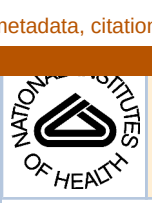

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Beta-blocker Use Following Myocardial Infarction: Low Prevalence of Evidence-based Dosing

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Abstract

Background—Quality improvement programs have shown increased use of beta-blockers post-myocardial infarction(MI), but there are no data on whether appropriate doses are administered.

Methods—In a prospective registry that enrolled consecutive patients with MI, we evaluated beta-blocker dosing at discharge after MI and 3-weeks later and assessed clinical predictors for treatment with very low doses. We studied 1971 patients (70.8% male) with mean age 63.9±13.7 years, of whom 48.2% had an ST elevation MI.

Results—Beta-blocker utilization rates following MI were 93.2% at discharge-20.1% received <25% of target dose, 36.5% received 25% of target dose, 26.4% received 26–50% of target dose, and 17.0% received >50% of target dose. Between discharge and three-weeks, 76.4% had no change in beta-blocker dose, with 11.9% and 11.6% having their dose reduced and increased, respectively. Absence of hypertension, acute PCI, older age, and no ACE-inhibitor therapy were consistent predictors of treatment with very low beta-blocker doses.

Conclusions—Underdosing of beta-blockers is highly prevalent among patients post-MI. This represents an important opportunity in quality improvement for the care of patients who have suffered a MI.

Keywords

myocardial infarction; beta-blockers; quality

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INTRODUCTION

For >20 years, it has been known that beta-blockers improve survival following myocardial infarction(MI). This has been established in multiple randomized trials[1–5] including CAPRICORN[2] performed in the reperfusion era. In addition, observational studies from large databases have confirmed that beta-blockers may confer up to a 40% survival advantage following MI[6–8]. For this reason, the 1990 ACC/AHA guidelines[9] first recommended (class I or IIa recommendation) beta-blocker therapy for essentially all post-MI patients, except those with contraindications. Despite these data and guideline recommendations, many studies published in the 1990s[7, 10–18] demonstrated that beta-blockers were under-prescribed following MI, with the largest studies showing utilization rates of approximately 50% or less. In order to enhance beta-blocker use post-MI, this was selected to be a key quality indicator tracked by several organizations, including the National Center for Quality Assurance, the Joint Commission on Accreditation of Healthcare Organizations, and the Center for Medicare and Medicaid Services, and this indicator was incorporated in the 2006 and 2008 ACC/AHA clinical performance measures for acute MI[19, 20]. Recent data document the effectiveness of this approach. The CRUSADE registry[21] demonstrated that 84% of patients were discharged on beta-blockers. The NCQA has also demonstrated an increase in beta-blocker utilization rates among eligible post-MI patients from approximately 60% in 1996 to >90% in 2005[22]. A recent European survey demonstrated that 80% of patients with acute MI were treated with beta-blockers [23]. It is important to note that the quality indicator tracked by these organizations is whether a beta-blocker was prescribed or not, with no consideration as to whether the dose administered is reflective of the doses shown to be effective in the randomized trials upon which this performance measure was based.

In addition to the underuse of beta-blockers, several studies[11, 18, 24, 25] have also documented underdosing of beta-blockers. These studies, predominantly reflecting practice patterns in the 1990s, showed that the vast majority of patients discharged on beta-blockers did not achieve the target doses demonstrated to be effective in the randomized trials. In fact, the majority received ≤50% of the target dose. This contrasts with the dosing used in randomized clinical trials of beta-blockers which have shown a very high success rate of achieving target dose; these trials and the dosing used serve as the basis for the survival benefit of post-MI beta-blocker therapy. Interestingly, the COMMIT trial[26], in which rapid titration to target doses was performed, demonstrated no survival benefit. The purpose of this report is to determine whether the evidence-based quality assurance initiatives that have increased beta-blocker use at discharge have also impacted the issue of underdosing of beta-blockers, by evaluating beta blocker doses in patients enrolled in the **PACE**maker & Beta-blocker Therapy Post-**M**yocardial **I**nfarction (PACE-MI) Trial Registry.

METHODS

The PACE-MI Trial is a randomized clinical trial evaluating whether pacemaker facilitated beta-blocker therapy after MI in patients who have bradycardia contraindications to beta-blockers improves survival[27]. The trial includes a registry of all patients with MI at most of the participating sites, in which detailed information regarding beta-blocker therapy is collected. Participating sites are listed in the appendix and include a cross-section of hospital types.

