Evaluation By Quantitative 99m-Technetium MIBI SPECT and Echocardiography of Myocardial Perfusion and Wall Motion Abnormalities in Patients With Dobutamine-Induced ST-Segment Elevation

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ST-segment elevation during exercise testing has been attributed to myocardial ischemia and wall motion abnormalities (WMA). However, the functional significance of ST-segment elevation during dobutamine stress testing (DST) has not been evaluated in patients referred for diagnostic evaluation of myocardial ischemia. DST (up to 40 μg/kg/min) with simultaneous echocardiography and technetium-99m sestamibi single-photon emission computed tomography (SPECT) was performed in 229 consecutive patients with suspected myocardial ischemia who were unable to perform an adequate exercise test; 127 (55%) had a previous acute myocardial infarction (AMI). ST elevation was defined as ≥1 mm new or additional J point elevations with a horizontal or upsloping ST segment lasting 80 ms. Reversible perfusion defects on SPECT and new or worsening WMA during stress on echocardiography were considered diagnostic of ischemia. ST elevation occurred in 40 patients (17%) during the test; 34 of them (85%) had previous AMI. All patients with ST-segment elevation had abnormal scintigrams (fixed or reversible defects, or both) and abnormal wall motion (fixed or transient defects, or both) at peak stress. In patients who had ST elevation and no previous AMI (n = 6), ischemia was detected in all by echocardiography and in 5 (83%) by SPECT. In patients with previous AMI, the prevalence of ischemia was not different with or without ST elevation (53% vs 43% by echocardiography and 53% vs 48% by SPECT, respectively). Baseline regional wall motion score in the infarct zone was higher in patients with ST elevation. In conclusion, myocardial perfusion defects and WMA at peak stress are a hallmark in patients with ST-segment elevation during DST. However, ST-segment elevation is a specific marker of ischemia only in patients without previous AMI.

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Exercise-induced ST-segment elevation in the electrocardiogram has been attributed to left ventricular dysfunction, myocardial aneurysm, or transmural myocardial ischemia due to severe coronary artery stenosis or coronary spasm.[1–11] Despite the known role of wall motion abnormalities (WMA) in patients with ST elevation during exercise,[4,6] previous studies have focused mainly on resting WMA, and little attention was paid to stress-induced WMA that may be associated with this electrocardiographic finding. It has been postulated that WMA due to either myocardial infarction or exercise-induced ischemia represent a common underlying mechanism of exercise-induced ST elevation. However, few data are available to support this contention.[8] In a few studies, ST elevation during dobutamine stress testing (DST) was attributed to myocardial ischemia in the absence of previous acute myocardial infarction (AMI)[12] and to stress-induced left ventricular asynergy in patients evaluated early after AMI.[13] However, the functional significance of this electrocardiographic finding has not been reported in patients referred for diagnostic evaluation of myocardial ischemia. Accordingly, the aim of this study was to evaluate the prevalence and functional significance of ST elevation during DST in patients with suspected myocardial ischemia undergoing DST with simultaneous echocardiography and technetium-99m sestamibi single-photon emission computed tomography (SPECT).

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METHODS

Study population: The study population comprised 229 consecutive patients (137 men and 92 women, mean age 59 ± 11 years) with known or suspected coronary artery disease unable to exercise or to perform an adequate exercise test, referred to our imaging laboratory for evaluation of chest pain by dobutamine technetium-99m sestamibi SPECT. Simultaneous echocardiography was performed in all patients as a part of a research protocol in our center. All patients gave informed consent to undergo the study. Patients with bundle branch block or ventricular hypertrophy were excluded. One hundred twenty-seven patients (55%) had a previous AMI, which

TABLE I Clinical Characteristics and Hemodynamic Variables During Dobutamine Stress Testing in Patients With (group A) and Without (group B) ST-Segment Elevation

	Group A (n = 40)	Group B (n = 189)	p Value
Men/women	31/9	106/83	<0.05
Mean age (yr)	58 ± 10	59 ± 12	NS
Previous AMI	34 (85)	93 (49)	<0.0001
Resting HR (beats/min)	72 ± 13	69 ± 14	NS
Peak HR (beats/min)	132 ± 16	134 ± 16	NS
85% of target HR reached	34 (79)	158 (82)	NS
Resting systolic BP (mm Hg)	136 ± 31	135 ± 21	NS
Peak systolic BP (mm Hg)	139 ± 35	146 ± 30	NS
Resting diastolic BP (mm Hg)	77 ± 13	79 ± 12	NS
Peak diastolic BP (mm Hg)	74 ± 15	75 ± 14	NS
Chest pain	12 (30)	48 (25)	NS
ST-segment depression	14 (35)	33 (17)	<0.05

Values are expressed as mean ± SD or number of patients (%).
AMI = acute myocardial infarction; BP = blood pressure; HR = heart rate.

TABLE II Echocardiographic and Scintigraphic Findings in 102 Patients Without Previous Infarction With (group A) and Without (group B) ST-Segment Elevation

	Group A (n = 6)	Group B (n = 96)	p Value
Normal echocardiography	0	70 (73)	<0.005
WMA at rest	2 (33)	12 (13)	NS
WMA at peak stress	6 (100)	26 (27)	<0.005
New or worsened WMA	6 (100)	22 (23)	<0.0001
Normal scintigraphy	0	63 (66)	<0.005
RPD with or without FPD	5 (83)	27 (28)	<0.005

Values are expressed as number of patients (%).
FPD = fixed perfusion defects; RPD = reversible perfusion defects; WMA = wall motion abnormalities.

