Annals of Internal Medicine | ORIGINAL RESEARCH

Prediction of Heart Failure Mortality in Emergent Care

A Cohort Study

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Background: Heart failure contributes to millions of emergency department (ED) visits, but hospitalization-versus-discharge decisions are often not accompanied by prognostic risk quantification.

Objective: To derive and validate a model for acute heart failure mortality applicable in the ED.

Design: Clinical data abstraction with development of a broadly applicable multivariate risk index for 7-day death using initial vital signs, clinical and presentation features, and readily available laboratory tests.

Setting: Multicenter study of 86 hospitals in Ontario, Canada.

Patients: Population-based random sample of 12 591 patients presenting to the ED from 2004 to 2007.

Measurements: Death within 7 days of presentation.

Results: In the derivation cohort (n = 7433; mean age, 75.4 years [SD, 11.4]; 51.5% men), mortality risk increased with higher triage heart rate (adjusted odds ratio [OR], 1.15 [95% CI, 1.03 to 1.30] per 10 beats/min) and creatinine concentration (OR, 1.35 [CI, 1.14 to 1.60] per 1 mg/dL [88.4 μmol/L]), and lower triage systolic blood pressure (OR, 1.52 [CI, 1.31 to 1.77] per 20 mm Hg) and initial oxygen saturation (OR, 1.16 [CI, 1.01 to 1.33] per 5%). Nonnormal serum troponin levels (OR, 2.75 [CI, 1.86 to 4.07]) were associated with increased mortality risk. Areas under the receiver-operating characteristic curves of the multivariate model were 0.805 for the derivation data set (bootstrap-corrected, 0.811) and 0.826 for validation data set (n = 5158; mean age, 75.7 years [SD, 11.4]; 51.6% men). In the derivation cohort, a multivariate index score stratified 7-day mortality with rates of 0.3%, 0.3%, 0.7%, and 1.9% in quintiles 1 to 4, respectively. Mortality rates in the 2 highest risk groups were 3.5% and 8.2% in deciles 9 and 10, respectively.

Limitation: Left ventricular ejection fraction was not included in the model.

Conclusion: A multivariate index comprising routinely collected variables stratified mortality risk with high discrimination in a broad group of patients with acute heart failure presenting to the ED.

Primary Funding Source: Canadian Institutes of Health Research.

Ann Intern Med. 2012;156:767-775. www.annals.org

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Heart failure (HF) is a global public health issue characterized by high mortality and increased rates of hospitalizations and rehospitalizations (1). Patients with decompensated HF frequently visit emergency departments (EDs) and have high rates of rehospitalization, which result in increased health care costs (2). Despite the substantial resource and economic implications of hospitalization of the patient with HF, acute care decisions may not be guided by evidence (3, 4). As a consequence, clinicians may hospitalize some low-risk patients who have HF and may discharge some high-risk patients without being able to accurately assess prognosis, largely because prognostic algorithms that can be applied before decisions are made about patient disposition are not available (5).

Although sole reliance on clinical judgment for the patient with acute HF, rather than an assessment also informed by prognostic quantification, is the current standard of care, it may result in excess hospitalization of low-risk patients (6). Conversely, some patients with acute HF who are discharged after an apparent initial response to treatment may die after leaving the ED (7). Indeed, there is substantial overlap in the prognostic profiles of patients with HF presenting to the ED who are subsequently discharged or hospitalized (7). Blood pressure, heart rate and rhythm, precipitants (such as myocardial ischemia), comorbid conditions, and clinical severity have been proposed as important considerations in the 6-axis model for initial assessment of acute HF syndromes (8). An evidence-based clinical risk score that incorporates facets of the initial assessment rubric and facilitates prognostication would benefit clinicians and patients.

Previous prognostic studies have focused on patients with HF who were hospitalized but have excluded those who were discharged from the ED (9–12). However, those who are discharged from the ED may be a substantial proportion of all patients with HF, who may also be at significant risk for acute mortality (7). This limits the use of previous hospitalization-based risk algorithms in the broader ED setting. There is no assurance that HF risk algorithms that were developed in patients who had already been hospitalized will identify those who can be safely discharged (13). The objective of this study was to derive and validate a risk model for predicting acute mortality in pa-

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Context

Although models to predict the risk for short-term mortality among patients presenting with acute heart failure have been developed, the factors included have been derived from patients who were already hospitalized. A model applicable to patients before the decision to hospitalize or discharge from the emergency department may help improve clinical decisions.

Contribution

A model derived from several emergency departments accurately predicted the 7-day mortality of both patients who were discharged and those who were hospitalized.

Implication

This model may be useful in assessing the appropriate setting for the care of patients presenting with an acute episode of heart failure.

—The Editors

tients with HF who present to the ED to guide acute clinical decision making.

METHODS

Study Cohort

We studied the data of approximately 12 500 patients who visited an ED for HF and were either discharged or hospitalized from 1 April 2004 to 31 March 2007, in Ontario, Canada, which has a provincial population of more than 13 million. We randomly selected patients, who were either discharged from the ED or hospitalized, for detailed chart abstraction by using stratified cluster sampling. The patients were from hospitals randomly selected from strata defined by hospital type (14). Approximately 125 hospitalized patients were selected for chart abstraction from each of the 86 hospitals. Patients who were not hospitalized were sampled from a random subsample of 52 hospitals with 50 or more HF visits per year, maintaining a representative proportion of hospital type in the province (that is, teaching, large, or small). Approximately 95 patients were randomly selected from each teaching hospital and large community hospital; similarly, 60 patients were sampled from each small hospital. Heart failure cases were initially identified using the International Classification of Diseases, Tenth Revision (code I50), from the National Ambulatory Care Reporting System for ED visits and the Canadian Institute for Health Information database for patients who were hospitalized. To be included in the study and to emulate ED-based HF diagnosis based on clinical presentation, patients were also required to fulfill the Framingham criteria for HF after examination of their clinical chart records (15). Patients who were palliative or had do-not-resuscitate orders before ED arrival and transfers from another acute care hospital were excluded. Those who were dialysis-dependent were also excluded because of their differences in HF pathophysiology and treatment.

To assess the performance of the prediction model in a separate cohort, we randomly split the overall sample into derivation and validation data sets. We derived the model on a random sample of approximately 7500 patients with HF, two thirds of whom were hospitalized and one third of whom were discharged from the ED, reflecting the hospitalization-to-discharge ratio from which the study population was sampled (7). The validation cohort comprised approximately 5000 patients with a similar distribution of hospitalized and discharged patients. Before study initiation, research ethics approval was obtained from Sunnybrook Health Sciences Centre and from hospitals where chart abstraction was done.

