CHAPTER FOURTEEN: VALIDATION STUDY OF ACUTE MYOCARDIAL INFARCTION, RESULTS

This chapter summarizes the key findings of the AMI validation study. The seven research questions are listed below, followed by a detailed description of all relevant findings. Where appropriate, the specific measures and methods used to answer the question are described.

QUESTION 1: What proportion of cases included in the 1993 AMI study should have been excluded because acute myocardial infarction was incorrectly reported or incorrectly diagnosed?[1]

Of the 1,005 records received, a total of 31 required exclusion. Eighteen records with a principal diagnosis of AMI on the original discharge abstracts were excluded because that diagnosis was never documented by a physician. Hospital coders apparently misinterpreted these records and mistakenly assigned an unsubstantiated diagnosis. The corrected principal diagnoses for these 18 cases are shown in Table 14.1. Four additional records were excluded when they were found to be post-transfer hospitalizations. The reasons for transfer were: infarct extension, coronary artery bypass grafting (CABG), recurrent angina requiring intravenous nitroglycerin, and routine post-infarct care complicated by pneumonia.

Finally, nine excluded records were derived from a sample of 22 cases with secondary, not principal, diagnoses of AMI. These cases qualified for the 1993 hospital outcomes study because their principal diagnoses (e.g., ventricular tachycardia, ventricular fibrillation, complete atrioventricular block) were presumed to represent AMI complications. In four of these nine cases, the secondary diagnosis of AMI was never documented by a physician, although the principal diagnoses were correct. One additional case with a principal diagnosis of cardiogenic shock contained a brief reference to "probable myocardial infarction" but no supportive documentation; it was also excluded. The last four cases represented postoperative AMIs that occurred after revascularization for arterial thrombosis (444.xx), which was inappropriately included in the 1993 list of acceptable principal diagnoses.

[1] Note that this validation study did not address the number of AMI cases that should have been included but were missed because of underreporting by hospitals.

All of the remaining 974 records carried a physician diagnosis of AMI and were therefore fully abstracted. However, many of these records failed to meet more rigorous criteria for the diagnosis of AMI. The international diagnostic criteria used in the World Health Organization's Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA) project[2] and adapted by the Corpus Christi Heart Project were reviewed, along with the comparable ARIC criteria[3] used in the Cardiovascular Health Study. These criteria were adapted to the AMI validation data in the following manner:

1. Chest pain was defined as a sensation of "pain,...tightening, pressure, discomfort, angina,...heaviness, crushing, squeezing, burning..." located in the chest or epigastric area with or without radiation to the arms, jaw, throat, or neck. The pain had to have occurred within 24 hours of presentation.

2. Positive enzymes were defined as a creatine kinase isoenzyme (CK-MB) at least twice the upper limit of normal or "positive" (if the exact value was not reported), or a CK-MB greater than or equal to 10% of the total CK. CK-MBs reported to be "weakly positive" were coded as "positive."

3. Borderline enzymes were defined as a total CK at least twice the upper limit of normal, a total lactate dehydrogenase (LDH) at least twice the upper limit of normal (if no CK was obtained), a CK-MB between 5% and 9% of the total CK, or a CK-MB between once and twice the upper limit of normal.

4. Normal enzymes were defined as a CK, CK-MB, and LDH that did not meet any of the criteria specified in (2) and (3).

5. Patients with positive enzymes and chest pain were automatically classified as "definite" AMIs. Patients with positive enzymes in the absence of chest pain, or borderline enzymes with chest pain, were classified as possible AMIs.

6. For patients with no enzymes, normal enzymes, or borderline enzymes without chest pain, the first and last electrocardiogram (ECG) within 24 hours after presentation were reviewed:

    a. If the ECGs showed an "evolving diagnostic" pattern (using specific Minnesota codes), then the case was classified as a "definite" AMI.

    b. If the ECGs showed a "diagnostic" pattern, an "evolving ST-T" pattern, or an equivocal pattern and the enzymes were borderline (in the absence of chest pain), then the case was classified as a "possible" AMI.

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[2] Gillum RF, Fortmann SP, Prineas RJ, et al. International diagnostic criteria for acute myocardial infarction and acute stroke. *Am Heart J* 1984; 108:150-158.

[3] Cohort Component Procedures, ARIC Protocol 2, version 2.0, 1988.

c. If the ECGs showed a "diagnostic" pattern or an "evolving ST-T" pattern, the enzymes were incomplete, and chest pain was present, then the case was classified as a "possible" AMI.

d. All other combinations of findings were classified as "no AMI."

Using this algorithm, the 974 abstracted cases were classified as shown in Table 14.2. The 74 doubtful cases were distributed across 22 hospitals, with 0 to 9 cases at each hospital. There was no difference in the proportion of physician-diagnosed AMIs that failed to meet clinical criteria across hospital mortality classes. However, medium-volume hospitals tended to have a higher percentage of these doubtful AMIs than high-volume hospitals (9.3% versus 5.9%, p=0.062). Doubtful AMIs had higher mortality than definite and possible AMIs (41.9% versus 23.1%, p<0.001), although only 9.5% of these 74 patients presented to the hospital in cardiac arrest.

Combining the results of these analyses, 31 cases from the original sample of 1,005 (3.1%) were definitely false positives using the inclusion and exclusion criteria from OSHPD's 1993 study of AMI mortality, and an additional 74 cases (7.4%) were suspected to be false positives using modified ARIC criteria. These suspected false positives were discharged with a diagnosis of AMI by a licensed physician, but lacked the necessary combination of chest pain, cardiac enzyme, and ECG findings. Reweighting these figures to the statewide population, OSHPD estimates that 2.2% of the cases included in its 1993 AMI mortality study are definitely false positives and an additional 7.2% are suspected false positives.

To explore whether modifications of the selection rules might further reduce the false positive rate, a special analysis was performed of the 22 cases with a reported principal diagnosis other than 410.xx (AMI). These cases were included in the California Hospital Outcomes Project because missequencing of diagnoses was suspected. In other words, AMI was thought to be the underlying reason for admission when a patient had a secondary diagnosis of AMI and a principal diagnosis of a known AMI complication, such as cardiac arrest or ventricular tachycardia (see Chapter Three of the 1993 report for a complete list).[4]

Of the 22 cases with a secondary diagnosis of AMI, 9 were excluded for the reasons shown in Table 14.1 and 13 (57%) were found to have had a qualifying AMI. The diagnoses for the latter set of patients were resequenced with AMI as the principal diagnosis. Among these 13 cases, 5 had a principal diagnosis of paroxysmal ventricular tachycardia (427.1), 3 had cardiac arrest (427.5), 2 had acute edema of the lung, unspecified (518.4), 1 had arterial embolism or thrombosis (444.xx), 1 had hypotension, unspecified (458.9), and 2 had other (785.59) or unspecified (785.50) shock. Sorted by principal diagnosis, the proportion of cases upheld as AMIs ranged from 1 of 5 with arterial embolism or thrombosis and 0 of 2 with complete atrioventricular block to 3 of 3 with cardiac arrest and 5 of 6 with ventricular tachycardia. In the current report, arterial embolism and thrombosis was removed from the list of acceptable principal diagnoses in

[4] Hsia DC. Accuracy of Medicare reimbursement for cardiac arrest. *JAMA* 1990; 264:59-62.

Table 3.1. This change would be expected to reduce the statewide (weighted) frequency of definite false positives to 2.1%.

QUESTION 2: What is the statewide reporting accuracy for important risk factors included in the risk-adjustment models?

Table 14.3 summarizes the accuracy of reporting for ICD-9-CM coded risk factors derived from original hospital discharge abstracts, based on a comparison with the same risk factors derived from CMRI's reabstraction of matched records in the validation data set. It includes all of the clinical risk factors that were in AMI Model B for cases with no prior admissions. At the bottom of the table, overall measures of coding accuracy for diabetes and hypertension are reported (these risk factors were categorized in multiple levels for risk modelling, which makes dichotomous measures of coding accuracy hard to interpret). Except as indicated, all numbers in this table are weighted to adjust for the oversampling of outlier hospitals and deaths.

Sensitivity and predictive value are measures of **validity** that presume the existence of a "gold standard." CMRI's reabstracted diagnoses are taken to represent the truth; the diagnoses reported to OSHPD are evaluated against this gold standard. Sensitivity equals the percentage of patients with a risk factor, according to CMRI's reabstracted ICD-9-CM codes, who were reported to have that risk factor on the abstract originally submitted to OSHPD. A sensitivity of 30% means that the hospital coded only 30% of the cases with that risk factor, when compared to the CMRI gold standard. Positive predictive value (PV+) equals the percentage of patients reported to have a risk factor on the original abstract who were confirmed by CMRI's reabstraction. A PV+ of 30% means that only 30% of the cases reported to have the risk factor should have been reported, when compared to the CMRI gold standard. The ideal value for both sensitivity and PV+ is 100%. Low sensitivity represents undercoding and low PV+ represents overcoding.

Table 14.3 also reports the specificity, negative predictive value, and likelihood ratios for each risk factor. These statistics are less useful than the sensitivity and PV+, but are offered for the sake of completeness. Specificity is the percentage of patients without a risk factor, according to CMRI's reabstract, who were accurately identified from the original abstract. Negative predictive value (PV-) is the percentage of patients reported not to have a risk factor on the original abstract who were confirmed through CMRI's reabstraction as not having the risk factor. The ideal value for both specificity and PV- is 100%. The likelihood ratio (LR+) equals the sensitivity divided by 1-specificity; this measure incorporates both sensitivity (coding accuracy among patients with the risk factor) and specificity (coding accuracy among patients without the risk factor) into a single number. Higher values are better, while a value of 1.0 represents "random" coding.

Finally, the kappa statistic is a measure of **reliability**. It does not presume the existence of a "gold standard." Instead, it is designed to assess agreement between two independent data sources when neither is clearly superior to the other. Kappa is equal to the proportion of records for which both data sources agree on the presence or

absence of a risk factor, corrected for the level of agreement expected by chance.[5] Values fall between 0 and 1, where 1.0 is ideal. If a risk factor has a prevalence of 1% in two data sources, these data sources would be expected to agree in (0.99*0.99)+(0.01* 0.01)=98.02% of cases. Hence, 98% agreement is no better than one would expect by chance. Because the kappa statistic removes this "chance" effect, it is quite conservative. Note that the kappa statistic does not indicate whether there is undercoding or overcoding, because it presumes that both data sources are equally valid.

The validity and reliability of coding range from excellent to poor, depending on the risk factor. These values are excellent (sensitivity>80% and κ>0.8) for infarct site and diabetes, although about 60% of patients reported to have "other or unspecified" site actually have documentation suggesting a specific site. The quality of coding is very good (sensitivity>60% and κ>0.6) for congestive heart failure (CHF), chronic renal disease, prior coronary bypass surgery, history of pacemaker, complete atrioventricular block, and shock. Several other risk factors, including epilepsy, other cerebrovascular disease, primary and secondary malignancy, and hypertension, have intermediate sensitivities with kappa statistics between 0.45 and 0.60. Six risk factors (chronic liver disease, late effects of cerebrovascular disease, hypotension, pulmonary edema, nutritional deficiency, and other valve disease) are poorly coded, with sensitivities under 40% and kappa statistics less than 0.45. The least reliably coded risk factor, hypotension, has a likelihood ratio of 7.4. This means that patients with hypotension are 7.4 times more likely to have that diagnosis reported than patients without hypotension.

QUESTION 3: Are important risk factors coded more thoroughly at hospitals with low risk-adjusted mortality than at hospitals with high risk-adjusted mortality? If so, does the variation in risk-adjusted mortality diminish when inter-hospital differences in risk factor coding are removed?

Table 14.4 shows the sensitivities and kappa statistics for all risk factors present in at least 5% of cases (n=49) according to CMRI's reabstracts, stratified by hospital mortality and hospital volume. The "n" next to each risk factor name represents the number of cases with that risk factor, according to CMRI's reabstracts. Diabetes and hypertension include both complicated and uncomplicated cases; these results are similar to those based on separate subcategories. Probability values are based on Fisher's 2-tailed exact test. A relatively high p value cutoff (p<0.10) is recommended because of the small sample size and exploratory nature of the study.

