



# Short- and Long-Term Cause of Death in Patients Treated With Primary PCI for STEMI

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## ABSTRACT

**BACKGROUND** Short-term mortality has been studied thoroughly in patients undergoing primary percutaneous coronary intervention (PCI), whereas long-term cause of death in patients with ST-segment elevation myocardial infarction (STEMI) remains unknown.

**OBJECTIVES** The goal of this study was to describe the association between time and cause of death in patients with STEMI undergoing primary PCI.

**METHODS** A centralized civil registration system, patient files, and public disease and death cause registries with an accurate record linkage were used to trace time and cause of death in 2,804 consecutive patients with STEMI (age  $63 \pm 13$  years, 72% males) treated with primary PCI.

**RESULTS** Patients were followed up for a median of 4.7 years. During a total of 13,447 patient-years, 717 patients died. Main causes of death within the first 30 days were cardiogenic shock and anoxic brain injury after cardiac arrest. Age, culprit vessel size and flow, and the presence of heart failure and diabetes were independent predictors of mortality. After 30 days, the annual cardiac mortality rate was  $<1.5\%$ . Causes of death beyond 30 days were noncardiac in 65% of cases (mainly malignancies and pulmonary diseases). The 30-day, 1-year, and 5-year all-cause (and cardiac) mortality rates were 7.9% (7.3%), 11.4% (8.4%), and 23.3% (13.8%), respectively.

**CONCLUSIONS** Patients who survive the first month after an STEMI treated with primary PCI have an excellent prognosis, with a  $<1.5\%$  annual risk of successive cardiac death. Noncardiac causes are responsible for the majority of later deaths in these patients. (J Am Coll Cardiol 2014;64:2101-8) © 2014 by the American College of Cardiology Foundation.

Primary percutaneous coronary intervention (PCI) is the preferred initial treatment of patients presenting with ST-segment elevation myocardial infarction (STEMI) within 12 h of symptom onset, provided treatment can be initiated expeditiously by an experienced team (1-3). Knowledge of the causes of death in patients treated with primary PCI is important to implement new strategies and

design clinical trials and cardiac rehabilitation and secondary prevention programs, with the goal of further reducing mortality in these patients (4,5). However, relations between time and different causes of death after primary PCI have not been thoroughly investigated in large all-comer cohorts. Thus, the objective of the current study was to describe the associations between the time and causes of cardiac and

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## ABBREVIATIONS AND ACRONYMS

**AMI** = acute myocardial infarction

**BMS** = bare-metal stent(s)

**CPR** = civil personal registration (number)

**DES** = drug-eluting stent(s)

**MODS** = multiorgan dysfunction syndrome

**PCI** = percutaneous coronary intervention

**STEMI** = ST-segment elevation myocardial infarction

**TIMI** = Thrombolysis In Myocardial Infarction

**VSD** = ventricular septal defect

noncardiac death in consecutive patients with STEMI treated with primary PCI.

## METHODS

**PATIENTS.** Patients were eligible for this study if treated with primary PCI at our institution between July 1998 and July 2008. A diagnosis of STEMI was made in patients who presented within 12 h of chest pain onset and ST-segment elevation in at least 2 contiguous electrocardiographic leads. All patients treated with primary PCI were entered into this analysis, including those in cardiogenic shock and those resuscitated after cardiac arrest. Before transportation to primary PCI with emergency medical services,

10,000 U of intravenous unfractionated heparin, 300 mg of aspirin, and 300 or 600 mg of clopidogrel (150 mg of ticlopidine in the first years of the study period) were administered to each patient. After primary PCI, patients were medically treated according to contemporary guidelines.

SEE PAGE 2109

Conscious patients were informed according to the requirements of The Joint Commission for accreditation/certification of our hospital. Unconscious or incapacitated patients were treated according to the recommendations of the Danish Council of Ethics.