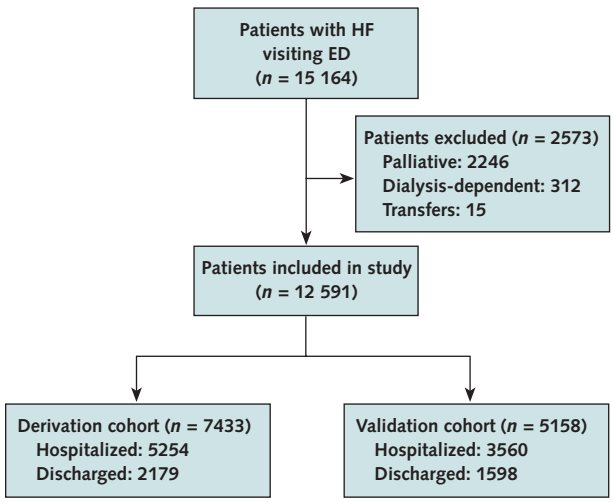
Outcome

The primary outcome was 7-day mortality after initial presentation, which is an end point that is important from the perspective of ED decision making and temporally close to the acute care episode (16).

Data Sources

The charts of patients who presented to the ED were abstracted by highly trained nurses to obtain clinical information pertaining to the patient and presenting features by using methods as previously described (14). Nurse abstractors were required to demonstrate high reliability on standardized chart abstractions before field deployment. Potential covariates considered in the analysis included age, sex, transport to ED by emergency medical services, origin from nursing home or long-term care facility, initial vital signs (systolic blood pressure, heart rate, respiratory rate, and oxygen saturation), symptoms (orthopnea and parosyxmal nocturnal dyspnea), signs (jugular venous pressure elevation, S_3, rales, and bilateral ankle edema), comorbid conditions (previous coronary revascularization, coronary artery disease, diabetes, hypertension, peripheral vascular disease, cerebrovascular disease, dementia, active cancer, and chronic pulmonary disease), laboratory results (hemoglobin, leukocytes, sodium, potassium, troponin, and creatinine), atrial fibrillation, QRS duration on 12-lead electrocardiogram, and pre-ED medications (angiotensin–converting enzyme inhibitor or angiotensin II–receptor blockers, β-adrenoreceptor antagonist, digoxin, furosemide, or metolazone) obtained by medical staff and recorded in the patient's chart. We also considered the Canadian Triage and Acuity Scale score, a nurse-rated score that is assigned at the time of triage for all patients seeking care in the ED (1 = resuscitation or critical, 2 = emergent, 3 = urgent, 4 = semi-urgent, or 5 = nonurgent) (17). A simple, random sample of more than 5% of the patients' charts from the derivation cohort were reabstracted to determine reliability. Those variables with a κ-statistic greater than 0.7 and crude agreement greater than 85% were examined in univariate analysis, whereas other variables were excluded from further analysis if deemed to be of low prognostic

Figure 1. **Study flow diagram.**



ED = emergency department; HF = heart failure.

importance. Through use of the patients' unique, encrypted health card number, these data were linked to the Registered Persons Database of vital statistics for determination of mortality in all patients.

Statistical Analysis

Continuous variables were reported as means (SDs) and compared by using a *t* test or the Kruskal–Wallis test for nonparametric distributions, whereas categorical variables were compared by using the chi-square statistic. In the derivation set, we used univariate logistic regression to examine the association of potential predictors, which are routinely available in the community, with 7-day mortality. Applying methods described by Harrell (18), potential predictors of mortality with *P* values less than 0.25 were considered for entry into a multiple logistic regression model based on statistical and clinical significance. Covariates were retained in the final multivariate model if the *P* value was less than 0.05 and if deemed clinically important or previous studies supported their prognostic importance. For continuous variables, the strength and shape of the relationship of potential predictors of death were examined by using cubic spline analyses with 5 knots at percentiles 5, 27.5, 50, 72.5, and 95 of the continuous covariate. On the basis of these analyses, upper and lower bounds were determined for the purposes of identifying truncation values above or below which there was no further contribution to the score calculation. Unmodified β-coefficients were reported from the multiple regression model, and odds ratios (ORs) were reported with estimates greater than unity reflecting increasing mortality risk.

Using a method of age-standardized β-coefficient–based weights similar to that used for the Framingham risk score (19), we developed a scoring system calculated by summing integer scores for categorical variables and weights for the value of continuous variables (where the value of the continuous variable was multiplied by its weight), called the Emergency Heart Failure Mortality Risk Grade (EHMRG). To derive the weights, model β-coefficients were divided by the age coefficient. The resultant weight was then used to determine the contribution of each variable to the overall risk score. Because the

Table 1. **Baseline Characteristics of the Derivation and Validation Cohorts**

Characteristic	Derivation Cohort (*n* = 7433)	Validation Cohort (*n* = 5158)
Mean age (SD), *y*	75.4 (11.4)	75.7 (11.4)
Men, *n (%)*	3825 (51.5)	2661 (51.6)
Transported by EMS, *n (%)*	2859 (38.5)	2240 (43.4)
Nursing home or long-term care, *n (%)*	525 (7.1)	427 (8.3)
Presenting features		
Median CTAS score (25th, 75th percentiles)	3 (2, 3)	3 (2, 3)
Mean SBP (SD), *mm Hg*	146.8 (31.0)	146.5 (30.7)
Mean heart rate (SD), *beats/min*	89.7 (24.4)	89.9 (24.4)
Mean respiratory rate (SD), *breaths/min*	23.5 (6.4)	23.5 (6.8)
Mean oxygen saturation (SD), %	93.7 (6.0)	92.9 (7.1)
Comorbid conditions, *n (%)*		
Coronary artery disease	3936 (53.0)	2608 (50.6)
Previous coronary revascularization*	1586 (21.3)	1053 (20.4)
Diabetes	2798 (37.6)	2004 (38.9)
Hypertension	4713 (63.4)	3313 (64.2)
Cerebrovascular disease†	1218 (16.4)	816 (15.8)
Peripheral artery disease	843 (11.3)	584 (11.3)
Chronic pulmonary disease	1619 (21.8)	1166 (22.6)
Dementia	448 (6.0)	336 (6.5)
Active cancer	653 (8.8)	367 (7.1)
Laboratory features		
Mean hemoglobin concentration (SD), *g/L*	124 (20)	124 (21)
Mean leukocyte count (SD), $\times 10^9$ *cells/L*	9.3 (4.2)	9.5 (4.8)
Mean sodium concentration (SD), *mmol/L*	138.5 (5.2)	138.3 (5.2)
Mean potassium concentration (SD), *mmol/L*	4.2 (0.6)	4.2 (0.6)
Mean creatinine concentration (SD)		
mg/dL	1.36 (0.72)	1.40 (0.80)
μmol/L	120.0 (63.3)	123.5 (70.6)
Nonnormal troponin level, *n (%)*‡	779 (10.5)	751 (14.6)
Electrocardiographic features		
Atrial fibrillation or flutter, *n (%)*	2208 (29.7)	1605 (31.1)
Mean QRS duration (SD), *msec*	110 (33)	109 (32)
Medications at home, *n (%)*		
ACE inhibitor or ARB	4281 (57.6)	3031 (58.8)
β-Adrenoreceptor antagonist	3322 (44.7)	2479 (48.1)
Digoxin	1441 (19.4)	935 (18.1)
Furosemide	3765 (50.7)	2826 (54.8)
Metolazone	112 (1.5)	108 (2.1)

ACE = angiotensin-converting enzyme; ARB = angiotensin II–receptor blocker; CTAS = Canadian Triage and Acuity Scale; EMS = emergency medical services; SBP = systolic blood pressure.

