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Antiarrhythmic Drug Therapy for Sustained Ventricular Arrhythmias Complicating Acute Myocardial Infarction

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Abstract

Objective—Few data exist to guide antiarrhythmic drug therapy for sustained ventricular tachycardia (VT)/ventricular fibrillation (VF) after acute myocardial infarction (MI). The objective of this analysis was to describe survival of patients with sustained VT/VF post-MI according to antiarrhythmic drug treatment.

Design & Setting—We conducted a retrospective analysis of ST-segment elevation MI patients with sustained VT/VF in GUSTO IIB and III and compared all-cause death in patients receiving amiodarone, lidocaine, or no antiarrhythmic. We used Cox proportional hazards modeling and inverse weighted estimators to adjust for baseline characteristics, beta-blocker use, and propensity to receive antiarrhythmics. Due to non-proportional hazards for death in early follow-up (0–3 hours after sustained VT/VF) compared with later follow-up (>3 hours), we analyzed all-cause mortality using time-specific hazards.

Patients & Interventions—Among 19,190 acute MI patients, 1126 (5.9%) developed sustained VT/VF and met the inclusion criteria. Patients received lidocaine (n=664, 59.0%), amiodarone (n=50, 4.4%), both (n=110, 9.8%), or no antiarrhythmic (n=302, 26.8%).

Results—In the first 3 hours after VT/VF, amiodarone (adjusted HR 0.39, 95% CI 0.21–0.71) and lidocaine (adjusted HR 0.72, 95% CI 0.53–0.96) were associated with a lower hazard of death—likely evidence of survivor bias. Among patients who survived 3 hours, amiodarone was

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associated with increased mortality at 30 days (adjusted HR 1.71, 95% CI 1.02–2.86) and 6 months (adjusted HR 1.96, 95% CI 1.21–3.16) but lidocaine was not at 30 days (adjusted HR 1.19, 95% CI 0.77–1.82) and 6 months (adjusted HR 1.10, 95% CI 0.73–1.66).

Conclusion—Among patients with acute MI complicated by sustained VT/VF who survive 3 hours, amiodarone, but not lidocaine, is associated with an increased risk of death; reinforcing the need for randomized trials in this population.

Keywords

ventricular arrhythmia; antiarrhythmic drug therapy; clinical trials; acute coronary syndrome; ventricular tachycardia; ventricular fibrillation

INTRODUCTION

Sustained ventricular arrhythmias remain a potentially lethal complication of acute myocardial infarction (MI),^{1,2} occurring in 5–10% of all patients who survive to hospitalization. Despite their frequency, however, there are few data to guide acute antiarrhythmic drug therapy for sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) in the setting of an acute MI. Prior analyses have suggested that prophylactic lidocaine may be associated with increased mortality and that amiodarone may be beneficial for the treatment of cardiac arrest due to VT/VF complicating acute MI.^{3,4}

Although there are no randomized trial data demonstrating the efficacy and safety of these agents for treatment of sustained VT/VF, antiarrhythmic drugs are commonly used when VT/VF occurs despite beta-blockade and revascularization. There are no large studies that compare commonly used antiarrhythmic drugs in the setting of acute MI for treatment of sustained VT/VF.⁵ The objective of this analysis is to describe the survival of patients with sustained VT/VF after acute MI according to antiarrhythmic drug treatment.

METHODS

Study Population

The details of the Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes (GUSTO) IIB and GUSTO III randomized controlled trials have been previously published.^{6,7} GUSTO IIB enrollment started on May 19, 1994, and ended on October 17, 1995. GUSTO III enrolled patients between October 13, 1995 and January 13, 1997. Briefly, GUSTO IIB compared recombinant hirudin and heparin in 12,142 patients with ST-segment elevation (n=4131) and non-ST-segment elevation acute coronary syndromes (ACS). GUSTO III compared reteplase and alteplase in 15,059 patients with ST-segment elevation MI. Data from GUSTO IIB and GUSTO III were merged in a common database. For the purpose of this analysis, patients with sustained VT or VF were selected to form the study cohort. Patients were further subclassified according to the antiarrhythmic drug therapy they received: lidocaine, amiodarone, both, or none. Patients who received prophylactic lidocaine (n=81) or antiarrhythmic drug therapy other than lidocaine or amiodarone (sotalol n=49; class I agents n=141) were excluded from this analysis.

