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Is Survival Better at Hospitals With Higher “End-of-Life” Treatment Intensity?

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Abstract

Background—Concern regarding wide variations in spending and intensive care unit use for patients at the end of life hinges on the assumption that such treatment offers little or no survival benefit.

Objective—To explore the relationship between hospital “end-of-life” (EOL) treatment intensity and postadmission survival.

Research Design—Retrospective cohort analysis of Pennsylvania Health Care Cost Containment Council discharge data April 2001 to March 2005 linked to vital statistics data through September 2005 using hospital-level correlation, admission-level marginal structural logistic regression, and pooled logistic regression to approximate a Cox survival model.

Subjects—A total of 1,021,909 patients 65 years old, incurring 2,216,815 admissions in 169 Pennsylvania acute care hospitals.

Measures—EOL treatment intensity (a summed index of standardized intensive care unit and life-sustaining treatment use among patients with a high predicted probability of dying [PPD] at admission) and 30- and 180-day postadmission mortality.

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Results—There was a nonlinear negative relationship between hospital EOL treatment intensity and 30-day mortality among all admissions, although patients with higher PPD derived the greatest benefit. Compared with admission at an average intensity hospital, admission to a hospital 1 standard deviation below versus 1 standard deviation above average intensity resulted in an adjusted odds ratio of mortality for admissions at low PPD of 1.06 (1.04–1.08) versus 0.97 (0.96–0.99); average PPD: 1.06 (1.04–1.09) versus 0.97 (0.96–0.99); and high PPD: 1.09 (1.07–1.11) versus 0.97 (0.95–0.99), respectively. By 180 days, the benefits to intensity attenuated (low PPD: 1.03 [1.01–1.04] vs. 1.00 [0.98–1.01]; average PPD: 1.03 [1.02–1.05] vs. 1.00 [0.98–1.01]; and high PPD: 1.06 [1.04–1.09] vs. 1.00 [0.98–1.02]), respectively.

Conclusions—Admission to higher EOL treatment intensity hospitals is associated with small gains in postadmission survival. The marginal returns to intensity diminish for admission to hospitals above average EOL treatment intensity and wane with time.

Keywords

intensive care; terminal care; mortality; hospitals; efficiency; quality

Greater spending on acute care and physician services among Medicare beneficiaries in the last 6 months of life has gained substantial traction as a measure of inefficiency.^{1–3} The general assumption is that the provision of intensive and expensive care to those who died was a waste of resources. Indeed, Fisher et al reported that Medicare beneficiaries who lived in hospital referral regions with higher last 6-months-of-life spending did not live longer.^{4,5} These investigators recently demonstrated marked variation in the use of acute care services in the last 6 months of life among cohorts of Medicare beneficiaries loyal to “highly respected” hospitals.¹ The hospital is a much more proximate measure than the hospital referral region, yet we do not know whether higher “end-of-life” (EOL) hospital treatment intensity impacts survival.

The purpose of the current study is to examine whether there is a survival benefit of admission to a high EOL intensity hospital. In a departure from the approach used by other investigators, we define EOL treatment intensity as the use of intensive care and life-sustaining treatments among patients who had a high probability of dying upon admission, rather than among those who died. By using intensive care and life-sustaining treatments, rather than spending, we measure treatment provided with life-saving (or prolonging), not palliative, intent. By using a cohort of patients who have a high probability of dying, rather than those who died, we capture treatment decisions for patients who may be dying that are made under conditions of prognostic uncertainty, rather than treatment decisions that (in hindsight) were made for patients who actually died.^{6,7} Furthermore, if higher treatment intensity reduces mortality, then the patients making up the decedent cohorts at higher compared with lower intensity hospitals would differ. Using noncomparable denominators would introduce a systematic bias into the intensity measurement that could confound the relationship between treatment intensity and survival.

