QS- and QR-Pattern in Leads V<sub>3</sub> and V<sub>4</sub> in Absence of Myocardial Infarction: Electrocardiographic and Vectorcardiographic Study

By Borys Surawicz, M.D., Robert G. Van Horne, M.D., John R. Urbach, M.D., and Samuel Bellet, M.D.

A QS or QR pattern in the absence of myocardial infarction is frequently present in lead V<sub>5</sub> and occasionally in lead V<sub>6</sub>. Exploration by means of multiple chest and abdominal unipolar leads and vectorcardiograms revealed that in almost all such cases, the vector of the initial portions of the QRS complex is directed downwards. Accordingly, in the absence of infarction, patients presenting this pattern almost invariably showed an initial R wave in the leads recorded from positions below the standard level of V<sub>3</sub> and V<sub>4</sub>. The vast majority of patients with myocardial infarction with a similar QRS pattern showed a Q wave in the lower leads. Consideration of vertical components of cardiac voltages may be helpful in the interpretation of the precordial leads.

The presence of a QS pattern or of an abnormally deep and wide Q wave (deeper than 25 per cent of succeeding R wave and wider than 0.04 second) in precordial leads V<sub>3</sub> to V<sub>6</sub> is usually, although not invariably, attributed to myocardial infarction. Occurrence of a QS pattern or of a significant Q wave in leads V<sub>3</sub> and V<sub>4</sub> and on some occasions even in leads V<sub>5</sub> and V<sub>6</sub>, in the absence of myocardial infarction, has been demonstrated in cases of left ventricular hypertrophy, hypertrophy or dilatation of the right ventricle, complete or “incomplete” left bundle branch block, right bundle branch block, and displacement of the heart. In his study of electrocardiograms which may be mistaken for myocardial infarction, Myers emphasized the occurrence of these patterns on several occasions. It seems to be justifiable to suspect that in many instances myocardial infarction has been diagnosed incorrectly on the basis of a QS, QR or QRS pattern in the precordial leads. (See fig. 1.) This suspicion is augmented by the fact that there are very few electrocardiographic diagnostic criteria which differentiate a QS or QR deflection of myocardial infarction from an identical deflection caused by other factors.

The present investigation was initiated in the hope of finding a method which might help to differentiate these patterns which are found with myocardial infarction from similar patterns not associated with infarction. At the same time we have attempted to obtain information regarding the factors responsible for the genesis of the QRS patterns which simulate myocardial infarction in the precordial leads.

Two major principles of electrocardiographic diagnosis were applied: (1) exploration of the precordium by means of multiple semidirect unipolar leads; and (2) vectorcardiograms representing the distant indirect leads and picturing the over-all or average electrocardiograms.

Method

The routine, 12-lead electrocardiograms made at the Heart Station of the Philadelphia General Hospital were screened daily during the period from
August 1953, until March 1954, for patterns displaying a QS complex or a significant Q wave in the standard precordial leads V2 and V4. Cases with a QS or QR pattern limited to leads V1 and V2 were not included because the occurrence of such patterns in the absence of myocardial infarction is widely known. Cases with a significant Q wave which is deeper than one-fourth of the R wave and wider than 0.04 second in leads V3 and V4 were not included because these findings are almost invariably due to infarction. As a result, the selected group included only cases of myocardial infarction in which the QRS changes due to infarction did not extend further to the left than lead V4 and cases of non-infarction with an absent initial R wave in leads V4 and V5 which could have been mistaken for the above infarction pattern. None of the features of the electrocardiogram, other than the QRS complex, were considered in the selection of cases.

During the selection of cases for this study, it became obvious that various observers differed in their judgment as to what is a discernible initial R wave. On several occasions a given electrocardiographic QRS complex was designated by some observers as R5 while others preferred to call the same complex QS. In order to determine the error which might be due to this factor of difference in interpretation, 15 different complexes with an absent or a very small initial R wave and a deep S or QS wave were presented to 20 experienced cardiologists who were asked whether, in their opinions, the 15 complexes should be designated QS, rS or undeterminable. The results of this inquiry are presented in figure 2. It has become obvious that an R wave smaller than 0.5 mm. can hardly be recognized as such even if the technical recording of the tracing

![Fig. 1. Electrocardiogram of a 57 year-old Negro man with pulmonary emphysema and a possible Addison's disease 24 hours before death. Myocardial infarction was suspected because of the QS pattern in leads V2, V3 and V4. Autopsy revealed no cardiac abnormalities except slight dilatation of the right ventricle. Heart weight 280 Gm. Thickness of the left ventricle 12 mm.; of the right ventricle, 4 mm.](image1)

![Fig. 2. Fifteen electrocardiographic complexes from one of the right precordial leads presented to 20 cardiologists. In the first line beneath each complex is shown the number of persons who designated this complex as RS; in the second line, the number of persons who designated this complex as undeterminable and in the third line, the number of persons who designated it as QS.](image2)
is good. The material selected for this study contains only cases in which all authors of this paper felt that there was no initial R wave in leads V3 or V4.

The patients selected for the study were subjected to the following examinations: (1) clinical evaluation, (2) recording of one or two leads synchronously with lead V3 or V6, (3) multiple chest and abdominal uninipolar electrocardiographic leads, (4) vectorcardiograms and (5) special roentgenologic studies. All these studies were performed on the same day.