Definitions

Sustained VT was defined as a regular wide-complex tachycardia of ventricular origin lasting 30 seconds or causing hemodynamic compromise requiring immediate cardioversion.⁸ VF was defined as irregular waveforms of varying shape and amplitude, without discrete QRS or T waves, resulting in acute hemodynamic compromise.⁸ Only patients with

sustained VT/VF occurring after randomization were included in this analysis. The primary outcome for this analysis was total mortality

Study Design & Statistical Analysis

The study design is shown in Figure 1. Baseline characteristics were compared between those patients with sustained VT/VF according to antiarrhythmic drug treatment: lidocaine, amiodarone, both, or none. Continuous variables were compared using the non-parametric Kruskal-Wallis test and categorical variables were compared using the Pearson's chi-square or Fisher's exact test. Continuous variables are expressed as the mean plus/minus standard deviation; discrete variables are expressed as number and percentage.

Our principle goal was to assess survival following a ventricular arrhythmia according to antiarrhythmic drug strategy. Extensive work has been done to identify clinical risk factors for 30-day mortality from GUSTO-I.⁹ Our analyses adjusted for significant clinical factors from the GUSTO-I model which were available for age, weight, systolic blood pressure, histories of coronary artery bypass graft surgery, diabetes, hypertension, MI, and/or cerebral vascular disease, heart rate, MI location, smoking, Killip class, and the interaction of Killip class with age, as well as creatinine clearance, beta-blocker therapy at enrollment, beta-blocker therapy in-hospital, and the type of ventricular arrhythmia (sustained VT versus VF). We examined the relationship between antiarrhythmic therapy and mortality at 30 days and 6 months post-arrhythmia. For each interval, an unadjusted, adjusted, and inverse propensity-weighted Cox model were fit. Continuous variables were tested for linearity by comparing the fit of linear models with restricted cubic spline models. When a non-linear relationship was identified, appropriate transformations were applied. In these cases, the variables were included in our models using linear splines.

Inspection of the hazard for each factor over time revealed non-proportional hazards for both antiarrhythmic therapies. It is also likely that deaths occurring very soon after the arrhythmia would be counted in the group of patients labeled as having no antiarrhythmic treatment, explaining, at least in part, the non-proportionality. To address both the non-proportional hazards and survivor bias, hazard functions for 30-day and 6-month mortality were examined with change points at 3 days, 2 days, 1 day, 12 hours, 6 hours, and 3 hours. Ultimately, a 3 hour dichotomy provided the greatest -2 log likelihood chi-square and satisfaction of the proportional hazards assumption. Models were generated including 2 time-dependent indicators for each treatment, 1 for the early (0–3 hours) and 1 for the later (>3 hours) periods. This method utilizes all patients when estimating the hazard ratio for the first 3 hours. But only those who survive to 3 hours influence the estimation of the hazard ratio for the later period, thus minimizing the potential effect of survivor bias during this period.

For the propensity weighted models, generalized logistic regression with the same baseline covariates as above was used to compute each individual's propensity to receive the antiarrhythmic treatment (none, lidocaine only, amiodarone only, or both) actually given. The interaction of quintiles of the propensity score with each factor in the logistic model was used to evaluate the ability of the score to create balance of covariates between the treatment arms. A Cox model was run, weighting individuals by the inverse of this propensity score. The use of inverse propensity weighting has been shown to lead to consistent results when the models are correctly specified.^{10–12}

Missing values of baseline characteristics were imputed using multiple imputation techniques. Five independent datasets were generated with missing values imputed using Markov chain Monte Carlo methods with the Jeffreys prior. All analyses were conducted using SAS statistical software version 9 (SAS Inc., Cary, NC). A P value of 0.05 was

considered statistically significant, bearing in mind the exploratory nature of this study. The study protocol was reviewed and approved by the Duke University Medical Center Institutional Review Board.

