Understanding the Coronary Heart Disease Versus Total Cardiovascular Mortality Paradox

A Method to Enhance the Comparability of Cardiovascular Death Statistics in the United States

Christopher J.L. Murray, MD, DPhil; Sandeep C. Kulkarni, AB; Majid Ezzati, PhD

Background—Coronary heart disease (CHD) represents the largest share of cardiovascular disease in the United States, but there are conspicuous discrepancies between CHD and total cardiovascular death rates across the states, possibly due in part to variations in physician assignment of causes of death. Our aim was to identify exogenous individual- and community-level predictors of cause-of-death assignment and variability and to use these predictors to improve the comparability of CHD mortality estimates across states.

Methods and Results—We performed a multinomial logistic regression analysis to estimate the effect of individual- and community-level factors on the likelihood of a death being certified as 1 of 3 ill-defined clusters (general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest) relative to being certified as CHD. The individual-level variables were the decedent’s race, sex, age, education, and place of death; the community-level variable was the number of cardiologists per capita. We used the model to estimate state-level CHD rates that are standardized with regard to the levels of individual- and community-level determinants of cause-of-death assignment. Decedents who died in hospitals and in counties with more cardiologists per capita were more likely to be assigned to CHD than to the ill-defined categories, as were white males relative to other race-sex combinations. Adjustment for these factors resulted in substantially improved correlation between death rates for CHD and all cardiovascular causes. Increases in CHD death rates across states after adjustment for external predictors of cause-of-death assignment ranged from 2% (North Dakota) to 72% (Washington, DC); New York had a decrease (1%) in CHD death rates after adjustment. Nationally, CHD death rates increased 10% for males and 15% for females. The total number of deaths in 2001 attributed to CHD in patients over 30 years of age rose from 433,625 to 489,836 after adjustment.

Conclusions—Greater presence of medical knowledge at the time of death, reflected by place of death and cardiologists per capita, reduces the use of the ill-defined cardiovascular clusters. Racial and gender effects on CHD assignment may reflect disparities in access to care and quality of care. By adjusting for differentials in these parameters, a comparable and consistent set of CHD mortality estimates can be created. The role of the exogenous predictors in validity and comparability of cause-of-death statistics should be confirmed in carefully designed validation autopsy studies.

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Key Words: cardiovascular diseases ■ coronary disease ■ statistics ■ heart diseases ■ mortality

Valid and comparable data on mortality and causes of death are essential for the design and evaluation of health policies and intervention programs. The death certificate serves as a key source of information for both overall and cause-specific mortality, but its utility may be compromised by variability in assignment practices for underlying cause of death between regions and over time. Lack of comparability in cause-of-death assignment is particularly important for cardiovascular diseases, because they are the largest contributors to mortality in the United States. In the year 2001, nearly 38% of all deaths in the United States were due to cardiovascular diseases; of these, 47% were attributed to coronary heart disease (CHD).

Although CHD constitutes the largest share of cardiovascular disease nationally, there are conspicuous discrepancies between CHD and total cardiovascular death rates at the state level (Figure 1). In 2001, the state of New York had the highest death rate from CHD among all the states, but the 17th highest rate of all-cause cardiovascular mortality; Washington, DC ranked 5th highest in overall cardiovascular mortality.
mortality but 44th for CHD; Alabama had the 7th highest overall cardiovascular mortality but ranked 28th for CHD. Is the poor correlation between CHD and total cardiovascular death rates due to actual epidemiological differences across states in the age-specific mortality from non-CHD cardiovascular causes, or is part of this paradox due to regional differences in cause-of-death assignment practices?1

Cardiovascular deaths in the United States are distributed across 8 major clusters of causes: CHD, stroke, hypertensive heart disease, inflammatory heart disease, general atherosclerosis and unspecified heart disease, heart failure, cardiac arrest, and other cardiovascular causes (Table 1; Figure 2). Epidemiological and autopsy studies suggest that a considerable fraction of deaths from 3 of the non-CHD clusters, general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest, may be due to underlying CHD.2–6 Variability in the use of these 3 “ill-defined” cause clusters in lieu of CHD may confound the comparability of detailed

