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COOPERATIVE STUDIES

Effects of Gender and Race on Prognosis After Myocardial Infarction: Adverse Prognosis for Women, Particularly Black Women

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Controversy has arisen concerning whether gender influences the prognosis after myocardial infarction. Although some studies have shown there to be no difference between the sexes, most have indicated a worse prognosis for women, attributing this to differences in baseline characteristics. It has been further suggested that black women have a particularly poor prognosis after infarction. To determine the contribution of gender and race to the course of infarction, 816 patients with confirmed myocardial infarction who were enrolled in the Multicenter Investigation of the Limitation of Infarct Size (MILIS) were analyzed. Of those patients, 226 were women and 590 were men, 142 were black and 674 were white.

The cumulative mortality rate at 48 months was 36% for women versus 21% for men ($p < 0.001$, mean follow-up 32 months). The cumulative mortality rate by race was 34% for blacks versus 24% for whites ($p < 0.005$). Both women and blacks exhibited more baseline characteristics predictive of mortality than did their male or white counterparts. It was possible to account for the greater mortality rate of blacks by identifiable baseline variables; however, even after adjustment, the mortality rate for women remained significantly higher ($p < 0.002$). The poorer prognosis for women was influenced by a particularly high mortality rate among black women (48%); the mortality rate for white women was 32%, for black men 23% and for white men 21%. The mortality for black women was significantly greater than that of the other subgroups. Thus, findings in the MILIS population indicate that the prognosis after myocardial infarction is worse for women, particularly black women.

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The effect of gender on the prognosis after myocardial infarction is unclear. Although several studies (1,2) have found either no difference in postinfarction prognosis between the sexes or a worse prognosis for men (3), most studies (4,5) have shown a higher mortality rate in women than in men. In the Framingham Study (4), the 12 month mortality rate after a recognized myocardial infarction was 34% for women compared with 19% for men. It has been suggested (6) that the worse prognosis observed in women is due to an increased prevalence in women of baseline predictors of mortality. Women with infarction are typically older and have a greater prevalence of hypertension and diabetes mellitus than do men with infarction (7–9).

Although no consistent gender differences in complications after infarction have been shown, individual studies (10) have suggested that women have a higher frequency of reinfarction, a selectively worse prognosis after inferior infarction (6) and a greater prevalence of cardiac rupture after infarction (11). Racial differences in the expression of coronary heart disease have also been described. Although black women seem to have a higher prevalence of, and

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mortality rate from, coronary heart disease than do white women, there appear to be no such differences between black and white men (12).

The data base from the Multicenter Investigation of the Limitation of Infarct Size (MILIS) provides a unique opportunity to investigate the influence of both gender and race on the course of a large number of patients with acute infarction. Although the primary purpose of the study was to assess the efficacy of hyaluronidase and propranolol therapy on acute myocardial infarction (13), extensive data, including baseline tests of left ventricular function and long-term mortality status, were collected on all patients.

The specific objectives of our study were to determine: 1) Whether gender or race influences survival after myocardial infarction; and 2) whether any observed differences in survival could be accounted for by differences in baseline characteristics between the genders and races.

Methods

The MILIS patient population. The structure of MILIS and a list of participating institutions are presented in the Appendix. Patients were eligible for enrollment in MILIS if they were <76 years old, had ≥30 minutes of pain typical of myocardial ischemia, had been evaluated <18 hours after the onset of chest pain and demonstrated electrocardiographic changes suggestive of acute ischemia or evolving infarction (new Q waves greater than 30 ms in width and ≥0.2 mV in depth, or ST segment elevation or depression, or both, of ≥0.1 mV in at least two related leads) or left bundle branch block or idioventricular rhythm. Exclusion criteria, methods of informed consent, guidelines for standard care and details of the administration of hyaluronidase or propranolol have been reported previously (13–15).

From the 9,450 patients screened, 985 were randomized. The study population in this report comprised 816 patients who were of the white or black race and in whom an acute infarction was confirmed by the creatine kinase (CK) core laboratory. Of these 816 patients, 226 were women and 590 were men, 142 were black and 674 were white. When grouped by race and gender, there were 63 black women, 79 black men, 163 white women and 511 white men.