This analysis shows no consistent differences in risk factor coding across hospital mortality and volume categories. Hospitals with high risk-adjusted mortality code anterior wall site with greater sensitivity (p=0.030) than hospitals with low or intermediate risk-adjusted mortality, but hospitals with intermediate mortality code chronic renal

[5] Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics* 1977; 33:159-174.

disease with the highest sensitivity (p=0.044) and reliability (p=0.043). Across hospital volume categories, high-volume hospitals code other valve disease more reliably (p=0.031), and hypertension (p=0.045) and shock (p=0.056) less reliably, than medium-volume hospitals. Only for CHF is a significant tradeoff between sensitivity (undercoding) and positive predictive value (overcoding) seen: high-volume hospitals code CHF with lower sensitivity (p=0.039) but higher predictive value (p=0.015) than medium-volume hospitals.

Overall, 65.0% of the original discharge abstracts have at least one missing clinical risk factor and 30.9% have at least two missing risk factors. There are no differences across hospital mortality categories in the percentage of original discharge abstracts with missing clinical risk factors, but this occurrence is more frequent at high-volume hospitals than at medium-volume hospitals (68.8% versus 61.2%, p=0.015). At the hospital level, the percentage varies from 45% to 87%.

Conversely, 31.5% of the original discharge abstracts have at least one unsupported clinical risk factor based on CMRI's reabstraction. Coding of unsupported clinical risk factors is more frequent at low-mortality hospitals than at intermediate or high-mortality hospitals (36.7% versus 29.2% and 29.0%, p=0.039), but is unrelated to hospital volume. At the hospital level, the percentage varies from 10% at a high-mortality hospital to 74% at a low-mortality hospital.

The aggregate impact of undercoding and overcoding risk factors was evaluated by recalculating risk-adjusted hospital mortality rates, using only risk factors that were identified through CMRI's reabstraction of the records in the validation data set. Eight models (e.g., four versions of Model A and four versions of Model B) were used in this analysis:

1a,1b The 1993 risk-adjustment models for AMI mortality (among cases with no prior admissions) were applied to the validation sample, thereby estimating each patient's risk of death using the ICD-9-CM codes reported to OSHPD and the coefficients listed in the 1993 report of the California Hospital Outcomes Project. For patients with no prior admissions, these estimates equal the predicted probabilities reported to hospitals in 1993.

2a,2b The same risk-adjustment models were applied to the same cases, but the ICD-9-CM codes reported to OSHPD were replaced by those reabstracted by CMRI. This procedure generated the predicted probabilities that would have been estimated **if an individual hospital had improved its coding practices to match CMRI's standard** (assuming that the hospital had too few cases to significantly affect the coefficient estimates in the risk-adjustment model). Although CMRI's reabstracts may not represent a true gold standard, they are at least coded uniformly across hospital categories.

3a,3b The 1993 risk-adjustment models for AMI mortality (among cases with no prior admissions) were reestimated on the validation sample, using the ICD-9-CM codes reported to OSHPD. Sampling variation explains the differences between

these models and the comparable models reported in the 1993 report of the California Hospital Outcomes Project.

4a,4b The same risk-adjustment models were reestimated on the same cases, but the ICD-9-CM codes reported to OSHPD were replaced by those reabstracted by CMRI. This procedure generated the predicted probabilities that would have been estimated **if all hospitals had coded their records as CMRI did**. By comparing these models with models 3a and 3b, one can evaluate whether reabstracted diagnoses yield a more powerful, less biased model than diagnoses reported to OSHPD.

In summary, models 1a, 1b, 2a, and 2b used the same regression coefficients that were originally estimated in 1993 using statewide data. These coefficients are quite reliable because they are based on nearly 29,000 AMIs, but they are biased by measurement error because hospitals do not report risk factors in a uniform manner. Models 3a and 4a were estimated on the 974 cases in the validation sample; models 3b and 4b were estimated on the 938 cases with nonmissing values of all risk factors. The regression coefficients in these models are unreliable because of the small sample size, but they are also less biased because risk factors are presumably measured more accurately. All four models were weighted to compensate for the oversampling of both deaths and cases from extreme-outcome hospitals.

Table 14.5 shows the results of all eight models, comparing risk-adjusted mortality across hospital outcome and volume strata. In this analysis, each stratum should be regarded as a single facility whose patients were drawn randomly from all hospitals in that stratum. The ISR represents the indirectly standardized mortality ratio comparing that subset of hospitals to the statewide experience (e.g., the number of observed deaths divided by the number of expected deaths). An ISR greater than one indicates higher than expected mortality, whereas an ISR less than one indicates lower than expected mortality. Asterisks denote the ISRs that significantly differ from one, at a 95% confidence level ($p<0.05$).

Using OSHPD data and 1993 OSHPD Model B coefficients (model 1b), "better" hospitals had 30% fewer deaths than expected (1-0.7007) while "worse" hospitals had 47% more deaths than expected (1.4701-1). Both of these ISRs differ significantly from one, as would be expected because hospitals were sampled based on their Model B risk-adjusted outcome classification from OSHPD's 1993 report. Using reabstracted data (model 2b), the same set of "better" hospitals had 37% fewer deaths than expected and the same set of "worse" hospitals had 18% more deaths than expected. The confidence limits for "better" hospitals still do not include one, but "worse" hospitals no longer differ significantly from expected. Another way to interpret these numbers is that the difference in risk-adjusted mortality between "better" and "worse" hospitals decreases by 28% if CMRI data are used in place of OSHPD data ((0.7694-0.5519)/0.7694).

This decrease was **not** found when Model A was applied instead of Model B. Using OSHPD data and 1993 OSHPD Model A coefficients (model 1a), "better" hospitals had 26% fewer deaths than expected while "worse" hospitals had 45% more deaths than expected. Using reabstracted data (model 2a), the same set of "better" hospitals had

17% fewer deaths than expected but the same set of "worse" hospitals had 55% more deaths than expected. Hence, the difference in risk-adjusted mortality between "better" and "worse" hospitals is virtually unchanged when CMRI data are used in place of OSHPD data (0.7208 versus 0.7052).

The lower half of Table 14.5 shows the results of models reestimated on the validation sample. As described above, reestimation removes bias in the regression coefficients but also decreases their reliability. Because all four patients with chronic liver disease, as reported to OSHPD, died before discharge, the risk-adjustment model based on OSHPD risk factors initially could not be reestimated. When chronic liver disease was omitted, model fit significantly deteriorated (c=0.835 versus 0.840). This problem was corrected by recoding the value of chronic liver disease for one case, which was randomly selected from the three cases that were identified by CMRI, but not reported to OSHPD, as having this condition.

Using OSHPD data and reestimated Model B coefficients (model 3b), "better" hospitals had 39% fewer deaths than expected while "worse" hospitals had 19% more deaths than expected. Only the ISR at "better" hospitals differs significantly from one, which is consistent with a modest ((0.7694-0.5849)/0.7694 = 24%) decrease in the risk-adjusted mortality difference between "better" and "worse" hospitals, relative to that obtained using the 1993 regression model. This decrease is attributable to random error in the risk-adjustment models and a phenomenon known as "regression to the mean."[6] Using reabstracted data (model 4b), the same set of "better" hospitals had 37% fewer deaths than expected and the same set of "worse" hospitals had 8% more deaths than expected. Hence, the difference in risk-adjusted mortality between "better" and "worse" hospitals decreases by 24% if CMRI data are used in place of OSHPD data. Using reestimated Model A coefficients (models 3a and 4a), this difference decreases by only 12% when CMRI data are used in place of OSHPD data.

These results suggest that unreliable coding (represented by the difference between original OSHPD data and reabstracted CMRI data) explains 24% to 28% of the difference in risk-adjusted mortality between low-mortality and high-mortality outlier hospitals based on Model B, but only 0% to 12% of the difference based on Model A. In other words, Model B is somewhat compromised by coding bias but Model A is not. Even with Model B, however, at least 72% of the gap in risk-adjusted mortality persists when coding variation is eliminated.

Several other findings from these models are of interest. First, the predicted probabilities of death calculated from OSHPD data are highly correlated with those calculated from reabstracted data at the individual level, regardless whether the 1993 regression models are applied (Spearman r=0.93 with Model A, r=0.91 with Model B) or reestimated

[6] "Regression to the mean" describes the observation that when outlier cases (e.g., hospitals) are selected using one measurement (e.g., risk-adjustment model) with a specific statistical threshold (e.g., $p<0.05$), other measurements on the same cases tend to demonstrate less extreme results.

(Spearman r=0.86 with Model A, r=0.83 with Model B). The correlations are slightly weaker with Model B because undercoding has more impact on the predictions generated by that model. Figures 14.1 and 14.2 show how reabstracting ICD-9-CM codes affects the ISRs of individual hospitals, based on either Model A or Model B. This is shown for illustrative purposes only, as too few patients were sampled to generate statistically significant results at the hospital level. However, the effects were generally consistent among the hospitals in each stratum.

Second, the effect of using reabstracted data on model performance can be described (Table 14.6). The original Models A and B developed in 1993 had c statistics of 0.766 and 0.844, respectively. Reestimating both models on the validation sample using OSHPD risk factors generated similar c statistics of 0.782 and 0.841, respectively. Reestimating both models using reabstracted risk factors generated higher c statistics of 0.814 and 0.881, respectively. The risk-adjustment models estimated using reabstracted data have significantly greater discrimination than those estimated using original OSHPD data.[7] Unfortunately, models that explain more deviance at the patient level do not necessarily explain more variation at the hospital level. A log-log linear regression analysis of the relationship between observed and expected weighted deaths across the 30 participating hospitals revealed that using reabstracted data increases the explanatory power of Model B (from partial R^2=0.190 to 0.489) but paradoxically decreases the explanatory power of Model A (from partial R^2=0.425 to 0.327).

Finally, it is instructive to evaluate how specific regression coefficients change when reabstracted data are used instead of original data. If reabstracted data represent a gold standard, then the regression coefficients based on those data approximate the true values and any significant changes reflect coding bias. The first step in this analysis is to examine the reestimated models (3a and 3b) based on OSHPD risk factors, shown in Tables 14.10 and 14.11. The coefficients from these models are generally similar to those published in OSHPD's 1993 report. The differences reflect sampling variation; in other words, the 974 cases randomly sampled for the validation study differ somewhat from the 30,958 cases in the original sample. For example, CHF was a major risk factor in the 1993 models, but had no effect in the validation sample. Conversely, uncomplicated diabetes had no effect in the 1993 models, but was an important risk factor in the validation sample.

Comparing models 3a and 4a (available upon request from OSHPD), complicated diabetes, chronic renal disease, other infarct site, and CHF-age interactions were more important risk factors with greater coefficients when reabstracted data were used instead of original data. Conversely, chronic liver disease and other valve disease were less important risk factors. Comparing models 3b and 4b, the same differences were confirmed but others were identified. Epilepsy and pulmonary edema became less important risk factors using reabstracted data, presumably because CMRI identified patients with milder forms of these risk factors. Other cerebrovascular disease became irrelevant because of better coding of "late effects" of cerebrovascular accidents (CVA).

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[7] DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: A nonparametric approach. *Biometrics* 1988; 44:837-845.

Conversely, hypotension became a more important risk factor. There were striking changes in the coefficients for demographic factors, such as race and payment source, possibly due to better adjustment for patients' clinical risk factors.

QUESTION 4: How often do the clinical characteristics used as risk factors in Model B actually represent conditions that developed after admission?

Table 14.7 shows the timing of each ICD-9-CM coded risk factor, including the first recorded date (relative to the date of emergency room arrival or admission, whichever came first) and whether the condition was documented in an emergency room (ER) or admission note. The results of these two approaches are generally compatible, but may differ because: (1) many patients are admitted one day after their presentation to the ER; (2) an admission note may be written one or more days after the date of admission; and (3) conditions may develop on the day of presentation (or the following day) which would not be documented in ER and admission notes. The second approach is more compatible with the new reporting mandate, which requires California hospitals to report, beginning with discharges on 1/1/96, whether each condition was "present at admission."