RESULTS

Baseline Characteristics

Among the 19,190 ST-segment elevation MI patients enrolled in GUSTO IIB and III, we identified 1126 patients (5.9%) who developed sustained VT/VF and met the inclusion criteria for this analysis. Sustained VT occurred in 36.8% (n=414). VF occurred in 48.4% (n=545), and 167 patients had both sustained VT and VF (14.8%). Patients were treated with lidocaine (n=664, 59.0%), amiodarone (n=50, 4.4%), both (n=110, 9.8%), or no antiarrhythmic drug therapy (n=302, 26.8%). As shown in Table 1, patients who received amiodarone were older, had a higher resting heart rate at admission, were more likely to have hypertension, and a history of prior MI. Beta-blocker use at enrollment was not significantly different across the 4 groups, however, in-hospital beta-blocker use was higher in those patients who did not receive amiodarone. The rates of in-hospital beta-blocker use were 59% (n=179/302) for those not receiving antiarrhythmic drug therapy, 62% (n=479/772) for those receiving lidocaine, and 48% (n=77/160) for those receiving amiodarone. Those patients who received both amiodarone and lidocaine had a lower left ventricular ejection fraction. Overall, 14% (n=161/1125) of the cohort had atrial fibrillation and 61% (n=691/1126) underwent cardioversion or defibrillation during the hospital course.

Outcomes at 3 Hours

Overall survival according to antiarrhythmic drug treatment is shown in Figure 2A and 2B. At 3 hours post-arrhythmia, the overall Kaplan-Meier survival rate was 81.7%. In patients treated with amiodarone, the 3-hour survival rate was 88.1% compared with 80.6% among those not receiving amiodarone (P=0.294). In patients treated with lidocaine, the 3-hour survival rate was 85.1% compared with 74.1% among those not receiving lidocaine (P<0.001). Due to differences in the proportional hazard for death early after sustained VT/VF, we assessed all-cause mortality using time-dependent treatment effects. In the first 3 hours after sustained VT/VF, amiodarone (unadjusted hazard ratio [HR] 0.59, 95% confidence interval [CI] 0.37–0.95) and lidocaine (unadjusted HR 0.55, 95% CI 0.42–0.72) were both associated with better survival (Figure 3). After adjusting for baseline patient characteristics,⁹ baseline beta-blocker, in-hospital beta-blocker use, and creatinine clearance, amiodarone was associated with better survival (HR 0.43, 95% CI 0.27–0.70). After the same adjustment, lidocaine was also associated with a trend towards better survival (HR 0.75, 95% CI 0.56–1.00). After adjusting for the propensity to receive antiarrhythmic drug therapy based on baseline covariates, baseline beta-blocker status, in-hospital beta-blocker use, and creatinine clearance, amiodarone (HR 0.39, 95% CI 0.21–0.71) and lidocaine (HR 0.72, 95% CI 0.53–0.96) were both associated with better survival.

Medications at Discharge

Pharmacotherapy at discharge according to in-hospital treatment is shown in Table 2. Overall, 40% of patients were discharged on a beta-blocker and 29% were discharged on an ACE-inhibitor. None of the patients in this cohort were discharged on Vaughan-Williams class I antiarrhythmics or sotalol. Patients who received amiodarone were more likely to be discharged on amiodarone and were less likely to receive beta-blockers. Only 23% of the amiodarone-treated patients were discharged on oral beta-blockade (n=36/160).

Outcomes at 30 Days

At 30 days, the Kaplan-Meier survival estimate was 69.5% among all patients. After excluding deaths within the first 3 hours, amiodarone was associated with a significantly higher risk of death between 3 hours and 30 days (unadjusted HR 2.71, 95% CI 1.88–3.89) (Figure 3). On the other hand, there was no evidence of a higher or lower risk of death with lidocaine when we excluded deaths in the first 3 hours following the arrhythmia (unadjusted HR 1.07, 95% CI 0.74–1.57). After accounting for baseline patient characteristics, baseline beta-blocker use, in-hospital beta-blocker use, and creatinine clearance, amiodarone was no longer associated with higher risk between 3 hours and 30 days (HR 1.27, 95% CI 0.86–1.88). However, in the propensity-weighted model, amiodarone was associated with a significantly higher risk of death between 3 hours and 30 days (HR 1.71, 95% CI 1.02–2.86). In the adjusted and propensity weighted analysis, lidocaine was again not associated with a higher or lower risk of death between 3 hours and 30 days (Figure 3).

In-hospital beta-blocker use was the only post-baseline variable included in the adjusted analyses. Due to its post-baseline status, we conducted a sensitivity analysis without the adjustment for this covariate. As shown in Figure 3, the results with or without adjustment for in-hospital beta-blocker use were similar.

