Long-Term Trends in Myocardial Infarction Incidence and Case Fatality in the National Heart, Lung, and Blood Institute's Framingham Heart Study

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Background—Whereas the prevalence of coronary heart disease risk factors has declined over the past decades in the United States, acute myocardial infarction (AMI) rates have been steady. We hypothesized that this paradox is due partly to the advent of increasingly sensitive biomarkers for AMI diagnosis.

Methods and Results—In Framingham Heart Study participants over 4 decades, we compared the incidence and survival rates of initial AMI diagnosis by ECG (AMI-ECG) regardless of biomarkers with those based exclusively on infarction biomarkers (AMI-marker). We used Poisson regression to calculate annual incidence rates of first AMI over 4 decades (1960 to 1969, 1970 to 1979, 1980 to 1989, and 1990 to 1999) and compared rates of AMI-ECG with rates of AMI-marker. Cox proportional-hazards analysis was used to compare AMI case fatality over 4 decades. In 9824 persons (54% women; follow-up, 212 539 person-years; age, 40 to 89 years), 941 AMIs occurred, including 639 AMI-ECG and 302 AMI-marker events. From 1960 to 1999, rates of AMI-ECG declined by ≈50% and rates of AMI-marker increased ≈2-fold. Crude 30-day, 1-year, and 5-year case fatality rates in 1960 to 1969 and 1990 to 1999 were 0.20 and 0.14, 0.24 and 0.21, and 0.45 and 0.41, respectively. Age- and sex-adjusted 30-day, 1-year, and 5-year AMI case fatality declined by 60% in 1960 to 1999 (P for trend <0.001), with parallel declines noted after AMI-ECG and AMI-marker.

Conclusions—Over the past 40 years, rates of AMI-ECG have declined by 50%, whereas rates of AMI-marker have doubled. Our findings offer an explanation for the apparently steady national AMI rates in the face of improvements in primary prevention. **(*Circulation*. 2009;119:1203-1210.)**

Key Words: biomarkers ■ electrocardiography ■ epidemiology ■ myocardial infarction

During the past 4 decades, death rates from coronary heart disease (CHD) have declined by >60%.[1–4] Between nearly one half to upwards of three quarters of the decline in CHD mortality has been attributed to improvements in primary prevention and risk factor modification.[5–10] Awareness, treatment, and control of 3 key risk factors—hypertension, hypercholesterolemia, and smoking—have improved in recent decades.[1,11] Despite these improvements, hospitalization rates for acute myocardial infarction (AMI) have remained relatively stable over the past 5 decades.[1,4,12] The reasons for the paradoxical stability of AMI rates in the face of declining CHD risk factor prevalence are not clear.

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Whereas ECG criteria for AMI have not changed appreciably over the past 50 years, several different biomarkers of varying sensitivity and specificity have been introduced for the detection of AMI. Early on, serum glutamic oxalacetic transaminase and lactic dehydrogenase were used, in conjunction with clinical information, to diagnose AMI. In more recent times, serum markers of myocardial cell damage, including creatine phosphokinase (CPK), lactic dehydrogenase isoenzymes, CPK-MB, and troponin, have been introduced sequentially to diagnose AMI and have been firmly

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incorporated into international guidelines for AMI case definition.[13] Compared with diagnosis based solely on history and ECG, AMI diagnosis based on serial biomarker measurements has substantially increased the detection of AMI cases.[14–16]

Previous investigations of US trends in AMI incidence comparing different diagnostic criteria have been hospital based and have encompassed limited time periods for their analysis.[14,17] The Framingham Heart Study, which has >50 years of physician-validated AMI data on a community-based cohort, offers a unique setting to study trends in AMI incidence and case fatality rates based on the following AMI diagnostic criteria: AMI by ECG diagnosis (AMI-ECG) regardless of biomarker elevation, which offers an unbiased assessment of long-term trends, and AMI by biomarker diagnosis (AMI-marker) in the absence of diagnostic ECG changes, which reflects changing methods in clinical practice. The sum of these 2 mutually exclusive approaches represents total AMI.

