# Twenty-five year trends (1986-2011) in hospital incidence and case-fatality rates of ventricular tachycardia and ventricular fibrillation complicating acute myocardial infarction

Hoang V. Tran, MD, MPH,[a] Arlene S. Ash, PhD,[a] Joel M. Gore, MD,[b] Chad E. Darling, MD, MS,[c] Catarina I. Kiefe, PhD, MD,[a] and Robert J. Goldberg, PhD[a] *Worcester, MA*

**Background** Long-term trends in the incidence rates (IRs) and hospital case-fatality rates (CFRs) of ventricular tachycardia (VT) and ventricular fibrillation (VF) among patients hospitalized with acute myocardial infarction (AMI) have not been recently examined.

**Methods** We used data from 11,825 patients hospitalized with AMI at all 11 medical centers in central Massachusetts on a biennial basis between 1986 and 2011. Multivariable adjusted logistic regression modeling was used to examine trends in hospital IRs and CFRs of VT and VF complicating AMI.

**Results** The median age of the study population was 71 years, 57.9% were men, and 94.7% were white. The hospital IRs declined from 14.3% in 1986/1988 to 10.5% in 2009/2011 for VT and from 8.2% to 1.7% for VF. The in-hospital CFRs declined from 27.7% to 6.9% for VT and from 49.6% to 36.0% for VF between 1986/1988 and 2009/2011, respectively. The IRs of both early (<48 hours) and late VT and VF declined over time, with greater declines in those of late VT and VF. The incidence rates of VT declined similarly for patients with either an ST-segment elevation myocardial infarction (STEMI) or non-STEMI, whereas they only declined in those with VF and a STEMI.

**Conclusions** The hospital IRs and CHRs of VT and VF complicating AMI have declined over time, likely because of changes in acute monitoring and treatment practices. Despite these encouraging trends, efforts remain needed to identify patients at risk for these serious ventricular arrhythmias so that preventive and treatment strategies might be implemented as necessary. (Am Heart J 2019;208:1-10.)

Ventricular tachycardia (VT) and ventricular fibrillation (VF) frequently complicate the clinical course of patients hospitalized with an acute myocardial infarction (AMI).[1,2] The development of these serious ventricular arrhythmias in patients hospitalized with an AMI is often associated with a larger infarct, ventricular wall dyskinesia and dysfunction, more extensive underlying coronary artery disease, and ST-segment elevation myocardial infarction (STEMI).[3-6] The development of VT and VF in patients hospitalized with AMI is associated with increased in-hospital and postdischarge morbidity and mortality.[7,8]

National data on either the incidence rates (IRs) or case-fatality rates (CFRs) of VT and VF complicating AMI in the United States are not presently available. In a medical records linkage study of 2,280 patients who were admitted to all hospitals in Olmsted County, Minnesota, for coronary heart disease between 1979 and 1998, the annual IRs of sustained VT remained stable at 1.8%, whereas the IRs of VF declined from 7.7% in the initial study years to 5.0% during the most recent years under study.[9] However, this study was limited by its small sample size and by the lack of more contemporary data. A number of changes in the more optimal management of patients hospitalized with acute coronary disease during recent years may have led to favorable changes in the magnitude and/or outcomes of serious ventricular arrhythmias among patients hospitalized for an AMI.[10,11]

Using data from a population-based surveillance study of patients hospitalized with confirmed AMI at all central Massachusetts medical centers, we examined 25-year trends (1986-2011) in the hospital IRs and CFRs of VT and VF, and compared differences in the in-hospital CFRs of

*From the [a]Department of Quantitative Health Sciences, University of Massachusetts Medical School, Worcester, MA, [b]Department of Medicine, University of Massachusetts Medical School, Worcester, MA, and [c]Department of Emergency Medicine, University of Massachusetts Medical School, Worcester, MA.*

*Submitted June 7, 2018; accepted October 6, 2018.*

*Reprint requests: Robert J. Goldberg, PhD, Department of Quantitative Health Sciences, University of Massachusetts Medical School, 368 Plantation St, Worcester, MA 01605.*
*E-mail: Robert.Goldberg@umassmed.edu*

*0002-8703*



*https://doi.org/10.1016/j.ahj.2018.10.007*

patients who developed with those who did not develop VT or VF complicating their AMI.

## Methods

### Study design and data collection methods

Data from the Worcester Heart Attack Study, an ongoing population-based investigation that is examining long-term trends in the incidence, in-hospital, and postdischarge CFRs of AMI among residents of the Worcester, Massachusetts, metropolitan area were used for this investigation. The details of this study have been described previously.[12-16] In brief, the medical records of residents of central Massachusetts hospitalized for possible AMI at the 11 medical centers serving residents of this large central New England metropolitan area were individually reviewed. The diagnosis of AMI was independently validated according to criteria developed by the World Health Organization with further subclassification into those with an ST-segment elevation AMI (STEMI) or non–ST-segment elevation AMI (NSTEMI).[17,18] This investigation was approved by the Institutional Review Board at the University of Massachusetts Medical School.

Trained nurses and physicians abstracted demographic and clinical data from the medical records of residents of the Worcester metropolitan area hospitalized with a confirmed AMI. Quality control activities were routinely conducted on all nurse and physician data abstractors. Abstracted information included patient's age, sex, medical history, physiologic factors, laboratory test results at the time of hospital admission, and length of hospital stay. Information about the in-hospital use of important cardiac medications, coronary angiography, percutaneous coronary intervention (PCI), and coronary artery bypass graft (CABG) surgery was collected. Development of several significant clinical complications (eg, atrial fibrillation, cardiogenic shock, stroke, heart failure, survival status) during the patient's index hospitalization was defined according to standardized criteria.[18-21]

### Study population

Among the 12,804 residents of the Worcester metropolitan area who were hospitalized with an independently confirmed AMI on an approximate biennial basis between 1986 and 2011, we excluded patients with missing data on age (n = 281), race (n = 476), serum potassium levels (n = 212) and length of hospital stay (n = 10). The final study sample consisted of 11,825 patients with an AMI.