All 974 valid cases are included; the total number of reported patients with a risk factor (in the second column) is based on CMRI's reabstracts. The risk factors fall into three groups:

1. **Conditions that are usually diagnosed after admission**
   Conditions in this group are documented in ER or admission notes in less than 50% of cases and are first diagnosed at least one day after presentation in more than 50% of cases. Examples include hypotension, other cerebrovascular disease, pulmonary edema, and other valve disease. Shock does not quite meet these criteria, with 46% of occurrences documented in ER or admission notes and 51% first diagnosed on the day of presentation. All of these risk factors except other valve disease are currently considered Model B variables. Other valve disease is an exception because it is often based on the findings of diagnostic tests, such as echocardiography or ventriculography, that are performed after admission. The underlying valve disease was almost certainly present at admission, even if the diagnosis was not established until several days later.

2. **Conditions that are usually present at admission but may be diagnosed later**
   Conditions in this group are documented in ER or admission notes in 50-80% of cases. Examples include congestive heart failure, chronic liver disease, complete atrioventricular block, epilepsy, secondary malignant neoplasm, nutritional deficiency, and skin ulcer. Complete atrioventricular block, epilepsy, and skin ulcer are currently considered Model B variables. Nutritional deficiency is so rare that it did not appear in the updated risk-adjustment models (Chapter Nine of this volume); secondary and primary malignant neoplasms were aggregated. The apparent timing of chronic liver disease presumably reflects delayed diagnosis of a preexisting problem.

3. **Conditions that usually represent preexisting risk factors**
   Conditions in this group are documented in ER or admission notes in at least 80% of cases. Examples include infarct site, chronic renal disease, diabetes, hypertension, late effects of CVA, prior CABG, primary malignant neoplasm, and history of pacemaker. Note that a significant proportion of these preexisting conditions are first noted on the day after presentation. All of these variables are currently used in Model A.

These findings confirm that the additional variables in Model B may be preexisting conditions. Using currently available OSHPD data, it is impossible to distinguish such cases from those that arose after admission. However, a subset of Model B variables that are especially likely to have been diagnosed after admission has been identified (e.g., group 1 above).

The impact of mislabeling conditions diagnosed after admission as risk factors was evaluated by recalculating expected and risk-adjusted hospital mortality rates, using only risk factors that were documented in the ER or admission notes. Four models were employed:

5a,5b The 1993 risk-adjustment models for AMI mortality (among cases with no prior admissions) were applied to the validation sample, keeping the coefficients fixed but using the ICD-9-CM codes reabstracted by CMRI. These models differ from models 2a and 2b in that only conditions documented in the ER or admission notes were used to code risk factors.

6a,6b The same risk-adjustment models for AMI mortality were reestimated on the validation sample, using the ICD-9-CM codes reabstracted by CMRI. These models differ from models 4a and 4b in that only conditions documented in the ER or admission notes were used to code risk factors.

Table 14.8 demonstrates the impact of mislabeling as risk factors those conditions diagnosed after admission, across hospital mortality and volume categories. Using the regression coefficients published in 1993 (models 5a and 5b), disregarding conditions diagnosed after admission has little effect on expected mortality rates from Model A and therefore little effect on the ISRs. It has a dramatic effect on expected mortality rates from Model B, which drop from 15.6% to 12.3% at low-mortality hospitals, from 14.6% to 11.2% at intermediate hospitals, and from 14.6% to 10.7% at high-mortality hospitals. As a result, disregarding conditions diagnosed after admission increases the difference in risk-adjusted mortality between low and high-mortality hospitals by 49% ((0.8232-0.5519)/0.5519).

After hospitals begin submitting data with "present at admission" indicators, effective with discharges on January 1, 1996, OSHPD will be able to estimate models that adjust only for preexisting conditions and disregard conditions diagnosed after admission. Models 6a and 6b in Tables 14.10 and 14.11 demonstrate the potential impact of this change. Disregarding conditions diagnosed after admission when coding Model A risk factors increases the importance of CHF (from an odds ratio (OR) of 0.85 to 1.42) and weakens

the interactions between CHF and infarct site, but has little effect on other associations. Disregarding these conditions when defining Model B risk factors has a similar effect on CHF, but also increases the importance of complete atrioventricular block (from OR=1.12 to 2.14) and decreases the importance of complicated diabetes (from OR=4.15 to 1.74), hypotension (from OR=1.64 to 1.00), pulmonary edema (from OR=1.59 to 1.13), and shock (from OR=22.6 to 10.0). These differences exemplify the significant bias in AMI Model B due to OSHPD's inability to distinguish conditions present at admission from those diagnosed later.

Adjusting only for pre-existing conditions compromises the discriminatory power of Model B (from c=0.879 to 0.815) more than that of Model A (from c=0.814 to 0.786). At the hospital level, adjusting only for pre-existing conditions similarly compromises the explanatory power of both Model A (from partial R^2=0.394 to 0.287) and Model B (from partial R^2=0.470 to 0.310) in log-log linear regressions. Using either approach, Model B remains more powerful than Model A even when conditions diagnosed after admission are disregarded.

Using these models with reestimated coefficients, the expected mortality rate at low-mortality hospitals increases (from 15.6% to 17.4% in model 6a; from 15.6% to 16.8% in Model 6b) whereas that at high-mortality hospitals decreases slightly (from 15.9% to 15.2% in Model 6a; from 16.1% to 15.4% in model 6b) when conditions diagnosed after admission are disregarded. As a result, removing the bias due to mislabeling of these conditions increases the difference in risk-adjusted mortality between low and high-mortality hospitals by 25% in Model A and by 20% in Model B. In other words, counting conditions diagnosed after admission as risk factors leads one to underestimate the true difference in risk-adjusted mortality, even when the regression coefficients are reestimated.

QUESTION 5: How do the risk-adjustment models change when additional clinical variables are used as risk factors?

As part of CMRI's review of records in the AMI validation study, many clinical data elements were abstracted. This process involved reviewing all components of the medical record, including emergency room notes, histories and physical examinations, laboratory results, radiology reports, echocardiography reports, and operative notes. Based on review of the clinical literature and discussions with the AMI Clinical Advisory Panel, the following variables (with the alternative specifications listed) were evaluated as potential risk factors for in-hospital death within 30 days after an AMI.

1. Historical findings:
    a. Prior AMI, number of prior AMIs, prior AMI within 6 months
    b. Peripheral vascular disease (PVD), PVD with prior revascularization
    c. Prior stroke, prior stroke within 12 months, prior transient ischemic attack
    d. Prior CABG, prior percutaneous transluminal coronary angioplasty (PTCA), prior CABG or PTCA
    e. Asthma
    f. Chronic obstructive pulmonary disease

g. Known or suspected aortic aneurysm
h. Cardiac arrest, cardiopulmonary resuscitation, defibrillation within 24 hours
i. Sudden death, cardiac arrest (ever)
j. Atrial fibrillation or flutter
k. Congestive heart failure (CHF)
l. Current smoker, ever smoker
m. Pericarditis
n. Cocaine use
o. Permanent pacemaker or automatic defibrillator in place
p. Duration of chest pain, absence of chest pain

2. Physical findings at presentation:
   a. Systolic heart murmur (any, grade III or louder)
   b. Pulmonary rales (any, more than halfway up)
   c. Heart rate
   d. Systolic blood pressure at presentation, diastolic blood pressure
   e. Respiratory rate
   f. Shock, cerebral hypoperfusion, peripheral cyanosis, Military Antishock (MAST) trousers or pressors to support blood pressure
   g. Bilateral peripheral or presacral edema
   h. S_3 or summation gallop

3. Laboratory values at presentation:
   a. First CK value, first CK value indexed to upper limit of normal
   b. Hematocrit, anemia
   c. Serum creatinine, blood urea nitrogen (BUN)
   d. Platelet count, thrombocytopenia

4. Radiographic findings at presentation:
   a. CHF, cardiomegaly, pulmonary edema, pulmonary vascular congestion
   b. Pleural effusion (unilateral, bilateral)
   c. Pulmonary infiltrate (unilateral, bilateral)

5. Electrocardiographic findings at presentation:
   a. Atrial fibrillation or flutter
   b. QRS widening (e.g., bundle branch block)
   c. Ventricular hypertrophy (left, right, biventricular)
   d. Left axis deviation, right axis deviation

6. Miscellaneous:
   a. Do not resuscitate (DNR) order, DNR on day of admission, DNR at admission
   b. Left ventricular ejection or shortening fraction
   c. Non-AMI by ARIC criteria

Bivariate chi square tables were used to determine the specification of each clinical risk factor that best discriminates between low-risk and high-risk patients. This specification was then tested in weighted and unweighted multivariate logistic regression models that included all Model B risk factors, based on original OSHPD data. Each potential clinical

risk factor was tested individually in these models. Risk factors that significantly ($p<0.10$) increased the discrimination (c statistic) of the weighted models or had a significant ($p<0.10$) Wald chi square statistic in the unweighted models were retained for further analysis.[8]

This procedure identified seven promising clinical risk factors, which were forced simultaneously into an expanded version of Model B (with risk factors coded from original OSHPD data). All of the other candidate risk factors were then tested one final time, using automatic variable selection procedures (backward elimination, forward selection, and stepwise selection with p to enter and exit of 0.10) on the unweighted validation sample. This final step was designed to ensure that no potentially useful risk factors were discarded. A total of nine clinical risk factors were identified. At the recommendation of the AMI Clinical Advisory Panel, these nine risk factors were divided into five "core" variables and four "secondary" variables. The secondary variables either had marginal statistical (e.g., $0.03<p<0.10$) or clinical significance (e.g., systolic murmur), or became insignificant when reabstracted ICD-9-CM codes were used instead of original OSHPD data (e.g., history of stroke).

The definitions of the core clinical variables are as follows:

1. Systolic blood pressure at presentation (in mm Hg). For statistical reasons, this variable was recoded to zero if a patient had a cardiac or respiratory arrest within 24 hours before presentation.

2. Heart rate at presentation (in beats per minute). For statistical reasons, this variable was recoded to zero if a patient had a cardiac or respiratory arrest within 24 hours before presentation.

3. Cardiac arrest within 24 hours before presentation. This variable was recoded to one if a patient had a heart rate or blood pressure equal to zero at presentation. All

[8] Both of these criteria offer unique advantages and disadvantages. Using unweighted models to evaluate the importance of clinical risk factors allows the researcher to apply variable selection rules based on traditional chi square and likelihood ratio statistics, which are particularly appropriate with maximum likelihood estimation techniques (e.g., logistic regression). However, the parameter estimates and odds ratios from unweighted models are biased because of the oversampling of patients who died and patients at selected hospitals. Using weighted models to evaluate clinical risk factors obviates this problem, but the p values, chi square statistics, and standard errors from such models are uninterpretable because they depend on the scale of the weighting factor (e.g., multiplying all weights by 10 changes the p values). A solution to this problem is to use changes in the receiver operating characteristic curve to identify risk factors that significantly improve discrimination.

discrepancies between this variable and related variables on pre-hospital cardiopulmonary resuscitation and defibrillation were reconciled.

4. Shock at presentation (based on the use of MAST trousers or pressors, or clinical evidence of both cerebral and peripheral hypoperfusion).

5. Do-not-resuscitate order on the day of presentation to the emergency room or the day of admission.

The definitions of the secondary clinical variables are as follows:

6. The ratio of the first CK value (within 24 hours of presentation) to the hospital's gender-specific upper limit of normal.

7. Pulmonary rales (regardless of extent) on the patient's first physical examination.

8. Systolic heart murmur, grade III or louder on the patient's first physical examination.

9. Any prior history of stroke.

This final set of core and secondary clinical risk factors was then added to multivariate risk-adjustment models in the following manner:

7a,7b Models 3a and 3b, which reestimated OSHPD's 1993 risk-adjustment models (for AMI cases with no prior admissions) on the validation sample using the ICD-9-CM codes reported to OSHPD, were augmented with the five core clinical risk factors.

8a,8b Models 7a and 7b were further augmented with the four secondary clinical risk factors.

9a,9b Models 4a and 4b, which reestimated OSHPD's 1993 risk-adjustment models on the validation sample using the ICD-9-CM codes reabstracted by CMRI, were augmented with the five core clinical risk factors.

10a,10b Models 9a and 9b were further augmented with the four secondary clinical risk factors.

11a,11b Models 6a and 6b, which reestimated OSHPD's 1993 risk-adjustment models on the validation sample using only risk factors that were documented in the ER or admission notes (according to CMRI's reabstracted ICD-9-CM codes), were augmented with the five core clinical risk factors.

