Clinical Significance of QS Complexes in V₁ and V₂ without Other Electrocardiographic Abnormality

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Background: In the absence of other electrocardiographic (ECG) abnormalities, QS deflections simultaneously in both of the leads V₁–V₂ may have multiple possible causes. Despite much information in the literature indicating that this is an unlikely pattern for pure septal infarction, such an ECG diagnosis is frequently given.

Methods: Ninety-nine cases having QS deflections in both leads V₁ and V₂ but no other ECG abnormality were compared to 99 other patients with entirely normal ECGs, to whom they were matched by age, gender, and the presence or absence of septal Q waves. Retrospective analysis of medical records was performed to determine the nature of any cardiovascular disease in these two groups, and to find a possible explanation for the ECG abnormality.

Results: Because of its intermittence in subjects with multiple ECGs, QS deflections in leads V₁–V₂ appeared most often to be an artifact of precordial lead placement. Prior myocardial infarction, or presence of clinical coronary disease was present in only about 20% of the cases. Neither the intermittence of Q wave in V₂ on repeated ECGs nor the absence of septal Q waves was useful in distinguishing between those with and without coronary heart disease.

Conclusions: This ECG pattern is a sign of prior myocardial infarction in only a minority of cases, and in the latter, infarction limited to the interventricular septum is exceptional. This ECG finding should be interpreted as a nonspecific QRS abnormality with multiple possible causes. Clinical correlation and repeat tracings with attention to lead placement will help to clarify its significance.

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septal infarction; right precordial QS complexes; precordial lead placement

In the absence of other electrocardiographic (ECG) abnormality, the presence of QS deflections simultaneously in both leads V₁ and V₂ is an abnormality of uncertain clinical significance. From a vectorcardiographic perspective, this pattern suggests a posterior orientation of initial QRS forces, an important cause of which could be myocardial infarction (MI) underlying these leads.¹ Septal, or mid-septal infarction is an ECG diagnosis that has been used.^{2–4} However, there is much evidence to indicate that chronic infarction *limited* to the interventricular septum (IVS) is rare, often electrically silent, affects the presence of Q waves in leads I, V₅, and V₆ ("septal" Q waves) unpredictably, and rarely produces right precordial Q waves.^{2,5–9} The latter, when due to infarction, indicate involvement of the anterior wall or apex of the left ventricle.¹⁰ In the absence of incomplete or complete left bundle branch block (LBBB), other possible causes of

QS deflections in leads V₁–V₂ include improper precordial lead placement,^{11,12} left ventricular hypertrophy (LVH),^{11,13,14} unusual chest conformation,¹² congenital anomalies associated with ventricular inversion or dextrocardia,¹⁵ emphysema,¹³ and an intraventricular conduction defect involving the median or septal fibers of the left bundle branch (left septal fascicular block).^{16,17}

Some commonly used software programs for ECG interpretation continue to diagnose "septal infarction" when QS deflections are present leads V₁–V₂,¹⁸ and in my experience, physician overreaders usually confirm such an interpretation. The following study was undertaken in an attempt to clarify the clinical significance of such an ECG pattern in tracings with no other abnormality, and to determine what may be a more accurate and clinically useful interpretation of such ECGs in the setting of a major university medical center.

METHODS

All 12-lead ECGs processed in the UCLA Medical Center ECG Laboratory between January 1, 2001 and December 31, 2002 were reviewed. About two-thirds of these were from outpatients, and one-third from inpatients. Cases were excluded from the study if the sentinel tracing showed the presence of other abnormality, especially *other* abnormal Q waves, incomplete bundle branch block; QRS duration > 100 ms; any LVH voltage criterion; mean frontal plane QRS axis > +90° or < -29°; any P, ST-T, or QT abnormality, or rhythm disorder; PR interval > 210 ms; or technical inadequacies. Age less than 11 years, insufficient clinical information in the medical record, or patient duplication also caused exclusion. Absence of septal Q waves, i.e., simultaneous absence of Q waves in leads I, V₅, and V₆, was not considered an abnormality for the purposes of this study.^{1,19} From about 40,000 tracings, 99 were identified that were normal except for the presence of QS deflections simultaneously in leads V₁ and V₂. Each of these 99 cases was matched by age, gender, and presence or absence of septal Q waves with a patient selected from the same large cohort because of an entirely normal ECG.