Outcomes at 6 Months

When examining long-term outcomes to 6 months after arrhythmia, we observed similar findings to those at 30 days. At 6 months of follow-up, the Kaplan-Meier estimated survival probability of those receiving amiodarone was 47.5% compared with 69.6% among those not receiving amiodarone ($P<0.001$). At 6 months, patients who received lidocaine had an estimated survival of 70.1% compared with 59.4% among those without lidocaine ($P<0.001$). After excluding deaths within 3 hours, amiodarone was associated with higher mortality (unadjusted HR 3.09, 95% CI 2.18–4.36) at 6 months and lidocaine was not (unadjusted HR 1.04, 95% CI 0.72–1.49). These relationships remained unchanged in the adjusted model for amiodarone (HR 1.48, 95% CI 1.02–2.15) and lidocaine (HR 1.14, 95% CI 0.78–1.66). Finally, the propensity-weighted model also demonstrated the same relationship for both amiodarone (HR 1.96, 95% CI 1.21–3.16) and lidocaine (HR 1.10, 95% CI 0.73–1.66) for mortality at 6 months.

DISCUSSION

Among patients with acute MI complicated by sustained VT/VF, early antiarrhythmic drug therapy with amiodarone or lidocaine was associated with better survival. Whether this was due to effects of the drugs or survival bias will require further investigation. Excluding these early deaths, however, amiodarone use was associated with a higher risk of death at 30 days and 6 months while lidocaine use was not associated with benefit or harm. Given the observational and retrospective nature of our study, these results should be considered hypothesis generating. Nonetheless, they raise the possibility that lidocaine might be preferred over amiodarone for the treatment of sustained VT/VF complicating acute myocardial infarction.

Sustained ventricular arrhythmias complicate 5–10% of all acute MIs.^{2,13,14} These arrhythmias remain a life-threatening complication of MI, with an in-hospital mortality rate of 16–20%, despite advances in reperfusion and pharmacotherapy.^{2,14} Patients with sustained VT/VF are often treated with antiarrhythmic drugs, most commonly amiodarone or lidocaine. Consistent with widespread practice patterns, the American College of Cardiology (ACC)/American Heart Association (AHA)/European Society of Cardiology (ESC) 2006 Guidelines for the Management of Ventricular Arrhythmias give amiodarone a

Grade IIA recommendation for the treatment of hemodynamically unstable sustained VT complicating acute MI and a Grade I recommendation for the treatment of polymorphic VT complicating acute MI—despite a paucity of evidence (Class C). Similarly, intravenous lidocaine has a Grade IIB recommendation for the treatment of sustained VT and polymorphic VT in the setting of ACS.⁵

Prior research has suggested that prophylactic lidocaine may be associated with increased mortality when used in patients with acute MI.³ Prophylactic lidocaine decreases the risk of VF; however, it may also lead to increased mortality via a vis increased risks of sinoatrial block and other conduction disorders.^{3,15} Subsequent to these findings, the guidelines have recommended against the use of prophylactic lidocaine, and some clinicians have suggested that its use (prophylactic and therapeutic) should be avoided altogether in acute MI patients, as reflected by the Grade IIB recommendations in the ACC/AHA/ESC ST-segment elevation MI guidelines.¹⁶ However, use of lidocaine in response to a ventricular arrhythmia is different from prophylactic administration. In this analysis, we found no evidence of higher (or lower) mortality when lidocaine was given for treatment of an acute sustained ventricular arrhythmia.

