PATTERN OF MYOCARDIAL HYPERTROPHY AS A POSSIBLE DETERMINANT OF ABNORMAL Q WAVES IN HYPERTROPHIC CARDIOMYOPATHY

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Echocardiographic and electrocardiographic findings in 74 adults with hypertrophic cardiomyopathy (HCM) were analyzed to identify the pattern of myocardial hypertrophy as a possible determinant of abnormal Q waves.

The pattern of septal hypertrophy along the left ventricular long axis was divided into 3 types based on the site of maximum septal hypertrophy: basal, diffuse and apical types. Abnormal Q waves defined by the revised Minnesota Codes (either I–I, I–II or I–III) were noted in 31 cases (42%). The total incidence of abnormal Q waves in the basal type (15/26, 58%) and in the diffuse type (12/22, 55%) was significantly higher (p < 0.001 and p < 0.01, respectively) than that in the apical type (4/26, 15%). The abnormal Q waves defined by the strict criteria of Code I–I were significantly more prevalent (p < 0.05) in the basal type than in the diffuse type, although there was no significant difference in the total incidence of abnormal Q waves between these 2 groups. Thirty-six patients with an extension of hypertrophy to the right ventricle (RVH) had a significantly higher incidence of abnormal Q waves than 22 patients without RVH (56% vs 27%, p < 0.05). Furthermore, close relationships of RVH to the location of abnormal Q waves were documented.

In conclusion, the abnormal Q waves in HCM may be related to the pattern of septal hypertrophy along the left ventricular long axis and to RVH.

ABNORMAL Q waves resembling those in myocardial infarction have been indicated as one of the characteristic features of hypertrophic cardiomyopathy in electrocardiography. The genesis of abnormal Q waves is not fully understood as yet, although an increase in the initial electrical force resulting from a hypertrophied interventricular septum has been widely considered as a principal factor. Furthermore, it has been pointed out in recent M-mode echocardiographic studies by Halpern et al. and Savage et al. that no relation exists between the prevalence of abnormal Q waves and the degree of asymmetric septal hypertrophy. In the present study, we attempted to identify the specific patterns of myocardial hypertrophy.

Key Words:
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attributable to the genesis of abnormal Q waves in hypertrophic cardiomyopathy using two-dimensional (2-D) and M-mode echocardiography.

SUBJECTS AND METHODS

Echocardiograms and standard 12-lead electrocardiograms of 74 adults with hypertrophic cardiomyopathy (49.5 ± 14.1 years old on the average) were analyzed and compared.

A wide angle phased array scanner (Toshiba SSH-11A) was employed for the echocardiographic studies. In the 64 patients, M-mode echocardiographic evidence of asymmetric septal hypertrophy was noted at either the subaortic level or the mid-ventricular level. In the remaining 10 patients, the baseline portion of the septum used for the M-mode echocardiographic measurement was not prominently hypertrophied (less than 15 mm in thickness), but marked hypertrophy of the apical septum was documented by 2-D echocardiography. In addition, left ventriculographic and electrocardiographic findings in these 10 patients were compatible with those of "apical hypertrophic type" reported by Yamaguchi et al.9

In all patients, an association of systemic hypertension of more than 160/100 mmHg, myocardial infarction or other cardiovascular diseases, which may cause asymmetric septal hypertrophy, was excluded by thorough clinical analyses. In addition to a septal-to-posterior wall thickness ratio of 1.3 or more, an absolute septal thickness of 15 mm or more was also used as a criterion of asymmetric septal hypertrophy in the present study, because a thickness ratio of 1.3 or more without an increase in absolute septal thickness is often noted in normal subjects.10

The hypertrophic pattern of the interventricular septum was evaluated from the 2-D left ventricular (LV) long axis and the short axis views with a scanning from the base to the apex. The interventricular septum, as viewed from the long axis, was divided into basal and apical segments, with reference to the root of the papillary muscles. As viewed from the short axis, it was almost equally divided into anterior and posterior halves.

The LV free wall was equally divided into 3 segments: the anterior wall, the lateral wall, and the posterior wall. The posterior wall thickness was less than 13 mm on the M-mode echocardiogram in 67 of 74 cases. Furthermore, the degree of hypertrophy in the posterior wall did not ex-

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Fig. 2. Echocardiograms indicating the prominent right ventricular moderator band.

