Journal of the American College of Cardiology
© 1999 by the American College of Cardiology
Published by Elsevier Science Inc.

Vol. 34, No. 2, 1999
ISSN 0735-1097/99/$20.00
PII S0735-1097(99)00197-7

Value of the Electrocardiogram in Localizing the Occlusion Site in the Left Anterior Descending Coronary Artery in Acute Anterior Myocardial Infarction

Domien J. Engelen, MD, Anton P. Gorgels, MD, Emile C. Cheriex, MD, Ebo D. De Muinck, MD, Anton J. Oude Ophuis, MD,* Willem R. Dassen, PHD, Jindra Vainer, MD, Vincent G. van Ommen, MD, Hein J. Wellens, MD, FACC

Maastricht and Nijmegen, The Netherlands

OBJECTIVES The study assessed the value of the electrocardiogram (ECG) as predictor of the left anterior descending coronary artery (LAD) occlusion site in relation to the first septal perforator (S1) and/or the first diagonal branch (D1) in patients with acute anterior myocardial infarction (AMI).

BACKGROUND In anterior AMI, determination of the exact site of LAD occlusion is important because the more proximal the occlusion the less favorable the prognosis.

METHODS One hundred patients with a first anterior AMI were included. The ECG showing the most pronounced ST-segment deviation before initiation of reperfusion therapy was evaluated and correlated with the exact LAD occlusion site as determined by coronary angiography.

RESULTS ST-elevation in lead aVR ($ST\uparrow_{aVR}$), complete right bundle branch block, ST-depression in lead V5 ($ST\downarrow_{V5}$) and $ST\uparrow_{V1} > 2.5$ mm strongly predicted LAD occlusion proximal to S1, whereas abnormal Q-waves in V4–6 were associated with occlusion distal to S1 ($p = 0.000$, $p = 0.004$, $p = 0.009$, $p = 0.011$ and $p = 0.031$ to 0.005, respectively). Abnormal Q-wave in lead aVL was associated with occlusion proximal to D1, whereas $ST\downarrow_{aVL}$ was suggestive of occlusion distal to D1 ($p = 0.002$ and $p = 0.022$, respectively). For both the S1 and D1, inferior $ST\downarrow \geq 1.0$ mm strongly predicted proximal LAD occlusion, whereas absence of inferior $ST\downarrow$ predicted distal occlusion ($p \leq 0.002$ and $p \leq 0.020$, respectively).

CONCLUSIONS In anterior AMI, the ECG is useful to predict the LAD occlusion site in relation to its major side branches. (J Am Coll Cardiol 1999;34:389–95) © 1999 by the American College of Cardiology

In acute anterior myocardial infarction (AMI), the site of occlusion in the left anterior descending (LAD) coronary artery is related to the extent of the myocardial necrosis and prognosis (1–5). Electrocardiographically, anterior myocardial infarction is classified as anteroseptal, anterolateral and apical (6). With a few exceptions, such as the study of Ideker et al. (7), most studies show poor correlation between the electrocardiogram (ECG) and the exact extent of myocardial involvement as determined by autopsy (8,9). In the present descriptive study, we assessed the value of the ECG to predict the occlusion site of the LAD in relation to the first septal perforator (S1) and/or the first diagonal branch (D1).

From the Department of Cardiology, University Hospital Maastricht, Cardiovascular Research Institute, Maastricht, The Netherlands; and *Department of Cardiology, Canisius Wilhelmina Hospital, Nijmegen, The Netherlands.

Manuscript received October 21, 1998; revised manuscript received March 4, 1999, accepted April 9, 1999.

METHODS

Patient group. One hundred consecutive patients admitted to the coronary care unit of the University Hospital Maastricht with the diagnosis of anterior AMI, defined as chest pain lasting more than 30 min accompanied by ST-segment elevation ($ST\uparrow$) ≥ 2.0 mm in V2 and V3 ($ST\uparrow_{v2-3}$), were studied. In 98 patients AMI was confirmed enzymatically. The remaining two patients were treated for AMI but did not show an enzyme rise because of early reperfusion (one after thrombolysis and the other after primary percutaneous transluminal coronary angioplasty). Patients with complete left bundle branch block, left ventricular hypertrophy (Sokolow index), ECG signs of an old MI or previous cardiac surgery were excluded.

JACC Vol. 34, No. 2, 1999
August 1999:389–95

Abbreviations and Acronyms

AMI = acute myocardial infarction
cRBBB = complete right bundle branch block
D1 = first diagonal branch
ECG = electrocardiogram
LAD = left anterior descending coronary artery
Q_x = abnormal Q-wave in lead x
ST ↑ = ST-segment elevation
ST ↓ = ST-segment depression
$ST\uparrow_x$ = ST-segment elevation in lead x
$ST\downarrow_x$ = ST-segment depression in lead x
S1 = first septal perforator

This study was approved by the institutional ethics committee, and informed consent was obtained in all patients.