**RECORD LINKAGE.** Patient records include a unique, personal 10-digit civil personal registration (CPR) number, assigned at birth or at registration within the Danish Centralized Civil Registration System, in which all vital events are recorded for each patient. The CPR number, patient characteristics, history, and PCI procedural data were entered into the clinical database by the operating physician and assistants in the catheterization laboratory in relation to the primary PCI procedure.

The CPR number is used in all Danish public registries, which enables accurate record linkage. The National Board of Health (case file No. 7-505-29-889/1) gave its permission to cross-check data from public registries, and the study was reported to the Danish Data Protection Agency (case file No. 2008-41-2113).

**CLINICAL TRIALS.** During the study period, several randomized clinical primary PCI trials were conducted within our department (6-11). Patients and procedures from these trials are included in the analysis. In total, 743 of 2,804 STEMI patients (26%) were enrolled in randomized clinical trials.

## ENDPOINTS AND COMPLETENESS OF DATA.

Endpoints of this study were time and causes of death obtained from the Danish Centralized Civil Registration System, patients' medical records, the Danish National Patient Registry, and the Cause of Death Registry. All patients were classified as dead or alive. When the Danish Centralized Civil Registration System receives notification of a death, the event is recorded in the system within 2 weeks. Because the minimum duration of follow-up was 2.5 years, it is highly unlikely that any death would be missing from our analysis. Emigrated patients (n = 17) were followed up until the day of their emigration.

**CLASSIFICATION OF DEATH.** Two physicians reviewed the files of all dead patients, ascertaining the causes of death independently. In cases of any doubt or disagreement, the files were reviewed by a third physician and discussed until a consensus was reached as to the most likely cause of death.

The cause of death was classified into 1 of the following mutually exclusive categories: cardiovascular or noncardiovascular cause. Cardiovascular death was classified as either cardiac or vascular death, and cardiac causes were subclassified into cardiogenic shock, including multiple organ dysfunction syndrome, sudden death/cardiac arrest, anoxic brain injury after cardiac arrest, new acute myocardial infarction, life-threatening arrhythmias (ventricular tachycardia, ventricular fibrillation, or advanced atrioventricular node block), or congestive heart failure (12,13). Sudden cardiac death was defined as death that followed an abrupt loss of consciousness within 1 h of the onset of possible cardiac symptoms. Any death that could not clearly be attributed to a noncardiac cause was classified as cardiac, including those that occurred during sleep. Unless another specific cause could be identified, the cause of death in a patient with reinfarction was classified as death due to reinfarction. Unwitnessed death was classified according to available files. For example, if a patient were undergoing antibiotic treatment for pneumonia, the death cause would be classified as pneumonia, and if a patient with a recent stroke were found dead, the cause of death would be classified as a stroke. Malignant arrhythmias, cardiogenic shock, and cardiac rupture were divided into subgroups depending on whether they were related to the index infarction or a reinfarction or were not infarct related.

Available files were not detailed enough to assess a specific cause of death in 38 patients. In 14 of these patients, death certificates were missing. These patients were classified in the sudden cardiac death category.

**EXCLUDED PATIENTS.** The only STEMI patients excluded from the analysis were those without a CPR number (e.g., tourists; n = 57).

**STATISTICAL ANALYSES.** SPSS version 18.0 (SPSS Inc., Chicago, Illinois) was used for statistical analyses. Continuous variables are delineated by their mean ± SD or by the median and interquartile range, and frequencies are expressed as percents, as appropriate. All endpoints were analyzed either until death, emigration, or the study termination date in December 2010 using all available follow-up data from the index STEMI. Kaplan-Meier curves were constructed for patients to show mortality over time. Cox proportional hazards regression analysis was used to estimate hazard ratios for each endpoint. Crude and mutually adjusted hazard ratios with 95% confidence intervals were computed. Except for hypertension and body mass index (proportion of missing values >6%), variables associated with time to all-cause death in the univariate Cox regression analysis (p < 0.1) were included in the multivariate Cox regression model, which resulted in the inclusion of 2,573 total patients in this model. A 2-tailed p < 0.05 was considered significant.