### Ventricular arrhythmias

The occurrence of VT and VF was based on physicians' progress notes. To reduce the possible misclassification of VT and VF due to the underreported occurrence of these arrhythmias in physicians' notes, the study research physicians also reviewed patients' ECG strips in their hospital medical records. *Ventricular tachycardia* was defined as a cardiac arrhythmia of 3 or more consecutive complexes originating in the ventricles at a rate of greater than 100 beats/min (cycle length less than 600 milliseconds).[2] Information was also collected about the date of initial VT occurrence since the time of hospital admission. *Ventricular fibrillation* was defined as a rapid, usually more than 300 beats/ min (cycle length 200 millisecond or less), grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.[2]

Following definitions used in prior investigations,[9,14,22] we further classified VT and VF as either occurring early (within 48 hours after hospital admission) or late (after the first 48 hours) and primary (occurring without concomitant heart failure or cardiogenic shock) or secondary to concurrent heart failure or cardiogenic shock. For patients who had multiple episodes of VT (582/1,731 patients, 33.6%) and VF (158/574 patients, 27.5%), only the first episode was counted. We classified the 348 patients who developed both VT and VF as having VF because VF is more clinically serious than VT.

### Statistical analysis

Patient characteristics, including demographic factors, clinical presentation, physiologic findings, and laboratory test results, were compared between patients who developed VT or VF and those who did not using $\chi^2$ tests for categorical variables and the *t* test or Kruskal-Wallis test for continuous variables. Changes in these characteristics between our respective comparison groups were also examined across study years using $\chi^2$ tests for trends.

The hospital IRs (eg, frequencies) of VT and VF complicating AMI were calculated as the proportion of new cases of VT and VF which occurred among all patients hospitalized with AMI.[22] The in-hospital CFRs for each condition were calculated as the number of in-hospital deaths/total number of cases with a particular arrhythmia.[23] The IRs and CFRs were graphed in a continuous manner between 1986 and 2011 using locally weighted least square smoothing.[24]

We controlled for the effects of changes in patients' demographic characteristics, clinical presentation, and medical therapies and coronary reperfusion practices on trends in hospital IRs and CFRs using multivariable adjusted logistic regression modeling. Study year was treated as an integer-valued variable in examining linear trends in these end points. Potentially confounding factors were identified based on differences in the baseline characteristics of hospitalized patients according to the presence or absence of VT or VF using a *P* value of <.05 as a cutoff. These variables were successively introduced into the multivariable adjusted logistic regression model in blocks to a full model. Because length of hospital stay may affect the possibility of capturing VT or VF during the patient's acute hospitalization, or serve as a proxy for disease severity, length of stay was also

**Table I.** Baseline characteristics of patients admitted to the hospital for an AMI according to the presence of VT or VF

| Characteristics | No VT/VF (n = 9520) | VT (n = 1731) | VF (n = 574) | *P*1 | *P*2 |
|---|---|---|---|---|---|
| Age (median [IQR], y) | 72 [60-81] | 70 [58-79] | 68 [58-77] | <.001 | <.001 |
| Age (y, %) | | | | <.001 | <.001 |
| <55 | 15.3 | 19.5 | 19.0 | | |
| 55-64 | 18.3 | 18.9 | 20.4 | | |
| 65-74 | 24.5 | 25.0 | 28.9 | | |
| ≥75 | 42.0 | 36.6 | 31.7 | | |
| Men (%) | 55.7 | 66.7 | 67.8 | <.001 | <.001 |
| White (%) | 94.4 | 96.0 | 95.1 | .010 | .49 |
| History of disease (%) | | | | | |
| Coronary artery disease | 36.5 | 36.1 | 35.2 | .75 | .53 |
| Heart failure | 20.8 | 21.1 | 14.8 | .83 | .001 |
| Diabetes | 32.8 | 27.8 | 23.9 | <.001 | <.001 |
| Hypertension | 65.2 | 60.2 | 57.5 | <.001 | <.001 |
| Angina | 21.4 | 20.8 | 17.3 | .56 | .018 |
| Stroke/transient ischemic attack | 12.6 | 10.3 | 8.7 | .009 | .006 |
| Admission potassium (median [IQR], mmol/L) | 4.2 [3.9-4.6] | 4.2 [3.8-4.6] | 4.0 [3.5-4.4] | .017 | <.001 |
| Length of stay (median [IQR], d) | 5 [3-9] | 7 [4-11] | 7 [2-13] | <.001 | <.001 |
| In-hospital complications (%) | | | | | |
| Atrial fibrillation | 15.8 | 24.7 | 27.9 | <.001 | <.001 |
| Heart failure | 35.7 | 43.9 | 47.6 | <.001 | <.001 |
| Pulmonary edema | 12.4 | 17.0 | 20.7 | <.001 | <.001 |
| Cardiogenic shock | 4.3 | 9.2 | 26.7 | <.001 | <.001 |
| Treatment (%) | | | | | |
| Aspirin | 82.2 | 81.2 | 67.6 | .29 | <.001 |
| ACE-I/ARBs | 47.4 | 48.6 | 38.0 | .33 | <.001 |
| Antiarrhythmic agents | 12.9 | 27.8 | 40.6 | <.001 | <.001 |
| β-Blockers | 77.0 | 77.9 | 62.5 | .41 | <.001 |
| Lipid-lowering agents | 40.3 | 39.3 | 24.6 | .40 | <.001 |
| Thrombolytic therapy | 11.3 | 21.4 | 22.0 | <.001 | <.001 |
| PCI | 25.6 | 27.7 | 28.1 | .06 | .19 |
| CABG surgery | 4.6 | 6.9 | 5.8 | <.001 | .19 |

*P* value from $\chi^2$ test and Fisher exact test for categorical variables and Wilcoxon rank-sum test for continuous variables; *P*1: no VT/VF versus VT, *P*2: no VT/VF versus VF. *IQR*, interquartile range.

included in the fully adjusted models as a potentially confounding factor.

We examined the overall magnitude, and changes over time therein, in the frequencies of VT and VF based on the presence of concomitant heart failure/cardiogenic shock or not. Because the occurrence of VT may differ between patients with a STEMI and NSTEMI, trends in the hospital IRs and CFRs of VT were also examined for these 2 groups, separately in patients admitted to all central Massachusetts hospitals between 2001 and 2011, when information about the type of AMI was collected. We examined trends in the hospital IRs and CFRs of VT and VF according to whether or not the patient received coronary reperfusion therapy, namely, thrombolytic therapy or a PCI, during the years under study. In addition, we examined trends in hospital IRs of early (during the first 2 days after hospital admission) versus late VT in patients further classified by type of AMI (eg, STEMI vs NSTEMI).

Given the large size of this study, almost all differences that were observed were highly statistically significant. Thus, all reported between-group differences without an explicit *P* value had a *P* value <.05.

