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Prior Hospitalization and the Risk of Heart Attack in Older Adults: A 12-Year Prospective Study of Medicare Beneficiaries

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Background. We investigated whether prior hospitalization was a risk factor for heart attacks among older adults in the survey on Assets and Health Dynamics among the Oldest Old.

Methods. Baseline (1993–1994) interview data were linked to 1993–2005 Medicare claims for 5,511 self-respondents aged 70 years and older and not enrolled in managed Medicare. Primary hospital *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*) 410.*xx* discharge codes identified postbaseline hospitalizations for acute myocardial infarctions (AMIs). Participants were censored at death or postbaseline managed Medicare enrollment. Traditional risk factors and other covariates were included. Recent postbaseline non-AMI hospitalizations (ie, prior hospitalizations) were indicated by a time-dependent marker, and sensitivity analyses identified their peak effect.

Results. The total number of person-years of surveillance was 44,740 with a mean of 8.1 (median = 9.1) per person. Overall, 483 participants (8.8%) suffered postbaseline heart attacks, with 423 participants (7.7%) having their first-ever AMI. As expected, significant traditional risk factors were sex (men); race (whites); marital status (never being married); education (noncollege); geography (living in the South); and reporting a baseline history of angina, arthritis, diabetes, and heart disease. Risk factors were similar for both any postbaseline and first-ever postbaseline AMI analyses. The time-dependent recent non-AMI hospitalization marker did not alter the effects of the traditional risk factors but increased AMI risk by 366% (adjusted hazards ratio = 4.66, $p < .0001$).

Discussion. Our results suggest that some small percentage (<3%) of heart attacks among older adults might be prevented if effective short-term postdischarge planning and monitoring interventions were developed and implemented.

Key Words: Heart attack—Recent hospitalization—Epidemiology—Prospective cohort study.

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EIGHTY million (M) Americans have cardiovascular disease (CVD), including high blood pressure (74M), coronary heart disease (17M), heart failure (6M), and stroke (7M) (1). Half of these individuals are aged 60 years and older. Acute myocardial infarctions (AMIs) are a major CVD component, with nearly 8M Americans having a history of heart attack. About 785 thousand (K) will have their first-ever diagnosed heart attack this year, 470K will have a diagnosed recurrent AMI, and 195K will have their first-ever silent (undiagnosed) heart attack (1). Thus, 87% of the 1.5M heart attacks occurring annually are diagnosed, and most of these result in a hospitalization episode.

AMI risk factors are well established and classified by the American Heart Association (AHA) as modifiable or controllable factors, risks that cannot be changed, and other contributing factors (2). Modifiable or controllable risks include diabetes, being overweight or obese, physical inactivity, hyperlipidemia, high blood pressure, and smoking. Unalterable risk factors include increasing age, being a man, and heredity and race. Contributing factors include stress, poor dietary patterns, and alcohol consumption. Despite efforts to raise public awareness of the modifiable or controllable AMI risk factors, their prevalence remains high (3), with 15% of older adults diagnosed with diabetes and another 7% undiagnosed diabetics, more than 25% being obese, 24% being physically inactive, 16% having hyperlipidemia, half having hypertension, and 28% of older women and 49% of older men having a history of smoking (1).

Given the prevalence of AMI risk factors among older adults and the challenge in modifying or controlling them, it is prudent to identify other potentially modifiable risk factors. Emerging literature suggests prior hospitalizations (which are referred to as the index hospitalizations in rehospitalization studies and discussions) (4–9). The suspected mechanism is that inefficient and ineffective postdischarge planning and monitoring increases short-term risks by failing to appropriately manage the underlying conditions that led to the prior hospitalizations, as well as by not integrating the treatments for that episode into ongoing therapeutic regimens (10–12).

