Acutely myocardial infarction (AMI) associated with left main coronary artery (LMCA) occlusion frequently results in poor prognosis because of the extensive area at risk with anterolateral wall involvement. Early diagnosis and subsequent treatment are essential to rescue these patients. In the current study, we retrospectively compared ECG findings in patients with LMCA occlusion, to those with proximal coronary artery occlusion.

METHODS
Of the 1880 patients undergoing coronary angiography within 24 hours of onset of AMI, 25 patients had LMCA occlusion. ECG findings on admission in these patients were compared to those in 30 consecutive patients with proximal coronary artery occlusion. Patients with previous AMI, left ventricular hypertrophy, or ≥ 75% stenosis in non-infarct arteries were excluded from this study. Patients with right bundle branch block (RBBB) were included, but those with left bundle branch block were excluded because of the difficulty of diagnosing AMI. ST elevation or depression was considered present if ST deviation was ≥ 0.1 mV. Left anterior fascicular block (LAFB) was defined by the mean QRS axis deviated to the left < 30° in the frontal plane. The culprit lesion was determined by coronary angiography and confirmed later by two cardiologists who were not aware of any ECG findings.

Data are expressed as mean (SD). Statistical analysis was performed with the χ² test for categorical variables. Analysis of variance and Student-Newman-Keuls test were used to compare continuous variables. Differences were considered significant if the probability value was p < 0.05.

RESULTS
Heart rate on admission was 89 (26) beats/min, and systolic blood pressure was less than 90 mm Hg in 18 patients (72%). There was no significant difference in baseline characteristics between the LMCA group, the left anterior descending artery (LAD) group, the left circumflex artery (LCx) group, and the right coronary artery (RCA) group (table 1). ST level in II, III, or aVF was significantly lower in the LMCA group than in the other groups (p < 0.01). ST level in V2 or V3 was significantly lower in the LCx group than in the LAD group (p < 0.01). ST depression in II, III, or aVF, ST elevation in aVR, ST elevation in both aVR and aVL, ST elevation in aVR with less that in V1, LAFB and RBBB did with higher specificity than that in only aVR in patients with AMI.

DISCUSSION
This study assessed ECG findings in a large number of patients with LMCA occlusion and demonstrated that: (1) ST depression in II, III or aVF and LAFB, predicted LMCA occlusion with high sensitivity; (2) ST elevation in both aVR and aVL, ST elevation in aVR with less than in V1, LAFB and RBBB did with high specificity; (3) ST elevation in both aVR and aVL did with higher specificity than that in only aVR in patients with AMI.

We observed ST depression in II, III, or aVF more frequently in the LMCA group than in the other groups and this result was consistent with previous reports. We also observed that the ST level in V2 or V3 was significantly lower in the LMCA group than in the LAD group. As patients with LMCA occlusion have both LAD and LCx occlusions at the same time, one possible mechanism is that reciprocal ST depression in precordial leads, because of LCx occlusion, may abolish ST elevation caused by LAD occlusion.

Yamaji and colleagues recently reported that ST elevation in aVR, less that in V1, was an important predictor of LMCA occlusion. In the current study, we observed this finding more frequently in the LMCA group than in the other groups. Regrettably, Yamaji and colleagues did not include patients with LCx occlusion. In the current study these patients were included and we demonstrated that ST elevation in aVR was relatively high in the LCx and LMCA groups (27% and 68%, respectively), compared to the LAD and RCA groups (7% and 3%, respectively). As described by Gorgels and colleagues, pronounced posterobasal wall ischaemia may cause ST elevation in aVR, especially in patients with proximal LCx occlusion. On the other hand, we demonstrated that ST elevation in aVL was uncommon in the LCx group. As shown in a previous study, it appears that aVL represents the area perfused by the first diagonal branch which LMCA occlusion always involves. This is why we advocate that ST elevation in both aVR and aVL predicts LMCA occlusion with higher specificity than that in only aVR in the current study.