Patient Population

All patients admitted with acute MI at participating sites were entered into the registry. Acute MI was diagnosed by:

1. Either CPK elevation >2 times or troponin elevation >3 times the upper limit of normal and
2. Either chest pain (or equivalent symptoms suggestive of MI) or ECG changes consistent with MI.

Peak total CPK and troponin values were recorded, as available (5 sites used troponin T); because of the multiple laboratories and the wide range of reported upper limits of normal among the sites, only a median value is provided and this was not used for further analysis. Basic demographic, historical, and hospitalization information, as well as information regarding the index MI, was collected. Sites were instructed to obtain information from the emergency room record, admission history, discharge summary, cardiac catheterization report, and medication sheets. Specific information regarding beta-blocker type and dose at discharge was collected. Specific reasons for not using beta-blockers at discharge were noted in patients not discharged on beta-blockers. In addition, at most sites, beta-blocker dose three weeks following MI was ascertained via the patient's physician. No specific beta-blocker was recommended. All data were collected at the site and de-identified patient information was entered in a web based electronic data capture system (Medidata, New York, NY). A waiver of consent for the registry was obtained at each participating site. The study was approved by the Northwestern University IRB and at each participating site IRB.

Registry data include patients admitted with MI between August 2007 and July1,2008. As sites received IRB approval, registry accrual began. In June2008, the Steering Committee and DSMB reviewed the progress of the study as well as registry data. Because of the potential lack of efficacy of sub-therapeutic beta-blocker dosing as well as changes in registry procedures (change of ascertainment of beta-blocker use from three-weeks to two-months), the Steering Committee approved publication of these data.

Data Analysis

Patient characteristics were compared using chi-square tests for categorical variables and analysis of variance for continuous variables. Multiple logistic regression was used to model predictors of no beta-blocker or very low beta-blocker dose at discharge and 3-week follow-up. As beta-blocker type was chosen by the managing physician, for the purposes of this study, we defined the target doses for the most commonly used beta-blockers: metoprolol 200mg/day[3], carvedilol50 mg/day[2] (CoregCR80mg/day), propranolol180mg/day[1], bisoprolol10 mg/day[28], and atenolol100 mg/day[18, 29]. Based on the doses administered, a proportion of target dose was calculated (dose administered/target dose). Data are reported for metoprolol, the most commonly prescribed beta-blocker, separately and for all beta-blockers for which target dose could be identified. Beta-blocker doses were divided into three groups: ≤25% target dose (defined as very low dose), 26–50% target dose, and >50% target dose. Analyses were performed using SAS (SAS Institute Inc., Cary, NC). A p-value <0.05 was considered significant.

RESULTS

Table 1 provides the demographic and clinical data. There were 1971 patients (70.8% male) with mean age 63.9±13.7 (range 26 to 97) years; 48.2% had an ST elevation MI. Of these patients, 73.9% were treated with acute PCI and 17.0% were treated with thrombolytic therapy. The median (inter-quartile range) for peak troponin was 5.2 (1.5–20.5) ng/ml. In-hospital mortality was 5.0%. Of the 1872 patients discharged alive, 93.2% were treated with beta-blockers, 92.7% with aspirin, 68.7% with ACE-inhibitors/ARB, and 83.8% with statins. Mean ejection fraction for patients discharged alive was 47.8±12.2%.

There were 128 patients(6.8%) discharged without beta-blocker therapy. Reasons provided for not administering beta-blockers are shown in Table 2. Sinus node disease/bradycardia and hypotension were the most common listed reasons.

Of the 1741 patients discharged on beta-blocker therapy, 70.7% were administered metoprolol, 19.5% carvedilol (including CoregCR), 3.5% atenolol, 0.3% propranolol, 5.2% bisoprolol, and 0.86% received other beta-blockers.

Figure 1 demonstrates the beta-blocker dose distribution. At discharge, 18.1% of the metoprolol-treated patients received <50 mg/day, 39.8% received 50 mg, 25.9% received between 51–100 mg, and 16.3% received >100 mg. The respective numbers for all beta-blockers are 20.1% (<25% target dose), 36.5% (25% target dose), 26.4% (26–50% target dose), and 17.0% (>50% target dose). Prior beta-blocker use was noted in 32% of patients and was associated with a higher discharge dose (48.6±32.3% versus 36.4±26.2% of target dose, $p<0.001$).