Data collection. After enrollment but before randomization, baseline measurements including a 12 lead electrocardiogram and a rest radionuclide ventriculogram were obtained. The left ventricular ejection fraction was calculated from the ventriculogram by a semiautomated count method from the left anterior oblique view after background correction (16). Radionuclide ventriculography was repeated on the 10th day. Serial blood sample collections for measurement of CK and the MB CK isoenzyme were initiated before randomization and were continued for the length of the hospital stay, as previously reported. Throughout the hospitalization, a MILIS data coordinator recorded details of the patients' history and physical examination, a summary of daily clinical events, vital signs and special procedures and results of routine laboratory tests.

Follow-up examinations were conducted 3 and 6 months after randomization. The vital status of all patients was ascertained at 6 month intervals by a questionnaire administered by telephone. Core laboratories unaware of treatment assignment and mortality outcome analyzed total CK activity, MB CK activity, electrocardiographic localization of infarction and radionuclide ventriculography.

Table 1. Preinfarction Characteristics of 816 Patients

	Gender			Race		
	Women (n = 226)		Men (n = 590)	Black (n = 142)		White (n = 674)
Age (yr)	60	*	56	56		57
Hypertension (%)	63	*	50	70	*	50
Diabetes mellitus (%)	28	*	15	27	*	17
Cigarette smoking (pack·years)	34	*	43	33	*	42
Cigarette smoking within 6 months of MI (%)	54		54	58		53
Family history of MI age (<60 yr) (%)	40	*	31	27		34
History of congestive heart failure (%)	12	*	7	14	*	7
History of hyperlipidemia (%)	10		10	9		11
History of angina >3 weeks before MI (%)	36		36	38		36
Previous MI (%)	19		25	25		23
Obesity (%)†	52		50	51		50

*$p < 0.05$. †An "obesity index" was calculated as weight in kilograms divided by height squared in meters. A cutoff of 26 kg/m^2 was taken as the upper 25th percentile for the population. MI = myocardial infarction.

Table 2. Hospital Course in 816 Patients

	Gender			Race		
	Women (n = 226)		Men (n = 590)	Black (n = 142)		White (n = 674)
LVEF						
Admission (%)	49	*	45	50	*	46
10 Day (%)	51	*	47	50		48
Q wave MI (%)	54	*	65	53	*	64
Anterior MI (%)	58		56	59		57
Infarct size index (MB CK gEq/m^2)	16		17	19		17
Atrioventricular block (%)	28	*	19	19		22
Intraventricular conduction delay (%)	21		20	20		20
Infarct extension (%)	11		9	12		9
In-hospital death (%)	13	*	7	10		9

*$p < 0.05$. LVEF = left ventricular ejection fraction; MI = myocardial infarction.

Statistical methods. Within the gender and race groups, chi-square and *t* tests were used to determine the significance of the differences between proportions and means, respectively. When multiple comparisons were made among the four gender-race groups for a particular response, simultaneous inference methods were used to control the type I error rate. Duncan's test (17) was applied when the response was of a continuous nature and a technique developed by Goodman (18) was used for categorical responses.

The Cox proportional hazards regression model (19) was used to assess the differences in mortality to 48 months for both gender and race while adjusting for the effects of a "risk score" composed of baseline variables that showed imbalances for sex or race or were found from the MILIS population to predict mortality. The variables included in the risk score were age, history of hypertension, diabetes, smoking, history of congestive heart failure, family history of myocardial infarction before age 60 years, level of education, Karnofsky score, history of cardiac arrhythmias, use of diuretics in the 3 weeks before admission, transmural ischemia/infarction, the presence of ST elevation at randomization, hypotension on admission and left ventricular ejection fraction on admission. Unadjusted plots of the estimated cumulative mortality were produced by the Kaplan-Meier method (20). Adjusted mortality curves were based on the Kalbfleisch and Prentice discrete hazard model (21). All probability (p) values were two-tailed and a p value of <0.05 was considered significant.