12a,12b Models 11a and 11b were further augmented with the four secondary clinical risk factors.

Table 14.9 shows how adding clinical risk factors affects the performance characteristics of both Model A and Model B, depending whether ICD-9-CM coded risk factors were

based on original data or reabstracted data. The statistics given for models 3a, 3b, 4a, and 4b differ slightly from those listed in Table 14.6, because missing values for race and the first CK limited the sample size to 925 for all of the models shown in Table 14.9.

Adding clinical risk factors clearly improves model discrimination, regardless whether the "base" model uses originally reported ICD-9-CM codes, reabstracted ICD-9-CM codes, or reabstracted codes from the ER or admission records. Not surprisingly, the magnitude of this improvement is smaller for Model B (e.g., from c=0.879 to c=0.898 with reabstracted codes) than for Model A (e.g., from c=0.814 to 0.864 with reabstracted codes). The core clinical variables contribute much more than the secondary clinical variables, although the latter factors further improve the discrimination of most models. Although the magnitude of improvement from adding core clinical variables appears to be smaller when reabstracted ICD-9-CM codes are used in the "base" model instead of original codes, limiting the analysis to codes reabstracted from the ER or admission notes actually increases the magnitude of improvement.

At the hospital level, adding clinical risk factors also improves the explanatory power of risk-adjustment models. For example, core clinical risk factors improve the proportion of variance in observed weighted deaths attributable to the risk-adjustment model from 0.394 to 0.494 with Model A (using reabstracted codes) and from 0.470 to 0.539 with Model B (using reabstracted codes). Secondary clinical variables provide little or no incremental benefit. Similar improvements are noted when core clinical variables are added to models that adjust only for risk factors reabstracted from ER or admission notes.

Tables 14.10 and 14.11 show the parameter estimates and odds ratios for each of the additional clinical risk factors in Models A and B, respectively. Given the best possible base model (e.g., ICD-9-CM codes reabstracted from ER or admission notes), a do-not-resuscitate order increases the odds of death 8.3 (Model A) or 9.9 (Model B) times. A recent cardiopulmonary arrest increases the odds of death 14.5 (Model A) or 19.6 (Model B) times. The odds of death increase 1.16 (Model A) or 1.17 (Model B) times with each 10 beat per minute increase in the heart rate, and increase 1.14 times with each 10 mm Hg decrease in the systolic blood pressure. Shock at admission increases the odds of death 4.2 (Model A) or 3.3 (Model B) times. The odds ratios for the secondary clinical variables are generally smaller: 1.4 or 1.5 for rales; 1.5 or 2.5 for a loud systolic murmur; 1.1 or 1.2 for a history of stroke; and 1.1 for each multiple of the upper limit of normal in the first CK. All of these values are relatively stable across models, except that a history of stroke is significant only when original ICD-9-CM codes were used to define "late effects" of a CVA. Confidence intervals for these odds ratios can be calculated using standard errors available upon request from OSHPD.

Tables 14.10 and 14.11 also show how adding clinical risk factors affects the parameter estimates and odds ratios for the risk factors that were included in the 1993 models. Most of these values change relatively little, which indicates that the odds ratios are not confounded by clinical risk factors. The major exceptions are as follows:

1. The unfavorable effect of CHF, which was only seen using ICD-9-CM codes reabstracted from ER or admission notes, disappears after adjustment for core

clinical risk factors (e.g., vital signs, cardiopulmonary arrest, DNR). The latter variables represent the clinical manifestations of poor cardiac function.

2. The unfavorable effect of other infarct site markedly diminishes after adjustment for core clinical risk factors.

3. The protective effect of being black and the unfavorable effect of being uninsured, according to model B, essentially disappear after adjustment for core clinical risk factors. In other words, the apparent racial and socioeconomic effects are largely explained by clinical differences.

4. Several of the additional risk factors based on ICD-9-CM codes in Model B lose their unfavorable effect or even become protective after adjustment for core clinical risk factors. For example, hypotension becomes irrelevant when the model adjusts for the actual value of the systolic blood pressure.

QUESTION 6: Do hospitals with significantly higher or lower than expected mortality appear closer to average after adjusting for additional clinical variables? How do the risk-adjusted mortality rates and p values for individual hospitals change when additional clinical variables are used as risk factors?

Table 14.12 demonstrates the impact of adding clinical risk factors to the risk-adjustment models based on ICD-9-CM data, across hospital mortality and volume categories. In general, neither core clinical variables nor secondary clinical variables systematically change expected mortality rates for these groups of hospitals. Starting with a reestimated version of Model A, based on the ICD-9-CM codes reported to OSHPD, the addition of both core and secondary clinical risk factors reduces the difference in risk-adjusted mortality between low-mortality and high-mortality hospitals by 10% ((0.5273-0.4743)/0.5273). Starting with a similarly reestimated version of Model B, the addition of both sets of clinical risk factors reduces this difference by 20%. By contrast, the addition of clinical risk factors to a reestimated version of Model A based on reabstracted ICD-9-CM data has a minimal effect on the difference in risk-adjusted mortality between low-mortality and high-mortality hospitals. The addition of clinical risk factors to a similarly reestimated version of Model B reduces this difference by 21% if conditions diagnosed after admission are used in coding risk factors, and by 14% if they are not.

Figures 14.3 and 14.4 show how adding clinical risk factors affects the ISRs of individual hospitals, based on either Model A or Model B. These figures demonstrate the impact of adding clinical variables to the best models based on ICD-9-CM data (e.g., models 6a and 6b, which include only conditions present at admission). Although too few patients were sampled from each hospital to generate statistically significant results at the hospital level, none of the low-mortality or high-mortality outliers show dramatic changes in risk-adjusted mortality when clinical variables are added to the model.

QUESTION 7: Do hospitals with low risk-adjusted mortality demonstrate better processes of care than hospitals with high risk-adjusted mortality?

Through literature review and discussion with clinical advisors, certain medications and invasive procedures were identified as process measures that might be associated with lower mortality among AMI patients. It was hypothesized that low-mortality hospitals use aspirin, heparin, thrombolytics, and beta blockers more often than high-mortality hospitals, controlling for hospital volume. It was also hypothesized that low-mortality hospitals perform coronary angiography, revascularization procedures (i.e., PTCA and CABG), and pulmonary artery (Swan-Ganz) catheterization for hemodynamic monitoring more often than high-mortality hospitals. These differences should be magnified by evaluating the promptness of therapy - particularly revascularization within 24 hours, which has recently been shown to improve outcomes for certain AMI patients.[9] Finally, it was hypothesized that low-mortality hospitals have more efficient emergency rooms than high-mortality hospitals, so they should have shorter times from presentation to the first ECG, the first CK determination, and admission.

The results are summarized in Tables 14.13 and 14.14. The former table provides unweighted statistics and includes p values to assess performance differences across hospital categories. The estimates in the latter table are weighted to reflect the statewide population of AMIs, but do not allow assessment of statistical significance. The contraindications used in HCFA's Cooperative Cardiovascular Project[10] (CCP) were applied, although a few minor contraindications could not be matched. For example, CCP distinguishes hemorrhagic from non-hemorrhagic strokes and identifies patients with prolonged prothrombin times and heparin allergies. However, these discrepancies should not affect the general results. For aspirin and thrombolysis, a revised version of the CCP criteria was created by dropping marginal or relative contraindications, such as age greater than 80 years, stroke more than 6 months prior to admission, and hematocrit less than 30%. The footnotes to Table 14.13 list these contraindications in detail.

All statistically significant differences across hospital outcome or volume categories are indicated with asterisks. High volume hospitals prescribe aspirin to a higher percentage of AMI patients than medium-volume hospitals, but aspirin use does not differ across hospital mortality categories. However, low-mortality hospitals start aspirin within 6 hours of presentation more often than intermediate or high-mortality hospitals. Thrombolytic use is associated with neither hospital volume nor hospital mortality. This result is unaffected by whether the CCP list of contraindications or the revised list is used. Low-mortality and high-mortality hospitals also do not differ in the use of aspirin and heparin as early adjunctive therapy with thrombolytics. Low-mortality hospitals do, however, administer heparin to a higher percentage of AMI patients than high-mortality hospitals.

AMI patients admitted to low-volume hospitals are less likely to undergo PTCA, but are just as likely to undergo CABG, as patients admitted to high-volume hospitals. Patients admitted to high-mortality hospitals are somewhat less likely to undergo CABG, but

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[9] Grines CL, Browne KF, Marco J, Rothbaum D, Stone GW, O'Keefe J, et al. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. *N Engl J Med* 1993; 328:673-679.

[10] Ellerbeck EF, Jencks SF, Radford MJ, Kresowik TF, Craig AS, Gold JA, Krumholz HM, Vogel RA. Quality of care for Medicare patients with acute myocardial infarctions. *JAMA* 1995; 273:1509-1514.

almost as likely to undergo PTCA, compared with those admitted to low-mortality hospitals. Revascularization (CABG or PTCA) within 24 hours of presentation is about twice as frequent in low-mortality as in high-mortality hospitals. Coronary angiography and pulmonary artery (Swan-Ganz) catheterization are also performed more frequently at low-mortality than at high-mortality hospitals. In separate analyses (not shown), it was demonstrated that low-mortality hospitals experience better outcomes than high-mortality hospitals even among their patients who do not undergo revascularization. Therefore, the observed differences in risk-adjusted outcomes cannot be attributed solely to differential rates of revascularization.

Finally, there were no systematic differences in the measurable efficiency of emergency services between low-mortality and high-mortality hospitals. Medium-volume hospitals experienced less delay to the first ECG and the first CK determination than high-volume hospitals, although this analysis may be confounded by differences in the clinical presentation of AMIs.

Table 14.1: Cases excluded from the validation sample

Reason	*Number*
Principal diagnosis miscoded as AMI, no documentation of AMI	
Unstable angina, 411.1	7
Cardiac dysrhythmia, 427.x	3
Coronary occlusion without AMI, 411.81	3
Other and unspecified angina pectoris, 413.9	1
Congestive heart failure, 428.0	1
Malignant essential hypertension, 401.0	1
Hypertensive heart and renal disease, 404.93	1
Vascular myelopathies, 336.1	1
Principal diagnosis of AMI, post-transfer hospitalization	4
Secondary diagnosis miscoded as AMI, no documentation of AMI	4
Secondary diagnosis miscoded as AMI, questionable documentation of AMI	1
Secondary diagnosis of AMI, postoperative AMI	4
TOTAL	31

Table 14.2: AMIs cases classified according to clinical criteria

Classification	*Definite*	*Possible*	*No AMI*
Chest pain present, positive enzymes	662		
Chest pain present, borderline enzymes		93	
Chest pain present, normal enzymes			
Evolving diagnostic ECG pattern	4		
All other ECG patterns			35
Chest pain present, incomplete enzymes			
Evolving diagnostic ECG pattern	0		
Diagnostic or evolving ST-T ECG pattern		5	
Equivocal, other, absent, or uncodable ECGs			4
No chest pain, positive enzymes		115	
No chest pain, borderline enzymes			
Evolving diagnostic ECG pattern	2		
Diagnostic, evolving ST-T, or equivocal ECG pattern		18	
Other, absent, or uncodable ECGs			7
No chest pain, normal enzymes			
Evolving diagnostic ECG pattern	1		24
All other ECG patterns			
No chest pain, incomplete enzymes			
Evolving diagnostic ECG pattern	0		4
All other ECG patterns			
TOTALS	669 68.7%	231 23.7%	74 7.6%

Table 14.3: Sensitivity and specificity of risk factor reporting

Risk Factor	*Number of cases*[1]	*Sensitivity (%)*[2]	*Specificity (%)*[2]	*PV+ (%)*[2]	*PV- (%)*[2]	*LR+*[2]	*Kappa*
Anterior wall	328	84	94	87	93	15.2	0.80
CHF	397	72	95	90	84	14.1	0.65
Chronic liver	15	8	100	82	99	636.0	0.31
Chronic renal	63	72	99	78	99	78.0	0.61
Complete AV block	43	62	100	91	98	203.3	0.79
Diabetes, complicated	74	55	97	48	98	17.4	0.50
Diabetes, uncomplicated	176	66	97	82	92	19.0	0.63
Epilepsy	41	37	99	67	98	70.5	0.66
Hypertension	494	60	93	91	66	8.4	0.51
Hypotension	218	25	97	68	82	7.4	0.20
Inferior wall	286	83	94	88	92	14.8	0.81
Late effects of CVA	45	20	99	49	97	26.0	0.44
Other cerebrovascular disease	61	45	99	81	96	53.8	0.56
Prior CABG	101	72	98	85	97	44.6	0.73
Pulmonary edema	82	31	98	60	95	18.5	0.43
Secondary malignant neoplasm	8	82	100	79	100	418.1	0.46
Shock	111	64	99	82	97	55.6	0.71
Other site of infarction	51	84	95	38	99	15.7	0.61
Other valve disease	178	21	99	83	85	23.2	0.31
Primary malignant neoplasm	6	28	100	87	100	1225.9	0.60
Nutritional deficiency	5	9	100	22	99	41.6	0.40
History of pacemaker	30	73	100	95	100	1022.0	0.77
Skin ulcer	20	48	100	76	99	230.6	0.60
Diabetes (any)	250	82	98	94	94	50.7	0.88
Hypertension (any)	554	64	95	95	65	12.3	0.54

[1] This column indicates the number of cases with the risk factor, according to CMRI's reabstraction of the records in the AMI validation data set.