Three-week follow-up data were available in 1433(77.3%) patients. Mortality at 3-weeks was 1.2% among those discharged alive. At 3-weeks, 55 patients(3.8%) were not taking beta-blockers. Of the 1378 patients who were taking beta-blockers at 3-week follow-up, metoprolol accounted for 69.5%, carvedilol 18.3%, propranolol 0.2%, bisoprolol 7.0%, atenolol 4.1%, and other 0.9%. Of those receiving metoprolol, 18.9% received <50mg/day, 39.4% 50mg/day, 26.7% 51–100mg/day, and 15.8% >100mg/day. Respective numbers for all beta-blockers were 18.4%, 35.6%, 29.8%, and 16.7%.

Between discharge and 3-weeks, 76.4% of patients had no change in beta-blocker dose. There were 338(23.6%) patients who had a dose change. Of these, 167 patients(49.4%) had an increase in dose, and 171(50.6%) had a decrease in dose (figure 2). For patients whose dosage increased, there was a mean 28.8±22.3% dosage increase. For those patients treated with metoprolol, the mean daily dosage at discharge was 50.5±38.3mg and at 3-weeks 91.0±70.6mg. For those patients whose dosage decreased, there was a mean 38.9±58.7% change. For those patients treated with metoprolol, the mean daily dosage at discharge was 105.2±78.1mg and at 3-weeks 49.5±45.6mg.

Tables 3 and 4 demonstrate univariate and multivariable predictors of beta-blocker dosage at discharge and at 3-weeks. In the multivariable analysis, absence of hypertension, acute percutaneous coronary intervention, older age, and lack of ACE-inhibitor therapy were consistent predictors of treatment with very low doses of beta-blockers.

DISCUSSION

The PACE-MI Registry provides contemporary data on patterns of beta-blocker therapy post-MI. This registry confirms recent findings that utilization rates for beta-blockers post-MI are excellent, exceeding 90%. In all likelihood, this can be attributed to the cumulative effect of evidence-based guidelines and multiple national quality improvement initiatives. The prescribing patterns also document that patients are generally discharged on substantially lower dosages than the target dose. In addition, the vast majority of patients do not undergo any dose titration between discharge and three-weeks. At the end of three-week follow-up period, only 46% patients were taking ≥50% of the target dose shown to be beneficial in clinical trials. Furthermore, 54% of patients were still being treated with ≤25% of the dose shown to be effective in clinical trials. There are no data demonstrating the efficacy of beta-blockers at these very low doses. While underdosing of beta-blocker therapy has been documented in the past, this was in the setting of beta-blocker underuse. These data demonstrate that despite the widespread success of current quality improvement initiatives, underdosing of beta-blocker

therapy remains a significant clinical issue. This represents an important opportunity in quality improvement for the care of patients who have suffered a MI.

Clinical trials evaluating beta-blocker use following MI have set a substantial target dose. In BHAT[1], 82% were assigned to 180mg/day dosing and 18% to 240mg/day dosing. At study end, 57% were receiving the full-protocol propranolol dose (85% still on a beta-blocker). Based on pill counts, >90% of the prescribed dose was taken by 78% of patients in the Goteborg metoprolol trial[3] and 85% of patients in the Norwegian Multicenter Study Group[30]. More contemporary data from CAPRICORN[2] show that 74% were taking the target dose of carvedilol (50mg/day) with 11% taking 25mg/day (50% of target) and 7% taking 12.5mg/day (25% of target). It should be noted that this study enrolled only patients whose left ventricular ejection fraction was ≤40%. It is also interesting to examine the dose distribution of metoprolol in MERIT-HF[31] at 3 month follow-up (including >90% of patients enrolled, over half of whom had NYHA functional class III heart failure). The 200 mg/day target dose was achieved in 56% of patients with 11% taking 150 mg/day and 19% taking 100 mg/day. The SENIORS trial[32] evaluated nebivolol in a placebo-controlled randomized trial in elderly (≥70 years) patients with heart failure. Of the 1031 patients allocated to nebivolol, 67% reached target dose and an additional 12% reached 50% of the target dose. Thus, randomized clinical trials support the ability of achieving target doses in the majority of patients, even the elderly and patients with heart failure, with the vast majority able to take ≥50% of the target dose.