TABLE III Echocardiographic and Scintigraphic Findings in 127 Patients With Previous Infarction With (group A) and Without (group B) ST-Segment Elevation

	Group A (n = 34)	Group B (n = 93)	p Value
Normal echocardiogram	0	16 (17)	<0.01
WMA at rest	32 (94)	69 (74)	<0.05
WMA at peak stress	34 (100)	77 (83)	<0.01
New or worsened WMA	18 (53)	40 (43)	NS
WMS at rest	25.0 ± 6.4	21.1 ± 4.8	<0.005
WMS at peak stress	26.7 ± 6.4	22.2 ± 6.4	<0.0001
Ischemic WMS	1.9 ± 3.2	1.8 ± 3.2	NS
Akinetic + dyskinetic segments at rest	2.9 ± 3	1.1 ± 1.7	<0.001
Akinetic + dyskinetic segments at peak	3.7 ± 3.1	1.7 ± 2.1	<0.001
Normal scintigram	0	11 (12)	<0.01
RPD with or without FPD	18 (53)	45 (48)	NS
Perfusion defect score at rest	3,023 ± 2,955	1,589 ± 1,345	<0.05
Perfusion defect score at peak stress	2,978 ± 2,563	1,775 ± 2,281	<0.05
Ischemic perfusion score	401 ± 737	389 ± 1,322	NS

Values are expressed as mean ± SD or number of patients (%).
WMS = wall motion score; other abbreviations as in Table II.

was recent (<1 month) in 34 patients. The diagnosis of AMI relied upon a typical history of chest pain, a diagnostic increase in serum creatine kinase and evolutional electrocardiographic changes. On the day of the test, 154 patients (67%) were receiving antianginal therapy; 113 of them were receiving β blockers.

Dobutamine stress test: Dobutamine was infused through an antecubital vein starting at a dose of 10 μg/kg/min, increasing by 10 μg/kg/min every 3 minutes to a maximum of 40 μg/kg/min. Atropine (up to 1 mg) was given to patients not achieving 85% of their age-predicted maximal heart rate.[14] A 12-lead electrocardiogram was recorded each minute. The level of ST segment was calculated after signal averaging by a computer-assisted system (Cardiovet CSG/12, Schiller, Baar, Switzerland). Cuff blood pressure was measured every 3 minutes. The electrocardiograms were revised by 2 experienced cardiologists unaware of clinical, echocardiographic, or scintigraphic data. Pathologic Q waves were defined according to established criteria.[15] ST elevation was defined as new or additional elevation ≥1 mm at the J point, with a horizontal or upsloping ST-segment lasting 80 ms during stress in ≥1 electrocardiographic lead (the PQ segment was considered the isoelectric line).[3] ST-segment depression was defined as ≥1 mm horizontal or downsloping depression 80 ms after the J point, and below the resting baseline level. The test was interrupted prematurely if severe chest pain, ST-segment depression >2 mm, ST elevation >2 mm in patients with normal baseline electrocardiogram, significant ventricular or supraventricular arrhythmia, or a systolic blood pressure decrease of >40 mm Hg occurred during the test.

Sestamibi single-photon emission computed tomography imaging: Approximately 1 minute before the termination of the stress test, an intravenous dose of 370 MBq of sestamibi was administered. Stress SPECT imaging was begun 1 hour after sestamibi injection. For the resting studies, 370 MBq of sestamibi was injected 24 hours after the first study. Left ventricular images were divided into 6 segments: anterior, lateral, inferoposterior, interventricular septum (subdivided in anterior and posterior septum), and apex. Image interpretation was performed by an experienced observer unaware of the patients' electrocardiographic or echocardiographic data. A persistent perfusion defect on both stress and resting imaging was classified as a fixed defect. A reversible defect was defined as a perfusion defect on stress images that partially or completely resolved at rest imaging. This was considered diagnostic of ischemia.[16] The interpretation of the scan was semiquantitatively performed by visual analysis, and assisted by the circumferential profiles analysis. To assess perfusion defect size, perfusion defect score was quantitatively calculated at rest and at stress images by measuring the area between the lower limit of normal values (±2 SD) and the actual circumferential

profile in 6 short-axis slices. Ischemic perfusion score was derived by subtracting rest from stress score in segments with reversible defects.

Stress echocardiography: Stress echocardiography was performed in all patients according to a previously described protocol.[16] For both rest and stress studies, the left ventricular wall was divided into 16 segments and scored using a 4-point scale: 1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia. Both inward endocardial motion and myocardial thickening were considered for analysis. Wall motion score was derived by the summation of the score of the 16 segments. The diagnosis of ischemia was based on the occurrence of new or worsening WMA during the test, compared with baseline, in ≥1 segment. As we have previously concluded,[17] ischemia was not considered when akinetic segments at rest became dyskinetic during stress. Ischemic wall motion score was defined as the difference between peak and rest regional wall motion score in ischemic segments. Assessment of images was performed by 2 experienced investigators without knowledge of scintigraphic or electrocardiographic data. In case of disagreement, a consensus was reached with a third investigator. In our center, the inter- and intraobserver variability for the interpretation of stress echocardiographic studies is 91% and 92%, respectively.[18]

Coronary angiography: Coronary angiography was performed, using the Judkins technique, within 3 months in 106 patients (46%). Significant coronary artery disease was defined as a diameter stenosis of ≥50% in ≥1 major epicardial artery.