We recently explored this possibility by adding time-dependent risk factors for prior hospitalization into standard epidemiological analyses of hip fracture (13) and stroke (14). Using the survey of Assets and Health Dynamics among the Oldest Old (AHEAD) (15,16), those results revealed substantially increased independent risks for hip fracture (adjusted hazards ratio [AHR] = 2.51, $p < .0001$) and stroke (AHR = 2.90, $p < .0001$). We extend that work here to the risk for AMI. Specifically, after adjusting for traditional AMI risk factors and other available covariates, we introduce a time-dependent covariate reflecting prior hospitalization, calibrate its peak effect period, and determine the extent to which prior hospitalization mediates the effects of other risk factors.

Methods

Data

Complete documentation for the design, procedures, and protocols for the nationally representative AHEAD study (15,16) can be found online at http://hrsonline.isr.umich.edu. A multistage cluster sampling design was used that over sampled African Americans, Hispanics, and Floridians. All analyses were weighted to adjust for the unequal probabilities of selection. An 80.4% response rate was obtained at baseline (1993–1994), yielding 7,447 participants, all of whom were aged 70 years and older.

Our analyses were limited to 5,511 (74%) of the AHEAD participants. We excluded 530 participants (7.1%) because their baseline interview data were provided by a proxy, 802 participants (10.8%) because their baseline interview data could not be linked to Medicare claims, and 604 participants (8.1%) in managed Medicare at baseline (17). Participants were subsequently censored at the time of two competing risks—death (2,637) or postbaseline enrollment into managed Medicare (837)—whichever came first.

Selection Bias

To adjust for the potential selection bias resulting from our three exclusion criteria, we used propensity score methods (18–21). This involved estimating a multivariable logit model of whether or not each of the 7,447 original AHEAD participants was included in the analytic sample. After finding that the fit of the model to the data was good, we determined the average participation rate within each predicted probability decile and used the inverse of these average rates to reweight the data. The propensity score weights were rescaled to equal the number of participants in the analytic sample (ie, 5,511).

Case Identification and Analysis

Surveillance began on the day after each individual's baseline interview, with postbaseline heart attacks (AMIs) identified using primary hospital *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*) 410.*xx* discharge codes (22). Analyses were conducted two ways: (a) time to the first postbaseline heart attack among all 5,511 AHEAD participants in the analytic sample, including a marker for self-reported prebaseline AMI histories and (b) excluding the 374 participants who self-reported prebaseline heart attack histories to focus solely on first-ever postbaseline heart attacks.

Heart attacks had to occur at least 1 day after the participant's baseline interview. Censoring occurred at the time of death (n = 2,637) or postbaseline enrollment into managed Medicare (n = 837), whichever came first, using vital and insurance plan status data from the Medicare claims denominator file. Multivariable proportional hazards regression with competing risks (23) was used to model time to heart attack, assuming that these competing risks were independent and censored. Model development and evaluation followed standard procedures (24,25), with all measures of the traditional risk factors forced into the proportional hazards regressions and the covariates (see subsequently) entering the regressions if they had statistically significant effects ($p < .05$) in predicting either the first postbaseline heart attack or the first-ever postbaseline AMI. All analyses were performed using the propensity score reweighting to adjust for potential selection bias, although analyses performed without the propensity score reweighting were equivalent.

Traditional Risk Factors

Risk factors for AMI are classified by the AHA as modifiable or controllable factors, risks that cannot be changed, and other contributing factors (2). Diabetes, being overweight or obese, physical inactivity, hyperlipidemia, high blood pressure, and smoking are the modifiable risk factors. Increasing age, being a man, and heredity and race are the unalterable risk factors. Stress, poor dietary patterns, and alcohol consumption are the principal contributing factors.

Of these traditional risks, data are available for all but hyperlipidemia, family history, and dietary patterns in the baseline AHEAD interviews. Although their absence is unfortunate, the limiting effect is modest for two reasons. First, we are unaware of any evidence to suggest that their absence appreciably alters the risk estimates obtained for other factors in the models. Second, our principal focus is on the effects of the dynamic prior hospitalization marker. Nonetheless, the absence of these risk factors and other associated key comorbidites (like renal insufficiency and peripheral artery disease) could potentially confound our findings.