Methods

Overview

We conducted a retrospective cohort analysis of general acute care hospital discharges in Pennsylvania between April 1, 2001 and March 31, 2005, linked to vital statistics data through September 30, 2005. All admissions among patients 65 and older comprised the study population. The dependent variable was postadmission survival (measured at 30 and 180 days). The primary independent variable was the EOL treatment intensity of the treating hospital, measured as an index of intensive care unit (ICU) and life-sustaining treatment use

among the subset of the 65 and older study population who were at the “end-of-life.” We used Pennsylvania Health Care Cost Containment Council (PHC4) data because it includes a predicted probability of dying (PPD) during the hospitalization. The PPD is calculated from key clinical findings abstracted from the medical chart in the first 48 hours of admission and has condition-specific *c*-statistics of 0.84 to 0.88, substantially outperforming traditional claims-based risk prediction.⁸ We used the PPD for 3 important purposes, which we describe in greater detail in the sections that follow: (1) to identify the admissions that were at a high probability of dying to calculate each hospital's EOL treatment intensity; (2) to risk-adjust the admission-level regressions testing the relationship between the treating hospital's EOL treatment intensity and postadmission survival; and (3) to calculate propensity-score weights to use in these regressions to address some of the weaknesses inherent in observational studies and in so doing, to improve the strength of causal inference.

Sample Selection

Admissions to Pennsylvania acute care hospitals among patients 65 and older between April 1, 2001 and March 31, 2005 comprised the study population. We restricted the sample to persons aged 65 years and older because this group incurs 85% of all deaths and is the group for which spending in the last year of life has garnered most policy attention.⁹ For patients transferred from one hospital to another, we excluded the first admission from the analysis if the transfer occurred within 12 hours of initial admission (ie, we treated the first admission as a “triaging” hospital). Otherwise, we retained both admissions as separate observations.

Dependent Variable

The dependent variable was death, assessed at 2 time points postadmission: 30 days or 180 days.

Independent Variable: Hospital EOL Treatment Intensity

We previously published a detailed description of the development and validation of the independent variable, hospital end-of-life treatment intensity.¹⁰ In short, it is an index calculated by empirically weighting 6 Bayes' shrunk¹¹ case-mix standardized (observed-to-expected) treatment ratios (ICU admission, ICU length of stay, intubation/mechanical ventilation, tracheostomy, hemodialysis, and gastrostomy) among the subset of admissions 65 and older at each hospital who were at the end-of-life. For the current analysis, we defined EOL as admissions with a high probability of dying (eg, the highest 5% of predicted probability of inpatient mortality, corresponding to a probability of 0.21 or higher; mean = 0.41, 25th percentile = 0.26, 75th percentile = 0.51). We assumed that patients with a high probability of dying reflected those who providers might reasonably characterize as potentially “dying.” We chose ICU admission, intubation/mechanical ventilation, hemodialysis, and gastrostomy to capture initiation of life-sustaining treatments, and ICU LOS and tracheostomy to capture continuation of life-sustaining treatments, given that a significant source of variation in EOL treatment is willingness to withdraw life-sustaining treatments in anticipation of death.¹²

Covariables

Patient-level covariables eligible for inclusion in the admission-level models were age, race, sex, insurance status, admission type (emergent, urgent, elective), admission source (including hospital transfer), off-hours admission, home-hospital distance above the 90th percentile (>16 miles), PPD, and diagnoses (classified into categories using the clinical classification system [CCS, 2007 Version] developed by the Agency for Health Care Research and Quality [AHRQ]). Diagnoses included indicator variables for each of the 25

most common diagnoses among decedents in the first discharge diagnosis field (mentioned in Appendix: Top 25 diagnoses, online only, Supplemental Digital Content 1, Available at: <http://links.lww.com/MLR/A49>) and Elixhauser comorbidities¹³ in the second through eighth field.

Hospital-level covariables eligible for model inclusion were those potentially associated with postadmission survival that could confound the relationship between EOL treatment intensity and postadmission survival among all patients 65 and older, including the hospital's resident-to-bed ratio, mechanical ventilation volume,¹⁴ hospital bed size, and severity of illness, measured as the mean PPD among all admissions 65 and older.