(1) The clinical evaluation was based on a careful history and physical examination. The majority of the examined individuals were ward patients whose hospital and out-patient records could be traced back for a varying length of time. From the day on which the patient was selected for study, his course was followed by means of periodic clinical and electrocardiographic examinations. All available autopsy findings were secured.

(2) In order to establish whether the initial negative QRS deflection in leads V3 and V4 represented the earliest part of the ventricular depolarization, leads V3 and/or V4 were recorded synchronously with lead I, aVf, and one of the precordial V leads by means of Sanborn-Polyviso direct-writing electrocardiograph.

(3) The electrocardiographic exploration included standard limb and augmented uninipolar limb leads, standard precordial leads V1 through V6, V5n and V7 and 26 additional chest and abdominal leads. The additional leads consisted of two groups: leads recorded from the chest at the levels above, and leads recorded from the chest and abdomen below, the standard levels. The high leads were taken from the second and third intercostal spaces at positions V7 through V9, from the fourth intercostal space at the position V3 through V5 and also from the level of the fifth rib at position V4. The low leads were taken from the fifth intercostal space at the position V9, from the ensiform (E) level at the positions V1 through V3 and from the epigastriac level (ep), which was determined by the mid-point between the ensiform process and the umbilicus, at the positions V2 through V4. In some of the later cases, leads from the mid-line at the levels of umbilicus ("O") and between umbilicus and ensiform process ("EO") recommended by Lambert were recorded in addition.

(4) Vectorcardiograms were obtained with Sanborn Vectorcardiograph consisting of the Poly-Viso Recorder Model 64-1300 A, Coupling unit Model 78-100, and the Dumont Cathode Ray Oscillograph Type 304H. The electrode attachment systems were those described by Wilson and his co-workers referred to in this paper as "tetrahedron" and by Grishman and associates referred to in this paper as "cube".

Analysis of the QRS loops was undertaken in the following manner: The initial deflection was taken to be the first four points (0.02 second) emerging from the central blob. The direction of the initial QRS deflection was expressed through reference to the x, y and z axes in both reference systems. The direction of progression of the electron beam was recorded as clockwise or counterclockwise in the horizontal and sagittal planes. The total number of points from the beginning to the end of the QRS loop were counted in each plane. Each plane was then divided into four quadrants and the number of points counted in each quadrant. In addition, in each plane, it was noted in which of the quadrants the major part of the QRS loop area was situated. Irregularities and indentations in the loops were noted and arbitrarily graded from 1 (completely smooth) to 4 (very irregular).

(5) Six-foot chest roentgenograms with the patient in the supine position were obtained with the sites of the standard precordial electrode positions on the chest and abdomen indicated by lead numbers.

**Material**

Six groups of individuals were studied: Group 1, 24 patients with myocardial infarction; group 2, four patients with possible myocardial infarction; group 3, 25 patients with absent myocardial infarction; and group 4 six patients in whom myocardial infarction was considered unlikely; group 5, 10 patients with left ventricular hypertrophy; and group 6, 10 normal persons without evidence of cardiovascular disease.

Classification of the material in the first four groups was made, without the consideration of the results of special studies, on the basis of the clinical evaluation, follow-up records and autopsy findings, which were available in 10 patients. The group with infarctions included patients with conclusive evidence of myocardial infarction gained from autopsy or a combination of a typical clinical course and serial electrocardiograms. Most of the infarctions had occurred within the preceding 12 months. The group with possible infarctions included patients in whom infarction was suggested by the serial electrocardiograms, but the remaining clinical data were not sufficiently conclusive. The group with absence of infarctions included five patients in whom the diagnosis was established by autopsy and those patients in whom there was no suspicion of infarction either in the history or in the serial electrocardiograms recorded during a period from one to several years prior to the study. The group designated as "infarction unlikely" included similar patients in whom infarction was at no time suspected clinically, but no previous serial tracings were available.

Fifty-nine patients whose data were subjected to final evaluation included 45 males and 14 females. Forty patients were white and 19 were Negroes. The age of the patients ranged from 39 to 84 years, averaging 64.5 years. The distribution of the sex, race and age factors within the two major groups of proven and absent infarction showed no significant differences.
Patients in group 5 were selected at random from the Hospital population. They had proved left ventricular hypertrophy, as determined by x-ray study and an electrocardiographic pattern of left ventricular hypertrophy and "strain". None of them had history of myocardial infarction or chest pain at any time. The group included four males and six females who were 52 to 76 years old with an average age of 62.

Individuals in the group 6 were 25 to 47 years old. Five normal persons had hearts in a vertical anatomical and five in a horizontal anatomical position.

**Results**

**Time of Onset of QRS**

The results of the synchronous recording of lead V₃ or V₄ with other leads in all groups of cases can be summarized by a statement that in no case was the initial QRS deflection in leads V₁ or V₂ preceded by an earlier deflection in some other lead. The beginning of the QRS complex in leads V₃ and V₄ coincided usually with the beginning of the QRS complex in other precordial leads (V₁ and V₂) but in more than half of the cases occurred 0.01 to 0.02 second earlier than the QRS onset in the limb leads aVₑ or I.