Coronary angiography. All patients underwent coronary angiography: 89 patients during the acute phase (70 of 89 after thrombolytic therapy) and eleven patients 3 to 14 days after the acute episode (10 of 11 after thrombolytic therapy). In the latter 11 patients, immediate angiography was planned but logistically not possible. The severity of the stenosis was graded by using the CAAS II (Coronary Angiogram Analyzing System II, Pie Medical, Maastricht, The Netherlands [10]). The culprit lesion in the LAD was defined as the most severe and/or that lesion with local dissection or thrombus and was related to the take-off of S1 and D1. Flow over the culprit lesion was graded using Thrombolysis in Myocardial Infarction Trial (TIMI) criteria (11) and collateral circulation was classified according to Rentrop et al. (12). The presence of a wrap-around LAD (partially supplying the left ventricular inferior wall) was also assessed.

Electrocardiography. The MAC VU electrocardiograph (Marquette Medical Systems, Milwaukee, Wisconsin) with a frequency range of 0.01 to 150 Hz was used. In the acute phase, every 10 min a 12-lead ECG was recorded and also whenever the clinical condition changed. The one showing the most pronounced ST-segment deviation before start of reperfusion therapy was evaluated. The TP-segment was used as the iso-electric line; the PR-segment was used when the T-wave and the P-wave merged. The J-point was determined for each lead independently. Using electronic vernier calipers (Mitutoyo, Kawasaki-Shi, Japan), both ST ↑ and ST-segment depression (ST ↓) were measured at the J-point in all leads (1 mm = 0.1 mV). The accuracy of these calipers is ±0.02 mm, and in practice we measured with an accuracy of 0.1 mm. Although this system is semi-automatic, the accuracy of our measurements was clearly higher than usual in clinical practice.

Besides ST-segment deviation, the incidence of complete right bundle branch block (cRBBB) and abnormal Q-waves on the acute ECG was assessed (13).

Statistics. The data were analyzed using SPSS 7.0 for Windows (SPSS Inc., Chicago, Illinois). Data were expressed as median plus minimum and maximum for continuous variables and as rates (%) for categorical variables. For comparison of continuous variables, the Mann-Whitney *U* test was used. For comparison of categorical variables, the chi-square test or the Fisher exact test was used. A probability value <0.05 was considered statistically significant.

RESULTS

The S1 may have its take-off from the LAD proximal to the D1 and in other patients distal to the D1. The culprit lesion was proximal to both S1 and D1 in 31 patients, proximal to S1 and distal to D1 in 11 patients, distal to S1 but proximal to D1 in 10 patients and distal to both S1 and D1 in 48 patients. For correlation with the ECG, patients were divided into those with LAD occlusion proximal (31 + 11 = 42) or distal to S1 (10 + 48 = 58), respectively. The analysis was thereafter repeated in the same group of patients, but this time the patients were allocated to subgroups based on the site of occlusion in relation to D1: patients with LAD occlusion proximal (31 + 10 = 41) or distal to D1 (48 + 11 = 59), respectively.

Demography and clinical data. The baseline characteristics of the patients are listed in Table 1. Subgroups were comparable with regard to gender, mean age and the time delay between onset of chest pain and recording of the ECG. When the culprit lesion was proximal to either S1 or D1, the peak serum creatine kinase was significantly higher.

Coronary angiography. Between proximal and distal lesions, there were no differences regarding the presence of single-, two- and three-vessel disease or a wrap-around LAD (Table 1). One patient had left main stenosis. No significant differences regarding TIMI or Rentrop flow grade were found. Angiographic signs of thrombus were present in 47 patients.

Electrocardiography. ECG PREDICTORS OF LAD OCCLUSION PROXIMAL TO S1. $ST\uparrow_{aVR}$ was present in 43% of the occlusions proximal to S1 and in only 5% of the occlusions distal to S1 ($p = 0.000$). The amount of ST ↑ was small, the median being 0.4 (0.2 to 1.8) mm (Table 2, Figs. 1 and 2).

ST-segment depression in leads II, III and aVF was present in 74%, 86% and 91% of the occlusions proximal to S1, and in 33%, 66% and 55% of the occlusions distal to S1, respectively. In proximal occlusions, the amount of ST ↓ was significantly higher: 0.9 (0.2 to 4.5) versus 0.4 (0.2 to 0.9) mm, 1.9 (0.2 to 7.7) versus 0.9 (0.2 to 4.0) mm and 1.3 (0.2 to 6.2) versus 0.6 (0.2 to 2.5) mm, for leads II, III and aVF, respectively ($p = 0.000$, $p = 0.002$ and $p = 0.008$). Inferior ST ↓ ≥ 1.0 mm was highly predictive of LAD occlusion proximal to S1 ($p \leq 0.002$).