Regional myocardial function and perfusion: In patients with myocardial infarction, the location of resting WMA was presumed to represent the infarction zone. To assess regional myocardial perfusion and function, 2 myocardial segments were identified: (1) the anterior segment, which included the anterior wall, the interventricular septum, and the apex (assigned to leads V_1 to V_4); and (2) the inferior segment, which included the inferior and posterior wall (assigned to leads II, III, and aVF). Because of the infrequent occurrence of resting WMA confined only to the lateral wall, this segment, together with leads I, aVL, V_5, and V_6, were added to either the anterior or the inferior segment in the presence of concomitant abnormalities in 1 of these segments. Myocardial segments at echocardiography and at SPECT were matched in these 2 locations.

Statistical analysis: Unless specified, data are presented as mean values ± SD. The chi-square and Fisher's exact tests were used to compare differences between proportions. The Student's *t* test was used for analysis of continuous data. A p value <0.05 was considered statistically significant.

RESULTS

Dobutamine stress test: Heart rate and systolic blood pressure increased significantly from rest to peak stress (70 ± 13 vs 134 ± 16 beats/min and 135 ± 25 vs 145 ± 32 mm Hg, respectively, p <0.0001 for both). In 30 patients, the test was interrupted prematurely before reaching the maximal dose or 85% of the target heart rate because of a limiting side effect (angina in 15 patients, ST-segment depression in 7 patients, hypotension in 6 patients, and significant tachyarrhythmias in 2 patients).

ST elevation of ≥1 mm occurred in 34 of 127 patients with (27%) and in 6 of 102 patients without (6%) previous AMI. These 40 patients formed group A. Group B comprised 189 patients without ST elevation; 93 of them (49%) had previous AMI. Patients in group A had a higher prevalence of previous AMI and male gender. In patients with previous AMI, pathologic Q waves were detected in 27 of 34 group A patients (79%) and in 59 of 93 group B patients (63%), p = NS. Clinical characteristics and hemodynamic data in both groups are listed in Table I. Echocardiographic and scintigraphic findings in both groups are listed in Tables II (patients without AMI) and III (patients with previous AMI).

Stress echocardiography: ST elevation was associated with a higher prevalence of myocardial ischemia in patients without (Figure 1) but not with (Figure 2) pre-

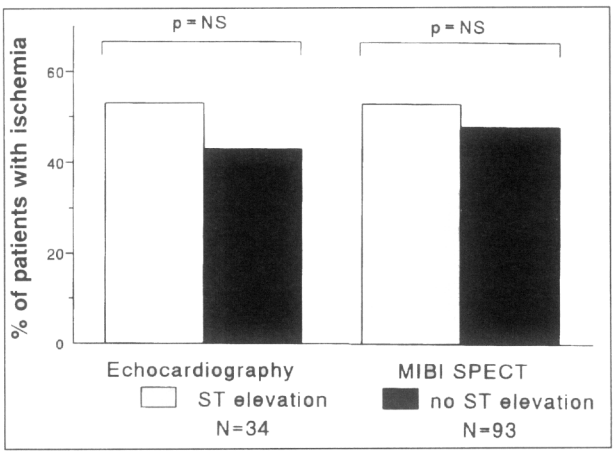

FIGURE 1. Prevalence of ischemia in patients without previous myocardial infarction with (n = 6) and without (n = 96) ST-segment elevation as diagnosed by echocardiography and technetium-99m sestamibi single-photon emission computed tomography (MIBI SPECT).

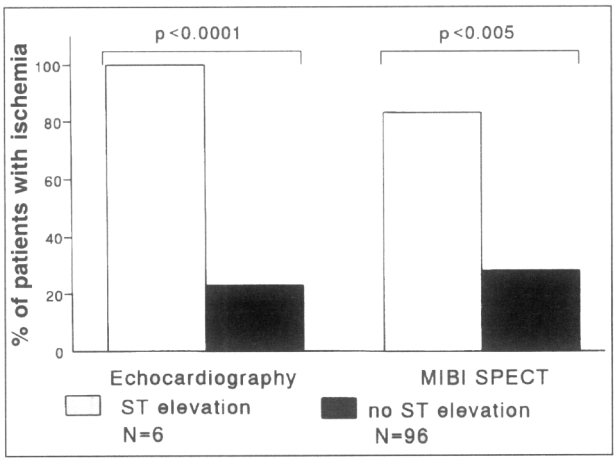

FIGURE 2. Prevalence of ischemia in patients with previous myocardial infarction with (n = 34) and without (n = 93) ST-segment elevation as diagnosed by echocardiography and technetium 99m sestamibi single-photon emission computed tomography (MIBI SPECT).