**Direction of the Initial QRS Deflection**

The differences between the group with myocardial infarctions and the group with possible infarctions on the one hand and the differences between the group with absent infarction and the group in which infarction was unlikely on the other hand were insignificant. It appears to be justifiable, therefore, to discuss only the differences between the group with myocardial infarction and the group with absence of infarction. Following are the more important results:

(a) **Standard Precordial Leads:** In the standard lead V₃, a QS pattern or a significant Q wave was present in 22 out of 24 patients with infarction and in 21 out of 25 patients without infarction. In standard lead V₄, the QS pattern or a significant Q wave was present in three patients without infarction and in nine with infarction. In standard precordial leads V₅ through V₇, the presence of a small Q wave was encountered in 71 per cent of the infarction cases and in only 32 per cent of the noninfarction cases. In standard precordial leads V₃, V₁ and V₂ the groups with infarction and with no infarction showed only insignificant differences. In the majority of the patients in both groups all three right precordial leads showed a QS pattern, but an initial R wave was present in leads V₃, V₁, and in some cases also in V₂ in eight patients with infarction and six without infarction.

In the group of 10 patients with left ventricular hypertrophy, without a QS pattern in leads V₃ and V₄, a QS pattern was present in four cases in lead V₃, and in three cases in leads V₁ and V₂.

(b) **Low Precordial Leads:** In lead Vₑ an initial R wave was present in 17 per cent of the patients with infarction and in 48 per cent without infarction. The situation was very similar in lead V₁ₑ (lead V₁ made at level of ensiform). In lead Vₑ₂ the difference between the QRS pattern in patients with infarction and without infarction was somewhat larger; the initial R wave was present in only 8 per cent of the patients with infarction and in 56 per cent of the patients without infarction.

The greatest difference between the QRS patterns in the infarction and noninfarction groups was present in lead V₃ₑ; the initial R wave was present in 12 per cent of the group with infarction and in 96 per cent of the group without infarction. The pattern in the lead between standard V₂ and V₃ₑ, at the level of the fifth intercostal space, was similar to that in lead V₃ₑ in the group with infarction, but the presence of the initial R wave in the group without infarction was less frequent than in lead V₃ₑ. An initial R wave in leads V₄ₑ, V₅ₑ, V₆ₑ (lead V₃ made at level of epigastrium) and Vₑ₆ in patients without infarction was found as frequently as in lead V₃ₑ, but a higher number of patients with infarction showed an initial R wave in leads V₄ₑ, V₅ₑ, V₆ₑ and Vₑ₆ than in lead V₃ₑ.

In the group of 10 patients with left ventricular hypertrophy without a QS pattern in lead V₃ and V₄ a QS pattern was present in three patients in lead V₁ₑ, in two cases in lead Vₑ and in one case in lead V₃ₑ.

In the group of 10 normal patients, the initial QRS deflection was positive in all low pre-
cordial leads with the exception of a small Q wave in leads V₄ₑ, V₅ₑ, and leads V₂ through V₃ made at the epigastric level in four cases in which a small Q wave was present in aVF.

(c) High Precordial Leads: Leads from a level one intercostal space higher than the level of the standard precordial leads showed generally great similarity of the initial QRS deflection in both the infarction and the noninfarction groups. In leads V₂ through V₄ made at the second intercostal space, the difference in pattern in the infarction and the noninfarction groups was slightly greater since the initial QRS deflection in these leads was invariably negative in the noninfarction group while it was positive in 36 to 45 per cent of the infarction cases.

In the group of 10 patients with left ventricular hypertrophy without a QS pattern in leads V₃ and V₄, a QS pattern was present in six patients in lead V₁ made at the third and second intercostal spaces, in five cases in lead V₂ taken at the third and second intercostal spaces, in four cases in lead V₃ taken at the third intercostal space and in one case in lead V₄ taken at the second intercostal space.

In the group of five normal subjects with vertical anatomic heart position the initial QRS deflection was positive in precordial leads V₁ through V₅, all made from points above the conventional level. In the group of five normal subjects with horizontal anatomic heart position, a QS or QR deflection was present in one subject in lead V₁ made at the third and second intercostal space and in two subjects in leads V₃ and V₄ made from the level of the second intercostal space.

**Vectorcardiograms**

Vectorcardiograms were taken on 46 patients of groups 1, 2, 3, and 4. In 43 of these patients, the "tetrahedron" coordination of Wilson (11, 64) was used and in 45 patients the cube modification of Grishman was used. Since the group with proved and possible infarction, on the one hand, and the groups with absent or unlikely infarctions on the other hand showed no significant difference, only differences between the two major groups with proved and absent infarction will be discussed in detail.

(a) **Initial Deflection.** (First 0.02 second of the ventricular depolarization.) The most significant difference between the group with proved and absent infarctions was in the direction of this deflection along the y axis in both coordinate systems. The initial deflection was directed downwards in 90 to 92 per cent of the group without, and in only 36 to 46 per cent of the group with infarction. In the majority of tracings of patients in both groups (64 to 83 per cent) the initial deflection was directed to the left. In the z axis, when the tetrahedron coordinates were used, 75 per cent of the patients with no infarction showed anterior progression, whereas only 36 per cent of the group with infarction showed this progression. With the cube coordinate system there was hardly any difference (57 and 62 per cent).

