Trends in Acute Coronary Heart Disease Mortality, Morbidity, and Medical Care From 1985 Through 1997

The Minnesota Heart Survey

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Background—Coronary heart disease (CHD) mortality continued to decline from 1985 to 1997.

Methods and Results—We tabulated CHD deaths (ICD-9 codes 410 through 414) in the Minneapolis/St Paul, Minnesota, area. For 1985, 1990, and 1995, trained nurses abstracted the hospital records of patients 30 to 74 years old with a discharge diagnosis of acute CHD (ICD-9 codes 410 or 411). Acute myocardial infarction (AMI) events were validated and followed for 3-year all-cause mortality. Between 1985 and 1997, age-adjusted CHD mortality rates in Minneapolis/St Paul fell 47% and 51% in men and women, respectively; the comparable declines in US whites were 34% and 29%. In-hospital mortality declined faster than out-of-hospital mortality. The rate of AMI (ICD-9 code 410) hospital discharges declined almost 20% between 1985 and 1995, whereas the discharge rate for unstable angina (ICD-9 code 411) increased substantially. The incidence of hospitalized definite AMI declined ~10%, whereas recurrence rates fell 20% to 30%. Three-year case fatality rates after hospitalized AMI decreased consistently by 31% and 41% in men and women, respectively. In-hospital administration of thrombolytic therapy, emergency angioplasty, ACE inhibitors, β-blockers, heparin, and aspirin increased greatly.

Conclusions—Declining out-of-hospital death rates, declining incidence and recurrence of AMI in the population, and marked improvements in the survival of AMI patients all contributed to the 1985 to 1997 decline of CHD mortality in the Minneapolis/St Paul metropolitan area. The effects of early and late medical care seem to have had the greatest contribution to rates during this time period. (Circulation. 2001;104:19-24.)

Key Words: heart diseases ■ myocardial infarction ■ incidence ■ survival

Age-adjusted mortality from coronary heart disease (CHD) in the United States has declined continuously between 1968 and 1997. During 1985 and 1997, CHD mortality declined ~30% in both men and women, but CHD has remained the leading cause of death in the United States. Monitoring total and in- and out-of-hospital CHD death rates, the incidence and recurrence rates of acute myocardial infarction (AMI), changes in diagnostic practices, and changes in medical care is important if we are to understand the nature of this dramatic decline, with a view toward continuing it.

Previous reports from the Minnesota Heart Survey (MHS)2-4 documented a substantial improvement in short- and long-term survival of hospitalized definite AMI in 1980 versus 1970, no further improvement in 1985 versus 1980, and substantial improved survival of AMI patients between 1985 and 1990. Both primary prevention and medical care played major roles in reducing CHD mortality between 1985 and 1990. We hypothesized that implementing medical technologies shown to be efficacious in clinical trials has led to reduced mortality after AMI since 1990. Among these important technologies were primary angioplasty and stent placements, which were rare before 1990, and continuing improvements in pharmacological therapy since 1990. Because reperfusion therapy limits the size and extent of the infarction, we further hypothesized that the increased use of thrombolytic therapy in 1990 compared with 1985 would result in a reduced recurrence of AMI and associated mortality in subsequent years.

In this report, we systematically examined trends in CHD mortality, morbidity, and medical care in a recent 12-year period (1985 to 1997). The study population comprised all residents aged 30 to 74 years in a large metropolitan area (Minneapolis/St Paul, Minn) and surrounding suburbs.

Methods

Study Population and Mortality Statistics

The 7-county metropolitan area of the Twin Cities (Minneapolis and St Paul) in Minnesota had a population of 2.29 million according to...
the 1990 census. In 1990, the target population for this study included 550,719 men and 576,690 women aged 30 to 74 years. The Twin Cities metropolitan area population in this age range is overwhelmingly white (>94% in both 1980 and 1990), with a median age for both sexes of 44 to 45 years in 1985, 1990, and 1995.

