageal leads had a diagnostic or suggestive Q_2Q_3 pattern in the standard leads. He implied that where Q_3 is the only abnormal finding, the esophageal lead is frequently equivocal.

Oram and Holt (14), in 1951, studied 27 cases in which posterior infarction had been established by history, physical, clinical, laboratory, and progressive electrocardiogram patterns. Using the esophageal leads they diagnosed 23 case of posterior infarction on the basis of the Q wave changes. Using QaVf, they diagnosed 24 correctly. Their criteria for a pathologic Q wave in either lead is that the Q wave be 25% or more of the corresponding R wave and have a duration of 0.04 second or more, and there should be an inverted T wave in the same lead. They concluded that esophageal leads are not as accurate as Lead aVf and, of course, not as convenient. Bain (15) and Sandbert (16) agree

that the esophageal lead adds nothing to Lead aVf.

Rubin et al (17) found no false positive diagnoses of posterior infarction using esophageal leads, but they got a false negative in 12% of the cases. Using the Q wave changes in Lead aVf, they obtained 7% false positives but fewer false negatives. They decided that esophageal leads were no more valuable than Lead aVf in the diagnosis of posterior infarction.

Other leads including back leads and deep epigastric leads (14,18) have been investigated and have been found to add nothing to the standard and unipolar extremity leads.

DEVELOPMENT OF THE Q WAVE

It can be noted from the above investigations that the Q wave can be misleading in the diagnosis of posterior myocardial infarction in a significant number of cases. Perhaps a review of the origin of the Q wave in the normal and posteriorly infarcted heart will help show why many of the discrepancies occur.

Non-Infarcted Heart: By definition, the Q wave is the initial downward deflection of the QRS complex--it may be found normally in any lead, and is usually not greater than 3.0 mm. in depth or greater than 0.03 seconds in duration (19). When appearing in the stand-

ard leads,, a Q wave indicates initial relative negativity in that lead, while in a unipolar lead, the Q wave represents initial negativity as measured from a zero potential. It is generally accepted that the impulse from the AV node is delivered to the left side of the septum,. and a wave of depolarization begins,, moving towards the right side of the septum. Since passage of electrical activity away from an electrode is recorded as negativity, leads from the left side of the septum (left arm, left leg, and esophagus), will exhibit a small negative deflection or Q wave. As soon as the excitation wave reaches the lateral ventricular wall and begins passage externally, a positive deflection (R wave) succeeds the Q wave (20). Thus, in the intermediate-positioned heart,, a Q wave may normally be seen in Leads I, II, III, VF or aVf, VL or aVl, and esophageal and precordial leads that face the left side of the septum.

However, in the vertical heart with right axis deviation,. the septum is rotated clockwise,. and more of the left septum faces the left leg than normally and less of it faces the left arm. This will cause a deep Q wave in Lead III because of the greater relative negativity between the left leg and left arm. There

may also be a Q_2 and a small QaVf. This is the reason no significance can be attached to a large Q_3 wave in right axis deviation.

In a horizontally positioned heart, the left side of the septum faces the left arm and the right side of the septum faces the left leg (6). Also, the direction of the excitation wave, as it travels through the ventricular musculature, is going to be more towards the left arm than the left leg, and may even be away from the left leg. Thus, a deep Q wave will be recorded in Lead III and maybe aVf, although in aVf there will usually be a small initial septal R wave followed by a deep S (9). Essentially the same pattern maybe seen inleft ventricular hypertrophy (20). Also, it might be added that in transversely positioned hearts with counterclockwise rotation, the right ventricle is the main component facing the left leg, and posterior infarcts of the left ventricle will hardly be recorded as a Q wave in the usual leads. This is why some authors (9,21) say records showing such cardiac position, cannot be correctly analyzed for posterior infarction on the basis of the Q wave changes.

<u>In Posterior Infarction</u>: Now let us consider the heart in infarction. Posterior myocardial infarction

is usually due to occlusion of the right coronary artery or its main branch, the posterior descending artery. In about 10% of subjects, the posterior portion of the left ventricle is supplied by the left coronary artery. The area involved is usually the diaphragmatic portion of the left ventricle and adjoining portion of the right ventricle (19).