## Results

### Baseline patient characteristics

The median age of the study population was 71 years old, most patients were white (94.7%), and 57.9% were men. Patients whose clinical course was complicated by either VT (n = 1731) or VF (n = 574) were several years younger than patients who did not develop these serious ventricular arrhythmias and were more likely to be male (Table I). Patients who developed VT or VF were less likely to have a medical history of diabetes, hypertension, or a stroke/transient ischemic attack. On the other hand, patients in whom these arrhythmias were diagnosed were more likely to have developed important clinical complications (eg, atrial fibrillation, heart failure, cardiogenic shock) during their acute hospitalization. Compared with patients who did not develop these ventricular arrhythmias, patients who developed VT or VF were more likely to have been treated with antiarrhythmic agents or thrombolytic therapy but were less likely to have received aspirin, angiotensin-converting enzyme inhibitor/angiotensin II receptor blockers (ACE-I/ARBs), β-blockers, and lipid-lowering agents.

**Table II.** Trends in the characteristics of patients admitted to the hospital for an AMI

| Characteristics | 1986-1990 (n = 2012) | 1997-2001 (n = 3074) | 2007-2011 (n = 2157) | *P* for trend |
|---|---|---|---|---|
| Age (median [IQR], y) | 70 [60-78] | 73 [61-82] | 68 [57-80] | .12 |
| Age (y, %) | | | | |
| <55 | 14.0 | 15.5 | 19.8 | |
| 55-64 | 21.8 | 16.1 | 21.1 | |
| 65-74 | 30.3 | 22.9 | 22.3 | |
| ≥75 | 33.9 | 45.5 | 36.9 | |
| Men (%) | 60.1 | 57.3 | 61.0 | .99 |
| White (%) | 97.6 | 95.4 | 91.7 | <.001 |
| History of disease (%) | | | | |
| Coronary artery disease | 19.1 | 52.4 | 32.0 | <.001 |
| Congestive heart failure | 14.8 | 23.5 | 20.6 | <.001 |
| Diabetes | 25.7 | 32.0 | 38.3 | <.001 |
| Hypertension | 49.9 | 65.0 | 74.9 | <.001 |
| Stable angina | 27.6 | 23.3 | 6.6 | <.001 |
| Stroke/transient ischemic attack | 9.1 | 13.5 | 13.1 | <.001 |
| Admission potassium (median [IQR], mmol/L) | 4.1 [3.7-4.4] | 4.2 [3.9-4.7] | 4.2 [3.8-4.6] | <.001 |
| Length of stay (median [IQR], d) | 10 [7-13] | 5 [3-8] | 3 [2-6] | <.001 |
| In-hospital complications (%) | | | | |
| Atrial fibrillation | 19.3 | 16.9 | 17.2 | .047 |
| Heart failure | 41.5 | 36.1 | 32.4 | <.001 |
| Pulmonary edema | 18.3 | 13.3 | 8.2 | <.001 |
| Cardiogenic shock | 6.9 | 6.3 | 5.1 | <.001 |
| Treatment (%) | | | | |
| Aspirin | 43.7 | 88.4 | 94.5 | <.001 |
| ACE-I/ARBs | 8.6 | 54.2 | 72.4 | <.001 |
| Antiarrhythmic agents | 20.3 | 14.6 | 11.1 | .11 |
| β-Blockers | 48.4 | 81.1 | 92.6 | <.001 |
| Lipid-lowering agents | 2.9 | 38.2 | 89.2 | <.001 |
| Thrombolytic therapy | 17.4 | 16.2 | 0.6 | <.001 |
| PCI | 2.8 | 22.6 | 53.6 | <.001 |
| CABG surgery | 1.7 | 6.6 | 7.5 | <.001 |

Years 1991-1995 and 2003-2005 were omitted for easy presentation of data. *P* values for trend were from logistic regression analysis for categorical variables and linear regression analysis for continuous variables with study year included as a linear predictor.

Between 1986 and 2011, the prevalence of several preexisting comorbidities (eg, heart failure, diabetes, hypertension, stroke) increased (Table II), as did the prescribing of aspirin, ACE-I/ARBs, β-blockers, lipid-lowering agents, PCI, and CABG surgery. During this period, there were declines in the frequency of several important in-hospital complications including atrial fibrillation, heart failure, and cardiogenic shock. Receipt of thrombolytic therapy and the average hospital length of stay also decreased over time (Table II).

## Trends in hospital IRs of VT and VF

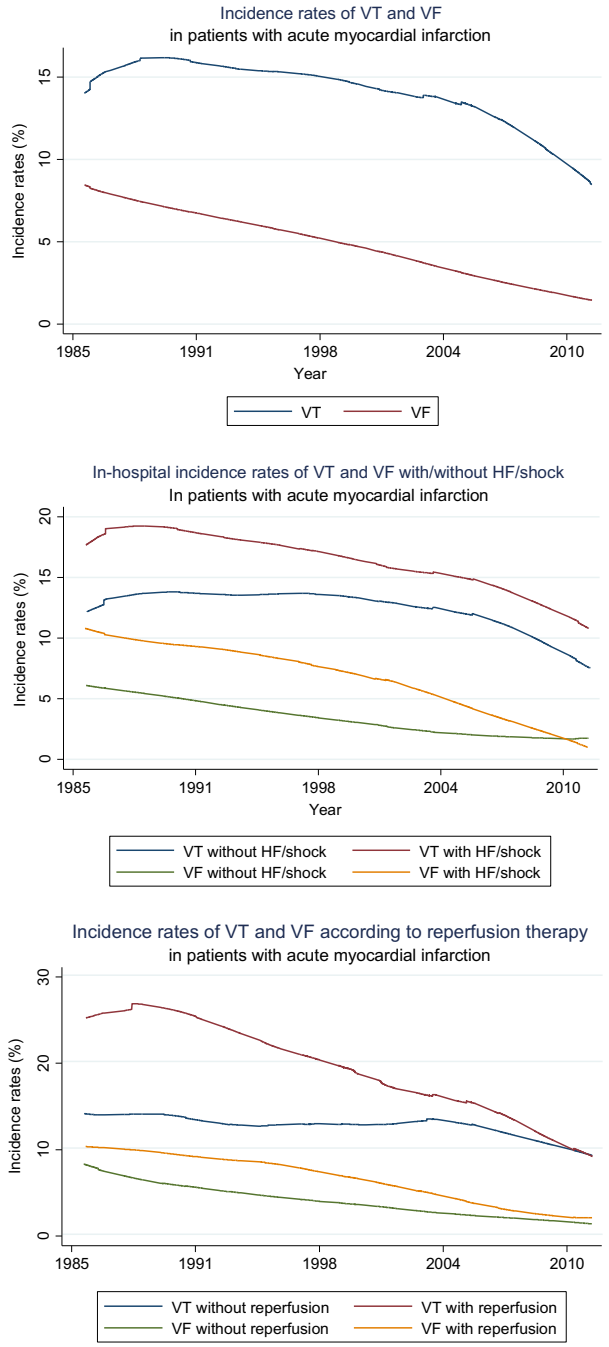
Between 1986 and 2011, VT developed in 14.6% and VF in 4.9% of all patients admitted to central Massachusetts hospitals for an AMI. During this period, the proportion of patients with AMI who developed VT declined from 14.3% in 1986/1988 to 10.5% in 2009/2011, whereas the proportion of patients who developed VF declined from 8.2% in 1986/1988 to 1.7% in 2009/2011 (Figure 1, top panel).