The resulting 198 cases were subdivided into the following groups: QSV₁₋₂-NSq indicates 62 cases with QS complexes in V₁ and V₂ but absent septal Q waves; QSV₁₋₂-Sq indicates 37 cases with QS complexes in V₁ and V₂ but with septal Q waves present; WNL-NSq indicates the 62 cases with normal ECGs but absent septal Q waves that were matched with cases in the QSV₁₋₂-NSq group; WNL-Sq indicates the 37 cases with normal ECGs including the presence of septal Q waves that were matched with cases in the QSV₁₋₂-Sq group. QSV₁₋₂-TOT indicates the entire group of 99 cases having QS complexes in V₁ and V₂, while WNL-TOT indicates the entire group of matching 99 cases with normal ECGs.

Retrospective analysis of medical records was made in all cases to determine the presence and nature of any cardiovascular disease, or other clinical characteristics that might affect the ECG. Cardiovascular risk factors were defined as presence of: documented history of hypertension requiring treatment or blood pressure measured at greater than 140 mmHg systolic or 90 mmHg diastolic; diabetes mellitus; active cigarette smoking within the preceding year; total cholesterol level > 200 mg/dL or history of elevated chole-

sterol requiring treatment. Comparisons of clinical and electrocardiographic characteristics between groups were performed. In analyzing the ECGs, a continuous recording of lead II, simultaneous with the other leads, provided a timing reference. Continuous variables were compared using ANOVA. Incidences amenable to expression in 2 × 2 tables were compared using a chi-square test, or a 2-tailed Fisher's exact test when numbers were small. Significant difference was set at P < 0.05. This protocol had the approval of the UCLA institutional review board.

RESULTS

The 99 cases with QS deflections in V₁ or V₂ but with no other abnormality represented about 0.2% of all the ECGs from which they were selected, and 24% of all 420 cases with QS in V₁ and V₂ from the same large cohort. An example is shown in Figure 1. Some clinical characteristics of the cases are summarized in Table 1.

Age

Compared to a large group of cases with entirely normal ECGs selected from the same patient population, patients in the QSV₁₋₂-TOT group were on average older: proportions of cases above 50-years old were, respectively, 1075 of 1894 (57%) versus 69 of 99 (70%), P < 0.02. The ratio of patients with QS deflections in leads V₁-V₂ without any other abnormality to those with entirely normal ECGs increased from 0.03 in patients under 40 years of age, to 0.06 in those with ages between 40-70 years, and to 0.13 in cases with ages over 70 years. These findings are consistent with this ECG abnormality being acquired in many cases.

Gender

The preponderance of women with isolated QS in V₁-V₂ was significantly greater in the QSV₁₋₂-TOT group than in a large randomly sampled group with entirely normal ECGs from a similar population at the same institution: 67 out of 99 cases (67.7%) versus 1160 out of 2042 cases (56.8%), respectively, P < 0.05.¹⁹ The ratio of women to men was particularly high in the QSV₁₋₂-NSq group, which was significantly greater than that in the QSV₁₋₂-Sq group.

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Figure 1. ECG of a 45-year-old woman with morbid obesity. She had hypertension but no other history of cardiovascular symptoms or disease. Radionuclide myocardial perfusion scan was normal. Tracing was interpreted as “septal MI of uncertain age.”

Height, Weight, and Body Mass Index

Cases with normal ECGs that had septal Q waves were on average taller than those without septal Q waves, and weighed more and had a higher body mass index (BMI) than those in the QSV₁₋₂-Sq group. Those in the WNL-NSq group had a greater BMI than those in the QSV₁₋₂-NSq group. For the total groups, cases with normal ECGs weighed more and had a greater BMI than those cases without R waves in V₁ and V₂. These differences were all significant.

Incidence of Cardiovascular Risk Factors

For the various groups there were a few risk factor differences that reached the minimum level of statistical significance. Incidences of current cigarette smoking were higher in the QSV₁₋₂-Sq group compared to the WNL-Sq group, and in the QSV₁₋₂-TOT group compared to the WNL-TOT group. A high total cholesterol level was more common in the WNL-TOT group than in the QSV₁₋₂-TOT group. In the latter two groups, there was no statistically significant difference in the proportion of cases having the presence of either docu-

mented coronary disease or two cardiovascular risk factors.

Presence of Coronary or Other Cardiovascular Disease

There was a trend for greater incidence of clinically apparent coronary disease in patients with QS in V₁₋₂ than in those with entirely normal ECGs, but this did not reach a level of statistical significance. The difference was not of a magnitude likely to be useful in ECG interpretation. Similarly, there were no significant differences between groups with regard to incidences of cases without coronary disease but with some other cardiovascular disorder, or of cases with either CAD or some other cardiovascular disorder.