TABLE IV Regional Wall Motion and Myocardial Perfusion Defect Score in Anterior Myocardial Segments With Baseline Dyssynergy With and Without ST-Segment Elevation During Dobutamine Stress Testing in Patients With Previous Myocardial Infarction

	ST Elevation (25 segments)	No ST Elevation (42 segments)	p Value
Rest WMS	18.3 ± 5.7	15.5 ± 4.5	<0.05
Stress WMS	19.1 ± 5.9	16.0 ± 5.0	<0.001
Ischemic WMS	0.9 ± 2.3	0.7 ± 2.1	NS
New and/or worsened WMA	10 (40)	15 (36)	NS
Rest perfusion defect score	1,668 ± 1,531	1,067 ± 1,193	<0.05
Stress perfusion defect score	1,680 ± 1,437	1,180 ± 1,154	<0.05
Ischemic perfusion score	338 ± 628	301 ± 1,003	NS
Reversible perfusion defects	10 (40)	16 (38)	NS

Values are expressed as mean ± SD or number (%).
Abbreviations as in Tables II and III.

TABLE V Regional Wall Motion and Myocardial Perfusion Defect Score in Inferior Myocardial Segments With Baseline Dyssynergy With and Without ST-Segment Elevation During Dobutamine Stress Testing in Patients With Previous Myocardial Infarction

	ST Elevation (17 segments)	No ST Elevation (40 segments)	p Value
Rest WMS	9.0 ± 2.1	7.2 ± 2.6	<0.05
Stress WMS	9.5 ± 2.5	7.8 ± 3.1	<0.05
Ischemic WMS	0.6 ± 1.6	0.7 ± 2.0	NS
New and/or worsened WMA	6 (35)	13 (33)	NS
Rest perfusion defect score	1,284 ± 1,491	899 ± 923	<0.05
Stress perfusion defect score	1,327 ± 1,494	972 ± 1,031	<0.05
Ischemic perfusion score	142 ± 216	144 ± 405	NS
Reversible perfusion defects	7 (41)	14 (35)	NS

Values are expressed as mean ± SD or number (%).
Abbreviations as in Tables II and III.

vious AMI. In patients with previous AMI, ST elevation was associated with a higher prevalence of resting WMA, a higher wall motion score at rest and at peak stress, a similar ischemic wall motion score (Figure 3), and more myocardial segments with akinesia or dyskinesia at rest and at peak stress (Table III). In patients with previous AMI and ST elevation, the prevalence of ischemia did not differ in the presence or absence of Q waves: 15 of 27 (56%) versus 3 of 7 (43%), respectively (p = NS).

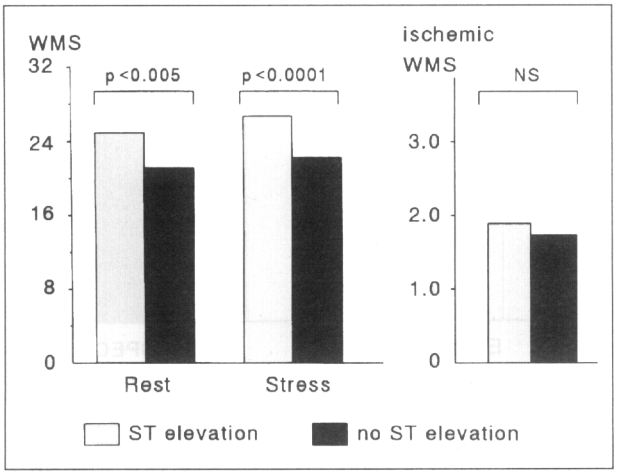

FIGURE 3. Rest, peak, and ischemic wall motion score (WMS) in patients with previous myocardial infarction with (n = 34) and without (n = 93) ST-segment elevation.

Results of sestamibi single-photon emission computed tomography: ST elevation was associated with a higher prevalence of ischemia in patients without (Figure 1) but not with (Figure 2) previous AMI. The electrocardiogram and perfusion scan of a patient without previous AMI, ST elevation during DST, and a completely reversible defect are shown in Figure 4. In patients with previous AMI, ST elevation was associated with a higher perfusion score at rest and at peak stress and a similar ischemic perfusion score compared with patients without ST elevation (Table III). In patients with previous AMI and ST elevation, the prevalence of ischemia did not differ in the presence or absence of Q waves: 16 of 27 (59%) versus 2 of 7 (29%), respectively (p = NS).

Regional myocardial perfusion and function: In patients with previous AMI, regional myocardial perfusion and function were compared in dyssynergic myocardial segments at rest with and without ST elevation during stress in the anterior (Table IV) and inferior (Table V) segments. In both locations, resting score was higher with ST elevation, whereas the ischemic score and prevalence of ischemia were not different with or without ST elevation.

The electrocardiograms and perfusion scans of 2 patients with previous anterior AMI, ST-segment elevation in Q leads during DST, and a perfusion defect on resting images with (Figure 5) and without (Figure 6) partial reversibility are presented.