### TABLE 1. Definitions and Corresponding ICD Codes for Major Cardiovascular Causes-of-Death Clusters

<table>
<thead>
<tr>
<th>Cardiovascular Causes of Death</th>
<th>ICD Codes</th>
</tr>
</thead>
</table>
| CHD: ischemic heart disease; angina pectoris; acute and subsequent myocardial infarction | ICD-9: 410–414  
ICD-10: I20–I25, excluding I25.0 |
| Stroke: cerebral infarction; intracerebral hemorrhage; subarachnoid hemorrhage | ICD-9: 430–438  
ICD-10: I60–I69 |
| Hypertensive heart disease: essential hypertension, hypertensive heart disease, hypertensive renal disease | ICD-9: 401–405  
ICD-10: I10–I15 |
| Atherosclerosis and unspecified heart disease: general and unspecified atherosclerosis; unspecified cardiovascular disease; unspecified heart disease | ICD-9: 429.2, 429.9, and 440.9  
ICD-10: I25.0, I51.6, I51.9, I70.9 |
| Heart failure: congestive heart failure; left ventricular failure; unspecified heart failure | ICD-9: 428  
ICD-10: I50 |
| Cardiac arrest: cardiac arrest; ventricular tachycardia; ventricular fibrillation | ICD-9: 427.1, 427.4, 427.5  
ICD-10: I46, I47.2, I49.0 |
| Inflammatory heart disease: cardiomyopathy; acute and subacute endocarditis; myocarditis; pericarditis | ICD-9: 429.0, 429.1, 420–425  
ICD-10: I51.4, I51.5, I30–I33, I38–I43 |

The variability in assigning CHD-related deaths to ill-defined cardiovascular causes could arise from a number of factors: ambiguous wording of the disease classification system; the clinical information available at the time of death, especially for out-of-hospital deaths; the training and skills of the person completing the death certificate; and regional practices and preferences in assigning particular causes of death.\(^{7–9}\) A number of studies, in the United States and internationally, have examined the validity of death certificates for cardiovascular causes and have concluded that cardiovascular death certificates and clinical diagnoses may disagree with physician reviews and autopsy findings, and that cause-of-death assignment practices vary from region to region.\(^{7–19}\)

An increased number of autopsies and physician follow-ups of death certificates would undoubtedly improve the validity and comparability of mortality statistics, but these measures are limited by financial and practical constraints. In fact, the percentage of deaths with autopsy has trended steadily down over the last 20 years in the United States, and the variable indicating whether an autopsy was performed on a given decedent was dropped from national death records in 1995 due to budget constraints.\(^{20}\) In the absence of autopsy-based information, one option for enhancing the comparability of cardiovascular mortality across the states is to use statistical models that rely on the individual- and community-level factors that influence cause-of-death assignment.

The Global Burden of Disease (GBD) study investigated cross-country variation in the level of ill-defined cardiovascular causes of death (which roughly overlap with general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest) and their relationship to intercountry variation in CHD mortality.\(^{21}\) This analysis found that the proportion of nonstroke cardiovascular deaths coded to ill-defined cardiovascular causes was lowest in New Zealand, Finland, and the United Kingdom (\(\approx 7\% \text{ to } 13\%)\) and highest in Japan, Spain, and Belgium (\(\approx 40\% \text{ to } 60\%\)); the proportion of nonstroke cardiovascular deaths coded to CHD mirrored these values and ranged from 25\% to 45\% for Japan, Spain, and Belgium to 75\% to 80\% for New Zealand, Finland, and the United Kingdom. Cross-national comparisons were used to redistribute some ill-defined cardiovascular deaths to CHD,\(^{21,22}\) but these models did not examine the determinants of cause-of-death assignment. In the present report, we used individual death records in the United States to analyze the effects of individual- and community-level factors on cause-of-death assignment across states in the United States. We then used the model to estimate CHD rates (by state, race, sex, and age) that are standardized with regard to the levels of individual- and community-level determinants of cause-of-death assignment.

