IS IT POSSIBLE TO RULE OUT EXTENSIVE ANTERIOR MYOCARDIAL INFARCTION IN THE ABSENCE OF ABNORMAL Q WAVES IN LEAD I AND aVL?: EFFECT OF INFERO-APICAL EXTENSION OF INFARCTION OVER APEX

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To determine whether abnormal Q wave in lead I or aVL may be of use to estimate the size of an extensive anterior myocardial infarction, electrocardiographic and left ventriculographic findings were analyzed in 45 patients with old extensive anterior infarction. All 45 patients had a significant narrowing in the proximal segment of left anterior descending coronary artery (LAD) and severe asynery in anterolateral segment. The patients were divided into two groups; Group I consisted of 35 cases with less involvement of the inferopical segment and Group II of 10 cases with remarkable extension of the anterolateral infarction into the inferopical segment due to occlusion of very long LAD supplying the anterior half of posterior interventricular groove. There were no statistical differences in the extent of anterolateral asynery, number of abnormal Q waves in precordial leads and left ventricular ejection fraction between the two groups. While abnormal Q wave in lead I or aVL was present in 28 cases (80%) of Group I, it was observed in only 3 cases (30%) of Group II (p < 0.01). Thus, we can't rule out extensive anterior myocardial infarction even if abnormal Q waves are absent in lead I or aVL, in which abnormal Q waves may be cancelled by loss of electromotive force of inferopical segment due to extension of the anterior infarction over the apex in cases with extraordinarily long LAD.

ABNORMAL Q waves on electrocardiogram (ECG) are utilized to diagnose myocardial infarction (MI) and their value in this regard has been proved by many authors. Conventionally, the number of Q waves in standard 12 lead ECG was utilized as an index for semiquantitative evaluation of the size of MI without definite proof until Wagner et al.1-3 compared pathological findings with number, duration and depth of abnormal Q waves. In their papers, abnormal Q waves in lead I and aVL were used only for estimation of anterior MI with same significance as abnormal Q waves in precordial leads.2 On the other hand, abnormal Q waves in lead I and aVL have been considered to indicate "high lateral" infarction since Myers4 described abnormal Q wave in lead aVL as index of lateral MI. However, except for his classic report, we can't find any description in which abnormal Q waves in lead I and aVL were compared with

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pathological, clinical or angiographical findings or other variable methods which could suggest the localization of MI. Griffith noted that in anterior infarction abnormal Q waves in lead I or aVL indicated occlusion of LAD proximal to the origin of the diagonal branch. However, there was no accurate description comparing ECG with angiogram in his paper. Thus, although the presence of abnormal Q waves in lead I and/or aVL in cases of anterior MI have generally been considered as a sign of extension of MI to “high lateral” wall, it still remains to be confirmed. In this paper, we tried to clarify the values and limitations of abnormal Q waves in lead I or aVL in estimating the size of infarction in cases with “extensive” anterior infarction in which “high lateral” wall as well as anteroseptal wall was considered to be involved in the infarction on angiogram.

METHODS
Patients: Of 3,000 serial cases who had selective coronary arteriography in our laboratory between March 1975 and October 1984, 45 study patients were selected according to the following criteria:
(1) The patient survived an acute MI at least 4 weeks prior to the angiographic study.

(2) The coronary arteriography showed a significant stenosis (a 75% or more narrowing of the luminal diameter) in LAD proximal to all its major branches.
(3) The left ventriculogram (LVG) showed severe asynergy (akinesia or dyskinesia) in the anterolateral and the septal segment?
(4) The coronary arteries other than LAD had no significant stenosis.

TABLE I PRESENCE OR ABSENCE OF ABNORMAL Q WAVE IN LEAD I OR aVL IN GROUP I AND GROUP II.

<table>
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<tr>
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<th>Lat. Q(+)</th>
<th>Lat. Q(−)</th>
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<tbody>
<tr>
<td>Group I (35 cases)</td>
<td>28 cases (80%)</td>
<td>7 (20%)</td>
</tr>
<tr>
<td>Group II (10 cases)</td>
<td>3 (30%)</td>
<td>7 (70%)</td>
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Abbreviations are the same as in Fig. 2.