Covariates

To obtain the independent effect of the dynamic prior hospitalization marker, we considered a number of covariates. In addition to the traditional risk factors of age, sex, and race, sociodemographic factors included living alone, marital status, and importance of religion. Socioeconomic factors included education, income, total wealth, number of health insurance policies, and perceived neighborhood safety (an indirect measure of stress). Place of residence was categorized by population density (another indirect measure of stress), geographic region, and dwelling type (another indirect measure of stress). Health behaviors included the traditional risk factors of being overweight or obese, smoking history, and alcohol consumption. Disease history included the traditional risk of factors of whether the participant had ever been told by a physician that she or he had diabetes, high blood pressure, or a prior heart attack, as well as angina, arthritis, cancer, other forms of heart disease (including coronary heart disease and congestive heart failure), a previous hip fracture, lung disease, psychological problems, or a stroke or transient ischemic attacks (TIA), and a binary comorbidity marker reflecting having four or more of these conditions. Functional status was measured by self-rated health; counts of the number of difficulties with activities of daily living (ADLs) and instrumental ADLs; number of reported depressive symptoms (another indirect measure of stress); falling; bothersome pain; ability to pick up a dime; and self-rated vision, memory, and urinary incontinence. Cognitive status included immediate and delayed word recalls and the Telephone Interview to Assess Cognitive Status (TICS) (26).

Prior Hospitalization

The dynamic prior hospitalization measure (13,14) was constructed using the postbaseline Medicare claims. This time-dependent covariate was switched “on” the day after the participant was discharged from a hospital for any primary *ICD-9-CM* diagnosis other than a heart attack at any time prior to study censoring. This prevented the marker from reflecting rehospitalization for an AMI, as well as from reflecting same day transfers from one hospital to another. Prior hospitalizations counted even if they were for non-AMI CVD because the baseline covariates should adjust for the majority of chronic CVD conditions. The prior hospitalization marker stayed “on” for *n* days after discharge and was then switched “off.” It could subsequently be switched back “on” at the onset of another precensoring non-AMI hospital admission. Sensitivity analyses were conducted to determine which of several values of *n* was most predictive of heart attack—1 and 2 weeks; 1, 2, 3, and 6 months; and 1, 2, and 3 years.

Results

Descriptive

The mean age in the analytic sample was 77 years; 38% were men, 10% were African American, 4% were Hispanic, and 41% were widowed. One fourth had only been to grade school, and mean income was $25K. Nearly half (47%) lived in cities of a million or more, 39% resided in multistory dwellings, and 12% rated the safety of their neighborhood as only fair or poor. Prebaseline heart attack histories were reported by 374 participants (7%). Diabetes was reported by 12%, high blood pressure by 46%, angina by 9%, other forms of heart disease by 28%, arthritis by 25%, cancer by 13%, a previous hip fracture by 4%, lung disease by 9%, psychological problems by 7%, a stroke or TIA by 9%, with 26% having endorsed at least three of the eight depressive symptoms, and 12% having at least four chronic conditions. One in seven (14%) was obese, and 36% were overweight. Smoking history was common, with 10% being current and 42% being former smokers. Daily consumption of one or more alcoholic drinks was reported by 11%. One fifth had less than good cognitive status on the TICS, and 23% were hospitalized in the year before their baseline interviews. Seventy-nine percent experienced postbaseline non-AMI hospitalizations. Postbaseline heart attacks were experienced by 483 participants (9%) and were rather uniformly distributed over the observation period, with 60% in 1993–1999 and 40% in 2000–2005, reflecting an increased incidence rate among survivors. Of the postbaseline AMIs, 423 were first-ever heart attacks. The total number of person-years of surveillance was 44,740, with a mean of 8.1 (median = 9.1) per person.