**Methods**

**Statistical Model**

We used multinomial logistic regression to estimate the relative risk ratios (RRRs) of nonstroke cardiovascular deaths being assigned to CHD or 1 of the ill-defined clusters of cardiovascular causes of death.
TABLE 2. Model Covariates, Descriptions, and Data Sources

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Values</th>
<th>Source</th>
<th>Reference Category for Adjusted Estimates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Race</td>
<td>White, black, Native American, Asian</td>
<td>Death record</td>
<td>White</td>
</tr>
<tr>
<td>Sex</td>
<td>Male, female</td>
<td>Death record</td>
<td>Male</td>
</tr>
<tr>
<td>Age</td>
<td>5-year intervals from 30 to ≥85</td>
<td>Death record</td>
<td>Decedent’s actual age</td>
</tr>
<tr>
<td>Place of death</td>
<td>In-hospital (defined as hospital or healthcare facility), out-of-hospital</td>
<td>Death record</td>
<td>In-hospital</td>
</tr>
<tr>
<td>Educational level</td>
<td>Less than high school, less than college, college or postgraduate</td>
<td>Death record</td>
<td>College education</td>
</tr>
<tr>
<td>Cardiologists</td>
<td>County-level estimate of cardiologists per 100,000 residents (range 1.9 to 12.7)</td>
<td>Dartmouth Atlas of Health Care*</td>
<td>80th Percentile (7.9 per 100,000)</td>
</tr>
</tbody>
</table>

*The Dartmouth Atlas of Health Care (year 1999; available at http://www.dartmouthatlas.org) provides estimates for hospital referral regions, which are linked to ZIP codes, which can in turn be mapped to county codes. Because ZIP codes do not map exactly to county boundaries, counties that did not have a value for cardiologists were assigned the same value as the adjacent county with the closest per capita income.

Data Sources

Data for causes of death and for individual-level variables were from the National Center for Health Statistics’ National Vital Statistics System, which maintains records for every death in the United States, including underlying cause of death and standard sociodemographic characteristics. All deaths between 1990 and 2001 that were attributed to CHD or to 1 of the 3 ill-defined cardiovascular clusters were included in the analysis to maximize predictive power (~7 million deaths). County identifiers for years after 2001 were not available for analysis because of changes in NCHS policy. The analysis was limited to deaths that occurred at ages ≥30 years, because very few cardiovascular deaths occur at younger ages.

Noncardiovascular Ill-Defined Causes

In addition to ill-defined cardiovascular causes, a death may also be assigned to other (general) ill-defined causes (ICD-9 codes 780 to 799; ICD-10 codes R00 to R99). Following the approach used in the GBD study, we reallocated deaths assigned to these causes for people above 30 years of age among noncommunicable diseases, proportional to their observed frequency. The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Regression Analysis

Table 3 shows the RRRs of deaths being assigned to 1 of the 3 ill-defined cardiovascular cause of death clusters relative to CHD for each of the individual- and community-level characteristics, after adjustment for other factors. In-hospital deaths had substantially lower probabilities than out-of-hospital deaths of being attributed to general atherosclerosis and unspecified heart disease (RRR = 0.48) and to heart failure (RRR = 0.82), consistent with the hypothesis that the availability of clinical information at the time of death in hospitals reduces the use of ill-defined cause groups. Assignment to cardiac arrest was slightly more likely for in-hospital deaths relative to out-of-hospital deaths (RRR = 1.07), possibly because of deaths in critical care units with monitoring capabilities being coded to various dysrhythmias.

The number of cardiologists was inversely associated with assignment to all ill-defined cause clusters, with an 11% reduction in the probability of being assigned to heart failure and a 7% reduction for cardiac arrest for each additional...
The introduction of the 10th revision of the ICD (ICD-10) in 1999 reduced the cardiologist per 100,000 residents. The introduction of the 10th revision of the ICD (ICD-10) in 1999 reduced the probability of death being coded to cardiac arrest (RRR = 0.76), but it increased the probability of death being assigned to general atherosclerosis and unspecified heart disease (RRR = 1.05) and heart failure (RRR = 1.31). This finding about the shift to the new revision requires further investigation.

Compared with white men, white females, blacks, and Native Americans had substantially higher probability of being assigned to all 3 cause groups, and Asians had a higher probability of their deaths being assigned to general atherosclerosis and unspecified heart disease or cardiac arrest. Assignment to ill-defined causes was particularly likely for blacks. Deaths among Asians had a lower probability of being assigned to heart failure, which may reflect assignment practice or differences in the natural history of heart disease in these populations.

