Giant T Wave Inversion as a Manifestation of Asymmetrical Apical Hypertrophy (AAH) of the Left Ventricle

Echocardiographic and Ultrasono-cardiotomographic Study

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SUMMARY

Left ventricular scanning by echocardiography and ultrasono-cardiotomography was performed to search the possible muscular abnormality in 9 cases with giant T wave inversion without documented cause. The deeply inverted T wave was more than 1.2 mV (average was 1.63 mV) in the left precordial leads. All the cases had electrocardiographic left ventricular hypertrophy of obscure origin and ischemic episode was absent. Conventional echo beam direction to measure the short axis of the left ventricle disclosed almost normal thickness and movement of both interventricular septum (IVS) and the posterior wall (PW), so that the report of these cases is frequently within normal limits. However, ultrasono-cardiotomography (sector B scan) disclosed the fairly localized hypertrophy near the left ventricular apex, and conventional echocardiography also revealed the same area of either IVS or PW or both below the insertion of the papillary muscles, when the scanning towards the apex was performed (asymmetrical apical hypertrophy: AAH).

Control study of 9 cases with IHSS showed asymmetrical septal hypertrophy (ASH) with almost equally hypertrophied IVS from base to apex. All cases had inverted T waves, but these were of lesser degree. Three cases had relatively deep T wave compatible with those of AAH, and these cases also had the apical hypertrophy of considerable degree (unusual type of IHSS, i.e., intermediate type between AAH and ASH). The close relationship between the depth of the inverted T waves and the Apex/Mid wall thickness ratios suggests that the altered recovery process of the hypertrophied apical musculature is responsible for the giant T wave inversion of heretofore unsolved origin. Until the connective link of AAH to the other forms of hypertrophic cardiomyopathy is disclosed, the cases with such a T wave and the apical hypertrophy may be designated as asymmetrical apical hypertrophy (AAH).
GIANT T wave inversion is an infrequent electrocardiographic finding, and many conditions have been attributed to the causes\(^1\) such as (1) Adams-Stokes attacks associated with complete heart block; (2) ischemic heart disease; (3) bradycardia; (4) right ventricular hypertrophy and right bundle branch block; (5) metabolic disturbances; (6) changes during coronary angiography; and (7) cerebral disturbance.\(^2\) However, the association of “localized” left ventricular hypertrophy with deeply inverted T waves in the left precordial leads has not been described yet. In view of the fact that the ultrasoundcardiography revealed a particular type of ventricular wall thickening in all of those with the giant negative T waves of unknown cause, the present report deals with the etiologic significance of such a particular left ventricular hypertrophy in the mechanism of this electrocardiographic abnormality.

**Materials and Methods**

Nine patients with giant T wave inversion with unknown cause were studied (Group I). They represent the total number of patients observed in our laboratory with giant T wave inversion and echocardiographic examinations. They were 7 males and 2 females, and the age ranged from 25 to 70 years with an average of 43. Electrocardiographic criteria for identification were a negative T wave of 1.2 mV or more in leads V\(_4\) to V\(_6\), no arrhythmia, and no widening of the QRS complex. No attempts were made to select patients by cardiac diagnosis.

All 9 patients underwent physical examination, standard 12 leads electrocardiography, vectorcardiography, phonoe- and mechanocardiography, chest X-rays, echocardiography and ultrasonocardiotomography. History taking revealed palpitation in 4, mild chest oppression in 1, and neither myocardial infarction nor cerebrovascular accident was present. All the cases had normal sinus rhythm and the heart rate was from 54 to 74 with a mean of 62 per min. Blood pressure was in the normal range with a mean of 138/68 mmHg, including 3 cases with mild hypertension. The cardiothoracic ratio was 51.6% in average, with cases of mild cardiomegaly.