Sensitivity Analyses

Sensitivity analyses identified the peak effect of the prior hospitalization marker, adjusting for all of the traditional risk factors and covariates described earlier. As shown in Table 1, AHRs for the prior hospitalization indicator were large. When the dynamic prior hospitalization marker was left “on” following the non-AMI hospitalization for 1 week, the effect was at its peak for both the first postbaseline heart attack (AHR = 4.66, $p < .0001$) and the first-ever postbaseline heart

Table 1. Adjusted Hazards Ratios (AHRs) for the Dynamic Prior Hospitalization Indicator Predicting Any and First-Ever Postbaseline AMIs by Selected Times After Discharge*

Selected Times After Discharge	Any Postbaseline AMI (n = 5,511)	First-Ever Postbaseline AMI (n = 5,137)
1 Week	4.66	5.38
2 Weeks	3.90	4.20
1 Month	2.86	2.91
2 Months	2.81	2.76
3 Months	2.58	2.39
6 Months	1.99	1.93
1 Year	1.82	1.74
2 Years	1.79	1.74
3 Years	1.65	1.60

Note: *AHRs adjusted for all of the traditional risk factors and covariates shown in Table 2, and all AHRs are statistically significant at the $p < .0001$ level. AMI = acute myocardial infarction.

attack (AHR = 5.38, $p < .0001$). The magnitude of the prior hospitalization effect dropped substantially as n (ie, left "on") increased, achieving relative stability from about 1–3 months, followed by a further drop at 6 months and then relative stability out to 3 years. Based on these results, n was set at 1 week.

Multivariable Hazards Models

Table 2 contains the results from the final multivariable hazards models with competing risks. Column 1 contains the AHRs for the static model, and column 2 contains the AHRs after adding the dynamic prior hospitalization marker for the first postbaseline heart attack among all 5,511 AHEAD participants in the analytic sample. Columns 3 and 4 contain comparable results for the first-ever heart attack after excluding the 374 participants who had self-reported prebaseline heart attack histories.

The results for the first postbaseline (ie, either recurrent or first-ever) heart attack static model (column 1) indicated statistically significant risks for men (AHR = 1.38); whites (AHR = 0.59 for African Americans); never married participants (AHR = 1.73); those with additional insurance policies (AHR = 1.20); participants who had not attended college (college AHR = 0.72); those who lived in the South (vs elsewhere); those who were overweight (AHR = 1.29); participants reporting baseline histories of diabetes (AHR = 1.46), heart disease (AHR = 1.45), heart attacks (AHR = 1.59), angina (1.38), or arthritis (AHR = 1.30); and scoring in the bottom of the delayed word recall test (AHR = 1.24). Introduction of the 1-week calibration of the dynamic prior hospitalization marker (AHR = 4.66, $p < .0001$) did not alter these risk estimates (column 2), although it significantly improved the fit of the model (ie, change in log likelihood ratio = 29.2 at 1 *df*; $p < .0001$). When these analyses were repeated (columns 3 and 4) after excluding those reporting prebaseline AMI histories (ie, focusing only on first-ever heart attacks), the results were essentially the same.

Ad Hoc Analyses

We conducted ad hoc analyses to separate the effects of non-AMI coronary heart disease from those of other prior hospitalizations. This involved breaking down the prior hospitalization marker into prior hospitalizations due to (a) non-AMI acute coronary syndrome (ACS; *ICD-9-CM* codes of 411.*xx* to 414.*xx*) versus (b) non-AMI non-ACS (*ICD-9-CM* codes other than 410.*xx* to 414.*xx*). We then reestimated the models shown in Table 2 substituting the non-AMI ACS and non-AMI non-ACS markers for the original measure. As expected, prior hospitalizations for ACS were even more likely to result in heart attacks, with AHRs (both $p < .0001$) for non-AMI ACS being 15.05 on first postbaseline AMI and 16.30 on first-ever postbaseline AMI. At the same time, the effects of prior hospitalizations for non-ACS remained quite large with AHRs (both $p < .0001$) for non-AMI non-ACS being 3.83 on first postbaseline AMI and 4.37 on first-ever postbaseline AMI.