Results

Influence of Gender on Myocardial Infarction

Preinfarction characteristics (Table 1). The mean age of the women was greater than that of the men and the women had a greater prevalence of hypertension, diabetes mellitus, family history of infarction at less than 60 years of age, and history of congestive heart failure before the index infarction. Men had a more extensive history of cigarette smoking than did women; however, there was no

Table 3. Follow-up

	Gender			Race		
	Women (n = 183)		Men (n = 531)	Black (n = 122)		White (n = 592)
At 6 months†						
Angina pectoris (%)	58	*	44	49		48
Congestive heart failure (%)	32	*	18	33	*	19
Recurrent MI (%)	19	*	12	15		14
At end of follow-up‡						
Mortality rate for hospital survivors (%)	27	*	15	27	*	17
Total cumulative mortality (%)	36	*	21	34	*	24

*$p < 0.05$; †significance of differences between proportions determined by chi-square test; ‡significance of differences for 48 month mortality based on life table analysis; mean duration of follow up 32 months. MI = myocardial infarction.

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difference in the percent of men and women smoking within the 6 months before infarction. The incidence of a prior infarction, of angina more than 3 weeks before infarction or of obesity was not significantly different between men and women.

Hospital course (Table 2). The left ventricular ejection fraction on admission was greater for women than for men and remained greater for women at 10 days. Although there was no difference in infarct size index between men and women, the infarct was more frequently associated with the development of Q waves among men than among women. There was no significant difference in location of infarction, prevalence of intraventricular conduction delay or infarct extension, although women had a greater prevalence of atrioventricular conduction disturbance. There was no gender difference in heart rate or blood pressure on admission. The mortality rate during hospitalization was significantly greater for women than for men.

Follow-up (Table 3). At the 6 month follow-up visit, women had a higher incidence of angina pectoris, congestive heart failure and reinfarction than did men. The frequency of cardiac rupture (including ventricular septal defect) was not significantly different between women (2.2%) and men (1.7%), but the total number of cardiac ruptures was too small to exclude a difference. There were no significant differences in sex ratio for mortality between patients with an anterior or an inferior infarction. The total mortality rate after anterior infarction was 42% for women and 28% for men ($p = 0.003$) and after inferior infarction it was 31% for women and 16% for men ($p = 0.001$). The mortality rate among hospital survivors was higher for women than for men during follow-up (27 versus 15%, $p < 0.001$). The 48 month cumulative mortality rate was 36% for women and 21% for men ($p < 0.001$) (Fig. 1A). After adjustment for the risk score, the mortality probability remained significantly higher for women ($p < 0.002$) (Fig. 1B). When the data were analyzed specifically for death because of cardiac causes, the mortality rate among women remained significantly higher than that for men (31 versus 18%, $p < 0.001$).

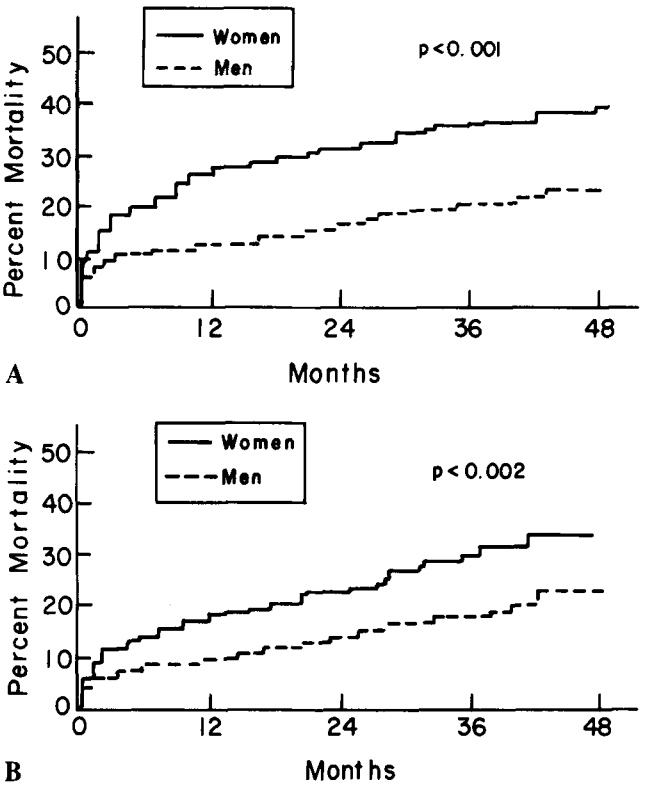

Figure 1. Mortality curves for women and men after myocardial infarction, unadjusted (**A**) and adjusted (**B**) by the risk score.