Table 1. Demographic, Clinical and Angiographic Data

	Proximal to S1	Distal to S1	p-Value	Proximal to D1	Distal to D1	p-Value
No. of pts	42	58		41	59	
Gender (M:F)	29:13	45:13	NS	30:11	44:15	NS
Age (yrs)	55 (22–77)	59 (35–80)	NS	54 (22–77)	59 (36–80)	NS
Time to ECG (h)	2.0 (0.1–9.5)	2.3 (0.2–19.4)	NS	2.1 (0.2–19.4)	2.3 (0.1–14.0)	NS
Peak CK (U/liter)	3948 (690–12,132)	2238 (78–10,364)	0.014	3333 (680–12,132)	2239 (78–10,364)	0.045
Percentage stenosis	99 (50–100)	100 (45–100)	NS	100 (50–100)	99 (45–100)	NS
Left main	2	0	NS	2	0	NS
RCA	10	9	NS	8	10	NS
RCX	14	22	NS	17	21	NS
RCA & RCX	12	14	NS	10	15	NS
Wrap-around LAD	52	42	NS	43	49	NS

Data are presented as median plus minimum and maximum for continuous variables and as rates (%) for categorical variables.
CK = creatine kinase; D1 = first diagonal branch; ECG = electrocardiogram; LAD = left anterior descending coronary artery; No. of pts = number of patients; RCA = right coronary artery; RCX = circumflex coronary artery; S1 = first septal perforator.

cRBBB was registered in six patients. In all of them, the culprit lesion was situated proximal to S1.

A similar number of patients with either an occlusion proximal or distal to S1 had $ST\uparrow_{V1}$ (88% vs. 86%), although the amount of ST ↑ was significantly higher in patients with the LAD occluded proximal to S1: 1.2 (0.2 to 5.1) versus 0.6 (0.2 to 2.5 mm; p = 0.004). $ST\uparrow_{V1} >$ 2.5 mm was only present in LAD occlusion proximal to S1, but the sensitivity of this criterion was very low. In occlusions proximal to S1, the amount of $ST\uparrow_{V2}$ was significantly higher: 4.9 (0.9 to 12.2) versus 3.2 (0.3 to 13.5 mm; p = 0.001). However, no cut-off points were found to discriminate between proximal and distal LAD lesion. No relation was found between the sum of ST ↑ in the precordial leads ($\Sigma ST\uparrow_{V1–V4}$ or $\Sigma ST\uparrow_{V1–V6}$) and the occlusion site.

$ST\downarrow_{V5}$ was very specific for LAD occlusion proximal to S1: it was present in 17% of the proximal lesions and only in 2% of the distal lesions (p = 0.009). The median ST ↓ in the former group was 0.5 (0.2 to 2.9) mm.

ECG PREDICTORS OF LAD OCCLUSION PROXIMAL TO D1. Considering the location of the culprit lesion in relation to D1, ST ↓ in leads II, III and aVF was present in 73%, 95% and 90% of the proximal and in 34%, 59% and 56% of the distal lesions, respectively. In proximal LAD disease, inferior ST ↓ was significantly more: 0.9 (0.2 to 4.5) versus 0.5 (0.2 to 1.6) mm, 1.7 (0.2 to 7.7) versus 0.9 (0.2 to 4.6) mm,

Table 2. Electrocardiographic Predictors of Left Anterior Descending Coronary Artery (LAD) Occlusion Proximal to the First Septal Perforator (S1) and/or the First Diagonal Branch (D1)

	Predictors of LAD Occlusion Proximal to S1					
	Sens	Spec	PPV	NPV	LR	p-Value
$ST\uparrow_{aVR}$	43	95	86	70	8.6	0.000
$ST\downarrow_{II} \geq 1.0$ mm	36	100	100	68		0.000
$ST\downarrow_{III} \geq 1.0$ mm	60	71	60	71	2.1	0.002
$ST\downarrow_{III} \geq 2.5$ mm	33	97	88	67	11.0	0.000
$ST\downarrow_{aVF} \geq 1.0$ mm	52	84	71	71	3.3	0.000
$ST\downarrow_{aVF} \geq 2.0$ mm	26	97	85	64	8.7	0.002
cRBBB	14	100	100	62		0.004
$ST\downarrow_{V5}$	17	98	88	62	8.5	0.009
$ST\uparrow_{V1} > 2.5$ mm	12	100	100	61		0.011
	Predictors of LAD Occlusion Proximal to D1					
$ST\downarrow_{II} \geq 1.0$ mm	34	98	93	68	17.0	0.000
$ST\downarrow_{III} \geq 1.0$ mm	66	75	64	76	2.6	0.000
$ST\downarrow_{III} \geq 2.5$ mm	32	95	81	67	6.4	0.001
$ST\downarrow_{aVF} \geq 1.0$ mm	54	85	71	72	3.6	0.000
$ST\downarrow_{aVF} \geq 2.0$ mm	27	97	85	66	9.0	0.001
Q_{aVL}	44	85	67	69	2.9	0.002

cRBBB = complete right bundle branch block; LR = likelihood-ratio; NPV = negative predictive value; PPV = positive predictive value; sens = sensitivity; spec = specificity; ST ↓ = ST-depression; ST ↑ = ST-elevation; Q = abnormal Q-wave.