Data tapes containing death certificate information from the Minnesota Department of Health were obtained annually. Information on the cause of death, age, sex, place of residence, and location of death was used to compute stratum-specific counts of CHD deaths (ICD-9 codes 410 to 414 for underlying cause of death) for Twin Cities residents. Persons who died outside a hospital, were dead on arrival to a hospital, or died in a hospital emergency department were considered out-of-hospital deaths. A previous study of out-of-hospital deaths in the Twin Cities indicated high levels of sensitivity and a positive predictive value for death certificate diagnoses of ischemic heart disease.5 Year-specific population denominators were obtained from the census data for 1970, 1980, and 1990; intercensal years were interpolated, and official census estimates were used for 1991 through 1997.

CHD death rates for whites in the United States, standardized to the projected age distribution in 2000, were obtained from the National Heart, Lung, and Blood Institute (Thomas Thom, BA, personal communication; May 2, 2000).

Hospitalized Acute CHD Data Collection
For 1985, 1990, and 1995, we obtained listings of all patients 30 to 74 years old who were discharged from the Twin Cities metropolitan area hospitals with a code of acute CHD among any of the discharge diagnoses. The target ICD-9 codes were 410 (AMI) and 411 (other acute and subacute forms of ischemic heart disease). We did not include as a target code an ICD-9 410.x2 code (x meaning any single digit), which was introduced in the late 1980s. This rubric identifies an uncomplicated rehospitalization for follow-up treatment for an infarction that had occurred within the previous 6 weeks, rather than a new infarction.4 Definite AMI, as defined below, was rare in these discharges (n=139 in 1990); it was diagnosed in only 25% of cases (35 persons) compared with 90% of individuals with a 410.x1 in the first position among discharge diagnoses (n=1,514 in 1990) and 72% of individuals with a 410.x1 in any other position (n=370 in 1990). Hospitals (31 of 31 in 1985, 25 of 25 in 1990, and 22 of 23 in 1995) provided the requested listings. The one omitted hospital in 1995 was a very small hospital that had <0.25% of the 1990 discharges from these sampling frames, we randomly selected 50% samples of men and women in 1985, a 50% sample of men and a 100% sample of women in 1990, and a 40% sample of men and an 80% sample of women in 1995. To enhance the efficiency of AMI case finding in 1995, full abstraction of sampled ICD-9 411 codes was restricted to a 15% random subsample, plus any 411 discharge in which any creatine kinase-MB (CK-MB) value exceeded the hospital-specific upper limit of normal.

The medical records of the selected hospitalizations were abstracted by trained nurses according to a written protocol. Information was obtained on signs and symptoms, medical history, cardiac enzyme levels, clinical complications, therapy, and (when available) autopsy results. Up to 4 ECGs were photocopied and coded according to the Minnesota Code6; in 1995, ECGs were only coded in the ICD-9 410 patients in whom enzyme findings were equivocal. The study protocol was approved by the University of Minnesota Institutional Review Board.

To maintain standardized identification of AMI cases over time, we applied a computer-based diagnostic algorithm to all abstracted acute CHD hospitalizations in each year using peak cardiac enzyme levels, Minnesota ECG codes, and autopsy findings. A case was declared a definite AMI according to the following algorithm: if CK-MB was above its upper limit of normal for the given hospital; failing this, if both creatinine kinase and lactate dehydrogenase were above twice their upper limit of normal; failing this, if a new Q-wave was detected (among ICD-410 discharges); and failing this, on the basis of positive findings at autopsy. Abnormal lactate dehydrogenase was downgraded when there was evidence of liver cirrhosis; abnormal creatinine kinase was downgraded when preceded by surgery, and abnormal CK-MB was downgraded when preceded by heart surgery. In 1995, we did not examine enzymes other than CK-MB among ICD-9 411 cases; this was justified by the observation that only 2 of 559 ICD-9 411 cases with abnormal enzymes in 1985 and 1990 were identified solely by having both creatinine kinase and lactate dehydrogenase levels greater than twice the upper limit of normal. Deaths that occurred within 48 hours of hospitalization and were not validated by the diagnostic algorithm (n=96) were further reviewed by a physician; if judged to be probable or definite AMI, they were classified as definite AMI. In each study year, incident (first-ever) and recurrent AMI were distinguished by searches of up to 2 prior hospitalization records. An explicit statement in the hospital record was used to classify 95% of cases as incident or recurrent; the remaining 5% of cases were assumed to be incident AMI.