As shown in the upper panel, a wide band-like echo extending from the septal surface is indicated along the short axis view of the 2-dimensional echocardiogram. On the M-mode echocardiogram, this produced non-continuous multiple echoes in front of the septal echoes (lower panel). In this particular case, the width was measured as 10 mm.

Abbreviations: RV = right ventricle; Mod. band = moderator band; IVS = interventricular septum; PW = posterior wall

Fig. 3. Three patterns of septal hypertrophy along the left ventricular long axis.

Representative 2-dimensional echocardiograms along the long axis view in patients with each type of septal hypertrophy are shown. The arrows in the schematic representations indicate the sites of maximum hypertrophy.

Abbreviations: IVS = interventricular septum; PW = left ventricular posterior wall; LA = left atrium; MV = mitral valve
Fig. 4. Incidence of abnormal Q waves and of absence of Q waves in the patients with each type of septal hypertrophy.

Solid bars indicate the incidence of abnormal Q waves defined according to the strict criteria of the Minnesota Code I–I. Striped bars are those defined according to the less strict criteria of Codes I–II or I–III. The incidence of "absence of Q waves" is shown by dotted bars.

proceed that in other LV segments on the 2-D echocardiographic short axis view in any of the patients. Therefore, the judgement of anterior wall and lateral wall hypertrophy was made by a qualitative comparison of their wall thickness with that of the posterior wall on the short axis view.

One of the characteristic findings in our patients was the extension of hypertrophy to the right ventricular (RV) structures adjacent to the anterior half of the septum. As shown in Fig. 1, thickness of the RV anterior free wall was measured on M-mode echocardiograms. Hypertrophy of the RV anterior free wall was defined as a thickness of 5 mm or more according to the pathological criteria. In addition to the RV anterior free wall, a prominent RV moderator band was frequently identified on the right side of the interventricular septum as shown by the arrow in the upper panel of Fig. 2. On M-mode echocardiograms, this structure produced noncontinuous multiple echoes in front of the ventricular septum. In the present study, a moderator band with a width of 5 mm or more on M-mode echocardiogram was judged as abnormal hypertrophy.

Standard 12-lead electrocardiograms were recorded within 2 weeks of each echocardiographic examination in all patients. The criteria for abnormal Q waves were based on the revised Minnesota Codes (I–I, I–II, I–III).13

RESULTS

Variation in Myocardial Hypertrophy

From the LV long axis view, 26 patients were characterized by a predominant hypertrophy of the basal septum as compared to that of the apical septum (Fig. 3A, basal type). The other 26 patients had predominant hypertrophy of the apical septum as shown in Fig. 3C (apical type). This group of patients consisted of 2 subgroups, that is, 16 patients with M-mode echocardiographic evidence of asymmetric septal hypertrophy and 10 patients without prominent hypertrophy in the basal septum (<15 mm in thickness). In 22 cases, the basal and the apical septums were almost equally hypertrophied (diffuse type, Fig. 3B). From the short axis view, 23 patients had predominant hypertrophy of the anterior half of the septum as compared with that of the posterior half, 3 having it in the posterior half and 48 having diffuse hypertrophy from the anterior to the posterior. There was no significant correlation between the pattern of septal hypertrophy on the long axis view and that on the short axis view.

Extension of the hypertrophy to the LV anterior wall was noted in most of the patients (67 cases, 91%), but hypertrophy of the lateral wall was rare (5 cases, 7%).

Hypertrophy of the RV anterior free wall and/or the moderator band was seen in 36 patients. Ten of these patients had hypertrophy of both structures. In 22 patients, neither RV anterior free wall nor moderator band was prominent. In the remaining 16 patients, the RV structures could not be assessed on echocardiograms.

Pattern of Myocardial Hypertrophy and Abnormal Q Waves

Abnormal Q waves were noted in 31 of the 74 patients (42%). The incidence of abnormal Q waves for each type of septal hypertrophy along

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Fig. 5. Three representative electrocardiograms for each type of septal hypertrophy. In a basal type case shown in the top, the amplitude of R waves decreases progressively in V1 to V4, and finally, abnormal QS pattern defined by Minnesota Code I–I appears in V6. Additional abnormal Q or QS patterns are also noted in limb leads. A diffuse type case shows deep Q waves in left precordial leads defined by the Code I–II, however, their widths are less than 0.04 sec (middle). In an apical type case shown in the bottom, abnormal Q waves are not documented, and moreover, even normal Q waves are absent in left precordial and limb leads.