When analysis was carried out by distribu-

---

**Fig. 3.** Position of the electrode in the lead V₂ with relation to cardiac silhouette of the tele-roentgenogram is presented in a schematic way. Sectors L.V., L.A. and P.A. correspond to the levels of the left ventricle, left auricle and pulmonary artery on the left side of the heart. The open circles represent cases without infarction, solid circles represent cases with infarction, triangles represent the cases with normal hearts in vertical anatomical position and squares represent cases with normal in horizontal anatomical position.
Fig. 4. Electrocardiogram of a 72 year old white man three weeks after myocardial infarction. Typical history of infarction and characteristic serial electrocardiographic changes. Note a QS pattern in leads V₁ through V₄ and V₅₆, V₆₇, but an RS pattern in aVF.

...tion in plane quadrants the biggest difference between the two groups in both coordinate systems was found in the sagittal plane. Only 4 to 10 per cent of the group without infarction showed progression of the initial deflection into the posterior superior quadrant of this plane, while 38 to 50 per cent of the patients with infarction showed such progression. The differences in the frontal plane were smaller and in the horizontal plane, negligible. In three dimensional analysis the differences were even smaller. The largest difference between the groups with and without infarction was that the initial deflection of the group with no infarction was directed into the posterior left superior octant in only 4 to 10 per cent, while in the group with infarction the direction into this octant occurred in 29 to 31 per cent.

(b) Orientation of the entire QRS loop. Analysis of the characteristics of the loops showed that in both groups the loops were most frequently found in the left posterior and superior octants. The differences in distribution between the infarction and noninfarction groups were insignificant in either coordinate system. Posterior orientation of the loop was encountered more frequently in the tracings recorded with the cube co-ordinate system.

(c) Direction of rotation of the QRS loop. In the tetrahedron system, in the horizontal plane, counterclockwise rotation was encountered slightly more frequently (80 per cent) in the group with no infarction than in the group with infarction (64 per cent), but there was practically no difference between the findings in the two groups in the cube system. In the sagittal plane clockwise rotation was slightly more frequently encountered in the noninfarction group than in the infarction group (43 to 54 per cent) in both systems. The difference between the cube and tetrahedron systems consisted of complete absence of counter-
clockwise rotation in tracings of the noninfarction group taken in the cube system, while it was found in 23 per cent of the tracings in the same group taken with the tetrahedron system. Another difference was a more frequent finding of clockwise rotation in the group without infarction (83 per cent) in the cube system, as compared with the tetrahedron system (65 per cent).

(d) Irregularities and indentations of the QRS loop. In both systems perfectly smooth loops were observed somewhat more frequently in the group without myocardial infarction (26 to 40 per cent), than in patients with infarction (14 to 15 per cent). Marked irregularities were no more common in the group with infarction (22 to 23 per cent) than in the group with no infarction (13 to 30 per cent) in both systems. Nor was there any significant difference in the distribution of moderate irregularities between the two groups.

Comparison of the Findings in Low Precordial Leads with Findings in Lead aVF of the Electrocardiogram

In view of the finding pointing to the conclusion that precordial leads V₃ and V₄ recorded at the ensiform and epigastric levels showed very significant differences in the direction of the initial QRS deflection between the group with infarction and the group with no infarction, it appeared necessary to compare the findings in these leads with those in lead aVF. Lead aVF is the only standard unipolar lead recorded routinely in which the electrode is placed below the level of the standard precordial leads.

In our patients without infarction, the initial QRS deflection in aVF was positive in 24 out of 25 cases and coincided in 96 per cent with the initial QRS deflection of lead V₃₉p and in 92 per cent with the initial QRS deflection of lead V₃₂.

Out of the 21 cases of infarction with an initial negative QRS deflection in lead V₃₂, only 10 cases showed an initial negative QRS deflection in aVF. In the remaining 11 cases, in which the initial QRS deflection was positive in aVF, two cases showed an initial negative QRS deflection in all examined low precordial leads, five cases showed an initial positive QRS deflection only in leads V₄₀p (fig. 4), four cases in leads V₄₀p and V₅₀p. Leads from the umbilical and epigastric levels in the midline (leads V₀ and V₁₅₀ of Lambert) were recorded.