Influence of Race on Myocardial Infarction

Preinfarction characteristics (Table 1). Before infarction, blacks had a greater prevalence of hypertension, diabetes mellitus and congestive heart failure than did whites. Whites had a more extensive history of cigarette smoking.

Hospital course (Table 2). Blacks had a lower incidence of Q wave infarction and a higher left ventricular ejection fraction on admission than did whites. In-hospital mortality was not significantly different between blacks and whites.

Follow-up (Table 3). Among hospital survivors of infarction, the mortality rate was greater for blacks than for whites (27 versus 17%, $p < 0.002$). Blacks exhibited a significantly higher cumulative mortality rate than did whites (34 versus 24%, $p < 0.005$) (Fig. 2A). However, when adjusted for baseline risk variables, the difference in mortality probability between blacks and whites was no longer significant (Fig. 2B). Blacks had a higher cardiac mortality rate than did whites (27 versus 21%, $p < 0.04$).

Combined Influence of Gender and Race on Myocardial Infarction

The ratio of women to men in this study was markedly higher for blacks than for whites (0.8:1 versus 0.3:1, $p < 0.001$); that is, 44% of the black patients and 24% of the white patients were women.

Preinfarction characteristics (Table 4). Differences between men and women similar to those observed in the total population were present for both the black and white races (Table 1). Statistical significance, however, was not always reached because of both the smaller numbers in each subgroup and the use of more stringent criteria for significance (Duncan's test and Goodman's technique to control against a type I error). Both black men and black women had a significantly greater prevalence of hypertension before infarction than did their white counterparts. A similar though not significant trend was present for diabetes mellitus.

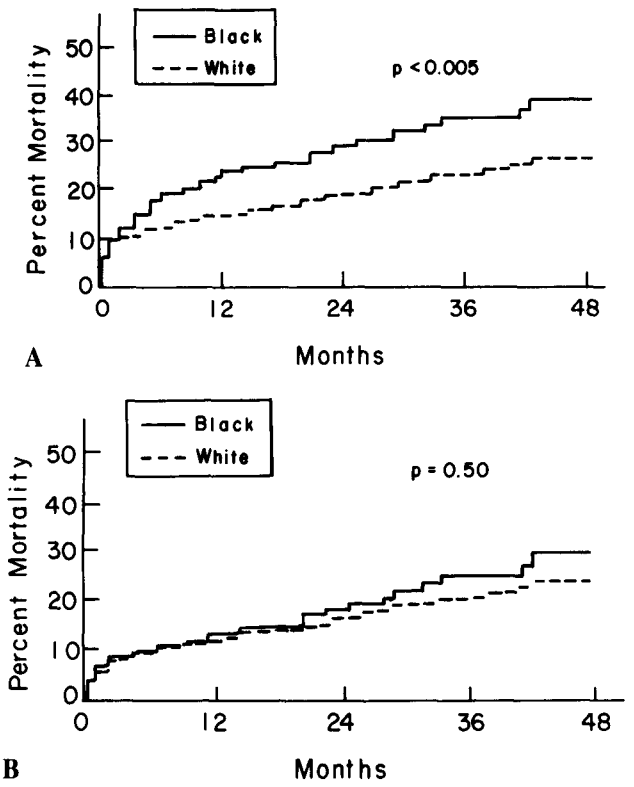

Figure 2. Mortality curves by race after myocardial infarction, unadjusted (**A**) and adjusted (**B**) by the risk score.

Hospital course (Table 5). White women had a lower incidence of Q wave infarction than did white men. The left ventricular ejection fraction was greater in both white and black women than in their male counterparts, although this difference was statistically significant only in black patients. Black women had a significantly greater infarct size index than did white women. Hospital course was not different for black and white men except that white men had more Q wave infarcts than did black men. The in-hospital mortality rate was markedly higher for black women than for black men.