After adjusting for changes in the demographic and clinical characteristics of our patient population over time, the proportion of patients who developed VT declined by approximately 11% (95% CI = 6%-17%), whereas the proportion of patients who developed VF declined by 37% (95% CI = 30%-43%) after each decade, respectively (Table III). After we adjusted for changes over time in the receipt of various cardiac medications and reperfusion therapies received at the time of hospital admission during the years under study, the proportion of patients who developed VT did not change over time, whereas adjustment for the receipt of these therapies attenuated the magnitude of the decline in the hospital IRs of VF after each decade to 20% (95% CI = 7%-32%).

## Trends in hospital IRs of VT and VF according to concomitant heart failure or cardiogenic shock

Approximately 41% of our 11,825 study patients developed either heart failure or cardiogenic shock during their hospitalization for AMI. The development of ventricular arrhythmias in these patients was more common than in patients who did not develop either heart failure or cardiogenic shock (17.2% vs 12.9% for VT and 7.3% vs 3.2% for VF, respectively). The proportion of patients who developed VT among those with concomitant heart failure

**Figure 1**




Trends in hospital IRs of VT and VF in patients with AMI: overall (top panel), according to the presence of heart failure/shock (middle panel), and reperfusion therapy (bottom panel).

or cardiogenic shock decreased from 17.7% in 1986/1988 to 13.6% in 2009/2011, whereas the hospital IRs for VT for those who did not develop these hemodynamic disturbances did not significantly decline ($P = .45$). The IRs of VF among those with concomitant heart failure or cardiogenic shock decreased from 10.7% in 1986/1988 to 2.1% in 2009/2011 compared with a decrease from 6.0% in 1986/1988 to 1.6% in 2009/2011 among patients without concomitant heart failure or cardiogenic shock (Figure 1, middle panel).

## Trends in hospital IRs of VT and VF according to time of occurrence

Data on the time of occurrence of VT and VF and type of AMI were collected in 5,764 patients admitted to all greater Worcester hospitals on a biennial basis with AMI between 2001 and 2011. During this period, VT occurred in 13.2%, whereas VF occurred in 2.8% of these patients, respectively. The frequency of VT declined from 15.8% in 2001/2003 to 9.9% in 2009/2011 and from 3.5% to 1.6% for VF. More than two-thirds of all VT and VF episodes occurred during the first 48 hours after hospital admission. The hospital IRs of both early and late VT declined over time, with greater declines noted for those with late VT in both relative and absolute terms (Figure 2, top panel). Similarly, the incidence rates of early and late VF significantly declined during the most recent decade under study, with a larger decline observed for late VF (Figure 2).

## Trends in hospital IRs of VT and VF according to type of AMI

During the most recent decade (2001-2011) under study in which data were collected on the type of AMI patients experienced, 32.0% of study patients (n = 1,161) were diagnosed with a STEMI, whereas the remainder (n = 3,930) were diagnosed with an NSTEMI. The occurrence of both VT and VF was more common in patients with a STEMI than for those with an NSTEMI (17.3% vs 11.3% for VT and 5.3% vs 1.6% for VF). Among patients with a STEMI, about 80% of episodes of VT and VF occurred within the first 48 hours after hospital admission, compared with only 60% of such episodes for those with an NSTEMI. The hospital IRs of VT and VF declined between 2001 and 2011 in patients with a STEMI (Figure 2, bottom panel). The IRs of VT also declined in patients with an NSTEMI, whereas the IRs of VF in these patients did not change significantly over time ($P = .25$, Figure 2).

## Trends in hospital IRs of VT and VF according to the receipt of coronary reperfusion therapy

We also examined trends in the hospital IRs of VT and VF during the years under study according to the receipt of coronary reperfusion therapy (eg, thrombolytic therapy or a PCI) during the patient's acute hospitalization.

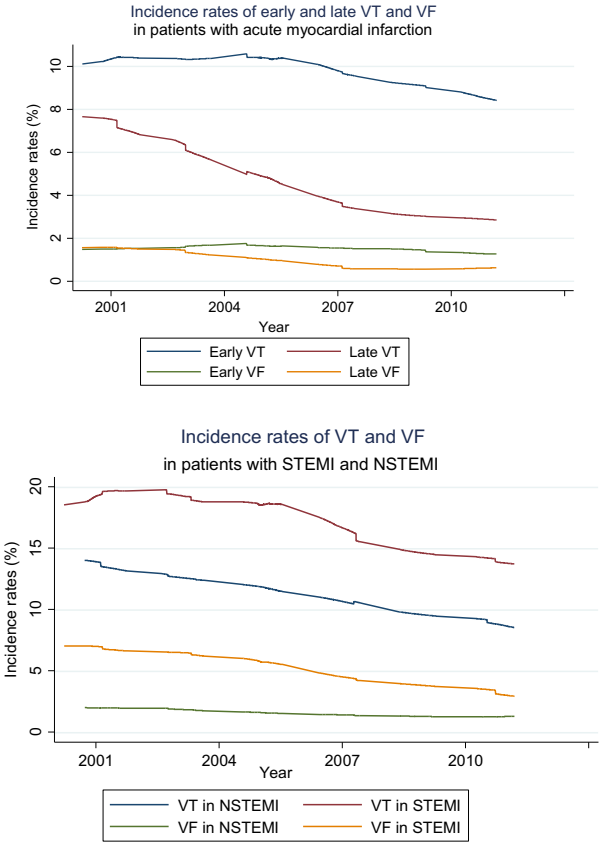
Patients who received either form of coronary reperfusion therapy were more likely to have developed either VT (17.8% vs 12.8%) or VF (5.9% vs 4.3%) than those who were not treated with coronary reperfusion therapy ($P < .001$). Encouragingly, there was a marked decline over time in the likelihood of developing either VT or