* Previous percutaneous coronary intervention or coronary artery bypass graft surgery.

† Stroke or transient ischemic attack.

‡ Greater than the upper limit of normal.

Table 2. **Multivariate Analysis of 7-Day Mortality**

Variable	Model β-Coefficient	OR Unit	OR (95% CI)	Wald Chi-Square	*P* Value
Age	0.0335	10-y increase	1.40 (1.16 to 1.69)	12.36	<0.001
Transport by EMS	1.044		2.84 (1.92 to 4.21)	27.15	<0.001
SBP	−0.021	20–mm Hg decrease*	1.52 (1.31 to 1.77)	30.69	<0.001
Heart rate	0.0143	10-beats/min increase†	1.15 (1.03 to 1.30)	5.75	0.017
Oxygen saturation	−0.0300	5% decrease‡	1.16 (1.01 to 1.33)	4.56	0.033
Creatinine	0.300	1-mg/dL (88.4-μmol/L) increase	1.35 (1.14 to 1.60)	11.59	<0.001
Potassium	NA	4.0 to 4.5 mmol/L	Reference	Reference	NA
	0.535	≥4.6 mmol/L	1.71 (1.13 to 2.58)	6.37	0.012
	0.089	≤3.9 mmol/L	1.09 (0.69 to 1.73)	0.14	0.70
Elevated troponin level§	1.013		2.75 (1.86 to 4.07)	25.67	<0.001
Active cancer	0.744		2.11 (1.33 to 3.33)	10.12	0.002
Metolazone at home	0.976		2.65 (1.07 to 6.61)	4.40	0.036

EMS = emergency medical services; NA = not applicable; OR = odds ratio; SBP = systolic blood pressure.
* Initial/triage SBP, maximum 160 mm Hg; OR shown is for decrease in SBP and, thus, is inverted compared with β-coefficient.
† Initial/triage heart rate, minimum of 80 beats/min and maximum of 120 beats/min.
‡ Lowest initial/triage oxygen saturation, maximum of 92%; OR shown is for decrease in oxygen saturation and, thus, is inverted compared with β-coefficient.
§ Greater than the upper limit of normal.

weights associated with systolic blood pressure and heart rate were approximately −0.5 and 0.5, respectively, we multiplied all weights by a factor of 2, so that the weight associated with each variable would be an integer. The weights for categorical variables were similarly multiplied by a factor of 2 and rounded to the nearest 5 points.

Model Evaluation

Model discrimination in both the derivation and validation data sets was evaluated using the c-statistic. In the derivation sample, both the apparent area under the receiver-operating characteristic (ROC) curve and the optimism-corrected areas under the ROC curve were estimated. Optimism-corrected measures were obtained using 200 bootstrap samples drawn with replacement from the derivation sample. We assessed calibration in several ways, including the Hosmer–Lemeshow chi-square statistic, the calibration slope, and calibration in the large (20). We examined linear shrinkage estimators to confirm no overfit ($\gamma > 0.85$) and evaluated model performance with the Brier score (21).

Comparisons between the EHMRG and other risk scores were performed in both derivation and validation data sets by examining the differences of areas under the empirical ROC curves for 7-day mortality using methods previously described (22). Given the paucity of published ED-based risk scores for HF mortality, we compared performance of the EHMRG model with hospitalization-based risk scores, which studied different cohorts of patients who had already been hospitalized (9–11). The predictiveness curve of the EHMRG was constructed in the validation data set by plotting ordered risk percentile (*x*-axis) versus risk for 7-day mortality (*y*-axis), using previously published methods (23). Sensitivity and negative predictive value were determined by examining deaths in higher or lower risk groups dichotomized at different EHMRG thresholds. All analyses were conducted using SAS, version 9.2 (SAS Institute, Cary, North Carolina), for Unix environments.

Table 3. **EHMRG 7-Day Mortality Risk Score**

Variable	Units	Additive or Multiplicative Component
Age	y	2 × age
Transported by EMS	If "yes"	+60
SBP	mm Hg*	−1 × SBP
Heart rate	beats/min†	1 × heart rate
Oxygen saturation	%‡	−2 × oxygen saturation
Creatinine	mg/dL§	20 × creatinine
Potassium	4.0 to 4.5 mmol/L	0
	≥4.6 mmol/L	+30
	≤3.9 mmol/L	+5
Troponin	>ULN	+60
Active cancer	If "yes"	+45
Metolazone at home	If "yes"	+60
Adjustment factor‖		+12
Total		**EHMRG score¶**

EHMRG = Emergency Heart Failure Mortality Risk Grade; EMS = emergency medical services; SBP = systolic blood pressure; ULN = upper limit of normal.
* Initial/triage SBP, maximum of 160 mm Hg.
† Initial/triage heart rate, minimum of 80 beats/min and maximum of 120 beats/min.
‡ Lowest initial/triage oxygen saturation, maximum of 92%.
§ If creatinine concentration is in μmol/L, divide by 88.4 to convert to mg/dL.
‖ Adjustment factor of +12 added to allow for an approximate 0 median score.
¶ All variables are required to calculate the score; users are cautioned against estimating component values. The EHMRG is not for use in patients who are dialysis-dependent.

Role of the Funding Source

The study was funded by the Canadian Institutes of Health Research. The funding source had no role in the design, conduct, and analysis of this study or in the decision to submit the manuscript for publication.

RESULTS

Derivation and Validation Cohorts

We examined 15 164 patients with HF who visited the ED and fulfilled the Framingham criteria for HF:

10 781 were hospitalized and 4383 were discharged. Of the 12 591 patients included in the study, 7433 constituted the derivation cohort (5254 hospitalized and 2179 discharged) and 5158 constituted the validation cohort (3560 hospitalized and 1598 discharged). **Figure 1** shows a flow diagram of the study cohort and exclusion criteria.

The mean age of the derivation cohort was 75.4 years (SD, 11.4), with 3825 men (51.5%). The mean age of the validation cohort was 75.7 years (SD, 11.4), with 2661 men (51.6%). Overall, 247 deaths occurred, with a 7-day mortality rate of 2.0%. The 7-day mortality rate was 1.8% in the derivation cohort (135 deaths) and 2.2% in the validation cohort (112 deaths). Although baseline characteristics of the 2 cohorts were similar, the validation cohort had marginally higher creatinine concentration, more patients transported by emergency medical services, a greater proportion of patients using diuretics, and more patients with troponin elevation (**Table 1**).