Despite widespread use of amiodarone for patients with sustained VT/VF in-hospital during an acute MI, the majority of evidence for amiodarone is extrapolated data from studies of out-of-hospital cardiac arrest.⁴ Surprisingly, there are no large observational studies and no randomized clinical trials comparing these agents in this patient population. Although amiodarone appears to be neutral with respect to all-cause mortality in several clinical settings, there are significant risks and adverse effects associated with chronic amiodarone use.¹⁷ As cardiologists well know, most trials of chronic antiarrhythmic therapy after acute MI have demonstrated harm, including significant toxicity and increased mortality.^{18–20} However, acute antiarrhythmic drug therapy has a very different therapeutic goal—to suppress recurrent ventricular arrhythmias during the period of greatest proarrhythmic risk. Since this approach avoids chronic drug exposure, acute therapy may spare the patient from long-term disadvantage. Recent work has highlighted the predominance of both arrhythmic and mortality risks early after sustained VT/VF.¹³ Therefore, acute antiarrhythmic therapy directed at preventing recurrent VT/VF and early mortality may bridge the patient through the period of greatest risk while avoiding end-organ toxicities and other potential harms. In our analysis, the association between amiodarone pharmacotherapy and increased mortality at 6 months could be explained by residual adverse effects from amiodarone or it could reflect persistent confounding (eg, sicker patients received amiodarone). Data from the European and Canadian Amiodarone Myocardial Infarction Trials (EMIAT & CAMIAT) suggest an important interaction between amiodarone and beta-blockers, such that the benefit of amiodarone may be restricted to those receiving concomitant beta-blockade. In light of these findings, it is important to note that patients who received acute amiodarone therapy were less likely to be discharged on beta-blockers.²¹ Any retrospective analysis is subject to selection and survival bias, however, other analyses of amiodarone use in the setting of ACS have also uncovered similar mortality risks.²²

Critics opposed to the use of antiarrhythmic drugs point out the numerous antiarrhythmic drug trials associated with increased mortality in an ischemic heart disease population,^{18,20} however, these studies did not examine short-term antiarrhythmic use in the setting of acute MI, nor did they study patients with refractory sustained arrhythmias. The patient who develops recurrent sustained VT/VF, despite treatments directed towards the underlying ischemia (beta-blockade and revascularization), oftentimes requires membrane-active antiarrhythmic drug therapy. Unfortunately, there are few data to help guide this treatment decision. There are more than 1 million MIs each year in the United States and, by extension, between 50,000 and 100,000 patients who experience sustained VT/VF after

arrival to the hospital. Furthermore, patients who present with sustained VT/VF are usually not included in randomized clinical trials and the incidence of these arrhythmias in clinical trials may under-represent general practice. There is a paucity of evidence in the area of antiarrhythmic drug treatment of sustained VT/VF complicating acute MI. Accordingly, randomized trial data of acute antiarrhythmic drug therapy for sustained VT/VF complicating ACS would help guide these common treatment decisions.

Our study is a retrospective analysis of prospectively collected data and is limited by potential selection bias, survival bias, and confounding. To address these limitations, we employed (1) temporal adjustment for changes in hazards, (2) multivariable adjustment, and (3) propensity scoring to reduce the effect of confounders. Results using multivariable models or propensity scores should be interpreted with caution, as there are likely numerous unmeasured factors associated with antiarrhythmic drug use in this population. For example, patients treated with amiodarone may have been sicker, as evidenced by the higher rate of in-hospital atrial fibrillation. Therefore, despite multiple methods of adjustment, we cannot exclude the possibility that the excess mortality observed with amiodarone, may have due to differences between patient groups. The relatively small number of amiodarone-treated patients may also have limited our analysis. The observed associations need to be evaluated in larger numbers of randomized patients. Our analysis was also limited by the lack of baseline left ventricular ejection fraction or data regarding the dose or duration of antiarrhythmic therapy. Finally, despite our exclusion of deaths within 3 hours and the use of time-dependent hazards, survival bias may have continued to affect the observed associations.

There have been improvements in the care of patients with acute MI since the conduct of the GUSTO IIB and III trials that may limit the generalizability of these results to current practice. This fact, however, illustrates the limitations of our current understanding of antiarrhythmic drug therapy in this population and underscores the need for contemporary clinical trials. Despite these limitations, our study represents the largest analysis of antiarrhythmic drug therapy in response to ventricular arrhythmia following acute MI.

CONCLUSIONS

In the acute period after VT/VF complicating acute MI, both amiodarone and lidocaine are associated with better survival. It is likely that this is at least partially due to survival bias. However, following this early phase, amiodarone, but not lidocaine, is associated with worse outcomes. These results are hypothesis generating and underscore the need for randomized clinical trial data to guide antiarrhythmic drug therapy in patients with acute MI complicated by VT/VF.

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Figure 1.
Study design.

Figure 2. Survival at 3 hours (inset) and 30 days. **A.** Survival at 3 hours (inset) and 30 days according to amiodarone pharmacotherapy. **B.** Survival at 3 hours (inset) and 30 days according to lidocaine pharmacotherapy.

Figure 3.