**Table III.** Trends in hospital IRs and CFRs of VT and VF for each decade among patients with AMI

| | VT | | VF | |
|---|---|---|---|---|
| IRs | (n = 11,251) | | (n = 10,094) | |
| In years 1986/1988 | 14.3 | | 8.2 | |
| In years 2009/2011 | 10.5 | | 1.7 | |
| Trends regression models | IRR | 95% CI | IRR | 95% CI |
| Unadjusted | 0.85 | 0.81-0.91 | 0.57 | 0.51-0.64 |
| Demographic factors (1) | 0.86 | 0.81-0.91 | 0.58 | 0.52-0.65 |
| History of disease (2) & (1) | 0.87 | 0.82-0.92 | 0.60 | 0.53-0.67 |
| Clinical presentation (3) & (1), (2) | 0.89 | 0.83-0.94 | 0.63 | 0.57-0.70 |
| In-hospital treatments (4) & (1), (2), (3) | 0.89 | 0.81-0.97 | 0.80 | 0.68-0.93 |
| CFRs | (n = 1731) | | (n = 574) | |
| In years 1986/1988 | 27.7 | | 49.5 | |
| In years 2009/2011 | 6.9 | | 37.5 | |
| Trends regression models | IRR | 95%CI | IRR | 95%CI |
| Unadjusted | 0.64 | 0.53-0.77 | 0.87 | 0.76-0.99 |
| Demographic (1) | 0.59 | 0.49-0.71 | 0.84 | 0.74-0.95 |
| History of disease (2) & (1) | 0.57 | 0.47-0.68 | 0.80 | 0.71-0.90 |
| Clinical presentation (3) & (1), (2) | 0.59 | 0.50-0.71 | 0.76 | 0.67-0.86 |
| In-hospital treatments (4) & (1), (2), (3) | 1.09 | 0.86-1.39 | 1.07 | 0.91-1.27 |

Each block of variables was added to precedent blocks in the successive models.
Block 1: Age, sex, and race.
Block 2: History of heart failure, diabetes, hypertension, and stroke/transient ischemic attack.
Block 3: In-hospital serum potassium levels and hospital complications of atrial fibrillation, heart failure, and cardiogenic shock.
Block 4: In-hospital treatment with aspirin, ACE-I, antiarrhythmic agents, β-blockers, lipid-lowering agents, thrombolytics, PCI, and CABG.

**Figure 2**




Trends in hospital IRs of VT and VF in patients with STEMI and NSTEMI (top panel) and by timing of VT and VF (early: in the first 48 hours of hospital stay, late: after the first 48 hours of hospital stay, bottom panel).

VF for patients who did or did not receive coronary reperfusion therapy (Figure 1, bottom panel). A greater decline was observed in the IRs of VT among patient who received coronary reperfusion therapy compared with patients who did not receive this treatment modality (Figure 1, bottom panel).

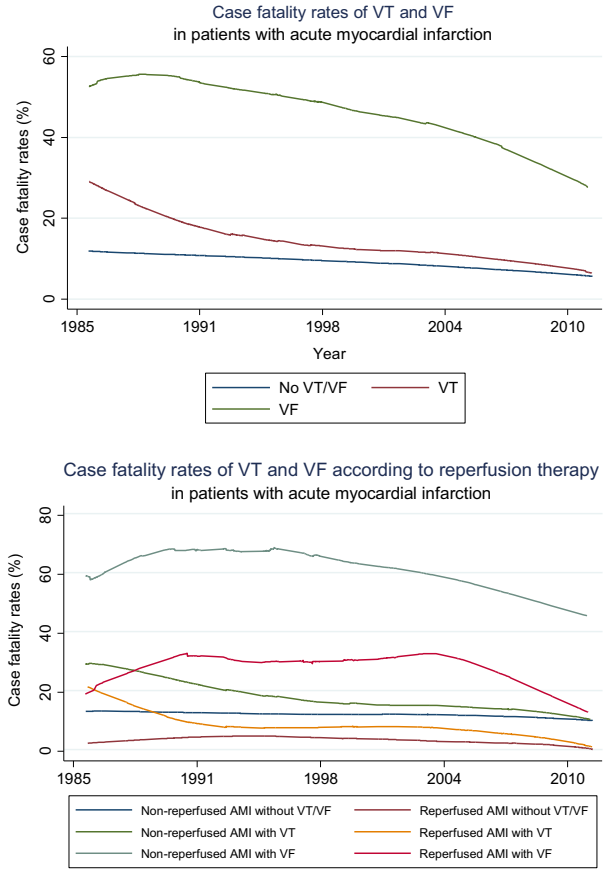
## Trends in hospital CFRs of VT and VF

The overall in-hospital CFRs were 48.3% among patients who developed VF, 13.9% in patients who developed VT, and 8.9% in patients who did not develop either of these ventricular arrhythmias during their acute hospital stay. The in-hospital CFRs decreased from 27.7% in our initial 2 study years (1986/1988) to 6.9% in our 2 most recent study years of 2009/2011 for patients who developed VT, from 49.6% to 36.0% for patients who developed VF, and from 11.5% to 4.6% for those whose hospital course was not complicated by either VT or VF (Figure 3). After adjusting for changes in patient's demographic characteristics and clinical presentation during the years under study, the hospital CFRs decreased by 41% (95% CI = 39%-50%) for patients who developed VT and by 24% (95% CI = 14%-32%) for those who developed VF after each study decade (Table III). However, the declining trends in the hospital CFRs of VT and VF were markedly attenuated after adjusting for the receipt of various cardiac medications and reperfusion treatments during the patient's index hospitalization for AMI.