**RESULTS**

Of 2,861 consecutive patients with STEMI treated with primary PCI at our institution, 57 were excluded (without a CPR number). Thus, 2,804 patients with a mean age of 62.7 ± 13.1 years were included in the study. The median time from symptom onset to initiation of the primary PCI procedure was 230 min (interquartile range: 159 to 345 min). Risk factors, emergency system delays, Thrombolysis In Myocardial Infarction (TIMI) flow in the culprit artery, number of vessels treated, concomitant medical treatment, and other procedural details are shown in **Table 1**.

In the median follow-up period of 4.7 years, 717 patients died. Whereas 30-day, 1-year, and 5-year all-cause mortality rates were 7.9%, 11.4%, and 23.3%, respectively, the corresponding cardiac mortality rates were 7.3%, 8.4%, and 13.8%, respectively. The median length of hospital stay was 5 days, and the 10-day and 20-day all-cause death rates were 5.8% and 6.3%, respectively.

Causes of death were cardiovascular in 61.6% (n = 442) and noncardiovascular in 38.4% (n = 275) of cases. The most frequent causes of death within the first 30 days (in % of the number of deaths within the entire follow-up period) were as follows: cardiogenic shock, 19.5%; anoxic brain damage, 3.1%; and malignant arrhythmia, 1.7%. The most frequent causes of death after 30 days were noncardiac in 64.8% of

**TABLE 1 Patient and Procedural Characteristics (N = 2,804)**

		Valid Cases, n* (%)†
Age, yrs	62.7 ± 13.1	2,804 (100)
Female	799 (28.5)	2,804 (100)
Hypertension	799 (33.6)	2,379 (84.8)
Hyperlipidemia	474 (32.3)	1,468 (52.4)
Active or previous smoker	1,768 (78.6)	2,250 (80.2)
Diabetes type 1 or 2	421 (15.0)	2,804 (100)
History of heart failure	107 (3.8)	2,804 (100)
Killip class I/II/III/IV, %	87/6/2/5	2,651 (94.5)
Body mass index, kg/m <sup>2</sup>	26.5 ± 4.4	1,154 ± 41
Median time delay, min (25th-75th percentile)		
Patient and first medical contact	128 (65-240)	1,531 (54.6)
Emergency service transportation	60 (32-90)	1,572 (56.1)
Symptom onset to PCI	230 (159-345)	1,532 (54.6)
Targeted coronary arteries		2,804 (100)
Left anterior descending	1,574 (44.5)	—
Circumflex	469 (13.2)	—
Right coronary	1,445 (40.8)	—
Left main	39 (1.1)	—
Saphenous vein graft	11 (0.3)	—
Arterial graft	2 (0.1)	—
Number of lesions treated		2,804 (100)
1	2,198 (78.4)	—
2	497 (17.7)	—
3	90 (3.2)	—
>3	19 (0.7)	—
Culprit artery		2,804 (100)
Left anterior descending	1,298 (46.3)	—
Circumflex	340 (12.1)	—
Right coronary	1,131 (40.3)	—
Left main	23 (0.8)	—
Saphenous vein graft	10 (0.4)	—
Arterial graft	2 (0.1)	—
Culprit vessel flow		
TIMI flow grade before 0/1/2/3, %	65/8/14/13	2,799 (99.8)
TIMI flow grade after 0/1/2/3, %	2/1/7/90	2,801 (99.9)
Intervention		2,804 (100)
Balloon angioplasty only	294 (10.5)	—
Any use of stent	2,506 (89.4)	—
Drug-eluting stent	1,255 (44.8)	—
Bare-metal stent	1,251 (44.6)	—
No use of stent or balloon	4 (0.1)	—
Largest balloon diameter, mm	3.3 ± 0.61	2,721 ± 97.0
Maximal balloon pressure, atm	15.9 ± 3.3	2,725 ± 97.2
Procedural medication		2,754 (98.2)
Clopidogrel	2,515 (91.3)	—
Aspirin	2,573 (93.4)	—
Glycoprotein IIb/IIIa inhibitor	1,664 (60.4)	—
Bivalirudin	82 (3.0)	—

Values are mean ± SD or n (%). \*Number of patients with the actual parameter available for analysis. †Percent = n × 100/total number of patients (N = 2,804).  
 PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction.