Risk of all-cause death at 30 days in patients with sustained VT/VF according to treatment with amiodarone or lidocaine. Shown are hazard ratios with 95% confidence intervals for all-cause death at 30 days according to treatment with amiodarone or lidocaine. Due to asymmetric hazards, these ratios are shown for early (0–3 hours after sustained VT/VF) and late (>3 hours) antiarrhythmic drug therapy.

Table 1

Patient characteristics according antiarrhythmic drug therapy

	None (n=302)	Amiodarone (n=50)	Lidocaine (n=664)	Both (n=110)	P Value [*]
Age, y	64.8±12.3	67.8±9.8	61.5±12.6	64.0±11.4	<0.001
Female, %	26.8	30.0	26.1	20.9	0.571
White, %	96.0	100	92.6	90.0	0.020
Heart rate, bpm	79.1±21.4	84.1±21.3	76.0±19.1	78.4±19.3	0.031
Systolic BP, mm Hg	129±27	132±28	126±26	125±23	0.171
Creatinine clearance, mL/min	72±32	69±36	79±31	74±28	<0.001
LVEF	48±17	46±19	47±14	40±13	0.033
Killip class III/IV	20 (7)	3 (6)	32 (5)	11 (10)	0.186
MI location, No. (%)					0.033
Anterior	123 (42)	25 (52)	263 (41)	58 (55)	
Inferior	131 (44)	17 (35)	264 (41)	35 (33)	
Other	41 (14)	6 (13)	122 (19)	12 (11)	
Atrial fibrillation in-hospital, No. (%)	35 (12)	21 (42)	75 (11)	30 (27)	<0.001
Diabetes, No. (%)	45 (15)	11 (22)	94 (14)	21 (19)	0.308
Hypertension, No. (%)	107 (35)	29 (58)	238 (36)	46 (42)	0.011
Prior MI, No. (%)	54 (18)	19 (38)	111 (17)	29 (26)	<0.001
Cerebrovascular disease, No. (%)	13 (4)	3 (6)	19 (3)	4 (4)	0.384
Heart failure, No. (%)	9 (3)	6 (12)	22 (3)	4 (4)	0.040
PCI, No. (%)	6 (2)	1 (2)	24 (4)	5 (5)	0.420
CABG, No. (%)	14 (5)	1 (2)	31 (5)	5 (5)	0.949
History of smoking, No. (%)					0.051
Never	84 (29)	18 (36)	163 (25)	33 (31)	
Former	84 (29)	18 (36)	182 (28)	38 (36)	
Current	127 (43)	14 (28)	304 (47)	36 (34)	
Beta-blocker use at baseline, No. (%)	50 (17)	12 (25)	119 (18)	20 (18)	0.616
Beta-blocker use in-hospital, No. (%)	179 (59)	27 (54)	429 (65)	50 (46)	0.001
Sustained ventricular arrhythmia, No. (%)					<0.001
Sustained VT only	95 (32)	17 (34)	249 (38)	53 (48)	

	None (n=302)	Amlodarone (n=50)	Lidocaine (n=664)	Both (n=110)	P Value*
Sustained VF only	182 (60)	24 (48)	320 (48)	19 (17)	
Both	25 (8)	9 (18)	95 (14)	38 (35)	

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

* P values shown are for any association. BP=blood pressure; bpm=beats per minute; CABG=coronary artery bypass grafting; LVEF=left ventricular ejection fraction; MI=myocardial infarction; PCI=percutaneous coronary intervention; VF=ventricular fibrillation; VT=ventricular tachycardia.

Table 2

Pharmacotherapy at Hospital Discharge

Medication	None (n=302)	Amiodarone (n=50)	Lidocaine (n=664)	Both (n=110)	P-value
ACE-inhibitors (%)	81 (27)	16 (32)	188 (28)	40 (36)	0.265
Beta-blockers	126 (42)	13 (26)	290 (44)	23 (21)	<0.001
Antiarrhythmics*					
Amiodarone	0 (0)	18 (36)	0 (0)	33 (3)	<0.001*
Sotalol	0 (0)	0 (0)	0 (0)	0 (0)	N/A
Class I agents	0 (0)	0 (0)	0 (0)	0 (0)	N/A

* Patients who received prophylactic lidocaine (n=81) or antiarrhythmic drug therapy other than lidocaine or amiodarone (sotalol n=49; class I agents n=141) were excluded from this analysis.