

# Value of Electrocardiographic Algorithm Based on “Ups and Downs” of ST in Assessment of a Culprit Artery in Evolving Inferior Wall Acute Myocardial Infarction

Miquel Fiol, MD, PhD, Iwona Cygankiewicz, MD, PhD, Andrés Carrillo, MD, Antoni Bayés-Genis, MD, PhD, Omar Santoyo, MD, Alfredo Gómez, MD, PhD, Armando Bethencourt, MD, PhD, and Antoni Bayés de Luna, MD, PhD

Acute myocardial infarction (AMI) of the inferoposterior wall is due to occlusion of the right coronary artery (RCA) or the left circumflex (LCx) coronary artery. The outcome of patients depends mainly on the culprit artery. Therefore, the presumptive prediction of a culprit artery based on the electrocardiogram recorded at admission is of clinical importance. The aim of this study was to develop a sequential algorithm based on the “ups and downs” of the ST segment in different leads to predict the culprit artery (RCA vs LCx) in cases of inferoposterior AMI. We analyzed electrocardiographic and angiographic findings of 63 consecutive patients with an evolving AMI with ST elevation in the inferior leads (II, III, and aVF) and a single-vessel occlusion. Specificity, sensitivity, and positive and negative predictive values of different electrocardiographic criteria (ups and downs of the ST segment) were studied individually and in com-

bination to find an algorithm that would best predict the culprit artery. The following electrocardiographic criteria were included in the 3-step algorithm: (1) ST changes in lead I, (2) the ratio of ST elevation in lead III to that in lead II, and (3) the ratio of the sum of ST depression in precordial leads to the sum of ST elevation in inferior leads [ $(\sum \downarrow \text{ST in leads V}_1 \text{ to V}_3)/(\sum \uparrow \text{ST in leads II, III, and aVF})$ ]. Application of this sensitive algorithm suggested the location of the culprit coronary artery (RCA vs LCx) in 60 of 63 patients (>95%). The few patients in whom this algorithm did not work were those with a very dominant LCx that presented ST depression of  $\geq 0.5$  mm in lead I. In conclusion, careful sequential analysis of an electrocardiogram of an inferoposterior AMI with ST elevation may lead to the identification of a culprit artery. ©2004 by Excerpta Medica, Inc.

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**A**cute myocardial infarction (AMI) of the inferoposterior wall is reflected on an electrocardiogram as ST elevation in the inferior leads accompanied by ST changes in other leads that may be a consequence of concomitant ischemia of other zones or a reciprocal image.<sup>1</sup> Different electrocardiographic criteria based on the analysis of ST elevation and ST depression (“ups and downs”) in different leads, including V<sub>3</sub>R to V<sub>5</sub>R, have been suggested to predict the culprit artery.<sup>2–10</sup> These criteria are based on the fact that in cases of AMI of the inferoposterior wall due to occlusion of the right coronary artery (RCA), the vector of injury is directed more downward than backward and more to the right than to the left (Figure 1); in contrast, in cases of occlusion of the left circumflex (LCx) coronary artery, the vector of injury points downward, predominantly posteriorly, and more to the left than to the right (Figure 1). In this work, we present a sequential electrocardiographic

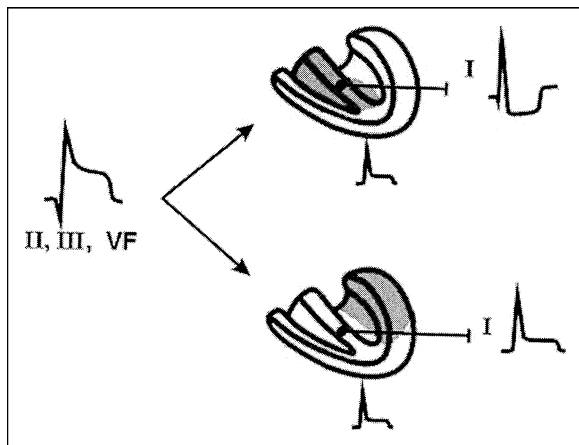
algorithm based on ST changes in different leads that has a very high sensitivity, specificity, and predictive value to predict the culprit artery (RCA vs LCx) in cases of an evolving inferoposterior AMI with ST elevation.