Incidence of Factors Predisposing to QS in V₁-V₂

Cases were analyzed for the presence of clinical abnormalities that might predispose to the presence of QS deflections in V₁-V₂. The following were considered such abnormalities: coronary or hypertensive heart disease; LVH of any other cause; pulmonary emphysema; morbid obesity; marked chest

Table 1. Clinical Characteristics of Cases

	QSV ₁₋₂ -NSq	WNL-NSq	QSV ₁₋₂ -Sq	WNL-Sq	QSV ₁₋₂ -TOT	WNL-TOT
Age (years)	60.6 ± 19.0 (n = 62) ^a	60.5 ± 18.7 (n = 62) ^a	63.1 ± 18.8 (n = 37) ^a	62.9 ± 18.6 (n = 37) ^a	61.5 ± 18.8 (n = 99) ^a	61.4 ± 18.6 (n = 99) ^a
Gender (M/F)	15/47	15/47	17/20 ^{§1}	17/20	32/67	32/67
Height (CM)	164.7 ± 11.2 (n = 50)	163.7 ± 8.6 (N = 50)	166.9 ± 10.7 (n = 30)	170.2 ± 12.2 ¹ (n = 28)	165.5 ± 11.0 (n = 80)	166.1 ± 10.4 (n = 78)
Weight (kg)	68.9 ± 18.9 (n = 58)	71.1 ± 17.6 (n = 62)	64.6 ± 12.4 (n = 35)	73.4 ± 16.0 ^{‡2} (n = 36)	67.0 ± 17.3 (n = 93)	71.9 ± 17.0 ^{¶1} (n = 98)
BMI	24.3 ± 4.6 (n = 50)	26.6 ± 6.9 ^{†1} (n = 50)	23.0 ± 3.8 (n = 30)	25.9 ± 4.3 ^{‡2} (n = 28)	23.8 ± 4.4 (n = 80)	26.3 ± 5.4 ^{¶2} (n = 78)
Cases with HTN	37	30	19	15	56	45
Cases of diabetes	10 (n = 60)	15	9	4	19 (n = 97)	19
Smokers	8 (n = 60)	5 (n = 59)	9 ^{‡1} (n = 35)	2 (n = 36)	17 ^{¶1} (n = 95)	7 (n = 95)
Elevated cholesterol	23 (n = 42)	30 (n = 46)	10 (n = 31)	18 (n = 31)	33 (n = 73)	48 ^{¶1} (n = 77)
Cases with CAD	9	8	10	4	19	12
No CAD, but some CVD	13	14	6	4	19	18

Values of continuous variables are expressed as mean ± standard deviation. BMI = body mass index; CAD = coronary artery disease; CVD = cardiovascular disease; HTN = hypertension; QSV₁₋₂-NSq = group with QS in V₁₋₂ and no septal Q waves; QSV₁₋₂-Sq = group with QS in V₁₋₂ and septal Q waves present; QSV₁₋₂-TOT = total group with QS in V₁₋₂; WNL-NSq = group with normal ECGs and no septal Q waves; WNL-Sq = group with normal ECGs and septal Q waves present; WNL-TOT = total group with normal ECGs.

^a(n) in this row represents total number of cases in this group. If data collection was incomplete, in other rows the number of cases for which data was available is specified.

Statistically significant differences between groups are indicated by two elements: (1) a symbol identifying the groups being compared:

†QSV₁₋₂-NSq vs. WNL-NSq,

‡QSV₁₋₂-Sq vs. WNL-Sq,

§QSV₁₋₂-NSq vs. QSV₁₋₂-Sq,

||WNL-NSq vs. WNL-Sq,

¶QSV₁₋₂-TOT vs WNL-TOT,

and (2) a superscript number indicating the degree of statistical significance of the difference found:

1 = P < 0.05; 2 = P < 0.01; 3 = P < 0.001.

wall deformity; chronic mediastinal shift; prior cardiac surgery, including orthotopic heart transplantation; and documented left septal fascicular block. One or more of these factors were present in 28 of the 62 patients (45%) of the QSV₁₋₂-NSq group, but in only 17 of the 62 cases (27%) of the WNL-NSq group, P < 0.05. One or more of these factors was present in 18 of 37 patients (49%) of the QSV₁₋₂-Sq group, but only 9 of 37 cases (24%) of the WNL-Sq group, P < 0.05. The incidences in the combined QSV₁₋₂-TOT and WNL-TOT groups were, respectively, 46 of 99 cases (46%) and 26 of 99 cases (26%), P < 0.01.