Follow-up (Table 6). After infarction, both black and white women had a greater incidence of angina and heart failure than did their male counterparts, although these differences were statistically significant only in black women. The overall mortality, as well as the mortality among hospital survivors, was significantly greater for black and white women than for black and white men, respectively. Although black and white men had a similar follow-up course, black women had a greater mortality than did white women. The mortality curves by gender and race are displayed as both unadjusted (Fig. 3A) and adjusted by risk score (Fig. 3B); by both methods, the mortality of black women by life table analysis was significantly greater than that of the other subgroups. The significantly greater mortality among black women persisted when only death from cardiac causes was considered.

Causes of Death (Table 7)

There were no significant differences in the mortality profiles of the various subgroups. Compared with whites, blacks showed a trend toward a greater incidence of death from a cardiovascular but noncoronary cause, which includes cerebrovascular-related death, and from noncardiovascular causes.

Table 4. Preinfarction Characteristics of 816 Patients by Gender and Race

	Gender Within Race						Race Within Gender					
	BW (n = 63)	vs.	BM (n = 79)	WW (n = 163)	vs.	WM (n = 511)	BW (n = 63)	vs.	WW (n = 163)	BM (n = 79)	vs.	WM (n = 511)
Age (yr)	58	*	55	61	*	56	58	*	61	55		56
Hypertension (%)	78		65	57		48	78	*	57	65	*	48
Diabetes mellitus (%)	37		19	25	*	14	37		25	19		14
Cigarette smoking (pack·years)	27	*	37	36	*	44	27	*	36	37		44
Cigarette smoking within 6 months of MI (%)	54		61	53		53	54		53	61		53
Family history of MI age <60 yr (%)	32		23	43	*	32	32		43	23		32
History of congestive heart failure (%)	18		11	10		6	18		10	11		6
History of hyperlipidemia (%)	11		8	10		11	11		10	8		11
History of angina >3 weeks before MI (%)	41		35	34		36	41		34	35		36
Previous MI (%)	24		27	18		25	24		18	27		25
Obesity (%)	62		42	49		51	62		49	42		51

*p < 0.05. BM = black men; BW = black women; MI = myocardial infarction; WM = white men; WW = white women.

Table 5. Hospital Course in 816 Patients by Gender and Race

	Gender Within Race						Race Within Gender					
	BW (n = 63)	vs.	BM (n = 79)	WW (n = 163)	vs.	WM (n = 511)	BW (n = 63)	vs.	WW (n = 163)	BM (n = 79)	vs.	WM (n = 511)
LVEF												
Admission (%)	53	*	47	48		45	53	*	48	47		45
10 Day (%)	52	*	48	51		47	52		51	48		47
Q wave MI (%)	56		50	53	*	67	56		53	50	*	67
Anterior MI (%)	62		56	57		57	62		57	56		57
Infarct size index (MB CK gEq/m^2)	21		18	15		17	21	*	15	18		17
Atrioventricular block (%)	21		18	30	*	20	21		30	18		20
Intraventricular conduction delay (%)	18		22	22		20	18		22	22		20
Infarct extension (%)	18		8	9		9	18		9	8		9
In-hospital death (%)	18	*	4	11		8	18		11	4		8

*$p < 0.05$. Abbreviations as in Tables 1, 2 and 4.

Discussion

Although coronary heart disease is the most common cause of death in women, its greater prevalence in men has tended to lead to an underestimate of the extent of the problem in women.

Influence of gender on prognosis. In the MILIS Study, women had a worse prognosis after myocardial infarction than did men. This difference was significant for both the hospital and follow-up periods. As has been reported in other studies (2,6,8), the women were older and had a greater prevalence of hypertension, diabetes mellitus and history of congestive heart failure before infarction. However, adjusting for these and other gender differences and baseline predictors of mortality failed to explain the difference in prognosis. This may be due to a number of reasons. First, there may be a differential effect of a particular risk factor on women. For example, when associated with coronary heart disease, diabetes mellitus has been shown to convey a poorer prognosis for women than for men (22,23). Second, there may be other known factors predictive of mortality that differ between the sexes and that were not evaluated in the Cox regression model. Third, there may be unidentified gender-related differences in the response to myocardial ischemia and infarction.

