The increased use of coronary angiography and the progress made in various diagnostic imaging techniques for the diagnosis of ischemic heart disease (IHD) currently allows highly accurate diagnosis and treatment during the early phase of myocardial infarction (MI). However, the standard 12-lead electrocardiogram (ECG) is still the most common, basic examination method in daily clinical practice. Because of the small number of lead points and the absence of points on the right chest and the back, the location of the coronary artery lesions in IHD is roughly divided into the anterior wall and inferoposterior wall regions,1–3 and a more detailed diagnosis of the location is often more difficult.4 In addition, for anterior wall infarction, when the lesion is located more proximal than distal to the left anterior descending coronary artery (LAD), the infarct area extends further. In general, inferoposterior wall infarction related to the left circumflex coronary artery (LCX) or right coronary artery (RCA) is viewed as having favorable prognosis,5 but an infarction of the proximal region of the RCA frequently involves the right ventricle and often the prognosis is poor.6,7 Therefore, bearing reperfusion therapy in mind, a more detailed diagnosis of the coronary artery lesions responsible for the MI is required.7,8

In the present study, we examined whether by adding more specific lead points it is possible to better estimate the location of the affected vessels, and thus improve the diagnostic accuracy, from the ECG findings of patients with IHD.

Methods

Patients With MI

The onset of a MI is diagnosed from typical chest pain with characteristic ECG findings and elevation of the cardiac enzymes in the serum. The present study enrolled 121 patients with a first episode of MI (71 anterior wall, 50 inferoposterior wall) who underwent revascularization of the coronary artery in the first 12 h by thrombolytic therapy or percutaneous transluminal coronary angioplasty (PTCA). Patients with bundle branch block, intraventricular conduction disturbance, atrial fibrillation or Wolf-Parkinson-White syndrome were excluded.

Patients With Angina Pectoris

We analyzed the findings from the leads showing ST segment elevation in 89 patients with angina pectoris (AP) who underwent PTCA (Table 1) and in 28 patients (31%)

Table 1 Clinical Details of the Study Subjects

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
<th>Age (years)</th>
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<tbody>
<tr>
<td>Myocardial infarction</td>
<td>121 (84, 37)</td>
<td>63.4±10.7</td>
</tr>
<tr>
<td>Anterior wall</td>
<td>71 (51, 20)</td>
<td>63.2±10.1</td>
</tr>
<tr>
<td>Inferoposterior wall</td>
<td>50 (33, 17)</td>
<td>63.6±11.5</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>89 (69, 20)</td>
<td>65.8±8.8</td>
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who had double vessel disease. Patients who had not had a previous MI, or who had bundle branch block, intraventricular conduction disturbance, atrial fibrillation or Wolf-Parkinson-White syndrome were excluded.

Standard 12-Lead ECG
In all 121 patients with MI, the location of the lesion responsible for the infarction was confirmed by coronary angiography. We examined the ECG findings from 42 of the 71 patients with an anterior wall infarction and from 20 of the 46 cases of inferoposterior wall infarction, all of whom were shown by ECG to be in the acute phase at admission. With regard to the occurrence of ST segment deviation during the acute phase and the occurrence of an abnormal Q wave (≥0.04 s), decreased R wave and embryonic r in the various lead points were examined. For all 117 patients, the ECG recordings from the subacute phase in the first 1–3 weeks were compared with the angiographic location of the coronary artery lesions. Significant ST segment deviation was defined by an ST shift of 0.1 mV or more, measured 60 ms after the J point.

The following ECG findings were examined.
- Anterior wall leads: ST segment elevation in leads V1–4, QS wave in lead V1.
- Posterior wall leads: R/S >1 in lead V1.
- Inferior wall leads: ST segment elevation and Q wave in leads II, III and aVF.

Body Surface Mapping
For 105 of 206 patients with MI or AP, body surface mapping (BSM) was performed from 87 lead points, with 59 on the chest and 28 on the back according to the method of Yamada et al. In MI, the BSM findings were examined using departure maps which express the deviation from the normal on each lead point as the departure index (DI), calculated according to the following formula:

\[ DI = \frac{\text{patient potential} - \text{mean potential in the normal group}}{\text{standard deviation for the normal group}} \]

In AP, BSM was recorded during PTCA and the areas of ST segment elevation were also examined using isopotential maps.