We hypothesized a priori that rates of AMI-ECG have declined in the long term (consistent with improvements in CHD risk factors) while the rates of AMI-marker have increased (owing to greater biomarker sensitivity), resulting in a relatively steady rate of total AMI incidence over a 40-year time interval. Accordingly, we analyzed 40-year trends in the incidence of first AMI and for the 2 mutually exclusive AMI subgroups of AMI-ECG and AMI-marker. Such an approach may shed light on the paradoxical stability of national AMI rates in the setting of improvements in CHD risk factors and declining rates of CHD mortality.

Secondarily, we assessed time-period trends in mortality after AMI and its subcomponents, AMI-ECG and AMI-marker. This analysis will help promote understanding of the relative effectiveness of secondary prevention efforts over time when considered in conjunction with analyses of time-period changes in the incidence of initial AMI, which reflect advances in primary prevention.

Methods

The Framingham Heart Study is a community-based prospective observational study that began in 1948, enrolling 5209 men and women in the original study cohort.[18] Original cohort members attended clinic examinations approximately every 2 years. In 1971, 5124 men and women enrolled in the Framingham Heart Study offspring cohort, which included the children and spouses of the children of the original cohort. Participant examinations for the offspring cohort occurred approximately every 4 to 8 years; the design and methodology have been described elsewhere.[19] This investigation included original and offspring cohort members.

We considered all original and offspring cohort members 40 to 89 years of age who were free of AMI (recognized and unrecognized) at their first Framingham clinic examination in each decade of study (1960s, 1970s, 1980s, 1990s). Our final sample size consisted of 9824 individuals. Each individual could enter the sample multiple times on the basis of eligibility for time period and age group. For example, a participant 35 years of age in 1960 would not contribute follow-up time to the first time period until he or she turned 40 in 1965, thereafter contributing 5 years. That participant would contribute 5 years to second time period and so on until the patient died or developed AMI. Similarly, a patient 75 years old in 1960 contributed at most 5 years to the last period. Participants provided written informed consent, and the study protocol was approved by the Boston University Medical Center Institutional Review Board.

Table 1. Characteristics of Framingham Heart Study Participants at the Start of Each Time Period of Study

	1960s	1970s	1980s	1990s
Men, n	1768	2205	2145	2147
Women, n	2366	2687	2662	2632
Mean age, y	53±8	56±10	60±12	60±13
Women, %	57	55	55	55
Total cholesterol, mg/dL	248±45	226±43	221±41	210±39
Systolic BP, mm Hg	136±23	136±22	133±20	133±21
Diastolic BP, mm Hg	84±12	82±11	79±10	78±10
Body mass index, kg/m^2	25.8±4.1	26.5±4.3	26.6±4.4	27.0±4.8
Glucose, mg/dL	82±23	106±18	95±39	99±30
Hypertension, %	43	47	47	48
Diabetes mellitus, %	4	8	10	8
Smoking, %	53	28	30	35

BP indicates blood pressure. Values are mean±SD when appropriate.

Risk Factor Assessment

At each routine clinic visit, participants underwent physical examination, 12-lead ECG, anthropometry, and laboratory assessment of vascular risk factors. Details on the ascertainment of risk factors have been previously described.[19] Participants with systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg (mean reading of 2 readings taken by an examining physician) or receiving medication for the treatment of hypertension were defined as having hypertension. Plasma glucose and total cholesterol were measured. Diabetes mellitus was defined (throughout the study period) as fasting plasma glucose ≥126 mg/dL, a nonfasting glucose of ≥200 mg/dL, or treatment with either insulin or hypoglycemic agents. Participants were considered to be current smokers if they smoked on average at least 1 cigarette per day during the year before examination.

Serum Biomarkers of MI

Several serum biomarkers were used for AMI diagnosis during the study time period. Specific diagnostic biomarkers and the decades during which they were used for AMI diagnosis in the Framingham Heart Study included the following: serum glutamic oxalacetic transaminase beginning in the mid-1950s, lactic dehydrogenase in the 1960s, CPK in the 1970s, CPK-MB and lactic dehydrogenase isoenzyme in the 1980s, and troponin in the late 1990s. We did not use prespecified cut points to determine biomarker elevation because variability was present in assays used in the various hospitals from which medical records were collected. Thus, we considered a biomarker elevated if it exceeded the reference limit provided by the hospital laboratory report at the time of AMI hospitalization, according to the available medical record/chart.