Control study was made on 9 patients with idiopathic hypertrophic subaortic stenosis (IHSS) without arrhythmia or bundle branch block (Group II). They were 7 males and 2 females, and the age ranged from 29 to 50 years with an average of 42. The diagnosis of IHSS was made by clinical grounds, and the echocardiographic diagnostic criteria were the thickness of interventricular septum (IVS) of 14 mm or more, its ratio to the posterior wall (PW) thickness of the left ventricle of 1.3 or more,\(^3\) and the presence of the unequivocal systolic anterior

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**Additional Indexing Words:**
- Hypertrophic cardiomyopathy
- Idiopathic hypertrophic subaortic stenosis (IHSS)
- Asymmetrical septal hypertrophy (ASH)
- Asymmetrical posterior wall hypertrophy (APWH)
- Unusual type of IHSS
- Ventricular gradient (G)
- Ventricular wall thickness
movement (SAM) of the so-called mitral valve echogram.\textsuperscript{4)}

Electrocardiogram recorded were, as mentioned above, standard 12 leads, Frank system scalar electro- and vectorcardiogram, and the depth of the inverted T waves was measured in the left precordial leads (V\textsubscript{4}-V\textsubscript{6}). QTc was obtained from the maximum value in the standard limb leads. Ventricular gradient (G) was measured in the Frank system scalar electrocardiogram with a paper speed of 100 mm/sec. Exercise test was performed using treadmill up to the level of 85% of the maximum heart rate standardized by the age.

Echocardiograms were obtained with an Aloka SSD 100 using an unfocused 2.25 MHz transducer of 10 mm in diameter, and recorded on a strip chart recorder. Examination was performed with the patient in supine position, and the transducer was placed in the third or fourth intercostal space at the left sternal border (i.e., 3L and 4L).

The left ventricular end-systolic and end-diastolic dimensions (LVDs and LVDd) were measured at the time of the aortic component of the second heart sound (IIA) and of the beginning of the QRS. The excursion of IVS and PW was measured at the point of their maximum amplitude. The mean Vcf was ob-

![Fig. 1. Demonstration of the 3 main positions of the echocardiographic measurement of the ventricular wall thickness. Ultrasound-cardiotomogram of end-diastolic phase is depicted to clarify the beam direction. 20 year-old normal subject.](image)

The echo beam directions are; B: base direction observing both anterior and posterior mitral leaflets (AML and PML), M: mid portion obtaining conventional short axis, and A: apex direction below the insertion of posterior papillary muscle. The wall thickness is measured in these 3 directions on the M-mode scan echocardiogram (cf. Fig. 2). RV: right ventricle, IVS: interventricular septum, LA: left atrium, LV: left ventricle, PW: posterior wall of left ventricle.
Fig. 2. Echocardiograms obtained from 3 directions demonstrated in Fig. 1. Case 1 in Group I, 33 year-old male.
Position B (left) and M (middle) show almost normal thickness of the wall, but position A (right) shows the markedly thickened wall and the narrowing of the ventricular cavity. Time lines: 0.05 sec. Paper speed: 50 mm per sec.
tained according to the formula of Fortuin et al. 5)

**Special procedures**: To compare the wall thickness of the various areas of the left ventricle, the M-mode scan was performed in all along the long axis and 3 main positions were selected. Namely, the beam was firstly directed inferiorly to obtain the mitral valve echo (both anterior and posterior leaflets) (Position 1: Base), secondly to obtain the ordinal short axis of the left ventricular cavity (Position 2: Mid), and finally to obtain the left ventricular apex as closer as possible (Position 3: Apex) (Figs. 1 and 2). The measurement of the thickness of the wall at these 3 sites was done at the timing of the beginning of the Q wave in the electrocardiogram, and the ratio of Apex/Base (or Apex/Mid) was calculated for either IVS or PW. Unfortunately, however, this ratio could not be obtained in some cases with extremely narrow ventricular cavity due to the hypertrophy of the wall (2 cases of Group I).