Jacobs et al7 developed the Predicting Risk of Death In Cardiac disease Tool (PREDICT) score for event severity on the day of admission. The score is a weighted sum of 15 abstracted indicators of cardiovascular disease history, shock, congestive heart failure, renal function, the Charlson comorbidity index, and age. As used in this article, the ECG was omitted from the PREDICT score. In 1985 and 1990 data, 6-year mortality rates after hospitalized AMI ranged from 4% for a PREDICT score of 0 to >80% for a PREDICT score ≥16.

Mortality Follow-Up
The patient’s vital status at the time of hospital discharge was ascertained from the medical record. Vital status after hospital discharge was determined by computer linkage with the Minnesota Death Index, a system that has 98% agreement with the National Death Index.8 Information on death was available through 1998, allowing for the evaluation of 3-year survival in all patients. All-cause death was considered the case fatality end point.

Statistical Methods
CHD mortality rates for whites in the United States were compared with corresponding rates in the Twin Cities; the latter were smoothed using a 3-year moving average.

We computed several types of rates, all of which were person-based (ie, counting only one of multiple events per patient in a given year). They were the hospital discharge rate of acute CHD (ICD-9 410 and 411 combined), the discharge rate of AMI alone (ICD-9 410), and the discharge rate of AMI as the primary diagnosis (ICD-9 410 in the first position). Next, we computed the overall attack rate and incident rate of definite hospitalized AMI. Finally, to take into account out-of-hospital CHD deaths (many of which are due to AMI), we combined out-of-hospital CHD deaths with definite hospitalized AMI events to achieve an overall estimate of the population rate of acute CHD.

Sex-specific rates were age-adjusted to the 1990 US population distribution by the direct method. The statistical significance of changes between 1985 and 1995 was assessed using Poisson regression.9 Trends in mortality after definite AMI were evaluated by computing relative odds of death in 1990 and 1995 compared with 1985 after adjusting for age.10 The statistical significance of changes in acute medical care was determined using χ² tests. All reported probability values are 2-tailed.

Results
Trends in CHD Mortality
Over the last quarter century, CHD mortality declined continuously in the Twin Cities, as it did in US whites (Figure 1). In the United States, the decline averaged ∼3% annually from 1970 and totalled 50% to 55% by 1997, assuming that most of the change between 1978 and 1979, when the ICD-8 coding system switched to ICD-9, was artifactual. In the Twin Cities, the rate of decline was somewhat faster, ∼3% to 4% per year through the mid-1980s, and it accelerated to
5.5% between 1985 and 1997. It totalled 70%, with a 50% decline between 1985 and 1997 alone. The death rate from all noncardiovascular causes remained virtually unchanged during the same period (data not shown).

The decline in the CHD mortality rate in the Twin Cities among 30- to 74-year-olds occurred both out-of-hospital and in-hospital, but since 1985, the percent decline was greater for in-hospital mortality in both sexes (Figure 2). In men, the out-of-hospital death rate declined 24% between 1985 and 1991 and 30% between 1991 and 1997, whereas the in-hospital death rate declined 43% and 39%, respectively. In women, the out-of-hospital CHD death rate declined 20% between 1985 and 1991 and 30% between 1991 and 1997, whereas the in-hospital death rate declined 31% and 49%, respectively.

**Trends in Acute CHD Rates and Severity**

The total number of abstracted acute CHD discharges (ICD-9 410 or 411) was 2784, 4097, and 3615 in 1985, 1990, and 1995, respectively. Trends in age-adjusted acute CHD rates between 1985 and 1995 are shown in Table 1. AMI (ICD-9 410, whether as the first diagnosis or any discharge diagnosis) declined by ~20% in both men and women. This decline was apparent in both 5-year periods (1985 to 1990 and 1990 to 1995). In contrast, unstable angina (ICD-9 411) hospital discharge rates increased by 56% in men and 30% in women between 1985 and 1995.