**TABLE 2 Causes of Death**

Main Groups and Subgroups	Main Groups	Subgroups	Median Time to Death, Days
<b>Cardiovascular</b>			
<b>Cardiac</b>			
Cardiogenic shock and MODS (index AMI)	140 (19.5)		1 (0-3)
Cardiogenic shock		125 (17.4)	1 (0-3)
Cardiogenic shock: VSD/myocardial rupture		8 (1.1)	3 (0-9)
Cardiac tamponade		7 (1.0)	0 (0-5)
Sudden death/cardiac arrest	111 (15.5)	111 (15.5)	1,005 (467-1,804)
Reinfarction	45 (6.3)		227 (6-1,096)
Reinfarction		22 (3.1)	300 (87-1,346)
Cardiogenic shock (reinfarction)		19 (2.6)	5 (3-790)
Cardiogenic shock: VSD/myocardial rupture (reinfarction)		1 (0.1)	1,099 (-)
Bradycardia/asystole (reinfarction)		2 (0.3)	10 (-)
Ventricular tachycardia/fibrillation		1 (0.1)	1,403 (-)
Congestive heart failure	29 (4.0)		717 (50-1,237)
Cardiogenic shock		6 (0.8)	515 (40-1,353)
Congestive heart failure/pulmonary congestion		15 (2.1)	760 (40-1,456)
Chronic heart failure		8 (1.1)	837 (138-1,218)
Anoxic brain damage (cardiac arrest, index AMI)	22 (3.1)	22 (3.1)	6 (4-11)
Other cardiac causes	19 (2.6)		932 (549-1,219)
Heart disease not specified		6 (0.8)	875 (433-1,024)
Ischemic heart disease		11 (1.5)	932 (97-1,782)
Ventricular tachycardia/fibrillation		2 (0.3)	1,718 (-)
Arrhythmias: index AMI	12 (1.7)		1 (0-3)
Ventricular tachycardia/fibrillation		3 (0.4)	1 (0-3)
Bradycardia/asystole		4 (0.6)	2 (1-11)
Pulseless electrical activity		5 (0.7)	1 (0-2)
<b>Vascular</b>			
Cerebral infarctions and bleedings	40 (5.6)	40 (5.6)	766 (143-1,652)
Aorta dissections/ruptured aneurysm	10 (1.4)	10 (1.4)	1,314 (585-2,127)
Abdominal and peripheral vascular causes	8 (1.1)		597 (5-1,396)
Bowel arterial occlusion		5 (0.7)	9 (3-677)
Peripheral vascular causes		3 (0.4)	1,512 (-)
Pulmonary embolism	6 (0.8)	6 (0.8)	1,783 (1,407-2,604)
<b>Noncardiovascular</b>			
Cancer and other malignancies	124 (17.3)	124 (17.3)	1,186 (718-1,691)
Other causes	81 (11.3)		643 (130-1,501)
Acute abdomen		13 (1.8)	542 (60-1,443)
Sepsis		19 (2.6)	1,222 (758-2,381)
Multiorgan failure		8 (1.1)	71 (37-1,411)
Other		41 (5.7)	622 (164-1,376)
Pneumonia and acute respiratory insufficiency	65 (9.1)		1,099 (535-1,972)
Pneumonia		51 (7.1)	1,232 (533-1,981)
Acute respiratory insufficiency		14 (2.0)	980 (541-1,749)
Gastrointestinal bleeding	5 (0.7)	5 (0.7)	375 (58-1,302)
<b>All</b>	<b>717 (100)</b>	<b>717 (100)</b>	<b>560 (10-1,402)</b>

