Published in final edited form as:
Am Heart J. 2009 April ; 157(4): 666–672. doi:10.1016/j.ahj.2008.12.004.

Impact of Stress Testing Prior to PCI or Medical Management on Outcomes of Patients With Persistent Total Occlusion After Myocardial Infarction: Analysis From the Occluded Artery Trial

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Abstract

Background—In the Occluded Artery Trial (OAT), 2201 stable patients with an occluded infarct-related artery (IRA) were randomized to percutaneous coronary intervention (PCI) or optimal medical treatment alone (MED). There was no difference in the primary endpoint of death, re-MI or heart failure (CHF). We examined the prognostic impact of pre-randomization stress testing.

Methods—Stress testing was required by protocol except for patients with single vessel disease and akinesis/dyskinesis of the infarct zone. The presence of severe inducible ischemia was an exclusion criterion for OAT. We compared outcomes based on performance and results of stress testing.

Results—598 (27%) patients (297 PCI, 301 MED) underwent stress testing. Radionuclide imaging or stress echocardiography was performed in 40%. Patients who had stress testing were younger (57 vs. 59 years), had higher ejection fractions (49% vs. 47%), and had lower rates of death (7.8% vs. 13.2%), class IV CHF (2.4% vs. 5.5%), and the primary endpoint (13.9% vs. 18.9%) than patients without stress testing (all $p<0.01$). Mild-moderate ischemia was observed in 40% of patients with stress testing, and was not related to outcomes. Among patients with inducible ischemia, outcomes were similar for PCI and MED (all $p>0.1$).

Conclusions—In OAT, patients who underwent stress testing had better outcomes than patients who did not, likely related to differences in age and LV function. In patients managed with optimal medical therapy or PCI, mild-moderate inducible ischemia was not related to outcomes. The lack of

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benefit for PCI compared to MED alone was consistent regardless of whether stress testing was performed or inducible ischemia was present.

Introduction

Previous studies have indicated a prognostic benefit for revascularization in patients who have severe inducible ischemia early after acute myocardial infarction (MI)[1, 2]. However, the role of revascularization for patients with only mild-moderate ischemia has not been fully established, particularly among patients with persistent total occlusions of the infarct-related artery (IRA). In the Occluded Artery Trial (OAT), 2201 stable patients with a persistently occluded IRA after MI were randomized to percutaneous coronary intervention (PCI) or optimal medical treatment alone (MED)[3]. Overall, there was no difference in the primary endpoint of death, recurrent MI or heart failure[4]. The objective of this analysis was to determine the impact of pre-randomization stress testing on clinical outcomes in OAT.

Methods

The design and results of OAT have been previously published[3, 4]. The trial was supported by grants from the National Heart, Lung, And Blood Institute. Eligibility required angiographically documented total occlusion (TIMI flow 0–1) of the IRA at 3–28 days after MI and either ejection fraction < 50% or proximal occlusion of a major epicardial vessel. Patients were randomized to undergo PCI or optimal medical therapy alone (MED). Patients in the PCI group underwent revascularization within 24 hours after randomization. Stenting was recommended whenever technically feasible.

Stress testing with low level exercise or pharmacological stress was required by protocol except for patients with single vessel disease and akinesis/dyskinesis of the infarct zone. The type of stress (exercise or pharmacological) and use of imaging (radionuclide or echocardiography) were based on local clinical practice and left to the discretion of treating physicians and the findings were based on local laboratory interpretations. Patients with post-MI angina at rest or severe inducible ischemia in the IRA distribution were ineligible for OAT. Severe ischemia was defined as in previous guidelines and reperfusion trials[1, 5–12]. For exercise stress testing, severe ischemia was defined as exercise-induced ST segment depression ≥ 2mm, ST segment elevation ≥ 1 mm in leads without Q waves, inability to complete stage 1 of the standard Bruce Protocol without angina, failure to achieve 3 or 4 METS, or exertional hypotension presumed due to ischemia. For stress myocardial perfusion imaging, severe ischemia was defined as increased lung uptake with any reversible perfusion defect, reversible perfusion defects in multiple territories, or an extensive reversible perfusion defect occupying ≥ 20% of the left ventricle[13].