Statistical Analysis
The diagnostic accuracy in identifying the location of the coronary artery lesions responsible for ischemia was indicated on each ECG index as its sensitivity, specificity and positive predictive value and then the indices were compared with each other using the test. A value of p<0.05 was considered statistically significant.

Results
Anterior Wall Leads
The responsible lesions were observed to be within the LAD in all 71 patients with an anterior MI, who presented with ST segment elevation, Q waves and decreased R waves in 3 consecutive precordial leads. In these patients, the location of the responsible lesion in either the proximal or distal section of LAD (#6 or #7 according to the AHA classification) could be identified from the ECG findings. Fig 1 shows representative ECGs.

We also evaluated the diagnostic accuracy of ST segment elevation in leads I and aVL, and QS wave in lead V1, which are regarded as the criteria for anterior wall infarction caused by a LAD #6 lesion.

Diagnosis of ST Segment Elevation in Leads I and aVL for LAD #6 Lesions
Of 42 patients with acute anterior wall infarction, 18 had LAD #6 lesions and 24 had LAD #7 lesions. ST segment elevation in leads I and aVL occurred in 16 of the 18 patients (89%) with LAD #6 lesions and in 10 of the 24 patients (42%) with LAD #7 lesions. This finding had a sensitivity of 89%, a specificity of 58% and a positive predictive value of 62% for LAD #6 lesions (p<0.01) (Table 2).

Diagnosis of QS Wave in Lead V1 for LAD #6 Lesions
Of the 71 patients with anterior wall infarction, including those who were in the subacute phase approxi-

![Fig 1. ECGs from patients with acute anterior wall infarction caused by a lesion in the left anterior descending coronary artery (LAD). (a) LAD #6 lesion: ST segment elevation and Q wave in leads I, aVL and V1–4. (b) LAD #7 lesion: ST segment elevation and regression of R wave in leads V1–3.](image-url)
ECG Findings in IHD

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mately 1–3 weeks after onset, 29 had LAD #6 lesions and 42 had LAD #7 lesions. QS wave in lead V1 occurred in 18 of the 29 patients (62%) with LAD #6 lesions and in 7 of the 42 patients (17%) with LAD #7 lesions. Thus, this finding had a sensitivity of 62%, a specificity of 83% and a positive predictive value of 72% for LAD #6 lesions (p<0.001) (Table 2).

Table 3  ST Variations During PTCA in Patients With Angina Pectoris

<table>
<thead>
<tr>
<th></th>
<th>No. of patients</th>
<th>No. of patients with ST elevation (%)</th>
<th>Lead showing ST elevation</th>
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<tr>
<td>LAD</td>
<td>44</td>
<td>43 (98%)</td>
<td>I,aVL,V5=V5</td>
</tr>
<tr>
<td>LCX</td>
<td>25</td>
<td>11 (44%)</td>
<td>I,aVL,V6/II,III,aVF,V5,V6</td>
</tr>
<tr>
<td>RCA</td>
<td>20</td>
<td>16 (80%)</td>
<td>II, III, aVF, V5</td>
</tr>
</tbody>
</table>

LAD, left anterior descending coronary artery, LCX, left circumflex coronary artery, RCA, right coronary artery, PTCA, percutaneous transluminal coronary angioplasty.

In 50 patients who had not shown any signs of anterior wall infarction and thus inferoposterior wall infarction was diagnosed, the lesion responsible for the infarction was found in the LCX for 12 patients and in the RCA for 38 patients.