### TABLE 1 SEPTAL THICKNESS AND THE PRESENCE OR ABSENCE OF ABNORMAL Q WAVES IN EACH TYPE OF SEPTAL HYPERTROPHY

<table>
<thead>
<tr>
<th></th>
<th>Q (+)</th>
<th>Q (−)</th>
</tr>
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<tbody>
<tr>
<td>Basal</td>
<td>19.0 ± 3.2</td>
<td>17.0 ± 1.8</td>
</tr>
<tr>
<td></td>
<td>n = 15</td>
<td>n = 11</td>
</tr>
<tr>
<td>Diffuse</td>
<td>18.5 ± 4.0</td>
<td>17.8 ± 1.0</td>
</tr>
<tr>
<td></td>
<td>n = 12</td>
<td>n = 10</td>
</tr>
<tr>
<td>Apical</td>
<td>17.5 ± 3.0</td>
<td>15.2 ± 3.4</td>
</tr>
<tr>
<td></td>
<td>n = 4</td>
<td>n = 22</td>
</tr>
</tbody>
</table>

Values are given as mean ± S.D. Abbreviations: IVS = interventricular septum; Q = abnormal Q waves; ns = not significant; n = number of patients

The LV long axis is indicated in Fig. 4. The total incidence of abnormal Q waves in the basal and the diffuse type patients was 58% (15/26) and 55% (12/22), respectively. These values are significantly higher (p < 0.001 and p < 0.01, respectively) than the incidence of 15% (4/26) in the apical type patients. There was no significant difference in the total incidence of abnormal Q waves between the basal type patients and the diffuse type patients. However, the abnormal Q waves defined according to the strict criteria of Code I–I were significantly more prevalent (p < 0.05) in the basal type patients (12 cases, 46%) than in the diffuse type patients (4 cases, 18%).

As shown by the dotted bars, normal Q waves were absent in the limb and the left precordial leads (the so-called “absence of Q waves”14) in one patient (4%) with basal septal hypertrophy and in 2 (9%) with diffuse septal hypertrophy, while in the apical type patients the incidence (14 cases, 54%) was significantly higher than

TABLE II  EXTENSION OF HYPERTROPHY AND THE INCIDENCE OF ABNORMAL Q WAVES

<table>
<thead>
<tr>
<th>Incidence of abnormal Q</th>
<th>(+)</th>
<th>(-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV lat. wall</td>
<td>25/2</td>
<td>29/69</td>
</tr>
<tr>
<td>LV ant. wall</td>
<td>30/67</td>
<td>17</td>
</tr>
<tr>
<td>RV structure</td>
<td>20/36</td>
<td>6/22</td>
</tr>
</tbody>
</table>

Abbreviations: LV lat. wall = left ventricular lateral wall; LV ant. wall = left ventricular anterior wall; RV structure = hypertrophy of RV anterior free wall and/or moderator band; ns = not significant

Abnormal Q (+) | Abnormal Q (-)
Basal N=26
Diffuse N=22
Apical N=26

Fig. 6. Relationship between abnormal Q waves and right ventricular hypertrophy in patients with each type of septal hypertrophy.
Solid bars indicate the incidence of the cases with right ventricular hypertrophy and open bars indicate that of the cases without right ventricular hypertrophy. In the cases shown by the striped bars, right ventricular structures could not be assessed echocardiographically.

TABLE III  RELATIONSHIP BETWEEN EXTENSION OF HYPERTROPHY TO THE RIGHT VENTRICLE AND LOCATION OF ABNORMAL Q WAVE

<table>
<thead>
<tr>
<th>RVH (+)</th>
<th>Mod. band RVAW</th>
<th>RV not visualized</th>
<th>RVH (-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral Q (n = 21) patients</td>
<td>14</td>
<td>12</td>
<td>5</td>
</tr>
<tr>
<td>Inferior Q (n = 9)</td>
<td>6</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Antero-septal Q (n = 3)</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: Lateral Q = abnormal Q waves in either lead I, aVL, V5 or V6; Inferior Q = abnormal Q waves in either lead II, III or aVF; Anterior Q = abnormal Q in either lead V1, V2 or V3; RVH = extension of hypertrophy to the right ventricle; Mod. band = moderator band hypertrophy; RVAW = right ventricular anterior free wall hypertrophy.

the left ventricular long axis. As shown in Table I, the mean values of absolute septal thickness in the patients with abnormal Q waves exceeded those in the patients without abnormal Q waves in all 3 groups, although the differences were not statistically significant.