### Table 1.—Subdivision of 25 Cases Without Infarction

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>HHD</td>
<td>8</td>
</tr>
<tr>
<td>HHD &amp; severe kyphoscol</td>
<td>1</td>
</tr>
<tr>
<td>HHD &amp; bullous emphys.</td>
<td>1</td>
</tr>
<tr>
<td>Cor pulmon. emphys.</td>
<td>1</td>
</tr>
<tr>
<td>Cor pulmon. pulm. fibrosis</td>
<td>2</td>
</tr>
<tr>
<td>Cor pulmon. sarcoaid</td>
<td>1</td>
</tr>
<tr>
<td>Aortic sten. &amp; insuff.</td>
<td>4</td>
</tr>
<tr>
<td>Aortic &amp; mitral dis.</td>
<td>1</td>
</tr>
<tr>
<td>IV sept. defect.</td>
<td>1</td>
</tr>
<tr>
<td>Senile degener. HD.</td>
<td>1</td>
</tr>
<tr>
<td>Thyrotoxic HD.</td>
<td>1</td>
</tr>
<tr>
<td>Emphysema, no heart disease</td>
<td>1</td>
</tr>
<tr>
<td>No heart &amp; lung dis.</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Anatomic Diagnosis</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH</td>
<td>13</td>
</tr>
<tr>
<td>LVH?</td>
<td>3</td>
</tr>
<tr>
<td>LVH &amp; RVH</td>
<td>3</td>
</tr>
<tr>
<td>RVH</td>
<td>1</td>
</tr>
<tr>
<td>Diffuse cardiomeg.</td>
<td>1</td>
</tr>
<tr>
<td>Normal heart</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG Pattern</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBBB</td>
<td>3</td>
</tr>
<tr>
<td>&quot;LV strain*&quot;</td>
<td>11</td>
</tr>
<tr>
<td>LVH without T inversion*</td>
<td>3</td>
</tr>
<tr>
<td>RVH</td>
<td>1</td>
</tr>
<tr>
<td>Normal with deep S2-3</td>
<td>3</td>
</tr>
<tr>
<td>Normal</td>
<td>3</td>
</tr>
<tr>
<td>RBBB</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

* These cases had an absent Q wave in V₅ through 7, but QRS duration was less than 0.10 sec.

Abbreviations: HHD—hypertensive heart disease. LVH—left ventricular hypertrophy. RVH—right ventricular hypertrophy. LBBB and RBBB—left and right bundle branch block.
ventricle only in one-half of the normal patients and in one-sixth of the patients without infarction.

**Lead V₃:** Figure 3 shows in a semischematic way the relation of the electrode to the cardiac silhouette in all cases.

**Lead V₄:** The electrode was located over the ventricle (presumably the left) or the area outside of the heart shadow at the level of the ventricles in all examined cases in all groups. In the normal subjects with horizontal hearts the electrode was close to the apex, in the other three groups the electrode positions varied from a site over the apex to a site over the upper border of the ventricle.

**Leads V₅ and V₆:** In all cases of all groups the electrode was located outside of the heart shadow at the level of the ventricles.

**Analysis of Individual Cases**

Twenty-five patients with no infarction represented a variety of clinical and anatomic conditions and electrocardiographic patterns which are summarized in table 1. A representative case is illustrated in figure 5.

In one of the patients without infarction, it was noted that the QS deflection in V₂ gave way to an rS deflection during deep expiration. In another case without infarction a QS pattern in lead V₂ changed into rS after removal of 500 cc. of right-sided pleural effusion.

**Discussion**

The number of tracings with an absent R wave in lead V₂ in patients without infarction was surprisingly high. The exact number of screened electrocardiograms is unknown, but it can be estimated at 5000 to 6000 tracings, which would give an incidence of the discussed pattern of about 0.5 per cent of all electrocardiographic tracings.* Accordingly a QS or QR pattern in lead V₂ in the absence of myocardial infarction is not very uncommon. This is in agreement with the impression gained from the review of literature.

The incidence of a QS or QR pattern in lead V₄, in the absence of myocardial infarction,

---

* This number of electrocardiograms does not represent the number of patients since several tracings might have been recorded in the same individual.
was much less frequent and did not exceed 0.015 per cent. No instances of a QS or QR pattern in lead V₅, in the absence of myocardial infarction, were encountered in this study, although proven cases are on record.¹⁰

The most important result of this study appears to be the finding of a method which enables one to determine, in a great majority of cases, whether or not a QS or QR pattern in leads V₃ and V₄ is due to infarction. The method consists in the recording of additional electrocardiographic leads at the ensiform and epigastric level below the positions on the chest at which V₃ and V₄ are conventionally recorded. For practical purposes recording of only one additional unipolar precordial lead, V₃E (lead V₃ recorded at the level of the ensiform), is probably sufficient.

In all of the patients without infarction, the transition between the initial negative and the initial positive QRS deflection in the leads placed along the vertical axis on the anterior body surface, occurred below the level at which the conventional leads V₃ and V₄ are recorded. This implies that, in all cases without infarction with an absent R wave in leads V₃ and/or V₄, the initial QRS vector was directed downward, and the anatomical point of the origin of this vector was situated below the level of the standard leads V₃ and V₄. This was supported by the vectorcardiograms which showed an initial downward spread of excitation in 90 to 92 per cent of the QRS loops of the cases without infarction.

Most of our cases with infarction showed a Q wave in the lead V₅E. If one assumes that the Q wave in the precordial leads appears when the electrode faces the infarcted area, this finding indicates that the infarction in the above mentioned cases probably affects the inferior portion of the anterior heart wall. Among the 24 cases of infarction there was only one unequivocal case with a QS pattern in leads V₃ and V₄ in which an R wave was present in the leads recorded from positions below the conventional leads.