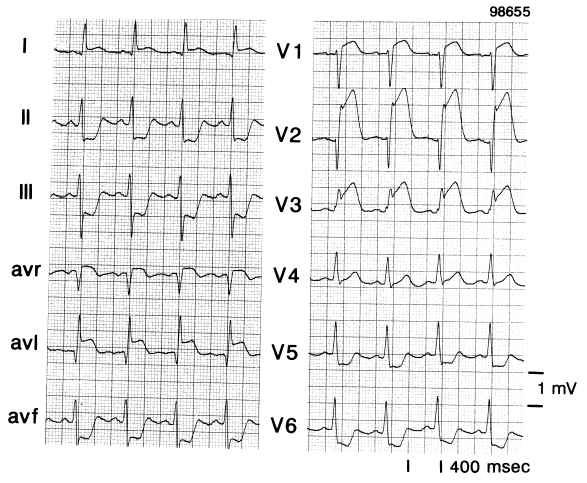

Figure 1. ECG of a patient with an anterior AMI as a consequence of LAD occlusion proximal to both the first septal perforator and the first diagonal branch, showing characteristic ST ↑ in aVR, ST ↑ >0.25 mV in V1, ST ↓ in V5, inferior ST ↓ >0.1 mV and an abnormal Q-wave in aVL.

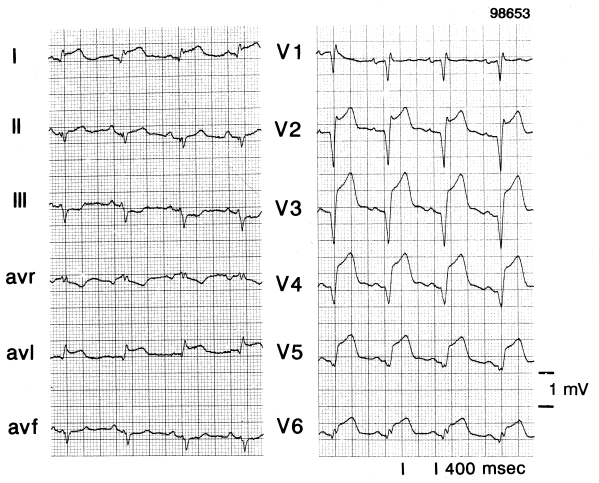

Figure 3. ECG of a patient with an anterior AMI as a consequence of LAD occlusion distal to the first septal perforator and proximal to the first diagonal branch showing characteristic abnormal Q-waves in V4, V5, V6 and aVL, ST ↓ >0.1 mV in III and absence of ST ↓ in II (and aVF).

1.3 (0.2 to 6.2) versus 0.4 (0.2 to 2.2) mm for leads II, III and aVF, respectively (p = 0.001, p = 0.005 and p = 0.001). Inferior ST ↓ ≥ 1.0 mm was also highly predictive of LAD occlusion proximal to D1 (p = 0.000). (See Table 2, Figs. 1 and 3.)

An abnormal Q-wave in lead AVL (Q_{aVL}; width ≥30 ms) was more frequent in LAD occlusion proximal to D1 in comparison with distal occlusion (44% vs. 15%; p = 0.002).

The ST ↑ in leads I and aVL was present in 83% of the LAD lesions proximal to D1 but also in 61% of the distal lesions (p = NS). The amount of ST $\uparrow_{aVL}$ was significantly higher in proximal occlusion: 1.2 (0.3 to 5.2) versus 0.5 (0.2 to 4.6 mm; p = 0.006). However, no cut-off point was found to discriminate between proximal and distal disease.