The relationship between the distribution of septal hypertrophy on the short axis view and the abnormal Q waves was also examined. The incidence of abnormal Q waves was 52% of 23 patients with a predominant hypertrophy in the anterior septum and 40% of 48 patients in which the anterior and posterior septums were equally hypertrophied. Abnormal Q waves were not noted in 3 patients with predominant hypertrophy in the posterior septum. Statistically significant differences were not found among these 3 groups.

As summarized in Table II, an extension of hypertrophy to the LV lateral wall and/or the anterior wall did not correlate with the prevalence of abnormal Q waves. Furthermore, the incidence of abnormal Q waves in 7 patients with posterior wall thickness of ≥ 13 mm (3 cases, 43%) was not significantly different from that in 67 patients, who had normal posterior wall thickness (42%). It is worthy of note that the incidence of abnormal Q waves in those patients with RV hypertrophy was significantly higher.
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(p < 0.05) than that in those without RV hypertrophy.

Figure 6 indicates the relation of abnormal Q waves to the extension of hypertrophy to the RV in each type of septal hypertrophy. RV hypertrophy was present in 7 of 15 cases (47%) with both basal septal hypertrophy and abnormal Q waves. In the diffuse and the apical type cases, RV hypertrophy appeared to be more closely related to the presence of abnormal Q waves than in the basal type cases: the incidence was 75% (9/12) in the diffuse type patients; in the apical type patients RV hypertrophy was found in all 4 patients with abnormal Q waves. The incidence of abnormal Q waves in the patients with RV hypertrophy was inversely related to the location of maximum septal hypertrophy. The incidence decreased from 88% (7/8) in the basal type to 64% (9/14) in the diffuse type and finally to 29% (4/14) in the apical type, and the difference between the basal and the apical type patients was statistically significant (p < 0.005).

Other clinical characteristics, including age, sex, the degree of symptom, LV outflow tract obstruction and so on, were not significantly related to abnormal Q waves and absence of Q waves.

Location of Abnormal Q waves

Abnormal Q waves were arbitrarily classified as lateral Q (Q waves in either lead I, aVL, V5 or V6 (21 cases)), inferior Q (Q waves in either lead II, III or aVF (9 cases)) and anteroseptal Q (Q waves in either lead V1, V2 or V3 (3 cases)). The relation of RV hypertrophy to the location of abnormal Q waves is indicated in Table III. RV hypertrophy was frequently noted in the patients with lateral Q (14/21, 67%) or inferior Q waves (6/9, 67%); in contrast, the incidence was relatively low in the patients with anteroseptal Q waves (1/3, 33%). RV anterior free wall hypertrophy was more prevalent in the patients with inferior Q (6 cases, 67%) than in the patients with lateral Q waves (5 cases, 24%, p < 0.05), while the incidence of moderator band hypertrophy was relatively high (12 cases, 57%) in the patients with lateral Q as compared with that in the patients with inferior Q waves (3 cases, 33%).

In addition to the appearance of abnormal Q waves, tall R waves in lead V1 are often noted in patients with hypertrophic cardiomyopathy. In the present study, R wave of 10 mm or more in lead V1 was noted in 9 cases, in 8 of whom an association with abnormal Q waves in limb leads and/or left precordial leads was found. This value is significantly higher than the incidence of Q waves in those patients with R wave of less than 10 mm in lead V1 (35% of the 65 patients) (p < 0.001).

DISCUSSION

Although abnormal Q waves are well known as one of the characteristics of hypertrophic cardiomyopathy, its overall incidence is as low as 40% according to Savage et al. This relatively low incidence of abnormal Q waves and the fact that there is no relation between the degree of asymmetric septal hypertrophy and abnormal Q waves suggest that the appearance of abnormal Q waves cannot be fully explained by the concept of an increased electrical force generated by the hypertrophied septum alone. The cancelative effect of free wall hypertrophy on the initial septal force and a diminution of the electrical force in the involved myocardium resulting from histological changes have been considered as possible explanations for this discrepancy. However, the relationships between these anatomical features to the abnormal Q waves have never been fully examined.