Values are n (%) or median (interquartile range).  
AMI = acute myocardial infarction; MODS = multiple organ dysfunction syndrome; VSD = ventricular septal defect.

cases; from 30 days to 1 year, reinfarction (6.3%) and cerebrovascular diseases (5.6%) were the most common causes of death; and after 1 year, congestive heart failure (4.0%), pneumonia/acute respiratory insufficiency (9.1%), sudden cardiac death (15.5%), and cancer/other malignancies (17.3%) accounted for the most deaths (Table 2).

Univariate and multivariate analyses appear in Tables 3 and 4. Slow TIMI flow in the culprit artery after PCI was a rather strong prognostic predictor, with a hazard ratio of 2.5 (95% confidence interval: 1.71 to 3.62). In addition to age, diabetes, previous and current signs of heart failure, and a large culprit vessel diameter were harbingers of worsened prognosis, whereas sex, TIMI flow in the culprit artery before PCI, and the presence of multivessel disease provided no independent information.

Cumulative survival curves showed <1.5% of annual cardiac mortality rates after 1 month (Figure 1). Analyses of time and cause of death relations showed that cardiovascular death due to cardiogenic shock (median 1 day) and anoxic brain damage after cardiac arrest (median 6 days), as well as malignant arrhythmia (median 1 day), after STEMI occurred mainly during the first week after the index procedure, whereas cardiovascular death due to reinfarction (median 8 months) and congestive heart failure (median 2 years) were most frequent between 6 months and 2 years after the index event (Figure 2, Table 2). Deaths attributable to noncardiovascular

**TABLE 3 Univariate Analysis of Death**

	Univariate HR (95% CI)	p Value	Valid Cases, n*
Age, per 1-yr increase	1.07 (1.06-1.08)	<0.001	2,804
Female	1.55 (1.33-1.80)	<0.001	2,804
Hypertension	1.31 (1.10-1.55)	0.002	2,379
Hyperlipidemia	1.11 (0.88-1.41)	0.39	1,468
Current or previous smoker	1.00 (0.80-1.24)	0.96	2,250
Diabetes	1.59 (1.33-1.90)	<0.001	2,804
History of heart failure	3.32 (2.57-4.29)	<0.001	2,804
Killip class I vs. II to IV	2.59 (2.17-3.10)	<0.001	2,651
Body mass index, kg/m <sup>2</sup>	0.93 (0.90-0.96)	0.001	1,154
Symptom onset to PCI, min	1.00 (1.00-1.01)	0.22	1,532
TIMI flow grade 0 to 1 before	1.05 (0.89-1.24)	0.57	2,799
TIMI flow grade 0 to 1 after	3.60 (2.74-4.72)	<0.001	2,801
Vessel size, per 1 mm	0.62 (0.55-0.70)	<0.001	2,721
Maximal balloon pressure, atm	0.95 (0.93-0.97)	<0.001	2,725
Multivessel treatment (vs. single vessel)	1.38 (1.17-1.63)	<0.001	2,804
DES vs. BMS	0.95 (0.80-1.13)	0.58	2,506

\*Number of patients with the actual parameter available for analysis.  
BMS = bare-metal stent(s); CI = confidence interval; DES = drug-eluting stent(s); HR = hazard ratio; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction.

