Reversal of Rest Asynergy during Exercise in Patients with Coronary Artery Disease

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SUMMARY

The diagnosis of ischemic heart disease by radionuclide ventriculography (RNV) is performed on the basis of an abnormal response of the left ventricular ejection fraction and the occurrence, or aggravation, of regional wall motion abnormality during exercise. However, the abnormal wall motion observed by RNV at rest is improved in some patients with coronary artery disease during exercise. We examined the clinical features of such patients who showed a paradoxical response of regional wall motion. The left ventricle was divided into 4 segments: anteroseptal, apical, inferior and posterolateral. The degree of wall motion of each segment was classified into 5 grades and scored according to a 5 point system: 4=normokinesis, 3=hypokinesis, 2=severe hypokinesis, 1=akinesis and 0=dyskinesis. The wall motion score (WMS) was calculated as the sum of each segment score. If the WMS increased by 2 points or more during exercise, the case was defined as having shown significant improvement of wall motion.

Improvement in WMS was found in 26 (12%) of 209 serial patients who underwent exercise RNV, exercise thallium myocardial scintigraphy and coronary angiography. Clinically, half of these patients had a variant form of angina pectoris. With respect to coronary lesions in the segments with reversible asynergy, 12 patients had 0 vessel disease, 8 had lesions with stenosis of less than 75% and 3 showed an adequate collateral circulation. Redistribution found on the exercise thallium myocardial scintigram at the same sites of improved wall motion was identified in only 1 patient. An analysis of patients with paradoxical improvement of wall motion during exercise suggests the involvement of coronary spasm, an improvement of coronary flow reserve, such as could be produced by regression or recanalization of the main lesions, or establishment of significant collateral circulation.

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EXERCISE tests are often performed to confirm the diagnosis[1] and to evaluate the severity of ischemic heart disease. It is believed that ischemia results from an imbalance between the increase of myocardial oxygen consumption and the coronary flow reserve. Among the various exercise tests, exercise RNV is thought to be useful to identify regional and global left ventricular dysfunction during stress.

For the evaluation of regional contraction, we used RNV to assess regional wall motion noninvasively. The validity of RNV for the diagnosis of wall motion has already been established.[2]-[5] In exercise RNV in patients with coronary artery disease, paradoxical phenomena[6] in which the abnormal wall motion observed at rest is improved during exercise are known to occur. We performed a comparative study of coronary angiography, RNV and thallium myocardial scintigraphy to examine the incidence and mechanisms of reversible asynergy.

Methods

1. Patient characteristics

The subjects were 209 consecutive patients with ischemic heart disease who underwent coronary arteriography, exercise RNV and exercise thallium myocardial scintigraphy. Coronary angiography and RI study were performed within about 3 weeks of each other without any coronary events between examinations.

No patients in this study had significant valvular disease or myocardial disease other than coronary artery lesions. Patients who had previously undergone coronary bypass surgery, percutaneous transluminal coronary recanalization or percutaneous transluminal coronary angioplasty were excluded from this study.

A diagnosis[7] of myocardial infarction was made in patients with typical chest pain, elevation of serum enzymes and typical ECG changes. Myocardial infarction was classified into 2 groups: transmural myocardial infarction and nontransmural myocardial infarction, according to the electrocardiographic findings.

2. Exercise test

Both exercise tests were performed after the patients had had no cardiac medication for at least 24 hours. A 12-lead electrocardiogram was moni-

tored during exercise and blood pressure was obtained by a sphygmomanometer at regular intervals. The exercise was stopped for subjective symptoms, changes of the ST segment, malignant arrhythmias, a fall in blood pressure or a rise in systolic blood pressure to more than 250 mmHg.

a) Exercise RNV

All subjects underwent ECG gated RNV[8] at rest after in vivo red blood cell labeling with 25 mCi of technetium-99 m. Cardiac imaging was accomplished in a modified left anterior oblique projection with a gamma camera equipped with an all-purpose collimator. Data acquisition was performed at 28 frames a beat for 5 min. Camera data were acquired in the frame format of a 64×64 matrix and stored in a Toshiba Nuclear Data Processor 90A System. Supine ergometer multistage exercise was performed at a work load of 25 watts and the work load was increased by 25 watts every 3 min. Cardiac imaging was also performed at each exercise stage for 2.5 min in the same way as it was performed at rest.