An electrocardiographic pattern of myocardial infarction in which QRS changes are present in standard precordial leads V₁ through V₄, and leads III and aVF, has been frequently encountered in the present study. This pattern has been described on numerous occasions:²⁷, ²⁷, ³¹, ³⁶, ³³, ²⁹, ³⁶, ³⁷, ⁴², ⁸, ¹, ⁴¹, ³² (these references are in chronological order) and designated as anteroseptal,²⁰, ³⁰ extensive infarction of septum involving the anterior and posterior wall,²⁰ anteroposterior,¹, ⁴², ⁷ and posteroinferior infarction.⁸

In two of our cases with such an electrocardiographic pattern, which came to autopsy, there was an occlusion of the descending branch of the left coronary artery with involvement of the anterior wall, inferior half of the septum in both and part of the posterior wall in addition in the other case.

**Value of High Precordial Leads**

Our study indicates that chest leads recorded from the levels above the standard precordial positions were of little value in differentiation between infarction and noninfarction patterns. The occurrence in normal subjects of a QS pattern in high right precordial leads¹, ⁸, ⁴₈ was confirmed by us. In normal persons with hearts in a horizontal position, we also found a QS pattern in leads V₃ and V₄ taken at the level of the second intercostal space.

High precordial leads have been utilized for diagnosis of myocardial infarction.³⁰, ⁴₃-⁴₅, ¹, ⁵ Our study leads to the conclusion that in the majority of instances a Q wave in high precordial leads does not necessarily indicate myocardial infarction. In contrast to cases without infarction, a significant percentage of our patients with infarction (28 to 40 per cent) showed the presence of an initial R wave in leads V₁ and V₅ made one to two intercostal spaces above the standard level.

**Absence of an Initial R Wave in Some Precordial Leads in the Presence of an R Wave in Leads made from Positions to the Right of these Leads**

Such patterns, as well as progressive diminution of R from right to left precordial positions, have been considered to be suggestive of anterior or anteroseptal myocardial infarction.², ¹⁸, ³³ It has been noted, however, that in cases of right ventricular dilatation without myocardial infarction, an R wave may be present in lead V₁ and either diminish in size or disappear in transitional leads toward the left precor_
In our cases with QS or QR pattern in leads V3 and V4, an initial R wave was present in leads V3R, V1, and occasionally in V2 in one-third of the patients with infarction and in almost one-fourth of the cases without infarction. This indicates clearly that this pattern is not specific for myocardial infarction. One or two of our cases without infarction, with the described pattern, might have had dilatation of the right ventricle but the majority had clinically and electrocardiographically an uncomplicated left ventricular hypertrophy.

Direction of the Initial QRS Deflection in Absence of Infarction

(a) Transverse Axis: In 14 of our cases without infarction, the initial deflection of QRS was caused by forces directed from right to left. Only three of these cases had a QRS duration exceeding 0.12 second, while in the remaining cases there was a left ventricular “strain” pattern with a QRS duration of 0.08 to 0.10 second.* In the remaining 11 cases of absent myocardial infarction, the initial QRS deflection was caused by forces directed from left to right, though the transition between the initial positive deflection on the right side and the initial negative deflection on the left side was not found in the standard precordial leads, but in the leads made from a site below the level of the standard precordial leads.

(b) Vertical Axis: In the cases of left ventricular hypertrophy selected at random, the initial deflection of QRS was caused by forces directed downward in 7 out of 10 cases. In 25 cases without infarction with an initial negative deflection of QRS in V3 and V4, the initial deflection of QRS was caused by forces directed downward in all cases as indicated by the results of the exploration and in 92 per cent as indicated by vectorcardiograms. The majority of these cases consisted of cases of left ventricular hypertrophy. Whether the development of left ventricular hypertrophy causes the vector of the initial deflection of QRS to assume a more downward direction in all cases remains to be proven. However, such a shift appears to be probable because of our observation of a progressive diminution of the size of the initial R wave in the right precordial leads in several cases in which serial tracings over a period of many years were observed.

Causes of Negative Initial Deflection of QRS in the Standard Precordial Leads in the Absence of Myocardial Infarction or Other Conditions in Which a Part of the Myocardium is Dead or Electrically Inactive

These may be the following: (1) atypical spread of excitation, e.g., the precordial electrode faces the same portions of the heart as in normal persons, but the electrical forces have changed their direction; (2) normal ventricular excitation but an altered position of the precordial electrode, e.g., the electrode faces such a portion of the heart in which the initial QRS deflection is normally negative.

(1) Atypical Spread of Excitation. A negative initial deflection of QRS in the precordial leads can be due either to the presence of an initial negative deflection instead of a positive one or to an absence of the normal positive deflection. The latter concept has been advanced in order to explain a QRS pattern in the transitional leads. It has been postulated that the initial deflection of QRS is nearly perpendicular to the axis of the exploring electrode and thus not recorded at all.4,10,1,5 Such a situation has been attributed to the depolarization of both sides of the septum at the same time and thus to cancellation of opposite vectors derived from a septal activation.10 If this were true, the initial deflection of QRS in the precordial leads from the transitional leads displaying QS pattern would be isoelectric. The results obtained in this study show that the beginning of QRS complex occurred at the same time in the leads with a QS or QR pattern as in other synchronously recorded precordial leads. Accordingly, the concept of an initial isoelectric deflection of QRS cannot be used to explain an initial negative QRS deflection in our cases.