The primary endpoint in OAT was the composite of death from any cause, reinfarction, or NYHA class IV heart failure. Secondary endpoints evaluated in this analysis included the individual components of the primary endpoint and the composite of death or myocardial infarction. The definition of reinfarction has been previously reported[3, 4]. Reinfarction was centrally adjudicated.

Baseline characteristics were summarized as frequencies and percentages for categorical variables and as means and standard deviations for continuous variables. Comparisons by stress test performance (stress test performed vs. stress test not performed) and by stress test results (ischemia present vs. ischemia absent) were performed using chi square/Fisher's exact test for categorical variables and Student t-test for continuous variables. Estimates of the cumulative event rates were calculated by the Kaplan-Meier product-limit method[14] and groups were compared by the log-rank test of the 5-year curves[15]. Hazard ratio and 99% confidence intervals

were calculated by Cox proportional hazards regression models[16]. To control for the Type I error rate, it was pre-specified by the study protocol that a p-value of less than 0.01 would be considered as showing evidence of differences in secondary analysis. SAS version 9.1.3 (SAS Institute, Cary, NC) was used for statistical analyses. The authors are solely responsible for the design and conduct of this study analysis, the drafting and editing of the paper and its final contents.

Results

A total of 2201 patients were enrolled in OAT, including the 2166 patients enrolled as of December 31, 2005 that were included in the main OAT results publication[4], and an additional 35 patients enrolled in the extension phase of the viability ancillary study in 2006. Stress testing was performed prior to randomization in 598 (27%) patients (297 in PCI group, 301 in MED group; p=0.84). The type of stress was exercise for 427 patients and pharmacological for 171 patients. The method for detecting ischemia was radionuclide imaging for 179 patients, echocardiography for 58 patients, and electrocardiography (ECG) for 357 patients. The details on the type of stress testing and the results are shown in Table 1.

The baseline clinical and angiographic characteristics of patients who did or did not undergo pre-randomization stress testing are compared in Table 2. Patients who underwent stress testing were younger, less likely to have ST-elevation MI at presentation, loss of R-waves on the electrocardiogram, and left anterior descending coronary artery as the IRA, and had lower heart rates and higher ejection fractions. Among the patients who had stress testing performed, there were no significant differences in baseline characteristics between the PCI and MED groups (data not shown).

Mild-moderate inducible ischemia was present in the IRA territory in 240 (40%) patients. Baseline characteristics for patients with and without inducible ischemia are shown in Table 3. Patients with inducible ischemia had shorter times from the index MI to randomization. Among the patients with inducible ischemia, there were no significant differences ($p < 0.01$) between the PCI and MED groups. Compared to the patients without IRA ischemia, the patients with inducible ischemia in the IRA territory had similar rates of non-protocol PCI of the IRA (11.6% vs. 10.0%; p=0.9) and of other vessels (5.2% vs. 7.0%; p=0.62).

Clinical outcomes at 5 years are shown in Tables 4–5 and Figure 1. Compared to patients who had no stress testing performed, patients who underwent stress testing had lower rates of death, class IV congestive heart failure (CHF), and the composite primary endpoint (death, MI or class IV CHF). There were no significant differences between the PCI and MED treatment groups for primary or secondary endpoints in the subgroups of patients who did and did not undergo stress testing, and those who did and did not have inducible ischemia. There were no significant differences in outcomes between patients who did and did not have inducible ischemia. In a multivariate model, inducible ischemia was not independently related to the primary endpoint (p=0.50). There were no significant interactions for any of the endpoints between treatment group, performance of stress testing, and presence of inducible ischemia.