The left ventricular ejection fraction was measured by a routine method and the left ventricular image was divided into 4 segments: anteroseptal, apical, inferior and posterolateral. Then, the wall motion of each segment was examined by 2 experienced observers, with no knowledge of the clinical diagnosis, using a phase image and a moving image in a closed loop movie format on the computer display.[4),5),9)] The wall motion was scored according to a 5 point system: 4=normokinesis, 3=hypokinesis, 2=severe hypokinesis, 1=akinesis and 0=dyskinesis. The sum of the scores of each segment was designated the wall motion score (WMS). When the WMS showed an improvement of 2 points or more during exercise, compared with the WMS at rest, reversible asynergy was considered to be present.

b) Thallium myocardial scintigraphy

Upright bicycle ergometer exercise was used for exercise thallium myocardial scintigraphy.[10)] Graded exercise was started at 50 watts and increased by 25 watts every 3 min. Thallium chloride (2.5–3.0 mCi) was injected intravenously 1 min before the end of exercise. Cardiac imaging was performed using single photon emission computed tomography (SPECT: Toshiba GCA 70A) 5 min after injection. SPECT imaging was collected up to 360° into a 64×64 matrix for 8 min. A redistribution image was obtained 3 hours after exercise in the same way. Three-dimensional tomograms of the sagittal long axial, short axial and horizontal long axial images were computed using a GMS-90 data processing system. The left ventricle was divided into 4 segments in the same manner as in the RNV study. The myocardial perfusion of these segments was classified visually by 2 experienced observers who were not given information on regional wall motion,

into normal, low uptake and defect. The presence of redistribution was also evaluated.

3. Coronary angiographic findings

Stenosis of more than 51% of the vessel diameter was considered to be a significant lesion according to the American Heart Association Classification. The angiographic criterion[11] for the presence of coronary collateral vessels was direct visualization of the distal segment of an occluded or stenotic artery for more than 50% of its length. Diagnosis of a variant form of angina pectoris[12] (coronary spasm) was made when an elevation of ST segment was observed during spontaneous attacks on a 12-lead ECG or Holter ECG, or when there was coronary spasm, occurrence of subjective symptoms and displacement of the ST segment on ECG by intravenous ergonovine during coronary arteriography.

4. Judgment of electrocardiographic findings

In the diagnosis of myocardial infarction, the infarction was determined to be transmural if abnormal Q waves were present in the infarction site lead. The abnormal Q waves were 0.04 sec or more in width and 25% of the height of the R wave in depth. Electrocardiographically, the location of the abnormal Q waves of each segment of the left ventricle was sought in the following manner: Q waves of the anteroseptal and apical sides in leads I and V_1–V_4; those of the inferior side in leads II, III and aV_F; and those of the posterolateral side in leads aV_L, V_5–V_6 and/or V_1, the R/S ratio being greater than or equal to 1.

5. Statistical analysis

The values in this paper are all indicated as mean values$\pm$1 standard deviation. A nonpaired Student's t-test was used to determine if there were significant differences between the normal group and the coronary lesion group. P values of less than 0.05 were considered to indicate significant differences.

Results

Although the number of vessels involved or the severity of coronary artery disease is an important determinant of left ventricular dysfunction during exercise, regional improvement of wall motion is the problem in this study. Specifically, the degree of the coronary artery lesions and the thallium myocardial scintigraphic findings at the site of improvement were analyzed carefully. Improvement of WMS was found in 26 (12%) of 209 serial patients. In addition, 21 normal subjects without coronary artery lesion were prepared as age-matched controls.

Table I. Coronary Arteriographic Findings in Segments with Reversible Rest Asynergy

Degree of narrowing (%)	Number of patients	Collateral circulation
0	8	0
-50	4	0
51-75	8	0
76-90	3	0
91-99	3	3

Table II. Location and Severity of Left Ventricular Rest Asynergy

Segment		Wall motion		Hypokinesis
		Dyskinesis akinesis	Severe hypokinesis	
Anteroseptal	(n=19)	1	7	11
Apical	(n=16)	1	5	10
Inferior	(n=3)		2	1
Posterolateral	(n=4)	1	2	1

n=number of cases.