Ascertainment of AMI and AMI Case Fatality

Framingham Heart Study participants are under continuous surveillance for cardiovascular disease events and death. The surveillance process included physician-administered questions about cardiovascular events during each routine follow-up Framingham Heart Study clinic visit and a mailed health history update questionnaire (which, before the late 1990s, consisted of a brief questionnaire for those who had not attended examinations and, after the late 1990s, included detailed sections about interim cardiac events and hospitalizations). If a participant reported a possible interim event, all pertinent medical records were collected and reviewed by an events adjudication committee consisting of 3 physicians who reviewed all available hospitalization records, physician office visit notes, and pathology reports.[20] AMIs were diagnosed on the basis of ischemic

Table 2. Decade-Specific Incidence Rates of Overall AMI, AMI-ECG, and AMI-Marker per 10 000 Person-Years Among Men

	1960–1969	1970–1979	1980–1989	1990–1999
Overall AMI				
Age, y				
40–49	43.80	22.05	29.43	24.71
50–59	62.62	53.86	59.75	39.78
69–69	74.05	84.33	97.90	55.11
70–79	152.17	83.84	135.56	117.36
80–89	*	98.09	129.90	166.03
Events, n	130	143	190	144
AMI-ECG				
Age, y				
40–49	39.39	18.17	23.75	15.96
50–59	53.50	40.63	43.80	21.67
69–69	63.25	63.59	71.74	30.01
70–79	121.15	56.31	87.65	52.05
80–89	*	66.69	85.10	75.20
Events, n	112	105	131	72
AMI-marker				
Age				
40–49	4.41	3.88	5.68	8.75
50–59	9.12	13.23	15.94	18.11
69–69	10.80	20.73	26.15	25.11
70–79	31.01	27.53	47.92	65.31
80–89	*	31.40	44.79	90.84
Events, n	18	38	59	72

Person-years of observation: 122 560.
*No events for age group/time period.

Table 3. Decade-Specific Incidence Rates of Overall AMI, AMI-ECG, and AMI-Marker per 10 000 Person-Years Among Women

	1960–1969	1970–1979	1980–1989	1990–1999
Overall AMI				
Age, y				
40–49	6.17	3.96	4.18	3.99
50–59	10.34	11.35	9.95	7.53
69–69	23.51	34.16	31.35	20.05
70–79	51.17	35.96	45.96	45.21
80–89	*	85.53	89.53	130.00
Events, n	47	81	100	106
AMI-ECG				
Age, y				
40–49	5.55	3.26	3.37	2.57
50–59	8.84	8.56	7.29	4.10
69–69	20.09	25.76	22.97	10.91
70–79	40.74	24.15	29.72	20.05
80–89	*	58.15	58.66	58.88
Events, n	38	59	72	50
AMI-marker				
Age				
40–49	0.62	0.70	0.81	1.4
50–59	1.51	2.79	2.66	3.43
69–69	3.43	8.40	8.37	9.13
70–79	10.43	11.81	16.25	25.16
80–89	*	27.38	30.87	71.13
Events, n	9	22	28	56

Person-years of observation: 89 979.
*No events for age group/time period.

chest discomfort with diagnostic ECG changes (based on chart review) with or without diagnostic biomarker changes (AMI-ECG) or ischemic chest discomfort with diagnostic serum biomarkers of infarction but without diagnostic ECG changes (AMI-marker). ECG criteria for AMI included development of pathological Q waves of ≥0.04 seconds, often accompanied by ST elevation and followed by serial changes indicating a reversion of these ECG changes toward normal. We chose to exclude persons with unrecognized/silent AMI because it is impossible to determine the exact date of occurrence, which is assigned a midpoint between the last ECG without an abnormality and the first one manifesting Q-wave changes.

Case fatality was assessed within 30 days and at 1 and 5 years. For 1- and 5-year mortality, deaths occurring within the first 30 days were excluded from analysis. We did this to obtain a truer sense of how many "later" case fatalities occurred after AMI (because a large proportion of post-AMI deaths occur within 30 days of the index event as opposed to later). Furthermore, pathophysiologically "early" death resulting from AMI is likely different from "later" deaths.