Analysis of subgroups: PATIENTS WITHOUT PREVIOUS MYOCARDIAL INFARCTION: In patients with reversible perfusion defects and no previous AMI, the ischemic perfusion score was significantly higher in patients with (n = 5) than without (n = 27) ST elevation (994 ± 652 vs 271 ± 407, p <0.01). Similarly, in patients with stress-induced WMA who had no previous AMI, the ischemic wall motion score was significantly higher in patients with (n = 6) than without (n = 22) ST elevation (4.2 ± 3.2 vs 2.7 ± 3.1, p <0.05).

PATIENTS WITH RECENT MYOCARDIAL INFARCTION: ST elevation occurred in 16 of 34 patients (47%) with recent AMI. Prevalence of ischemia was not different with or without ST elevation: 9 of 16 (56%) versus 9 of 18 patients (50%) by echocardiography and 9 of 16 (56%) versus 11 of 18 (61%) by SPECT, respectively.

PATIENTS WITH ST-SEGMENT DEPRESSION: ST-segment depression occurred in 29 patients with and in 18 without previous AMI (14 in group A and 33 in group B). ST depression was associated with a higher prevalence of ischemia in patients without previous AMI (12 of 18 [67%] versus 16 of 84 [19%] [p <0.0001 by echocardiography] and 13 of 18 [72%] versus 19 of 84 [23%] [p <0.0001 by SPECT]) and with previous AMI (18 of 29

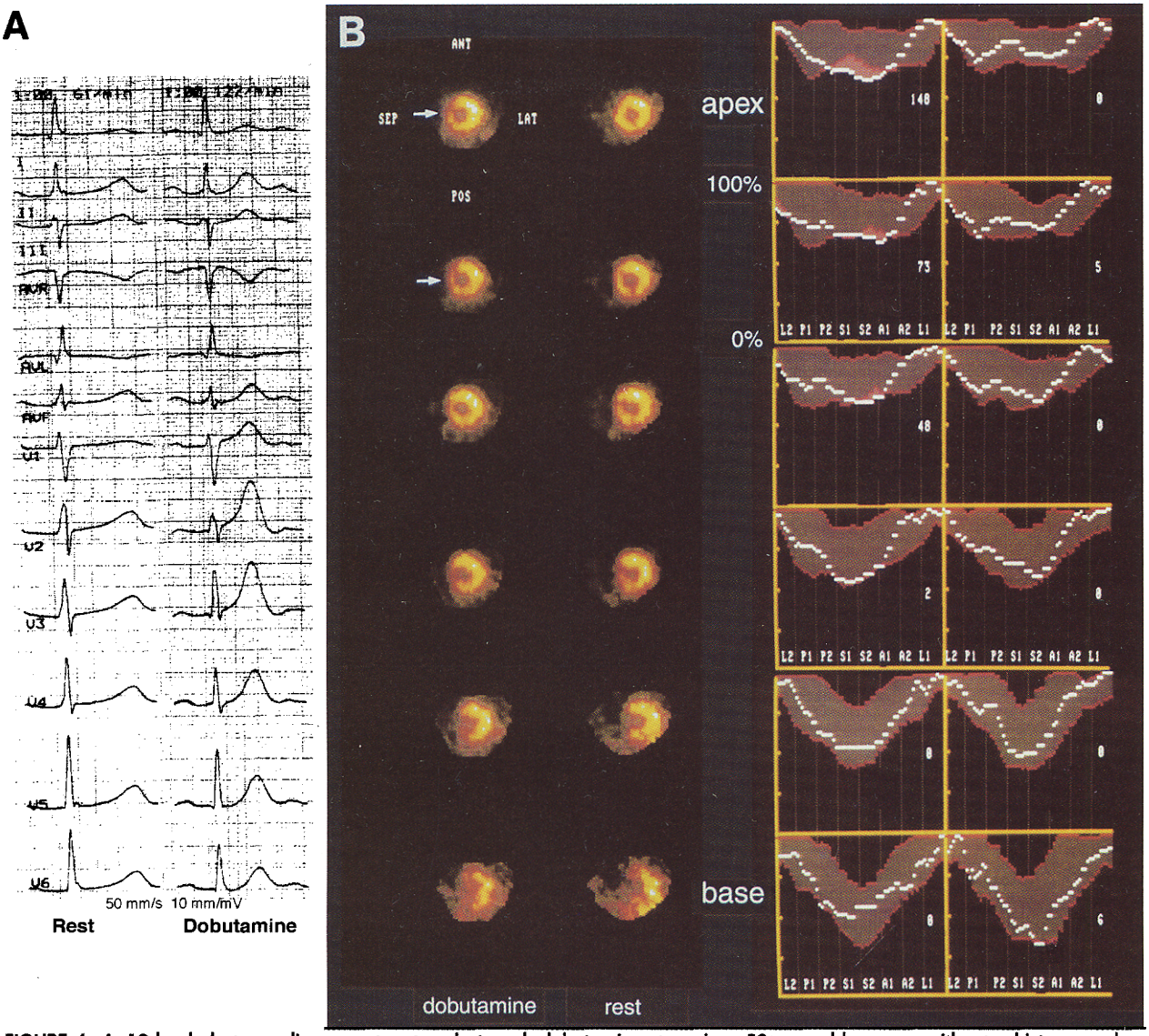

FIGURE 4. *A*, 12-lead electrocardiogram at rest and at peak dobutamine stress in a 53-year-old woman without a history or electrocardiographic evidence of previous myocardial infarction, showing ST-segment elevation in leads V_1 to V_3. *B*, dobutamine stress and rest sestamibi single-photon emission computed tomographic images in the 6 short-axis slices represented together with the corresponding circumferential profile analysis of the same patient, showing a completely reversible perfusion defect in the apical septum *(arrows)*. ANT = anterior; LAT = lateral; POS = posterior; SEP = septal.