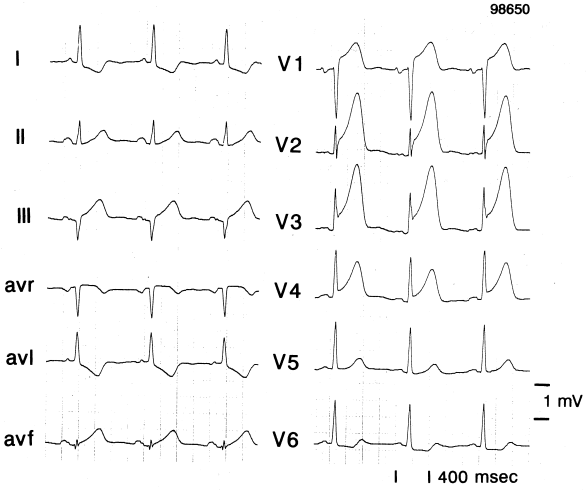

Figure 2. ECG of a patient with an anterior AMI as a consequence of LAD occlusion proximal to the first septal perforator and distal to the first diagonal branch, showing characteristic ST ↑ in aVR, ST ↑ >0.25 mV in V1, probably ST ↓ in V5, ST ↓ in aVL and absence of ST ↓ in III and aVF.

ECG PREDICTORS OF LAD OCCLUSION DISTAL TO S1. Absence of inferior ST ↓, particularly in leads II and aVF, was strongly related to LAD disease distal to S1. Except for one, all patients without inferior ST ↓ had a wrap-around LAD. (Table 3, Figs. 3 and 4.)

Q-waves in V4 (width ≥ 20 ms), V5 (width ≥ 30 ms) and V6 (width ≥ 30 ms) were rather specific for LAD occlusion distal to S1 and were found in 55%, 24% and 17% of the occlusions distal to S1 and in only 31%, 7% and 0% of the proximal occlusions (p = 0.015, p = 0.031 and p = 0.005).

ECG PREDICTORS OF LAD OCCLUSION DISTAL TO D1. Absence of inferior ST ↓, particularly in lead III, was also strongly related to LAD disease distal to D1. (See Table 3, Figs. 2 and 4). Also, ST $\downarrow_{aVL}$ was rather specific for occlusion distal to D1. The amount of ST ↓ was 0.3 (0.2 to 1.1) mm.

DISCUSSION

This article describes several new findings regarding the electrocardiographic prediction of the LAD occlusion site in anterior AMI (Table 4). Lead aVR was found to be very useful to identify LAD occlusion proximal to S1. Besides ST $\uparrow_{aVR}$, both cRBBB, ST $\downarrow_{V5}$ and ST $\uparrow_{V1}$ > 2.5 mm were strongly predictive of LAD occlusion proximal to S1, whereas abnormal Q-waves in V4 through V6 were indicative of occlusion distal to S1. Considering the LAD occlusion site in relation to D1, an abnormal Q-wave in lead aVL was suggestive of proximal occlusion, while ST ↓ in the same lead was associated with distal occlusion. For both

Table 3. Electrocardiographic Predictors of LAD Occlusion Distal to S1 and/or D1

	Predictors of LAD Occlusion Distal to S1					
	Sens	**Spec**	**PPV**	**NPV**	**LR**	**p-Value**
Absence of ST $\downarrow_{II}$	67	74	78	62	2.6	0.000
Absence of ST $\downarrow_{III}$	34	86	77	49	2.4	0.020
Absence of ST $\downarrow_{aVF}$	45	90	87	54	4.5	0.000
Q_{V6}	17	100	100	47		0.005
Q_{V5}	24	93	82	47	3.4	0.031
Q_{V4}	55	69	71	53	1.8	0.015
	Predictors of LAD Occlusion Distal to D1					
Absence of ST $\downarrow_{II}$	66	73	78	60	2.4	0.000
Absence of ST $\downarrow_{III}$	41	95	92	53	8.2	0.000
Absence of ST $\downarrow_{aVF}$	44	90	87	53	4.4	0.000
ST $\downarrow_{aVL}$	22	95	87	46	4.4	0.022

Abbreviations as in Table 2.

S1 and D1, marked ST $\downarrow$ in the inferior leads predicted proximal occlusion, whereas absence of inferior ST $\downarrow$ predicted distal occlusion.

ST-elevation in lead aVR. ST $\uparrow_{aVR}$ in unstable angina in three-vessel or left main stem disease has previously been reported (14). However, we did not find literature on ST $\uparrow_{aVR}$ as an indicator to localize ischemia of the left ventricular anterior wall. In the only two articles on ST $\uparrow_{aVR}$ in the setting of ischemia in the perfusion area of the LAD, ST $\uparrow_{aVR}$ was not used to define the exact LAD occlusion site in relation to the major side branches (15,16). Kataoka et al. (17) found that in anterior AMI, ST $\uparrow$ in the right precordial leads was associated with LAD occlusion proximal to S1, but the ST-segment in lead aVR was not mentioned. When present, the amount of ST $\uparrow_{aVR}$ is usually small (<1 mm), particularly when measured at the J-point and not 40 to 80 ms thereafter. Therefore, in this selected population with anterior AMI, any ST $\uparrow_{aVR}$ is associated with LAD occlusion proximal to S1 and is probably the result of transmural ischemia of the basal part of the septum (injury current directed toward the right shoulder). This theory is supported by our finding that none of the 10 patients with LAD occlusion proximal to D1 but distal to S1 showed ST $\uparrow_{aVR}$. ST $\uparrow_{aVR}$ was absent in 57% of the occlusions proximal to S1. The reason for the rather low sensitivity of this ECG criterion may be due to the rare dominance of the basal septum, being needed for this criterion to become positive, because of the counter balance of the septal ischemia current by ischemia in other large areas of the left ventricle perfused by the LAD such as the lateral and apical inferior wall.

