JACC Vol. 16, No. 2
August 1990:368–74

Q Waves in Hypertrophic Cardiomyopathy in Relation to the Distribution and Severity of Right and Left Ventricular Hypertrophy

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The cause of abnormal Q waves in hypertrophic cardiomyopathy remains unclear. Myocardial wall thickness was assessed by two-dimensional echocardiography at 8 left ventricular and 10 right ventricular sites in 67 patients (mean age 40 years) with hypertrophic cardiomyopathy and the findings were analyzed in relation to the presence of abnormal Q waves on the 12 lead rest electrocardiogram (ECG). Nineteen (28%) of the 67 patients had abnormal Q waves. Right ventricular hypertrophy was significantly more common in patients without abnormal Q waves (25 [52%] of 48 versus 2 [11%] of 19, $p < 0.001$).

With univariate analysis, there were six measurements that were significantly associated with abnormal Q waves: an increase in upper anterior septal thickness ($p < 0.005$) and maximal left ventricular wall thickness ($p < 0.02$), a decrease in mean and maximal right ventricular wall thickness (both $p < 0.005$) and an increase in the ratio of both upper anterior septal to mean right ventricular wall thickness ($p < 0.005$) and upper anterior septal to upper posterior wall thickness ($p < 0.005$). With multivariate analysis, only the ratios of upper anterior septal to mean right ventricular wall thickness ($p < 0.005$) and to upper posterior wall thickness ($p < 0.05$) were significantly related to the presence of abnormal Q waves and predicted Q wave location with a sensitivity, specificity and predictive accuracy of 90%, 85% and 89%, respectively.

In hypertrophic cardiomyopathy, the presence of abnormal Q waves on the 12 lead ECG is primarily a function of the relation of right ventricular wall thickness and upper anterior septal thickness.

(J Am Coll Cardiol 1990;16:368–74)

Abnormal Q waves in hypertrophic cardiomyopathy were described more than 25 years ago (1,2). Their mechanism remains uncertain despite electrocardiographic (ECG) (2–7), echocardiographic (4,6,7), electrophysiologic (8–10) and surgical/pathologic (9,11,12) correlative studies. Such abnormal Q waves are generally thought to be caused by abnormal electrical activation of the hypertrophied interventricular septum (8–12); however, there does not appear to be a relation between the magnitude of septal hypertrophy and the presence of pathologic Q waves (4,6,7). In this study, echocardiographic measurements of the right and the left ventricle in patients with hypertrophic cardiomyopathy were related to the presence of abnormal Q waves.

Methods

Study patients. Eighty-three consecutive patients with hypertrophic cardiomyopathy underwent left and right ventricular two-dimensional echocardiographic and 12 lead ECG evaluation. Ten patients were excluded because the echocardiographic recordings were technically inadequate. Another six patients who had ECG abnormalities that could mask the presence of Q waves were excluded (left bundle branch block in two and pre-excitation in four). The final study group included 67 patients. Thirty-six were male and 31 female; they were aged 17 to 67 years (mean 40). No patient had a history of myocardial infarction. Hypertrophic cardiomyopathy was diagnosed 1 to 20 years (mean 6) before this study on the basis of characteristic clinical, hemodynamic and angiographic findings (2). The diagnosis was confirmed in all patients by two-dimensional echocardiographic demonstration of unexplained left ventricular hyper-

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Manuscript received November 20, 1989; revised manuscript received February 7, 1990, accepted March 13, 1990.

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0735-1097/90/$3.50

trophy (13,14). Informed consent was obtained in all patients, and the institutional Committee on Human Research approved the study protocol.

Echocardiography. Two-dimensional echocardiography was performed using a phased array system (GE Pass IC) with a 3.3 MHz transducer. Images of the examination sequence together with standard lead II of the ECG were stored on videotape (Sony Betamax) for subsequent analysis. Each study included parasternal long- and short-axis views with the transducer positioned at the third or fourth intercostal space, two and four chamber views with the transducer at the apex, long- and short-axis views from the subcostal window as well as specific views of the right ventricular inflow and outflow tracts. Ten right ventricular wall thickness measurements were obtained as previously described (13). All measurements were made at end-diastole defined by the onset of the R wave on a simultaneous ECG. Right ventricular hypertrophy was considered to be present when at least two right ventricular wall thickness measurements exceeded 2 standard deviations of the mean of a previously established control group (13).