## Trends in hospital CFRs of VT and VF according to the receipt of coronary reperfusion therapy

Patients who were treated with coronary reperfusion therapy in the form of either thrombolytic therapy or a PCI experienced markedly lower in-hospital death rates than those who did not receive this form of therapy, irrespective of whether they did not develop either VT or VF (3.5% vs 11.7%) or if they developed VT (7.7% vs 18.9%) or VF (29.4% vs 63.3%), respectively. The in-hospital CFRs declined over time for all patients, regardless of whether they received reperfusion therapy or if they developed VAs, with more significant declines observed in patients who developed VT who were treated with either conventional or coronary reperfusion therapy (Figure 3, bottom panel). However, caution needs to be exercised in the interpretation of the in-hospital death rates among

**Figure 3**




Trends in hospital CFRs of VT and VF in patients with AMI: overall (top panel) and according to receipt of coronary reperfusion therapy (bottom panel).

patients who developed VT, and especially VF, given the declining frequency of these serious VAs among patients with AMI which resulted in relatively small sample sizes of several patient groups during the most recent years under study.

## Discussion

In this community-based study of approximately 12,000 patients hospitalized with AMI at all central Massachusetts medical centers, we found that the incidence rates of both VT and VF, especially among patients with concomitant heart failure or cardiogenic shock, declined between 1986 and 2011. Although the in-hospital death rates for patients whose hospital course was complicated by VT or VF have declined during the study period, mortality remained high at approximately 40% in patients who developed VF in 2009/2011. Between 2001 and 2011, trends in the magnitude of early versus late VT differed, whereas trends in the frequency of VF complicating AMI differed according to the type of AMI patients developed.

### Trends in the hospital IRs of VT complicating AMI

We observed declining trends in the frequency of sustained and nonsustained VT among patients admitted to all central Massachusetts hospitals for an AMI between 1986 and 2011. Published data examining long-term or recent trends in the magnitude or death rates associated with VT complicating AMI are extremely limited. In a study of 2,280 patients who were admitted to all hospitals in Olmsted County, Minnesota, for an AMI between 1979 and 1998, sustained VT occurred in 1.8% of hospitalized patients.[9] Among the more than 3,000 patients enrolled in the Primary Angioplasty in Myocardial Infarction trials who underwent a primary PCI, VT/VF occurred in 4.3% of studied patients.[25] In the large multicenter randomized HORIZONS-AMI trial of patients with a STEMI who underwent a primary PCI,[26] 5.2% of patients developed VT/VF after undergoing their PCI, with most episodes (85%) occurring during the first 48 hours of their procedure. In a subgroup analysis of data from the multicenter, multicountry, APEX AMI trial,[6] among the 5,745 patients with a STEMI who underwent a primary PCI, 5.7% of patients developed VT/VF.

Notably, our observed declines in the frequency of VT were primarily driven by declines in VT among patients who had concomitant heart failure or cardiogenic shock. The declines we observed in the frequency of VT in patients with heart failure or cardiogenic shock might be due to increases in the use of coronary reperfusion therapies, including thrombolytic therapy and PCI, which have been shown to improve cardiac function and lower the risk of developing heart failure.[27] Indeed, in our study, the proportion of patients who developed heart failure after AMI decreased from 44% in 1986 to 29% in 2011.

Although the use of coronary reperfusion therapies might reduce the frequency of occurrence of primary and secondary VT after AMI, reperfusion arrhythmias, including VT, might offset these benefits.[6,28] An increasing occurrence of reperfusion arrhythmias might also explain the smaller decrease in the incidence of early VT, where reperfusion VTs are more prevalent, compared with the incidence rates of late VT that were observed between 2001 and 2011.

Whereas evidence-based treatment modalities may have an impact on preventing early-onset VT, which is often induced by transient abnormalities of automaticity or triggered activity following acute myocardial ischemia, these treatments can reduce infarct size and remodeling of the arrhythmic substrate or reentry pathways, thereby reducing the risk of late VT.[29] Discontinuation of the use of prophylactic lidocaine and other antiarrhythmic agents in the mid-1990s might also have contributed to declines in the frequency of VT.[30,31] The trends we observed are consistent with these hypotheses; however, disentangling the interplay of these factors on trends in the incidence rates of VT is complex and remains a topic for further study.

## Trends in the hospital IRs of VF complicating AMI

The frequency of primary VF (ie, VF which occurs in patients without heart failure or cardiogenic shock) and secondary VF among patients with an AMI also decreased between 1986 and 2011, with a larger decrease in the frequency of secondary VF. This finding was in contrast to our prior analysis of data from this community-wide study which failed to find a significant change in the incidence rates of primary VF complicating AMI between 1975 and 1997[14,15] or with the results of the Rochester Epidemiology study between 1979 and 1998.[9]

Declining trends in the magnitude of VF that we observed may be due, in part, to the favorable effects of increased utilization of evidence-based treatments for AMI during the most recent years under study. In contrast with relatively stable trends in the frequency of VT between 1986 and 2011, declines in the occurrence of primary VF might be explained by the fact that reperfusion-induced VF occurred in a considerably smaller proportion of treated patients compared with the frequency of VT.[32]

## Trends in hospital IRs of VT and VF according to type of AMI

Consistent with prior reports,[7,33,34] we found that VT and VF occurred more commonly in patients with a STEMI than in patients with an NSTEMI. Most instances of VT and VF tended to occur early within the first 48 hours of hospitalization in patients with a STEMI, whereas slightly more than one-half of all episodes of VF occurred during this high-risk period among patients with an NSTEMI. The greater frequency of ventricular arrhythmias in patients with a STEMI is possibly due to the more common occurrence of electrical or inflammatory disturbances that result after the complete blockage of a coronary artery compared to less common disturbances due to residual coronary flow in patients with an NSTEMI.[35] In addition, emergent PCI is more common in patients with a STEMI, which may result in a greater frequency of reperfusion-induced VT as compared to patients with an NSTEMI, where a "watchful waiting" strategy is more commonly used. Differences in the timing of these ventricular arrhythmias may have implications for when to discharge high-risk patients with an NSTEMI. However, later discharge and/or increased postdischarge surveillance for patients with an NSTEMI needs to be considered from a cost-benefit perspective given the fairly low incidence rates of VF (1.6%) among these patients.

Patients with either a STEMI or an NSTEMI experienced a decline in the hospital frequency of VT between 2001 and 2011. Similar trends were observed with regard to the development of VF among patients with a STEMI, whereas the occurrence of VF among patients with an NSTEMI did not change during this period. It is unclear why the frequency of VF did not decrease in patients with an NSTEMI during the study period. However, the low frequency of VF (1.6%) among these patients might have been difficult to detect in our study. Differences in the trends of VT and VF among patients with an NSTEMI may also suggest distinct underlying mechanisms and risk factors for the development of these serious arrhythmias among these patients.