Area-level variables eligible for model inclusion were the rural-urban continuum code of the hospital, the county-level Herfindahl-Hirschman Index, a measure of local hospital competition,¹⁵ and share of residents 65 and older below the poverty line in the patient's ZIP code from the 2000 census, a crude measure of socioeconomic status.^{16,7}

Statistical Analysis

Using an admission-level data file, we tested for differences in the characteristics of admissions to higher, compared with lower, EOL treatment intensity hospital, using ordinary least squares regression. Then, using a hospital-level data file, we similarly tested for differences in the characteristics of higher, compared with lower, EOL treatment intensity hospitals.

We then conducted 3 types of analyses, each of increasing statistical complexity, to explore the relationship between the treating hospital's EOL treatment intensity and postadmission mortality. First, we used a hospital-level data file to calculate a Pearson correlation coefficient between EOL treatment intensity and standardized 30- and 180-day postadmission mortality ratios (SMRs). We calculated SMRs using Bayes' shrinkage estimates of the observed-to-expected 30- and 180-day postadmission mortality rates for each hospital. Expected mortality models included only patient, not hospital, covariables.

Second, we used an admission-level data file to assess the multivariable relationship between hospital EOL treatment intensity and 30- and 180-day postadmission mortality. We adjusted for the correlation among admissions for the same patient using Huber-White robust standard errors. We did not adjust standard errors for clustering of patients within hospitals due to imperfect clustering of patients within hospitals: over one-third (37.3%) of patients with 2 hospitalizations were admitted to 2 different hospitals. To simultaneously address selection bias (eg, severity of illness varying systematically across hospitals of varying EOL treatment intensity) and time-varying confounders (eg, severity of illness being affected by the intensity of treatment received during a prior hospitalization), we used marginal structural modeling. Marginal structural models are a new class of epidemiologic models that increase the strength of causal inference in observational data. To implement the marginal structural models, we calculated generalized inverse probability weights, which are analogous to propensity scores, and then used these to weight the logistic regression.^{18,19} Our propensity-score weight was based on a model that included the demographic and clinical characteristics of the current admission as well as information from the immediate past prior hospitalization, including the prior hospital's EOL treatment intensity index and the patient's receipt of intensive care and life-sustaining treatments during that admission. This allowed us to capture the time-varying nature of the confounding relationship between treating hospitals' EOL treatment intensity and survival (given that the majority of patients in our sample had multiple admissions). We then trimmed the upper 1% of adjustment weights to reduce the effect of influential observations before applying them to the 30- and 180-day logistic regression mortality models. We included identical admission, hospital, and

area covariables in the 30- and 180-day models, retained if statistically significant at the P 0.05 level in either of the models.

Because patients could be admitted to more than one hospital in the 30- or 180-day window, it is theoretically possible that the admission-level analysis described above could be confounded by readmission rates if readmission and EOL treatment intensity were correlated.²⁰ So, in our third type of statistical approach, we used pooled logistic regression to approximate time-dependent Cox regression and to estimate a daily mortality hazard.²¹ Specifically, we conducted bootstrapped analysis of 500 repetitions using a 1% sample of patients (with replacement) in which we expanded each patient's hospitalization record to include 1 record per day. We then used pooled logistic regression (propensity-score-weighted, as before) to calculate the daily mortality hazard, conditional on the intensity of the treating hospital. Thus, the relative hazard, or risk, is only attributable to a single hospital, since patients are not admitted to more than one hospital in a day. We used identical admission and hospital covariables in the pooled logistic regression “Cox” models as in the previously described logistic regression models.

For the multivariable models, we did not want to assume that the relationship between EOL treatment intensity and survival was linear. Hence, we used visual inspection of crude mortality curves, fitting of these mortality curves with piecewise linear splines, and empirical model-fitting procedures to identify the optional functional forms for EOL treatment intensity (intensity, intensity², intensity³, 1/intensity, log(intensity)).