[62%] versus 40 of 98 [41%] [p <0.05 by echocardiography], and 20 of 29 [69%] versus 43 of 98 [44%] [p <0.05 by SPECT]). In group A patients with previous AMI (n = 34), the prevalence of ischemia by both techniques was not different in patients with (n = 12) and without (n = 22) concomitant ST-segment depression.

Coronary angiography: Coronary angiography was performed in 19 group A patients (48%). Three of them had no previous AMI. All patients had significant coronary artery disease: 3-vessel (n = 7), 2-vessel (n = 3), and 1-vessel (n = 9) disease. The site of ST elevation was predictive of significant disease in the corresponding coronary artery in all patients.

Eighty-seven group B patients (46%) underwent coronary angiography. Significant coronary artery disease was detected in 59 patients; 3-vessel (n = 6), 2-vessel (n = 17), and 1-vessel (n = 36) disease. No significant disease was detected in 28 patients. In patients with previous AMI, ST elevation was associated with a higher prevalence of total occlusion of ≥1 major artery (50% vs 14%, p <0.005), whereas the prevalence of multivessel disease was 56% in patients with (9 of 16) and 29% in patients without (14 of 48) ST elevation (p = NS).

DISCUSSION

In the present study, the prevalence and functional significance of ST elevation during DST was evaluated in patients with known or suspected coronary artery disease in whom the test was performed with simultaneous echocardiography and technetium-99m sestamibi SPECT for diagnostic evaluation of myocardial ischemia. Our results show that dobutamine-induced ST elevation is a common occurrence in patients with previous AMI. In such patients, ST elevation is associated with more se-