Gender difference in left ventricular ejection fraction. Despite having a worse prognosis, women had a higher left ventricular ejection fraction than did men, both on admission and at 10 days after infarction. A gender difference in ejection fraction is not present in normal individuals at rest (24). However, in one angiographic study (25) of subjects with chest pain but normal coronary arteries, the women had a

Table 6. Follow-up in 714 Patients

	Gender Within Race						Race Within Gender					
	BW (n = 48)	vs.	BM (n = 74)	WW (n = 135)	vs.	WM (n = 457)	BW (n = 48)	vs.	WW (n = 135)	BM (n = 74)	vs.	WM (n = 457)
At 6 months†												
Angina pectoris (%)	69	*	36	55		46	69		55	36		46
Congestive heart failure (%)	48	*	22	26		17	48	*	26	22		17
Recurrent MI (%)	22		11	18		12	22		18	11		12
At end of follow-up‡												
Mortality for hospital survivors (%)	37	*	20	23	*	14	37	*	23	20		14
Total cumulative mortality (%)	48	*	23	32	*	21	48	*	32	23		21

*$p < 0.05$; †significance of difference between proportions determined by chi-square test with Goodman's correction for multiple tests; ‡significance of differences in 48 month mortality rate based on life table analysis; mean duration of follow up 32 months. Abbreviations as in Table 5.

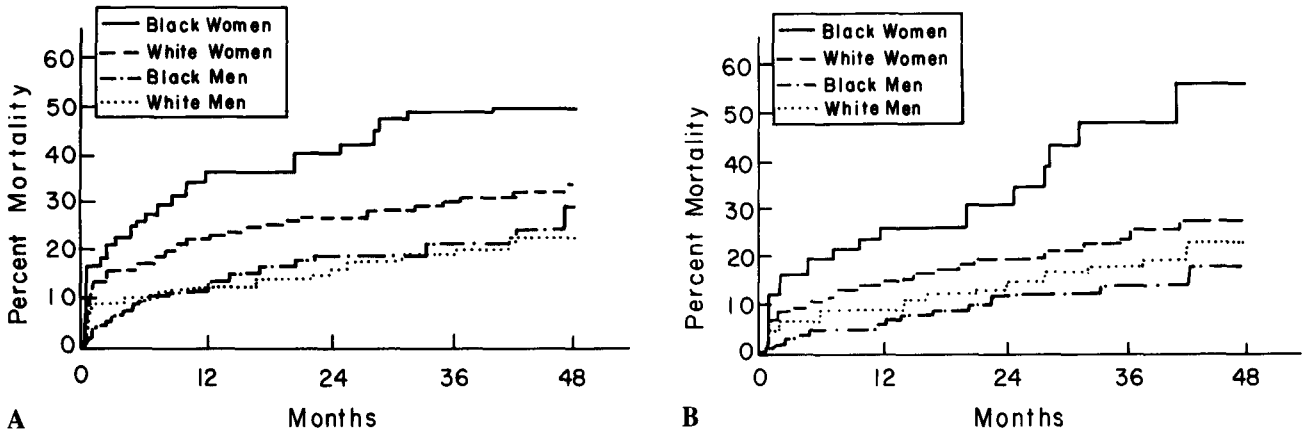

Figure 3. Mortality curves by gender and race after myocardial infarction. **A,** Unadjusted by the risk score. When considered separately as pairs within gender and race, the mortality curves were significantly different for black women and black men ($p < 0.001$); white women and white men ($p < 0.004$); and black women and white women ($p < 0.02$). **B,** After adjustment by the risk score. When considered separately as pairs within gender and race, the mortality curves were significantly different for black women and black men ($p < 0.001$); and black women and white women ($p < 0.01$).