1. Features of the 26 patients and degree of coronary artery disease

Twenty-six of the 209 patients met the clinical criteria for diagnosis of improvement of abnormal wall motion. The clinical pictures of the 26 patients were: angina pectoris in 9 (vasospastic angina in 8), nontransmural myocardial infarction in 9 (vasospastic angina in 4) and transmural myocardial infarction in 8 (vasospasm in 1). With respect to coronary arterial lesions, 0 vessel disease was found in 11 patients, single vessel disease in 7 and multivessel disease in 8.

2. Coronary artery lesions and the degree of severity of wall motion in the segments with reversible asynergy

The coronary artery lesions at the site of improvement in the 26 patients are shown in Table I. Stenosis of 50% or less was observed in 12 patients, stenosis of between 51–75% in 8 patients, 76–90% stenosis in 3 patients and 99% stenosis in 3 patients. Significant collateral circulation was found in all 3 patients with 99% stenosis. Therefore, 23 of 26 patients were considered to have no or a less severe lesion, or to have the possibility of established collateral blood flow.

Segments with reversible asynergy are shown in Table II. Nineteen anteroseptal, 16 apical, 3 inferior and 4 posterolateral segments were detected. The anteroseptal and apical segments accounted for 35 of the 42 segments as shown in Table II. As for the degree of improvement of wall

Table III. Improvement of Rest Asynergy during Exercise

	Rest (n=42)	Exercise (n=42)
Normokinesis		23 + 10 = 33
Hypokinesis	23	10 + 3 = 13
Severe hypokinesis	16	6 + 3 = 9
Akinesis	3	0
Dyskinesis	0	0

n=number of cases.

Table IV. Hemodynamic Measurements during Exercise

		HR (/min)	BP (mmHg)	LVEF (%)	WMS
Patients (n=20)	Rest	63± 9	126±15	59± 8	12.3±1.8
	Exercise	111±19	180±20	68±10	14.8±1.5
Control (n=21)	Rest	65± 9	125±15	62± 8	
	Exercise	115±15	186±26	72± 8	

n=number of cases; WMS=wall motion score; HR=heart rate; BP=blood pressure.

motion, as shown in Table III, hypokinesis improved to normal in 23 segments, severe hypokinesis changed to normal in 10 and to hypokinesis in 6, and akinesis became hypokinesis in 3 segments. In fact, hypokinesis and severe hypokinesis accounted for 39 of the 42 examples of reversible abnormal wall motion.

3. Heart rate and systolic blood pressure during exercise

Work load is an important factor in the development of myocardial ischemia during exercise. In the 26 patients with reversible asynergy, the heart rate increased from 63±9 per minute at rest to 111±19 per minute during exercise, and the systolic blood pressure rose from 126±15 mmHg at rest to 180±20 mmHg during exercise. In the age-matched normal control group, the heart rate increased from 65±9 per minute at rest to 115±15 per minute during exercise, and the systolic blood pressure rose from 125± 15 mmHg at rest to 186±26 mmHg during exercise. There was no significant difference between the 2 groups. So all 26 patients in this study underwent sufficient exercise stress as shown in Table IV.

4. Electrocardiographic findings in 26 patients

Abnormal Q waves were observed in the 12-lead ECG in 9 patients at rest. However, the presence of abnormal Q waves in the segment showing

Table V. ^{201}Thallium Myocardial Images in Segments with Reversible Rest Asynergy

Wall motion at rest	Normal (n=12)	Thallium image		Redistribution (+) (n=1)
		Persistent low uptake (n=24)	Defect (n=5)	
Hypokinesis (n=23)	3	18	2	
Severe hypokinesis (n=10)	8	4	3	1
Akinesis (n=3)	1	2		

n=number of segments.

improvement in abnormal wall motion was noted in only 3 patients. The regional wall motion in the above 3 patients was akinetic. Depression of ST segment during exercise was found in 5 patients. The left ventricular ejection fraction decreased in 1 of the 5, but increased in the other 4.

5. Changes in WMS and response of LVEF during exercise

The WMS of the 26 patients showed a significant increase, from 12.3±1.8 at rest to 14.8±1.5 during exercise. The LVEF also increased significantly from 59±8% at rest to 68±10% during exercise. The response of the LVEF of individual patients was: an increase of 5% or more in 20 patients, an increase of 0–5% in 5 patients and a decrease in only 1 patient. The LVEF in the normal group rose significantly from 62±8% at rest to 72±8% during exercise.