We present results by subgroups—patients who had an average predicted probability of death at admission (overall mean, or 4.6%), low predicted probability (mean of the fifth percentile, or 0%), and high predicted probability (mean of the 95th percentile, or 41%) to explore the hypothesis that higher EOL treatment intensity affects these populations differently. Specifically, we hypothesized that higher EOL treatment intensity might cause iatrogenic harm to the lowest risk patients.

We used SAS version 9.1.3 (SAS Institute, Cary, NC) and STATA version 9.2 (STATA Corp, College Station, TX) for all analyses.

Sensitivity Analyses

We repeated identical analyses after excluding the 17 facilities that reported data from multiple locations to avoid misspecification bias, and after excluding hospitals outside the 2 major cities, Pittsburgh and Philadelphia. We also repeated analyses restricted to each patient's first hospital admission. This is one way to address the concern that high readmission rates could artificially inflate survival estimates, if intensity and readmission rate are correlated, although it reduces both the power and the strength of causal inference. Finally, we repeated all of the primary and sensitivity analyses using the decedent-based EOL treatment intensity index, rather than the high-probability of dying-based EOL treatment intensity index.

Human Subjects and Role of the Sponsor

We conducted the study under a data use agreement with PHC4. The University of Pittsburgh Institutional Review Board reviewed and approved the protocol. Dr. Barnato, Professor Chang, and Mr. Farrell had full access to the data and take responsibility for the integrity of the analyses. We had complete independence from the funding agency, the National Institutes of Health, in designing, analyzing, and reporting the study.

Results

Sample Characteristics

Of 188 acute care facilities reporting data to PHC4 during the study period, we calculated EOL treatment intensity for 169 (89%) (eg, they treated 50 high probability of dying admissions and 50 decedents). Admissions to these hospitals were normally distributed by hospital EOL treatment intensity, so that the majority of admissions in PA were to hospitals of average treatment intensity. The study sample included 1,021,909 patients aged 65 years and older who incurred 2,216,815 admissions at these 169 hospitals (Fig. 1).

We modeled hospital EOL treatment intensity as a continuous variable with several functional forms. For simplicity, we depict results using figures and summarize findings for hospitals 1 standard deviation (SD) below and above average treatment intensity for illustrative purposes. Due to the large sample size, some admission-level differences are statistically, but not clinically, different (Table 1). Among notable large differences, admissions to higher intensity hospitals were more likely to be among black patients, to be emergent, and to have insurance suggestive of lower socioeconomic status (eg, Medicare without commercial Medigap, Medicare with Medicaid, or Medicaid only). The predicted probability of death among admissions to higher intensity hospitals was slightly lower. Hospitals 1 SD above average EOL treatment intensity had higher crude and standardized use of the ICU and life-sustaining treatments than hospitals 1 SD below average (Table 2). Higher intensity hospitals were larger, more likely to be teaching hospitals, and to have a higher annual volume of mechanical ventilation (Table 2).

Hospital Intensity and Postadmission Survival

Hospital Level Correlation—Crude 2-way correlations at the hospital level demonstrated an association between greater EOL treatment intensity and lower hospital 30-day SMR (-0.21 , $P = 0.005$), but not 180-day SMR ($P = 0.71$).

Admission-Level Logistic Regression—At the admission level, there was a nonlinear relationship between greater EOL treatment intensity and survival after adjustment for demographics and clinical characteristics, hospitalization history, and hospital characteristics (Fig. 2, Panels A–D). Patients who were at the highest PPD upon admission experienced the greatest benefit (Fig. 2, Panels B and D). In Table 3, we report relative and absolute comparisons in mortality if admissions with low, average, or high PPD had been admitted to a hospital 1 SD above or below average, instead of being admitted to an average EOL treatment intensity hospital.