The initial deflection of QRS in the right and in the transitional precordial leads may be negative if the spread of excitation is directed...
from right to left which presumably takes place in high left bundle branch block. Our non-infarction cases with an initial negative QRS deflection in leads V3 and V4 included only 14 cases in which the initial deflection of QRS was considered to be caused by forces directed from right to left while in the remaining 11, the initial deflection of QRS was caused by forces directed from left to right. Therefore, it appears to be doubtful whether one can interpret an electrocardiographic deflection in the unipolar chest leads without taking into consideration the vertical components of the cardiac voltages.

Our findings indicate that in all cases without infarction the initial QRS vector was directed inferiorly. This explains the negative initial deflection of QRS in all leads recorded from sites above the anatomical point of the origin of this vector and the positive initial deflection of QRS in all leads made from sites below this point, regardless of whether the downward spread has a right-to-left direction, a left-to-right direction or is vertical. This explanation holds if the initial QRS vector in all such instances is directed anteriorly.

For the sake of completeness, one has to mention some other concepts concerning the same problem. An initial Q wave in right precordial leads has been attributed to congenital variation in the distribution of conduction fibers. An absent R wave in precordial leads in certain cases of right and left ventricular hypertrophy has been attributed to a decreased density of the junctions between Purkinje fibers and ordinary muscle as a result of dilatation of the affected chamber. It has been postulated recently that absence of an initial R wave in right precordial leads in cases of left ventricular hypertrophy is due to a posterior spread of the initial deflection of QRS as a result of stretching and bowing of the inflow tract of the left ventricle.

(2) Change in position of the electrode in relation to the heart. The negative initial deflection of QRS in the standard precordial leads cannot be satisfactorily explained without taking into consideration this second factor. This can theoretically occur either because of a change of heart position with relation to the electrode or because of a change of the electrode position with relation to the heart. The last factor may play some role in certain chest deformities in which the upper ribs anteriorly are closer together than normally, thus making the intercostal spaces narrower and the position of the standard electrodes higher than normal. In one of our cases such a situation was believed to be present. The factor of change of the position of the heart with relation to the electrodes appears to be of more practical importance. Pardee has demonstrated on x-ray films of six individuals that the electrode in leads V2 and V3 in the sixth intercostal space lies over the ventricles more frequently than in standard leads V2 and V3 which overlay supraventricular structures. Figure 10 of reference 19 shows the x-ray film of a patient with right ventricular hypertrophy in whom the position of the V1 electrode is close to the pulmonary artery segment. On the other hand, fairly numerous postmortem determinations in which the electrode position was correlated with the heart position have demonstrated the position of the V3 electrode to be near the interventricular septum in persons with normal hearts, to the right of the septum in patients with left ventricular hypertrophy, and to the left of the septum in subjects with right ventricular hypertrophy. The horizontal level of the electrode in the last study was not mentioned, but the illustrated variations of position appear to be of considerable magnitude. Six teleroentgenograms in normal students were made by Kossman and Johnston and in one illustrated case electrode V3 overlies the lower part of the left ventricle.

The results of our x-ray study support the opinion of Pardee that in order to have the electrode closer to the ventricles one has to record leads V2 and V4 at lower levels than the present standard level used for these leads. It is difficult to establish with any degree of accuracy whether the absence of an initial R wave in our cases without infarction was due in an appreciable number of cases to a high electrode position with relation to the heart. The comparison with the small control group of normal persons, which show a similar electrode position in relation to the heart, suggests that this is not a crucial factor. It appears to us that the
low position of the anatomic point of the origin of the initial QRS vector was of greater importance as a factor producing the QS deflections in the precordial leads than the low position of the diaphragm or other changes of the anatomic position of the whole heart.

Correlation Between Vectorcardiographic Loops and Scalar Electrocardiographic Patterns Obtained With the Chest Leads

In view of the many theoretical and practical difficulties of vectorcardiography, it is not surprising that the vectorcardiogram did not differentiate between patients with and those without myocardial infarction. The initial deflection of the QRS complex corresponds to the first 0.02 second or more of the QRS sE-loop. In many of the photographs, the white spot made by the P and T waves is sufficiently large to cover a portion of the QRS loop. This is often a significant factor as proved by a count of the time dots in the same loop in different planes. The QRS duration varied at times as much as 100 per cent in the three planes. It is, therefore, frequently impossible to be sure that the time dots interpreted to be the recording of the potential of depolarization in the first 0.02 second are actually recorded at that time. In general, the vectorcardiogram correlated poorly with leads V3 and V4, frequently showing an anterior direction of the initial QRS vector when no initial R waves were recorded in these scalar leads. Although this anterior direction of the initial QRS vector occurred much more frequently in the absence of infarction, the correlation with the clinical findings was not sufficiently good to be of differential diagnostic importance. On the other hand, correlation of vector loops with high and low precordial leads was very much better. Inferior direction of the initial QRS vector was usually seen in patients with initial R waves in leads made from sites below the standard positions, and superior direction of the initial QRS vector in patients with initial R waves in high chest leads. This better correlation of the y axis of the vector and scalar electrocardiogram may be due to the lesser skewing of this axis by cardiac eccentricity.* None of the other frequently mentioned signs of infarction (irregularity of the loop, change in rotation of the QRS sE) significantly differentiated between the subjects of the infarction and noninfarction groups.