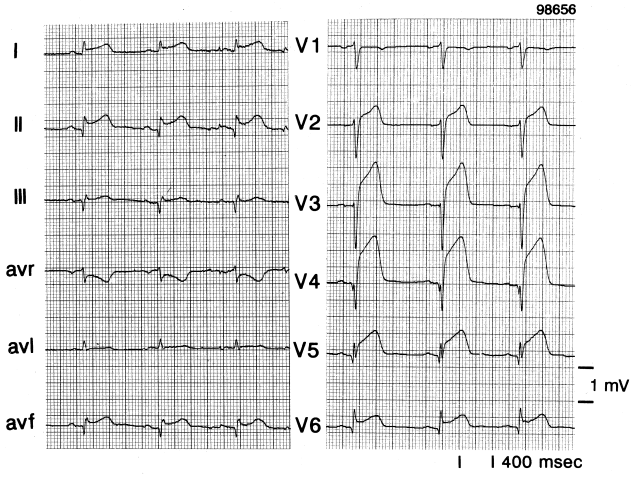

Figure 4. ECG of a patient with an anterior AMI as a consequence of LAD occlusion distal to both the first septal perforator as well as the first diagonal branch showing characteristic abnormal Q-waves in V4, V5 and V6 and absence of inferior ST $\downarrow$ in all inferior leads.

ST-deviation in leads II, III and/or aVF. The amount of inferior ST $\downarrow$ was significantly higher in proximal LAD disease, and particularly ST $\downarrow \geq 1.0$ mm was highly predictive of a proximal LAD lesion. This finding is consistent with previous studies on inferior ST $\downarrow$ in anterior AMI (18,19). Inferior ST $\downarrow$ was not associated with disease of the right coronary artery. This supports the theory that inferior ST $\downarrow$ in anterior AMI represents reciprocal changes associated with transmural ischemia in the high anterobasal region (20,21). Despite the presence of a wrap-around LAD

Table 4. Electrocardiographic Predictors of LAD Occlusion Site

ST $\uparrow_{V1}$ >2.5 mm	proximal to S1
cRBBB	proximal to S1
ST $\uparrow_{aVR}$	proximal to S1
ST $\downarrow_{V5}$	proximal to S1
Q_{aVL}	proximal to D1
Inferior ST $\downarrow \geq 1.0$ mm	proximal to S1/D1
$Q_{V4–6}$	distal to S1
ST $\downarrow_{aVL}$	distal to D1
Absence of inferior ST $\downarrow$	distal to S1/D1

Abbreviations as in Table 2.

JACC Vol. 34, No. 2, 1999
August 1999:389–95

and thereby transmural ischemia in a part of the inferior left ventricular wall, almost all occlusions proximal to D1 still showed inferior ST ↓ . The most likely explanation for this finding is the magnitude of the ischemia current produced by the transmural ischemia of the anterobasal wall exceeding the current of the inferior wall. Consequently, besides a wrap-around LAD, a more distally occluded LAD is thought to be a prerequisite of an iso-electric or even elevated ST-segment inferiorly. Indeed, in 90% of the patients without inferior ST ↓ (defined as absence of ST ↓ in ≥2 inferior leads) the culprit lesion was situated distally to D1, and in all but one (97%) of the patients without inferior ST ↓ the coronary angiogram revealed a wrap-around LAD. Similar findings were recently published by other investigators (22–24). In addition, a recent study by Porter et al. (25) showed that the presence of lead III ST ↓ with a positive T-wave associated with ST ↑ $_{aVL}$ could serve as an early ECG marker of prediagonal occlusion of a wrap-around LAD.

ST-deviation in leads I and/or aVL. In the individual patient, ST ↑ in the lateral leads did not discriminate between occlusions proximal and distal to D1: ST ↑ $_{aVL}$ was not only present in 83% of the LAD occlusions proximal to D1, but also in 66% and 47% of the occlusions distal to D1 and D2, respectively. In contrast, ST ↓ $_{aVL}$ was rather specific for LAD occlusion distal to D1 and probably represents reciprocal changes associated with transmural ischemia in the inferoapical region: 6% of the patients with and 43% of the patients without inferior ST ↓ showed ST ↓ $_{aVL}$, respectively ($p = 0.000$). The ST-segment shift in aVL could not be explained by the presence or absence of an anterolateral branch.