Admission-Level Cox-Approximation—The pooled logistic regression approach approximating a Cox survival model revealed a similar relationship between intensity and overall survival, though more pronounced, as might be expected given the timeframe of comparison. Compared with admission to a hospital of average EOL treatment intensity, the 1-day mortality hazard for admission to a hospital -1 SD and $+1$ SD above average EOL treatment intensity was 1.80 (1.39–2.26) and 0.38 (0.30–0.48), respectively.

Sensitivity Analyses—The relationship between EOL treatment intensity and postadmission mortality did not qualitatively change in multiple sensitivity analyses (mentioned in Appendix: Sensitivity Analyses, online only, Supplemental Digital Content 1, Available at: <http://links.lww.com/MLR/A49>). The size of the relationship was larger with the decedent-based EOL intensity index. It was no longer statistically significant in a model restricted to the first hospitalization only.

Discussion

In this observational study of over 2 million admissions among more than 1 million patients to Pennsylvania acute care hospitals between 2001 and 2005, we found that hospitals with higher EOL treatment intensity—treatment typically assumed to be futile—actually have higher survival rates. The benefits were greatest for the patients at the highest probability of dying upon admission, but patients at low and average probability also benefited. Our findings were robust to 3 distinct statistical approaches, including new classes of models that strengthen causal inference in observational data. Importantly, the relationship between intensity and postadmission survival was nonlinear, with decreasing marginal returns to intensity above average intensity. Furthermore, the benefits to intensity attenuated with longer follow-up, indicating that the survival benefit is fleeting.

These findings highlight the critical balance between provision of life-saving and futile care, and demonstrate that different hospitals in the same state strike that balance differently. The implications for clinical or public policy of these findings are in the “eye of the beholder.” On the one hand, intensive care providers might interpret the findings to suggest that more is better, citing a 2% absolute increase in 30-day postadmission survival among the highest risk patients. On the other hand, palliative care providers might wonder whether high-intensity hospitals are doing harm by prolonging death, rather than prolonging life, citing relatively small and time-limited gains.

Our findings of a survival benefit to higher EOL treatment intensity are similar to Cher and Lenert's study of “potentially ineffective care” (defined as the concurrence of in-hospital death or death within 100 days of hospital discharge and total hospital costs above the 90th percentile). Specifically, they found a higher rate of “potentially ineffective care” among fee-for-service Medicare beneficiaries admitted to the ICU, compared with HMO beneficiaries, was associated with higher rates of survival to 100 days.²²

In contrast, our findings are different from Fisher et al who found no relationship between hospital referral region intensity (as measured by acute care and physician spending for patients in the last 6 months of life) and 1-year survival.^{4,5} There are several possible explanations for this difference. First, there is substantial within-region variation of intensity among individual hospitals which is lost in regional analyses. Second, our measure of intensity is fundamentally different. It is based on specific clinical activities designed to extend life rather than palliate: intensive care and life-sustaining treatments. These treatments may be more directly linked to survival than overall physician and acute care spending, which represents a heterogeneous group of services with more variable impact on survival. Third, their time horizon (1 year) was relatively long; indeed, the benefit to EOL treatment intensity that we observe waned significantly by 6 months.

Our measure is a theoretically less biased proxy for the treatment of “dying” patients⁷ because it is based upon the treatment of patients with a high probability of dying upon hospital admission rather than patients retrospectively identified as having died (decedents), the cohort used in the studies by Cher and Lenert and Fisher et al. A high-probability of dying-based definition is more closely aligned to real-world decision making under conditions of uncertainty. Furthermore, this approach ensured comparability (ie, risk adjustment) across hospitals, since the composition of decedent cohorts will be different between hospitals given that intensity indeed improves survival. Given the heterogeneous case mix of decedents (one-third at high probability of dying and two-thirds not),¹⁰ a decedent-based measure would actually overestimate the survival benefit of EOL intensity, because it would mix the treatment seen among patients with lower probability of dying (eg, those who might be saved with intensive care) with those at higher probability of dying (eg,

those who may be “harder” to save). Indeed, in sensitivity analyses, the impact of intensity on postadmission survival was larger when the hospital's intensity was measured based upon its treatment of decedents.