The technique for recording and assessing the extent and distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy has been previously described (14). In brief, eight wall thickness measurements were made from short-axis views at each of the quadrants at the mitral valve tip and papillary muscle levels. The distribution of left ventricular hypertrophy was classified as asymmetric septal, symmetric or predominantly distal ventricular according to established criteria (14).

To assess the relation of left and right ventricular hypertrophy in patients with and without abnormal Q waves, we obtained eight left ventricular measurements (Fig. 1), mean left ventricular wall thickness and mean and maximal right ventricular wall thickness. We then calculated the following ratios: each left ventricular wall thickness measurement divided by the other left ventricular measurements, each left ventricular wall thickness measurement as well as the sum of two values obtained at the papillary or mitral valve level divided by mean and maximal right ventricular measurement. A total of 66 combinations were assessed.

Electrocardiography. A standard 12 lead ECG was recorded at the time of the study with the patient in the supine position during quiet respiration, using either a Marquette microcomputer-augmented cardiograph (Mac II) or a Hewlett-Packard three channel recorder (1513A or 1513B). The following features were documented: rhythm, mean frontal plane axis, conduction defects, giant T wave inversion (T wave inversion >0.1 mV or >50% of the amplitude of the R wave) and the presence of abnormal Q waves. These were defined as >0.04 s in duration and >25% of the R wave in depth. The degree of left ventricular hypertrophy was assessed with voltage criteria as well as the point score system of Romhilt and Estes (15). Right ventricular hypertrophy was assessed using right axis deviation of ≥110° and an R/S ratio ≥1 in lead V_1 (16).

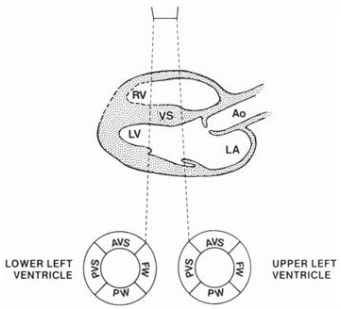

Figure 1. Diagrammatic representation of left ventricular cross sections obtained at the level of the mitral valve and papillary muscle, resulting in eight measurements. Right ventricular measurements include both mean and maximal right ventricular thickness obtained from 10 views (13). Ao = aorta; AVS and PVS = anterior and posterior ventricular septum, respectively; LA = left atrium; LV = left ventricle; PW and FW = posterior and free wall, respectively; RV = right ventricle; VS = ventricular septum. Adapted from references 13 and 14.

Statistical analysis. Group data are presented as the mean value ± SD. Student's *t* test or chi-square *t* test was used to analyze the difference between variables. A stepwise discriminant analysis was performed to identify measurements that were most significantly associated with the presence of abnormal Q waves. We then applied these values to calculate the sensitivity, specificity and predictive accuracy for the presence and location of Q waves. A p value <0.05 was used to indicate statistical significance.

Results

Location of Q waves. Nineteen (28%) of the 67 patients had abnormal Q waves on the 12 lead ECG. Figure 2 shows that abnormal Q waves were located either inferiorly, over precordial leads V_2 to V_6 or in leads I and aVL. The majority (13 patients) had Q waves in inferior leads (II, III or aVF), whereas only 4 patients had Q waves in leads I and aVL.