6. Thallium myocardial scintigraphic findings (Table V)

Thallium myocardial scintigrams at the sites of improved abnormal wall motion revealed that there were normal findings in 12 segments, persistent low uptake with no redistribution in 24, persistent low uptake with redistribution in 1 and a permanent defect in 5. These findings showed that the myocardium was probably viable in 37 of the 42 segments, with improved rest asynergy during exercise RNV and that, on the other hand, myocardial ischemia was induced by exercise in only 1 patient.

Discussion

1. Radionuclide ventriculography

Although the validity in the assessment of wall motion is the most important factor in this investigation, the accuracy of regional wall motion analysis using RNV has been established. Karsch et al[2] used qualitative (A), semiquantitative (B) and quantitative (C) analyses of regional wall motion by RNV and compared them with biplane cine ventriculograms.

The sensitivity of method A was 88%, while that for method B was 84%. The specificities of methods A and B were 83% and 100%. The concordance of methods B and C was 92.8%. In the report of Okada et al,[3] the agreement with contrast ventriculography was 88% in normokinetic segments and 79% in asynergy segments. Furthermore, RNV has been used as a routine method for the noninvasive assessment of regional wall motion during exercise in recent investigations.[4),5]

In exercise RNV of patients with coronary artery disease, the response of the LVEF shows varying results,[13] including increase, no change or reduction. The abnormal left ventricular response is usually produced by aggravated regional wall motion due to exercise induced ischemia. However, abnormal wall motion at rest may improve in some cases[6] among a large enough series of patients. In general, the mechanisms of reversible asynergy in patients with ischemic heart disease are considered to be as follows: a) improvement of coronary flow in the segment with resting hypoperfusion after AC-bypass surgery,[14),15] b) reduction of left ventricular preload and/or afterload,[16] c) administration of inotropic agents,[16] d) passive improvement caused by enhanced contraction of the surrounding myocardium, e) abnormal wall motion at rest observed in athletes' hearts.[6] Among these possibilities, the mechanism of reversible asynergy during exercise should be clarified. We investigated retrospectively the types of patients who demonstrated improvement in left ventricular regional wall motion abnormalities during exercise testing.

2. Problem of patient selection

In the assessment of regional wall motion during exercise RNV in 85 patients with coronary artery disease in the report of Kimchi,[6] 48 patients (56%) had no change or further deterioration of wall motion at peak exercise, 15 (18%) showed both improvement and deterioration and 22 (26%) showed only improvement. The incidence of reversible asynergy in the above report was higher than our results.

In patients with multivessel disease, signs of regional ischemia may develop in the most jeopardized segment with a severe stenotic lesion during the early stage of exercise. On the other hand, rest asynergy may improve in a fair number of these patients during the early stage of exercise due to an inotropic effect in a segment with a less significant coronary artery lesion. So the incidence of the segment with reversible asynergy would be increased. For this reason, we decided to regard the improvement of WMS (reflecting total left ventricular wall motion) as an indicator of reversible asynergy. Therefore, the number of patients with reversible asynergy during exercise might be significantly less compared with the report of Kimchi.[6]

3. Features of the coronary artery lesions

It is interesting that coronary spasm was involved in the occurrence of symptoms in 13 of the 26 patients studied here. Furthermore, 17 patients with myocardial infarction had less significant coronary lesions: no significant lesion in 4, 51–75% stenosis in 7, 76–90% stenosis in 3 and 99% stenosis with collaterals in 3.

PTCR has come into widespread use and the coronary findings during acute myocardial infarction have become clear. In the coronary angiograms of 76 patients with acute myocardial infarction,[17] 69 patients showed complete occlusion and 7 patients showed highly stenotic lesions. But in the report of DeWood[18] of 517 patients with acute myocardial infarction, the incidence of coronary arterial occlusion decreased during the first 24 hours after acute myocardial infarction. The incidence of complete occlusion was 86% (320 of 368) within the first 6 hours, fell to 68% (58 of 85) in the 6–12-hour period and to 64% (41 of 64) in the 12–24-hour period. These results suggest that coronary spasm or thrombus formation with subsequent recanalization is seen often in the evolution of acute infarction. Coronary lesions in our 17 patients with myocardial infarction are less significant than the above results. Our 17 patients with myocardial infarction included 5 with coronary spasm, 10 with less significant coronary artery stenosis and 3 with adequate collateral circulation. Furthermore, nontransmural infarction occurred in 9 of the 17 patients.