Predictors of HF Mortality in the ED

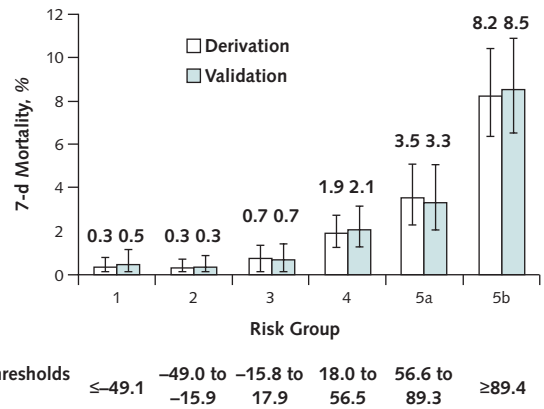
Of 37 covariates considered clinically and for reliability (543 patients), 31 (84%) met criteria for univariate analysis in the derivation cohort. The **Appendix Table** (available at www.annals.org) shows covariates assessed by univariate analysis for the outcome of 7-day mortality. Predictors of 7-day death included lower initial systolic blood pressure, oxygen saturation, and hemoglobin concentration. Higher leukocyte count and potassium, creatinine, and nonnormal troponin levels were associated with increased risk for death. Patients who were transported by emergency medical services and those presenting with acute HF despite being prescribed furosemide or metolazone before ED arrival also had higher mortality risk.

Multivariate Analysis

Table 2 shows predictors of 7-day mortality. **Appendix Figure 1** (available at www.annals.org) shows cubic regression splines adjusted for multivariate model covariates. The relationships of age and creatinine concentration with the log odds of 7-day mortality were linear. Systolic blood pressure and oxygen saturation were inversely related to the log odds of death, with an attenuated slope at higher values. Potassium concentration displayed a U-shaped relationship with mortality. In the derivation set, the c-statistic of the multivariate model was 0.805, suggesting high discrimination. The bootstrap-corrected unbiased estimate of the ROC area was 0.811 (95% CI, 0.770 to 0.847). There was no lack of model fit (Hosmer–Lemeshow chi-square statistic, 4.31; $P = 0.828$) and no overfit, as determined by a heuristic linear shrinkage estimator ($\gamma = 0.946$).

In the validation data set, the c-statistic of the multivariate model was 0.826, with no lack of model fit (Hosmer–Lemeshow chi-square statistic, 2.99; $P = 0.935$). In the validation data set, the calibration slope was 0.970 (CI, 0.810 to 1.131) and the calibration in the large P value was 0.923, denoting no miscalibration. The Brier score was 0.20.

Figure 2. **Absolute 7-day mortality rates and 95% CIs, by EHMRG score.**



Error bars are 95% CIs. EHMRG = Emergency Heart Failure Mortality Risk Grade.

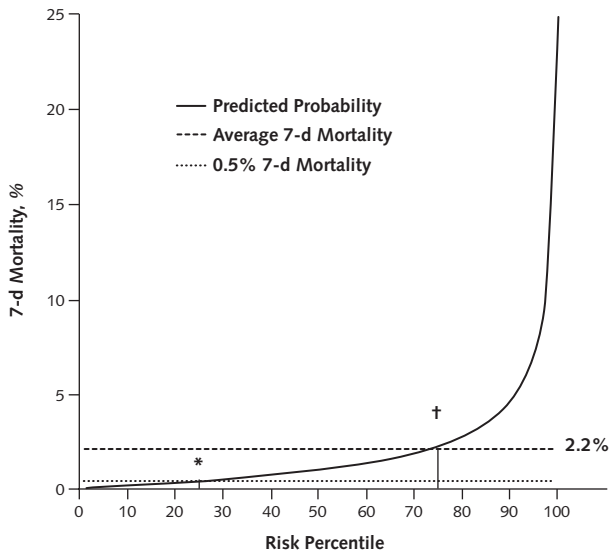
EHMRG Score

The EHMRG score comprises multiplicative and additive variables, with the method for calculation shown in **Table 3** (online calculator available at www.annals.org). An adjustment factor of 12 points was added to shift the median value to approximately 0, such that higher and numerically positive EHMRG scores conferred increased risk for death. The EHMRG score was normally distributed (see **Appendix Figure 2**, available at www.annals.org), with a mean score of 5.9 (SD, 62.0) and a median score of 0.5 (25th, 75th percentiles: −40.2, 44.6). For each 20-point increase in the EHMRG score, the odds of 7-day death increased by 41% (OR, 1.41 [CI, 1.34 to 1.48]) in the derivation cohort and by 39% (OR, 1.39 [CI, 1.32 to 1.47]) in the validation cohort (both $P < 0.001$). For each 1-SD increase in EHMRG, the odds of 7-day mortality were increased 2.9-fold in the derivation (OR, 2.88 [CI, 2.47 to 3.36]) and validation (OR, 2.92 [CI, 2.44 to 3.49]) cohorts (both $P < 0.001$). The c-statistic of the EHMRG was 0.807 in the derivation cohort and 0.806 (CI, 0.761 to 0.842) after bootstrap correction. The c-statistic was 0.803 in the validation cohort and 0.804 (CI, 0.763 to 0.840) after bootstrap correction.

EHMRG Risk Categories

Figure 2 shows mortality rates and 95% CIs according to risk score quintiles. Mortality rates were 0.3% in quintiles 1 (score, ≤−49.1) and 2 (score, −49.0 to −15.9) in the derivation cohort. Mortality was higher in quintiles 3 (score, −15.8 to 17.9) and 4 (score, 18.0 to 56.5), with the latter approximating the overall 7-day mortality rate of 2%. Quintile 5 (score, ≥56.6) contained the 2 highest risk deciles, with mortality rates of 3.5% in decile 9 (risk group 5a score, 56.6 to 89.3) and 8.2% in decile 10 (risk group

Figure 3. **Predictiveness of the EMHRG showing ordered distribution of 7-day mortality risk.**



Vertical bars indicate 25th (*) and 75th (†) risk percentiles. EHMRG = Emergency Heart Failure Mortality Risk Grade.

5b score, ≥89.4). The results were similar when the same score thresholds were applied to the validation data set (**Figure 2**). Examining the EHMRG predictiveness curve (**Figure 3**), 27% of all patients had predicted risks that exceeded the average 7-day mortality rate, whereas 25% had lower than 0.5% mortality risk. At the 25th, 50th, and 75th percentiles of risk (probabilities of death were 0.5%, 1.1%, and 2.3%, respectively), sensitivities were 95.5%, 90.2%, and 69.6%, whereas negative predictive values were 99.6%, 99.6%, and 99.1%, respectively.

Regardless of whether the HF subcohort was discharged from the ED or hospitalized, the EHMRG score was similarly effective in stratifying mortality risk. In a post hoc analysis combining validation and derivation cohorts, the 7-day mortality rate in the 2 lowest risk quantiles was 0.2% among those discharged from the ED, with greater than 8-fold risk in quantile 5a and 21-fold risk in the highest risk group (quantile 5b), as shown in **Table 4** (both $P < 0.001$ vs. quantiles 1 and 2 combined). Among those who were hospitalized, mortality in the 2 lowest risk quantiles was 0.4%, with nearly 10-fold risk in quantile 5a and 23-fold risk in the highest risk group (quantile 5b), as shown in **Table 4** (both $P < 0.001$ vs. quantiles 1 and 2 combined).