Our study revealed several correlations between myocardial hypertrophy and abnormal Q waves by means of a comparative assessment of echocardiographic and electrocardiographic findings. First, the septal hypertrophic pattern along the LV long axis was closely related to the appearance of abnormal Q waves. Abnormal Q waves were significantly more prevalent in the basal type patients than in the diffuse type patients and the apical type patients, while "absence of Q waves" was more frequently noted in the apical type patients than in the other 2 groups. These data, in conjunction with findings in recent M-mode echocardiographic studies, indicate that the determinant for the development of abnormal Q waves is not the absolute thickness of the interventricular septum, but rather the relative degree of hypertrophy in the basal septum and the apical septum. In other words, basal septal hypertrophy may intensify the initial septal force directed right anteriorly and superiorly or inferiorly while the apical myocardium may counteract the appearance of Q waves by intensifying the electrical component directed left anteriorly and inferiorly. This hypothesis seems to be compatible both with the concept of an increased septal force as the mode of genesis and with the fact that abnormal Q waves...
waves are relatively low in patients with septal hypertrophy.

A variation of myocardial hypertrophy in hypertrophic cardiomyopathy has been indicated in recent 2-D echocardiographic studies18—20. An extension of hypertrophy is frequently observed in the LV anterior wall19 and papillary muscles18 but can rarely be seen in the postero-inferior wall. In the present study, it was demonstrated that myocardial hypertrophy often extended even to the RV structures adjacent to the anterior half of the interventricular septum and this RV involvement can be related to the abnormal Q waves. The incidence of abnormal Q waves was significantly higher in the patients with RV hypertrophy than in the patients without RV hypertrophy. Furthermore, close relationships between the RV anterior free wall hypertrophy and the inferior Q wave and those between moderator band hypertrophy and the lateral Q wave were documented. These data, in conjunction with a disproportionate distribution of hypertrophy in this disease, predominantly involving the interventricular septum and the LV anterior wall, could further support the idea that accentuation of the electrical force in the hypertrophied myocardium is the primary mechanism for the genesis of abnormal Q waves in this disease, that is, the increased electrical force resulting from the septal hypertrophy associated with RV anterior free wall hypertrophy is probably in the direction opposite to that of the LV inferior wall, and similarly the force generated by an association with RV moderator band hypertrophy may counteract and overcome that of the LV lateral wall.

Our data does not support the idea that the relatively low incidence of abnormal Q waves can be explained by the cancellative effect of LV free wall hypertrophy. This could be further confirmed by the fact that the precordial QRS voltage (SV2 + RV5) in the patients with and without abnormal Q waves was not significantly different (55.8 ± 22.2 vs 62.0 ± 22.5 mm).

Thus, the abnormal Q waves in hypertrophic cardiomyopathy appeared to be explained by their anatomical characteristics. However, some exceptions were also noted. For example, the right-anteriorly directed initial force could not be considered as the mechanism for the genesis of antero-septal Q waves. Furthermore, the absence of septal Q waves observed in a patient with basal septal hypertrophy suggests that the abnormality in the early electrical activation of the ventricle can not be entirely explained by the relative degree of hypertrophy in the basal septum and the apical septum. Burch et al14 have described a close relationship between septal fibrosis and the absence of septal Q waves. Blackhill et al21 have demonstrated a vectorcardiographic similarity between hypertrophic cardiomyopathy and aortic stenosis, and proposed the electrical internal cancellation within the septum in hypertrophic cardiomyopathy as the reason for this similarity. Some evidence of an abnormal electrophysiological property in the cardiomyopathic septal muscle has also been found22,23. Coltart and Meldrum24 have reported a decrease of maximum upstroke velocity and a reduction in the maximum rate of follow in septal muscles excised at the time of surgery. Furthermore, disappearance of abnormal Q waves occurring after an atrial stimulus of relatively late coupling or atrial pacing has been reported by Cosio et al23. In one of the present subjects with a typical subaortic stenosis, disappearance of abnormal Q waves in limb and left precordial leads associated with a development of QS pattern in V1 was documented without any apparent changes in cardiac status during the observation period. It is tempting to speculate that abnormal electrophysiological properties in the bizarre disarrayed septal muscles affect the overall direction and magnitude of the initial vector as the mechanism for the genesis of abnormal Q waves, especially in patients in whom the abnormal properties can not be explained by the myocardial hypertrophic pattern.

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