## Trends in hospital CFRs of VT and VF complicating AMI

We observed an encouraging decline in the hospital death rates of patients who did not develop VT or VF, as well as in patients who developed either VT or VF, between 1986 and 2011. Our prior work also found lower odds of dying among patients who developed primary VF in the late 1990s and early 2000s compared with the late 1970s.[22]

Improvements in the timing and use of in-hospital cardiopulmonary resuscitation might have contributed to the decreased in-hospital CFRs of VT and VF that we observed during the years under study. Data from 17,490 patients with in-hospital VT or VF who were studied in the Get With The Guidelines–Resuscitation registry showed that immediate survival after resuscitation increased from 58% in 2001 to 72% in 2009.[36-39] Several factors, including early recognition and rapid defibrillation, greater availability of trained personnel, better chest compressions, and therapeutic hypothermia, likely contributed to improved survival rates.[33-36] For patients with hemodynamically stable VT, changes in patient management, including correction of electrolyte and acid/base abnormalities and treatment of important clinical complications, during the period under study may have further reduced the risk of deterioration into pulseless VT or VF and death.

It is important to note that, in the present study, changes in hospital management practices, namely, the increased use of aspirin, ACE-I/ARBs, β-blockers, and lipid-lowering agents, as well as increased use of PCI and CABG surgery during the period under study, primarily accounted for the improvement in the short-term survival rates we observed among patients who developed either VT or VF. Our findings provide encouragement for the continued high use of effective hospital treatments for AMI to reduce the risk of dying from these ventricular arrhythmias.

## Study strengths and limitations

The present study has several strengths. It is among the very few studies that have provided more generalizable community-based epidemiologic data that can furnish insights into the magnitude and impact of the major ventricular arrhythmias in patients hospitalized with AMI. All patients admitted to the coronary care unit were telemonitored for their entire stay, making it unlikely that episodes of asymptomatic and nonsustained VT were missed. However, our study also has several limitations. Because of considerable missing data on duration of prehospital delay, we were unable to systematically classify the time of onset of VT or VF according to acute symptom onset. However, in an analysis of 1,501 patients with information available about duration of prehospital delay,

the classification of early from late VT or VF based on acute symptom onset agreed well with that based on the timing of hospital admission. Because of our methods of data collection, we were unable to disentangle patients who developed sustained VT from those who developed nonsustained VT, which may, in part, explain the relatively high IRs of VT that we observed in the present study compared with prior investigations. Finally, caveats need to be placed on the interpretation of our data that examined trends in the hospital CFRs of VT and VF according to the receipt of coronary reperfusion therapy during the years under study given the sample sizes involved and unclear temporarily of administration of this therapy and development of the serious VAs under study.

## Conclusions

The results of this community-wide study found declining multidecade-long trends in the incidence and mortality of VT and VF in a community-wide population of patients hospitalized with AMI. The encouraging trends that we observed are likely due to changes in hospital treatment practices during the years under study. However, efforts remain needed to identify patients at risk for these serious ventricular arrhythmias so that effective preventive and treatment strategies could be implemented as necessary.[40]

## Funding support and disclosures

Partial support for R. J. G. was provided by National Institutes of Health (NIH)/National Heart, Lung, and Blood Institute grants 1R01HL126911-01A1, 5R01HL125089-02, and 5R01HL115295-05. Partial support for C. I. K. was provided by Patient-Centered Outcomes Research Institute ME-1310-07682; NIH/National Center for Research Resources U54 RR 026088; and National Heart, Lung, and Blood Institute R01 HL126911. Partial support for A. S. A. was provided by NIH grant 5UL1TR001453. All authors have declared no conflicts of interest.

## References

1. Newby KH, Thompson T, Stebbins A, et al. Sustained ventricular arrhythmias in patients receiving thrombolytic therapy. Circulation 1998;98:2567-73, https://doi.org/10.1161/01.CIR.98.23.2567.
2. Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death). J Am Coll Cardiol 2006;48:e247-346, https://doi.org/10.1016/j.jacc.2006.07.010.
3. Khairy PP. Prognostic significance of ventricular arrhythmias post-myocardial infarction. Can J Cardiol 2003;19:1393-404.
4. Scirica BM, Braunwald E, Belardinelli L, et al. Relationship between nonsustained ventricular tachycardia after non–ST-elevation acute coronary syndrome and sudden cardiac death. Circulation 2010;122:455-62, https://doi.org/10.1161/CIRCULATIONAHA.110.937136.
5. Timmer J, Breet N, Svilaas T, et al. Predictors of ventricular tachyarrhythmia in high-risk myocardial infarction patients treated with primary coronary intervention. Neth Heart J 2010;18:122-8, https://doi.org/10.1007/bf03091750.
6. Mehta RH, Starr AZ, Lopes RD, et al. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. JAMA 2009;301:1779-89, https://doi.org/10.1001/jama.2009.600.
7. Al-Khatib SM, Granger CB, Huang Y, et al. Sustained ventricular arrhythmias among patients with acute coronary syndromes with no ST-segment elevation. Circulation 2002;106:309-12, https://doi.org/10.1161/01.CIR.0000022692.49934.E3.
8. Volpi A, Cavalli A, Turato R, et al. Incidence and short-term prognosis of late sustained ventricular tachycardia after myocardial infarction: results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-3) Data Base. Am Heart J 2001;142:87-92, https://doi.org/10.1067/mhj.2001.115791.
9. Henkel DM, Witt BJ, Gersh BJ, et al. Ventricular arrhythmias after acute myocardial infarction: a 20-year community study. Am Heart J 2006;151:806-12, https://doi.org/10.1016/j.ahj.2005.05.015.
10. Bradley EH, Nallamothu BK, Stern AF, et al. The door-to-balloon alliance for quality: who joins national collaborative efforts and why? Jt Comm J Qual Patient Saf 2009;35:93-9, https://doi.org/10.1016/S1553-7250(09)35012-6.
11. Liang JJ. Temporal evolution and implications of ventricular arrhythmias associated with acute myocardial infarction. Cardiol Rev 2013;21:289-94, https://doi.org/10.1097/CRD.0b013e3182a46fc6.
12. Goldberg RJ, Yarzebski J, Lessard D, et al. Decade-long trends and factors associated with time to hospital presentation in patients with acute myocardial infarction: the Worcester Heart Attack study. Arch Intern Med 2000;160:3217-23, https://doi.org/10.1001/archinte.160.21.3217.
13. Crowley A, Menon V, Lessard D, et al. Sex differences in survival after acute myocardial infarction in patients with diabetes mellitus (Worcester Heart Attack Study). Am Heart J 2003;146:824-31, https://doi.org/10.1016/S0002-8703(03)00406-X.
14. Chiriboga D, Yarzebski J, Goldberg RJ, et al. Temporal trends (1975 through 1990) in the incidence and case-fatality rates of primary ventricular fibrillation complicating acute myocardial infarction. A communitywide perspective. Circulation 1994;89:998-1003, https://doi.org/10.1161/01.CIR.89.3.998.
15. Thompson CA, Yarzebski J, Goldberg RJ, et al. Changes over time in the incidence and case-fatality rates of primary ventricular fibrillation complicating acute myocardial infarction: perspectives from the Worcester heart attack study. Am Heart J 2000;139:1014-21, https://doi.org/10.1067/mhj.2000.106160.
16. Goldberg RJ, Gore JM, Alpert JS, et al. Recent changes in attack and survival rates of acute myocardial infarction (1975 through 1981): the Worcester heart attack study. JAMA 1986;255:2774-9, https://doi.org/10.1001/jama.1986.03370200076031.
17. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Circulation 2012;126:2020-35, https://doi.org/10.1161/CIR.0b013e31826e1058.
18. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-Elevation myocardial infarction.