Hospitalized definite AMI in 30- to 74-year-old Twin Cities residents declined by ~5% between 1985 and 1990 and by ~10% between 1990 and 1995 (Table 1). Findings were similar for men and women. The median age of AMI patients was 60, 61, and 60 years in 1985, 1990, and 1995, respectively, in men and 66, 66, and 64 years in women, respectively. Declines in incident AMI were seen in men in both 5-year periods but in only the first period in women. Substantial declines in recurrent AMI were observed, particularly in the latter period. When we combined out-of-hospital CHD deaths with hospitalized definite AMI cases (an estimate of total AMI), the rate declined by 21% over 10 years in both sexes.

Hospital record-keeping has improved steadily since 1985. In hospitalized definite AMI patients, an age-adjusted average of 2.4 of 15 targeted historical and clinical items were not recorded in 1985, 1.7 items were not recorded in 1990, and 1.0 items were not recorded in 1995. A similar amount of information was missing for men and women, and differences between years were all highly significant. Event severity (mean PREDICT score), after adjustment for age and sex, was unchanged among recurrent cases. Among incident cases, event severity was constant between 1985 and 1990 but declined by 0.41 points between 1990 and 1995 (P=0.005).

**Trends in Mortality After Hospitalized Definite AMI**

Three-year mortality curves for definite hospitalized AMI demonstrated substantial improvements in the long-term survival of AMI in both men and women between 1985 and 1995 (Figure 3). There were substantial declines in case fatality, both soon after admission and through the 3 years of follow up. The age-adjusted 3-year death rate was 22% higher for women than for men in 1985. After a decline of 31% in men and 41% in women between 1985 and 1995, 3-year death rates were similar for both sexes. These favorable trends were evident for both 28-day case fatality and 3-year mortality among 28-day survivors and in incident and recurrent cases (Table 2). Declines in 3-year mortality among 28-day survivors and in 3-year mortality among incident cases seemed to be greater in women than in men. By 1995, the 28-day case fatality among hospitalized definite AMIs was 7% to 10%. Further adjustment for event severity on the day of admission had little impact on these results.

Hospitalized definite AMI patients in 1995 who subsequently died within 3 years were less likely to have a CHD diagnosis (ICD-9 410 to 414) as the underlying cause of death compared with their 1985 counterparts (54% versus 67%; P<0.001). Stroke, other forms of cardiovascular disease, and cancer all increased as causes of death.

**Trends in Medical Care of Hospitalized Definite AMI**

Between 1985 and 1995, major changes occurred in the age- and severity-adjusted frequencies of medication use during
AMI hospitalization (Table 3), with increases in β-blockers, ACE inhibitors, high-dose heparin, and aspirin. More than 90% of AMI patients received aspirin, and >80% received high-dose heparin in 1995. Use of calcium-channel blockers and lidocaine decreased. Rates of diagnostic and therapeutic procedures changed markedly. Angiography and echocardiography use both increased to ~60% in 1995, and the frequency of coronary bypass graft surgery doubled in men but remained stable in women. Angioplasty use increased to 1 in 3 patients in 1995, the majority of which was performed within 24 hours of admission (emergency angioplasty). Stents were placed in ~5% of patients in 1995; this procedure was not queried before 1995. A small but increasing percentage of incident patients had a prior history of either coronary bypass graft surgery or angioplasty (after age adjustment, men: 4.5% in 1985, 7.2% in 1990, and 8.4% in 1995; women: 0.8% in 1985, 3.0% in 1990, and 5.2% in 1995). Thrombolytic therapy increased from 1985 to 1990 but showed little change thereafter. A clinical reading of the ECG in all AMI patients in 1995 revealed ST elevation in 94% of those receiving thrombolysis in 1995 (373 of 396 patients).