Comparison With Hospitalization-Based Algorithms

The EHMRG (derivation c-statistic = 0.807, validation c-statistic = 0.803) had superior discrimination than hospitalization-based risk algorithms (9–11), which had c-statistics that ranged from 0.528 to 0.665 in the derivation data set and from 0.598 to 0.707 in the validation data set (all comparisons $P < 0.001$ vs. the EHMRG score). Compared with a previously developed hospitalization-based model (Enhanced Feedback for Effective Cardiac Treatment-HF risk score) intended for 30-day mortality prediction (12), the EHMRG had superior discrimination overall (c-statistic, 0.808 vs. 0.755; $P < 0.001$) when patients who were hospitalized or discharged from the ED were included.

DISCUSSION

Clinical decisions pertaining to the acute care of patients with HF in the ED may be improved by availabil-

Table 4. **Mortality Rates and ORs for 7-Day Mortality Stratified by Discharge Versus Hospitalization Status in the Derivation and Validation Data Sets Combined**

Risk Quantile	Score Range	7-Day Mortality Rate (95% CI)	*P* Value*	OR (95% CI)	*P* Value*
Discharged from ED†‡					
1 or 2	≤–15.9	0.21 (0.06 to 0.53)	–	Reference	–
3	−15.8 to 17.9	0.25 (0.03 to 0.91)	0.82	1.22 (0.22 to 6.67)	0.82
4	18.0 to 56.5	1.45 (0.67 to 2.74)	≤0.001	7.03 (2.16 to 22.92)	≤0.001
5a	56.6 to 89.3	1.71 (0.56 to 3.95)	<0.01	8.32 (2.22 to 31.16)	<0.01
5b	≥89.4	4.27 (1.73 to 8.60)	≤0.001	21.29 (6.17 to 73.51)	≤0.001
Hospitalized†§					
1 or 2	≤–15.9	0.41 (0.21 to 0.72)	–	Reference	–
3	−15.8 to 17.9	0.93 (0.53 to 1.50)	<0.05	2.26 (1.07 to 4.79)	<0.05
4	18.0 to 56.5	2.13 (1.53 to 2.89)	≤0.001	5.25 (2.74 to 10.03)	≤0.001
5a	56.6 to 89.3	3.88 (2.80 to 5.22)	≤0.001	9.72 (5.09 to 18.57)	≤0.001
5b	≥89.4	8.89 (7.37 to 10.60)	≤0.001	23.52 (12.92 to 42.84)	≤0.001

ED = emergency department; OR = odds ratio.
* *P* values for comparison with quantiles 1 and 2 (reference).
† Exact percentages not shown because cell sizes ≤5 persons cannot be reported, as per Institute for Clinical Evaluative Sciences regulations under the Ontario Personal Health Information Protection Act.
‡ Rounded to the nearest 5%, approximately 50%, 20%, 15%, 10%, and 5% of discharged cohort were in quantiles 1 or 2, 3, 4, 5a, and 5b, respectively.
§ Rounded to the nearest 5%, approximately 35%, 20%, 20%, 10%, and 15% of hospitalized cohort were in quantiles 1 or 2, 3, 4, 5a, and 5b, respectively.

ity of prognostic information. However, evidence-based decision-support tools for the broad group of patients presenting to the ED with HF have not been available. In this study, we developed and validated a prediction model specifically for patients with acute HF in the ED. An online calculator is available at www.annals.org. Unlike previous studies that examined only patients who were hospitalized, the EHMRG was developed in a study cohort whose entry point was presentation to the ED and included those who were ultimately discharged or hospitalized. The EHMRG performed well in a validation data set and can assist in acute decision making by providing the risk for death in the next 7 days after ED presentation. Mortality risks in the highest 2 deciles were more than 10-fold (9th decile) and 25-fold (10th decile) higher than the 0.3% event rate in the 2 lowest risk quintiles.

Previous studies have suggested a mortality effect of some of the EHMRG predictor covariates, including initial systolic blood pressure, heart rate, potassium, and creatinine (9–11). However, although oxygen saturation was cited in guidelines as a potential reason for hospitalization, it has not been associated with prognosis (24). Furthermore, despite demonstration of higher mortality with troponin elevation (25), multivariate HF risk models have not included troponin as a predictor of outcome. As seen with other conditions studied in the ED setting (26), patients who required paramedic transport were also at substantially higher risk for death. Finally, diuretic use is known to be an important prognostic factor that reflects greater severity of HF, dysfunction of the cardiorenal axis, or both (27). Our data suggest that the occurrence of acute HF despite use of metolazone was a more potent factor associated with early death in multivariate analysis.

A major strength of EHMRG is that it encompasses all patients presenting to the ED, regardless of whether they were hospitalized or discharged. If a model is intended to guide hospitalization-versus-discharge decisions based on acute prognosis, it is important to examine a patient sample whose inception is presentation to the ED and not only those who were hospitalized. The examination of a broad cohort of ED patients with HF was probably a major contributor to the superior discrimination of EHMRG compared with other models that examined hospitalized cohorts (9–11), which were not designed to ensure accuracy in those with different HF trajectory or severity, also known as spectrum transportability (13). Consensus-based considerations to assist hospitalization or discharge decisions have been published in HF guidelines; however, these recommendations were based on disparate studies of isolated predictors (24).

Many variables included in the EHMRG reflect biological perturbations that predispose to increased mortality risk. Among patients with HF who are acutely ill, lower initial oxygen saturation reflects greater respiratory compromise and pulmonary congestion (28). Lack of elevation of initial systolic blood pressure in the patient with acute HF is a predictor of death, reflecting underlying left ventricular contractile dysfunction (29). Higher initial heart rates may reflect several contributing processes, including the need for increased chronotropy to maintain cardiac output and sympathoadrenergic response (30). The mechanism for the biphasic effect of potassium is complex, reflecting cardiorenal disturbances of the renin–angiotensin–aldosterone system, effects of medications, or alterations in renal function (31).