General atherosclerosis and unspecified heart disease were more commonly used diagnoses in younger age groups. This finding may have been due to the higher utilization of cardiovascular diagnostic studies among individuals over 65 years of age through Medicare, leading to more specific cardiovascular diagnoses in the older age groups. The use of heart failure as a cause of death increased with age, probably reflecting an age-gradient for this sequela of CHD, hypertension, and other heart diseases. Cardiac arrest, however, was relatively more commonly used in the younger age groups. This practice or differences in the natural history of heart disease in these populations.

The observed divergent age patterns of the 3 ill-defined cause clusters require further investigation.
Adjusted Cause-of-Death Statistics (National)

Nationally, CHD, as a proportion of nonstroke cardiovascular deaths, rose from 57% to 64% after we standardized the individual- and community-level determinants to the reference values shown in Table 2. Of the ill-defined categories, “general atherosclerosis and unspecified heart disease” and “heart failure” had larger relative reductions (30% and 38%, respectively, nationally) than cardiac arrest (18%), which made up the smallest share of nonstroke cardiovascular deaths before adjustment. Adjustment resulted in a 10% increase in age-standardized CHD death rates (from 5.5 to 6.1 per 1000) for males and a 14% increase (3.4 to 3.9 per 1000) for females. The total number of CHD deaths above age 30 increased from 433,625 to 489,836 deaths for 2001. Of this increase, 5341 deaths were due to redistribution of deaths certified to noncardiovascular ill-defined codes and the remaining 50,870 to redistribution among CHD and the 3 ill-defined clusters after standardization of the individual- and community-level determinants. CHD as a proportion of nonstroke cardiovascular deaths increased more for females (6 to 8 percentage points) than for males (5 to 7 percentage points) in every age group, possibly because CHD is more commonly assigned to males (Table 4).

CHD proportions of nonstroke cardiovascular deaths increased most for blacks and for Native American females (10 to 12 percentage points) after adjustment (Table 4); Asians and white males had the smallest increase (5 percentage points each). Most of the increase in CHD proportions came from shrinking the shares of “general atherosclerosis and unspecified heart disease” and “heart failure.” The relative contributions of the 3 groups of ill-defined causes to the increase in CHD, however, varied with race, with blacks and Native Americans having the largest relative decline in general atherosclerosis and unspecified heart disease. The reduction in the cardiac arrest cluster varied the least across races.

Percent increases between unadjusted and adjusted CHD death rates were lower for whites and Asians (ranging from 7% to 12% from 1990 to 2001) than for blacks and Native Americans (16% to 24%; Figure 3). During the interval 1990 to 2001, observed CHD mortality for blacks and whites was similar, with some divergence only at the end of the interval (11%). After adjustment, black CHD death rates ranged from 13% to 21% higher than those of whites each year.

Adjusted Cause-of-Death Statistics (State Patterns)

Washington, DC, Alaska, and Nevada had the greatest increase in CHD death rates after adjustment, with adjusted

### TABLE 4. Unadjusted Proportions (%) of Nonstroke Cardiovascular Deaths Assigned to Different CHD-Related Cause Clusters and Change (in Percentage Points) After Adjustment (2001)