We conducted further ad hoc analyses to characterize the prior hospitalizations that were directly linked to subsequent AMIs. Twenty (4.1%) of the 483 postbaseline heart attacks occurred within 1 week of a prior hospitalization, and Table 3 shows their primary *ICD-9-CM* discharge diagnosis code and description, whether it was a medical versus surgical admission, whether any time was spent in an intensive care unit (ICU), and length of stay (LOS). Medical versus surgical admissions and intensive ICU use were determined using standard algorithms (27,28). These data are mixed. On the one hand, there were seven prior hospitalizations (cases 4, 5, 12–14, 18, and 19) that involved ICU stays, four that involved surgical admissions (cases 7, 8, 12, and 13), four that involved ACS (cases 6, 10, 12, and 16), and three for chronic bronchitis (cases 5, 11, and 17). On the other hand, there were single cases of congestive heart failure (CHF), pneumonia, rectal cancer, lung cancer, epistaxis, and malnutrition. Furthermore, mean LOS was a robust 7 (median = 6) days.

Discussion

Our principal finding involves the dynamic prior hospitalization marker (13,14). When calibrated (ie, left "on") for 1 week after discharge from the prior non-AMI hospitalization, its effect was substantial (AHR = 4.66, $p < .0001$). Moreover, its introduction did not mediate the effects associated with traditional heart attack risk factors or other covariates. Rather, the prior hospitalization marker clearly represents a previously untapped independent risk for heart attack. And even when non-AMI ACS prior hospitalizations are removed from the dynamic marker, its effect remained large (AHR = 3.83, $p < .0001$).

These results have potentially important implications for health care policy. Of the 483 postbaseline heart attacks experienced by the 5,511 participants in the AHEAD cohort, 20 (4.1%) were directly linked to hospitalizations that had occurred in the past week for something other

Table 2. Adjusted Hazards Ratios (AHRs) From Final Models of Time to the First Postbaseline AMI (n = 5,511) and Time to the First-Ever Postbaseline AMI (n = 5,137)

	Static Model AHRs First Postbaseline AMI	Dynamic Model AHRs First Postbaseline AMI	Static Model AHRs First-Ever Postbaseline AMI	Dynamic Model AHRs First-Ever Postbaseline AMI
Variable				
Prior hospitalization (calibrated at ≤7 d)		4.66***		5.38***
Sociodemographics				
Age, y				
69–74 (RG)	1.00	1.00	1.00	1.00
75–79	1.06	1.06	1.20	1.20
80–84	1.28	1.27	1.45*	1.44*
85+	1.39	1.38	1.43	1.42
Sex (men)	1.38**	1.38**	1.40**	1.40**
Race				
African American	0.59**	0.59**	0.58**	0.58**
Hispanic	0.84	0.85	0.86	0.87
White (RG)	1.00	1.00	1.00	1.00
Marital status				
Widowed	1.14	1.13	1.13	1.12
Divorced/separated	1.11	1.11	1.26	1.26
Never married	1.73*	1.72**	1.50	1.49
Married (RG)	1.00	1.00	1.00	1.00
Socioeconomics				
Education				
Grade school	0.97	0.97	0.90	0.90
High school (RG)	1.00	1.00	1.00	1.00
College	0.72**	0.72**	0.70**	0.70**
No. of Health insurance policies	1.20*	1.20*	1.20*	1.20*
Neighborhood safety				
Poor	0.85	0.84	0.96	0.96
Fair	1.07	1.07	1.02	1.02
Good to excellent (RG)	1.00	1.00	1.00	1.00
Residence characteristics				
Population more than 1,000,000	0.98	0.98	0.99	0.99
Region of the United States				
Northeast	0.47***	0.48**	0.46**	0.47**
North central	0.45***	0.46***	0.44***	0.45***
West	0.65*	0.65*	0.59*	0.60*
South (RG)	1.00	1.00	1.00	1.00
Risk factors				
Health behaviors				
Body mass				
Obese	1.07	1.07	1.13	1.12
Overweight	1.29*	1.30*	1.27*	1.28*
Normal (RG)	1.00	1.00	1.00	1.00
Underweight	1.49	1.47	1.39	1.38
Smoking history				
Former smoker	1.03	1.02	1.02	1.01
Current smoker	1.57**	1.55**	1.51*	1.50*
Never smoked (RG)	1.00	1.00	1.00	1.00
Drinking history				
3 or more drinks per day	0.48	0.49	0.38	0.38
1–2 drinks per day	1.25	1.25	1.27	1.28
Less than 1 drink per day (RG)	1.00	1.00	1.00	1.00
Disease history				
Angina	1.38*	1.36*	1.38	1.36
Arthritis	1.30*	1.30*	1.38**	1.37**
Diabetes	1.46**	1.46**	1.52**	1.51**
Hypertension	1.24*	1.24*	1.18	1.18
Heart attack	1.59**	1.58**		
Heart disease	1.45**	1.45**	1.41**	1.40**
Psychological problems	1.21	1.21	1.34	1.33
Stroke	1.30	1.29	1.48*	1.47*
Functional status				
CESD-8 count				
0 (RG)	1.00	1.00	1.00	1.00
1	0.91	0.90	0.84	0.84

