In-hospital mortality rates from acute myocardial infarction by race in U.S. hospitals: findings from the National Hospital Discharge Survey

EULALIA ROIG, M.D., ANGEL CASTANER, M.D., BRIAN SIMMONS, M.D., RASHMI PATEL, M.D., EARL FORD, M.D., AND RICHARD COOPER, M.D.

ABSTRACT Mortality rates in the United States from coronary artery disease are higher among blacks than whites at younger ages, with a crossover to lower rates above the age of 70. The factors that determine this crossover of age-specific death rates have not been elucidated. Selection from the black population of younger individuals who are sicker by virtue of being more coronary prone might leave a relatively healthier group of older persons. Support for this hypothesis would consist in part of evidence that coronary artery disease has an earlier onset in the black population. We examined data from the National Hospital Discharge Survey for the years 1973–1984 to determine if age-related differences in case-fatality rates existed between whites and nonwhites. In-hospital case fatality rates were 10% to 70% higher for each of the 10 year age groups for nonwhites up to age 70, at which time a crossover occurred. The median age at death from myocardial infarction was approximately 5 years younger in nonwhites compared with whites. National estimates of hospitalization rates for myocardial infarction from these data likewise suggest that nonwhites receive less health care for coronary artery disease than whites relative to recorded fatal events. The age-specific trends in case-fatality support the hypothesis that a cohort selection effect in part determines the black/white differentials in coronary artery disease. Relative susceptibility of the black and white population is thus not appropriately estimated by age-adjusted rates, but should be examined on an age-specific basis within the framework of selection effects on a cohort.


THE EPIDEMIOLOGY of coronary artery disease (CAD) in the black population of the United States has been the subject of significant recent controversy. From the cohort studies that have been reported to date it is clear that the standard coronary risk factors influence the development of CAD among blacks to a reasonably similar degree to that described for U.S. whites and other populations. Major inconsistencies have arisen, however, when efforts have been made to compare quantitatively the disease burden from CAD in whites and blacks. Given the twofold greater prevalence of hypertension among U.S. blacks, and similar levels of hypercholesterolemia and cigarette smoking, it would be anticipated that blacks should suffer higher rates of CAD. Comparison of vital statistics data, however, suggest nearly equal age-adjusted CAD mortality in black and white men, with only modestly higher rates among black women compared with their white counterparts. Data from several community studies have further shown that the in-hospital death rates from CAD is lower among blacks than whites, accompanied by an excess of out-of-hospital deaths. Death certificate coding for out-of-hospital and sudden CAD death is not highly reliable and it has been suggested that the higher CAD rates reported among blacks in the vital records are an artifact, resulting primarily from misclassification of these medically unattended events.

Estimated differentials in CAD mortality between the black and white populations have generally been based on only age-adjusted rates. Age adjustment tends to weight these comparisons toward deaths among the elderly and does not account for the potential effects of selection and competing cause. These
concerns led to the formulation of the following hypothesis as an explanation for the observed racial differentials presently observed in the United States with respect to this disease: CAD is in fact more severe among blacks than whites, as would be anticipated from the distribution of coronary risk factors. Among blacks the disease would therefore be expected to have its onset at a younger age (i.e., a higher proportion of cases will occur "prematurely") and at any given age the disease will be more severe relative to that in whites. To test this hypothesis we examined racial differences in age-specific case fatality rates from myocardial infarction in a representative sample of U.S. hospitals. In addition, the age trends in mortality from CAD from the two populations were further studied through use of vital statistics data.

Methods

Data sources

Hospital discharge data. Hospital discharge data were provided by the National Center for Health Statistics (NCHS) through the National Hospital Discharge Survey (NHDS). Detailed descriptions of the methods used for the NHDS have been published elsewhere.\textsuperscript{15,16} The NHDS is an ongoing sample survey drawn from a frame of 7500 short-stay hospitals in the United States, exclusive of military and Veterans Administration facilities. Hospitals are stratified on the basis of size (number of beds) and geographic location, and within each hospital a random sample of discharges are selected. Data are abstracted either by local hospital staff or by representatives of the NCHS. In 1984, from a total of approximately 6000 hospitals, 533 were selected to participate, and data were ultimately collected from 407 of those institutions. Approximately 192,083 abstracts of medical records were entered into the survey file that year.