Clinical and ECG features in patients with and without Q waves. When comparing both groups of patients, there was no significant difference in age and gender. A history of chest pain was present in 26 (54%) of the 48 patients without Q waves compared with 9 (47%) of the 19 patients with Q waves (p = NS). Sixty-two patients were in sinus rhythm and 5 had atrial fibrillation. Giant T wave inversion in the

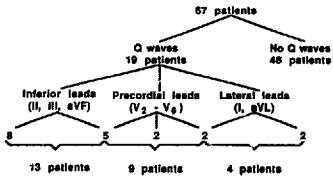

Figure 2. Localization of Q waves in 19 patients with hypertrophic cardiomyopathy. Q waves were most common in inferior leads (13 patients) but were also present in precordial leads in 9 patients and leads I and aVL in 4 patients. The total number of patients with Q waves according to site is >19 because of overlap between sites in some patients.

precordial leads was seen in nine patients and none of these had Q waves; three of these patients had documented right ventricular hypertrophy and six did not. The absence of normal Q waves in leads I and aVL or V_5 and V_6 was seen in 6 (42%) of 14 patients with abnormal Q waves inferiorly or anteriorly, compared with 27 (56%) of 48 patients without abnormal Q waves (p = NS). Table 1 reveals similar ECG features of right and left ventricular hypertrophy in patients with and without abnormal Q waves.

Echocardiographic features in patients with and without Q waves. Left ventricular hypertrophy was asymmetric in 37 patients (55%), symmetric in 24 (36%) and predominantly distal ventricular in 6 (9%). Right ventricular hypertrophy was present in 27 patients (40%) with a strong correlation between maximal right and mean left ventricular wall thickness ($r = 0.643$, $p < 0.001$). Right ventricular hypertrophy was not associated with a particular pattern of left ventricular hypertrophy.

Abnormal Q waves were present in 14 (38%) of 37 patients with asymmetric ventricular hypertrophy, 4 (17%) of 24 patients with symmetric hypertrophy and in none of the 6 patients with apical hypertrophy. Specifically, patients with abnormal Q waves had greater upper anterior septal and maximal left ventricular wall thickness measurements, with a lower mean and maximal right ventricular wall thickness (Table 2). On multivariate analysis, however, none of these wall thickness measurements were associated with abnormal Q waves. Right ventricular hypertrophy was seen in only 2 (11%) of 19 patients who had abnormal Q waves compared with 25 (52%) of 48 patients without abnormal Q waves ($p < 0.001$).

Significant differences in echocardiographic measurements in patients with and without abnormal Q waves were seen when the relation of left and right ventricular wall thickness was analyzed (Table 2). Multivariate analysis revealed that 4 of the 66 ratios assessed were significantly different in patients with and without Q waves (Table 3). In particular, the ratios of upper anterior septal to mean right ventricular wall thickness and of upper anterior septal to upper posterior wall thickness were significantly greater in patients with abnormal Q waves, and when entered into a stepwise discriminant analysis had a high sensitivity, specificity and predictive accuracy for the prediction of abnormal Q waves (Table 3).

Table 1. Electrocardiographic Features of Ventricular Hypertrophy and Mean Frontal Axis in 67 Patients With and Without Abnormal Q Waves

	Q Waves	No Q Waves	p Value
No. (%)	19 (28)	48 (72)	—
Mean frontal plane axis (°)	9.6 ± 52.4	24.1 ± 40.3	NS
Romhilt-Estes score	5.5 ± 2.5	6.2 ± 2.6	NS
R wave in lead aVL (mV)	12.7 ± 8.5	9.8 ± 6.6	NS
S wave in lead V_1 + R wave in leads V_5 or V_6 (mV)	36.7 ± 22.7	41.5 ± 18.9	NS
R/S in lead V_1	0.39 ± 0.55	0.27 ± 0.42	NS

Data are mean values ± SD.

Discussion

Predictors of abnormal Q waves. The presence of abnormal Q waves in hypertrophic cardiomyopathy was predicted by the interrelation of right ventricular, upper anterior septal and posterior wall thickness. In particular, an increase in the ratios of upper anterior septal to right ventricular wall and upper posterior wall thickness was predictive of abnormal Q waves. This is consistent with the interpretation that Q waves are present when electrical forces of the upper anterior septum are relatively unopposed by right ventricular and upper posterior wall electrical forces (moderate septal hypertrophy with no right ventricular hypertrophy and normal posterior wall thickness) or are of such magnitude that they cancel out right ventricular and posterior wall electrical forces (marked septal hypertrophy with mild right ventricular hypertrophy or posterior wall hypertrophy) (Fig. 3).