Discussion

This analysis was conducted to determine the impact of pre-randomization stress testing on clinical outcomes in patients with persistent total occlusions of the IRA following myocardial infarction in OAT. The three main findings of the analysis were: i) study patients who underwent stress testing had better outcomes than those who did not; ii) mild-moderate inducible ischemia was not associated with any difference in outcomes; iii) the lack of clinical

benefit for PCI over MED seen overall was consistent among subgroups regardless of whether stress testing was performed or inducible ischemia was present.

The higher event rates for patients who did not undergo pre-randomization stress testing in OAT is consistent with previous studies showing worse outcomes for patients unable to perform exercise testing[12, 17]. However, the higher event rate in OAT is most likely related to the differences in baseline clinical characteristics and left ventricular function. The OAT protocol did not require pre-randomization stress testing for patients with akinesis/dyskinesis of the infarct zone (unless multivessel disease was present). Not surprisingly, patients who underwent stress testing were younger, had less ST elevation and loss of R-waves, were less likely to have LAD as the IRA, had lower heart rates and higher ejection fractions. The patients who underwent pre-randomization stress testing were therefore a lower risk cohort with smaller infarct size and more preserved left ventricular function.

Previous studies, including the DANAMI trial, have demonstrated that patients with severe inducible ischemia early after MI benefit from revascularization[1], albeit in a period before the modern era of optimal medical therapy. It is unclear whether patients with only mild-moderate or delayed inducible ischemia after MI derive benefit from revascularization,. In the COURAGE trial, there was no benefit of PCI compared with optimal medical therapy in the 876 patients with previous myocardial infarction and objective evidence of ischemia on stress testing[18]. Patients with early markedly positive stress tests were excluded from COURAGE. The use of anti-anginal medications, aspirin, and ACE-inhibitors in OAT was similar to COURAGE, with the exception of lower use of calcium-channel blockers[4, 18]. In this analysis, PCI showed no benefit over MED for OAT patients with mild-moderate ischemia on pre-randomization stress testing. One potential explanation for the lack of benefit with PCI is that mild-moderate inducible ischemia does not help identify a subset of higher-risk patients who would benefit from revascularization. This is supported by the absence of any significant relationship between inducible ischemia and clinical outcomes on univariate or multivariate testing in this analysis. Furthermore, for patients undergoing PCI for persistent total occlusion of the IRA, any potential benefit may be offset by the risk of distal embolization leading to periprocedural MI and the loss of rapidly recruitable collateral flow, increasing the risk for subsequent ischemic events if restenosis or reocclusion occurs. The results of this analysis, along with the main OAT results, indicate that patients with persistent occlusion of the IRA and no post-MI angina at rest or severe inducible ischemia should be treated medically, which is consistent with the revised ACC/AHA and ESC guidelines[19, 20].

In contrast to other previous studies[5, 6, 11], we did not demonstrate any relationship between the presence of inducible ischemia and clinical outcomes in this analysis. Since patients with severe inducible ischemia were ineligible for the trial, this analysis only evaluated patients with mild-moderate ischemia. Several studies have shown that only severe inducible ischemia is associated with worse outcomes after MI[7, 8, 10]. The COURAGE nuclear substudy demonstrated confirmed the relationship between ischemia in >10% of the LV and increased risk of events, and suggested that revascularization may benefit this group [21]. In OAT, mild-moderate inducible ischemia was documented in 40% of patients who underwent pre-randomization stress testing, but was not related to prognosis. It is possible that inducible ischemia after MI is only associated with worse clinical outcomes in patients with patent IRA. The presence of inducible ischemia in the IRA territory was not associated with higher rates of non-protocol (cross-over) PCI that may have confounded our ability to identify a relationship between inducible ischemia and outcomes.

Although these results indicate that the presence of mild-moderate inducible ischemia in this patient population does not provide prognostic information or an indication for PCI of a persistently occluded IRA, stress testing may still be warranted after myocardial infarction to

rule out severe inducible ischemia. Previous studies and the recent COURAGE substudy suggest the possibility that patients with severe inducible ischemia after MI may benefit from revascularization[1, 7, 8, 10, 21].