4. Features of segments with improved asynergy during exercise

The frequency of improved asynergy during exercise was 57 (20%) of 279 segments in the report of Kimchi.[6]

Did these segments with asynergy improve during exercise? Three observations support our conclusion. 1) Hypokinetic or severely hypokinetic segments accounted for 39 of 42 segments. 2) Abnormal Q waves were detected in only 3 segments. 3) Persistent thallium defect was observed in only 5 of 42 segments. Even the thallium-persistent defect does not always represent a myocardial scar. In the report of Liu et al,[19] regions of persistent defect on exercise thallium scan reverted to normal after PTCA in 75% of cases.

When coronary angiographic findings, regional wall motion abnormalities and myocardial scintigraphic findings are considered together, improved segments with asynergy are considered to be viable. Previous reports indicate that reversible wall motion is described after surgery,[14] by postextrasystolic potentiation,[20] response to nitroglycerin[16] and postexercise radionuclide ventriculography.[21] They conclude that these segments are probably viable.

In our patients, improvement is considered to occur when abnormal wall motion is caused by coronary artery spasm without significant lesion, or in patients exhibiting regression, or recanalization of coronary lesions or the establishment of collateral circulation. Furthermore, the mechanism of reversible asynergy during exercise may be readily conjectured to be that contraction reserve is responsive to inotropic action in the regional myocardium, and that even during exercise contractility is improved when myocardial ischemia is not induced. It is possible that coronary spasm may cause such a phenomenon clinically, and that early recanalization of the coronary artery or the formation of collateral circulation may promote this phenomenon.

5. Problems in this study

The problems are that wall motion and thallium myocardial scintigrams were all evaluated subjectively. Regional ejection fraction can be used for the objective evaluation of wall motion, and circumferential profile curves or washout rates can be used to evaluate thallium myocardial scintigrams. However, there were no differences in our experience and reporting[22] between visual assessment and quantitative analysis.

Secondly, tomograms could be evaluated in thallium myocardial scintigraphy, but only the left anterior oblique planar images were evaluated in RNV. Therefore, improvements in the anteroseptal and apical regions, which are easily assessable, might be relatively increased, while improvements in the inferior and posterolateral regions might be relatively reduced.

6. Clinical usefulness of this study

At present, aggressive myocardial salvage by PTCR or with a tissue plasminogen activator[23] is performed for acute myocardial infarction. Invasive studies are used to determine whether these treatments are effective in the acute phase. Noninvasive evaluations include determination of the blood flow distribution and the remaining viable myocardium using thallium myocardial scintigraphy, and observation of improvements in regional wall motion abnormalities by RNV and echocardiography. In this study the presence of a sufficient coronary reserve can be presumed, in addition to the presence of a significant portion of viable left ventricular myocardium in patients in whom improvements of abnormal wall motion were observed on exercise RNV.

References

1. Becker LC: Diagnosis of coronary artery disease with exercise radionuclide imaging: State of the art. Am J Cardiol **45**: 1301, 1980
2. Karsch KR, Schicha H, Rentrop P, Blanke H, Kreuzer H, Emirch D: Qualitative und

Semiquantitative Analyse der lokalen Wandfunktion mittels der Gated-blood-pool-Methode im Vergleich zur Cineventrikulographie. Z Kardiol **69**: 334, 1980