[2] The figures in these columns are weighted to compensate for the oversampling of outlier hospitals and deaths; the weighted estimates approximate the true value of these parameters among all AMI patients admitted to California hospitals (except low-volume hospitals).

Table 14.4: Sensitivity and predictive value of risk factor reporting, by hospital volume and outcome category

Risk factor (cases)[1]	*Hospital mortality category*				*Hospital volume category*		
Measure	*Better*	*Neither*	*Worse*	*p value*	*High*	*Medium*	*p value*
Anterior wall (n=328)							
Sensitivity, %	84	82	93	0.030*	85	88	0.627
Adjusted sensitivity[2]	80	83	92		83	86	
PV+, %[3]	85	88	86	0.776	85	87	0.634
Kappa	0.76	0.79	0.84	0.218	0.78	0.81	0.449
CHF (n=397)							
Sensitivity, %	69	74	65	0.329	64	74	0.039*
Adjusted sensitivity	66	70	66		66	71	
PV+, %	83	89	90	0.291	93	83	0.015*
Kappa	0.60	0.69	0.64	0.388	0.64	0.65	0.738
Chronic renal (n=63)							
Sensitivity, %	40	80	52	0.044*	59	50	0.614
Adjusted sensitivity	36	75	47		54	41	
PV+, %	67	80	80	0.749	81	71	0.503
Kappa	0.47	0.79	0.61	0.043*	0.66	0.56	0.360
Hypotension (n=218)							
Sensitivity, %	17	27	14	0.125	22	17	0.396
Adjusted sensitivity	11	18	12		15	14	
PV+, %	48	65	50	0.425	65	45	0.110
Kappa	0.16	0.29	0.14	0.160	0.23	0.15	0.211
Inferior wall (n=286)							
Sensitivity, %	84	83	86	0.898	86	82	0.417
Adjusted sensitivity	85	83	88		88	84	
PV+, %	87	88	92	0.577	90	88	0.564
Kappa	0.80	0.79	0.85	0.413	0.84	0.79	0.221
Other cerebrovascular (n=61)							
Sensitivity, %	47	50	44	0.947	47	48	1.000
Adjusted sensitivity	42	42	46		42	49	
PV+, %	69	81	70	0.721	83	63	0.264
Kappa	0.54	0.59	0.52	0.871	0.58	0.52	0.661
Prior CABG (n=101)							
Sensitivity, %	64	74	75	0.602	64	77	0.193
Adjusted sensitivity	62	69	76		63	74	
PV+, %	81	83	81	1.000	79	84	0.590
Kappa	0.68	0.76	0.76	0.619	0.68	0.78	0.156
Pulmonary edema (n=82)							
Sensitivity, %	54	31	45	0.226	38	49	0.374
Adjusted sensitivity	36	36	45		36	41	
PV+, %	44	67	56	0.345	59	47	0.461
Kappa	0.43	0.39	0.47	0.833	0.42	0.44	0.859

Table 14.4: Sensitivity and predictive value of risk factor reporting, by hospital volume and outcome category, continued

Risk factor (cases)[1]	*Hospital mortality category*				*Hospital volume category*		
Measure	*Better*	*Neither*	*Worse*	*p value*	*High*	*Medium*	*p value*
Shock (n=111)							
Sensitivity, %	67	67	56	0.589	57	71	0.166
Adjusted sensitivity	48	56	54		48	61	
PV+, %	97	83	79	0.068*	81	93	0.173
Kappa	0.76	0.71	0.63	0.425	0.63	0.78	0.056*
Other site (n=51)							
Sensitivity, %	83	88	73	0.548	81	80	1.000
Adjusted sensitivity	89	85	76		81	81	
PV+, %	50	44	67	0.258	61	43	0.173
Kappa	0.61	0.56	0.67	0.609	0.67	0.54	0.198
Other valve dis (n=178)							
Sensitivity, %	27	19	23	0.599	30	18	0.076*
Adjusted sensitivity	20	22	18		26	15	
PV+, %	82	83	74	0.830	86	71	0.194
Kappa	0.34	0.27	0.29	0.759	0.39	0.22	0.031*
Diabetes (any, n=250)							
Sensitivity, %	92	82	88	0.203	87	88	0.852
Adjusted sensitivity	91	82	87		86	88	
PV+, %	95	95	97	0.780	97	95	0.537
Kappa	0.91	0.84	0.90	0.281	0.89	0.88	0.928
Hypertension (any, n=554)							
Sensitivity, %	64	63	60	0.781	57	68	0.011*
Adjusted sensitivity	64	61	59		59	67	
PV+, %	94	94	95	0.960	97	92	0.114
Kappa	0.53	0.54	0.55	0.957	0.50	0.59	0.045*

* Statistically significant at p<0.10

[1] Only risk factors present in at least 5% of cases (n=49) are shown in this table.

[2] Adjusted sensitivities reflect Choi's correction for sample selection bias. There is no procedure for evaluating the statistical significance of differences in the adjusted sensitivity across hospital categories, because the variance of the adjusted sensitivity is unknown. The results of the adjusted and unadjusted sensitivity analyses are generally similar.

[3] Oversampling of deaths does not bias the positive predictive value, so no adjustment is necessary.

Table 14.5: Weighted indirectly standardized mortality ratios by hospital mortality and volume category, using risk-adjustment models based on ICD-9-CM coded data[1]

	Hospital mortality category				*Hospital volume category*		
Risk-adjustment model	*Better*	*Neither*	*Worse*	*Difference*	*High*	*Medium*	*Difference*
Model 1a (OSHPD data, 1993 coefficients)	0.7441	1.2052	1.4494*	0.7052	1.1114	1.2412	0.1299
Model 2a (CMRI data, 1993 coefficients)	0.8269	1.4556*	1.5476*	0.7208	1.3547*	1.4159*	0.0612
Model 1b (OSHPD data, 1993 coefficients)	0.7007*	1.2015	1.4701*	0.7694	1.0690	1.2692	0.2002
Model 2b (CMRI data, 1993 coefficients)	0.6270*	1.0334	1.1789	0.5519	0.9627	1.0293	0.0666
Model 3a (OSHPD data, reestimated coefficients)	0.6775*	1.0273	1.2012	0.5237	0.9700	1.0390	0.0690
Model 4a (CMRI data, reestimated coefficients)	0.6151*	1.0534	1.0736	0.4585	1.0090	0.9893	0.0197
Model 3b (OSHPD data, reestimated coefficients)	0.6084*	1.0467	1.1933	0.5849	0.9307	1.0920	0.1613
Model 4b (CMRI data, reestimated coefficients)	0.6282*	1.0523	1.0751	0.4469	1.0210	0.9772	0.0438

* This indirectly standardized mortality ratio is statistically significantly different from one, which represents the average statewide mortality experience under this model.

[1] Risk-adjustment models 1a, 2a, 3a, and 4a are based on Model A, whereas models 1b, 2b, 3b, and 4b are based on Model B. Model B differs from Model A in that it includes race, expected principal source of payment, source and type of admission, and clinical factors that may represent either risk factors or complications. Models 1a, 2a, 3a, and 4a include 974 cases; models 1b, 2b, 3b, and 4b include 938 cases.

Table 14.6: Performance characteristics of risk-adjustment models based on ICD-9-CM coded data

Risk-adjustment model	*c statistic*[1]		*Calibration coefficients*[2]			*Partial R^2 (Type II) for hospital-level mortality*[3]
	Estimate	*95% confidence interval*	*Intercept*	*Linear slope*	*Quadratic slope*	
Model 1a (OSHPD data, 1993 OSHPD coefficients)	0.766[4] 0.775[5]	0.742 - 0.808	0.248	1.022	-0.003	0.388
Model 2a (CMRI data, 1993 OSHPD coefficients)	0.799[5]	0.768 - 0.830	1.099	1.437	0.029	0.395
Model 1b (OSHPD data, 1993 OSHPD coefficients)	0.844[4] 0.836[5]	0.806 - 0.866	0.180	0.904	-0.025	0.133
Model 2b (CMRI data, 1993 OSHPD coefficients)	0.869[5]	0.842 - 0.896	0.055	0.987	-0.023	0.435
Model 3a (OSHPD data, reestimated coefficients)	0.782	0.749 - 0.815	0.045	1.149	0.050	0.425
Model 4a (CMRI data, reestimated coefficients)	0.814	0.783 - 0.845	0.000	1.036	0.014	0.327
Model 3b (OSHPD data, reestimated coefficients)	0.841	0.810 - 0.871	0.000	0.997	-0.001	0.190
Model 4b (CMRI data, reestimated coefficients)	0.881	0.855 - 0.908	0.009	0.985	-0.007	0.489

[1] The c statistic is a measure of discrimination, or a model's ability to distinguish individuals who had a poor outcome from those who had a good outcome. It represents the proportion of all randomly selected pairs of observations with different outcomes in which the patient who died had a higher expected probability of death than the survivor. This statistic is equivalent to the area under a receiver operating characteristic curve, which plots sensitivity versus 1-specificity at various cutoff values for the predicted probability (see Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* 1982; 143:29-36). The c statistic takes on values between 0 and 1; higher values indicate greater discrimination but there is no cutoff that identifies inadequate models. A value of 0.5 can be obtained by random selection.

[2] Calibration coefficients assess the agreement between predicted probabilities generated by a logistic model and observed outcomes. A weighted logistic model is to used to regress the logit of observed mortality against the logit and logit squared of predicted mortality across all covariate patterns (see Miller ME, Hui SL, Tierney WM. Validation techniques for logistic regression models. *Stat Med* 1991; 10:1213-1226). The ideal values of the intercept and the quadratic slope are zero; the ideal value of the linear slope is one (see Miller ME, Langefeld CD, Tierney WM, Hui SL, McDonald CJ. Validation of probabilistic predictions. *Med Decis Making* 1993; 13:49-58). Because deaths were oversampled in the validation study, weighted analyses are essential and the statistical significance of the

coefficients cannot be determined. Instead, the reader should use the statistics reported in these columns to compare the calibration of hierarchical models. The same problem precludes use of the Hosmer-Lemeshow goodness-of-fit test (see Hosmer DW, Lemeshow S. *Applied Logistic Regression*. New York: John Wiley & Sons, 1989).

[3] The partial R^2 represents the squared Type II partial correlation between the observed and expected numbers of weighted deaths at each hospital, controlling for the total number of weighted cases. All variables are logarithmically transformed to reduce heteroscedasticity. The method of Kronmal (Kronmal RA. Spurious correlation and the fallacy of the ratio standard revisited. *J Royal Stat Assoc* 1993; 156(3):379-392) was used to avoid spurious correlations that may appear when ratios are regressed.

[4] These c statistics were derived from the original AMI sample included in the 1993 report of the California Hospital Outcomes Project.

[5] These c statistics were derived by applying OSHPD's 1993 models to the validation sample. They differ from the c statistics derived from the original statewide sample only because of sampling variation.