## METHODS

**Study population:** We retrospectively analyzed electrocardiographic and angiographic findings of consecutive patients admitted to the emergency unit with an evolving AMI with ST elevation in the inferior leads. The study population consisted of patients who met the following inclusion criteria: no history of a myocardial infarction, <6 hours from the onset of symptoms, sinus rhythm on electrocardiogram, ST elevation >1 mm in  $\geq 2$  of 3 leads (II, III, and aVF), no left or right bundle branch block, coronary angiography performed within 12 hours of symptom onset, and critical (>70%) single-vessel stenosis (RCA or LCx). Diagnosis of AMI was based on clinical symptoms (anginal pain >20 minutes), electrocardiographic findings, and enzymatic changes. In all patients, data were acquired concerning demographics, clinical characteristics, time from symptom onset to the first recorded electrocardiogram, and outcome (Table 1).

From the Hospital Universitario Son Dureta, Palma de Mallorca; and the Hospital de la Santa Creu i Sant Pau, Barcelona, Spain. Manuscript received February 13, 2004; revised manuscript received and accepted May 27, 2004.

Address for reprints: Antoni Bayés de Luna, MD, PhD, Hospital de la Santa Creu i St Pau, St. Antoni Ma. Claret 167, E-08025 Barcelona, Spain. E-mail: abayesluna@hsp.santpau.es.



**FIGURE 1.** Vectors of injury in an evolving inferoposterior AMI due to occlusions of the RCA (*top*) and LCx (*bottom*).

**TABLE 1** Clinical Characteristics of Patients

	All Patients (n = 63)	RCA Occlusion (n = 50)	LCX Occlusion (n = 13)
Age (yrs)	58 ± 11	59 ± 11	58 ± 9
Men/women	50/13	41/9	9/4
Time from symptom onset to first ECG (min)	150 ± 73	130 ± 69	180 ± 69
Antecedents			
Active smoker	34 (53.9%)	27 (54%)	7 (53.8%)
Diabetic	8 (12.6%)	6 (12%)	2 (15.3%)
Arterial hypertension	26 (41.2%)	21 (42%)	5 (38.4%)
Dyslipidemia	23 (36.5%)	17 (34%)	6 (46%)
Previous angina	5 (9.5%)	5 (10%)	1 (7.6%)
Familial ischemic heart disease	8 (12.6%)	8 (16%)	0 (0%)
Cocaine	1 (1.5%)	0 (0%)	0 (0%)
Stroke	0 (0%)	3 (6%)	0 (0%)
Left ventricular ejection fraction at time of admission (%)	60 ± 11	59 ± 12	60 ± 9
Enzyme peaks			
CPK (IU/L)	2,317 ± 1,854	2,386 ± 1,722	2,017 ± 2,105
CK-MB (IU/L)	285 ± 240	299 ± 229	285 ± 251
Primary angioplasty	34 (53.9%)	25 (50%)	9 (69.3%)
Rescue angioplasty	28 (44.4%)	24 (48%)	4 (30.7%)
Facilitated angioplasty	1 (1.5%)	1 (2%)	0
Hemodynamic complications	8 (12.6%)	8 (16%)	0
Atrioventricular block (%)	7 (11%)	7 (14%)	0
Death in the acute phase	1 (1.5%)	1 (2%)	0

p = NS.  
CK-MB = creatine kinase-MB; CPK = creatine phosphokinase; ECG = electrocardiogram.

**Electrocardiographic recordings:** Standard 12-lead electrocardiograms were recorded at a speed of 25 mm/s and a voltage of 10 mm/mV at the time of admission. Electrocardiographic tracings were reviewed by 2 independent investigators blinded to patients' clinical and angiographic data. In cases of discrepancy, the final decision was made by the third investigator. ST changes were measured at 20, 40, 60, and 80 ms from the J point in all leads. Because there were no significant differences in sensitivity, specificity, and predictive values across results obtained at different points, we used changes at 60 ms for the final analysis. Measurements were made to the nearest 0.5 mm (0.05 mV). The TP segment on the electrocardio-

gram was used as an isoelectric line. The ST segment between <0.5 mm depression and <0.5 mm elevation was considered isoelectric.