The blood supply to the proximal segment of the right bundle is derived from the atroventricular node artery, whereas that for the remaining two thirds of the right bundle and for the left anterior fascicle is provided by the septal branches of the left anterior descending coronary artery.

Abbreviations: AMI, acute myocardial infarction; LAD, left anterior descending; LAFB, left anterior fascicular block; LCx, left circumflex; LMCA, left main coronary artery; RBBB, right bundle branch block; RCA, right coronary artery.
Sgarbossa and colleagues reported that RBBB, especially that associated with LAFB, was associated with LAD occlusion and high 30 day mortality. This is primarily considered to be because of extensive myocardial damage rather than the conduction disorder itself. In the current study, we first demonstrated that the incidences of LAFB and RBBB were strikingly higher in the LMCA group than in the LAD group.

This study included only patients with proximal coronary artery occlusion and did not include those with distal or mid-coronary artery occlusion. Moreover, selection bias could have influenced the enrolment of patients with LMCA occlusion because these patients died before undergoing coronary angiography more frequently than those with single vessel occlusion.

This study demonstrated that ST depression in II, III, or aVF and LAFB predicted LMCA occlusion with high sensitivity, and that ST elevation in both aVR and aVL, ST elevation in aVR with less that in V1, LAFB and RBBB predicted LMCA occlusion with high specificity in patients with AMI.

**Table 1** Baseline characteristics and ECG findings

<table>
<thead>
<tr>
<th></th>
<th>LMCA group (n = 30)</th>
<th>LAD group (n = 30)</th>
<th>Lx group (n = 30)</th>
<th>RCA group (n = 30)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62 (9)</td>
<td>62 (14)</td>
<td>64 (12)</td>
<td>63 (13)</td>
<td>NS</td>
</tr>
<tr>
<td>Male sex</td>
<td>24 (96%)</td>
<td>27 (90%)</td>
<td>22 (73%)</td>
<td>26 (87%)</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes</td>
<td>4 (16%)</td>
<td>6 (20%)</td>
<td>7 (23%)</td>
<td>9 (30%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3 (12%)</td>
<td>11 (37%)</td>
<td>9 (30%)</td>
<td>9 (30%)</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>10 (40%)</td>
<td>22 (73%)</td>
<td>15 (50%)</td>
<td>21 (70%)</td>
<td>NS</td>
</tr>
<tr>
<td>Time to ECG (hours)</td>
<td>3.5 (5.3)</td>
<td>4.8 (5.0)</td>
<td>7.3 (6.2)</td>
<td>3.9 (4.2)</td>
<td>NS</td>
</tr>
<tr>
<td>ST depression &gt;0.1 mV in II, III or aVF</td>
<td>22 (71%)</td>
<td>21 (71%)</td>
<td>8 (27%)</td>
<td>1 (3%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ST depression &gt;0.2 mV in II, III or aVF</td>
<td>19 (76%)</td>
<td>6 (20%)</td>
<td>27 (7%)</td>
<td>0 (0%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ST elevation &gt;0.1 mV in aVR</td>
<td>17 (68%)</td>
<td>4 (13%)</td>
<td>8 (27%)</td>
<td>1 (3%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ST elevation &gt;0.1 mV in both aVR and aVL</td>
<td>10 (40%)</td>
<td>1 (3%)</td>
<td>1 (3%)</td>
<td>0 (0%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ST elevation in aVR with less that in V1</td>
<td>10 (40%)</td>
<td>2 (7%)</td>
<td>26 (20%)</td>
<td>0 (0%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Left anterior fascicular block</td>
<td>20 (80%)</td>
<td>3 (17%)</td>
<td>2 (7%)</td>
<td>2 (7%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right bundle branch block</td>
<td>13 (52%)</td>
<td>6 (20%)</td>
<td>1 (3%)</td>
<td>2 (7%)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

LAD, left anterior descending artery; Lx, left circumflex artery; LMCA, left main coronary artery; NS, not significant; RCA, right coronary artery.

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