(5) ECG showed no bundle branch blocks, Wolff-Parkinson-White pattern or left anterior hemiblock.

ECG: Standard 12 lead ECG was recorded within 48 hours prior to angiography, and q wave equal to or wider than 30 msec was defined as abnormal Q wave.

Angiography: Selective cine coronary arteriography and left ventriculography were performed at 30 degree right anterior oblique (RAO) and 60 degree left anterior oblique projections with an injection of 21 to 28 ml of sodium meglumine diatrizoate. Cinefilms were recorded at 30 frames/sec using 35 mm film. Severe asynergy included akinesis and dyskinesis which were defined with quantitative measurement using chord system. The severe asynergies of inferoapical segment (B in Fig. 1) as well as of anterolateral segment (D in Fig. 1) were measured in the end-diastolic frames. The extent of severe asynergy was expressed as a ratio in relation to the entire inferior segment (Fig. 1 B/A) or to the entire anterolateral segment (Fig. 1, D/C). Left ventricular ejection fraction (LVEF) was measured using biplane method.

Statistical analysis: Statistical analyses were performed using chi-square method or unpaired Student t test. B/A, D/C and LVEF values were expressed as mean ± one standard deviation.

RESULTS

Figure 2 shows the correlation of the presence or absence of abnormal Q wave in lead I and/or aVL (Lat. Q) with the extent of inferoapical asynergy (B/A). The 45 cases were divided into

A Case 1
No.1972 K. A. 54 y.o. male

I — V1
II — V2
III — V3
V — V4
V6

Fig.3. Cases 1 (Group I) K.A. 54 y.o. male
The patient had an acute MI without any complications 2 months prior to the angiographic study. (Fig. 3A); QS pattern and inverted T waves are present in lead aVL as well as in precordial leads (V1-V4). An ECG recorded 2 months prior to the acute infarction showed normal ECG with RS pattern and upright T in lead aVL. (Fig. 3B): Left upper panel: LAD has a 90% narrowing proximal to all its branches. However, it is not long and terminates just at the apex. Left lower panel: Right coronary artery is intact and its posterior descending branch is long. Right panels: Left ventriculography reveals anterolateral akinesis which does not extend into the inferoapical segment.
two groups; Group I (35 cases) with B/A less than 0.4, and Group II (10 cases) with B/A equal to or more than 0.4. There was no difference in the anterolateral extension of severe asynergy between the two groups (D/C = 0.70 ± 0.11 for Group I, 0.78 ± 0.09 for Group II; NS.) Also, there was no difference in LVEF between the two groups; LVEF = 47.2 ± 9.8% for Group I, 42.6 ± 12.2% for Group II; NS.) Table I shows the number of patients in Groups I and II with or without Lat. Q; whereas Lat. Q was present in 80% of cases (28/35) in Group I (Fig. 3), it was found in only 30% (3/10) of cases in Group II (Fig. 4). This difference was statistically significant (p < 0.01). The number of abnormal Q waves in precordial leads did not differ between the two groups (3.5 ± 1.0 for Group I, 4.0 ± 0.9 for Group II; NS).

Thus, in cases with large anterolateral infarction caused by occlusion of LAD proximal to all major branches, Lat. Q is more often absent in cases whose anterior MI extends into the inferoapical segment than in cases whose MI is confined to the anterolateral segment. There was no definite difference of ventriculographic or electrocardiographic manifestations between these two groups other than the inferoapical extension of MI and the presence or absence of Lat. Q.

DISCUSSION

In addition to conventional ECG or vectorcardiography, new methods such as echocardiography, nuclear cardiology, computed tomography and nuclear magnetic resonance imaging have been proven useful for estimation of the extent of MI.10-15 Body surface potential mapping is also said to be valuable for its estimation.16,17 However, standard 12 lead ECG is the most widely available and economic approach and seems relatively free of interobserver difference and it is very important to recognize advantages and limitations of ECG in estimating semiquan-
Abnormal Q in I and aVL

In this study, LVG was adopted as the standard for estimating the size of MI. Although pathological examination of the heart is mandatory for the ideal standard of estimation of MI in practice it is very difficult to have an opportunity to examine a necropsied heart with old MI associated with single vessel disease and recent ECG recording.