**TABLE 4 Multivariate Analysis of Death**

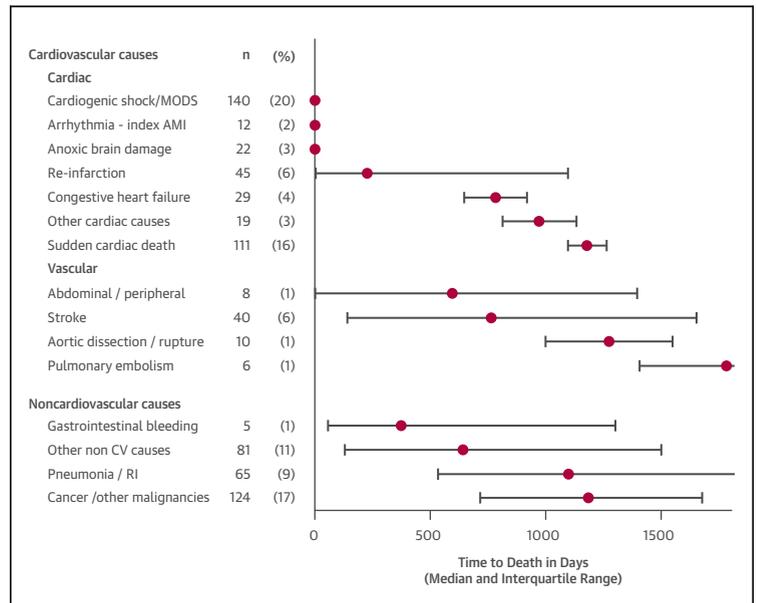
	Multivariate HR (95% CI)	p Value
Age, per 1-yr increase	1.07 (1.06–1.07)	<0.001
Diabetes, type 1 or 2	1.62 (1.32–1.97)	<0.001
History of heart failure	1.66 (1.32–1.97)	0.001
TIMI flow grade 0 to 1 after PCI	2.49 (1.71–3.62)	<0.001
Vessel size, per 1 mm	0.77 (0.68–0.88)	0.04
Killip class >I	1.44 (1.33–1.56)	<0.001

Abbreviations as in Table 3.

causes were relatively rare in the acute phase of STEMI treatment, whereas pneumonia (median 3 years) and cancer (median 3.2 years) were the most frequent causes of late death (Figure 2, Table 2).

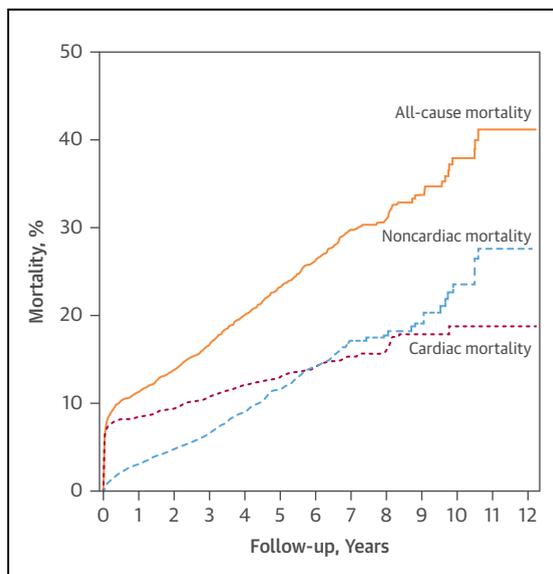
**DISCUSSION**

The pattern of early and long-term mortality after STEMI in the era of primary PCI is well described from large randomized trials comparing invasive treatment with fibrinolysis and evaluating different medical regimens and access site approaches in selected patients (14–19). In the present study, we focused on a detailed description of both the acute and long-term causes of death in a large cohort of



**FIGURE 2 Temporal Relation of Causes of Death in Patients With ST-Segment Elevation Myocardial Infarction After Primary Percutaneous Coronary Intervention**

Median times to death with interquartile ranges are indicated. AMI = acute myocardial infarction; CV = cardiovascular; MODS = multiorgan distress syndrome; RI = respiratory insufficiency.