All discharge diagnoses and procedures were listed on the abstract in the order of the principal one, or the first-listed one if the principal one was not identified, followed by the order in which all diagnoses were entered on the discharge sheet. Coding was then carried out by NCHS staff according to the Eighth Revision of the International Classification of Diseases, Adapted (ICDA-8) for 1973-1978, and the Ninth Revision (ICD-9-CM) for 1979–1983. Race, which is not formally required on discharge summaries, was missing from 8.9% of records in 1984; persons with race not stated were excluded from the present study in all years. Procedures for identifying nonwhites varied from year to year but consisted primarily of aggregating blacks and native Americans; Hispanics were coded "white". The category of "nonwhite" is taken to represent "black" in this analysis, as in all studies based on the national vital statistics data before 1979. For the year 1978 data were available on computer tapes on which race was coded as black and nonwhite; 88% of the individuals in the nonwhite category were identified as black. It is assumed, therefore, that the vast majority of individuals in the nonwhite category in each year are black and this segment of the population weights the outcome.

Analysis was restricted to persons over the age of 35, given the low incidence of myocardial infarction before this age. In-hospital fatality rates were age-adjusted to a standard population of all cases. In the entire sample, including all 12 years of data, 43,849 discharge forms contained the mention of myocardial infarction (ICD Code 410) at any rank order (i.e., not restricted to principal or first diagnosis), and of this number 3146 (13.9%) were coded as race "other." Because of those methodologic problems the NCHS does not routinely report data from the NHDS by race, and the present findings must be interpreted with caution for this reason. The survey did not differentiate acute myocardial infarction during the hospitalization being examined from any mention of a prior event; therefore the same individual admitted more than once to a study hospital would be counted as a case on each occasion if acute myocardial infarction appeared on the discharge summary. To obtain data on a sufficient number of deaths in each age-sex-race cell, data from the 12 year period 1973 to 1984 were pooled. Separate examination of the data in 4 year subgroups did not reveal any important trends over time in the patterns of age or racial differentials. As a result of the design of the NHDS, estimates of hospitalization rates based on the civilian noninstitutionalized population could be obtained, and these were calculated for each age-sex-race group.

Mortality rates and vital statistics. Mortality data from acute myocardial infarction for the resident U.S. population were obtained from the NCHS. Death rates from myocardial infarction (ICD Code 410) in 1983 were examined by age-sex-race subgroups, and the ratio of black to white deaths was calculated. Population estimates for 1980 were obtained from the Bureau of the Census and used to estimate discharge rates for myocardial infarction in conjunction with the NHDS data for the years 1978 to 1982. Blacks were used as the population base to calculate nonwhite rates.

Statistical analysis. In reporting data from the NHDS the NCHS advises that estimates based on a sample size less than 60 be regarded with caution. The smallest number of discharges for all 12 years combined was encountered in the age group 35 to 39 among blacks, in which 77 patients were discharged and five died. Estimates of sampling error used in statistical testing do not account for weighing or sample design, providing additional reason to generalize beyond the sample with caution. Differences in case fatality rates were tested based on point estimates for each 5 year age group with the chi-square test. Differences in the relationship between case-fatality rates and age were examined by comparing the slopes of the rate between whites and nonwhites after fitting the curve to a linear regression line plot with the use of programs available with SAS. Although the relationship between fatality rates and age is curvilinear, the purpose of this analysis was not to estimate the shape of the curve but to compare the slopes in the two groups; programs were not available to compare two curvilinear relationships, so straight line plots were estimated. Since the vital statistics represent data from entire population, not a sample, no statistical tests were performed on those data.