Statistical Methods

Prevalence rates and means (±SD) of cardiovascular disease risk factors were calculated for the study sample at the first examination cycle in each decade of study. We used Poisson regression to calculate annual incidence rates of first AMI over 4 time periods (1960 to 1969, 1970 to 1979, 1980 to 1989, and 1990 to 1999) and compared rates of AMI-ECG with rates of AMI-marker. We tested for sex×age group, sex×time period, age group×time period, age group×AMI type, and time period×AMI type interactions for incidence rate trends; given multiple statistically significant probability values for these interactions, we present age- and sex-specific AMI incidence rates for each time period. Additionally, with small numbers of events for the oldest and youngest age groups, we provide trends in (log-transformed) event rates across the 4 time periods for the age groups of 50 to 59, 60 to 69, and 70 to 79 years for men and women separately. We calculated tests of trend for overall AMI, AMI-ECG, and AMI-marker across time periods, with the 1960s serving as the referent decade (using a model accounting for the interactions listed above). We used Cox proportional-hazards models to calculate age- and sex-adjusted case fatality curves and 30-day, 1-year and 5-year case fatality rates after all AMI, AMI-ECG, and AMI-marker for each of the 4 time periods (with 1960 to 1969 serving as the referent period). The follow-up period for case fatality was until the end of 2006. The assumption of proportionality of hazards was satisfied over the 5-year follow-up period after AMI (P for time to death×period interaction >0.32 for overall AMI, AMI-ECG, and AMI-marker). A 2-sided value of $P<0.05$ was considered to indicate statistical significance. All statistical analyses were performed with the use of the SAS statistical software (version 9.0; SAS Institute, Inc, Cary, NC).

Dr Levy had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors have read and agree to the manuscript as written.

Results

Characteristics of Study Sample

Study participant characteristics by decade are shown in Table 1. Of the 9824 participants, 54% were women;

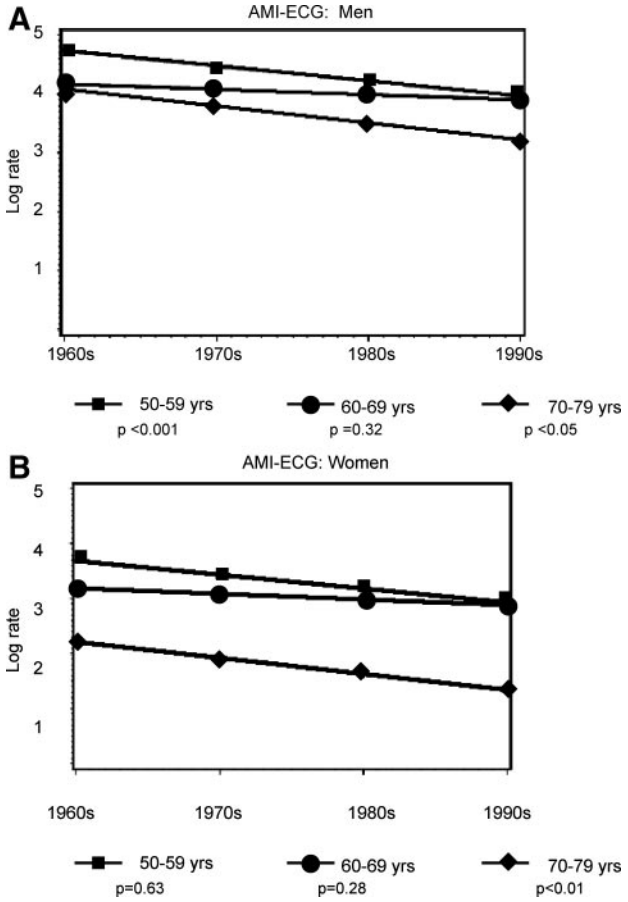

Figure 1. Temporal trends for age-range–specific incidence rates in AMI-ECG from 1960 to 1999 among men (A) and women (B).