Our work is situated within the ongoing controversy regarding the effectiveness and cost-effectiveness of greater treatment intensity. The magnitude the relationship between intensity and survival are smaller than those found in studies by authors such as Cutler et al²³ and Doyle,²⁴ who argue that increases in the intensity of treatment, as measured by advancing technology and overall spending, provide cost-effective survival advantage. Our findings of a nonlinear relationship between intensity and survival suggest that reducing intensity among high intensity hospitals might be cost-saving, without substantial harm, whereas increasing intensity among low-intensity hospitals might save lives. Indeed, the heterogeneity of the relationship between intensity and survival found in our study suggests a hypothesis to explain competing findings in the literature. Perhaps regions with lower medical care intensity experience gains to greater intensity whereas regions with higher medical care intensity at baseline do not. Future research informing this controversy should explore the impact on both survival and quality of life of the more complex interplay of the intensity of specific services, the organizational model in which those services are delivered, and costs.

Our study is subject to many of the limitations frequently reported in analyses of hospital discharge data, such as variability of coding between hospitals, not to mention both random and systematic errors. PHC4 tries to anticipate random errors by limiting response options in each data field to exclude nonsensical options and by conducting hospital audits. Potential systematic sources of imprecision include nonrandom missingness of key clinical findings at lower intensity hospitals that might lead to an underestimate of illness severity (eg, troponin levels for acute myocardial infarction). Such differences, though, would be expected to bias our findings toward the null if lower intensity hospitals had sicker patients than advertised. Furthermore, although the inclusion of a validated risk prediction tool that uses prospective clinical information is one strength of our study, this variable was missing for 10% of admissions. The systematic exclusion of these admissions from our analyses may reduce the generalizability of our findings. Moreover, even among the admissions for which predicted probability of inpatient death was available, there were few with a high enough PPD to deem treatment futile.

We do not know whether the survival benefit we observed represents the direct effect of greater use of the specific services included in the index, or whether it is a marker for intensity across a much broader domain of clinical services. Importantly, the value of the observed survival benefit is unclear, since we have no information about quality of life, nor do we have sufficient information about postdischarge costs to calculate cost-effectiveness of greater hospital EOL treatment intensity. Finally, we have no information about the impact of hospital EOL treatment intensity on the quality of dying.

In conclusion, admission to higher EOL treatment intensity hospitals is associated with small gains in postadmission survival. The marginal returns to intensity diminish for admission to hospitals above average EOL treatment intensity and wane with time.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Sample selection. Four years of hospital discharge data included over 7 million admissions among more than 3 million individual patients. After restriction of the sample to in-state residents over age 65 and subjecting the data to cleaning, verification, and weighting, the analytic sample included over 2 million admissions among over 1 million patients.

Figure 2.

Panels A–D. Adjusted odds ratio 30- and 180-day postadmission mortality, by hospital treatment intensity among admissions with a high probability of dying. The figures represent the adjusted odds ratio for death at 30 days (A, B) and 180 days (C, D) postadmission, given treatment in a hospital with a particular end-of-life treatment intensity compared with if the patient had been admitted instead to a hospital with the average intensity. Adjustment covariables included patient demographics and clinical characteristics, hospitalization history, and hospital characteristics. Panels A and C depict a patient with an average predicted probability of death (PPD) upon admission (4.6% predicted probability of inpatient death; black line with 95% confidence interval in shaded gray), panels B and D depict patients at lowest (0% predicted probability of inpatient death; blue line with 95% confidence interval in shaded blue) and highest (41% predicted probability of inpatient death; red line with 95% confidence interval in shaded red) PPD upon admission (confidence interval overlap in shaded purple) (note: as the length of follow-up increases, the odds ratio cannot be interpreted as the risk ratio because the event rate exceeds 5%).