Results

As presented in table 1A and figure 1, the in-hospital case fatality rates for whites and nonwhites were calculated as a percentage by 5 year age groups from 35 to 39 and over. Rates were higher for nonwhites than whites until the age of 70, at which time a crossover for the two oldest age categories was observed. Fatality rates were significantly higher for nonwhites compared with whites only among persons 35 to 39 years old (p = .03). At the same time, the age-adjusted mortality was very slightly higher for whites for the entire age spectrum because of the greater number of deaths among the elderly in the white population and the higher fatality rate in this oldest age group. The
TABLE 1A
In-hospital case-fatality rates from myocardial infarction by race and age: the NHDS, 1973–1984

<table>
<thead>
<tr>
<th>Age group (yr)</th>
<th>White</th>
<th>Nonwhite</th>
</tr>
</thead>
<tbody>
<tr>
<td>35–39</td>
<td>19/615 (3)</td>
<td>5/77 (6)</td>
</tr>
<tr>
<td>40–44</td>
<td>57/1,315 (4)</td>
<td>11/175 (6)</td>
</tr>
<tr>
<td>45–49</td>
<td>154/2,223 (17)</td>
<td>20/248 (8)</td>
</tr>
<tr>
<td>50–54</td>
<td>227/3,460 (7)</td>
<td>30/348 (9)</td>
</tr>
<tr>
<td>55–59</td>
<td>443/4,691 (11)</td>
<td>50/425 (16)</td>
</tr>
<tr>
<td>60–64</td>
<td>771/5,541 (14)</td>
<td>72/443 (16)</td>
</tr>
<tr>
<td>65–69</td>
<td>1,095/5,952 (18)</td>
<td>90/435 (21)</td>
</tr>
<tr>
<td>70–74</td>
<td>1,374/5,897 (23)</td>
<td>84/391 (21)</td>
</tr>
<tr>
<td>75+</td>
<td>3,591/10,909 (33)</td>
<td>177/604 (29)</td>
</tr>
<tr>
<td>Crude rate</td>
<td>7,776/40,703 (19)</td>
<td>539/3,146 (17)</td>
</tr>
<tr>
<td>Age-adjusted rate</td>
<td>(19)</td>
<td>(18)</td>
</tr>
</tbody>
</table>

*aTotal discharges/deaths.
*bPercent in-hospital deaths.

dratios of mortality rates at various ages, nonwhite to white, were as follows: 35 to 44, 1.7:1.0; 45 to 54, 1.3:1.0; 55 to 64, 1.1:1.0; 65 to 74, 1.0:1.0; and 75+, 0.8:1.0. The relationship between the case fatality rate and age in the two groups was compared by fitting the age-specific rates (table 1A) to a linear regression plot. For whites the beta coefficient (±SE) was 0.701 ± 0.083, while for nonwhites it was 0.567 ± 0.065; the difference between these two slopes was highly significant statistically (t = 24).

Among nonwhite men the in-hospital fatality rate was higher than among whites for all but one of the 5 year age groups under 70, with a crossover observed at that point (figure 1, B). Only sixteen nonwhite women under 40 were included in the sample, and in this group three (19%) died in the hospital; except for higher rates in this youngest age group, age-specific death rates were similar in women of both races until age 70, at which time nonwhite women experienced lower rates (figure 1, B). A somewhat higher proportion of nonwhite vs white patients were women — 43% vs 37%, respectively. Overall in-hospital death rates were 16% for white men, 15% for nonwhite men, 24% for white women, and 20% for nonwhite women. Women experienced higher death rates than men in all age groups below 70. The ratio of total discharges, men to women, was also highly age dependent; thus, at age 35 to 39 the mortality ratio for men to women was 6.1:1.0 for whites and 3.8:1.0 for nonwhites, while over age 70

![IN HOSPITAL MORTALITY FROM ACUTE MYOCARDIAL INFARCTION (1973–1984)](image)

**FIGURE 1.** Top, In-hospital case-fatality rate from acute myocardial infarction by race, sex, and age, in the United States, 1973–1984 (%). Bottom, Ratio of in-hospital case-fatality rates from acute myocardial infarction, nonwhite/white, by sex and age.
the ratio was 0.8:1.0 for both whites and nonwhites. An estimate of the rate of additional complicating conditions was sought by examining the frequency with which other disorders were listed on the discharge sheet in the four sex-race groups for the year 1984. With a single exception, the following diagnostic codes (ICD-9-CM) appeared among the first five listed as second diagnoses for all groups: other forms of chronic ischemic heart disease (414), cardiac arrhythmias (427), heart failure (428), diabetes mellitus (250), and essential hypertension (410) (table 1B). Further examination of third-listed diagnoses likewise did not provide evidence of any differential pattern of associated comorbid conditions by sex or race. The rate at which other comorbid conditions were listed was also identical in all four groups, ranging from 52% to 55% cumulative frequency for the above-mentioned five conditions. These data do not lend support to the hypothesis that differential fatality rates can be attributed to the more frequent association of other medical illnesses among blacks.