ECG Characteristics

Some ECG characteristics are summarized in Table 2. Absence of an initial R wave in V₁-V₂ is not

strictly synonymous with a QS deflection in those leads, as the initial QRS forces could be isoelectric.¹¹ This latter condition was present in only 6 of the 99 cases with "QS" deflections in V₁-V₂. There was an absence of septal Q waves in cases with QS in V₁-V₂ much more frequently than in cases with normal ECGs derived from a similar population (62 out of 99 cases or 63% vs 95 of 1535 cases or 6.2%, P < 0.0001).¹⁹ As judged by the polarity of the initial deflection in lead aVF, the direction of the initial 10-ms QRS vector was inferior in a much greater proportion of cases with QS in V₁-V₂ than in cases with entirely normal ECGs. Counterclockwise rotation of the QRS loop in the frontal plane was also much more common in cases with QS in V₁-V₂ than in those with normal ECGs. These latter two very significant differences were present independently of whether septal Q waves were present.

Table 2. ECG Characteristics of Cases

	QSV ₁₋₂ -NSq	WNL-NSq	QSV ₁₋₂ -Sq	WNL-Sq	QSV ₁₋₂ -TOT	WNL-TOT
No. of cases	62	62	37	37	99	99
Initial inferior QRS direction	50† ¹	37	28‡ ²	15	78¶ ³	52
CCW frontal plane QRS loop	34† ¹	21	26‡ ¹	17	60¶ ²	38
Mean frontal QRS axis (°)	29.2 ± 35.3	37.6 ± 30.4	26.1 ± 32.6	35.0 ± 26.2	28.0 ± 34.2	36.6 ± 28.8
Mean frontal T wave axis (°)	43.2 ± 26.7	42.1 ± 19.2	44.2 ± 20.7	44.2 ± 16.8	43.6 ± 24.5	42.9 ± 18.3
Angle between QRS and T axes (°)	14.0 ± 32.0	5.3 ± 25.4	18.1 ± 31.1	9.3 ± 24.8	15.6 ± 31.6¶ ¹	6.8 ± 25.1
Inverted T in V ₁	39	35	26	22	65	57

Values of continuous variables are expressed as mean ± standard deviation. CCW = counterclockwise; QSV₁₋₂-NSq = group with QS in V₁₋₂ and no septal Q waves; QSV₁₋₂-Sq = group with QS in V₁₋₂ and septal q waves present; QSV₁₋₂-TOT = total group with QS in V₁₋₂; WNL-NSq = group with normal ECGs and no septal Q waves; WNL-Sq = group with normal ECGs and septal Q waves present; WNL-TOT = total group with normal ECGs.

Statistically significant differences between groups are indicated by two elements: (1) a symbol identifying the groups being compared:

†QSV₁₋₂-NSq vs WNL-NSq,

‡QSV₁₋₂-Sq vs. WNL-Sq,

¶QSV₁₋₂-TOT vs. WNL-TOT,

and (2) a superscript number indicating the degree of statistical significance of the difference found:

1 = P < 0.05; 2 = P < 0.01; 3 = P < 0.001.

Although mean frontal plane QRS and T wave axes did not differ significantly between the patient groups, the angle between the QRS and T-wave axes was definitely greater in the patients with QS in V₁-V₂ compared to those with an entirely normal ECG.

Intermittence of QS in V₂

A major characteristic of cases having QS in V₁₋₂ was intermittence of the QS in V₂ (Fig. 2). When more than one ECG was available for comparison, this occurred in 49 of 77 (63.6%) cases. When an initial R wave intermittently appeared in V₂, it was usually not accompanied by appearance of an R in V₁. The presence or absence of septal Q waves appeared to have no relation to whether initial R waves in V₂ were intermittently present in multiple ECGs. Five cases were found in which clinical features and serial ECGs documented that the QS in V₁-V₂ were permanently acquired due to a pathologic process: acute MI in two cases; severe emphysema, hypertensive LVH, and left septal fascicular block in one case each. Figure 3 illustrates such a case in a patient with myotonic dystrophy, in which loss of septal Q waves and appearance of QS in V₁-V₂ was probably due to the development of left septal fascicular block. Cardiac involvement in my-

otonic dystrophy is frequently associated with the development of intraventricular conduction abnormalities such as fascicular or bundle branch blocks.^{20,21}

Of the 36 cases in the QSV₁₋₂-NSq group having more than one ECG and also one or more factors predisposing to the presence of QS in V₁-V₂, an R wave was intermittently present in lead V₂ in 19 cases (53%). As there were no clinical events or other evidence of altered ventricular activation to explain the intermittence of R wave in V₂, this suggests that right precordial lead placement was important in the presence or absence of R waves in V₂ in these cases.