The implications of the EHMRG relate to its ability to support decision making in the ED by complementing the physician's assessment of symptomatic response and clinical factors with prognostic data. Methods for acute prognostication that are not contingent on knowledge of underlying HF cause and ventricular functional status are needed, because these may be unknown in the ED setting. The potential effect of the EHMRG depends on the baseline rate of hospitalization of patients with HF from the ED. At institutions with higher rates of hospitalization of low-risk patients, use of the score may identify patients who can be safely discharged with appropriate postdischarge care (32). At EDs with higher baseline rates of discharge, the EHMRG may identify high-risk patients who may have been otherwise discharged and might benefit from rapid diagnostic testing and therapy provided in the acute hospital setting (32). In our study population (**Table 4**), if the EHMRG was applied to the discharged cohort and patients with scores of 18.0 or greater (risk quantiles 4 and 5) were hospitalized, 28.5% of patients would be reclassified to hospitalization. A more stringent score of 56.6 or greater (risk quantile 5) would have resulted in 12.1% of patients being reclassified to hospitalization. The caveat that applies to all decision-support tools is pertinent, because the EHMRG is intended to assist the clinician, who should ultimately synthesize the risk score with clinical judgment because the decision to hospitalize or discharge patients with HF weighs several potential factors. Symptomatic improvement, ability of the patient to seek follow-up care, and social circumstances should also be considered, along with quantification of acute prognosis. However, published data suggest that there is potential for improvement in hospitalization-versus-discharge decision making, because many low-risk patients with HF are hospitalized and some high-risk patients are discharged (7).

Our study has some limitations. The EHMRG was designed for evaluation of patients with either new or recurrent episodes of acute HF, and not chronic HF, and thus its performance was not compared with risk models for symptomatically stable disease (33). Brain natriuretic peptide testing was not frequently performed in the ED setting and was not included in the risk model. Indeed, routine testing is not suggested in HF guidelines (34) and has not been demonstrated to be beneficial in the acute setting (35, 36). Determination of medication use was based on prescriptions in the medical record before ED arrival, and adherence to these drugs was not verified by

using administrative pharmacare databases. Left ventricular function was not included in this analysis because the acute evaluation of systolic function is often not available in the ED setting. However, inclusion of left ventricular ejection fraction of 0.45 or less ($n = 1527$), more than 0.45 ($n = 969$), or unknown ($n = 2662$) to the EHMRG model increased the c-statistic only minimally, from 0.826 to 0.828, in the validation data set ($P = 0.309$). Finally, because the 7-day mortality rate of patients who were hospitalized was approximately 2-fold that of those discharged from the ED (**Table 4**), other unmeasured factors that may have had an effect on mortality probably remained (for example, reduced β-blocker dosage or nonuse resulting in higher heart rate). However, decision-support tools require a balance between variable inclusiveness and model simplicity, limiting the incorporation of an exhaustive list of potential factors in the model.

In conclusion, the EHMRG is a simple clinical risk model that can predict, with high accuracy, acute mortality among patients with HF who present to the ED. The importance of this risk model for prognostication is underscored by the inclusion of patients who present to the ED regardless of disposition and its utility in guiding acute care decisions. The care and outcomes of patients with acute HF may be substantially improved if clinical judgment is supported by prognostic quantification in emergent care.

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Disclaimer: The opinions, results, and conclusions are those of the authors and no endorsement by the Ministry of Health and Long-Term Care or the Institute for Clinical Evaluative Sciences is intended or should be inferred.

Acknowledgment: The authors thank Dr. David Henry for reviewing and providing comments on an earlier draft of the manuscript.

Grant Support: The Institute for Clinical Evaluative Sciences is supported in part by a grant from the Ontario Ministry of Health and Long-Term Care. This research was supported by an operating grant from the Canadian Institutes of Health Research (CIHR MOP 114937), a Canadian Institutes of Health Research clinician-scientist award (Dr. D.S. Lee), a Career Investigator Award from the Heart and Stroke Foundation of Ontario (Dr. Austin, Dr. Tu), a Canadian Institutes of Health Research Applied Chair in Health Services and Policy Research (Dr. Schull), and a Canada Research Chair in Health Services Research (Dr. Tu).

Potential Conflicts of Interest: Disclosures can be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=M11-2177.

Reproducible Research Statement: *Study protocol:* Available from Dr. D.S. Lee (e-mail, dlee@ices.on.ca). *Data set and statistical code:* Not available.

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References

1. **Jencks SF, Williams MV, Coleman EA.** Rehospitalizations among patients in the Medicare fee-for-service program. N Engl J Med. 2009;360:1418-28. [PMID: 19339721]
2. **Sanderson JE, Tse TF.** Heart failure: a global disease requiring a global response [Editorial]. Heart. 2003;89:585-6. [PMID: 12748201]
3. **Krumholz HM, Parent EM, Tu N, Vaccarino V, Wang Y, Radford MJ, et al.** Readmission after hospitalization for congestive heart failure among Medicare beneficiaries. Arch Intern Med. 1997;157:99-104. [PMID: 8996046]
4. **Collins SP, Schauer DP, Gupta A, Brunner H, Storrow AB, Eckman MH.** Cost-effectiveness analysis of ED decision making in patients with non-high-risk heart failure. Am J Emerg Med. 2009;27:293-302. [PMID: 19328373]
5. **Peacock WF, Braunwald E, Abraham W, Albert N, Burnett J, Christenson R, et al.** National Heart, Lung, and Blood Institute working group on emergency department management of acute heart failure: research challenges and opportunities. J Am Coll Cardiol. 2010;56:343-51. [PMID: 20650354]
6. **Butler J, Hanumanthu S, Chomsky D, Wilson JR.** Frequency of low-risk hospital admissions for heart failure. Am J Cardiol. 1998;81:41-4. [PMID: 9462604]
7. **Lee DS, Schull MJ, Alter DA, Austin PC, Laupacis A, Chong A, et al.** Early deaths in patients with heart failure discharged from the emergency department: a population-based analysis. Circ Heart Fail. 2010;3:228-35. [PMID: 20107191]
8. **Gheorghiade M, Braunwald E.** A proposed model for initial assessment and management of acute heart failure syndromes. JAMA. 2011;305:1702-3. [PMID: 21521852]
9. **Fonarow GC, Adams KF Jr, Abraham WT, Yancy CW, Boscardin WJ; ADHERE Scientific Advisory Committee, Study Group, and Investigators.** Risk stratification for in-hospital mortality in acutely decompensated heart failure: classification and regression tree analysis. JAMA. 2005;293:572-80. [PMID: 15687312]
10. **Chin MH, Goldman L.** Correlates of major complications or death in patients admitted to the hospital with congestive heart failure. Arch Intern Med. 1996;156:1814-20. [PMID: 8790075]
11. **Auble TE, Hsieh M, Gardner W, Cooper GF, Stone RA, McCausland JB, et al.** A prediction rule to identify low-risk patients with heart failure. Acad Emerg Med. 2005;12:514-21. [PMID: 15930402]
12. **Lee DS, Austin PC, Rouleau JL, Liu PP, Naimark D, Tu JV.** Predicting mortality among patients hospitalized for heart failure: derivation and validation of a clinical model. JAMA. 2003;290:2581-7. [PMID: 14625335]
13. **Justice AC, Covinsky KE, Berlin JA.** Assessing the generalizability of prognostic information. Ann Intern Med. 1999;130:515-24. [PMID: 10075620]
14. **Tu JV, Donovan LR, Lee DS, Wang JT, Austin PC, Alter DA, et al.** Effectiveness of public report cards for improving the quality of cardiac care: the EFFECT study: a randomized trial. JAMA. 2009;302:2330-7. [PMID: 19923205]
15. **McKee PA, Castelli WP, McNamara PM, Kannel WB.** The natural history of congestive heart failure: the Framingham study. N Engl J Med. 1971;285:1441-6. [PMID: 5122894]
16. **Guttmann A, Schull MJ, Vermeulen MJ, Stukel TA.** Association between waiting times and short term mortality and hospital admission after departure from emergency department: population based cohort study from Ontario, Canada. BMJ. 2011;342:d2983. [PMID: 21632665]
17. **Van Spall HG, Atzema C, Schull MJ, Newton GE, Mak S, Chong A, et al.** Prediction of emergent heart failure death by semi-quantitative triage risk stratification. PLoS One. 2011;6:e23065. [PMID: 21853068]
18. **Harrell FE Jr.** Regression Modeling Strategies: With Applications to Linear Models, Logistic Regression, and Survival Analysis Series. New York: Springer; 2001.
19. **Sullivan LM, Massaro JM, D'Agostino RB Sr.** Presentation of multivariate data for clinical use: The Framingham Study risk score functions. Stat Med. 2004;23:1631-60. [PMID: 15122742]
20. **Steyerberg EW.** Clinical Prediction Models: A Practical Approach to Devel-