Given the steeper relationship between age and case-fatality for whites and the more youthful age structure of the black populations, proportional mortality was distributed upward for whites compared with nonwhites (table 2); the median age at death from myocardial infarction in this sample fell between 70 and 74 for whites and between 65 and 69 for nonwhites.

Incidence rates based on hospital discharge rates after myocardial infarction in the white and nonwhite populations were estimated for the 5 years around 1980. For both sexes incidence rates were virtually identical for the two racial groups at all ages, with a slight excess among nonwhites during the age decade of the 40s (figure 2, A). For men considered alone, a slightly lower rate was observed between ages 45 and 70, although these rates were unstable because of the small number of cases. Population-based in-hospital death rates were estimated by combining the number of discharges, the case-fatality rates and, the population estimates by age, sex, and race (table 3). Hospital data for the five years around 1980 were again used, together with the 1980 census data. The actual rate of death from myocardial infarction in the hospital, given higher case fatality and similar incidence, is twofold higher for nonwhites under age 45, with the expected age trend.

At least half of the deaths coded to myocardial infarction occur outside the hospital. Trends in the death rate in 1983 for the entire United States by age and race are presented in figure 3, A. (Race as black is available for these data.) Consistently higher rates were noted until the crossover at age 70. The ratio of age-specific death rates, nonwhite/white, were greater than 3.0:1.0 for the younger age groups for both sexes; age-adjusted rates were similar, however, among men and only 20% higher for women (white men = 196, black men = 176, white women = 89, black women = 107, per 100,000). Again, the variable racial differentials by age are a result of the crossover phenomenon observed among the elderly. The exponential increase in the number of CAD deaths with age account for the weighting of the age-adjusted rates by the 70+ age group.

Table 2

<table>
<thead>
<tr>
<th>Age group (yr)</th>
<th>White</th>
<th>Nonwhite</th>
</tr>
</thead>
<tbody>
<tr>
<td>35–39</td>
<td>0.2</td>
<td>0.9</td>
</tr>
<tr>
<td>40–44</td>
<td>0.7</td>
<td>2.1</td>
</tr>
<tr>
<td>45–49</td>
<td>2.0</td>
<td>3.7</td>
</tr>
<tr>
<td>50–54</td>
<td>2.9</td>
<td>5.6</td>
</tr>
<tr>
<td>55–59</td>
<td>6.3</td>
<td>9.2</td>
</tr>
<tr>
<td>60–64</td>
<td>9.9</td>
<td>13.4</td>
</tr>
<tr>
<td>65–69</td>
<td>14.1</td>
<td>16.7</td>
</tr>
<tr>
<td>70–74</td>
<td>17.7</td>
<td>15.6</td>
</tr>
<tr>
<td>75+</td>
<td>46.2</td>
<td>32.8</td>
</tr>
</tbody>
</table>

Values are proportions of all deaths occurring in a given age stratum (%).

Discussion

Comparison of the relative disease burden for a common illness in two populations is a complicated undertaking. Selection of healthy survivors will play an important role in determining rates of common illness among the elderly. For the predominant cause of death, such as CAD in the United States, large age-related variations could be introduced by this phenom-

### Table 1B

<table>
<thead>
<tr>
<th>Rank order</th>
<th>White men</th>
<th>Nonwhite men</th>
<th>White women</th>
<th>Nonwhite women</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>414 (26.3)²</td>
<td>414 (19.1)</td>
<td>414 (22.2)</td>
<td>414 (15.6)</td>
</tr>
<tr>
<td>2</td>
<td>427 (36.7)</td>
<td>427 (32.6)</td>
<td>428 (35.1)</td>
<td>428 (27.3)</td>
</tr>
<tr>
<td>3</td>
<td>428 (45.9)</td>
<td>401 (41.1)</td>
<td>250 (45.5)</td>
<td>427 (35.9)</td>
</tr>
<tr>
<td>4</td>
<td>250 (50.6)</td>
<td>428 (48.9)</td>
<td>427 (50.6)</td>
<td>250 (43.8)</td>
</tr>
<tr>
<td>5</td>
<td>410 (55.1)</td>
<td>250 (53.9)</td>
<td>401 (54.2)</td>
<td>401 (50.8)</td>
</tr>
</tbody>
</table>