Table 2. (*Continued*)

	Static Model AHRs First Postbaseline AMI	Dynamic Model AHRs First Postbaseline AMI	Static Model AHRs First-Ever Postbaseline AMI	Dynamic Model AHRs First-Ever Postbaseline AMI
2	1.03	1.03	1.04	1.04
3 or more	0.97	0.97	0.97	0.96
Cognitive status				
Low ½ delayed word recall	1.24*	1.24*	1.29*	1.29*
High ½ delayed word recall	1.00	1.00	1.00	1.00
Refused to answer delayed recall	0.60	0.60	0.65	0.65

Notes: Among 5,511 Assets and Health Dynamics among the Oldest Old self-respondents (at baseline) with linked Medicare claims who were not in managed care at their baseline interviews for the first postbaseline AMI analysis, there were 483 AMI cases. Among 5,137 self-respondents (at baseline) with linked Medicare claims who were not in managed care at their baseline interviews for the first-ever postbaseline AMI analysis, there were 423 AMI cases. All variables were binary coded (1 = yes, 0 = no) except the number of health insurance policies (actual number). AMI = acute myocardial infarction; CESD = Center for Epidemiologic Studies Depression scale; RG = reference group.

$^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$.

than an AMI. Because 75% of these directly linked prior hospitalizations were not for ACS, it is possible that some small percentage (<3%) of heart attacks among older adults might be prevented if effective short-term postdischarge planning and monitoring interventions consistent with the suggestions of Coleman and colleagues (10–12) were developed and implemented. But certainly, not all of the prior hospitalizations linked to subsequent AMIs are preventable. Some likely result from the stress of the prior hospital episode's underlying cause. For example, the initial hospital episode could trigger ischemia in susceptible participants much like vigorous exercise increases AMI risk. It is unlikely that postdischarge planning and monitoring would prevent these, although some might be detected and treated earlier.