R/S>1 in lead V1 revealed a LCX lesion in 6 of the 12 patients (50%) and a RCA lesion in 4 of the 38 patients (11%). Therefore, this finding had a sensitivity of 50%, a specificity of 99% and a positive predictive value of 60% for inferoposterior wall infarction caused by LCX lesions (p<0.001) (Table 2). Fig 2 shows the ECGs of a patient with a LCX #11 lesion, which has R/S>1 in lead V1 in the subacute phase. Because of the small number of patients with acute MI caused by LCX lesions, patients with AP who had undergone PTCA were used to study the ECG findings of LCX lesions (Table 3). For acute LCX lesions, ST segment elevation occurred in 11 of 25 patients (44%) and the relevant leads were either I, aVL, and V5, or II, III, aVF, V5 and V6. Fig 3a shows the ECG recording during PTCA for a LCX #13 lesion.

Inferior Wall Leads

ST segment elevation or Q wave in leads II, III and aVF occurred in 42 patients, of whom 38 (90%) had the RCA as the affected vessel (Fig 3b). In addition to these findings, 11 of 19 patients with an acute inferior wall infarction showed ST segment elevation in leads II, III and aVF, and ST segment depression in aVL, which were considered to be characteristic findings of RCA lesions (Table 3).

Differentiation of LCX and RCA Lesions Based on ST Segment Elevation in Leads II, III and aVF

The diagnostic accuracy of ST segment elevation in leads II, III and aVF, and ST segment depression in lead aVL, for identifying the affected vessels was evaluated in a total of 69 cases, including 24 patients with acute inferoposterior wall infarction and 45 other patients who underwent PTCA (25 patients with LCX lesions and 20 patients with RCA lesions). These ECG findings were observed in 35 of the 39 patients (90%) with RCA lesions and in 3 of the 30 patients (10%) with LCX lesions. Thus, the diagnostic accuracy was 90% in sensitivity, 90% in specificity and 92% in positive predictive value (p<0.001) (Table 2).

Lateral and Posterior Wall Leads

Fig 4a shows the ECG of a patient with lateral wall infarction from a LAD #9 (D1) lesion, recorded during the acute phase, and the departure map (Fig 4b) recorded on the eighth day after onset. The ECG shows slight ST segment elevation in leads I and aVL, and the departure map shows the infarct area restricted to the upper region of one intercostal space above V4 and decreased potentials around this region. The ECG findings do not differ markedly from...
those described for a LCX #11 lesion (Fig 2a). Thus, differentiation of the vessel responsible for lateral and posterior wall infarction was difficult.

The isopotential maps of ST segment elevation recorded during PTCA in 2 patients with LCX #13 lesions showed potential maxima toward the left lower thoracic region with similar patterns extending over the entire back. Fig 5a shows the expansion of the ST segment elevation area up to lead V6, but in Fig 5b this area remains restricted to the region below the lead. Taking these findings into account, there are different implications for the same LCX #13 lesion. The most useful lead, with marked ST segment elevation in both cases, was one intercostal space below lead V6.

Discussion

In the present study, we divided the IHD patients into 2 groups: anterior lesions and inferoposterior lesions. Our objective was a more detailed diagnosis of, and improved accuracy in identifying, the responsible coronary artery lesions by diagnosis according to the ECG findings. The ECG findings of MI sequentially show tall T wave, ST segment elevation, abnormal Q wave and coronary T wave, and their occurrence in certain leads suggests the site of the infarct.12 In general, the occurrence of an abnormal Q wave in a certain lead will correspond to the site of the MI and the leads are roughly divided into 2 groups: anterior wall leads (I, aVL, V1-6) and inferior wall leads (II, III, aVF).12 In addition, a high R wave in leads V1 and V2 for the posterior wall leads has also been taken as an indication of posterior wall infarction.13

However, the location of the affected vessel and the abnormal ECG leads do not necessarily coincide; because of the great anatomical differences in coronary arterial circulation among individuals. For example, the lateral wall of the left ventricle may be supplied by the LAD or the LCX, and in the inferior wall the anatomical dominance of the posterior descending branch may lead to inconsistent results.14 Accordingly, a Q wave in leads II, III and aVF also appears in pure posterior wall infarction and a high R wave in leads V1 and V2 occurs in inferior or lateral wall infarctions.