Comparison with previous ECG studies. The early description of hypertrophic cardiomyopathy by Braunwald et al. (2) was based on an analysis of 64 patients and revealed 16 patients (25%) who exhibited abnormal deep and broad Q waves. When that series was updated 4 years later in 1968 (17), 56% of 123 patients had abnormal Q waves. These investigators (2,17) and others (8–12) attributed the abnormal Q waves in hypertrophic cardiomyopathy to the massive hypertrophy observed in the interventricular septum. However, the study by Maron et al. (4) concluded that abnormal Q waves were seen more frequently when hypertrophy involved regions of the left ventricle other than the anterior basal septum. In their study (4), 23 (18%) of 126 patients had abnormal Q waves when hypertrophy involved the anterior

Table 2. Echocardiographic Measurements in Patients With and Without Abnormal Q Waves

	Q Waves	No Q Waves	p Value*	
			Uni	Multi
No. (%)	19 (28)	48 (72)		
Left ventricle				
Upper anterior septum	23.4 ± 4.3 (17–35)	18.3 ± 5.3 (9–30)	<0.005	NS
Upper posterior septum	17.1 ± 4.9 (11–28)	15.8 ± 3.9 (8–30)	NS	NS
Upper posterior wall	12.4 ± 3.4 (8–20)	13.7 ± 3.7 (10–30)	NS	NS
Upper free wall	16.8 ± 4.0 (10–28)	16.2 ± 4.5 (8–30)	NS	NS
Lower anterior septum	22.3 ± 5.7 (13–35)	20.8 ± 5.3 (13–32)	NS	NS
Lower posterior septum	18.1 ± 4.0 (13–28)	17.6 ± 4.4 (10–28)	NS	NS
Lower posterior wall	14.8 ± 4.8 (8–28)	15.7 ± 4.3 (10–32)	NS	NS
Lower free wall	16.5 ± 4.7 (10–23)	17.8 ± 4.0 (10–32)	NS	NS
Maximal ventricular thickness	24.9 ± 4.8 (20–35)	21.7 ± 4.9 (15–32)	<0.02	NS
Right ventricle				
Mean ventricular thickness	3.7 ± 0.5 (3.0–5.0)	5.0 ± 1.7 (3.2–14.2)	<0.005	NS
Maximal ventricular thickness	4.0 ± 0.4 (4–5)	5.6 ± 2.0 (3–18)	<0.005	NS

*p value by univariate (Uni) and multivariate (Multi) analysis. All values given in mm (mean ± SD and range).

basal septum alone or other areas of the left ventricle (so-called type I-II-III hypertrophy). In patients without hypertrophy of the anterior basal septum (type IV hypertrophy, 15 (56%) of 27 had abnormal Q waves. The apparent discrepancy between their results and ours may be related to the site of the abnormal Q waves. Although Maron et al. (4) do not describe the location of abnormal Q waves, it is interesting that in the example shown in their Figure 3, Q waves occur in the lateral leads only. In our study, the location of abnormal Q waves in the lateral leads was not associated with significant hypertrophy of only the anterior septum (Table 3). The overall 18% incidence rate of abnormal Q waves in patients with hypertrophy involving the anterior septum in the report by Maron et al. (4) is in accordance with the usual reported incidence of abnormal Q waves in patients with hypertrophic cardiomyopathy. The

Table 3. Relation of Left and Right Ventricular Wall Thickness Measurements in Patients With and Without Abnormal Q Waves*

		Abnormal Q Waves				
	All Patients	II, III, aVF and Other Leads	II, III, aVF Only	V_2–V_6 or I and aVL and Other Leads	V_2–V_6 or I and aVL Only	No Abnormal Q Wave
No.	19	13	8	11	6	48
A. Ratio of the thickness of						
1. Upper anterior septum/mean right ventricular wall	6.6 ± 1.3‡	6.7 ± 1.55‡	6.4 ± 1.3‡	6.1 ± 1.0‡	5.9 ± 1.1	3.8 ± 1.2
2. Upper anterior septum/upper posterior wall	2.0 ± 0.4§	2.0 ± 0.2§	2.0 ± 0.4§	1.9 ± 0.5	1.9 ± 0.6	1.4 ± 0.3
3. Upper posterior septum/maximal right ventricular wall	4.2 ± 1.2	3.8 ± 1.2	3.9 ± 1.4	4.5 ± 1.1	5.0 ± 1.1§	3.0 ± 9.9
4. Upper anterior + posterior septum/upper posterior wall	3.4 ± 0.8	3.3 ± 0.4	3.2 ± 0.4	3.6 ± 1.0§	3.7 ± 1.3‡	2.5 ± 0.5
B. Value of echocardiographic measurements for predicting abnormal Q waves†						
Sensitivity (%)	90	90	90	90	95	—
Specificity (%)	88	100	100	89	60	—
Predictive accuracy (%)	89	92	92	90	91	—