Table 1
Characteristics of Admissions, HPD Cohort, by Hospital Treatment Intensity

Characteristic	Study Sample	Admissions, by Hospital Treatment Intensity		Difference in means between +/-1 SD (p-value)
		~1SD below average [†]	average [‡] ~1SD above average [§]	
Admissions, N	2,216,815	278,707	708,920	
Age, mean (sd)	78.0 (7.6)	78.5 (7.7)	78.2 (7.6)	< .001
Male, %	57.7	58.8	57.7	< .001
Race, %				< .001
Caucasian	88.1	96.0	93.0	
Black	7.3	2.7	2.9	
Hispanic	1.3	0.1	0.6	
Asian	0.2	0.1	0.1	
Other or Unknown	3.1	1.2	3.4	
Admission Type, %				< .001
Emergency	59.3	51.0	61.6	
Urgent	22.1	31.2	20.0	
Elective	18.6	17.8	18.3	
Admission Source, %				< .001
Referral	33.1	36.6	34.2	
Transfer from SNF	1.2	0.5	1.9	
Emergency Room	62.5	61.8	61.2	
Other	3.3	1.2	2.7	
Insurance, %				< .001
Medicare only	30.6	26.7	27.9	
Medicare & Medicaid	8.8	9.2	7.6	
Medicare & Commercial	56.6	61.4	60.9	
Medicaid only	0.7	0.3	0.4	
Commercial only	3.2	2.4	3.2	
Uninsured	0.1	0.1	0.1	

Characteristic	Study Sample	Admissions, by Hospital Treatment Intensity			Difference in means between ± 1 SD (p-value)
		~ 1 SD below average [†]	average [‡]	~ 1 SD above average [§]	
Predicted probability of inpatient mortality, mean (sd)	.046 (.10)	.048 (.10)	.046 (.10)	.044 (.10)	< .001 *
Primary diagnosis, %					
Congestive heart failure	7.6	8.2	7.5	7.7	< .001
Coronary artery disease	5.5	4.1	5.2	6.2	< .001
Pneumonia	5.2	6.5	5.5	4.6	< .001
Cardiac dysrhythmia	4.7	4.3	4.5	5.0	< .001
Acute myocardial infarction	3.9	4.0	3.8	3.8	< .001
Chronic obstructive pulmonary disease	3.4	4.0	3.4	3.4	< .001
Cerebrovascular accident	3.2	3.2	3.1	3.1	.002
Osteoarthritis	3.2	3.1	3.6	3.0	.020
Crude 30-day mortality, %	8.0	8.8	8.1	7.5	< .001
Crude 180-day mortality, %	18.8	19.7	18.9	18.3	< .001

* statistical comparison based upon log-transformed value

[†] -1.33 SD to -0.67 SD

[‡] -0.33 SD to 0.33 SD

[§] 0.67 SD to 1.33 SD

Table 2
Characteristics of Hospitals, HPD Cohort, by Treatment Intensity

Characteristic	Study Sample	Hospital Treatment Intensity			Difference in means between +/-1 SD (p-value)
		~ 1SD below average [†]	average [‡]	~ 1SD above average [§]	
Hospitals, N	169	35	48	27	
Beds, mean (sd)	190 (130)	108 (61)	195 (112)	255 (112)	< .001
Resident-to-bed ratio, mean (sd)	.10 (.20)	.01 (.04)	.05 (.08)	.23 (.27)	< .001 *
Teaching Status, %					
Major Teaching	14.8	0	4.2	40.7	
Minor Teaching	29.6	14.3	37.5	37.0	
Non-teaching	55.6	85.7	58.3	22.2	< .001
Annual volume of mechanically ventilated patients, mean (sd)	670 (786)	205 (169)	626 (522)	1142 (855)	< .001
Crude intensity-of-treatment measures, proportion (sd)					
ICU Admission Rate	.57 (.17)	.44 (.15)	.58 (.11)	.71 (.11)	< .001
ICU Length of Stay	5.7 (2.1)	4.2 (1.4)	5.2 (0.9)	7.8 (1.5)	< .001
Intubation and mech. vent. rate	.22 (.10)	.13 (.04)	.22 (.06)	.30 (.08)	< .001
Hemodialysis rate	.04 (.03)	.02 (.02)	.04 (.02)	.06 (.02)	< .001
Tracheostomy rate	.02 (.02)	.004 (.004)	.02 (.01)	.03 (.02)	< .001
Gastrostomy rate	.04 (.02)	.02 (.01)	.04 (.01)	.05 (.02)	< .001
Smoothed, standardized (observed/expected) intensity-of-treatment ratios					
ICU Admission Rate	.99 (.27)	.82 (.27)	1.02 (.20)	1.18 (.13)	< .001
ICU Length of Stay	1.33 (.43)	1.04 (.26)	1.26 (.26)	1.75 (.30)	< .001
Intubation and mech. vent. rate	.93 (.30)	.64 (.15)	.95 (.20)	1.18 (.17)	< .001
Hemodialysis rate	.83 (.56)	.46 (.50)	.93 (.49)	1.25 (.40)	< .001
Tracheostomy rate	.77 (.65)	.24 (.21)	.70 (.39)	1.20 (.36)	< .001
Gastrostomy rate	.90 (.38)	.63 (.29)	.93 (.22)	1.03 (.26)	< .001