While guidelines and performance measures for the care of post-MI patients clearly indicate that virtually all such patients should be treated with beta-blocker therapy, the guidelines are silent on dosing[9, 19, 20, 33, 34]. As the strong recommendations of the guidelines and performance measures committees were based on the evidence accumulated from multiple randomized clinical trials, it is reasonable to presume that the committees' intent was for beta-blocker therapy to be used as in those trials. Although clinical trials show that target doses can be achieved in substantial numbers of patients, the present data indicate this does not happen in clinical practice. Although quality improvement initiatives have had enormous impact on the problem of beta-blocker underuse following MI, underdosing remains a significant problem. It is therefore important to explore both the clinical implications of underdosing, potential reasons for not achieving higher doses, and further steps to address this issue.

Because dose-response studies have not been performed in survival trials following MI, there are no data to evaluate the efficacy of lower beta-blocker doses, but there are some data from heart failure trials. The MOCHA trial[35] evaluated three carvedilol doses in patients with heart failure–12.5, 25, and 50mg/day. Although this trial was not designed to evaluate carvedilol's effect on survival, the lowest 6 month mortality (1.1%) was noted in the highest dose group. The groups receiving intermediate doses had mortalities of 6–6.7%. Other large-scale heart failure trials have performed post-hoc analyses evaluating the effect of dose on mortality reduction. In MERIT-HF[31], the high-dose group (mean 192mg/day) experienced 6.2% deaths/patient-year, while the low-dose group (mean 76mg/day), experienced 8% deaths/patient-year. This compared to the 10.8% deaths/patient-year in the 1845 placebo patients. There were 22% and 29% reductions in all-cause mortality plus all-cause hospitalization in the low- and high-dose subgroups, respectively, and 40% and 62% reductions in hospitalization due to worsening heart failure. Dose was also evaluated in COMET[36] comparing metoprolol (target 100mg/day) and carvedilol (target 50mg/day) in patients with heart failure. In multivariate analysis, the relative risk associated with target versus below target dose treatment was 0.78 ($p<0.0025$). Patients on target dose had a two-year mortality of approximately 10%, while those at below target dose had a two-year mortality of approximately 20%. Of note, there is a paucity of data even in these trials regarding patients taking very low doses of beta-blockers, specifically those <25% of target dose.

While this registry demonstrated that most patients did not have any dose titration between discharge and three-week follow-up, the reasons for the lack of dose titration are unclear. It is certainly possible that patients could not tolerate higher doses. This seems unlikely given the dramatically lower percentage of patients in this Registry achieving ≥50% of target dose compared to what has been achieved in prior trials. Alternative explanations could include physician reluctance to up-titrate beta-blockers due to the variety of other medications patients were taking. Another possibility is the lack of an infrastructure to effect dose titration systematically following discharge. As the average length of stay following MI has declined, the hospital setting does not provide as adequate an opportunity for dose-titration as in the earlier era of longer hospital stays. In contrast to the clinical practice setting, in clinical trials, patients are seen frequently and can have their dose escalated at each visit. It should be noted that avoiding excessively rapid dose titration is also a very important issue, as the COMMIT trial[26] demonstrated both positive and negative clinical outcomes in post-MI patients whose dose was escalated rapidly during the in-hospital phase following MI. Specifically, they observed a 22% reduction in arrhythmic death but an increase in cardiogenic shock; overall there was no survival benefit when beta-blockers were given in this fashion. Similarly, the POISE trial[37] evaluated the effects of peri-operative beta-blocker therapy for non cardiac surgery. The study demonstrated that rapid titration to target dose therapy can be associated with both beneficial (less MIs) and harmful (more deaths and strokes) effects. Finally, there could be reluctance among patients to have their dose increased. If this is a significant issue, efforts at patient education could be implemented to address this.

While quality improvement initiatives have had clear impact on beta-blocker use, new initiatives to deal with underdosing could be more challenging. As target doses are not likely to be achieved by hospital discharge, a true assessment of dose achieved must be performed at some later time. Because patients may be followed by multiple physicians at multiple locations, it is unclear when and how to obtain the follow-up beta-blocker information. Innovative hospital discharge programs involving nurses and pharmacists can impact outcomes[38]. It is likely that modifications of our current approach to the post-discharge management of the patient with MI will be required in order to incorporate target doses of beta-blockers in the quality assessment program. Based on past performance, setting these targets will also drive the medical community to accommodate this objective.

Limitations

It is possible that dose up-titration occurred beyond the three-week window evaluated in this study, as routine office visits often occur beyond this time in this cohort. However, studies that have evaluated long term beta-blocker use post-MI have generally shown attrition and not increasing use[24, 39]. Gislason[24] reported only a 2–7% change in beta-blocker and ACE-inhibitor dosing over a period of years post-MI and concluded that doses are “seldom adjusted during long-term therapy”.