Diagnostic codes: 414 = ischemic heart disease; 427 = cardiac arrhythmias; 428 = heart failure; 250 = diabetes mellitus; 410 = acute myocardial infarction; 401 = essential hypertension.
enon of selection. In recent years increased attention has been focused on the question of the relative rates of CAD in the black and white populations.\textsuperscript{21-24} The data from the NHDS presented here, in combination with the vital statistics, demonstrate that blacks suffer higher rates of CAD at younger ages. From these findings we conclude that CAD has an earlier onset among blacks and at any given age is more severe. The only potential explanation for the decrease with age relative to whites in both incidence and case-fatality would be the effect of selection.\textsuperscript{19, 20} The paradox of lower age-adjusted CAD rates among blacks is therefore a statistical artifact yielded by the method of analysis. In evaluating the relative disease burden, two distinctly different approaches should be taken, based respectively on the age-adjusted and age-specific data. While the age-adjusted data provide an estimate of the overall burden for the population relative to other causes and the age-structure of the population, age-specific rates more accurately reflect the force of the disease-causing factors to which the population is exposed.

Studies of mortality crossover have been carried out primarily by demographers,\textsuperscript{19, 20, 25-27} Manton and Stal-lard in particular have explored the effect of "heterogeneity of frailty" on the age-related increase in mortality.\textsuperscript{25} While it could be assumed that the rate of development of a disease process with age for an individual could be imputed from the rates of increase for the whole population, this assumption ignores the effect of selection.\textsuperscript{25} This concept implies that susceptible individuals will be removed from the cohort at a younger age, leaving a group of relatively more robust survivors. A convergence of age-specific mortality rates — or in the special case an actual crossover — is therefore observed in the comparison of the black and white populations of the United States,\textsuperscript{26-28} and this has

### TABLE 3
In-hospital mortality rates from myocardial infarction by race and age in the United States, 1978–1982 (rate per 100,000)

<table>
<thead>
<tr>
<th>Age group (yr)</th>
<th>White</th>
<th>Nonwhite</th>
</tr>
</thead>
<tbody>
<tr>
<td>35–39</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>40–44</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>45–49</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td>50–54</td>
<td>28</td>
<td>36</td>
</tr>
<tr>
<td>55–59</td>
<td>66</td>
<td>72</td>
</tr>
<tr>
<td>60–64</td>
<td>126</td>
<td>110</td>
</tr>
<tr>
<td>65–70</td>
<td>180</td>
<td>210</td>
</tr>
<tr>
<td>70–74</td>
<td>230</td>
<td>210</td>
</tr>
</tbody>
</table>

Rate based on the average in-hospital rate for 1978–1982 and the population base of the 1980 census.
been shown not to be a result of inaccuracies in the census data or age reporting.\textsuperscript{19, 28, 29} The crossover/convergence relationship observed in the trajectory of age-specific mortality rates exists not only for all-cause mortality, but for the major individual causes as well.\textsuperscript{19, 29} Stroke mortality, for example, which begins at much higher rates among blacks, likewise converges with that for whites among the elderly.\textsuperscript{30} This relationship also applies in the comparison of the two sexes; thus, among whites a 400\% excess mortality for CAD in the early 40s is reduced to 25\% over age 85 in the national rates,\textsuperscript{26} and male/female prevalence crossed over at age 65 in the Framingham study.\textsuperscript{31}