Between 1985 and 1995, the geometric mean length of stay for definite AMI patients in the Twin Cities decreased by ~40%, from 8.3 to 5.0 days in men and from 8.4 to 5.5 days in women (P<0.001).

Discussion

The decline in CHD mortality rates in the Twin Cities during the 1970s and 1980s largely mirrored the national trends, but there is evidence that the decline since the mid-1980s has been faster in the Twin Cities. In general, time trends in out-of-hospital CHD mortality likely echo the success of primary prevention measures, whereas trends in in-hospital CHD mortality are more likely tied to early medical care. We previously reported4 that between 1985 and 1990, the rate of decline of in-hospital mortality in the Twin Cities accelerated considerably and was greater than the decline of out-of-hospital mortality. These patterns continued through 1997.

Although rates of hospitalized AMI declined between 1985 and 1995, rates of unstable angina continued to increase steadily. This may reflect real increases in the occurrence of unstable angina, either because of improved AMI survivorship or aborted AMI in an era of medical care innovation in the treatment of CHD. It is possible that patients with angina pectoris were more likely to have been hospitalized and aggressively treated in 1995 than in the past. In addition, increased use of acute interventions may have resulted in aborted AMIs that were labeled unstable angina. In the combined 1990 and 1995 data, we observed 254 patients with definite AMI who were coded to ICD-9 411; of these, 13 (5%) had thrombolytic therapy. Nonetheless, the absolute increases in unstable angina rates were similar in magnitude to the decreases observed in rates of stable angina and other chronic coronary disease (ICD-9 412 to 414; data not shown), suggesting the possibility of diagnostic coding drift.11 Such diagnostic drift might reflect greater reimbursement for the ICD-9 411 category.

The decline in the rate of hospitalized definite AMI was consistent with a concurrent favorable trend in the population risk profile, as reported in Minnesota4 and elsewhere.12–14 However, this decline was more pronounced in the category of recurrent events, which is less likely to reflect the impact of risk factors. The increased use of thrombolytic therapy in 1990 compared with 1985 may have resulted in a reduced recurrence of AMI by 1995.

### Table 1. Age-Adjusted Rates of Acute CHD Among Twin Cities Residents, 30 to 74 Years of Age, 1985 to 1995*

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<td>410 in first position</td>
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<td>411 (no 410)</td>
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<td>134</td>
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<td>410 and 411</td>
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<td>879</td>
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<td>322</td>
<td>310</td>
<td>322</td>
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<td>Hospitalized definite AMI</td>
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<td>Attack rate</td>
<td>469</td>
<td>438</td>
<td>394</td>
<td>163</td>
<td>155</td>
<td>137</td>
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<td>Incident</td>
<td>309</td>
<td>289</td>
<td>272</td>
<td>111</td>
<td>106</td>
<td>105</td>
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<tr>
<td>Recurrent</td>
<td>161</td>
<td>149</td>
<td>122</td>
<td>53</td>
<td>49</td>
<td>32</td>
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<td>Out-of-hospital CHD death (ICD-9 410 to 414) or definite hospitalized AMI attack rate</td>
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<td>619</td>
<td>561</td>
<td>491</td>
<td>213</td>
<td>188</td>
<td>168</td>
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Values are n per 100 000.

*Age-adjusted to 1990 US population.

Figure 3. Age-adjusted 3-year case fatality after definite hospitalized AMI.
There are 2 other sources of data on trends in acute CHD rates in the late 1980s and early 1990s in the United States. Rosamond and colleagues, using data on 35- to 74-year-olds from 4 communities (including the northwestern suburbs of Minneapolis), reported that between 1987 and 1994, the age-adjusted attack rate of definite or probable hospitalized AMI fell by 10% to 20% in white men and women, with a greater decline in recurrence (versus incidence) rates in both sexes. It is important to note that parallel increases of 10% were found in black men and women. A report from Worcester, Massachusetts, which examined trends in that community through 1995, observed a small decline of 3% to 5% in the age-adjusted incidence of hospitalized definite AMI over the entire period between 1984/1986 and 1993/1995.