Table 14.7: Timing of risk factors

Risk Factor	Total	Date of Documention: ER/admit[1] date, no. (%)	Date of Documention: Next day, no. (%)	Date of Documention: >1 day after ER/admit, no. (%)	Source of Documention: ER/admit note, no. (%)
Anterior wall	328	295 (89.9)	31 (9.5)	2 (0.6)	324 (98.8)
CHF	397	267 (67.3)	58 (14.6)	72 (18.1)	271 (68.3)
Chronic liver	15	9 (60.0)	3 (20.0)	3 (20.0)	8 (53.3)
Chronic renal	63	53 (84.1)	6 (9.5)	4 (6.3)	59 (93.7)
Complete AV block	43	28 (65.1)	4 (9.3)	11 (25.6)	24 (55.8)
Diabetes, complicated	74	60 (81.1)	11 (14.9)	3 (4.1)	69 (93.2)
Diabetes, uncomplicated	176	145 (82.4)	24 (13.6)	7 (4.0)	168 (95.5)
Epilepsy	41	27 (65.9)	4 (9.8)	10 (24.4)	28 (68.3)
Hypertension without CHF or renal failure	494	434 (87.9)	53 (10.7)	7 (1.4)	473 (95.8)
Hypotension	218	103 (47.2)	34 (15.6)	81 (37.2)	98 (45.0)
Inferior wall	286	265 (92.7)	17 (5.9)	4 (1.4)	279 (97.6)
Late effects of CVA	45	37 (82.2)	1 (2.2)	7 (15.6)	37 (82.2)
Other cerebrovascular dz	61	27 (44.3)	10 (16.4)	24 (39.3)	28 (45.9)
Prior CABG	101	93 (92.1)	8 (7.9)	0 (0.0)	100 (99.0)
Pulmonary edema	82	31 (37.8)	17 (20.7)	34 (41.5)	31 (37.8)
Secondary malignant neoplasm	8	5 (62.5)	1 (12.5)	2 (25.0)	6 (75.0)
Shock	111	57 (51.4)	21 (18.9)	33 (29.7)	51 (46.0)
Other site of infarction	51	37 (72.5)	10 (19.6)	4 (7.8)	44 (86.3)
Other valve disease	178	48 (27.0)	45 (25.3)	85 (47.8)	33 (18.5)
Primary malignant neoplasm	6	5 (83.3)	0 (0.0)	1 (16.7)	5 (83.3)
Nutritional deficiency	5	3 (60.0)	0 (0.0)	2 (40.0)	3 (60.0)
History of pacemaker	30	22 (73.3)	8 (26.7)	0 (0.0)	30 (100.0)
Skin ulcer	20	10 (50.0)	4 (20.0)	6 (30.0)	11 (55.0)
Hypertension (any)	554	485 (89.0)	60 (11.0)	0 (0.0)	530 (95.7)

[1] The day of presentation is the day of arrival in the emergency room or the day of admission, if the patient was not admitted through the emergency room.

Table 14.8: Weighted indirectly standardized mortality ratios by hospital mortality and volume category, using risk-adjustment models that include only risk factors reabstracted from ER or admission notes[1]

	Hospital mortality category				*Hospital volume category*		
Risk-adjustment model	*Better*	*Neither*	*Worse*	*Difference*	*High*	*Medium*	*Difference*
Model 2a (CMRI data, 1993 coefficients)	0.8269	1.4556*	1.5476*	0.7208	1.3547*	1.4159*	0.0612
Model 5a (CMRI data from ER/admit notes, 1993 coefficients)	0.8174	1.4992*	1.5957*	0.7783	1.5069*	1.3481*	0.1588
Model 4a (CMRI data, reestimated coefficients)	0.6151*	1.0534	1.0736	0.4585	1.0090	0.9893	0.0197
Model 6a (CMRI data from ER/admit notes, reestimated coefficients)	0.5506*	1.0683	1.1236	0.5731	1.0629	0.9535	0.1093
Model 2b (CMRI data, 1993 coefficients)	0.6270*	1.0334	1.1789	0.5519	0.9627	1.0293	0.0666
Model 5b (CMRI data from ER/admit notes, 1993 coefficients)	0.7967	1.3407*	1.6199*	0.8232	1.2665	1.3181	0.0516
Model 4b (CMRI data, reestimated coefficients)	0.6282*	1.0523	1.0751	0.4469	1.0210	0.9772	0.0438
Model 6b (CMRI data from ER/admit notes, reestimated coefficients)	0.5820*	1.0617	1.1174	0.5355	0.9991	1.0010	0.0019

* This indirectly standardized mortality ratio is statistically significantly different from one, which represents the average statewide mortality experience under this model.

[1] Risk-adjustment models 2a, 4a, 5a, and 6a are based on Model A, whereas models 2b, 4b, 5b, and 6b are based on Model B. Model B differs from Model A in that it includes race, expected principal source of payment, source and type of admission, and clinical factors that may represent either risk factors or complications. Models 2a, 4a, 5a, and 6a include 974 cases; models 2b, 4b, 5b, and 6b include 938 cases because of missing values.

Table 14.9: Performance characteristics of risk-adjustment models with and without additional clinical variables[1]

Risk-adjustment model	*c statistic*[1]		*Calibration coefficients*[2]			*Partial R^2 (Type II) for hospital-level mortality*[3]
	Estimate	*95% confidence interval*	*Intercept*	*Linear slope*	*Quadratic slope*	
Model 3a (OSHPD data, no clinical variables)	0.782	0.749 - 0.815	0.066	1.182	0.059	0.476
Model 7a (OSHPD data, core clinical variables)	0.845	0.816 - 0.875	0.008	1.132	0.047	0.296
Model 8a (OSHPD data, core & secondary clinical variables)	0.854	0.825 - 0.883	-0.004	1.128	0.047	0.321
Model 4a (CMRI data, no clinical variables)	0.814	0.783 - 0.845	0.001	0.985	-0.006	0.394
Model 9a (CMRI data, core clinical variables)	0.860	0.831 - 0.889	-0.017	1.071	0.027	0.494
Model 10a (CMRI data, core & secondary clinical variables)	0.864	0.835 - 0.892	-0.026	1.099	0.038	0.488
Model 6a (CMRI data from ER/admit notes, no clinical variables)	0.786	0.753 - 0.820	0.018	1.087	0.031	0.287
Model 11a (CMRI data from ER/admit notes, core clinical variables)	0.843	0.814 - 0.873	-0.002	1.071	0.027	0.406
Model 12a (CMRI data from ER/admit notes, core & secondary clinical variables)	0.845	0.814 - 0.875	0.003	1.129	0.046	0.371
Model 3b (OSHPD data, no clinical variables)	0.837	0.806 - 0.868	-0.001	1.008	0.003	0.170
Model 7b (OSHPD data, core clinical variables)	0.870	0.842 - 0.897	-0.013	1.055	0.021	0.249
Model 8b (OSHPD data, core & secondary clinical variables)	0.877	0.851 - 0.904	-0.016	1.060	0.023	0.276

Table 14.9: Performance characteristics of risk-adjustment models with and without additional clinical variables[1], continued

Risk-adjustment model	*c statistic[1]*		*Calibration coefficients[2]*			*Partial R^2 (Type II) for hospital-level mortality[3]*
	Estimate	*95% confidence interval*	*Intercept*	*Linear slope*	*Quadratic slope*	
Model 4b (CMRI data, no clinical variables)	0.879	0.852 - 0.907	0.009	0.986	-0.007	0.470
Model 9b (CMRI data, core clinical variables)	0.898	0.872 - 0.923	0.028	0.966	-0.017	0.539
Model 10b (CMRI data, core & secondary clinical variables)	0.898	0.873 - 0.923	0.016	0.981	-0.009	0.556
Model 6b (CMRI data from ER/admit notes, no clinical variables)	0.815	0.782 - 0.848	-0.007	1.063	0.025	0.310
Model 11b (CMRI data from ER/admit notes, core clinical variables)	0.852	0.822 - 0.882	0.002	0.993	-0.003	0.359
Model 12b (CMRI data from ER/admit notes, core & secondary clinical variables)	0.859	0.829 - 0.888	-0.013	1.071	0.028	0.357

[1] The c statistic is a measure of discrimination, or a model's ability to distinguish individuals who had a poor outcome from those who had a good outcome (see Table 14.6 for additional description).

[2] Calibration coefficients assess the agreement between predicted probabilities generated by a logistic model and observed outcomes. A weighted logistic model is to used to regress the logit of observed mortality against the logit and logit squared of predicted mortality across all covariate patterns (see Table 14.6 for additional description). The ideal values of the intercept and the quadratic slope are zero; the ideal value of the linear slope is one. The reader should use the statistics reported in these columns to compare the calibration of hierarchical models; statistical significance cannot be assessed.

[3] The partial R^2 represents the squared Type II partial correlation between the observed and expected numbers of weighted deaths at each hospital, controlling for the total number of weighted cases (see Table 14.6 for additional description).

Table 14.10: Acute myocardial infarction Model A, no prior admission (reestimated parameter estimates and odds ratios using validation sample)

Risk Factor	*1993 model*		*Model 3a* *(OSHPD data, no additional clinical variables)*		*Model 8a* *(OSHPD data, all clinical variables)*		*Model 6a* *(CMRI data from ER/admit notes, no additional clinical variables)*		*Model 12a* *(CMRI data from ER/admit notes, all clinical variables)*	
	Value	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*
Intercept	-3.0808		-2.9244		-3.3565		-3.1710		-3.2614	
Female	0.1574	1.17	0.2881	1.33	0.0781	1.08	0.0702	1.07	-0.0387	0.96
Age 18-40[1]	-1.5206	0.22	-1.8021	0.16	-2.4325	0.09	-1.8201	0.16	-2.6201	0.07
Age 41-55[1]	-1.2456	0.29								
Age 56-65	-0.6150	0.54	-1.1684	0.31	-1.5598	0.21	-1.5194	0.22	-1.8675	0.15
Age 76-85	0.5045	1.66	0.2453	1.28	0.1886	1.21	0.3378	1.40	0.5009	1.65
Age >86	1.0780	2.94	0.5143	1.67	0.7381	2.09	0.5342	1.71	-0.0728	0.93
Anterior wall	1.4185	4.13	1.2577	3.52	1.2812	3.60	1.9736	7.20	2.1582	8.66
CHF	0.5670	1.76	-0.0064	0.99	-0.3307	0.72	0.4950	1.64	-0.0854	0.92
Chronic liver	1.1743	3.24	4.1570	63.88	3.2771	26.50	2.5741	13.12	2.5091	12.29
Chronic renal	0.3244	1.38	0.6425	1.90	0.8240	2.28	1.1145	3.05	0.9471	2.58
Diabetes, complicated	0.4658	1.59	0.6665	1.95	0.8769	2.40	0.7387	2.09	0.8004	2.23
Diabetes, uncomplicated	0.0383	1.04	0.9775	2.66	1.3025	3.68	0.9280	2.53	1.4391	4.22
Hypertension	-0.5779	0.56	-0.5462	0.58	-0.3505	0.70	-0.6605	0.52	-0.5966	0.55
Inferior wall	1.0944	2.99	1.1931	3.30	1.3340	3.80	1.4757	4.37	1.3893	4.01
Late effects of CVA	0.3648	1.44	1.9226	6.84	1.5169	4.56	2.1009	8.17	2.0370	7.67
Prior CABG	-0.0841	0.92	-0.4600	0.63	0.1975	1.22	-0.4521	0.64	-0.4619	0.63
Secondary malignant neoplasm	0.7533	2.12	-0.0749	0.93	-1.2044	0.30	-1.9750	0.14	-1.9364	0.14
Other site of infarction	2.2115	9.13	1.4880	4.43	0.7808	2.18	3.0216	20.52	2.3451	10.43
CHF & Age 41-55	0.7695	2.16	3.0330	20.76	2.9258	18.65	2.8052	16.53	3.8588	47.41
CHF & Age 56-65	0.4149	1.51	-0.1442	0.87	-0.1565	0.86	0.4868	1.63	0.2474	1.28
CHF & Age >86	-0.4371	0.65	0.3411	2.15	0.6922	2.00	0.1189	1.13	1.7001	5.47
CHF & Anterior wall	-0.2397	0.79	0.7660	2.40	1.0598	2.89	1.2780	3.59	-0.2617	0.77
Female & Age 56-65	0.2138	1.24	0.8742	0.05	-2.5547	0.08	-3.6915	0.02	1.4414	4.23
Inferior wall & Age 41-55	-0.7049	0.49	-3.0469	1.23	0.4024	1.50	0.3170	1.37	-3.9990	0.02