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age &amp; Sex</th>
<th>HR (beats/min)</th>
<th>BP (mm Hg)</th>
<th>CTR (%)</th>
<th>$S_{v_r} + R_{v_r}$ (mm)</th>
<th>Maximum Negative T (mV)</th>
<th>QTc (sec)</th>
<th>LVDD (mm)</th>
<th>LVDs (mm)</th>
<th>Excursion of IVS (mm)</th>
<th>Excursion of PW (mm)</th>
<th>Mean Vcf (circ/sec)</th>
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<tr>
<td>12</td>
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<td>18+22</td>
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<td>59</td>
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<td>54.0</td>
<td>28+36</td>
<td>-0.76</td>
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<td>39.0</td>
<td>20.4</td>
<td>6.7</td>
<td>14.7</td>
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</table>

Ultrasono-cardiotomogram\(^6\),\(^7\) (two-dimensional echocardiogram; sector B scan) was obtained with an Aloka SSD 100 along the long axis of the heart placing the focused (10 cm) 2.25 MHz transducer of 13 mm in diameter over the site 1 intercostal space lower than the usual echocardiographic position. The tomogram was mainly obtained at the timing of the Q wave.

**RESULTS**

Tables I and II summarize the data obtained from the various measurement of the individual case.

I. Electrocardiographic findings

1) Electrocardiogram and vectorcardiogram at rest (Fig. 3)

Usually, giant T wave inversion was markedly observed in V\(_5\), except

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Interventricular Septum (IVS)</th>
<th>Posterior Wall (PW)</th>
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<tr>
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<td>Thickness (mm)</td>
<td>Thickness (mm)</td>
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<td>Base  Mid  Apex</td>
<td>Apex/Base  Apex/Mid</td>
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<td>Apex/Base Apex/ Mid</td>
<td>Apex/Base Apex/ Mid</td>
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<td>Group I: Giant T wave inversion (AAH)</td>
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<td>12 12 30</td>
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<td>9</td>
<td>9 10 —</td>
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<tr>
<td>Average</td>
<td>11.8 11.9 23.4</td>
<td>12.0 11.6 26.9</td>
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Group II: IHSS (ASH)

<table>
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<th>Case No.</th>
<th>Interventricular Septum (IVS)</th>
<th>Posterior Wall (PW)</th>
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<tbody>
<tr>
<td></td>
<td>Thickness (mm)</td>
<td>Thickness (mm)</td>
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<td>Base  Mid  Apex</td>
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<tr>
<td>Average</td>
<td>20.8 18.1 22.8</td>
<td>12.8 15.7 23.8</td>
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3 cases in which $V_4$ showed the deepest T wave (Case 9 in Group I, Cases 12 and 15 in Group II). There was no case in which $V_6$ had the deepest T. The negativity was $-1.63\, \text{mV}$ ($-1.2$ to $-2.1\, \text{mV}$) in Group I, whereas it was

![Image of electrocardiogram and vectorcardiogram](image)

**Fig. 3.** An example of electrocardiogram and vectorcardiogram in Group I. Case 2, 50 year-old male.

The negativity of T in $V_4$ is $-2.0\, \text{mV}$ (left). Frank system scalar electrocardiogram (upper right) is for the measurement of ventricular gradient. Frank system vectorcardiogram (lower right) shows the rightward deviation of T loop in the frontal and horizontal planes.
-0.76 mV (-0.3 to -1.4 mV) in Group II. There were 3 cases in Group II, in which relatively deep T wave inversion was present (Cases 16~18), and the deepest was -1.4 mV (Case 17). As mentioned in the item of echocardiographic findings, these 3 cases were the important link between 2 groups.

The duration of the QRS was less than 0.10 sec in both groups, and there was no significant difference in the prolonged QTc and the increased amplitude of SV₁ or RV₅ between the 2 groups.

Vectorcardiogram in Group I disclosed large and rightward T loop in all cases (Fig. 3).

2) Follow-up observation (Fig. 4)
A daily or weekly follow-up of the electrocardiogram disclosed no change

Fig. 4. Serial electrocardiogram in the follow-up study.
Left (Case 2): In the 4 stages, the height of R wave was 2.5, 3.2, 4.6 and 4.6 mV, respectively, whereas that of T wave was -0.4, -0.6, -1.6 and -2.0 mV, respectively. Progressive change in the T wave is much more marked than that of R wave.