Anterior Wall Leads

In the comparison of the ECGs of the LAD #6 and #7 lesions responsible for anterior wall infarction, leads I and aVL showed ST segment elevation in a patient with a LAD #6 lesion, not in a patient with LAD #7 lesion. Thus, it is conceivable that LAD #6 lesions are located in the proximal LAD, including D1, from the standpoint that in characteristic ECG findings the ST segment elevation in leads I and aVL resulted from the D1 lesion. However, the diagnostic accuracy for patients with LAD #7 lesions showed false-positive results in 10 of 24 cases, resulting in the low rate of 50% in specificity and a positive predictive value of 62%. These findings were related to the anatomical characteristic of D1 bifurcating from the distal site of LAD #7. In 2 patients with LAD #6 lesions, ST segment elevation was not observed in leads I and aVL, and in both cases, coronary angiography revealed that spontaneous recanalization had already occurred during the acute phase. These results suggested that the anatomical location of the D1 bifurcation and spontaneous recanalization of infarct related vessels will effect the occurrence of ST segment elevation in leads I and aVL.

We also examined the possibility of differentiating LAD #6 and LAD #7 lesions based on the QS wave in lead V1. Because the proximal site of the first septal branch of the LAD in patients with #6 lesions is the lesion responsible for anterior wall infarction, the initial septal vector loss is probably transformed into the QS wave in lead V1. Howev-
er, 11 of 29 patients with LAD #6 lesions did not show a QS wave in lead V1. Coronary angiography was performed in 8 of those 11 patients during the acute phase and in 4 patients spontaneous recanalization had already occurred, which might contribute to the false-negative results. The reason for the other 4 false-negative results, and the 7 false-positives from 42 patients, is attributed to normal individual differences in the anatomical position of the heart. The diagnostic accuracy based on the ECG findings of either ST segment elevation in leads I and aVL or the occurrence of QS wave in lead V1 was not affected by the presence of collateral flow.

**Posterior Wall Leads**

When R/S>1 in lead V1 is defined as the ECG finding indicative of cases with posterior wall infarction, this finding was observed in 6 of 12 patients (50%) with LCX lesions, but among the patients with RCA lesions this was found in only 4 of 38 patients (11%). This variation is the reason why posterior wall infarction rarely occurs as a purely localized event, but in many cases, occurs together with lateral or inferior wall infarction.

Examination of the ECG recorded during the acute phase of a pure posterior wall infarction did not allow identification of ST segment elevation. Therefore, we examined the occurrence of ST segment elevation in cases complicated with an inferolateral wall infarction. ST segment elevation was observed in leads I, aVL, V5, and V6 in 3 of 5 patients with acute MI because of LCX lesions, in leads II, III, aVF, V5, and V6 in one of the remaining 2 and in leads I, II, III, aVR, V5, and V6 in the another. A small number of patients with this type of infarction were included with the patients with AP who underwent PTCA and in approximately 44% of the cases (11 of 25 patients), ST segment elevation was observed during PTCA for LCX lesions. LCX lesions had a low incidence, compared with the corresponding rate of 98% for LAD lesions and 80% for RCA lesions. In 8 of 11 patients who showed ST segment elevation during PTCA for LCX lesions, ST segment elevation was observed in more than 2 of the I, aVL, aVF, and V6 leads, and in the 3 other patients, the ST segment elevation occurred in more than 2 of II, III, aVF, V5 and V6. No characteristic differences between the leads were observed for LCX #11 and LCX #13 lesions. According to similar examinations during PTCA for LCX lesions, ST segment elevation was reportedly observed in 32% of the cases for leads V5, V6 or II, III, aVF and V4, but not in leads I and aVL. In addition, ST segment elevation during the same type of procedure was reportedly observed in 44% of the cases for leads II, III, aVF, V5, and V6. In neither of those studies was the site of the PTCA for LCX clearly stated, preventing a clear distinction. However, the anatomical position of the heart or difficulties in the ischemic regions manifesting on the ECG were the assumed reasons for the low incidence of ST segment elevation in the standard 12-lead recording. LCX shows many anatomical variations and is also involved in the dominance of the posterior descending branch, so diagnosis of the location of the LCX lesion based on ST segment elevation of specific leads remains extremely difficult and the ECG here reaches its limitations.