*Groups with abnormal Q waves each compared with the group with no abnormal Q waves. †When analyzing the most significant variables obtained during multivariate analysis. For the entire group of 19 patients with Q waves, A1 was taken for the analysis of sensitivity, specificity and predictive accuracy; for the subgroup of 13 patients, A1 was analyzed; for the subgroup of 8 patients, A1 was analyzed; for the subgroup of 11 patients, A1 was analyzed; for the subgroup of 6 patients, A4 was chosen for analysis. ‡$p < 0.005$ and §$p < 0.05$ by stepwise discriminant analysis. See text for explanation.

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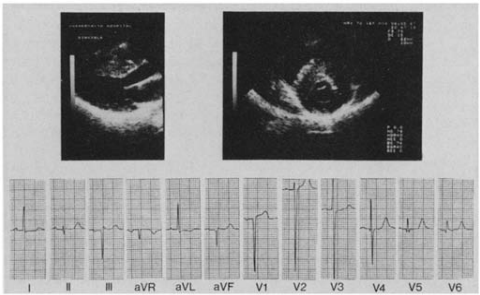

Figure 3. Long- (left) and short- (right) axis two-dimensional echocardiographic views at the mitral valve level from a patient with asymmetric septal hypertrophy who does not have right ventricular hypertrophy. Abnormal Q waves are present in leads III, aVF and V_1.

predominant inferior location of abnormal Q waves in our study may also be related more closely to specific involvement of the anterior septum.

A recent two-dimensional echocardiographic study (7) of patients with hypertrophic cardiomyopathy and severe left ventricular hypertrophy with anterior ventricular septal thickness of 30 to 50 mm (mean 37 ± 5) revealed that only 5 (15%) of 34 patients had abnormal Q waves. Right ventricular wall thickness measurements were not made in these patients. The strong correlation of left and right ventricular wall thickness measurements in hypertrophic cardiomyopathy (13) suggests that the patients of Louie and Maron (7) may have had coexistent right ventricular hypertrophy. Our report indicates that abnormal Q waves are uncommon in the presence of increased right ventricular wall thickness and are strongly predicted by an increased ratio of upper anterior septal to right ventricular wall thickness. The low incidence of Q waves in patients with massive septal hypertrophy could be explained by the coexistence of right ventricular hypertrophy (Fig. 4).

Comparison with previous electrophysiologic studies. van Dam et al. (9) recorded epicardial and intramural electrograms from 10 patients with hypertrophic cardiomyopathy, 5 of whom had abnormal Q waves. They observed a delay in subendocardial activation over the anterior paraseptal left ventricular wall, prolongation of total septal activation because of hypertrophy and an increased contribution of the right ventricle to septal excitation. Septal activation normally takes 40 ms; they observed a delay of activation of up to 70 ms (9). They suggested that abnormal Q waves were the result of irregular activation in the inner layers of the septum, caused by hypertrophy and myocardial disarray. However, they did not find a correlation between abnormal Q waves and a particular pattern of septal activation.