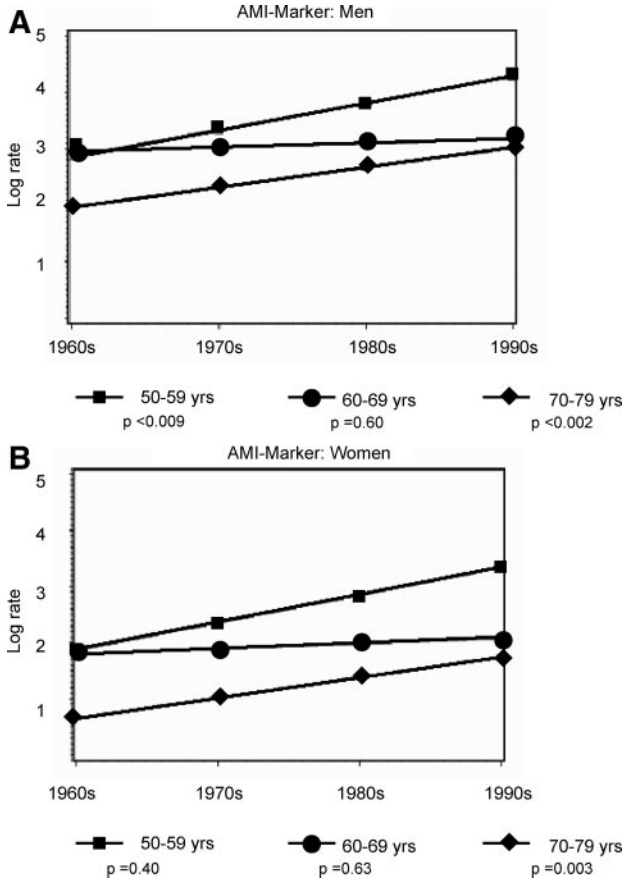

Figure 2. Temporal trends for age-range–specific incidence rates in AMI-marker from 1960 to 1999 among men (A) and women (B).

follow-up time was 212 539 person-years. The mean age of participants at the start of each time period ranged from 53 years in the 1960s to 60 years in 1990s. Smoking rates, total cholesterol concentrations, and systolic and diastolic blood pressures decreased from 1960 to 1999.

Trends in Overall AMI, AMI-ECG, and AMI-Marker Rates

Overall, 941 first AMIs occurred, including 639 AMI-ECG events (68%) and 302 AMI-marker events (32%). Age- and sex-specific incidence rate trends from the 1960s to the 1990s are presented in Tables 2 and 3 for men and women, respectively.

Rates of AMI-ECG declined by ≈50% and rates of AMI-marker doubled over the study period (Tables 2 and 3, Figures 1 and 2). Among men, statistically significant declines in AMI-ECG were noted in the age groups of 50 to 59 years (P for trend <0.001) and 70 to 79 years (P for trend <0.05) (Figure 1A). In women, statistically significant declines in AMI-ECG were noted among those 70 to 79 years of age (P for trend <0.01) (Figure 1B). Among men, statistically significant increases in AMI-marker were noted in those 50 to 59 and 70 to 79 years of age (P for trend <0.01 for both) (Figure 2A); in women, statistically significant increases in AMI-marker were noted among those 70 to 79 years of age (P for trend <0.01) (Figure 1B). Trends for AMI-ECG and AMI-marker were largely flat for the 60-to 69-year-old group.

Trends in 30-Day, 1-Year, and 5-Year AMI Case Fatality

Five-year case fatality rates after overall AMI decreased steadily from 1960 to 1999 (P for trend <0.001) (Table 4, Figure 3A). Trends in 5-year case fatality rates after AMI-ECG and after AMI-marker mirrored overall 5-year AMI case fatality trends (Figure 3B and 3C). Similarly, decreases were seen in 30-day and 1-year case fatality rates after overall AMI, AMI-ECG, and AMI-marker (Table 4). A particularly large shift was found toward decreased case fatality between the 1970s and 1980s (Figure 3A through 3C).

Discussion

Principal Findings

In a community-based cohort of 9824 men and women followed up for a 4-decade interval, we found that AMI-ECG rates declined ≈50% with a concomitant 2-fold increase in rates of AMI-marker. The 30-day, 1-year, and 5-year case fatality rates after overall AMI declined by 50% to 75% from 1960 to 1999, with parallel declines in case fatality after both AMI-ECG and AMI-marker over this period. We conclude that national MI trend data may be biased by a diagnostic drift resulting from the advent of diagnostic biomarker tests for

Table 4. Age-and Sex-Adjusted 30-Day, 1-Year, and 5-Year Mortality Rates for Overall AMI, AMI-ECG, and AMI-Marker Among Framingham Heart Study Participants