In 1933, Wilson et al (22) pointed out that the interior of the ventricular cavity is negative. Thus a large transmural infarct of the posterior wall acts as a window, transmitting the negativity of the cavity to the leads facing the infarcted area and giving a QS deflection. However, if the entire thickness of the wall isn't destroyed, and the depolarization wave encounters responsive muscle, the initial Q wave is followed by an R wave. The amplitude of the R wave is reduced and the Q wave increased in rough proportion to the amount of muscle destroyed. When the posterior infarct is patchy in distribution, there may be a normal Q wave followed by a notched or slurred prolonged R wave, presumably reflecting a circuitous pathway of the impulse between islands of preserved myocardium. A small posterior infarct may not be manifested by diagnostic electrocardiographic changes in the left leg

lead, depending upon whether the potential variations of the left leg are derived chiefly from the involved or intact portion of the posterior wall (9,23).

Burch and Winsor (19) describe an area of infarction as consisting of three zones: The zone of necrotic tissue, the zone of injury, and the zone of ischemia. The first electrocardiographic changes after posterior infarction are an elevation of the S-T segment in Leads III and aVf and, usually Lead II, and a depression of the S-T segment in Lead I--these changes are due to the current of injury or zone of injury. In a few hours to a few days, Q waves tend to appear in Leads III and aVf, and often in Lead II, and the corresponding R's become smaller--this is due to the zone of necrotic tissue. As the infarct heals, the Q waves become more prominent, the S-T segments approach the isoelectric line, T_3 and TaVf become negative, and T_1 peaked. The infarct may eventually heal and the Q waves become smaller or disappear, or persist for many years after the acute episode, as the only sign that infarction has occurred in the past. Thus, the dead zone or infarcted area per se is indirectly responsible for the QRS changes encountered in myocardial infarction, and without them, a definite electrocardiographic

diagnois or infarction cannot be made.

CAUSES OF ERRORS IN Q WAVE INTERPRETATION

Now let us look at some of the reasons why interpretation of the Q wave in electrocardiographic records may lead to a wrong diagnosis as regards posterior infarction.

As pointed out earlier, a heart in the horizontal or semi-horizontal position may show deep Q waves in the absence of infarction, or there may be no significant Q wave in Lead aVf even in the presence of infarction of the posterior wall of the left ventricle. Thus, patients with ascites, pregnancy, obesity, or left ventricular hypertrophy may show misleading Q waves. It has been suggested that if the deep Q wave will disappear on deep inspiration, it is not due to infarction (5)--however, it has been proven that the Q wave of infarction may disappear on deep inspiration (7,9).

Probably some errors are due to calling an S wave a Q wave--if the deflection is preceeded by an upstroke, no matter how small, this initial upstroke is an R and the downstroke is an S wave (5,6,24).

Deep Q waves may normally occur in children. The reason is not known, but may be due to the greater

thickness of the interventricular septum (25,26).

Infarcts located high on the posterior wall adjacent to the auriculo-ventricular junction may be missed because of the absence of normal activity in this region causing little, if any, electrocardiographic change. This is especially apt to occur in clockwise, vertical hearts (9,27,28). The only clue to infarction in this area may be reciprocal S-T depression in the precordial leads.

In cases of slight posterior infarction, the infarct may be superficial with only injured muscle, and R-T and T changes may be the only electrocardiographic signs (28,29). The effort test may bring out a Q wave in Leads III and aVf in such cases (30).

In left bundle-branch block, the left side of the interventricular septum is not activated first, and the initial QRS deflections in those leads facing the left side of the septum will be distorted. Thus no accurate diagnosis of posterior infarction can be made in the presence of LBBB. However, the diagnosis of infarction can be made in right bundle-branch block because the left side of the septum is still activated first (6,9,27).

When the QRS deflection is small (4.0 to 6.0 mm.),

the Q/R ratio may be over 25% in normal subjects. Thus Q waves occurring with small QRS complexes, should be interpreted with caution (9,26).

An anterior infarction imposed upon a posterior infarct may cause a diminution of the Q waves, and the diagnosis of posterior infarction may be missed (27,28,29).

Pulmonary embolism and vitamin deficiencies may cause a pattern resembling posterior infarction (28, 31).

DISCUSSION

So far in this paper, I have tried to point out the origin of the Q wave, its relation to posterior infarction, and the various criteria offered for its diagnostic value. Some of the criteria have been so liberal as to include many cases who have no infarction, while others have been so strict as to exclude many with posterior infarcts. It is evident that no sharp dividing line can be drawn between the Q wave of the normal heart and that of infarction. Yet when several factors are considered, the diagnostic value of the Q wave in posterior infarction is significant.