The ideal time for dose titration post-MI is unknown. While dose titration for the randomized trials[2, 3, 30, 40] was generally reported to be done within several days to weeks post-MI, most of these trials were done at a time when many of the other current therapies were not used, making it easier to focus on up-titration of beta-blockers.

Conclusions

The PACE-MI Registry documents excellent impact of quality improvement initiatives to increase beta-blocker use following MI. However, underdosing remains a significant issue. Focusing only on the dichotomous question of “use” does not necessarily optimize quality if the majority of patients receive low doses, in the absence of data demonstrating a benefit of these doses. Until and unless it becomes clear that low dose beta-blockers provide the same

large benefit as the target doses used in clinical trials, every effort should be made to achieve target doses. As it seems unlikely that very low doses (≤25% target) will result in the full benefit achievable with higher doses, a new phase in the quality improvement programs to address this issue could have dramatic impact on survival of patients following MI.

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Appendix

The following investigators, coordinators, and centers participated in the study: P. Desai, M. Betzen, *Amarillo Heart Clinical Research Institute, Amarillo;* J. Whitehill, L. Janak, M. Lytle, M. Samendinger, *Austin Heart P.A., Austin;* E. Johnson, C. Allbritton, T. Bacon-Howard, J. Sparks, *Baptist Memorial Hospital, Memphis;* G. Nair, T. Strickland, *Clarian Health/ Methodist Research Institute, Indianapolis;* D. Lustgarten, B. Alemy, S. Forsell, L. Chadwick, M. Rowen; *Fletcher Allen Health Care, Burlington;* D. Dan, K. Picardi, *Fuqua Heart Center, Atlanta;* C. Schuger, J. Dzidowski, L. Louzon, *Henry Ford Hospital, Detroit;* J. Messenger, B. Easley, J. Morrison, *Long Beach Memorial Medical Center, Long Beach;* F. Kusumoto, B. Little, *Mayo Clinic, Jacksonville;* D. Mehta, E. Pe, A. Simon, *Mount Sinai School of Medicine, New York;* D. Fintel, E. Karpf, *Northwestern Memorial Hospital, Chicago;* D. Bello, S. Harrison, S. Sawyer, I. Viera-Fleetwood, *Orlando Regional Medical Center, Orlando;* J. Simonson, S. Barnes, J. LeTexier, *Park Nicollet Institute, Minneapolis;* M. Romanelli, L. Froehlich, S. Teller, *St. John Hospital and Medical Center, Detroit;* E. Herzog, R. Knox, *St. Luke's-Roosevelt Hospital, New York;* W. Warnica, D. Eichman, D. Scarcelli, B. Smith, *University of Calgary, Calgary;* J. Mounsey, C. Blyth, C. Schuler, *University of North Carolina, Chapel Hill;* R. Germany, J. Hubbart, *University of Oklahoma Health Sciences Center, Oklahoma City;* M. Panday, S. Harris, *The University of Texas Health Science Center at San Antonio, San Antonio;* A. Warner, J. Johnson, *West Los Angeles VA Medical Center, Los Angeles.*