<table>
<thead>
<tr>
<th></th>
<th>General Atherosclerosis and Unspecified Heart Disease</th>
<th>Heart Failure</th>
<th>Cardiac Arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unadjusted Change</td>
<td>Unadjusted Change</td>
<td>Unadjusted Change</td>
</tr>
<tr>
<td><strong>By sex and age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30–44 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>44 5 12</td>
<td>–5 2 0</td>
<td>3 0</td>
</tr>
<tr>
<td>Female</td>
<td>32 6 8</td>
<td>–3 3 2</td>
<td>3 1</td>
</tr>
<tr>
<td>45–64 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>57 7 15</td>
<td>–6 3 1</td>
<td>3 1</td>
</tr>
<tr>
<td>Female</td>
<td>49 7 11</td>
<td>–3 4 2</td>
<td>4 1</td>
</tr>
<tr>
<td>65 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>61 5 11</td>
<td>–3 7 2</td>
<td>2 0</td>
</tr>
<tr>
<td>Female</td>
<td>55 8 11</td>
<td>–3 9 4</td>
<td>2 0</td>
</tr>
<tr>
<td><strong>By sex and race</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>61 5 11</td>
<td>–3 6 2</td>
<td>2 0</td>
</tr>
<tr>
<td>Female</td>
<td>55 8 11</td>
<td>–3 9 4</td>
<td>2 0</td>
</tr>
<tr>
<td>Black</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>47 11 16</td>
<td>–8 5 2</td>
<td>3 1</td>
</tr>
<tr>
<td>Female</td>
<td>48 10 13</td>
<td>–5 7 3</td>
<td>3 1</td>
</tr>
<tr>
<td>Native American</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>55 11 17</td>
<td>–8 5 1</td>
<td>3 1</td>
</tr>
<tr>
<td>Female</td>
<td>51 12 16</td>
<td>–7 8 4</td>
<td>3 1</td>
</tr>
<tr>
<td>Asian</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>59 5 13</td>
<td>–5 3 1</td>
<td>4 2</td>
</tr>
<tr>
<td>Female</td>
<td>57 5 11</td>
<td>–3 5 0</td>
<td>4 2</td>
</tr>
</tbody>
</table>
Figure 3. CHD death rates per 1000 by race for 1990 to 2001. Deaths among those aged >45 years are included. Rates in each state are age-standardized to the 2000 US population. Native Am indicates Native American.
rates 51% to 72% higher than the unadjusted (observed) rates (Figure 4). The smallest increases in CHD death rates occurred in North Dakota, Wisconsin, and Missouri (2% to 4% of the original values). New York was the only state with a reduction in CHD death rate after adjustment (~1%). After adjustment, the proportion of nonstroke cardiovascular deaths coded to CHD across states ranged from 59% to 69% compared with 33% to 68% in unadjusted values. The proportion of ill-defined causes was between 14% and 16% compared with 15% to 41% before adjustment. The remaining variation between CHD and ill-defined cardiovascular causes was due to regional differences in other nonstroke cardiovascular deaths: inflammatory heart disease, hypertension, valvular disorders, and cardiac dysrhythmias. After standardization for the effects of exogenous determinants of cardiovascular cause-of-death assignment, the relationship between all cardiovascular mortality and CHD mortality (Figure 5) across states was strengthened substantially.

Mississippi had the highest adjusted CHD death rate and the highest death rates due to all cardiovascular diseases; Oklahoma ranked second for both (Figures 5 and 6). Generally, there was a more consistent relationship between CHD and total cardiovascular death rates after adjustment. Alabama, which had ranked 28th for highest CHD mortality before adjustment, ranked 5th and 7th for CHD and total cardiovascular death rate after adjustment, respectively. The highest adjusted CHD death rates for males occurred in the Mississippi River Delta and Appalachia regions, whereas the lowest CHD death rates were in the southwestern and northwestern states. Adjusted rates for females showed a cross-state pattern similar to that for males but with less variation.

**Discussion**

Definitive identification of CHD deaths that might have been coded as being due to ill-defined cardiovascular causes requires detailed prospective autopsy studies. In this analysis, we retrospectively investigated some of the determinants of variation in cardiovascular death assignment with the aim of enhancing the comparability of CHD death rates across race, sex, and state. Adjusting the assignment of cardiovascular deaths in this analysis led to significant changes in the CHD pattern across states and race-sex groups and partially addressed the “CHD versus total cardiovascular mortality” paradox. The findings are consistent with the clinical literature that suggests that a substantial fraction of deaths assigned to general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest have CHD as the underlying pathology.2–6

The current literature on healthcare disparities almost invariably suggests that whites, males, and people of higher socioeconomic status have better access to healthcare and higher quality of healthcare.25 Even after we controlled for place of death and number of cardiologists per capita, which are proxies for access to care and medical center infrastructure at the community level, race and sex had significant associations with assignment to ill-defined cardiovascular causes, with blacks and Native Americans being the most likely to be assigned to ill-defined causes. The finding may reflect differences in quality of care experienced by minority
patients and differential practices in cause-of-death assignment by race. At the same time, some of the observed racial and gender variation of recorded causes of death may represent actual epidemiological differences in the natural history of CHD, including differential prevalence of risk factors, which cannot be captured in our model. Determination of the exact contributions of cause-of-death assignment versus biological and risk factor determinants would require further epidemiological investigations that include autopsies for cause-of-death assignment. We also examined the interactions between individual-level sociodemographic variables and place of death (results not shown). Some of the interactions were statistically significant, but the final results of the analysis, including the relationships by race and state, were not sensitive to these additional analyses.