higher left ventricular ejection fraction than did the men. Women in that study also had a higher heart rate than did men, suggesting that the women exhibited a hyperdynamic state causing both tachycardia and vigorous systolic function. However, in our study there was no gender difference in heart rate at presentation. A study (26) of patients undergoing coronary artery bypass grafting reported that women have a higher preoperative left ventricular ejection fraction than do men, although this may be due to the less extensive coronary artery disease and a lesser degree of previous infarction in the women undergoing surgery. Hakki and Iskandrian (27) did not find a difference in ejection fraction between men and women with a comparable extent of coronary artery disease. Although left ventricular systolic function may have been better in women than in men, the left ventricular diastolic function in women may have been more impaired. Heart failure due to diastolic stiffness in the presence of preserved systolic function has been well described in association with hypertension (28,29) and may also be of significance in diabetes mellitus (30). Women with infarction had a greater prevalence of both hypertension and diabetes mellitus than did men. A greater degree of diastolic dysfunction in women, if present, may contribute to the higher mortality rate observed among women despite preserved systolic function.

Women had a higher incidence of initial non-Q wave infarction in our study, as has been reported in other studies (31,32). This finding would contribute to a better preserved left ventricular ejection fraction among women. In addition, it may explain in part the prevalence of reinfarction among women within the first 6 months of follow-up. Marmor et al. (10) reported that early recurrent infarction correlated significantly with female sex in addition to non-Q wave infarction. Cardiac rupture has also been proposed to occur more commonly in women (11). Although this complication

Table 7. Causes of Death in 122 Patients*

	Gender		Race		Gender and Race			
	Women (n = 83)	Men (n = 139)	Black (n = 50)	White (n = 172)	BW (n = 30)	WW (n = 53)	BM (n = 20)	WM (n = 119)
Arrhythmia (%)	17	23	16	22	17	17	15	24
Coronary heart disease other than primary arrhythmia (%)	64	59	54	63	60	65	45	61
Cardiovascular other than coronary heart disease (%)	7	6	14	5	10	6	20	4
Noncardiovascular (%)	7	6	10	5	10	6	10	5
Unknown (%)	5	6	6	5	3	6	10	6

*Refers to the profile of cause of death within each group. There were no significant differences ($p < 0.05$ by chi-square test) detected within the groups. Abbreviations as in Table 5.

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did not occur more frequently in women in our study, there were too few cases of cardiac rupture to clarify this point. It has been suggested (6,33) that women with inferior infarction have an excess mortality over men, (that is, above that seen with anterior infarction), owing to a greater prevalence of right coronary artery dominance. In our study, women with inferior infarction did not have such an excess mortality. The cause of death was not significantly different between women and men. It has been found (26) that women have a higher complication rate after coronary artery bypass surgery, due in part to the smaller size of their coronary arteries. The numbers were too small in our study to adequately address this question.

Influence of race on prognosis. Although coronary heart disease is the leading cause of death among blacks, there is a lack of accurate information concerning its epidemiology in the United States black population (12). We found that blacks had a worse prognosis than did whites after infarction. However, blacks also had a greater prevalence of prior infarction, hypertension, diabetes mellitus and congestive heart failure than did whites. In contrast to our findings with respect to gender, when mortality rate was adjusted for baseline differences, there was no significant difference between the races. Analysis of the cause of death by race showed interesting trends, although the numbers were too small to achieve significance. Blacks showed a trend toward a greater proportion of deaths due to both noncoronary cardiovascular disease, such as cerebrovascular disease, and noncardiovascular-related cause. Although the former trend may reflect the greater prevalence of hypertension and diabetes seen in blacks, both trends may be a manifestation of a more generalized increased mortality among blacks that is not confined to cardiovascular disease (34). The ratio of the number of women to men in the study was significantly higher among blacks. This phenomenon, which has been noted previously (35,36), has not been adequately explained but may reflect selection bias resulting from factors such as delay among black men in seeking medical attention. The concept that factors other than left ventricular systolic function are responsible for subsequent mortality is underscored by the observation that black women had the lowest survival rate of the four groups despite having the highest ejection fraction. Although blacks had a worse prognosis overall than did whites, black men showed no difference in prognosis from white men. This supports the hypothesis that the higher mortality rate in blacks after infarction may not be due to intrinsic racial differences (37).