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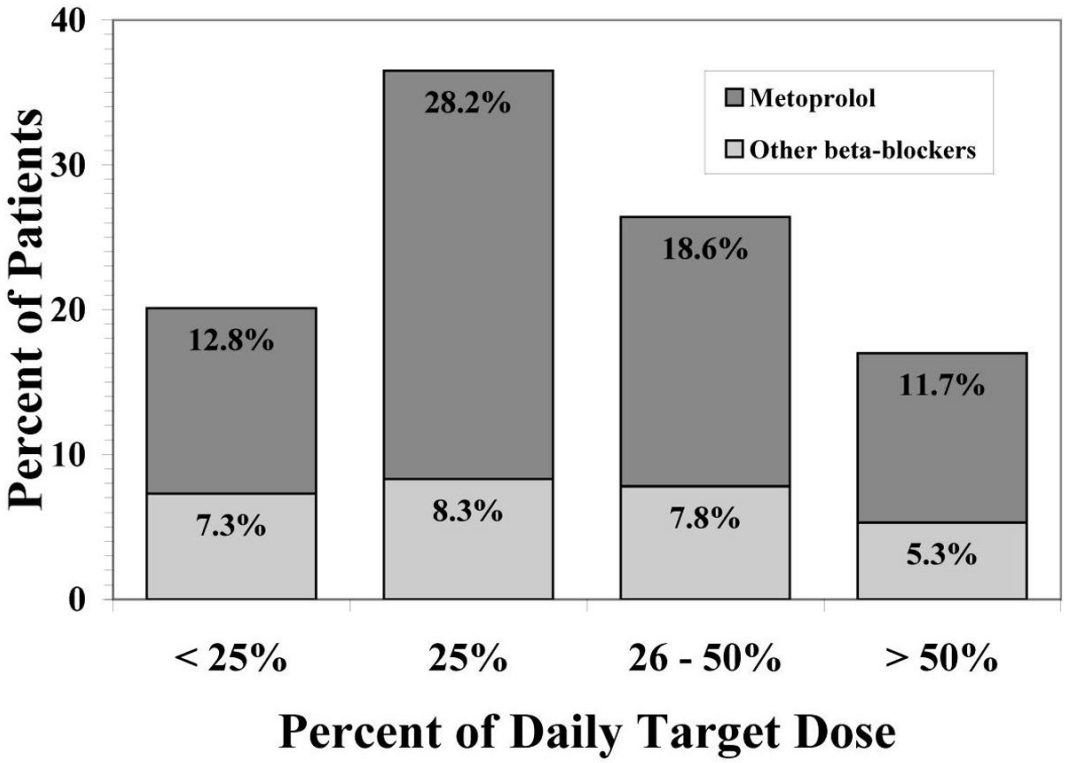

Figure 1.
The graph shows the percent of patients taking various doses of beta-blockers at discharge. Doses are shown as the proportion or percent of daily target dose - <25% - n=347; 25% - n=629; 26–50% - n=454; >50% - n=293. Each bar is subdivided to indicate metoprolol dosing and dosing of other beta-blockers.