opment, Validation, and Updating Series. New York: Springer; 2009.
21. **Harrell FE Jr, Lee KL, Mark DB.** Multivariable prognostic models: issues in developing models, evaluating assumptions and adequacy, and measuring and reducing errors. Stat Med. 1996;15:361-87. [PMID: 8668867]
22. **DeLong ER, DeLong DM, Clarke-Pearson DL.** Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. Biometrics. 1988;44:837-45. [PMID: 3203132]
23. **Pepe MS, Feng Z, Huang Y, Longton G, Prentice R, Thompson IM, et al.** Integrating the predictiveness of a marker with its performance as a classifier. Am J Epidemiol. 2008;167:362-8. [PMID: 17982157]
24. **Lindenfeld J, Albert NM, Boehmer JP, Collins SP, Ezekowitz JA, Givertz MM, et al; Heart Failure Society of America.** HFSA 2010 Comprehensive Heart Failure Practice Guideline. J Card Fail. 2010;16:e1-194. [PMID: 20610207]
25. **Peacock WF 4th, De Marco T, Fonarow GC, Diercks D, Wynne J, Apple FS, et al; ADHERE Investigators.** Cardiac troponin and outcome in acute heart failure. N Engl J Med. 2008;358:2117-26. [PMID: 18480204]
26. **Perry JJ, Stiell IG, Sivilotti ML, Bullard MJ, Lee JS, Eisenhauer M, et al.** High risk clinical characteristics for subarachnoid haemorrhage in patients with acute headache: prospective cohort study. BMJ. 2010;341:c5204. [PMID: 21030443]
27. **Abdel-Qadir HM, Tu JV, Yun L, Austin PC, Newton GE, Lee DS.** Diuretic dose and long-term outcomes in elderly patients with heart failure after hospitalization. Am Heart J. 2010;160:264-271.e1. [PMID: 20691831]
28. **Park M, Sangean MC, Volpe Mde S, Feltrim MI, Nozawa E, Leite PF, et al.** Randomized, prospective trial of oxygen, continuous positive airway pressure, and bilevel positive airway pressure by face mask in acute cardiogenic pulmonary edema. Crit Care Med. 2004;32:2407-15. [PMID: 15599144]
29. **Gheorghiade M, Abraham WT, Albert NM, Greenberg BH, O'Connor CM, She L, et al; OPTIMIZE-HF Investigators and Coordinators.** Systolic blood pressure at admission, clinical characteristics, and outcomes in patients hospitalized with acute heart failure. JAMA. 2006;296:2217-26. [PMID: 17090768]
30. **Eisenhofer G, Friberg P, Rundqvist B, Quyyumi AA, Lambert G, Kaye DM, et al.** Cardiac sympathetic nerve function in congestive heart failure. Circulation. 1996;93:1667-76. [PMID: 8653872]
31. **Packer M.** Potential role of potassium as a determinant of morbidity and mortality in patients with systemic hypertension and congestive heart failure. Am J Cardiol. 1990;65:45E-51E. [PMID: 2178378]
32. **Lee DS, Stukel TA, Austin PC, Alter DA, Schull MJ, You JJ, et al.** Improved outcomes with early collaborative care of ambulatory heart failure patients discharged from the emergency department. Circulation. 2010;122:1806-14. [PMID: 20956211]
33. **Levy WC, Mozaffarian D, Linker DT, Sutradhar SC, Anker SD, Cropp AB, et al.** The Seattle Heart Failure Model: prediction of survival in heart failure. Circulation. 2006;113:1424-33. [PMID: 16534009]
34. **Thygesen K, Mair J, Mueller C, Huber K, Weber M, Plebani M, et al.** Recommendations for the use of natriuretic peptides in acute cardiac care: A position statement from the Study Group on Biomarkers in Cardiology of the ESC Working Group on Acute Cardiac Care. Eur Heart J. 2011. [PMID: 21292681]
35. **Schneider HG, Lam L, Lokuge A, Krum H, Naughton MT, De Villiers Smit P, et al.** B-type natriuretic peptide testing, clinical outcomes, and health services use in emergency department patients with dyspnea: a randomized trial. Ann Intern Med. 2009;150:365-71. [PMID: 19293069]
36. **Singer AJ, Birkhahn RH, Guss D, Chandra A, Miller CD, Tiffany B, et al.** Rapid Emergency Department Heart Failure Outpatients Trial (REDHOT II): a randomized controlled trial of the effect of serial B-type natriuretic peptide testing on patient management. Circ Heart Fail. 2009;2:287-93. [PMID: 19808351]