The current report documents substantially improved survival of hospitalized AMI patients. Between 1985 and 1995, both the 28-day and 3-year risk of death after hospitalized definite AMI was reduced by ≈35% in both men and women. This and the substantial decline in out-of-hospital death played major roles in the recent continued decline of CHD mortality. The improved 28-day AMI survival, reduced in-hospital death rates, and improved 3-year survival likely further contributed to the reduced out-of-hospital death rates. Furthermore, for those who died within 3 years of hospitalization, CHD was less likely to have been the underlying cause of death. This may reflect improved treatment of CHD, allowing people to survive until another cardiovascular or noncardiovascular illness occurred.

Our findings of substantially improved survival of hospitalized AMI patients are similar to those reported by others. The 4-community study of 35- to 74-year-olds found age-adjusted reductions in 28-day mortality between 1987 and 1994 of 30% and 60% in white men and women, respectively. However, the trends for black men and women showed little or no improvement. The Worcester community study found an almost 40% decrease in multivariable-adjusted hospital case fatality of AMI between 1986/1988 and 1993/1995, but no difference in 2-year death.

Trends in medical care for AMI, similar to those reported here, have been reported elsewhere. The dramatic changes in therapy for hospitalized AMI, with relatively little change in severity, as documented in this report, support an important contribution of early medical care to the decline of CHD mortality rates. Previous analyses suggested that some of the decline in case fatality was due to increased use of thrombolytic therapy. Other therapies with established impact on AMI survival, such as aspirin, ACE inhibitors, β-blockers, anticoagulants, and emergency angioplasty were also used much more frequently in 1995 than in 1985. Two striking features of the trends in the short-term care of hospitalized AMI presented here are (1) their consistency with published results of major clinical trials in the same period and (2) the little to no sex difference in medical intervention in AMI patients in 1995.

The more rapid decline in age-adjusted CHD mortality between 1985 and 1997 in the Twin Cities (49%) compared
with US whites (30%) suggests that either the incidence of acute CHD declined faster in the Twin Cities or that medical care of hospitalized AMI patients underwent a greater improvement in the Twin Cities than across the United States as a whole. We did observe reductions in incident AMI and out-of-hospital death that were suggestive of ongoing improvements in primary prevention. However, medical care, as suggested by declining hospital case fatality, recurrent AMI, and 3-year mortality, seems to account for a greater fraction of the improvement. Effective treatment with thrombolysis, other pharmacological therapy, and emergency angioplasty is expensive and dependent on the local medical care delivery system.

The greatest limitation of this study concerns the ability to define definite AMI consistently across study years. This consistency is the groundwork for inferences about trends. In more recent years, CK-MB enzyme mass rather than activity was assayed, but we thought that it was reasonable to assume an equal likelihood of exceeding the hospital- and method-specific upper limit of normal, regardless of the assay type. We considered it unreasonable to base inferences on the extent of CK-MB elevation, because the mass assay produces a greater elevation above the upper limit of normal than the activity assay. A related issue was whether to count AMIs that occurred after coronary artery bypass grafting or percutaneous transluminal coronary angioplasty procedures.26,27 This issue was addressed by sensitivity analysis. The time trend in AMI was qualitatively very similar whether all, some, or none of the grafting procedures were included as definite AMIs and whether marginally elevated CK-MB was downgraded after angioplasty or not. Another limitation is the regional nature of this study, which pertains primarily to whites. Studies in other regions and of minorities are of interest.

We conclude that the approximate 50% decrease in the CHD mortality rate between 1985 and 1997 among Twin Cities residents aged 30 to 74 years can be explained by declining out-of-hospital deaths, declining occurrences of hospitalized AMI in the population, and improved survival of AMI patients. The dramatic improvements in short- and long-term case fatality after hospitalized AMI may have been due to greater use of beneficial therapies, including thrombolytic agents, antiplatelet therapy, aspirin, beta-blockers, and emergency angioplasty.

Acknowledgments

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