ST-deviation in leads V1–V6. In 57% and 23% of the patients showing an ECG pattern traditionally termed "anteroseptal infarction" (ST ↑ $_{V1–V3}$), the LAD occlusion was located distal to the first and second septal perforator, respectively. This is in agreement with the study by Shalev et al. (26). Analyzing the ST-deviations in leads V1 thru V4, ST ↑ $_{V1}$ >2.5 mm was the only ECG parameter that could be defined to discriminate between proximal and distal LAD occlusion in the individual patient. Furthermore, ST ↓ $_{V5}$ appeared to be very specific for LAD occlusion proximal to S1. The occurrence of ST ↓ $_{V5}$ was interpreted as reciprocal changes associated with transmural ischemia high in the anteroseptal area. Although ST ↓ $_{V6}$ was seen more often in proximal LAD disease, this difference was not significant.

Complete right bundle branch block. The development of cRBBB in anterior AMI is an independent marker of poor prognosis (27). This is primarily considered to be due to extensive myocardial damage rather than the conduction disorder itself. Only if cRBBB comes together with left anterior hemiblock is there an increased risk for progression into complete AV-block (28). In our study, cRBBB was registered exclusively in LAD occlusions proximal to S1. As S1 is the main blood supply to the distal part of the bundle of His and the proximal bundle branches, the cRBBB is most likely acquired in the setting of anterior AMI.

Abnormal Q waves. In leads V4, V5 and V6, Q waves were rather specific for LAD disease distal to S1. In distal LAD disease, normal activation of the ventricles starting in the interventricular septum and spreading transversely from left to right through the septum usually remains intact. In case of myocardial necrosis beneath the anterolateral leads, the septal vector in (V5)V6 will facilitate Q-wave formation in those electrodes. In contrast, in proximal to S1 occlusion the septal vector will decrease or even disappear and thereby hinder Q-wave formation. The Q-waves occurred already early after onset of AMI and may indicate local conduction delay rather than necrosis.

Clinical significance. The ECG signs described in this study may prove useful in defining the site of LAD occlusion in relation to S1 and D1 in the setting of anterior AMI. In particular, ST ↑ $_{aVR}$ and ST-deviation in the inferior leads were found to be very helpful. We consider our findings useful in identifying patients with proximal LAD occlusion who need a more aggressive approach to revascularization to prevent extensive myocardial damage resulting in pump failure, the possible development of sub-AV-nodal conduction disturbances and the occurrence of life-threatening ventricular arrhythmias in the second and third week after AMI (29).

Study limitations. The number of patients studied is limited. Therefore, we are now evaluating our findings prospectively in a larger study population. Several ECGs were recorded in the acute stage, and the one showing the most pronounced abnormalities was selected for our study. Therefore, our findings may not be reproducible in a setting where fewer ECGs are recorded. Most of the study population had onset of chest pain within 4 h before the recording of the ECG, and coronary angiography allowing identification of the culprit lesion was performed in the majority of our cases shortly after admission. Thus, we cannot be certain whether our findings are applicable when patients come in later.

Reprint requests and correspondence: Domien J.M. Engelen, Department of Cardiology, University Hospital Maastricht, Box 5800, 6202 AZ, Maastricht, The Netherlands. E-mail: D.Engelen@cardio.azm.nl.

REFERENCES

1. Gaudron P, Eilles C, Kugler I, Ertl G. Progressive left ventricular dysfunction and remodeling after myocardial infarction: potential mechanisms and early predictors. Circulation 1993;87:755–63.
2. Jeremy RW, Allman KC, Bautovitch G, Harris PJ. Patterns of left ventricular dilation during the six months after myocardial infarction. J Am Coll Cardiol 1989;13:304–10.
3. Pirolo JS, Hutchins GM, Moore GW. Infarct expansion: pathologic