It has been well extablished that a diagnosis of posterior infarction should not be based on a deep Q_3

alone, even if it conforms to Pardee's criteria. One reason is that a Q_3 does not necessarily reflect initial negativity of the diaphragmatic surface of the left ventricle. For instance, it may be present when both the left leg and left arm are positive, if the left arm is predominantly positive. Thus, if there is no Q wave in the unipolar left leg lead, there is probably no infarction. When posterior infarction is present, the left arm lead should show initial positivity, and since Lead III = (aVf - aVl)2/3, Q_3 should always be greater than QaVf in infarction. Probably if Q_3 is less than, or equals QaVf, posterior infarction is not present (24, 32). Just from this, it can be noted that diagnosis should not be based on the study of the EKG pattern in one lead alone. In addition to examining Leads III and aVf, Lead II should be examined, too. The presence of a Q_2 enhances the diagnosis. Also, if previous electrocardiograms are available for study, a lowering or absence of Q_1 is expected in posterior infarction (24).

Determination of cardiac position is important before making a diagnosis of posterior infarction on the basis of the Q waves alone, because horizontal or semi-horizontal position of the heart may cause erroneous

Q wave changes.

Other factors that lead to errors have been pointed out in the previous section. When these are all considered, it can be seen that the rather high percentage of errors made by some investigators is more excusable. Let us review the work of Myers, Klein, et al (9). They found that 35 of their 110 cases had transverse cardiac position, and observed that Q waves were of no value in the diagnosis of infarction in these cases. Of the remaining 75 cases, sixteen were classed as being negative for posterior infarction. Table II shows an analysis of the cases with regard to position of the infarcts.

Extent of Infarction	Number Missed	Total Number of Cases
2/3 or more of the length of posterior wall	1	31
Middle 1/3 of posterior wall	1	5
Posteroapical infarct extended to middle 1/3	2*	14
Confined to apical 1/3	8	14
Confined to basal 1/3	3	3
Posterobasal lesion continued into middle 1/3	1	8

*These 2 had left bundle-branch block

TABLE II

It has been noted before that infarcts which are small (as those confined to the apical 1/3 would necessarily be), those which are located in the basal region, and those in the presence of LBBB are often missed. When these factors are considered, we can see that, in reality, Myers et al had a high correlation. Applying the criteria of Myers et al to the observations of Yu and Blake, it will be noted that 59% could be classed as diagnostic of posterior infarction, with an additional 32% as suggestive to borderline. Thus it would seem to me that, while none of the criteria suggested are ideal, Myers, Klein, et al have offered the best criteria for a significant Q wave.

SUMMARY AND CONCLUSIONS

Since Pardee first stressed the importance of a deep Q wave in Lead III and listed his criteria for the differentiation of a normal from a pathological Q wave, other authors have tried to list more exacting criteria, utilizing not only the standard leads, but unipolar extremity, esophageal, and other leads. The size of a significant Q wave has varied in the opinions of the various clinicians. Pardee stated that it should be at least 25% of the QRS complex; Durant suggested 50%, with a Q_2 of at least 25% of R_2; Wallace

and Bayley confirmed Durant's observations. Goldberger thinks that a QaVf should be 40% of the corresponding QRS complex and have a duration of 0.04 second to be pathologic. Lyle,, Myers and Oren,, and Yu and Blake have suggested that a Q wave in the left leg lead,, which is 25% or more of the corresponding R and has a duration of at least 0.04 second,. is diagnostic of posterior infarction.

Myers,. Klein, et al seem to have worked out the best criteria to date, which is as follows:

1. QRS voltage in Lead aVf must be 0.05 millivolt or more.
2. Duration of QaVf must be 0.03 second or more as measured from onset to nadir.
3. QaVf must be 25% or more of the corresponding R as measured from the bottom of the base line to nadir.

The record is classed as borderline to suggestive if:

1. It fulfills two of the above requirements.
2. It fulfills number one and has a distinct Q wave present in Lead aVf followed by a prolonged notched or coarsely slurred R wave.

Esophageal leads have been investigated by Hamilton and Nyboer, Burchell,. Bain, Sandberg, Oram and

Holt, and Rubin et al, and it has been concluded that this lead has nothing to offer that cannot be learned from the left leg lead. Back leads and deep epigastric leads have contributed nothing.

Records which show horizontal or semi-horizontal position of the heart, left ventricular hypertrophy, or left bundle-branch block cannot be correctly analyzed for posterior infarction on the basis of the Q wave changes. Significant Q waves may not be manifested in cases when an anterior infarct is imposed on a posterior infarction, when the infarct is not transmural, or when the infarct is small or located high on the posterior wall. Deep Q waves may be obtained in non-infarcted hearts in children, pulmonary embolism, vitamin deficiencies, or when the QRS voltage is low.

Note: The suggestions and assistance received from Dr. Edward Langdon, M. D., in the preparation of this thesis is acknowledged and greatly appreciated.

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