Results of Myocardial Imaging

Ninety-two patients had prior echocardiographic, angiographic, or nuclear perfusion imaging bearing on the presence or absence of prior MI. Those cases with QS in V₁-V₂ were more likely to have imaging evidence of MI than cases with entirely normal QRS (16 of 58 cases or 27.6% vs 4 of 37 or 10.8%, P < 0.05). They were also more likely to have documented either clinical or imaging evidence of MI (19 of 58 or 32.8% vs 7 of 37 or 18.9%, P < 0.05). In the 16 cases in the QSV₁₋₂-TOT group showing either a regional wall motion abnormality or myocardial

Figure 2. Same patient as in Figure 1. Lead V₂ from multiple ECGs over a period of 7 years showed QRS morphology varying from QS to qR to rS. Interpretations of ECGs ranged from “septal infarction” to “within normal limits.”

Figure 3. Serial ECGs in a 47-year-old (in 2002), premenopausal woman with myotonic muscular dystrophy, which had been diagnosed at age 13 years. She had no symptoms or signs of cardiovascular disease. There was evolution from prominent septal Q waves, to absent septal Q waves with QS in V₁ and V₂, without evidence of change in mean QRS axis or duration, or ventricular activation time. This latter pattern was present on all tracings after 1994. This is consistent with development of left septal fascicular block.

perfusion scan defect at rest, the IVS was involved in only 6 cases, and the abnormality was *limited* to the IVS in only 1 case. In the cases having multiple ECGs in the QSV₁₋₂-TOT group, cases with myocardial imaging evidence of prior infarction were no less likely to have intermittent appearance of an R wave in V₂ than were patients without clinically apparent cause for QS in V₁₋₂ (12 of 15 cases vs 22 of 38 cases, respectively, $P > 0.17$).

Importance of Additional ECG Abnormality

To estimate the effect of an additional ECG abnormality on the specificity of the finding of QS in leads V₁-V₂, 12 cases were studied that had late, relatively symmetrical T-wave inversions in one or more precordial leads, including lead V₂

(designated as group QSV₁₋₂-Tinv). Evidence of MI was present in 7 of these 12 (58%) cases, which is significantly more frequent than was found in the cases having QS in V₁-V₂ without any other abnormality (19 out of 99 or 19%, $P < 0.01$). An exception to this was encountered in a group of patients with a unique appearance of right precordial complexes. Eleven cases were found with QS deflections and late T-wave inversion limited to leads V₁ and V₂, but with P wave inversions in the same leads, giving P-QRS-T complexes that greatly resembled those of lead aVR (Fig. 4). This suggests a high placement of those precordial leads, and the intermittence of QS and T inversion in V₂ shown in Figure 4 is consistent with this.¹² The incidence of MI in this group was only 1 out of the 11 cases or 9%, which is less than was found in the above QSV₁₋₂-Tinv group ($P < 0.05$), or in the QSV₁₋₂-TOT group ($P < 0.01$).

Figure 4. ECG of a 74-year-old woman with no evidence of cardiovascular disease. On May 9, 2002 left ventricular size and wall motion were judged to be normal by radionuclide cardiac blood pool scan. On bottom row, lead V₂ from multiple ECGs over a 10-year period showed varying morphologies from QS to rS, suggesting changes due to varying right precordial lead placement.

DISCUSSION

Limitations of the Methods

The retrospective nature of the data collection resulted in some incompleteness of data available. Patients were not available to test the effects of precordial lead placement on their ECG pattern. Myocardial imaging of one sort or another was available less often in patients with an entirely normal ECGs than in cases with QS in V₁-V₂. The presence of the latter ECG abnormality itself undoubtedly prompted a more thorough cardiac work-up in those patients, thereby eliciting a greater incidence of an otherwise not apparent coronary heart disease than was diagnosable in patients with entirely normal ECGs. In vivo imaging methods available in this study may not be sensitive enough to detect small infarcts of the IVS.²² Numbers of cases in most of the groups were too small to give much statistical power to negative findings. The findings in this study may not be applicable to ECGs from other types of populations.