Right (Case 1): Throughout the 3 stages, no change in the height of R wave was observed (3.6 mV in all), whereas the negativity of T wave progressively increased (-0.7, -1.2 and -1.8 mV, respectively).
in both groups. However, semi-annual or annual follow-up disclosed significant change in 2 cases of Group I. The progressive increase in both height of RV5 and negativity of T was observed in 1 (Case 2) and the increased negativity of T without significant change in RV5 in another case (Case 1). Unfortunately, the complete echocardiographic study including left ventricular scan was not performed in the past, the follow-up observation of the special area described below was not performed.

3) Treadmill stress test (TST) (Fig. 5)

During or after the exercise, further depression of ST segment was not observed. The deeply inverted T wave tended to be less marked during exercise. Neither anginal episode nor arrhythmias were observed despite the maxmum load (MTST).

4) Ventricular gradient (G)

The measurement values of G of 5 cases in Group I were; $X = -23.2$ to $-80$ (average $-45.5) \mu V\text{sec}$; $Y = -1.3$ to 40 (average 14.5) $\mu V\text{sec}$; and $Z = 1.7 \mu V\text{sec}$ in average. As shown in Fig. 6, these were far out of the normal range. $^8$ Exercise did not cause the change in the direction of G, though the decrease in the amplitude was observed.

![Fig. 5. An example of treadmill stress test. Case 2.](image)

Heart rate changed from 58 to 115 per min, but no ST change was observed, T wave became slightly less inverted.
Fig. 6. Ventricular gradient. See text.
Shaded area: normal range. Five dots were obtained from Case 1, 2, 4, 5 and 6, respectively.

II. Echocardiographic and ultrasono-cardiotomographic findings
1) Routine observation
LVDD was slightly increased or near the upper normal limits in Group I and LVDs was within the normal range. The excursion of IVS and PW

Fig. 7 (opposite page). M-mode (sector) scan demonstrating the changes in wall thickness and the cavity of the left ventricle.
Top: Normal subject. Left ventricular cavity gradually becomes narrow towards the apex, but the wall thickness remains unchanged.
Middle: Asymmetrical apical hypertrophy (AAH). Case 3. Normal echogram is obtained at the base and mid positions, but there is marked thick-
ening of PW near the apex, resulting in the narrowing of the ventricular cavity.

Bottom: Asymmetrical septal hypertrophy (ASH). Case 10. IVS is most conspicuously thickened at the base, but also at the apex. PW shows thickening of mid and apex positions. SAM is typical.

Paper speed: 25 mm per sec, time lines: 0.1 sec.
was not impaired in Group I, whereas the excursion of IVS was inevitably reduced in Group II. Mean Vcf was increased significantly in both groups (normal is 1.14 in average).

2) Special procedures

Fig. 7 demonstrates the M mode scan from the left ventricular apex to the aorta in the representative cases of normal (top), Group I (middle) and Group II (bottom).

In normal cases, the M mode scan along the long axis of the left ventricle showed almost the same thickness of the wall of both IVS and PW and the same diameter of the cavity from the apex to base. The narrowing of the cavity near the apex was not so conspicuous. On the other hand, cases with giant negative T wave (Group I) showed a peculiar pattern. Namely, the wall had no thickening, therefore the movement was well reserved, as mentioned above, from the base to the mid-portion of the left ventricle. However, there was a marked thickening of the wall near the apex, giving the striking narrowing of the cavity. The cases with IHSS (Group II) showed conspicuous thickening of the IVS at the base, mid-portion as well as apex, so that the abrupt change in the cavity size towards the apex was never observed.

Fig. 8 illustrates the above-mentioned situation by ultrasono-cardiotomogram taken at the end-diastolic phase along the long axis of the left ventricle. The enormously thickened posterior wall of the left ventricle was well depicted, the location of which was far from the base in this case of Group I (Fig. 8 middle).