Outcome	1960–1969	1970–1979	1980–1989	1990–1999	*P*, Trend Test
At 30 d					
All AMI					
Deaths, n	35	45	47	34	
HR (95% CI)	Referent	0.66 (0.42–1.05)	0.45 (0.28–0.71)	0.27 (0.16–0.45)	<0.0001
AMI-ECG					
Deaths, n	32	38	35	23	
HR (95% CI)	Referent	0.71 (0.44–1.16)	0.50 (0.30–0.82)	0.38 (0.21–0.69)	0.0004
AMI-marker					
Deaths, n	3	7	12	11	
HR (95% CI)	Referent	0.72 (0.18–2.81)	0.46 (0.12–1.82)	0.22 (0.06–0.89)	0.006
At 1 y					
All AMI					
Deaths, n	(42)	(66)	(73)	(54)	
HR (95% CI)	Referent	0.83 (0.56–1.23)	0.58 (0.39–0.87)	0.35 (0.22–0.54)	<0.0001
AMI-ECG					
Deaths, n	37	49	55	29	
HR (95% CI)	Referent	0.80 (0.52–1.25)	0.68 (0.44–1.05)	0.42 (0.25–0.71)	<0.001
AMI-marker					
Deaths, n	5	17	18	25	
HR (95% CI)	Referent	1.11 (0.41–3.04)	0.43 (0.15–1.22)	0.31 (0.11–0.87)	<0.001
At 5 y					
All AMI					
Deaths, n	80	104	111	104	
HR (95% CI)	Referent	0.73 (0.54–0.98)	0.47 (0.35–0.64)	0.36 (0.26–0.50)	<0.006
AMI-ECG					
Deaths, n	70	78	72	46	
HR (95% CI)	Referent	0.70 (0.50–0.98)	0.47 (0.33–0.66)	0.36 (0.24–0.53)	<0.001
AMI-marker					
Deaths, n	10	26	39	58	
HR (95% CI)	Referent	1.06 (0.51–2.21)	0.59 (0.28–1.22)	0.45 (0.22–0.93)	0.001

HR indicates hazard ratio.

AMI; we were able to identify and quantify the possible magnitude of this effect within our study setting. These findings may explain the paradoxical stability of AMI rates in the United States despite concomitant improvements in CHD risk factors.

Temporal Trends in AMI

Several epidemiological studies conducted in United States have demonstrated steady rates of AMI from the 1970s to the 1990s,[4,12,17,21] whereas data from the World Health Organization Monitoring Trends and Determinants in Cardiovascular Disease (WHO-MONICA) project demonstrated modest declines in rates of AMI from 1985 to 1991.[22] Data from the Worcester Heart Attack Study similarly demonstrated modest declines in the incidence of first AMI.[23] Differences in study design and event ascertainment may have accounted for the differing results between these prior studies. In our study, particularly in men, overall AMI trends appear to be decreasing in a parallel fashion compared with AMI-ECG. In women, overall AMI rates were steady to decreased.

Defining AMI in population studies and clinical research is essential for accurate disease surveillance, clinical trial design and conduct, and healthcare resource allocation.[13,24,25] Several prior studies demonstrating trends in AMI rates have used international diagnostic codes (*International Classification of Diseases* [ICD]) for hospital discharges to identify AMI cases[21,26–28] and may be subject to "diagnostic drift".[29] Specifically, diagnostic coding of AMI during hospitalizations may have increased as a result of changes in reimbursement practices and by the use of more sensitive biomarkers of infarction.[24,29] Temporal-trend estimates of AMI based on ICD codes have shown steady rates[4,21] over the past several decades. In contrast, AMI-ECG rates in our study sample declined by ≈50% from 1960 to 1999. AMI-ECG represents a relatively "unbiased" estimate of AMI that has not been influenced by the advent of increasingly sensitive biomarkers of infarction in recent decades.[17] Not surprisingly, AMI-marker rates in our study increased over this same time period in a manner consistent with prior data in the WHO-MONICA