Jencks and colleagues (29) have recently provided substantial evidence consistent with our and Coleman and colleagues' interpretation (10–12) that the primary mechanism through which the effect of prior hospitalization is manifested involves inefficient and ineffective postdischarge planning and monitoring. Although focused on rehospitalization in general, they have shown that fully half of all Medicare patients who were rehospitalized within 30 days of their index hospitalization had no evidence (ie, Medicare claims) reflecting their having been seen in a physician's office during the period between their index and rehospitalizations. In the AHEAD cohort, we found that 1,724 (31%) of the 5,511 participants in the analytic sample had at least one postbaseline 30-day rehospitalization. Of these 1,724 AHEAD participants, 575 (33%) had no evidence of ambulatory care visits (in either their Medicare Outpatient or Carrier Statistical Analytic Files) during the 30 days between their first prior and any (ie, all cause, rather just an AMI) rehospitalization pairings. Moreover, of the 1,724 participants with one or more 30-day rehospitalizations, 80 involved subsequent AMIs, and 35 (44%) of those had no evidence of outpatient visits in the interim. Therefore, we agree with Jencks and colleagues (29) that (a) the troubling absence of evidence reflecting no physician visits during the immediate posthospitalization period for one third (our finding) to one half (their finding) of rehospitalized Medicare patients, combined with (b) extant evidence from controlled trials (30–32) that rehospitalization can be reduced by appropriate discharge planning interventions focusing on transitional care, then (c) a sizable portion of the excess independent risk captured by our prior hospitalization marker is likely preventable.

This leads to two questions from a policy perspective: What should an intervention target, and how might it be supported in the long run? To answer the former question, we note that it is well known that the transition from the hospital to the home is often difficult for vulnerable elders, especially in an era of shortened hospital stays when they are discharged at greater risk (33). Problems that have previously been identified in the literature include inadequate communication between hospital physicians and primary care professionals, failure to maintain appropriate medication regimens postdischarge, cognitive impairment leading to failure to understand discharge instructions as well as poor health literacy among patients and their families, acute functional declines that lead to functional vulnerability, and reemergence of partly controlled conditions in the home setting, like congestive heart failure (34–36). These problems suggest the need for an effective institutionalized program for physician communication and monitoring during the postdischarge period for older adults.

For the answer to the latter question, our examinations of the risk of rehospitalization for AMI, hip fracture (13), and stroke (14) have identified that the peak risk occurred during the first week or the second after the prior hospitalization. Accordingly, we recommend consideration of a Medicare demonstration project that would provide additional reimbursement incentives to both the hospital and primary care physicians of record. To receive reimbursement, the hospital physician would have to provide, both verbally and in writing (manual or electronic), an appropriately detailed accounting of the hospital episode and subsequent treatment recommendations directly to the primary care physician. And the primary care physician would have to actually see the patient in the office and generate appropriate evaluation and management codes at least once during the first postdischarge week.

Table 3. Characteristics of the 20 Prior Hospitalizations Linked to Subsequent Acute Myocardial Infarctions

Case	Primary Diagnosis—*ICD-9* Code	Primary Diagnosis—Description	Medical or Surgical Admission	Intensive Care Unit	Length of Stay (d)
1	486	Pneumonia	Medical	No	6
2	154.1	Malignant neoplasm of rectum	Medical	No	9
3	295.34	Schizophrenic disorder—paranoid type	Medical	No	3
4	428.0	Congestive heart failure	Medical	Yes	6
5	491.21	Chronic bronchitis with acute exacerbation	Medical	Yes	6
6	414.00	Other forms of chronic ischemic heart disease (of unspecified type of vessel, native or graft)	Medical	No	3
7	162.3	Malignant neoplasm of upper lobe, bronchus, or lung	Surgical	No	7
8	784.7	Epistaxis	Surgical	No	2
9	V57.89	Other care involving use of rehabilitation procedures	Medical	No	19
10	414.00	Other forms of chronic ischemic heart disease (of unspecified type of vessel, native or graft)	Medical	No	2
11	491.21	Chronic bronchitis with acute exacerbation	Medical	No	4
12	414.01	Other forms of chronic ischemic heart disease (of native coronary artery)	Surgical	Yes	10
13	550.10	Inguinal hernia, with obstruction	Surgical	Yes	15
14	812.00	Fracture of upper end of humerus	Medical	Yes	3
15	972.1	Poisoning by agents primarily affecting the cardiovascular system (cardiotonic glycosides and drugs of similar action)	Medical	No	5
16	411.1	Intermediate coronary syndrome	Medical	No	1
17	491.21	Chronic bronchitis with acute exacerbation	Medical	No	15
18	786.50	Chest pain, unspecified	Medical	Yes	3
19	531.40	Chronic or unspecified gastric ulcer with hemorrhage	Medical	Yes	9
20	263.9	Unspecified protein–calorie malnutrition	Medical	No	4