A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61:e78-e140, https://doi.org/10.1016/j.jacc.2012.11.019.

19. Bonow RO, Mann DL, Zipes DP, et al. *Braunwald's heart disease: a textbook of cardiovascular medicine*. Elsevier Health Sciences. 2011.
20. Goldberg RJ, Gore JM, Alpert JS, et al. Cardiogenic shock after acute myocardial infarction: incidence and mortality from a community-wide perspective, 1975 to 1988. N Engl J Med 1991;325:1117-22, https://doi.org/10.1056/NEJM199110173251601.
21. Saczynski JS, Spencer FA, Gore JM, et al. Twenty-year trends in the incidence of stroke complicating acute myocardial infarction: Worcester heart attack study. Arch Intern Med 2008;168:2104-10, https://doi.org/10.1001/archinte.168.19.2104.
22. Goldberg RJ, Yarzebski J, Spencer FA, et al. Thirty-year trends (1975-2005) in the magnitude, patient characteristics, and hospital outcomes of patients with acute myocardial infarction complicated by ventricular fibrillation. Am J Cardiol 2008;102:1595-601, https://doi.org/10.1016/j.amjcard.2008.08.008.
23. Kelly H, Cowling BJ. Case fatality: rate, ratio, or risk? Epidemiology 2013;24:622-3, https://doi.org/10.1097/EDE.0b013e318296c2b6.
24. Cleveland WS. Robust locally weighted regression and smoothing scatterplots. J Am Stat Assoc 1979;74:829-36, https://doi.org/10.1080/01621459.1979.10481038.
25. Mehta RH, Harjai KJ, Grines L, et al. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. J Am Coll Cardiol 2004;43:1765-72.
26. Mehta RH, Yu J, Piccini JP, et al. Prognostic significance of postprocedural sustained ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention (from the HORIZONS-AMI Trial). Am J Cardiol 2012;109:805-12.
27. Goldberg RJ, Spencer FA, Okolo J, et al. Long-term trends in the use of coronary reperfusion strategies in acute myocardial infarction: a community-wide perspective. J Thromb Thrombolysis 2007;23:163-71, https://doi.org/10.1007/s11239-006-9029-0.
28. Aufderheide TP. Arrhythmias associated with acute myocardial infarction and thrombolysis. Emerg Med Clin North Am 1998;16:583-600, https://doi.org/10.1016/S0733-8627(05)70019-5.
29. Thomas D, Jex N, Thornley A. Ventricular arrhythmias in acute coronary syndromes—mechanisms and management. Contin Cardiol Educ 2017;3:22-9, https://doi.org/10.1002/cce2.51.
30. Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). J Am Coll Cardiol 1996;28:1328-428, https://doi.org/10.1016/S0735-1097(96)00392-0.
31. Antman EM. Declining incidence of ventricular fibrillation in myocardial infarction. Implications for the prophylactic use of lidocaine. Circulation 1992;86:764-73, https://doi.org/10.1161/01.CIR.86.3.764.
32. Gressin V, Louvard Y, Pezzano M, et al. Holter recording of ventricular arrhythmias during intravenous thrombolysis for acute myocardial infarction. Am J Cardiol 1992;69:152-9, https://doi.org/10.1016/0002-9149(92)91295-F.
33. Steg PG, Goldberg RJ, Gore JM, et al. Baseline characteristics, management practices, and in-hospital outcomes of patients hospitalized with acute coronary syndromes in the Global Registry of Acute Coronary Events (GRACE). Am J Cardiol 2002;90:358-63, https://doi.org/10.1016/S0002-9149(02)02489-X.
34. Gheeraert PJ, De Buyzere ML, Taeymans YM, et al. Risk factors for primary ventricular fibrillation during acute myocardial infarction: a systematic review and meta-analysis. Eur Heart J 2006;27:2499-510, https://doi.org/10.1093/eurheartj/ehl218.
35. Di Stefano R, Di Bello V, Barsotti MC, et al. Inflammatory markers and cardiac function in acute coronary syndrome: Difference in ST-segment elevation myocardial infarction (STEMI) and in non-STEMI models. Biomed Pharmacother 2009;63:773-80, https://doi.org/10.1016/j.biopha.2009.06.004.
36. Girotra S, Nallamothu BK, Spertus JA, et al. Trends in survival after in-hospital cardiac arrest. N Engl J Med 2012;367:1912-20, https://doi.org/10.1056/NEJMoa1109148.
37. Jentzer JC, Clements CM, Wright RS, et al. Improving survival from cardiac arrest: a review of contemporary practice and challenges. Ann Emerg Med 2016;68:678-89, https://doi.org/10.1016/j.annemergmed.2016.05.022.
38. Callaway CW, Donnino MW, Fink EL, et al. Part 8: post–cardiac arrest care. 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2015;132:S465-82, https://doi.org/10.1161/cir.0000000000000262.
39. Chan PS, Berg RA, Tang Y, et al. Association between therapeutic hypothermia and survival after in-hospital cardiac arrest. JAMA 2016;316:1375-82, https://doi.org/10.1001/jama.2016.14380.
40. Bhar-Amato J, Davies W, Agarwal S. Ventricular arrhythmia after acute myocardial infarction: the perfect storm. Arrhythm Electrophysiol Rev 2017;6:134-9.