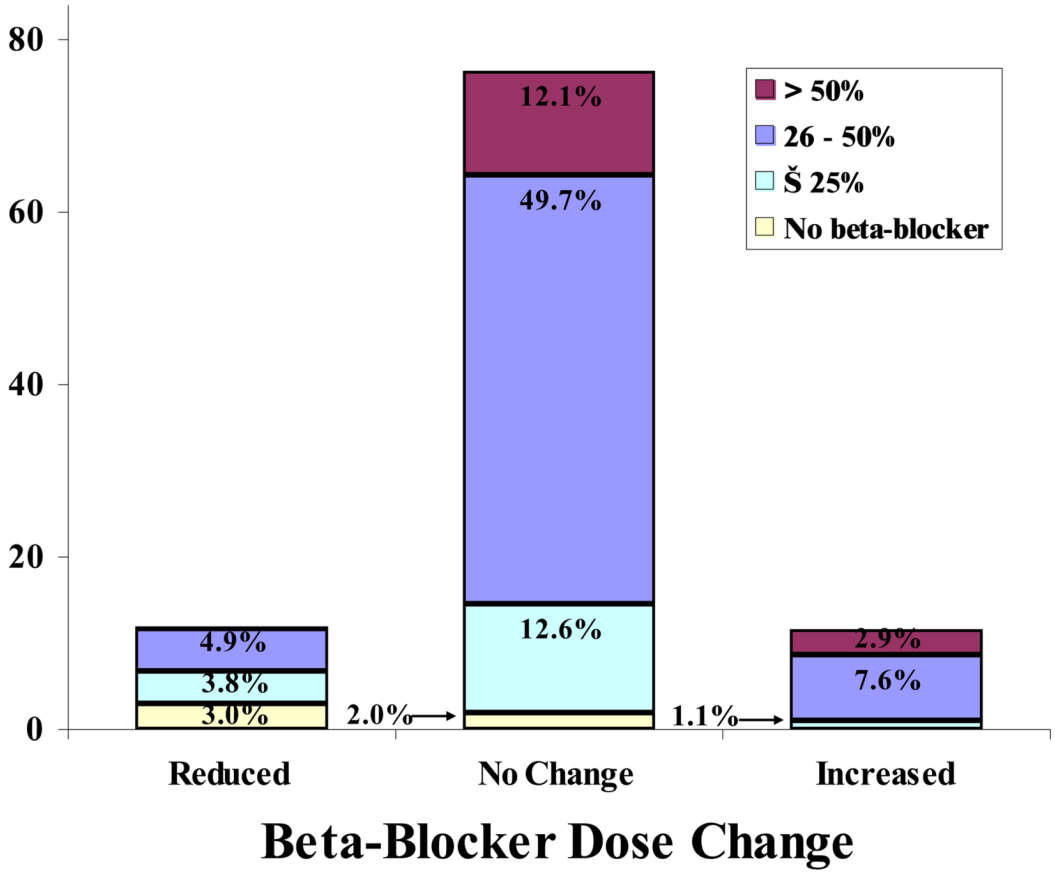

Figure 2.
The graph shows the distribution of beta-blocker dosing (shown as percent of daily target dose – see legend) at 3 weeks stratified by whether the dose was reduced (n=171), remained the same (n=1095), or increased (n=167) since hospital discharge.

Table 1

Clinical characteristics of the population, also stratified by beta-blocker dose at discharge (p-values indicate whether there are significant differences based on beta-blocker dose).

DEMOGRAPHICS	All Patients N=1971	Beta-blocker dose at discharge (as % of target dose)				
		None n=128	≤25% n=976	26–50% n=454	>50% n=293	p-value
Age (years) —Mean±SD	63.9±13.7	64.6±14.2	63.3±13.9	62.8±13.6	63.7±13.1	p=0.58
Male—N(%)	1396 (70.8)	83 (64.8)	678 (69.5)	337 (73.2)	224 (76.4)	**p=0.019**
Race—N(%) White	1562 (79.2)	98 (76.6)	774 (79.3)	371 (81.7)	230 (78.5)	p=0.52
American Indian	23 (1.2)	1 (0.8)	13 (1.3)	7 (1.5)	1 (0.3)	
Black	214 (10.9)	16 (12.5)	103 (10.6)	43 (9.5)	38 (13.0)	
Asian	83 (4.2)	4 (3.1)	30 (3.1)	23 (5.1)	16 (5.5)	
Hawaiian	4 (0.2)	0	1 (0.1)	2 (0.4)	0	
Unknown	86 (4.4)	9 (7.0)	55 (5.6)	8 (1.8)	9 (3.1)	
Mixed	1 (0.1)	0	0	0	1 (0.3)	
Hispanic—N(%);	119 (6.1)	11 (8.6)	80 (8.3)	12 (2.6)	11 (3.8)	**p<0.001**
LVEF (%)—Mean±SD	47.5±12.4	48.5±12.7	47.6±12.8	47.1±11.7	49.3±10.6	p=0.10
DM—N(%);	595 (30.2)	36 (28.1)	274 (28.1)	125 (27.5)	112 (38.2)	**p=0.006**
HTN—N(%);	1310 (66.5)	83 (64.8)	607 (62.2)	309 (68.1)	229 (78.2)	**p<0.001**
Hypercholesterolemia—N(%)	1041 (52.9)	67 (52.3)	494 (50.6)	247 (54.5)	179 (61.3)	**p=0.014**
STEMI—N(%);	949 (48.2)	49 (38.3)	497 (50.9)	224 (49.3)	114 (38.9)	**p<0.001**
Anterior	274 (31.5)	6 (13.0)	134 (29.6)	71 (34.3)	32 (31.1)	
Inferior	387 (44.5)	26 (56.5)	206 (45.5)	94 (45.4)	43 (41.8)	
Lytic—N(%)	195 (9.9)	14 (10.9)	108 (11.1)	45 (9.9)	24 (8.2)	p=0.54
Primary PCI—N(%);	1053 (53.5)	68 (53.1)	590 (60.4)	237 (52.3)	114 (38.9)	**p<0.001**
Discharge Medications—N(%)						
ASA;	1734 (88.1)	112 (87.5)	915 (93.8)	428 (94.3)	263 (89.8)	**p=0.007**
ACEI/ARB;	1285 (65.3)	74 (57.8)	661 (67.7)	333 (73.4)	205 (70.0)	**p=0.007**
Statin	1564 (83.8)	98 (76.6)	818 (84.0)	382 (84.3)	250 (85.3)	p=0.14
Clopidogrel	1387 (74.9)	77 (60.2)	753 (77.2)	353 (77.8)	204 (69.6)	**p<0.001**

LVEF left ventricular ejection fraction;

DM diabetes mellitus

HTN hypertension

STEMI ST-elevation myocardial infarction

PCI percutaneous coronary intervention

ASA aspirin

ACEI/ARB angiotensin-converting enzyme inhibitors/angiotensin receptor blocking agents

Table 2

Reasons provided for not initiating beta-blockers at discharge (n=128)