Several limitations must be acknowledged. Stress testing was performed in only 27% of patients in OAT. The possibility that this study is underpowered to identify a significant association between ischemia and outcomes cannot be excluded. Nevertheless, this is the largest study to date to the impact of inducible ischemia on outcomes for patients with persistently occluded IRA undergoing PCI or receiving optimal medical therapy. The type of stress testing was based on local clinical practice. The majority of patients in this analysis underwent only low-level exercise testing without radionuclide or echocardiographic imaging. Given the lower sensitivity of low-level exercise testing, it is possible that the number of patients with inducible ischemia was underestimated. However, this would not alter the observed lack of benefit of PCI over MED, since there was no signal of benefit and possibly a trend toward higher event rates with PCI irrespective of whether inducible ischemia was documented. The presence and localization of inducible ischemia in the IRA territory vs. non-IRA territory was based on site interpretation. The ability of sites to accurately localize ischemia may have been limited by the low use of radionuclide or echocardiographic imaging. However, since 82% of patients who underwent stress testing had single vessel disease, the localization of ischemia to the IRA territory should have been correct in the vast majority of cases. The anti-anginal medications taken at the time of stress testing were not documented. The findings of this analysis apply only to patients with persistent total occlusion of the IRA after MI and without severe inducible ischemia or post-MI angina at rest.

Conclusions

In OAT, patients who underwent stress testing had better outcomes than patients who did not, likely related to differences in age and LV function. In patients managed with optimal medical therapy or PCI, mild-moderate inducible ischemia was not related to clinical outcomes. The lack of benefit for PCI over MED was consistent regardless of whether stress testing was performed or mild-moderate inducible ischemia was present.

Acknowledgments

We thank the investigators and the staff at the study sites for their important contributions, Zubin Dastur and Erika Laurion for assistance in the preparation of the manuscript.

The project described was supported by Award Numbers U01 HL062509 and U01 HL062511 from the National Heart, Lung, And Blood Institute. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Heart, Lung, And Blood Institute or the National Institutes of Health.

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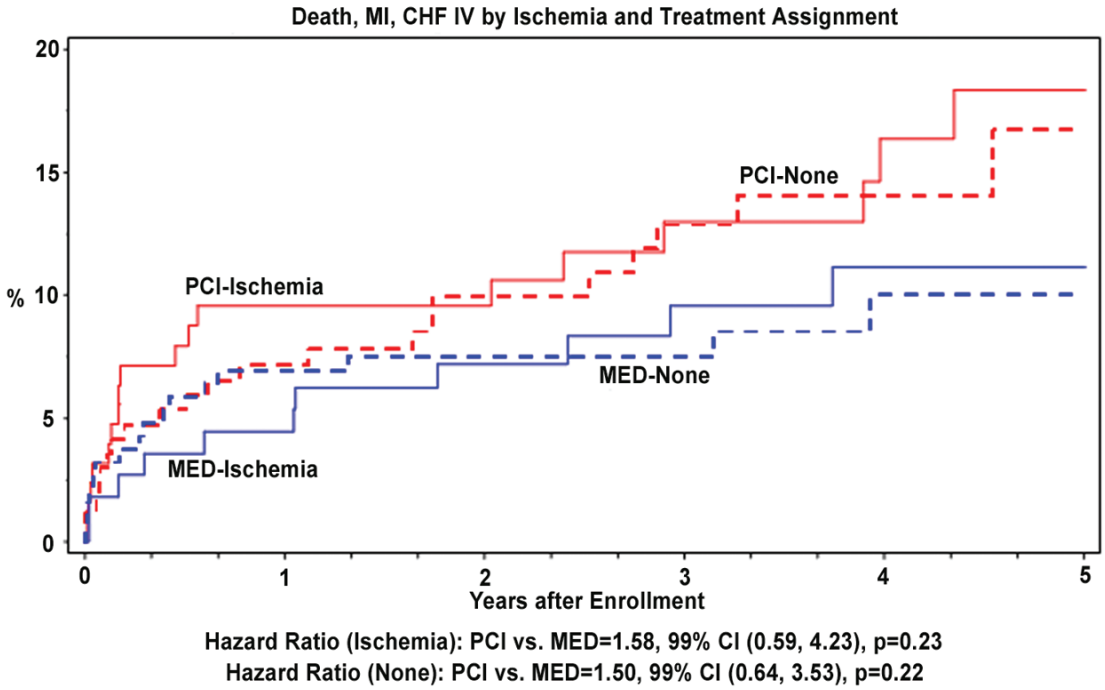