The specificity, sensitivity, and positive and negative predictive values for different electrocardiographic criteria were studied individually (Table 2) and in combination to find the most sensitive algorithm that would produce the best results for the prediction of a culprit artery (Figure 2). The electrocardiographic criteria used in our algorithm considered the most useful for the identification of the culprit artery were (1) ST changes in lead I, (2) the ratio of ST elevation in lead III to that in lead II, and (3) ratio of the sum of ST depression in the precordial leads to the sum of ST elevation in the inferior leads [ $(\sum \downarrow \text{ST in } V_1 \text{ to } V_3) / (\sum \uparrow \text{ST in II, III, and aVF})$ ].

**Coronary angiography:** All patients underwent coronary angiography within the first 12 hours from symptom onset. Angiographic findings were evaluated by 2 independent investigators blinded to the clinical and electrocardiographic data. In cases of discrepancy concerning the results, the final decision was made by the third investigator. Critical stenosis was defined as >70% narrowing of the coronary artery luminal diameter, and only patients with critical stenosis in 1 vessel were recruited. A culprit artery was determined from the correlation between the electrocardiograms and the characteristics of occlusion (occlusion due to thrombosis formation and/or ulceration with decreased contrast density). Patients were classified into 2 groups, those with occlusion of the RCA and those with occlusion of the LCx.

**Statistical analysis:** Groups were compared with chi-square test and Fisher's correction when appropriate. A p value <0.05 was considered statistically significant. Sensitivity, specificity, and positive and negative predictive values were assessed for

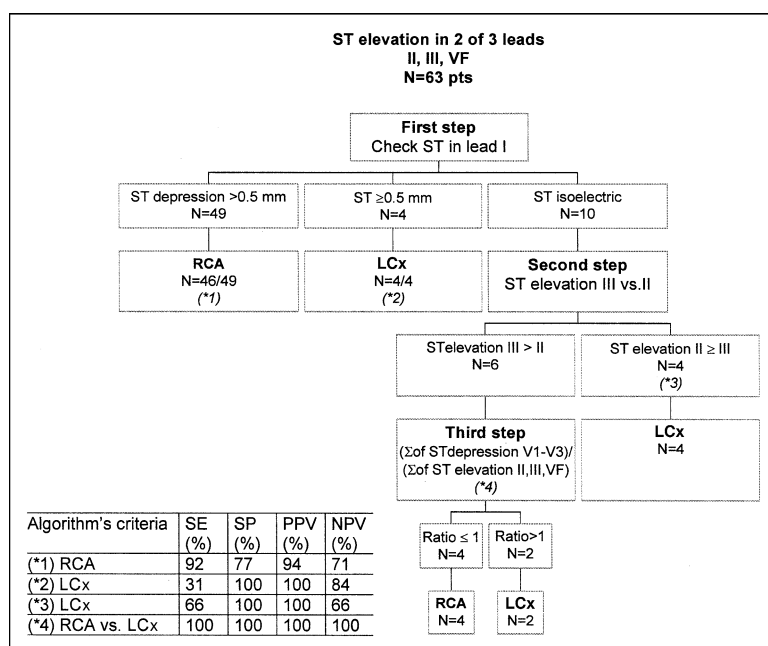
all criteria and for the sequential analysis of combinations produced by the algorithms.

## RESULTS

**Clinical characteristics of patients:** Sixty-three patients (50 men and 13 women, mean age of 58 ± 11 years) met the inclusion criteria. Table 1 lists the clinical characteristics of the study groups.

**Electrocardiographic and angiographic findings:** The mean time from symptom onset to recording of the initial electrocardiogram was 150 ± 73 minutes. Angiography showed RCA occlusion in 50 patients and LCx occlusion in 13 patients. RCA was a dominant artery in 40 patients.