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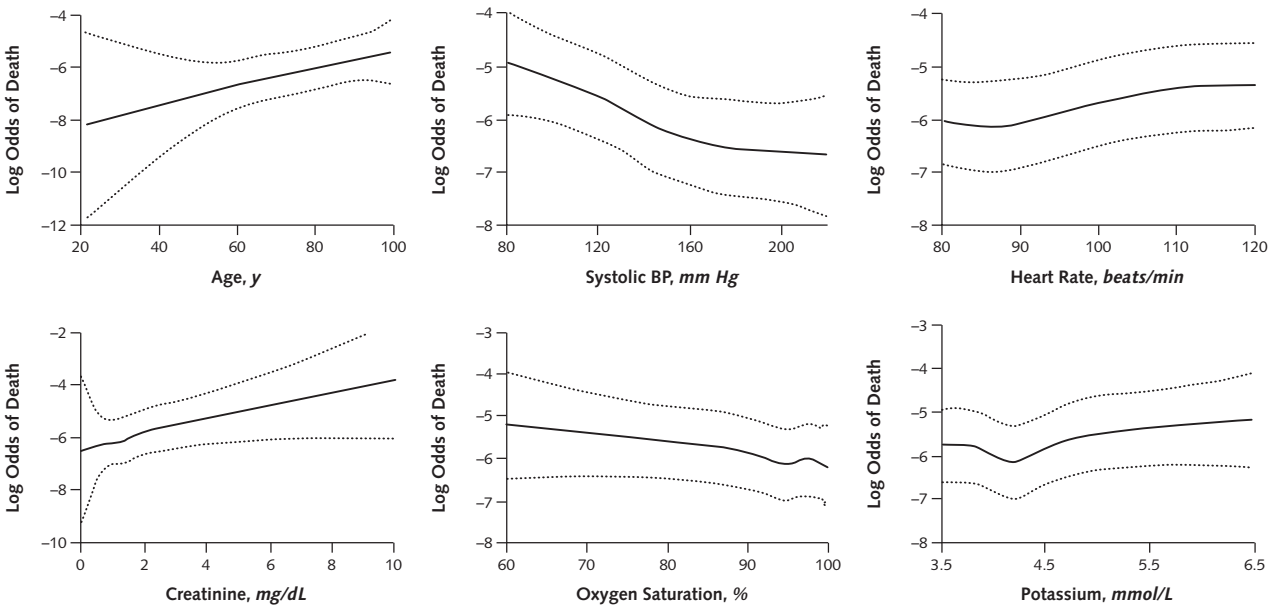
Appendix Table. **Univariate Analysis for 7-Day Death (All Variables)**

Variable	Units or Reference Category	OR (95% CI)	*P* Value
Age	10-y increase	1.53 (1.28–1.83)	<0.001
Male	vs. female	1.11 (0.79–1.57)	0.54
Transported by EMS	vs. no EMS	3.90 (2.69–5.65)	<0.001
Nursing home or long-term care	vs. from home	3.09 (1.98–4.82)	<0.001
CTAS score			
1 (triage critical)		2.72 (0.82–9.01)	0.103
2–3 (urgent or emergent)		1.58 (0.64–3.87)	0.32
4–5 (nonurgent)	Reference	Reference	NA
SBP	20–mm Hg decrease	1.39 (1.23–1.57)	<0.001
Heart rate	10-beats/min increase	1.06 (0.99–1.14)	0.077
Respiratory rate	5-breaths/min increase	1.07 (0.95–1.21)	0.29
Oxygen saturation	5% decrease	1.21 (1.09–1.34)	<0.001
Coronary artery disease		1.19 (0.84–1.69)	0.34
Previous coronary revascularization*		0.53 (0.32–0.88)	0.013
Diabetes		0.78 (0.54–1.12)	0.173
Hypertension		0.76 (0.54–1.08)	0.127
Cerebrovascular disease†		1.55 (1.03–2.33)	0.035
Peripheral artery disease		0.97 (0.56–1.66)	0.90
Chronic pulmonary disease		1.41 (0.96–2.06)	0.077
Dementia		1.85 (1.05–3.24)	0.033
Active cancer		2.41 (1.55–3.76)	<0.001
Hemoglobin	10-g/L increase	0.86 (0.79–0.94)	<0.001
Leukocyte count	1×10^9 cells/L increase	1.03 (1.01–1.06)	0.012
Sodium	5-mmol/L increase	0.98 (0.84–1.14)	0.81
Potassium	1-mmol/L increase	1.64 (1.31–2.04)	<0.001
Creatinine	1-mg/dL (88.4-μmol/L) increase	1.53 (1.33–1.77)	<0.001
Troponin	>ULN vs. normal	4.32 (2.99–6.24)	<0.001
Atrial fibrillation or flutter		1.33 (0.94–1.90)	0.111
QRS duration	10-msec increase	1.00 (0.95–1.05)	0.96
Medications at home			
ACE inhibitor or ARB		0.81 (0.57–1.14)	0.22
β-Adrenoreceptor antagonist		0.87 (0.62–1.23)	0.43
Digoxin		1.41 (0.95–2.09)	0.089
Furosemide		1.73 (1.21–2.47)	0.003
Metolazone		3.16 (1.36–7.32)	0.007

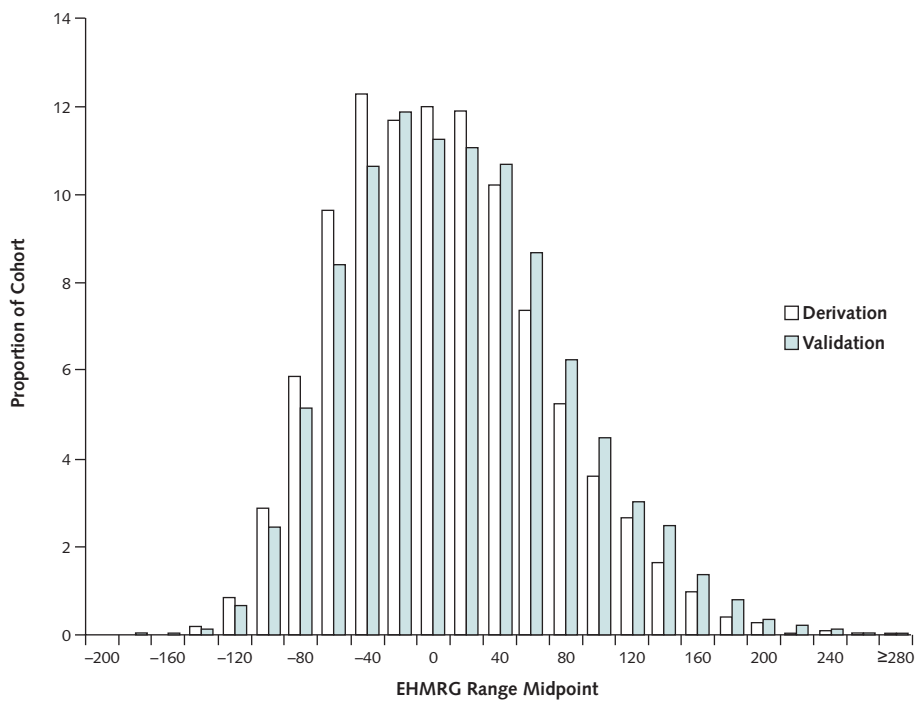
ACE = angiotensin-converting enzyme; ARB = angiotensin–II receptor blocker; CTAS = Canadian Triage and Acuity Scale; EMS = emergency medical services; NA = not applicable; OR = odds ratio; SBP = systolic blood pressure; ULN = upper limit of normal.
* Previous percutaneous coronary intervention or coronary artery bypass graft surgery.
† Stroke or transient ischemic attack.

Appendix Figure 1. **Cubic spline curves showing adjusted log odds of 7-day mortality for continuous covariates.**



To convert the creatinine units from mg/dL to μmol/L, multiply value by 88.4. BP = blood pressure.

Appendix Figure 2. **Distribution of EHMRG score in derivation and validation data sets.**



EHMRG = Emergency Heart Failure Mortality Risk Grade.