analysis of 204 patients with a single myocardial infarct. J Am Coll Cardiol 1986;7:349–54.
4. Voth E, Tebbe U, Schicha H, et al, and the ISAM Study Group. Intravenous streptokinase in acute myocardial infarction (I.S.A.M.) Trial: serial evaluation of left ventricular function up to 3 years after infarction estimated by radionuclide ventriculography. J Am Coll Cardiol 1991;18:1610–6.
5. White HD, Norris RM, Brown MA, et al. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987;76:44–51.
6. Roberts WC, Gardin JM. Location of myocardial infarcts: a confusion of terms and definitions. Am J Cardiol 1978;42:868–72.
7. Ideker RE, Wagner GS, Ruth WK, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size: II. correlation with quantitative anatomic findings for anterior infarcts. Am J Cardiol 1982;49:1604–14.
8. Sullivan W, Vlodaver Z, Tuna N, et al. Correlation of electrocardiographic and pathologic findings in healed myocardial infarction. Am J Cardiol 1978;42:724–32.
9. Savage RM, Wagner GS, Ideker RE, et al. Correlation of postmortem anatomic findings with electrocardiographic changes in patients with myocardial infarction. Circulation 1977;55:279–85.
10. Haase J, Escaned J, Van Swijndregt EM, et al. Experimental validation of geometric and densitometric coronary measurements on the new generation Cardiovascular Angiography Analysis System (CAAS II). Catheter Cardiovasc Diagn 1993;30:104–14.
11. Mueller HS, Dyer A, Greenberg MA, et al. The thrombolysis in myocardial infarction (TIMI) trial: phase I findings. N Engl J Med 1985;312:932–6.
12. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. J Am Coll Cardiol 1985;5:587–92.
13. Selvester RH, Wagner GS, Hindman NB. The Selvester QRS scoring system for estimating myocardial infarct size: the development and application of the system. Arch Intern Med 1985;145:1877–81.
14. Gorgels APM, Vos MA, Mulleneers R, et al. Value of the electrocardiogram in diagnosing the number of severely narrowed coronary arteries in rest angina pectoris. Am J Cardiol 1993;72:999–1003.
15. Nakamori H, Iwasaka T, Shimada T, et al. Clinical significance of ST segment elevation in lead aVR in anterior myocardial infarction: assessment by thallium-201 exercise scintigraphy. Cardiology 1995;86:147–51.
16. Karakawa M, Iwasaka T, Kamihata H, et al. Transient ST elevation in lead aVR during left anterior descending artery angioplasty as a marker of the adequacy of the coronary circulation. J Cardiovasc Technol 1992;10:233–9.
17. Kataoka H, Tamura A, Yano S, et al. ST elevation in the right chest leads in anterior wall left ventricular acute myocardial infarction. Am J Cardiol 1990;66:1146–7.
18. Birnbaum Y, Solodky A, Herz I, et al. Implications of inferior ST-segment depression in acute anterior myocardial infarction: electrocardiographic and angiographic correlation. Am Heart J 1994;127:1467–73.
19. Tamura A, Kataoka H, Mikuriya Y, Nasu M. Inferior ST-segment depression as a useful marker for identifying proximal left anterior descending coronary artery occlusion during acute anterior myocardial infarction. Eur Heart J 1995;16:1795–9.
20. Fletcher WO, Gibbons RJ, Clements IP. The relationship of inferior ST depression, lateral ST elevation, and left precordial ST elevation to myocardium at risk in acute anterior myocardial infarction. Am Heart J 1993;126:526–35.
21. Quyyumi AA, Crake T, Rubens MB, et al. Importance of reciprocal electrocardiographic changes during occlusion of the left anterior descending coronary artery: studies during percutaneous transluminal coronary angioplasty. Lancet 1986;15:347–50.
22. Sapin PM, Musselman DR, Dehmer GJ, Cascio WE. Implications of inferior ST-segment elevation accompanying anterior wall acute myocardial infarction for the angiographic morphology of the left anterior descending coronary artery morphology and site of occlusion. Am J Cardiol 1992;69:860–5.
23. Tamura A, Kataoka H, Nagase K, et al. Clinical significance of inferior ST elevation during acute anterior myocardial infarction. Br Heart J 1995;74:611–4.
24. Lew AS, Hod H, Cercek B, et al. Inferior ST segment changes during acute anterior myocardial infarction: a marker of the presence or absence of concomitant inferior wall ischemia. J Am Coll Cardiol 1987;10:519–26.
25. Porter A, Sclarovsky S, Ben-Gal T, et al. Value of T-wave direction with lead III ST-segment depression in acute anterior myocardial infarction: electrocardiographic prediction of a wrapped left anterior descending artery. Clin Cardiol 1998;21:562–6.
26. Shalev Y, Fogelman R, Oettinger M, Caspi A. Does the electrocardiographic pattern of "anteroseptal" myocardial infarction correlate with the anatomical location of myocardial injury? Am J Cardiol 1995;75:763–6.
27. Ricou F, Nicod P, Gilpin E, et al. Influence of right bundle branch block on short- and long-term survival after acute anterior myocardial infarction. J Am Coll Cardiol 1991;17:858–63.
28. Lie KI, Wellens HJJ, Schuilenburg RM, et al. Factors influencing prognosis of bundle branch block complicating acute antero-septal infarction: the value of His bundle recordings. Circulation 1974;50:935–41.
29. Braat SH, De Zwaan C, Brugada P, Wellens HJJ. Value of left ventricular ejection fraction in extensive anterior myocardial infarction to predict development of ventricular tachycardia. Am J Cardiol 1983;52:686–9.