<b>TABLE 2</b> Values of Sensitivity (SE), Specificity (SP), and Predictive Positive values (PPV) of Different ECG Criteria to Predict Occlusion of a Culprit Artery									
ECG Criteria	RCA Occlusion				LCx Occlusion				
	SE	SP	PPV	NPV	SE	SP	PPV	NPV	
ST in lead I ↓ ST ≥0.5 mm ST isoelectric ↑ ST ≥0.5 mm	92	77	94	71	54	92	64	88	
Ratio of ST elevation in lead III vs lead II >1 ≤1	88	69	92	60	31	100	100	85	
Σ ↓ ST in leads V <sub>1</sub> –V <sub>3</sub> Vs Σ ↑ ST in leads II, III, and aVF >1 ≤1	69	92	69	92	61	94	73	90	
	94	61	90	73					
ECG = electrocardiographic.									



**FIGURE 2.** Sequential electrocardiographic algorithm to predict the culprit artery involved in an evolving AMI with ST elevation in leads II, III, and aVF. Numbers in parentheses correspond to global values of sensitivity (SE), specificity (SP), positive predictive value (PPV), and negative predictive value (NPV) of the electrocardiographic criteria that were assessed in successive steps of the algorithm (table).

**Electrocardiographic criteria for the prediction of a culprit artery:** Table 2 presents the sensitivity, specificity, and predictive values of different electrocardiographic criteria corresponding to a culprit artery.

Different sequential algorithms were developed, and those that seemed to identify a culprit artery were chosen. The algorithm shown in Figure 2 correlated the best, with angiographic findings showing the highest accuracy to predict a culprit artery. With this algorithm, we were able to identify the culprit artery in >95% of patients. Figure 3 presents examples of electrocardiographic changes found in patients with occlusion in the RCA or LCx that corresponded to the sequential algorithm. In Figure 3, the diagnosis of a culprit artery was achieved in the first step, the culprit artery was identified in the second step of the algo-

riethm, and the diagnosis and culprit artery were established in the third step in case these were not clear in the first and second steps.