Table 14.10: Acute myocardial infarction Model A, no prior admission (reestimated parameter estimates and odds ratios using validation sample), continued

Risk Factor	*1993 model*		*Model 3a* *(OSHPD data, no additional clinical variables)*		*Model 8a* *(OSHPD data, all clinical variables)*		*Model 6a* *(CMRI data from ER/admit notes, no additional clinical variables)*		*Model 12a* *(CMRI datafrom ER/admit notes, all clinical variables)*	
	Value	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*
Inferior wall & Age 56-65	-0.4120	0.66	0.2101	6.63	2.2513	9.50	0.9041	2.47	0.3344	1.40
CHF & Other site	-0.8908	0.41	1.8914	0.30	-1.6541	0.19	-1.4412	0.24	1.7111	5.54
Other valve disease	-0.4078	0.67	-1.2127	0.52	-1.2360	0.29	-1.8137	0.16	-1.0746	0.34
Other site & Age 56-65	0.4199	1.52	-0.6611		2.2257	9.26			-0.6528	0.52
DNR					2.5887	13.31			2.1172	8.31
Cardiopulmonary arrest					-0.0130	0.99			2.6756	14.52
Systolic blood pressure					0.0177	1.02			-0.0130	0.99
Heart rate					1.5841	4.87			0.0145	1.01
Shock (clinical)					0.0926	1.10			1.4259	4.16
First CK index					0.4627	1.59			0.0883	1.09
Rales					0.3742	1.45			0.4079	1.50
Systolic heart murmur					1.0366	2.82			0.4195	1.52
History of stroke (clinical)									0.1531	1.17

[1] These two variables were combined into one risk factor (age 18-55 years) for all models estimated on the validation sample.

Table 14.11: Acute myocardial infarction Model B, no prior admission (reestimated parameter estimates and odds ratios using validation sample)

	1993 model		*Model 3b* *(OSHPD data no clinical variables)*		*Model 8b* *(OSHPD data all clinicalvariables)*		*Model 6b* *(CMRI data from ER/admit notes, no clinical variables)*		*Model 12b* *(CMRI data from ER/admit notes, all clinical variables)*	
Risk Factor	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*
Intercept	-3.4275	0.03	-3.1802		-3.4451		-3.1707		-2.8891	
Female	0.1470	1.16	0.3346	1.40	0.2880	1.33	0.0526	1.05	-0.0552	0.95
Age 18-40[1]	-1.4958	0.22	-1.7079	0.18	-2.7790	0.06	-2.1848	0.11	-3.4902	0.03
Age 41-55[1]	-1.0211	0.36								
Age 56-65	-0.4704	0.62	-1.1738	0.31	-1.5811	0.21	-1.6018	0.20	-2.1330	0.12
Age 76-85	0.6067	1.83	0.8677	2.38	0.8002	2.23	0.5233	1.69	0.7501	2.12
Age >86	1.1307	3.10	0.7573	2.13	0.6993	2.01	0.7892	2.20	0.1854	1.20
Race: black	-0.0171	0.98	0.5714	1.77	0.8779	2.41	-1.0165	0.36	-0.3440	0.71
Race: Hispanic	0.0854	1.09	0.0861	1.09	0.0021	1.00	-0.6266	0.53	-0.6097	0.54
Race: other nonwhite	-0.0476	0.95	0.4503	1.57	0.1320	1.14	0.2734	1.31	-0.2450	0.78
Type: urgent or elective	-0.3881	0.68	-0.2728	0.76	-0.1324	0.88	0.4774	1.61	0.4634	1.59
Source: ER	0.0200	1.02	-0.3948	0.67	-0.6076	0.54	0.4485	1.57	0.4645	1.59
MediCal	0.3522	1.42	-0.5776	0.56	-1.2217	0.29	-2.8854	0.06	-2.7192	0.07
Medicare	0.1782	1.20	-0.0781	0.92	-0.2970	0.74	-0.6461	0.52	-1.0865	0.34
Uninsured	0.2949	1.34	0.4998	1.65	0.1784	1.20	0.6066	1.83	0.0375	1.04
Anterior wall	1.2160	3.37	1.3351	3.80	1.5687	4.80	1.7928	6.01	2.0051	7.43
CHF	0.3335	1.40	-0.0624	0.94	-0.3407	0.71	0.5737	1.77	-0.1697	0.84
Chronic liver	1.1069	3.02	4.6002	99.50	3.7791	43.78	2.6778	14.55	2.7178	15.15
Chronic renal	0.3279	1.39	0.3180	1.37	0.6092	1.84	1.0024	2.72	0.7880	2.20
Complete AV block	0.5835	1.79	-0.1835	0.83	-0.4758	0.62	0.7729	2.17	0.3434	1.41
Diabetes, complicated	0.3906	1.48	1.0578	2.88	1.1397	3.13	0.5423	1.72	0.6607	1.94
Diabetes, uncomplicated	0.0557	1.06	1.3159	3.73	1.6567	5.24	1.1349	3.11	1.6320	5.11
Epilepsy	1.2591	3.52	0.8924	2.44	0.1912	1.21	0.0108	1.01	-0.6665	0.51
Hypertension	-0.4740	0.62	-0.5441	0.58	-0.4247	0.65	-0.6611	0.52	-0.5943	0.55
Hypotension	0.4911	1.63	0.3195	1.38	-0.2624	0.77	-0.1048	0.90	-0.9531	0.39
Inferior wall	0.8124	2.25	0.8778	2.41	1.0878	2.97	1.2951	3.65	1.2974	3.66
Late effects CVA	0.2137	1.24	2.1433	8.53	1.9352	6.93	2.2282	9.28	1.9812	7.25
Other cerebrovascular disease	0.7112	2.04	1.2398	3.45	1.1137	3.05	-0.3033	0.74	0.0242	1.02
Prior CABG	-0.0507	0.96	-0.6043	0.55	-0.0147	0.99	-0.6590	0.52	-0.6442	0.53
Pulmonary edema	0.9532	2.59	1.3112	3.71	0.9079	2.48	0.1734	1.19	-0.4246	0.65

	1993 model		Model 3b (OSHPD data no clinical variables)		Model 8b (OSHPD data all clinicalvariables)		Model 6b (CMRI data from ER/admit notes, no clinical variables)		Model 12b (CMRI data from ER/admit notes, all clinical variables)	
Risk Factor	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*

Table 14.11: Acute myocardial infarction Model B, no prior admission (reestimated parameter estimates and odds ratios using validation sample), continued

	1993 model		Model 3b (OSHPD data no clinical variables)		Model 8b (OSHPD data all clinical variables)		Model 6b (CMRI data from ER/admit notes, no clinical variables)		Model 12b (CMRI data from ER/admit notes, all clinical variables)	
Risk Factor	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*	*Value*	*OR*
Secondary malignant neoplasm	0.9146	2.50	-1.5436	0.21	-2.2980	0.10	-0.8713	0.42	-0.9963	0.37
Shock	2.5734	13.11	3.1970	24.46	3.3009	27.14	2.3212	10.19	1.6943	5.44
Other site of infarction	2.0517	7.78	0.7801	2.18	0.1218	1.13	3.1675	23.75	2.3290	10.27
CHF & Age 41-55	0.4567	1.58	2.8144	16.68	3.4310	30.91	3.3612	28.82	4.9900	146.9
CHF & Age 56-65	0.1514	1.16	-0.8766	0.42	-0.3630	0.70	0.2321	1.26	0.2350	1.26
CHF & Age >86	-0.2368	0.79	0.4723	1.60	-1.0276	0.36	0.5479	1.73	1.2819	3.60
CHF & Anterior wall	-0.2180	0.80	0.3501	1.42	0.1992	1.22	-0.1508	0.86	-0.2832	0.75
Female & Age 56-65	0.1235	1.13	0.8024	2.23	0.9829	2.67	1.1942	3.30	1.6747	5.34
Inferior wall & Age 41-55	-0.7117	0.49	-2.3718	0.09	-2.1078	0.12	-3.8124	0.02	-4.0970	0.02
Inferior wall & Age 56-65	-0.4268	0.65	0.6914	2.00	0.5918	1.81	-0.0743	0.93	-0.1354	0.87
CHF & Other site	-0.8179	0.44	2.6475	14.12	2.7769	16.07	0.7790	2.18	1.8660	6.46
Other valve disease	-0.3662	0.69	-1.7239	0.18	-1.7306	0.18	-1.1301	0.32	-1.0242	0.36
Other site & Age 56-65	0.4528	1.57	0.2914	1.34	-0.4806	0.62	-1.9354	0.14	-1.3261	0.27
DNR					2.3244	10.22			2.2970	9.94
Cardiopulmonary arrest					3.1404	23.11			2.9759	19.61
Systolic blood pressure					-0.0112	0.99			-0.0127	0.99
Heart rate					0.0166	1.02			0.0158	1.02
Shock (clinical)					1.4161	4.12			1.2084	3.35
First CK index					0.0939	1.10			0.0964	1.10
Rales					0.2952	1.34			0.3310	1.39
Systolic heart murmur					0.6628	1.94			0.9186	2.51
History of CVA (clinical)					1.0895	2.97			0.1144	1.12

[1] These two variables were combined into one risk factor (age 18-55 years) for all models estimated on the validation sample.

Table 14.12: Weighted indirectly standardized mortality ratios and confidence intervals, by hospital mortality and volume category, using risk-adjustment models with and without additional clinical risk factors[1]

	Hospital mortality category				*Hospital volume category*		
Risk-adjustment model	*Better*	*Neither*	*Worse*	*Difference*	*High*	*Medium*	*Difference*
Model 3a (OSHPD data, no additional clinical variables)	0.6757*	1.0292	1.2030*	0.5273	0.9631	1.0437	0.0805
Model 7a (OSHPD data, core clinical variables)	0.6832*	1.0347	1.1222	0.4391	0.9976	1.0026	0.0050
Model 8a (OSHPD data, core & secondary clinical variables)	0.6684*	1.0362	1.1426	0.4743	0.9956	1.0048	0.0092
Model 4a (CMRI data, no additional clinical variables)	0.6430*	1.0518	1.0519	0.4090	1.0192	0.9798	0.0395
Model 9a (CMRI data, core clinical variables)	0.6819*	1.0419	1.0544	0.3724	1.0684	0.9345	0.1339
Model 10a (CMRI data, core & secondary clinical variables)	0.6759*	1.0407	1.0791	0.4032	1.0689	0.9341	0.1348
Model 6a (CMRI data from ER/admit notes, no additional clinical variables)	0.5689*	1.0704	1.0871	0.5182	0.9633	1.0435	0.0802
Model 11a (CMRI data from ER/admit notes, core clinical variables)	0.6149*	1.0565	1.0799	0.4650	1.0097	0.9896	0.0200
Model 12a (CMRI data from ER/admit notes, core & secondary clinical variables)	0.5993*	1.0583	1.1074	0.5082	1.0099	0.9894	0.0205
Model 3b (OSHPD data, no additional clinical variables)	0.6137*	1.0447	1.2085	0.5955	0.9251	1.0971	0.1720
Model 7b (OSHPD data, core clinical variables)	0.6282*	1.0519	1.0880	0.4598	0.9414	1.0730	0.1316
Model 8b (OSHPD data, core & secondary clinical variables)	0.6287*	1.0499	1.1060	0.4773	0.9453	1.0675	0.1222
Model 4b (CMRI data, no additional clinical variables)	0.6386*	1.0487	1.0915	0.4529	1.0179	0.9811	0.0368
Model 9b (CMRI data, core clinical variables)	0.6720*	1.0457	1.0418	0.3698	1.0366	0.9628	0.0739
Model 10b (CMRI data, core & secondary clinical variables)	0.6823*	1.0436	1.0388	0.3565	1.0509	0.9497	0.1011
Model 6b (CMRI data from	0.5875*	1.0603	1.1246	0.5371	0.9927	1.0081	0.0153

Risk-adjustment model	Hospital mortality category				Hospital volume category		
	Better	*Neither*	*Worse*	*Difference*	*High*	*Medium*	*Difference*
ER/admit notes, no additional clinical variables)							
Model 11b (CMRI data from ER/admit notes, core clinical variables)	0.6115*	1.0611	1.0477	0.4362	1.0060	0.9935	0.0124
Model 12b (CMRI data from ER/admit notes, core & secondary clinical variables)	0.6086*	1.0591	1.0731	0.4645	1.0051	0.9944	0.0107

* This indirectly standardized mortality ratio is statistically significantly different from one, which represents the average statewide mortality experience under this model.