	# of subjects (%)
Sinus node disease/bradycardia	31 (24.2%)
Hypotension	26 (20.3%)
Airway disease	19 (14.8%)
Congestive heart failure	15 (11.7%)
Unknown	15 (11.7%)
Other	9 (7.0%)
Illicit drug use	8 (6.2%)
AV Block	7 (5.5%)
Depressed mood	1 (0.8%)
Lightheadedness	1 (0.8%)
Fatigue	1 (0.8%)

Table 3

Baseline univariate predictors of no or very low dose beta-blocker treatment at discharge and 3-week follow-up. The odds ratio (OR) and 95% confidence intervals (CI) are listed with the specific criterion indicating use of very low dose beta-blockers.

Variable	Criterion	DISCHARGE (N=1851)			3-week FOLLOW-UP (N=1450)		
		OR	95% CI	p value	OR	95% CI	p value
ACEI	No	1.70	1.36–2.11	<0.0001	1.53	1.17–1.99	0.002
HTN	No	1.60	1.29–1.98	<0.0001	1.78	1.38–2.31	<0.0001
PCI	Yes	1.43	1.77–1.16	0.001	1.31	1.69–1.02	0.04
LVEF	10% Decrease	1.13	1.23–1.03	0.008	1.04	0.93–1.16	0.48
Ethnicity	Hispanic/Latino	1.70	1.14–2.52	0.009	1.19	0.68–2.08	0.54
Gender	Female	1.30	1.04–1.63	0.02	1.08	0.81–1.42	0.61
DM	No	1.26	0.99–1.59	0.06	1.24	0.93–1.64	0.15
Age	10 year increase	1.07	1.00–1.16	0.07	1.07	0.97–1.18	0.16
Statin*	No	1.36	0.98–1.90	0.07	0.87	0.55–1.38	0.56
Aspirin	No	1.22	0.83–1.80	0.32	1.14	0.70–1.85	0.59
HC	No	1.08	0.87–1.33	0.48	1.18	0.91–1.51	0.21
White*	No	1.09	0.84–1.41	0.51	0.63	0.43–0.91	0.01
Lytic	Yes	1.09	0.78–1.53	0.60	1.09	0.72–1.65	0.69
STEMI*	No	1.03	0.84–1.27	0.76	0.96	0.75–1.24	0.77

*Variable changes direction from increased likelihood of very low dose beta-blocker treatment to decreased probability of very low dose beta-blocker treatment

ACEI angiotensin-converting enzyme inhibitor

HTN hypertension

PCI percutaneous coronary intervention

LVEF left ventricular ejection fraction

DM diabetes mellitus

HC hypercholesterolemia

STEMI ST-elevation myocardial infarction

Table 4

Multivariable predictors of no beta-blocker or very low dose beta-blocker therapy at discharge (N=1680)

Variable	Criterion	Discharge (n=1,680)			3-week Follow-Up (n=1,027)		
		OR	95% CI	p value	OR	95% CI	p value
ACEI	No	1.73	1.36–2.20	<0.0001	1.87	1.39–2.52	<0.0001
Age	Decade Increase	1.12	1.02–1.22	0.01	1.6	1.20–2.14	0.002
Aspirin	No	1.38	0.90–2.12	0.14	1.18	1.06–1.32	0.003
DM	No	1.22	0.94–1.60	0.14	1.38	0.80–2.38	0.24
Ethnicity	Hispanic/Latino	1.97	1.27–3.06	0.003	1.1	0.79–1.52	0.58
Gender	Female	1.36	1.06–1.75	0.02	1.8	0.97–3.34	0.06
HC	Yes	1.09	0.86–1.37	0.49	1.11	0.82–1.50	0.5
HTN	No	1.64	1.28–2.10	<0.001	1.03	0.78–1.36	0.86
LVEF	10% Decrease	1.18	1.08–1.30	<0.001	1.08	0.96–1.22	0.17
Lytic	Yes	1.28	0.89–1.86	0.19	1.16	0.73–1.84	0.52
PCI	Yes	1.6	1.24–2.06	<0.001	1.46	1.07–1.99	0.02
Statin	No	1.32	0.92–1.91	0.13	1.12	0.68–1.86	0.65
STEMI	No	1.16	0.90–1.50	0.24	1.12	0.81–1.53	0.49
White	No	1.05	0.79–1.40	0.72	1.59	1.05–2.42	0.03

Abbreviations as in Table 3.