Although there have been no reports comparing the site of infarction between necrosis of myocardium on autopsy and asynergy on ventriculogram, it is generally accepted that the "anterolateral" segment and the "inferior" segment in RAO LVG reflect anterolateral and diaphragmatic wall of left ventricle, respectively. Recently, we proved that asynergies of anterolateral segment coincided with Lat. Q and probably also with anatomically "high lateral" wall, by comparing ECG and LVG in MI caused by occlusion of diagonal branches of LAD.

Griffith reported that Lat. Q in anterior MI indicated occlusion of LAD proximal to the first diagonal branch and large anterolateral MI. More recently, Wagner et al. proposed a scoring system for estimation of the extent of MI. Their scoring system utilizes Q waves in lead I and aVL as well as precordial leads in estimating the size of anterolateral MI. In that system, the presence of Lat. Q is regarded as one index of a large anterolateral MI. In fact, in the present study, 31 cases out of 45 (68.9%) anterior MI caused by occlusion of LAD proximal to its major branches had Lat. Q.

However, as seen in Table I, in very large anterolateral MI involving a large area of the inferoapical segment with its blood perfusion supplied by very long LAD, Lat. Q is frequently absent.

Accordingly, even if Lat. Q is absent we cannot rule out the possibility of the presence of a large anterior MI, especially when clinical features suggest pump failure. It is relatively well known that abnormal Q waves of a previous MI occasionally disappear when a new infarction takes place. This phenomenon is generally thought to be caused by electrical cancellation arising when the same changes in electromotive force occur at electrically opposite sites in the heart. This electrical cancellation might, at least partially, explain the apparently paradoxical results of this study; the loss of electromotive force in the inferoapical segment might cancel out that of the "high lateral" segment, which occupies anatomically (and probably electrically) a position opposite the inferior segment and might make ECG diagnosis confusing, as in cases of multiple MI caused by multiple coronary artery involvement.

In order to simplify the study, all the selected cases had anterior MI with a lesion in LAD proximal to all its major branches (diagonal branches, septal perforators). And in these cases both the lateral (or "high lateral") wall as well as the septum were thought to be infarcted, because, as described in methods, severe asynergies (akinesis or dyskinesis) were present in a large area of the anterolateral segment on RAO LVG, which, as described in results as D/C, involved at least about two-thirds of the whole anterolateral segment.

It is possible that electrical cancellation affects the presence or absence of Lat. Q more remarkably in cases of anterior MI caused by more distal occlusion of LAD. In such cases, loss of electromotive force of the lateral ("high lateral") wall is less extensive and more easily overcome by loss of electromotive force of the inferoapical wall, if LAD is long. In these cases, abnormal Q waves might appear in leads II, III or aVF instead of lead I or aVL when loss of electromotive force of the inferior wall overlapped that of the "high lateral" wall.

Of the 45 cases in this study, such a pattern of so-called "anteroapical" infarction was observed in only one patient in Group II; an additional 4 cases in Group II had rsR pattern in inferior leads, suggesting involvement of the inferior wall. No cases in Group I showed such patterns. Thus, in most cases of "extensive" anterior infarction, deep extension of infarction into the inferior segment was not definite on ECG, probably because of large loss of electromotive force in the "high lateral" wall.

LVEF is often utilized as an index of the size of MI in LVG as well as in echocardiogram and in radionuclide ventriculography. It is curious that there was no difference of LVEF between Groups I and II. One explanation of this apparent contradiction is the presence of compensatory hypercontractility of the inferoapical segment, which makes global LVEF a rather inaccurate index of MI size. Or, alternatively, the relatively small number of patients included in Group II (10 cases) may blur the possible difference between the two groups.

Clinical implications:
In cases of anterior infarction without previous inferoposterior MI, the presence of Lat. Q is, to some extent, one index suggesting necrosis of the “high lateral” wall supplied by diagonal branches of LAD. However, if Lat. Q is absent in anterior MI with congestive heart failure, shock or prominent elevation of cardiac enzymes, one should suspect that the MI may extend into the inferoapical segment, which may be confirmed with echocardiography, radioisotope ventriculography, or, contrast ventriculography.

REFERENCES
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