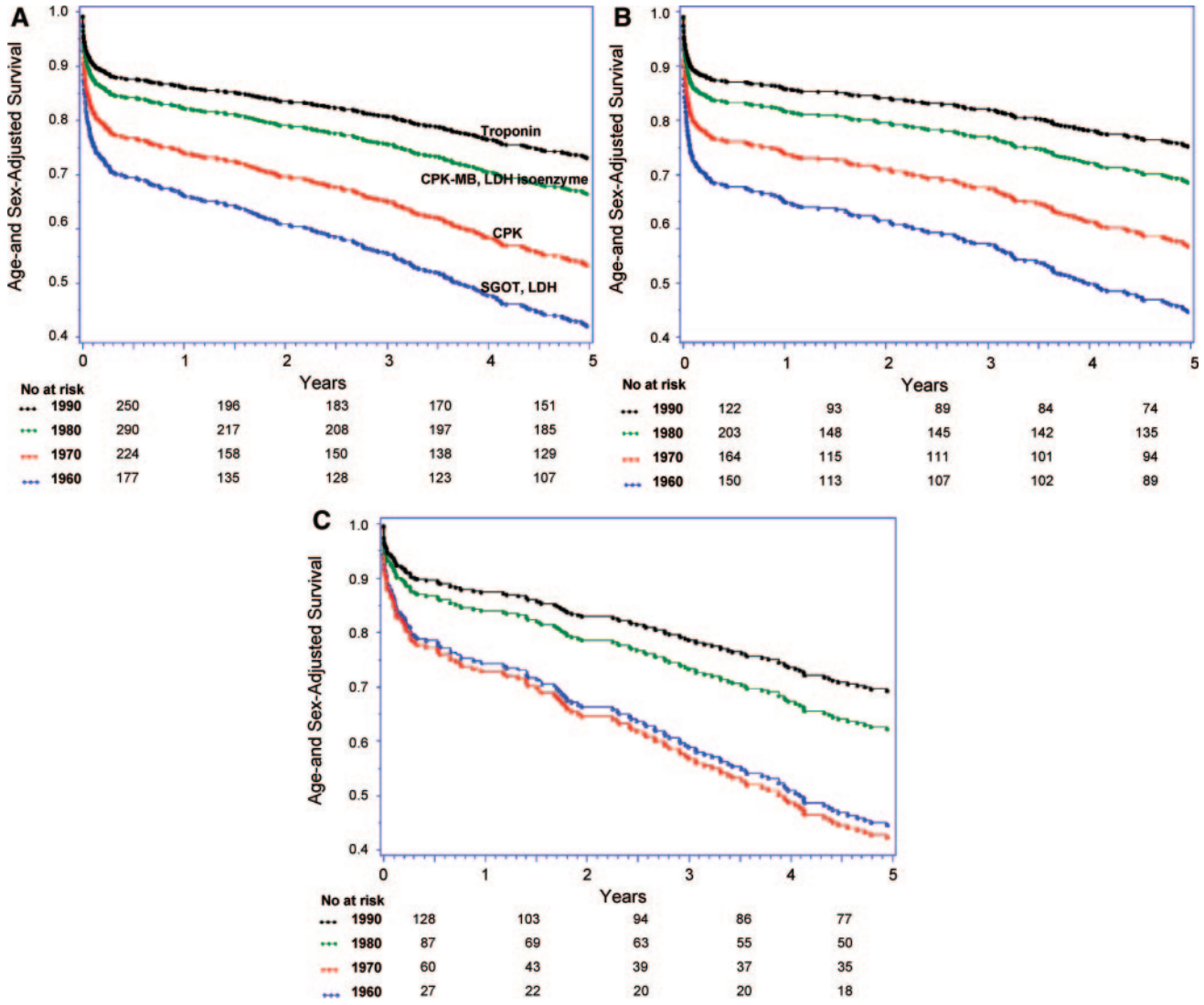

Figure 3. Up to 5-year case fatality after overall AMI (A), after AMI-ECG by decade (B), and after AMI-marker by decade (C), with the major biomarker used during each decade.

study, which showed higher AMI rates using biomarker-based definitions (troponin) compared with ECG-based definitions.[15] Similarly, an investigation in the Minnesota Heart Study demonstrated a 50% increase in AMI detection in 1980 when CPK and CPK-MB information was added to the Minnesota Heart Study AMI diagnostic algorithm.[14] Additional studies have mirrored these findings, showing that troponin-influenced AMI diagnosis has increased the AMI detection rate compared with AMI diagnosis based on CPK-MB and total CPK.[30,31] Our data extend these findings by demonstrating that biomarker-influenced AMI diagnosis has yielded a doubling in rates of AMI-marker over the 40-year period spanning 1960 to 1999.