[1] Risk-adjustment models 3a, 7a, 8a, 4a, 9a, 10a, 6a, 11a, and 12a are based on Model A, whereas models 3b, 7b, 8b, 4b, 9b, 10b, 6b, 11b, and 12b are based on Model B. Model B differs from Model A in that it includes race, expected principal source of payment, source and type of admission, and clinical factors that may represent either risk factors or complications. To maximize comparability, all models include 925 cases without missing values.

Table 14.13: Unweighted process of care characteristics, by hospital mortality and volume category (including p values)

Process (eligible cases)[1]	*Hospital mortality category*				*Hospital volume category*		
Restriction	*Better*	*Neither*	*Worse*	*p value*[2]	*High*	*Medium*	*p value*[3]
Aspirin if CCP-eligible (n=809)[4]							
Any time, %	80	68	77	0.568	81	69	<0.001*
Within 6 hours, %	35	23	26	0.032*	31	24	0.034*
Mean hours to first dose	21.8	25.5	23.1	0.024*	25.4	21.1	0.655
Aspirin if eligible (n=850)[5]							
Any time, %	77	68	75	0.570	79	68	<0.001*
Within 6 hours, %	33	23	25	0.035*	30	24	0.037*
Mean hours to first dose	21.8	25.8	22.9	0.044*	25.6	20.9	0.613
Thrombolytic if CCP-eligible (n=302)[6]							
Any time, %	40	57	46	0.475	45	50	0.356
Within 2 hours of arrival, %	31	44	31	0.806	30	40	0.115
Mean hours to first dose	2.2	2.2	4.9	0.672	4.8	1.7	0.469
Thrombolytic if eligible (n=381)[7]							
Any time, %	34	44	42	0.216	39	42	0.532
Within 2 hours of arrival, %	26	35	28	0.770	27	33	0.178
Mean hours to first dose	2.7	2.1	4.7	0.506	4.5	1.9	0.393
IV heparin if thrombolysed (n=230)							
Within 6 hours, %	86	81	86	0.893	79	89	0.045*
Aspirin if thrombolysed (n=230)							
Within 6 hours, %	57	40	52	0.696	55	44	0.089*
Heparin if CCP-eligible (n=861)[8]							
Any time, %	77	59	67	0.016*	68	68	0.999
Within 24 hours, %	63	43	55	0.089*	50	57	0.056*
Mean hours to first dose	17.0	23.0	18.6	0.160	22.4	16.2	0.409
PTCA (n=974)							
Any time, %	16	14	13	0.298	17	12	0.014*
Within 24 hours, %	7	3	4	0.034*	7	2	<0.001*
PTCA if eligible (n=700)[9]							
Any time, %	18	15	17	0.681	20	13	0.011*
Within 24 hours, %	7	4	5	0.230	8	3	0.003*
CABG (N=974)							
Any time, %	14	12	9	0.100*	12	12	0.921
Within 24 hours, %	3	1	1	0.040*	2	1	0.116*
CABG if eligible (n=700)[9]							
Any time, %	15	14	11	0.289	14	13	0.825
Within 24 hours, %	3	1	1	0.252	3	1	0.083*
PTCA or CABG (n=974)							
Any time, %	27	25	22	0.094*	28	22	0.045*
Within 24 hours, %	9	4	4	0.021*	9	3	<0.001*

Table 14.13: Unweighted process of care characteristics, by hospital mortality and volume category (including p values), continued

Process (eligible cases)[1]	*Hospital mortality category*				*Hospital volume category*		
Restriction	*Better*	*Neither*	*Worse*	*p value*[2]	*High*	*Medium*	*p value*[3]
PTCA or CABG if eligible (n=700)[9]							
Any time, %	30	28	27	0.417	32	25	0.024*
Within 24 hours, %	8	5	6	0.266	10	3	<0.001*
Swan-Ganz catheterization, % (n=974)	22	18	13	0.004*	19	16	0.152
Coronary angiography, % (n=974)	38	34	25	<0.001*	40	24	<0.001*
Beta blocker if eligible, % (n=530)[10]	38	40	51	0.015*	48	39	0.066*
Mean hours to admission (n=887)	3.4	3.0	3.2	0.817	3.3	3.1	0.887
Mean hours to first ECG (n=841)	1.8	0.8	1.3	0.492	1.4	1.1	0.025*
Mean hours to first CK (n=879)	2.7	1.9	2.7	0.001*	2.7	2.2	0.003*

* Statistically significant at p<0.10.

1 All therapies are ascertained exclusively from the index or initial hospitalization, except that CABG and PTCA are ascertained from the index hospitalization or any subsequent transfer hospitalization.

2 For dichotomous factors, this p value represents a test of the hypothesis that there is a monotonic relationship between hospital process and risk-adjusted outcomes (i.e., Mantel-Haenzel chi square for trend, df=1). For continuous factors, this p value represents a test that the distributions differ across hospital categories (i.e., Kruskal-Wallis rank sum test).

3 For dichotomous factors, this p value represents a test of the hypothesis that there is an association between hospital process and volume (i.e., 2-tailed Fisher's exact test). For continuous factors, this p value represents a test that the distributions differ across hospital categories (i.e., Wilcoxon rank sum test).

4 Exclusion criteria for this analysis include death or transfer on the day of presentation (if the patient is obtunded or experienced a cardiac arrest before or at arrival), bleeding diathesis or coagulopathy, aspirin allergy, gastrointestinal or genitourinary bleeding within the prior six months, guaiac positive or bloody stool at admission, warfarin at admission, thrombocytopenia at admission (platelet count below 100,000), any history of intracranial neoplasm or neurosurgery, chronic liver disease, head trauma within the prior six weeks, serum creatinine greater than 3 mg/dl, hematocrit less than 30% or hemoglobin less than 10 g/dl, and a history of metastatic cancer.

5 Exclusion criteria for this analysis include all of those listed above except the threshold platelet count is lowered to 50,000 and the thresholds for serum creatinine and hematocrit are eliminated.

6 Exclusion criteria for this analysis include chest pain for less than 30 minutes or more than 6 hours at presentation, bleeding diathesis or coagulopathy, gastrointestinal or genitourinary bleeding within the prior six months, guaiac positive or bloody stool at admission, warfarin at admission, any history of intracranial neoplasm or neurosurgery, chronic liver disease, head trauma or major surgery within the prior six weeks, cardiopulmonary resuscitation within the prior 24 hours, known or suspected aortic aneurysm, any history of stroke, uncontrolled hypertension (systolic blood pressure greater than 200 mm Hg or diastolic blood pressure greater than 120 mm Hg at presentation), age greater than 80 years, or any other specified contraindication or refusal of therapy.

7 Exclusion criteria for this analysis include all of those listed above except less than 30 minutes of chest pain at presentation, stroke more than six months before admission, age greater than 80 years, and other specified contraindications or refusal of therapy.

8 Exclusion criteria for this analysis include bleeding diathesis or coagulopathy, guaiac positive or bloody stool at admission, warfarin at admission, thrombocytopenia at admission (platelet count below 100,000), any history of

intracranial neoplasm or neurosurgery, head trauma within the prior six weeks, and hematocrit less than 30% (or hemoglobin less than 10 g/dl).

[9] Exclusion criteria for this analysis include age greater than 80 years, a "do not resuscitate" order on the date of presentation or the date of admission, and death or transfer on the day of presentation (if the patient is obtunded or experienced a cardiac arrest before or at arrival).

[10] Exclusion criteria for this analysis include a history of asthma or chronic obstructive pulmonary disease, diabetes mellitus requiring insulin at admission, congestive heart failure or pulmonary edema by the first chest radiograph, systolic blood pressure less than 100 mm Hg at presentation, second or third degree atrioventricular block on the first or last ECG in the first 24 hours (unless a permanent pacemaker was in place or inserted during this hospitalization), shock at any time during the hospitalization, and poor left ventricular function (ejection fraction less than 25%, shortening fraction less than 15%, or severe/very severe dysfunction).

Table 14.14: Weighted process of care characteristics, by hospital mortality and volume category[1]

Process (eligible cases)[2]	*State wide*	*Hospital mortality category*			*Hospital volume category*	
Restriction		*Better*	*Neither*	*Worse*	*High*	*Medium*
Aspirin if CCP-eligible (n=809)[3]						
Any time, %	73	86	71	79	81	64
Within 6 hours, %	26	37	25	27	31	20
Mean hours to first dose	24.5	26.7	24.1	24.3	22.0	28.6
Aspirin if eligible (n=850)[3]						
Any time, %	73	83	71	77	80	64
Within 6 hours, %	26	35	25	26	30	20
Mean hours to first dose	24.8	26.5	24.6	24.1	22.9	27.9
Thrombolytic if CCP-eligible (n=302)[3]						
Any time, %	51	37	54	47	40	68
Within 2 hours of arrival, %	39	26	42	32	29	54
Mean hours to first dose	2.5	3.1	2.2	5.2	3.3	1.9
Thrombolytic if eligible (n=381)[3]						
Any time, %	41	32	42	44	32	56
Within 2 hours of arrival, %	32	23	33	30	24	44
Mean hours to first dose	2.5	3.2	2.2	5.0	3.1	1.9
IV heparin if thrombolysed (n=230)						
Within 6 hours, %	81	87	80	86	71	90
Aspirin if thrombolysed (n=230)						
Within 6 hours, %	44	56	42	53	54	36
Heparin if CCP-eligible (n=861)[3]						
Any time, %	63	79	60	70	61	65
Within 24 hours, %	46	60	43	58	43	51
Mean hours to first dose	22.5	19.6	23.5	18.6	23.9	20.9
PTCA (n=974)						
Any time, %	17	18	17	15	20	12
Within 24 hours, %	5	8	4	4	8	1
PTCA if eligible (n=700)[3]						
Any time, %	18	21	17	18	22	12
Within 24 hours, %	5	8	4	4	9	0
CABG (N=974)						
Any time, %	12	14	12	10	12	12
Within 24 hours, %	1	3	1	1	2	0
CABG if eligible (n=700)[3]						
Any time, %	13	15	13	12	14	13
Within 24 hours, %	1	2	1	1	2	0
PTCA or CABG (n=974)						
Any time, %	28	28	28	24	31	23
Within 24 hours, %	6	9	5	5	9	1

Table 14.14: Weighted process of care characteristics, by hospital mortality and volume category[1], continued

Process (eligible cases)[2]	*State wide*	*Hospital mortality category*			*Hospital volume category*	
Restriction		*Better*	*Neither*	*Worse*	*High*	*Medium*
PTCA or CABG if eligible (n=700)[3]						
Any time, %	29	32	29	29	35	22
Within 24 hours, %	6	9	6	6	11	1
Swan-Ganz catherterization, % (n=974)	37	45	37	28	41	32
Coronary angiography, % (n=974)	37	45	37	28	41	32
Beta blocker if eligible, % (n=530)[3]	43	41	42	53	44	42
Mean hours to admission (n=887)	3.0	3.6	2.9	3.2	3.3	2.7
Mean hours to first ECG (n=841)	0.9	1.6	0.7	1.4	0.8	0.9
Mean hours to first CK (n=879)	2.1	3.0	1.9	2.7	2.4	1.7

[1] Weighting compensates for the oversampling of outlier hospitals and deaths; the weighted estimates approximate the true value of these parameters among all AMI patients admitted to this subset of hospitals statewide.

[2] All therapies are ascertained exclusively from the index or initial hospitalization, except that CABG and PTCA are ascertained from the index hospitalization or any subsequent transfer hospitalization.

[3] These exclusion criteria are described in the notes to Table 14.13.