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Table 1

Type and Results of Pre-Randomization Stress Testing

	PCI Group (n=297)	MED Group (n=301)	P value
A. Type of stress			0.93
Exercise	215 (72%)	212 (70%)	
Dobutamine	27 (9%)	28 (9%)	
Dipyridamole	36 (12%)	40 (13%)	
Adenosine	13 (4 %)	12 (4%)	
Other	6 (2%)	9 (3%)	
B. Method of recording			0.77
Radionuclide	83 (28%)	96 (32%)	
Echocardiography	30 (10%)	28 (9%)	
Electrocardiography	182 (61%)	175 (58%)	
Other	2 (1%)	2 (1%)	
C. Stress test results for ischemia in IRA distribution:			0.21
Severe (Ineligible)	0 (0%)	1 (0.3%)	
Moderate	27 (9%)	32 (11%)	
Mild	100 (34%)	80 (27%)	
None	170 (57%)	188 (63%)	
D. Stress test results for ischemia in non-IRA distribution:			0.20
Severe	0 (0%)	4 (1%)	
Moderate	12 (4%)	16 (5%)	
Mild	39 (13%)	40 (13%)	
None	246 (83%)	241 (80%)	

IRA = infarct-related artery; PCI = Percutaneous Coronary Intervention; MED = Medical Therapy Alone

Table 2

Baseline Clinical & Angiographic Characteristics by Stress Test Performed

	Stress Test Performed (n=598)	No Stress Test Performed (n=1603)	P value
Age	57 ± 11	59 ± 11	0.002
Caucasian	498 (83%)	1265 (79%)	0.02
Male	483 (81%)	1234 (77%)	0.06
Diabetes	108 (18%)	346 (22%)	0.07
Hypertension	288 (48%)	783 (49%)	0.77
Hyperlipidemia	326 (55%)	816/1602 (51%)	0.14
Family History of Coronary Disease	227 (38%)	656 (41%)	0.21
Current Smoker	229 (38%)	630 (39%)	0.67
Previous Angina	136 (23%)	359 (22%)	0.86
Prior Myocardial Infarction	69 (12%)	178 (11%)	0.77
Cerebrovascular Disease	22 (4%)	60 (4%)	0.94
Peripheral Vascular Disease	29/596 (5%)	54 (3%)	0.10
Renal Insufficiency	12 (2%)	18 (1%)	0.11
Prior Congestive Heart Failure	8/596 (1%)	44 (3%)	0.05
Prior Percutaneous Coronary Intervention	33 (6%)	72 (5%)	0.31
Thrombolysis in 1st 24 hours	123 (21%)	301 (19%)	0.35
New York Heart Association Class 2–4	130/597 (22%)	329 (21%)	0.52
New Q waves	395 (66%)	1080 (67%)	0.56
ST segment Elevation	344/576 (60%)	1066/1550 (69%)	<0.0001
Loss of R waves	214/576 (37%)	699/1549 (45%)	0.001
Left Anterior Descending Infarct-Related Artery	168 (28%)	625 (39%)	<0.0001
Collateral Vessels Present	531/588 (90%)	1391/1585 (88%)	0.10
Multivessel Coronary Disease	109/591 (18%)	270/1592 (17%)	0.42
Ejection Fraction (%)	49 ± 11	47± 11	0.0004
Number of Days from Myocardial Infarction to Randomization	15 ± 8	10 ± 7	<0.0001
Glomerular Filtration Rate (mL/min/1.73m^2)	80 ± 20	81 ± 22	0.60
Fasting Glucose (mg/dL)	113 ± 38	122 ± 43	0.001
Heart Rate	70 ± 12	73 ± 12	<0.0001
Systolic Blood Pressure (mmHg)	120 ± 17	121 ± 19	0.26
Diastolic Blood Pressure (mmHg)	72 ± 11	72 ± 12	0.95
Killip Class II–IV	113/595 (19%)	304/1597 (19%)	0.98
Body Mass Index	28 ± 5	29 ± 5	0.51