**Inferior Wall Leads**

In 11 of 19 patients with acute inferior wall infarction, ST segment elevation was also observed in the precordial leads in addition to leads II, III and aVF. ST segment elevation in leads V1-3 raises the suspicion of right ventricular involvement, whereas in leads V5 and V6 it suggests a posterolateral branch lesion of the RCA. However, differentiation of RCA #1 to #3 based on the ECG findings was impossible.

**Differential Diagnosis of LCX and RCA Lesions**

The sensitivity in diagnosing RCA lesions based on the findings of ST segment elevation in leads II, III and aVF, and ST segment depression in lead aVL, was extremely high at 90%. In the false-negative cases (4 of 40 patients), ST segment elevation was not observed during PTCA for RCA lesions. ST segment elevation in acute MI or during PTCA for LCX lesions was observed in leads II, III and aVF in 4 of 38 patients with acute MI because of LCX lesions, and in one of them there was complicated ST segment depression in lead aVL. The posterior descending branch had left dominant circulation. ST segment elevation was not observed in 2 of 5 patients with acute MI because of LCX lesions, and in one of them there was complicated ST segment depression in lead aVL and the posterior descending branch had left dominant circulation.

In the present study, ST segment elevation occurred in leads II, III and aVF in 3 of 11 patients with LCX lesions and an ST segment depression in lead aVL was observed in 2 of those 3 cases. One of the 2 patients had an obvious left dominant circulation of the posterior descending branch, and the other patient also had a balanced circulation of the posterior descending branch. Thus, the dominance of the posterior descending branch was considered a contributing factor in the ST segment elevation in leads II, III and aVF of 30 patients with LCX lesions, and ST segment depression in lead aVF because of LCX lesions and therefore the finding was not significant for the differential diagnosis of LCX and RCA lesions. In addition, it was reported that ST segment depression of 0.1 mV or more in lead aVL can assist in the diagnosis of LCX lesions, but the sensitivity of this finding in the present study was also insufficient because only 3 of 16 LCX lesions were detected. Senaratne et al suggested that the finding may point to a large area of supply of the culprit coronary artery.

**Study of New Lead Points to Improve Diagnostic Accuracy**

ST segment elevation in leads I and aVL was observed in both LAD #9 (D1) and LCX #11 lesions, which made it difficult to identify the vessel responsible for the infarction. In this context, we examined the utility of BSM also using leads on the back to compensate for the shortcomings of the ECG. On the departure map of a patient with an LAD #9 (D1) lesion, the infarcted area was restricted to the region of one intercostal space above lead V4 (Fig. 4). Recording from a lead or 2 intercostal spaces above the left lateral chest leads was reportedly useful for the diagnosis of high lateral wall infarction and the present result supports that suggestion.
Moreover, the area of ST segment elevation in 2 patients during PTCA for LCX#13 lesions had roughly the same pattern. However, expansion of the area toward lead V6 on the left lateral wall showed minor differences and the lead showing a common ST segment elevation was located one intercostal space below V6, which suggests that this specific lead may be useful for the diagnosis of LCX lesions.

Conclusions

The accurate diagnosis and presumptive identification of the affected coronary artery branch from the ECG findings in the standard 12-lead system can be affected by the occurrence of spontaneous recanalization and the presence of collateral circulation in addition to anatomical factors such as the branching of D1, the dominance of the posterior descending branch and normal subtypes. If there is ST segment depression in lead aVL in addition to ST segment descending branch and normal subtypes. If there is ST segment elevation in leads II, III and aVF, the RCA is probably the coronary artery responsible for the ischemia, and when the finding is because of a LCX lesion, the posterior descending branches are also involved. Therefore, it is necessary to consider broader ischemic areas.

To improve the accuracy in diagnosing the affected coronary artery branch, 2 additional leads, placed one intercostal space above lead V4 and one intercostal space below lead V6, are proposed in addition to the 6 precordial leads.

References