The proportion of overall AMI diagnosed by ECG (68%) was similar to figures reported in a prior report from the Minnesota Heart Survey.[17] That investigation concluded that incident AMI-ECG rates were steady from 1975 to 1985 and declined from 1985 to 1995.[17] We extend these findings by providing data from 2 additional decades of observation. Our results demonstrate a 50% to 60% decline in AMI-ECG rates from 1960 to 1999. AMI-ECG likely represents a more advanced form of MI; declines demonstrated in out-of-hospital sudden cardiac death[2,12,32–34] (attributable to improved primary prevention efforts)[32] have likely contributed to some degree to the declines in the incidence of AMI-ECG.

Another possible explanation for the decline in AMI-ECG and the relative rise in AMI-marker may have to do with decreases in time from the onset of symptoms to hospital presentation and treatment (data from the National Registry of Myocardial Infarction),[35] which are thought to be due to public health education efforts and guideline implementation, which have collectively stressed the need to decrease door-to-intervention time for AMI.[35] On the other hand, other studies of community-based individuals and clinical trial participants have shown no temporal declines in prehospital delay during AMI.[36–38]

Case Fatality Rates

Our finding that AMI case fatality declined from 1960 to 1999 is consistent with studies conducted in the United

States[1,3,4,23] and Europe[22] that demonstrated declines in overall AMI case fatality over the past 20 to 40 years. Several studies have demonstrated that out-of-hospital sudden cardiac death has declined substantially over the past several decades.[2,12,32–34] We extend these findings by demonstrating that case fatality rates after AMI-ECG and after AMI-marker have declined to a similar degree.

Prior studies have suggested that improvements in primary prevention account for 40% to 50% of the reduction in CHD mortality in the Unites States from 1968 to 2000.[6,10] Our finding of a 50% decline in incidence of first AMI using an AMI definition for which bias is inherently low (ie, AMI-ECG) implies that primary prevention efforts also have influenced the incidence of AMI.

Strengths and Limitations

The availability of 4 decades of physician-validated AMI and case fatality data and the ability to separate AMI-ECG and AMI-marker are unique strengths of our investigation. Indeed, AMI diagnosis relying on ICD coding may have a sensitivity of only 60% compared with physician-validated AMI diagnosis.[39] Our adjudication committee had access to simultaneous ECG and biomarker information; therefore, the ECG adjudication could have been biased by knowledge of biomarker information. However, we believe that if such a bias were introduced, it would have biased results toward a greater proportion of AMI-ECG over time. We could not separate the contribution of specific biomarkers to AMI diagnosis among AMI-marker cases. Our study sample is largely white of European descent; therefore, our findings may not be applicable to other ethnic groups or other geographic regions. We had a relatively small number of events when sex, specific age groups, and 4 time periods are considered, possibly limiting our statistical power to detect differences. We had a limited number of subjects in each sex, age group, and time period. We did not provide confidence intervals for the trend analyses for the incidence rates of AMI-ECG and AMI-marker; in addition, because of limited statistical power, we did not test the interaction term of MI type with time period.

Implications of our Findings

The diagnosis of AMI is evolving; therefore, it is a challenge to accurately characterize the "true" epidemiology of AMI. However, our data demonstrate that although AMI-ECG rates have declined, this decline was offset by rising AMI-marker rates. Because the most sensitive biomarkers (ie, troponin) were not available in the earlier study decades (1960s to 1980s), AMI-marker earlier on may have been underdiagnosed. Regardless, the advent of increasingly sensitive biomarkers for AMI diagnosis has substantially influenced AMI detection rates in the United States over the past several decades.

Conclusions

Over the past 40 years, AMI-ECG rates have declined by 50% and AMI-marker rates have doubled, offering a possible explanation for apparently steady national rates of overall AMI in the face of improvements in primary prevention.

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Disclosures

None.

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CLINICAL PERSPECTIVE

Whereas the prevalence of coronary heart disease risk factors has declined over past decades in the United States, acute myocardial infarction (AMI) rates have been steady. Because the diagnosis of AMI is evolving, it is a challenge to characterize the "true" epidemiology of AMI accurately. Among Framingham Heart Study participants, we found that over the past 40 years, rates of AMI diagnosed by ECG have declined by 50%, whereas rates of AMI diagnosed by biomarkers have doubled. The advent of increasingly sensitive biomarkers for AMI diagnosis has substantially influenced AMI detection rates in the United States over the past several decades. Our findings offer an explanation for the apparently steady national AMI rates in the face of improvements in primary prevention.