The location and the size of the localized thickening of the left ventricular wall were varied from case to case. Fig. 9 illustrates the localized thickening of marked degree of the IVS near the apex, giving the selective narrowing at the apex (Case 5). Fig. 10 illustrates the same finding of both IVS and PW near the apex, giving rather immobile wall and narrowed apical cavity (Case 4). In these 2, the mid-portion and the base showed normal thickness as well as movement of the wall and no narrowing was present, so that routine echocardiography in the past could not detect the serious abnormality.

3) Comparison of the wall thickness

To outline the unusual situation in 2 groups, the thickness of the base, mid-portion and apex of the left ventricular wall was measured and compared with each other (Table II).

Though the cases of Group I showed normal or occasionally (Cases 6 and 7 with mild hypertension) slightly increased thickness of the base and mid-portion, unproportionally thickened wall was observed near the apex of either IVS or PW. In 2 cases (Cases 7 and 9), the accurate measurement of the abnormally thickened wall in the apical portion was not done, however,
Fig. 8. Ultrasono-cardiotomograms demonstrating the changes in the wall thickness and the cavity of the left ventricle. Same patients as in Fig. 7. Tomograms were taken along the long axis of the left ventricle and obtained at the end of diastole. A: Normal, B: AAH, and C: ASH.
Fig. 9. Illustration of the apical narrowing by atypical hypertrophy.
Case 5.
Mid to apical thickening of IVS and subsequent narrowing of the left ventricular cavity are demonstrated by ultrasono-cardiotomography. Note the absence of the thickening of the base of IVS, resulting in no outflow obstruction.

Fig. 10. M-mode scan in a case of AAH. Case 4.
Both IVS and PW show the apical hypertrophy of marked degree and the movement is less marked. The mid portion has normal echogram.

the tendency that the apex was almost twofold thicker than the other portion was clearly observed.

On the other hand, the cases of Group II showed thickened IVS almost
Fig. 11. Comparison of the thickness of IVS (left) and PW (right). The Apex/Mid wall thickness ratio was plotted against the depth of the inverted T wave (lower panel). Three cases of IHSS (ASH) are in between the 2 groups (dotted line).
equally from the base to the apex. Noteworthy, 3 cases (Cases 16, 17, and 18) showed minor hypertrophy of the basal and mid-portion of IVS and PW, whereas the unproportionally thicker wall was present at the apex, and these cases had relatively deep T wave inversion (Table I).

III. Echocardiographic correlates of the giant T wave inversion

Fig. 11 demonstrates the correlation between the absolute value of the inverted T wave of the individual case and the grade of the apical hypertrophy judged by the ratio to the other portion (i.e., Apex/Mid ratio). The ratio was calculated in both IVS (Fig. 11 left) and PW (Fig. 11 right), respectively. The figures clearly demonstrates that the deeper the negative T wave, the more marked the unproportional thickening of the apical portion. Three cases of Group II, in which the relatively deep T wave inversion was present as mentioned above, were in between the Group I and the remainder of Group II.

DISCUSSION

As the cause of the giant T wave inversion in the present study, coronary heart disease should be excluded, because the possibility of any other causes
are easily eliminated on the clinical ground. Moreover, as Pruitt et al\textsuperscript{11} stated, there was neither clinical picture compatible with the diagnosis of ischemic heart disease, nor the characteristic sequence of the T wave alteration at least within a month. Though the study of the ventricular gradient suggested that the T wave change in question is primary, the exercise test in Group I was negative, and the change in the T wave was progressive but very slowly, as demonstrated in Fig. 4. Therefore, the cause of the giant T wave inversion in the present study should be different from those proposed by the previous paper.\textsuperscript{21} In this respect, it is noteworthy that there was unproportional thickening of the wall near the apex of the left ventricle in cases with giant negative T wave.