Values are presented as number (%) of patients or mean ± standard deviation.

Table 3

Baseline Clinical & Angiographic Characteristics by Inducible Ischemia

	Inducible Ischemia Present (n=240)	No Inducible Ischemia Present (n=358)	P value
Age	57 ± 11	58 ± 11	0.79
Caucasian	194 (81%)	304 (85%)	0.19
Male	204 (85%)	279 (78%)	0.03
Diabetes	48 (20%)	60 (17%)	0.31
Hypertension	125 (52%)	163 (46%)	0.12
Hyperlipidemia	144 (60%)	182 (51%)	0.03
Family History of Coronary Disease	97 (40%)	130 (36%)	0.31
Current Smoker	91 (38%)	138 (39%)	0.88
Previous Angina	64 (27%)	72 (20%)	0.06
Prior Myocardial Infarction	33 (14%)	36 (10%)	0.17
Cerebrovascular Disease	9 (4%)	13 (4%)	0.94
Peripheral Vascular Disease	14/239 (6%)	15/357 (4%)	0.36
Renal Insufficiency	4 (2%)	8 (2%)	0.63
Prior Congestive Heart Failure	5/239 (2%)	3/357 (1%)	0.19
Prior Percutaneous Coronary Intervention	16 (7%)	17 (5%)	0.31
Thrombolysis in 1st 24 hours	48 (20%)	75 (21%)	0.78
New York Heart Association Class 2–4	47 (20%)	83 (23%)	0.29
New Q waves	151 (63%)	244 (68%)	0.18
ST segment Elevation	133/232 (57%)	211/344 (61%)	0.34
Loss of R waves	79/232 (34%)	135/344 (39%)	0.21
Left Anterior Descending Infarct-Related Artery	62 (26%)	106 (30%)	0.31
Collateral Vessels Present	210/235 (89%)	321/353 (91%)	0.53
Multivessel Coronary Disease	44/238 (18%)	65/353 (18%)	0.98
Ejection Fraction (%)	50 ± 10	49± 11	0.07
Number of Days from Myocardial Infarction to Randomization	14 ± 8	16 ± 7	0.0001
Glomerular Filtration Rate (mL/min/1.73m^2)	81 ± 19	80 ± 21	0.77
Fasting Glucose (mg/dL)	118 ± 40	111 ± 37	0.03
Heart Rate	69 ± 12	70 ± 12	0.60
Systolic Blood Pressure (mmHg)	119 ± 17	121 ± 17	0.32
Diastolic Blood Pressure (mmHg)	72 ± 11	73 ± 10	0.33
Killip Class II–IV	40/239 (17%)	73/356 (20%)	0.25
Body Mass Index	29 ± 5	28 ± 5	0.02

Values are presented as number (%) of patients or mean ± standard deviation.