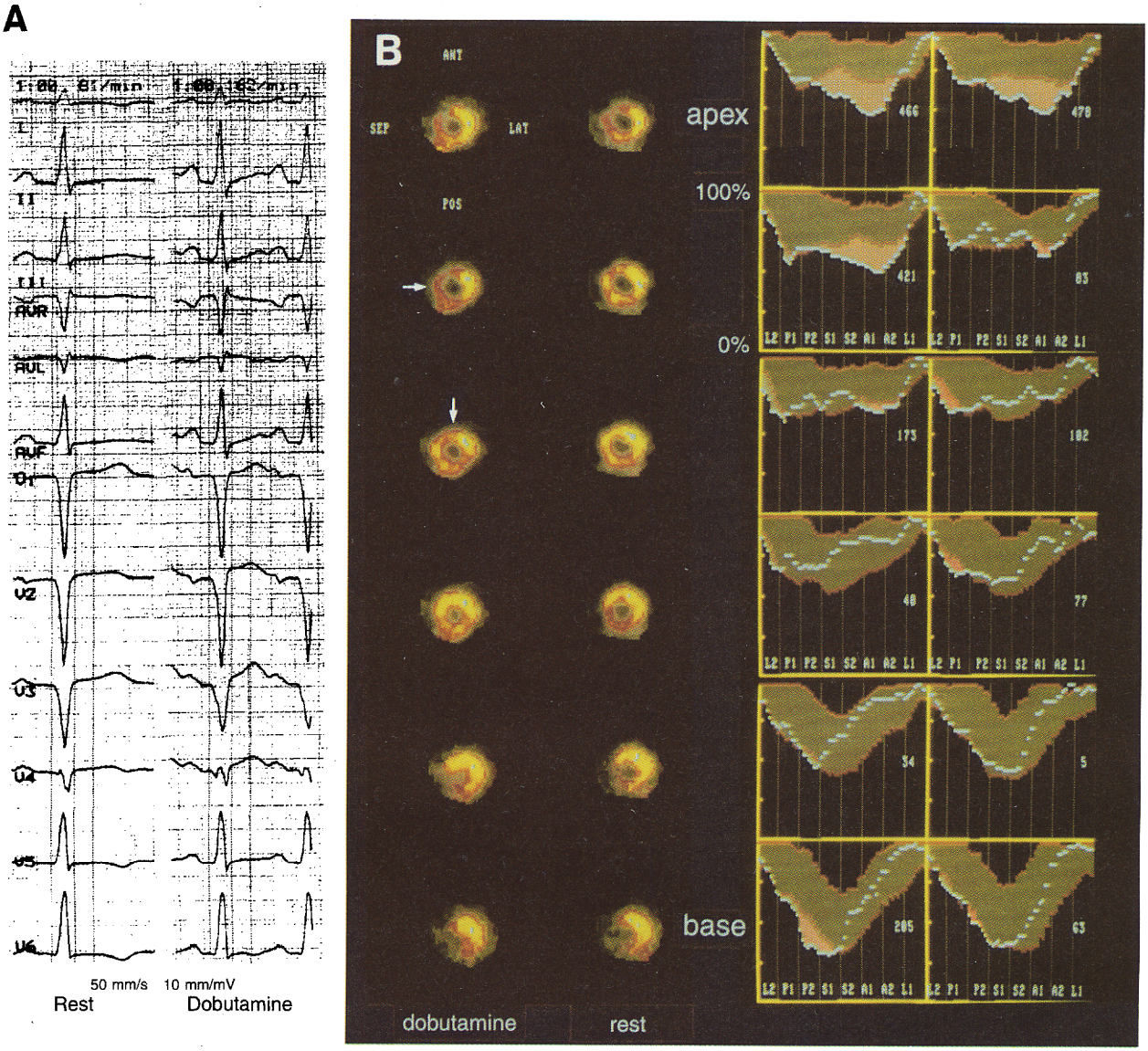

FIGURE 5. *A,* resting and peak dobutamine stress electrocardiogram of a 41-year-old man with a history of old anterior myocardial infarction and Q waves in leads V_1 to V_3, showing ST-segment elevation in V_2 to V_3 on the stress electrocardiogram. *B,* dobutamine stress and rest sestamibi single-photon emission computed tomographic images of the same patient, showing a partially reversible perfusion defect in the apical septum *(horizontal arrow)* and a completely reversible defect in the anterior wall *(vertical arrow).* Abbreviations as in Figure 4B.

vere global and regional left ventricular dysfunction, and a higher prevalence of total occlusion of ≥1 coronary artery. Results also show that despite being compatible with ischemia, ST elevation in patients with recent or old myocardial infarction is not specific for stress-induced overall or peri-infarction ischemia assessed by echocardiography and SPECT. The low specificity for ischemia was also noted in patients without Q waves. This can be explained by the low sensitivity of Q waves for the detection of baseline WMA, especially in patients with old myocardial infarction.[19] In contrast, ST depression was associated with a higher prevalence of ischemia. However, patients with concomitant ST-segment depression and elevation did not have a higher prevalence of ischemia than patients with isolated ST elevation. This may be explained by the occurrence of reciprocal ST depression without true ischemia.[20] In patients without previous AMI, ST elevation was not a usual occurrence (6%) and was associated with ischemia in the corresponding myocardial segments. In patients without previous AMI who had ischemia on echocardiography or SPECT, ischemia was more severe in patients with than without ST elevation.

Comparison with previous studies: Coma-Canella[12] reported ST elevation during DST in 20 of 90 patients with angina and no previous AMI. All had severe coronary artery stenoses. The high prevalence of ST elevation in that study may be related to the selection of patients with a high prevalence of unstable angina. However, no imaging technique was applied for the detection of ischemia. Coma-Canella et al[13] reported that in patients with recent AMI who underwent DST with thal-