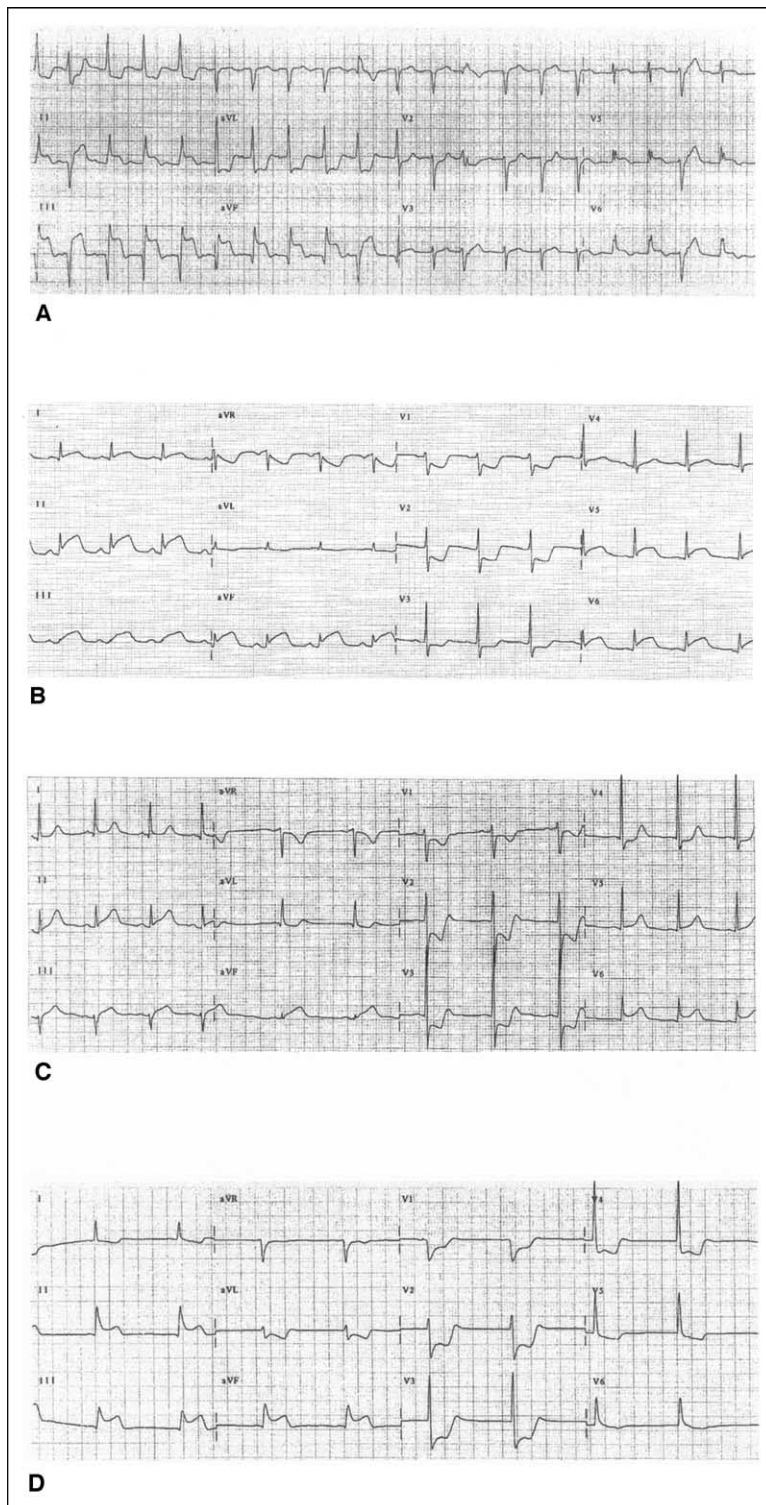
## DISCUSSION

The outcome of patients with AMI in the inferior wall depends in large part on the occluded artery (RCA or LCx). Patients with AMI due to occlusion in the RCA (~80% of cases) usually have a poorer outcome than do those with occlusion of the LCx, mainly due to associated complications (hemodynamic changes due to right ventricular involvement and conduction disturbances).<sup>11–13</sup> An electrocardiogram may detect right ventricular involvement, but true right ventricular infarction may be ascertained by assessment of hemodynamic parameters and confirmed by imaging techniques.<sup>14</sup> Therefore, it is important from a clinical point of view to predict the culprit artery during evolving AMI. The RCA supplies blood mainly to the posterior part of the septum and the inferior part of the inferoposterior wall of the myocardium, whereas the LCx supplies blood mainly

to the posterior part of the inferoposterior wall and the posterior part of the lateral wall (Figure 4). Because ST elevation in leads II, III, and aVF is key to diagnosing an evolving AMI that affects the inferoposterior wall of the heart, some characteristic ST changes in these leads, reciprocal leads (I, aVL, and V<sub>1</sub> to V<sub>3</sub>), and the right precordial leads (V<sub>3</sub>R to V<sub>5</sub>R) may provide information that identifies the culprit artery (RCA or LCx).

To identify the extent of injury to the myocardium caused by the culprit artery, one must assess ST changes in the inferior leads (inferior part of the inferoposterior wall), leads V<sub>1</sub> to V<sub>3</sub> (ST depression reflects the posterior part of the inferoposterior wall), and the lateral leads (I, aVL, and V<sub>5</sub> to V<sub>6</sub>). Leads V<sub>3</sub>R to V<sub>5</sub>R are also very useful for the assessment of