Implications of Findings

QS deflections limited to lead V₁ can be seen in some normal ECGs, and are more common when initial QRS forces are directed inferiorly and to the left.^{13,19} When an obvious intraventricular conduction delay is absent, QS deflections present simultaneously in both leads V₁ and V₂ have been said to suggest septal MI.²⁻⁴ However, the present study is consistent with the work of others showing that this pattern has multiple other possible causes and is an unreliable indicator of MI.^{10,11,13,14,17} When it does exist in the setting of MI, it usually indicates involvement of the anterior wall or apex of the left ventricle, the IVS itself is involved in a minority of cases, and the infarction is rarely *limited* to the IVS.^{10,22} Infarction limited to the IVS is often not apparent on the 12-lead ECG, and in some cases may involve only loss of septal Q waves.^{5,8,10,23,24} Since the absence of the latter is a not rare variant of normal, and can be acquired in conditions such as important LVH or left septal fascicular block (Fig. 3),^{1,14,17} the diagnosis of MI limited to the IVS is usually not possible from a single ECG, and requires comparison with other tracings and usually also correlation with other clinical information such as myocardial imaging studies.⁸

A high frequency of intermittence of an initial R wave in V₂ was found in this study in cases with

QS complexes in V₁ and V₂ in the sentinel tracing. Since this was usually not associated with change in septal Q-wave status, and since most of these cases did not have any explanatory cardiac pathology, it is likely that, with no other ECG abnormalities present, the intermittence of the R in V₂ was an artifact of differing lead placement. This is consistent with the high frequency with which these cases were associated with downward direction of initial QRS forces, which would predispose to negative initial deflections in right precordial leads if the latter were placed too high.^{11,13,19} It is also consistent with the speculation that QS complexes in V₁ and V₂ might be more frequent in conditions where the position of the heart is lower relative to the position of right precordial electrodes (e.g., emphysema), or those in which usual lead placement is complicated by marked thoracic deformity.

The absence of septal Q waves, present in nearly two-thirds of cases in the study group, in most cases was not related to the presence or absence of infarction of the IVS or presence of LVH. Most of these cases had no clinical evidence of heart disease. Rather than being a result of acquired cardiac disease, it is more likely that absence of septal Q waves was the result of a leftward direction of initial QRS forces, which can be seen in some normal persons.^{1,13,19} If right precordial electrodes are placed high, or if the heart position is lower relative to electrode placement, inferior and leftward orientation of initial forces sets the stage for right precordial electrodes to fall in a region where anteriorly directed initial QRS forces may project a negative potential on the body surface.^{11,13}

The apparent greater frequency of QS deflections in V₁-V₂ in women than in men is not readily explained, unless it is because the presence of breasts impedes correct electrode placement.¹² For that same reason, it was hypothesized at the start of this study that obesity would contribute to the occurrence of this pattern. However, the observed results were not consistent with this, as subjects with a QS in V₁-V₂ on an average actually weighed less and had a lesser BMI than matched controls with normal ECGs.

CONCLUSIONS

When other ECG abnormalities are absent, QS deflections in leads V₁ and V₂ should not be interpreted by an ECG reader as indicative of infarction of the IVS. Computer software that interprets this

pattern thus should be changed. In this study, subjects with such an ECG pattern had infarction of some part of the heart in less than one-fifth of the cases. In the latter, myocardial imaging showed the infarction involved the IVS in less than a third of these, and infarction *limited* to the IVS was rarely present. This ECG pattern can be due to multiple causes, the most frequent of which appears to be high placement of right precordial electrodes in patients with inferiorly directed initial ECG forces. It should be interpreted as a nonspecific QRS abnormality of multiple possible etiologies, one of which in a minority of cases can be MI of uncertain age involving at least some part of the anterior or apical left ventricle. Comparison with prior ECGs should be sought. To assess the significance of this ECG finding in a given case, the attending physician should be requested to correlate it with all other clinical information. The ECG should be repeated with special attention to correct right precordial lead placement, which will often generate an entirely normal tracing. When QS deflections in V₁ and V₂ are accompanied by *other* ECG abnormalities, especially ischemic-type precordial T wave inversions, the probability of underlying MI is greatly increased.

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