Recent years, an estimate of wall thickness of IVS and PW can be made from determination of the transverse axis as recorded echocardiographically.\textsuperscript{9)-13)} Generally, left ventricular hypertrophy causes symmetrical hypertrophy, i.e., proportional hypertrophy of both IVS and PW. An important exception is observed in cases with IHSS, in which asymmetrical septal hypertrophy (ASH) is regarded as the pathognomonic anatomic abnormality.\textsuperscript{31} However, the changes in the wall thickness of the other echo beam direction have not been quantitatively investigated yet. For some time past, the present authors have been used to scan echocardiographically as well as ultrasono-cardiotomographically the whole cavity of the left ventricle to detect the specific foci of the movement responsible for the diastolic heart sounds.\textsuperscript{14,15)} Care must be taken, however, for the measurement of the wall thickness of the area far from the usual transverse echo beam direction, because the oblique projection of the beam may erroneously result in the exaggerated wall thickness. In this regard, one should be extremely careful to obtain the long axis of the left ventricle in such a way that the cavity must be depicted as large as possible during the scanning. As a rule, thick wall has relatively poor movement as seen in the IVS of IHSS.\textsuperscript{16)} An erroneously obtained thick wall and narrow cavity by the oblique projection usually accompanies with the movement of the structure without decreased amplitude, so that the incorrectness of the echo beam direction is readily noticed. Furthermore, the real thickness of the apical portion should be assessed by the echo beam direction towards the areas below the papillary muscles. This study is also accomplished by the use of ultrasono-cardiotomography, by which the correctness of echocardiography was verified.

The hypertrophy of the left ventricular wall may cause the inversion of T waves in the left precordial leads. In fact, all cases with IHSS (Group II) had the inverted T in V\textsubscript{6}, but the degree was not so marked except a few cases. In Group II, the thickening of the wall was present overall IVS
(from base to apex), and the thickening of the PW was also present, though
the hypertrophy is asymmetrical (ASH). On the other hand, the distribu-
tion of the hypertrophy in Group I was quite different, because there was
the localized hypertrophy of marked degree below the insertion of the papil-
lary muscles, giving the high Apex/Mid thickness ratio (asymmetrical apical
hypertrophy: AAH) (Fig. 11).

The present study may suggest 2 possibilities, i.e., the T wave inversion
in hypertrophic cardiomyopathy may depend on the disparity of the wall
thickness, and there may be the close connection between Groups I and II,
i.e., AAH and ASH.

For the first possibility, the prolonged QTc may have some significance.
In both AAH and ASH, the prolonged QTc implies the prolonged recovery
process and this may be due to the hypertrophy of the ventricular wall in both
groups. However, the duration of the recovery process of the hypertrophied
muscles in various areas is almost the same in cases with diffusely hypertro-
phied wall in ASH, resulting in the inversion of the T wave of lesser degree.
On the other hand, AAH has localized apical hypertrophy, so that there may
be the difference in the duration of the recovery process between the apex
and the other areas, giving rise to the giant T wave inversion. The facts that
there were 3 cases with large T wave inversion in ASH group, in which the
Apex/Mid thickness ratio was abnormally high, and that there was a close
relationship between the negativity of the T wave and the unproportional
hypertrophy of the apical region, may imply the validity of the first possi-
bility. The fact that the ventricular recovery process had right, superior and
anterior direction (Fig. 3) further supports this hypothesis.

The second possibility is the problem of many speculations. The case
with giant T wave inversion of unknown origin has undoubtedly the localized
hypertrophy of the apical area of the left ventricle (AAH), and this may
progress slowly and result in more typical electrocardiographic and echocardi-
ographic appearance. At the present time, there is no basis to link AAH and
ASH, though intermediate form was present (Cases 16~18 in Group II). In
order to conclude, we should have the individual patients with development
from AAH to ASH. It should be emphasized, however, that there are several
patterns of left ventricular hypertrophy, in which the apical\textsuperscript{17,18} or mid-ventricu-
lar\textsuperscript{19} hypertrophy was included. In rare instance, the localized hyper-
trophy of the posterior wall is observed\textsuperscript{20} (Fig. 12) (asymmetrical posterior
wall hypertrophy: APWH). Until the connective link of these disease entities
is solved, the designation of AAH (asymmetrical apical hypertrophy) may
be proposed to a special type of left ventricular hypertrophy, which is
characterized by the giant T wave inversion in the left precordial leads.
ACKNOWLEDGEMENT

The authors wish to thank Dr. S. Mashima for his advice.

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