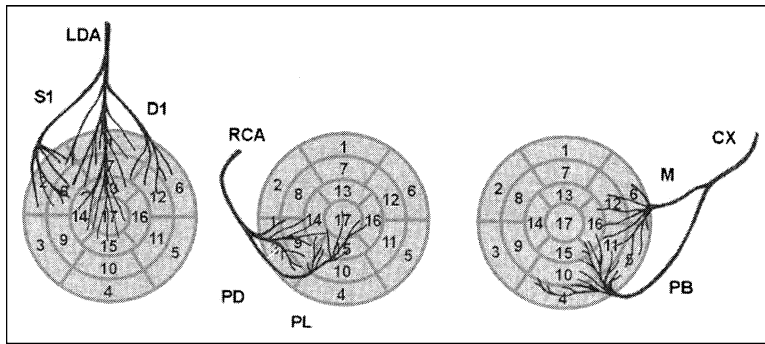
**FIGURE 3.** Electrocardiographic tracings of patients who had inferoposterior AMI (see text for details). (A) ST depression in lead I (first step) identifies the RCA as the culprit artery (\*1 in Figure 2). ST elevation that is higher in lead III than in lead II also may be observed. (B) ST elevation in lead I (first step) identifies the LCx as the culprit artery (\*2 in Figure 2). (C) ST elevation that is equal in leads II and III (second step) identifies the LCx as the culprit artery in an isoelectric ST segment in lead I (\*3 in Figure 2). (D) A ratio  $>1$  for  $\sum \downarrow$  ST in leads V<sub>1</sub> to V<sub>3</sub> versus  $\sum \uparrow$  ST in leads II, III, and aVF (third step) identifies the LCx as the culprit artery when the first and second steps do not provide a clear diagnosis (isoelectric ST segment in lead I, i.e.,  $<0.5$  mm depression, and ST elevation that is higher in lead III than in lead II; \*4 in Figure 2).

right ventricular involvement.<sup>15</sup> Nevertheless, the electrocardiographic changes in these leads are very specific but disappear in the early stage in the evolution of AMI. Further, sometimes there are no changes in leads V<sub>3</sub>R to V<sub>5</sub>R due to the presence of concomitant lateral or posterior involvement.<sup>16</sup> Another important disadvantage of a diagnosis based on these leads is that they are often not recorded in most emergency rooms. Hence, different electrocardiographic criteria based on the ups and downs of ST changes on 12-lead electrocardiograms have been developed to identify the culprit artery.<sup>2–10</sup> Based on the highest sensitivity, specificity, and predictive values and on our experience, we have proposed a sequential algorithm to identify the culprit artery with the highest accuracy. The remainder of this work discusses this algorithm.

An injury vector in the presence of ST elevation in leads II, III, and aVF and a ST depression in lead I identifies the RCA as the culprit artery. In this case, the injury vector is directed to the right and provokes a ST depression in lead I (Figure 1). A ST elevation in lead I suggests occlusion of the LCx, because the injury vector is directed leftward (Figure 1). An isoelectric ST segment in I lead may be found with occlusion of the RCA or the LCx.

The importance of ST depression in leads I and aVL (ST depression is usually greater in lead aVL than in lead I) as a marker of occlusion in the RCA has been stressed by many investigators<sup>3–7</sup> who observed that the criterion of ST depression in lead I strongly favors AMI caused by occlusion of the RCA. Chia et al<sup>3</sup> found that occlusion of the RCA never presents ST elevation in lead I, whereas ST depression was present in most patients in whom the RCA was the culprit artery. These observations were supported by Bairey et al,<sup>4</sup> who found elevated or isoelectric ST segments in lead I in 100% of patients who had AMI of the inferior wall with occlusion of the LCx and only 28% of patients who had occlusion of the RCA. Berry et al<sup>5</sup> observed ST-segment depression in leads I and aVL only during occlusion of the RCA, and Huey et al<sup>6</sup> reported that ST depression in lead I was significantly less common in LCx-related AMI. Similar results have been reported by Hasdei et al.<sup>7</sup>

In our study, ST depression  $\geq 0.5$



**FIGURE 4.** Heart walls and segments and coronary artery blood supply including only the most important branches. CX = left circumflex artery; D1 = first diagonal artery; LDA = left anterior descending artery; M = marginal artery; PB = posterobasal artery; PD = posterior descending artery; PL = posterolateral artery; S1 = first septal artery.

mm in lead I had the greatest accuracy for predicting occlusion of the RCA. The criterion of ST depression  $\geq 0.5$  mm in lead I was present in 46 of 49 patients who had occlusion of the RCA (Figure 2). The other 4 patients who presented this criterion had very dominant occlusion of the LCx. This observation may be explained by the injury vector pointing slightly to the right; alternatively, all 4 patients with ST elevation in lead I may have presented with occlusion of the LCx. The other 10 patients presented isoelectric ST or ST elevation/depression  $< 0.5$  mm in lead I, and for these patients, we had to perform additional steps and use another criterion to identify the culprit artery (Figure 2). Therefore, in the first step, we were able to identify the culprit artery in 50 of 63 patients.