Note: *ICD-9 = International Classification of Diseases, Ninth Revision.*

Although the introduction of our recent non-AMI hospitalization measure is a promising development that underscores the need to shift from static to dynamic risk modeling approaches (13,14), further research is needed. That research should proceed along two fronts. First, the underlying etiologic mechanisms through which the prior hospitalization marker operates need to be explored and clarified. In this regard, we suggest studies in which the risk mechanisms for different subsequent health events (ie, rehospitalizations) are compared and contrasted. For example, we have found similar effects of prior hospitalization on subsequent stroke and hip fracture using the AHEAD cohort, although those effects were notably smaller (ie, AHRs = 2.91 and 2.51, respectively, both $p < .0001$) than those observed here for subsequent AMIs (AHR = 4.66, $p < .0001$). The only common covariates with significant independent effects in these models, however, were older age (increased risk) and better cognitive function [lowered risk; as measured by the TICS (26)], both of which are traditional risk factors for nearly all hospitalizations among older adults. Thus, at least in these data, our suggested approach appears not to be fruitful. Second, research is needed to identify whether all non-AMI hospitalizations are equally risky for subsequent heart attacks. That research should clarify whether restrictions to surgical versus medical admissions, shorter versus longer stays, or other dimensions of the prior hospitalization events are associated with differential risks for subsequent heart attacks. As shown in Table 3, ICU use, surgical admissions, ACS, and chronic bronchitis may be promising targets.

It is also worth noting what we did and did not find among the traditional risk factors (and covariates). We found that the greatest risks were for men; whites; the never married; those living in the South, and those with prebaseline histories of diabetes, angina, and heart disease. Of these, only the elevated risk for whites is surprising. It may be that in such a cohort of older adults, this reflects a survivor effect among African Americans. African Americans enrolled in the AHEAD were survivors relative to their racial peers and may have advantaged health stocks. What we did not find were significant risks associated with increased age, smoking, and obesity. The failure to find age and smoking risks appears to be due to statistical power. That is, all three of the age contrasts reflected increasing risk, but all were marginally insignificant (ps = .06). The same was true for the risk associated with smoking ($p = .06$). In contrast, the failure to find increased risk associated with obesity is not likely an artifact of limited statistical power because 14% of the analytic sample was obese at baseline. Rather, this may reflect a beneficial robustness among obese individuals who reach very old age.

In conclusion, we have shown that when calibrated (ie, left "on") for 1 week after discharge from the prior non-AMI hospitalization, the dynamic prior hospitalization marker had a substantial independent effect on the risk for subsequent AMIs (AHR = 4.61, $p < .0001$). Moreover, this risk did not result from mediating the effects of well-established risk factors for heart attack, but from bringing new information to the table. Although a case-by-case examination of the offending prior hospitalizations did identify some commonalities, including ICU use (35%), surgical admissions (20%), ACS (20%), and chronic bronchitis (15%), there was also considerable uniqueness among these cases (ie, single cases of CHF, pneumonia, rectal cancer, lung cancer, epistaxis, and malnutrition). Based on these results, we recommend consideration of a Medicare demonstration project that would provide additional reimbursement incentives to both the hospital and primary care physicians of record to minimize the rehospitalization risk that we have identified here which results in subsequent AMIs, and elsewhere in strokes (14), and hip fractures (13).

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