Clinical implications. Women, particularly black women, have a significantly worse prognosis after myocardial infarction than do men. There is a need for further investigation into the possible mechanisms for the gender and race differences described. In addition, physicians must be conscious of the need for close attention to risk factor modification and clinical management of women, particularly black women, with coronary artery disease.

We are indebted to Kathleen Carney for assistance in the preparation of this manuscript.

Appendix
Multicenter Investigation of the Limitation of Infarct Size (MILIS) Study Personnel

Clinical Centers

Barnes Hospital, Washington University School of Medicine, St. Louis, MO. Allan S. Jaffe, MD, Principal Investigator; Robert Roberts, MD, Principal Investigator; Edward Geltman, MD, Co-Investigator; Dan Biello, MD, Nuclear Medicine Coordinator; Rosanne Wettach, RN, MNP, Research Nurse Coordinator; Ava Ysaguire, Susan Payne and Linda Wilson, Data Coordinators.

Massachusetts General Hospital, Boston, MA. Herman K. Gold, MD, Principal Investigator; Robert C. Leinbach, MD, Principal Investigator; Tsunehiro Yasuda, MD; Wendy Werner, RN and Mary McHugh, RN, Research Nurse Coordinators; Harry Garabedian, Data Coordinator.

Medical Center Hospital of Vermont, University of Vermont College of Medicine, Burlington, VT. Daniel S. Raabe, Jr., MD, Principal Investigator; Walter Gundel, MD; Marian Dornell, RN, Maureen Hawley, RN, Patricia Beecher, RN, Kathleen Cornell, RN, and Karen Helminger, RN, Research Nurse Coordinators; Raina Maynard, Data Coordinator.

Harvard Medical School/Brigham and Women's Hospital, Boston, MA. Eugene Braunwald, MD, Principal Investigator; Peter H. Stone, MD, Joseph S. Alpert, MD and Robert Rude, MD, Clinical Unit Directors; Nancy E. Taplin, RN, Kathryn Shea, RN, Debbie Shiner, RN, Research Nurse Coordinators.

Parkland Memorial Hospital, University of Texas Health Science Center at Dallas, TX. James T. Willerson, MD, Principal Investigator; Robert E. Rude, MD, Clinical Unit Director; Charles Croft, MD; Robert Dillon, MD; Kevin Wheelan, MD; Christopher Wolfe, MD; Barbara Moses, RN, Sandra Cochran, RN, Marvin Akers, RN, Joan Reinert Corey, RN, Vicki Gillespie, RN and Barbara Fitzpatrick, RN, Research Nurse Coordinators; Kris Kraft, Unit Clerk.

Creatine Kinase Core Laboratory

Washington University School of Medicine, St. Louis, MO. Burton E. Sobel, MD, Principal Investigator; Robert Roberts, MD, Principal Investigator; Allan Jaffe, MD; Cynthia Ritter, Laboratory Coordinator; Steven Mumm, Laboratory Technician.

Cardiovascular Pathology Core Laboratory

Duke University Medical Center, Durham, NC. Donald B. Hackel, MD, Principal Investigator; Raymond E. Ideker, MD, PhD; Keith A. Reimer, MD, PhD; Eileen Mikat, PhD.

Technetium-99m Pyrophosphate Myocardial Scintigram Core Laboratory

University of Texas Health Science Center at Dallas, Dallas, TX. James T. Willerson, MD, Principal Investigator; Samuel E.

Lewis, MD, Laboratory Director; Robert W. Parkey, MD, Laboratory Co-Director; Irma Dobbins, Laboratory Coordinator.

Holter Recording Core Laboratory

Washington University School of Medicine, St. Louis, MO. Lewis J. Thomas, Jr., MD, Principal Investigator; Robert Roberts, MD, Co-Principal Investigator; Kenneth W. Clark, Laboratory Director; Kathleen Madden, Laboratory Coordinator; J. Phillip Miller, Biostatistician.

Radionuclide Ventriculogram Core Laboratory

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U.S. Public Health Service Supply Service Center

Perry Point, MA. James Grigdesby; Salvatore Grasbia; Sidney Hamet; E. C. Brennan.

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