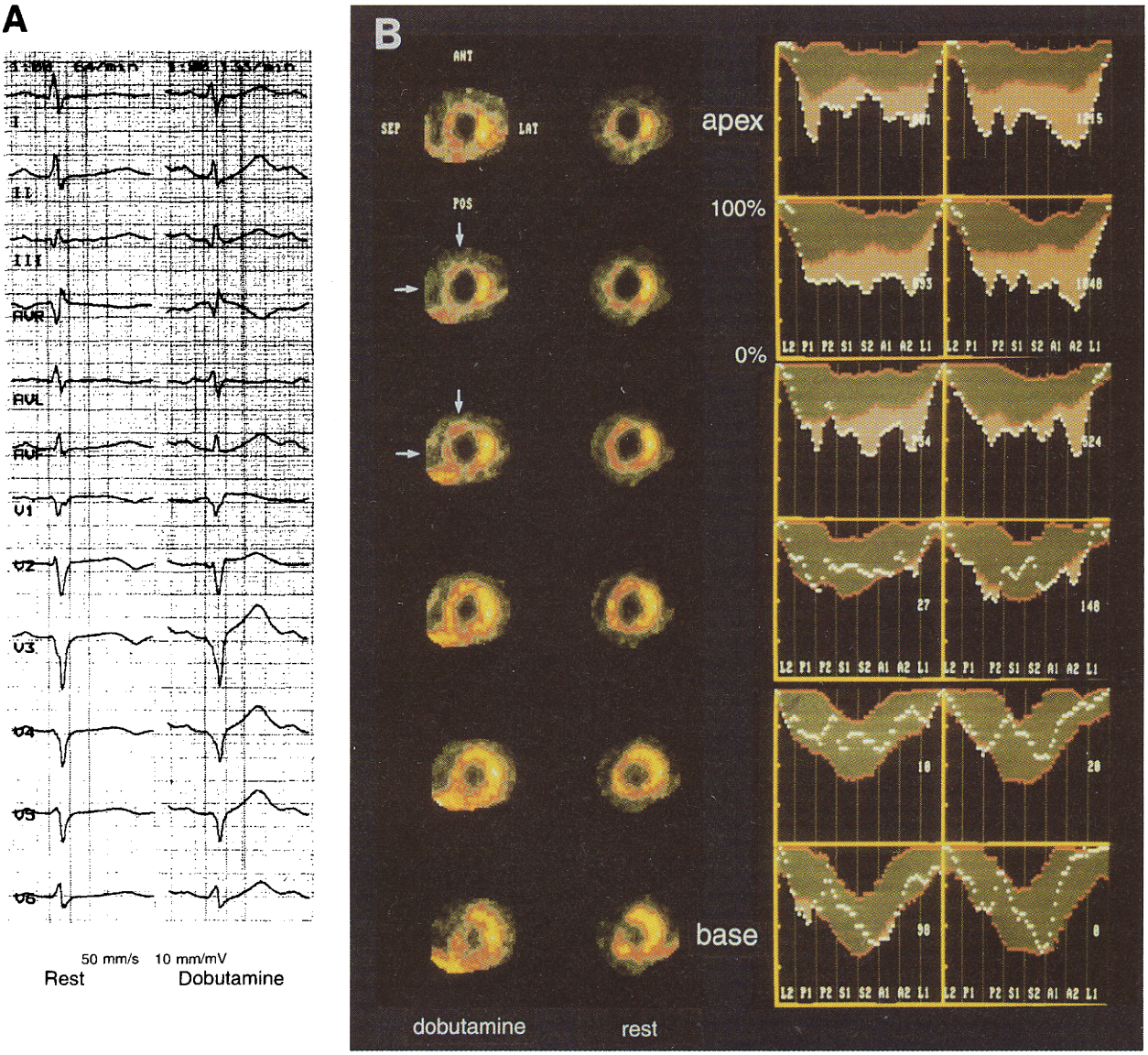

FIGURE 6. *A,* resting and peak dobutamine stress electrocardiogram of a 62-year-old man with a history of old anterior myocardial infarction and Q waves in leads V_1 to V_4, showing ST-segment elevation in V_1 to V_4. *B,* dobutamine stress and rest sestamibi single-photon emission computed tomographic images of the same patient, showing a large and severe fixed perfusion defect in the anterior wall *(vertical arrows)* and septum *(horizontal arrows).* Abbreviations as in Figure 4B.

lium scintigraphy and radionuclide ventriculography, ST elevation was not related to ischemia but to stress-induced left ventricular asynergy. However, the inverse correlation between the change in regional ejection fraction and the level of ST elevation in their study may represent a poor contractile response in myocardial segments corresponding to ST elevation rather than stress-induced asynergy, which is a specific marker of ischemia. Previtali et al[21] reported a case of high-dose dobutamine-induced ST elevation and akinesia of the inferior wall in a patient with 2-vessel disease. Coronary angiography performed during ischemic episodes revealed patency of coronary arteries, excluding coronary spasm as an underlying mechanism. They concluded that dobutamine may induce transmural myocardial ischemia in the presence of severe coronary lesions by increasing myocardial oxygen demand and inducing myocardial blood flow maldistribution.

ST elevation during exercise has been attributed to left ventricular WMA, myocardial aneurysm, or transmural myocardial ischemia due to either severe coronary artery spasm or fixed coronary artery disease.[1–11] Gallik et al[7] reported that exercise-induced ST elevation is an ominous sign of severe reversible hypoperfusion in patients without previous AMI. In our study, ST elevation identified a population with more severe reversible hypoperfusion and stress-induced WMA than patients who had ischemia without ST elevation. The absence of a history or electrocardiographic finding of AMI appears to be the most common characteristic that separates patients in whom stress-induced ST elevation reflects severe ischemia from those with marked abnormality of

left ventricular function.[8] However, exercise-induced ST elevation in patients after acute AMI has been attributed in some studies to peri-infarction ischemia.[10–11] Margonato et al[10] reported that exercise-induced ST elevation after acute AMI correlated with reversible thallium-201 perfusion defects in the peri-infarction area. A limitation of that study is the absence of a control group with the same clinical characteristics without ST elevation or T-wave normalization.

Mechanism of ST-segment elevation: Chahine et al[8] postulated that WMA that are either permanent in patients with previous AMI or transient due to ischemia in the absence of AMI constitute a common mechanism underlying ST elevation during stress. The results of our study support this hypothesis, because we detected WMA in all patients with ST elevation at peak stress in corresponding myocardial segments in the presence or absence of previous AMI.

Clinical implications: With regard to an ischemic response to dobutamine infusion, ST-segment elevation in patients without previous AMI would help with diagnosis in the presence of suboptimal or equivocal echocardiographic or scintigraphic images, and would identify patients with more severe ischemia. Because ST elevation is not a specific marker of ischemia in patients with previous AMI, it should not be used as a criterion for termination of DST.

Study limitations: The diagnosis of ischemia in this study relied on stress echocardiography and SPECT without performing coronary angiography in all patients. However, these techniques were reported as accurate methods for the diagnosis of coronary artery disease.[16,22–25] Echocardiographic detection of ischemia may be difficult in severely dyssynergic segments. However, echocardiography accurately detects ischemia in the normal peri-infarction region represented in the same electrocardiographic segment. Furthermore, there was a double check on the occurrence of ischemia using 2 imaging techniques; with these 2 techniques, a similar prevalence of ischemia was encountered. The intake of medications may have reduced the prevalence of ischemia at echocardiography and SPECT. However, it is unlikely that ischemia was seen on the electrocardiogram in the absence of transient perfusion or WMA, or both, because perfusion and WMA occur earlier than electrocardiographic changes in the ischemic cascade.[26]

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