* statistical comparison made on log-transformed variable

[†] -1.33 SD to -0.67 SD

[‡] -0.33 SD to 0.33 SD

[§] 0.67 SD to 1.33 SD

Table 3
The Relationship between “End-of-Life” Treatment Intensity and Mortality: Adjusted
Odd Ratios and Absolute Risk Differences

Mortality Measure and Population	Hospital EOL Treatment Intensity		
	1SD below average	average	1SD above average
Adjusted * Odds Ratio (OR); (95% CI)			
30-day post-admission mortality			
Patients at low † probability of dying	1.06 (1.04 – 1.08)	1.0 (reference)	0.97 (0.96 – 0.99)
Patients at average ‡ probability of dying	1.06 (1.04 – 1.09)	1.0 (reference)	0.97 (0.96 – 0.99)
Patients at high ¶ probability of dying	1.09 (1.07 – 1.11)	1.0 (reference)	0.97 (0.95 – 0.99)
180-day post-admission mortality			
Patients at low † probability of dying	1.03 (1.01 – 1.04)	1.0 (reference)	1.00 (0.98 – 1.01)
Patients at average ‡ probability of dying	1.03 (1.02 – 1.05)	1.0 (reference)	1.00 (0.98 – 1.01)
Patients at high ¶ probability of dying	1.06 (1.04 – 1.09)	1.0 (reference)	1.00 (0.98 – 1.02)
Adjusted * absolute risk difference, %; (95% CI)			
30-day post-admission mortality			
Patients at low † probability of dying	1.4 (0.9 – 1.9)	0 (reference)	-0.7 (-1.1 to -0.3)
Patients at average ‡ probability of dying	1.6 (1.1 – 2.1)	0 (reference)	-0.7 (-1.1 to -0.3)
Patients at high ¶ probability of dying	2.1 (1.6 – 2.7)	0 (reference)	-0.8 (-1.2 to -0.4)
180-day post-admission mortality			
Patients at low † probability of dying	0.6 (0.2 – 1.0)	0 (reference)	-0.1 (-0.4 to 0.2)
Patients at average ‡ probability of dying	0.8 (0.4 – 1.2)	0 (reference)	-0.1 (-0.004 to 0.2)
Patients at high ¶ probability of dying	1.5 (0.9 – 2.0)	0 (reference)	-0.1 (-0.5 to 0.4)

* Models directly adjusted for patient demographic and clinical characteristics and treating hospital characteristics; Regressions weighted by inverse-probability-of-treatment weights derived from models using patient demographic and clinical characteristics from the current and immediate past prior hospitalization plus the EOL treatment intensity index of the prior hospitalization and specific intensive treatments received during the prior hospitalization.

† 0%,

‡ 4.6%, and

¶ 41% predicted probability of inpatient death