In a second step to confirm the presumptive diagnosis provided by the ups and downs of the ST segment in lead I but specifically in cases of an isoelectric ST segment in lead I, one should examine the magnitude of ST-segment elevation in lead III versus that in lead II. If ST elevation in lead II is equal to or greater than that in lead III, the LCx is the likely culprit because the injury vector is directed leftward; conversely, if ST elevation is higher in lead III than in lead II, then the RCA is the likely culprit. This was observed by Herz et al<sup>8</sup> who found higher ST elevation in lead III than in lead II in 80% of patients with occlusion of the RCA versus that of the LCx. According to these investigators, the criterion of higher ST elevation in lead III than in lead II and a lower ST depression in lead aVL than in lead I is a sensitive and specific marker for RCA-related AMI. Nair et al<sup>9</sup> observed that an ST elevation that is higher in lead III than in lead II is more sensitive (96% vs 86%) than ST elevation in lead V<sub>4</sub>R for the identification of the RCA as the culprit artery. The criterion of higher ST elevation in lead III than in lead II was used not only to predict the culprit artery but also to precisely locate the place of occlusion. Proximal occlusion of the RCA is usually associated with right ventricular involvement. Saw et al<sup>9</sup> demonstrated that higher ST elevation in lead III than in lead II is more sensitive than higher ST elevation in lead V<sub>4</sub>R for the diagnosis of

right ventricular AMI. Our study has confirmed the value of a ST elevation that is higher in lead III than in lead II for the prediction of the RCA as the culprit artery. Most patients who had ST depression in lead I also had ST elevation that was higher in lead III than in lead II, and 42 of 46 patients who presented with the 2 criteria (ST depression in lead I and ST elevation that was higher in lead III than in lead II) had occlusion of the RCA. The application of this criterion seems to be very useful for identifying the culprit artery on electrocardiograms in which ST is isoelectric in lead I, which occurred in 10 of 63 patients (16%). When electrocardiograms showed ST elevation that was at least as high in

lead II as in lead III, the LCx was the culprit artery (4 of 10 patients).

In the remaining 6 patients, we had to proceed to the third step, which compares ST depression in leads V<sub>1</sub> to V<sub>3</sub> with ST elevation in the inferior leads. Kosuge et al<sup>2</sup> distinguished between the RCA and the LCx as the culprit artery by assessing the ratio of ST depression in lead V<sub>3</sub> to ST elevation in lead III. A ratio  $> 1.2$  indicated the LCx as the culprit artery with high sensitivity and specificity. We assessed this criterion by using the ratio of the sum of ST changes  $[(\sum \downarrow \text{ST of leads V}_1 \text{ to V}_3) / (\sum \uparrow \text{ST of leads II, III, and aVF})]$ . This criterion compared favorably with that of Kosuge et al<sup>2</sup> by being more sensitive (61% vs 31%) and as specific (94% vs 96%) for detecting the LCx as the culprit artery. When assessed in a sequential algorithm in the third step, the sensitivity and specificity of our criterion reached 100%. Our modified criterion effectively compared ST depression in the precordial leads with ST elevation in the inferior leads in the 6 patients whose culprit artery could not be identified by ST changes in lead I and by the ratio of ST elevation in lead II to that in lead III. In these patients who had an isoelectric ST segment in lead I and a ST elevation that was higher in lead III than in lead II, a ratio  $\leq 1$  for  $\sum \downarrow \text{ST in leads V}_1 \text{ to V}_3$  versus  $\sum \uparrow \text{ST in leads II, III, and aVF}$  identified the RCA as the culprit artery in 4 patients and a ratio  $> 1$  identified the LCx as the culprit artery in the remaining 2 patients.

In this study, we focused on prediction of a culprit artery and did not analyze electrographic criteria for prediction of right ventricular involvement. Nevertheless, an isoelectric or elevated ST segment in lead V<sub>1</sub> was a better predictor than higher ST elevation in lead III than in lead II of a proximal occlusion of the RCA.<sup>17</sup>

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