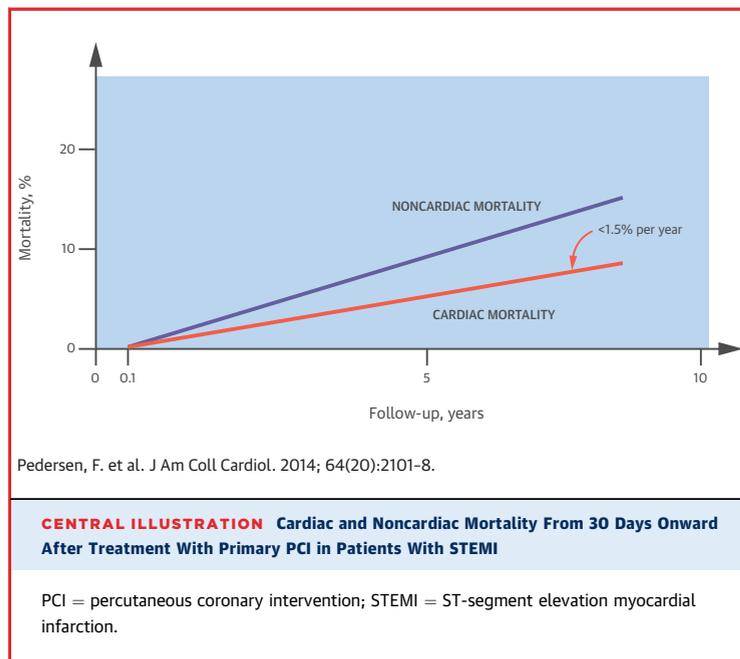
**FIGURE 1 Long-Term Mortality of Patients With ST-Segment Elevation Myocardial Infarction After Primary Percutaneous Coronary Intervention**

Kaplan-Meier curves showing all-cause mortality (orange line), noncardiac mortality (blue line), and cardiac mortality (red line).

unselected consecutive patient with STEMI undergoing primary PCI.

As might be expected, cardiac mortality was relatively high (>7%) during the first month in our all-comers cohort. The main causes of death in connection with the index event were cardiogenic shock, cerebral anoxia after cardiac arrest, and malignant arrhythmias. However, after the first month, cardiac mortality fell considerably (to <1.5 % per year), which suggests that patients who survive the acute phase of an STEMI treated with primary PCI have an excellent late cardiac prognosis and that late cardiac mortality in unselected all-comers is similar to that of selected participants of previous trials (18,19) (Central Illustration). Our findings encourage concentration of resources for prevention and treatment of cardiac complications primarily in the early phase of an STEMI.

Our findings also stress the importance of careful interpretation of interventional studies that focus on the effect of different treatment strategies on long-term clinical outcomes. Because late cardiac mortality may be quite low, very large patient sample sizes may be required to show any differences in treatment effects. However, noncardiovascular death is likely to be influenced by cardiovascular disease, and analyses of death causes are limited by difficulties



in detecting the exact cause of death, particularly in patients with multiple organ dysfunction and in patients who are found dead. Therefore, a continued focus on all-cause mortality in patients with STEMI receiving contemporary invasive and medical treatment seems warranted. Interestingly, as demonstrated by the Study of Platelet Inhibition and Patients Outcomes investigators (20), it seems possible to interfere with mortality, even after 1 month, by varying medical regimens. In contrast to our cohort, PCI was performed within 12 h of symptom onset in <math><75\%</math> of STEMI patients in that trial.

The other main findings of our study were that noncardiac causes of death were rare in the early phase after STEMI treated with primary PCI but considerably more frequent later, and that late noncardiac causes of death were mainly due to malignancies and lung diseases, including pneumonia.

Cardiac rehabilitation programs probably play a role in the explanation of the low cardiac mortality after primary PCI for STEMI, but legislation, funding, and guidelines seem insufficiently implemented in many regions of Europe (21). In Denmark, routine visits to outpatient clinics and general practice are free of charge for patients, and local hospitals (to which patients are transferred after primary PCI) are required by law to offer personalized secondary prevention. However, we were unable to evaluate whether the low cardiac mortality seen after primary PCI of our patients was due to particular effective secondary prevention programs. On the contrary, underuse of recommended medical

treatment after STEMI has been documented in our country (22,23).