Cosio et al. (10) performed electrophysiologic studies in six patients with hypertrophic cardiomyopathy and abnor-

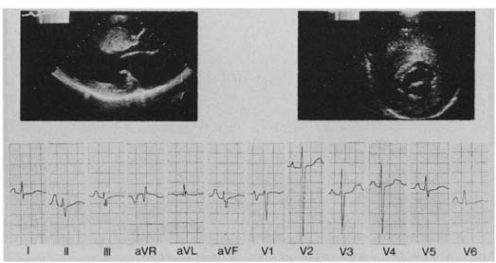

Figure 4. Long- (left) and short- (right) axis two-dimensional echocardiographic views at the mitral valve level from a patient with asymmetric septal hypertrophy and right ventricular hypertrophy. The 12 lead ECG indicates sinus rhythm and biatrial enlargement; there are no abnormal Q waves. Note the absence of normal Q waves in leads I, V_5 and V_6, a finding seen equally in patients with and without abnormal Q waves (see text).

mal Q waves on the surface ECG. The Q waves were rate dependent and decreased in size or disappeared after late coupled atrial extrastimuli and during atrial pacing at rates >115 to 155 beats/min. They suggested that abnormal Q waves in their patients were due to a disturbance in septal activation caused by abnormal electrophysiologic properties of the myopathic myocardium. Zalman et al. (18) reported the rate-dependent appearance of abnormal Q waves during exercise testing in two patients with hypertrophic cardiomyopathy. They suggested that such Q waves may have been caused by exercise-induced ischemia or a shift of the QRS vector, or both. The discrepancy between both of these studies (10,18) may be related to different techniques—exercise testing produces a greater increase in catecholamines and a greater decrease in cardiac volume than seen with pacing; refractoriness of normal and diseased myocardium may vary accordingly.

Several studies (9,10,18,19) support the concept of abnormal septal activation producing abnormal Q waves in patients with hypertrophic cardiomyopathy. Two studies (11,12) reported on the effects of ventriculomyotomy in three patients with hypertrophic cardiomyopathy who had abnormal Q waves. Surgical incision in line with the commissure between the right and left coronary cusps without removal of muscle was associated with disappearance of the abnormal Q waves. They (11,12) postulated that septal hypertrophy produced dominance of anterosuperior over posteroinferior electrical forces that could be altered by septal incision.

Our findings in patients with hypertrophic cardiomyopathy show that abnormal Q waves are seen infrequently in patients with right ventricular hypertrophy. Presumably, the electrical forces of the hypertrophied right ventricle combined with prolonged activation of the diseased septum produce a mean QRS vector posteriorly and rightward, with attenuation or cancellation of electrical forces from the septum (16). Our results are in contrast to those of Mori et al. (6), who noted that abnormal Q waves were associated with basal septal hypertrophy and right ventricular hypertrophy. The discrepancy may reflect different techniques: they used M-mode echocardiography and included the moderator band in the right ventricular measurement. The shape of the right ventricle makes it difficult to image, particularly from a single plane. Ten right ventricular wall thickness measurements were attempted from multiple views in our patients. A total of 4 to 10 measurements (median 7) were obtained in each patient (13). The maximal left and right ventricular thickness measurements differed by only 3.2 and 1.6 mm, respectively. Precise assessment of ventricular thickness is essential when comparing both groups of patients. The results of this study are limited by this small difference in right ventricular measurement between both groups and the lack of comparable reports. Further assessment of the right ventricle by echocardiography and magnetic resonance imaging is needed to substantiate our findings.

Role of myopathic changes in conduction. Our report does not assess the influence of myopathic changes on conduction and refractoriness. Also, the sensitivity analysis applies to this group of patients and needs to be verified in a separate group of individuals. Furthermore, although the majority of patients had anterior ventricular septal hypertrophy and 60% of patients did not have right ventricular hypertrophy, Q waves were present in only 28%. The significant association between abnormal Q waves and an increased ratio of the upper anterior septal to mean right ventricular wall thickness suggests that abnormal Q waves in patients with hypertrophic cardiomyopathy are determined to a greater extent by the distribution of myocardial hypertrophy than by primary or secondary conduction abnormalities. However, the absence of abnormal Q waves in patients with anterior ventricular septal hypertrophy who do not have right ventricular hypertrophy suggests that other factors or mechanisms may also be important for predicting abnormal Q waves in patients with hypertrophic cardiomyopathy.

We thank Shaughan Dickie for technical assistance and Lise de Repentigny for secretarial work.

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