3. Okada RD, Pohost GM, Nichols AB, Mckousick KA, Strauss HW, Boucher CA, Block PC, Rosenthal SV, Cinsmore RE: Left ventricular regional wall motion assessment by multigated and end-diastolic, end-systolic gated radionuclide left ventriculography. Am J Cardiol **45**: 1211, 1980
4. Dilsizian V, Bonow RO, Cannon RO III, Tracy CM, Vitale DF, McIntosh CL, Clark RE, Bacharach SL, Green MV: The effects of coronary artery bypass grafting on left ventricular systolic function at rest: Evidence for preoperative subclinical myocardial ischemia. Am J Cardiol **61**: 1248, 1988
5. Currie PJ, Kelly MJ, Harper RW, Federmen J, Kalff V, Anderson ST, Pitt A: Incremental value of clinical assessment, supine exercise electrocardiography, and biplane exercise radionuclide ventriculography in the prediction of coronary artery disease in men with chest pain. Am J Cardiol **52**: 927, 1983
6. Kimchi A, Rozanski A, Fletcher C, Maddahi J, Swan HJC, Berman DE: Reversal of rest myocardial asynergy during exercise: A radionuclide scintigraphic study. J Am Coll Cardiol **6**: 1004, 1985
7. Beta blocker heart attack study protocol. National Institute of Health Publication **81**: 2209, 1980
8. Schneider RM, Weintraub WS, Klein LW, Seelaus PA, Katz RI, Agarwal JB, Helfant RH: Multistage analysis of exercise radionuclide angiography in coronary artery disease. Am J Cardiol **58**: 36, 1986
9. Konishi T, Ichikawa T, Yamamuro M, Koyama T, Futagami Y, Nakano T, Takezawa H: Incidence and clinical course of right ventricular infarction: Assessment with radionuclide ventriculography. Angiology **38**: 741, 1987
10. Tamaki S, Kambara H, Kadota K, Suzuki Y, Nohara R, Kawai C, Tamaki N, Torizuka K: Improved detection of myocardial infarction by emission computed tomography with thallium-201: Relation to infarct size. Br Heart J **52**: 621, 1984
11. Elayda MA, Mathur VS, Hall RJ, Masumi GA, Garcia E, Castro CM: Collateral circulation in coronary artery disease. Br Heart J **55**: 58, 1985
12. Raiser AE, Chahine RA, Ishimori I, Verani M, Zacca N, Jamal N, Miller R, Luchi R: Provocation of coronary artery spasm by cold pressor test: Hemodynamic, arteriographic and quantitative angiographic observations. Circulation **62**: 925, 1980
13. Hammond HK, Kelly TL, Froelicher VF: Noninvasive testing in the evaluation of myocardial ischemia: Agreement among tests. J Am Coll Cardiol **5**: 59, 1985
14. Chatterjee K, Swan HJC, Parmley WW, Sustain H, Maraus HS, Matloff J: Influence of direct myocardial revascularization on left ventricular asynergy and function in patients with coronary heart disease. Circulation **47**: 276, 1973
15. Kolibash AL, Goodenow JS, Bush CA, Tetalman MR, Lewis RP: Improvement of myocardial perfusion and left ventricular function after coronary artery bypass grafting in patients with unstable angina. Circulation **59**: 66, 1979
16. Ramanathan KB, Bodenheimer MM, Banka VS, Helfant RH: Severity of contraction abnormalities after acute myocardial infarction in man: Response to nitroglycerin. Circulation **60**: 1230, 1979
17. Timmis GC, Gangadharan V, Hauser AM, Ramos RG, Westveer GC, Gordon S: Intracoronary streptokinase in clinical practice. Am Heart J **104**: 925, 1982
18. DeWood MA, Spores J, Hensley GR, Simpson CS, Eugster GS, Sutherland KI, Grunwald RP, Shields P: Coronary arteriographic findings in acute transmural myocardial infarction. Circulation **68** (suppl I): I-39, 1983
19. Liu P, Kiess MC, Okada RD, Block PC, Strauss HW, Pohost GM, Boucher CA: The persistent defect on exercise thallium imaging and its fate after myocardial revascularization: Does it represent scar or ischemia? Am Heart J **110**: 996, 1985

20. Popio KA, Gorlin R, Bechtel D, Levine JA: Postextrasystolic potentiation as a predictor of potential myocardial viability. Am J Cardiol **39**: 944, 1977
21. Rozanski A, Berman D, Gray R, Diamond G, Raymond M, Prause J, Maddahi J, Swan HJC, Matloff J: Preoperative prediction of reversible myocardial asynergy by postexercise radionuclide ventriculography. New Engl J Med **307**: 212, 1983
22. Doherty PW, Lipton MJ, Berninger WH, Skiolderand CG, Carlsson E, Redington RW: Detection and quantification of myocardial infarction in vivo using transmission computed tomography. Circulation **63**: 579, 1981
23. Topol EJ, O'Neill WW, Langburd AB, Walton JA Jr, Bourdillon PDV, Bates ER, Grines CL, Schork AM, Kline E, Pitt B: A randomized, placebo-controlled trial of intravenous recombinant tissue-type plasminogen activator and emergency coronary angioplasty in patients with acute myocardial infarction. Circulation **75**: 420, 1987