Whether a death occurred in the hospital and the number of cardiologists per capita were valuable proxies for clinical information and diagnostic skills at the community level. However, better information on the attributes of the hospitals in which individuals died (eg, training and diagnostic facilities) would allow a more direct assessment of these factors on assignment practices. Such information would become available if death records were linked to hospital characteristics.

The death certificate itself contains other sources of error that may undermine the applicability of this type of model. The examples of potential errors in the death certificate include educational attainment and race, which are provided by next of kin. These errors may have affected the findings of our regression analysis. We did not include deaths assigned to the other 3 nonstroke cardiovascular clusters (hypertensive heart disease, inflammatory heart disease, and other) because these clusters were considered less likely to be cross-coded with CHD. The percentage of nonstroke cardiovascular deaths assigned to these other categories ranged from 15% in Oklahoma to 29% in Washington, DC. Some deaths in these categories may be related to CHD and should be investigated in detailed investigations that rely on autopsies.

Our statistical model to enhance the comparability of cardiovascular cause-of-death estimates might be applied periodically to produce estimates of trends by state or race. As with any statistical model, the results may depend in part on the years from which the data are taken. When year of death was included in the model, a given death was consistently more likely to be assigned to heart failure relative to CHD later in the interval (RRR = 0.72 in 1990 compared with RRR = 0.95 in 2000, with 2001 as the reference year). There was no association with year for general atherosclerosis or cardiac arrest.

Above and beyond comparability, improving the overall accuracy of death certificates is important for public health planning. Improving accuracy would require autopsy validation studies, especially those that examine cause-of-death assignment practices and their determinants across states and races rather than simply for 1 community. However, the number of autopsies, the “gold standard” for accuracy, has declined in the United States over the last 20 years. This unfortunate trend makes the use of statistical models essential in accounting for possible biases. A related strategy for improving cardiovascular death assignment would be more

Figure 5. Adjusted CHD vs all cardiovascular disease death rates for the states in the United States and the District of Columbia (2001). Deaths among those aged ≥45 years are included. Rates in each state are age-standardized to the 2000 US population. $r=0.95$. 

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extensive training and better standardization of assignment practices for those who complete death certificates.

The multinomial logistic model used in the present report can be used for a number of other purposes, including the variation of cardiovascular cause-of-death assignment across countries. Such application requires unit record data that are currently difficult to obtain for many countries. The analytical strategy may also be useful for enhancing the comparability of other clusters of causes, such as diabetes mellitus, renal failure, and related heart disease.

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Disclosures
None.

References

**CLINICAL PERSPECTIVE**

Valid and comparable data on mortality and causes of death are essential for the design and evaluation of health policies and intervention programs. The death certificate is a key source of information for cause-specific mortality, but its utility may be compromised by variability in assignment practices for underlying cause of death between regions and over time. For example, there are conspicuous discrepancies between coronary heart disease (CHD) and total cardiovascular death rates across the United States. The present analysis demonstrates that the personal characteristics of the deceased, such as race and gender, whether the death took place in hospital or not, and health system factors, such as the number of cardiologists, influence the assignment of cause of death between CHD and the following ill-defined cardiovascular causes of death: general atherosclerosis and unspecified heart disease, heart failure, and cardiac arrest. After correction for these factors that undermine the comparability of cause-of-death assignment, the increase in CHD death rates across the United States ranged from 2% in North Dakota to 72% in Washington, DC; New York had a decrease (1%) in CHD death rates after adjustment. Nationally, CHD death rates increased 10% for males and 15% for females. Ideally, detailed clinical history and autopsies would be used more frequently in cause-of-death assignment and certification. With the declining trend in the number of autopsies, uniform and standardized training for those who complete death certificates would help reduce the variability of cause-of-death assignment and the associated impacts on the use of health and medical resources.
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