The majority of our patients had a history of smoking, and it has been suggested that habitual smokers have improved early survival after an acute myocardial infarction. However, it is also well known that smokers are at greater risk for development of cancer and other pulmonary disease during follow-up. Although this issue might be relevant to the low early cardiac mortality and late noncardiac mortality in our study, our data do not allow an assessment of the proportion of habitual smokers, and recent studies do not support the existence of a “smoker’s paradox” (24,25).

The present patient cohort included participants enrolled in randomized trials evaluating possible improvements of the primary PCI procedure, trials with different inclusion criteria and varying 30-day and 1-year mortality rates (from 1% to 10%) (6-11). In addition, post-PCI mortality might be influenced not only by medication but also by anatomic and procedural variables (16,17,26). A Swedish registry reported a successive decrease in 30-day and 1-year mortality rates over time, and Terkelsen et al. (28) studied subgroup mortality rates after primary PCI in a Danish patient cohort and found an association with field triage and emergency services transportation time (27,28). In our center, which covered primary PCI for southeastern Denmark, field triage was initiated after completion of the DANAMI-2 trial (DANish trial in Acute Myocardial Infarction-2), and a fully developed field triage organization has been in place since 2003 (29,30). Subsequently, primary PCI has been offered to all patients with STEMI, independent of age, socioeconomic status, or place of residence. Every ambulance and helicopter is equipped to perform electrocardiographic and telemedicine transmission of electrocardiograms of all patients with acute-onset chest pain to the regional PCI center to facilitate field triage. Together with a limited geographic size and a well-developed infrastructure, these facilities might explain our study’s relatively low mean time delays to primary PCI (Table 1).

The leading cause of cardiac death in the present study was cardiogenic shock and its consequences, including multiple organ dysfunction syndromes (Tables 2 and 3). Cardiogenic shock affects 5% to 10% of patients with acute myocardial infarction and remains associated with high mortality, despite successful revascularization and use of intra-aortic balloon pump (31). It is well known that percutaneous left ventricular assist devices seldom completely restore cardiac output, and it remains unknown whether a left ventricular assist device that

completely restores cardiac output in patients with cardiogenic shock will increase myocardial salvage and reduce early cardiovascular death, when used as a bridge to effective revascularization in the acute phase of myocardial infarction (32,33). Contemporary antithrombotic regimens might tend to reduce all-cause mortality in the early phase of an STEMI at the cost of increased frequency of bleeding complications. Whether these early changes will translate into improvement of long-term prognoses or be counterbalanced by other complications will be elucidated by future surveys.

**STUDY LIMITATIONS.** In general, the completeness of per procedural data was relatively high, whereas the completeness of time delay and some risk factor data was lower, which might influence the results of the survival analysis. Thus, to reduce the risk of bias from missing data, hypertension and body mass index were not included in the multivariate survival analysis.

## CONCLUSIONS

Cardiac deaths after primary PCI occur mainly within the first month. The main causes of early death are

cardiogenic shock, anoxic brain damage due to cardiac arrest, and malignant arrhythmias. Beyond 1 month, cardiac mortality declines to <1.5% per year. Noncardiac causes are responsible for the majority of late deaths in these patients.

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## PERSPECTIVES

**COMPETENCY IN MEDICAL KNOWLEDGE:** Deaths of cardiac cause after primary PCI among patients with STEMI occur mainly within the first month. Those surviving beyond the first month generally have a lower cardiac mortality rate of <1.5% annually.

**TRANSLATIONAL OUTLOOK:** Large studies of risk factor interventions to improve outcomes among survivors of STEMI must focus on noncardiac causes of death.

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