The differences between the loops inscribed by the cube and the tetrahedron coordinate systems were often considerable. Nevertheless, there was no very significant difference between them in the ability to differentiate patients with from patients without infarction.

Summary

1) A QS pattern or a significant Q wave in the lead V3 was found in 25 patients in whom myocardial infarction was considered to be absent (in five patients the findings were proved at autopsy) and in six patients in whom myocardial infarction was considered to be unlikely. A similar QRS pattern in lead V4 was found in only three of these patients. The majority of the patients in this group had left ventricular hypertrophy.

2) The initial QRS deflections of the electrocardiogram and the vectorcardiogram of the group of patients with a QS or QR pattern in leads V3 and V4 who had no infarction and of a group of patients with infarction who had a similar QRS pattern were compared. The electrocardiogram included 26 additional chest and abdominal unipolar leads. The vectorcardiograms were recorded by means of the tetrahedron and cube reference systems.

3) The differences between the groups of patients with and without infarction with regard to the direction of the initial QRS deflection and the features of the vectorcardiographic QRS sE loop are discussed. The most significant differences between the group with infarction and the group without infarction were found in the low chest leads V3 and V4 recorded at the ensiform and the epigastric levels. Lead V3 made at level of ensiform (V3E) showed the greatest difference: an initial R wave was present in 24 out of 25 cases without infarction and in only 3 out of 24 cases with infarction. Thus, the direction of the initial QRS deflection in lead V3E differentiated in 84 per cent patients with infarction from those without infarction.
even when the standard lead V₃ showed the same QRS pattern in both groups of patients.

(4) In chest leads made from sites above the standard level, the QRS pattern was not significantly different in the group with and the group without infarction, although the presence of an initial R wave in the high leads occurred more commonly in patients with infarction.

(5) No vectorcardiographic feature differentiated the infarction and noninfarction groups in a significant number of cases. The greatest difference between the two groups concerned the initial 0.02 second of the QRS loop, which was directed inferiorly in 90 to 92 per cent of the patients without infarction and in only 36 to 46 per cent of those with infarction.

(6) Fifty per cent of patients with infarction who had a Q wave in the low chest leads made at the ensiform or epigastri level showed an initial R wave in lead aV₂.

(7) The teleroentgenograms recorded in the supine position with electrode positions marked on the chest revealed that the electrode for lead V₃ faced the level of the ventricles in only 5 out of 18 patients without infarction who showed a QS pattern in leads V₃ and V₄. In the remaining 13 cases, the electrode (V₃) faced higher levels of the heart. However, the position of the electrodes with relation to the cardiac silhouette was fairly similar in a control group of five patients with infarction and five normal persons with a vertical anatomical heart position. In a group of five normal subjects with horizontal anatomical heart position, the electrodes faced generally lower portions of the heart shadow than in the other groups.

(8) The causes of an initial negative QRS deflection in the absence of myocardial infarction are discussed. The inferior direction of the initial QRS vector and the low location of the point of origin of this vector rather than the low position of the whole heart appeared to be responsible for the absent initial R wave in leads V₃ and V₄ in our patients without infarction.

(9) The consideration of the vertical components of the cardiac voltages may be useful in explanation of the electrocardiographic patterns in the unipolar chest leads.

**Summario in Interlingua**

Un configuration QS o QR in le absentia de infarctamento myocardial es frequentemente presente in le derivation V₃ e a vices in le derivation V₄. Un exploration per medio de multiple derivationes unipolar e vectorcardiogrammas del thorace e abdomine revelava que in quasi omne tal casos le vector del portion initial del complexo QRS exhibi un direction in basso. Consequentemente, in le absentia de infarctamento, patientes monstrante iste configuration exhibiva quasi invariabilmente un unda R initial in le derivationes obtenite ab positiones infra le nivello standard pro V₃ e V₄. Le grande majoritate del casos de infarctamento myocardial con simile configurationes QRS monstrava un unda Q in le derivationes inferior. Le consideration de componentes vertical de voltages cardiac es possiblemente de adjuta in le interpretation del derivationes precordial.

**Acknowledgment**

The authors wish to express their gratitude to Dr. Eugene Lepeschkin of Burlington, Vermont for his very helpful criticism, valuable suggestions and review of the paper; to Dr. Harold Braun for his helpful criticism; to Dr. Herbert W. Copelan for his assistance in the working up of several cases; and to the staff of the x-ray department of the Philadelphia General Hospital for their very kind cooperation.

**REFERENCES**


10 Myers, G. B.: QRS-T patterns in multiple precordial leads that may be mistaken for myocardial infarction. II. Right ventricular hypertrophy and dilatation. Circulation 1: 800, 1950.


QS- and QR-Pattern in Leads V3 and V4 in Absence of Myocardial Infarction: Electrocardiographic and Vectorcardiographic Study
BORYS SURAWICZ, ROBERT G. VAN HORNE, JOHN R. URBACH and SAMUEL BELLET

Circulation. 1955;12:391-405
doi: 10.1161/01.CIR.12.3.391

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1955 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/12/3/391

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at: http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at: http://circ.ahajournals.org//subscriptions/