Table 4

5-year Life Table estimates of Clinical Outcomes by Treatment Group & Stress Test Performed

		PCI Group Stress done (n=297) Not done (n=804)	MED Group Stress done (n=301) Not done (n=799)				P-Value (HR;99%CI) for stress test done vs stress test not done	
				p-value	HR	99% CI	Unadjusted	Adjusted BL
Death, MI or Class IV CHF	Stress Test	39 (17.6%)	27 (10.5%)	0.08	1.55	0.81–2.95	0.007 (1.46;1.02–2.08)	0.38 (1.14;0.78–1.66)
	No Stress Test	126 (19.4%)	119 (18.4%)	0.71	1.05	0.76–1.46		
Death	Stress Test	17 (9.7%)	13 (6.2%)	0.37	1.39	0.54–3.59	0.0004 (2.02;1.21–3.38)	0.01 (1.66;0.97–2.83)
	No Stress Test	75 (12.4%)	79 (14.1%)	0.66	0.93	0.61–1.41		
Nonfatal MI	Stress Test	19 (8.0%)	15 (5.9%)	0.40	1.33	0.55–3.26	0.15 (0.74;0.43–1.27)	0.03 (0.60;0.33–1.08)
	No Stress Test	39 (6.4%)	26 (4.3%)	0.11	1.50	0.78–2.88		
Death or MI	Stress Test	35 (16.9%)	25 (9.9%)	0.13	1.49	0.76–2.93	0.05 (1.34;0.92–1.96)	0.56 (1.09;0.73–1.63)
	No Stress Test	108 (17.7%)	99 (17.2%)	0.55	1.09	0.76–1.56		
Class IV CHF	Stress Test	8 (3.1%)	5 (1.7%)	0.37	1.67	0.38–7.24	0.007 (2.25;1.04–4.87)	0.15 (1.56;0.70–3.49)
	No Stress Test	36 (5.6%)	39 (5.3%)	0.69	0.91	0.50–1.66		

PCI = Percutaneous Coronary Intervention; MED = Medical Therapy Alone; HR=Hazard Ratio; MI = Myocardial Infarction; CHF = Congestive Heart Failure

Table 5

5-year Life Table Estimates of Clinical Outcomes by Treatment Group & Inducible Ischemia

		PCI Group Ischemia (n=127) No Ischemia (n=170)	MED Group Ischemia (n=113) No Ischemia (n=188)				P-Value (HR;99%CI)for ischemia vs no ischemia	
				p-value	HR	99% CI	Unadjusted	Adjusted BL
Death, MI or Class IV CHF	Ischemia	18 (18.4)	11 (11.2)	0.23	1.58	0.59–4.23	0.67 (1.11;0.59–2.11)	0.77 (1.08;0.56–2.05)
	No ischemia	21 (16.7)	16 (10.0)	0.22	1.50	0.64–3.53		
Death	Ischemia	8 (10.1%)	3 (3.4%)	0.18	2.49	0.43–14.2	0.48 (0.77;0.29–2.04)	0.52 (0.78;0.29–2.10)
	No ischemia	9 (8.8%)	10 (8.3%)	0.93	1.04	0.32–3.40		
Nonfatal MI	Ischemia	9 (9.2%)	7 (6.9%)	0.67	1.24	0.31–4.55	0.47 (1.28;0.53–3.11)	0.72 (1.14;0.46–2.78)
	No ischemia	10 (6.8%)	8 (5.2%)	0.47	1.41	0.42–4.78		
Death or MI	Ischemia	17 (19.1%)	10 (10.2%)	0.21	1.64	0.59–4.59	0.61 (1.14;0.59–2.24)	0.73 (1.10;0.56–2.16)
	No ischemia	18 (14.6%)	15 (9.8%)	0.38	1.36	0.55–3.35		
Class IV CHF	Ischemia	2 (1.9%)	2 (1.8%)	0.95	0.95	0.07–12.4	0.48 (0.66;0.14–3.09)	0.57 (0.71;0.15–3.38)
	No ischemia	6 (3.9%)	3 (1.7%)	0.26	2.20	0.36–13.8		

PCI = Percutaneous Coronary Intervention; MED = Medical Therapy Alone; MI = Myocardial Infarction; CHF = Congestive Heart Failure

Am Heart J. Author manuscript; available in PMC 2010 April 1.