How does heterogeneity of frailty explain the differential pattern of CAD mortality seen in blacks and whites? The increased population risk of CAD, a result primarily of excess hypertension, leads to premature atherosclerosis among blacks. Evidence for the earlier onset of CAD is derived from two sources, i.e., increased death rates at younger ages, and a higher case-fatality rate among those admitted to the hospital with the diagnosis of myocardial infarction. While it is possible, as noted above, that increased mortality based on vital records could be an artifact of miscoding of out-of-hospital deaths, it is unlikely that this differential applies to hospitalized cases. The only plausible explanations of the increased case-fatality rates are more severe CAD, the presence of other complicating medical conditions, and/or poorer quality health care. Our estimate of complicating conditions suggested no race difference in this sample; we were unable to evaluate quality of care. Neither of these latter two explanations, however, is consistent with a subsequent crossover to lower rates among blacks. Since the development of CAD is an age-related process, the onset of the disease at an earlier age in a given population implies that the average patient will have more serious disease at any given age. Invoking the explanation of more severe disease to explain higher case-fatality among younger blacks is likewise consistent with the vital statistics data suggesting more deaths in the population. Selection against the more coronary-prone individuals as a result of exposure to a greater force of mortality from atherosclerosis would leave a healthier cohort. At the same time, coronary-prone individuals would be more likely to die from other cardiovascular
causes, such as stroke, heart failure, and renal disease, and cancer associated with cigarette use; this latter phenomenon is probably more important quantitatively. The effect of this high competing mortality from diseases related to hypertension and smoking would further accentuate the selection of a cohort over 65 less susceptible to CAD.

Both all-cause mortality rates and CAD rates are lower in elderly blacks compared with whites. With the most straightforward approach of dividing the population at age 65, it is possible to account for only a fraction of the deficit in CAD deaths among elderly blacks by transfer of excess CAD deaths from persons under 65. About half of the difference, however, can be accounted for by excess deaths from the broader category of cardiovascular diseases, and on the basis of competing cause it is reasonable to assume that premature stroke and heart failure have eliminated some of the coronary-prone individuals. An additional 5% to 30% of the deaths, however, must be assumed to have resulted from other, noncardiovascular, causes. While it is likely that lung cancer, which is twice as common in black as white men under age 65, may eliminate potential CAD victims, it is difficult to identify other specific death categories that are of sufficient size to compete with CAD. Furthermore, all such arguments must remain theoretical since an individual can die only once and all assumptions about competing cause must remain unproven empirically.

In-hospital case fatality rates from myocardial infarction are generally reported to be in the range of 10% to 20% and have changed little over the last two decades. The lower limit of rates based on recent data were reported by the community surveillance program in Minneapolis, where 9% of men and 11% of women admitted with a documented myocardial infarction died before discharge. Rates in the range of 20% are more commonly reported, however. The 30 day fatality from myocardial infarction in the Framingham study was 16% for men and 28% for women. Based on 17,000 admissions to 65 hospitals in the Boston area, case-fatality rates were 22% in 1973–1974 and 23% in 1978–1979. Three previous reports including black patients demonstrated rates between 20% and 25%, and these did not differ from those for whites in the same geographic area or institution. Comparisons by age group were not carried out, and the samples were too small individually to permit such an analysis, so it is not possible to determine if the age-related racial differentials in case-fatality described here were present in these other studies. There are data suggesting that long-term survival after myocardial infarction is shorter in blacks, although this question requires further study.

As an attempt to gain insight into population-based event rates the present data set is not without significant limitations. As noted in the Methods section, a sizable hospital nonresponse rate exists for each year, and data on race are missing for 5% to 10% of records. Separate analysis of discharge summaries in which race was not stated suggest that they follow the pattern of all summaries combined, i.e., they are drawn proportionally from white and nonwhite categories, but this cannot be proven. Further bias may have been introduced by exclusion of all nonhospital cases. There is evidence that out-of-hospital death rates are higher for blacks than whites and delay before arrival at the hospital is markedly prolonged. Whether this phenomenon results in a mix of hospitalized cases with greater or lesser risk of dying cannot be determined with the data presently available, however. Local studies with direct population surveillance will be required to answer these questions.

Incidence rates for myocardial infarction in blacks compared with those in whites have been difficult to determine and are the subject of some controversy. Seven population studies reviewed by Gillum reported comparative data by race collected before 1978. Blacks were noted to have lower age-adjusted rates in all of these studies; the sample size of each study was not large, however, and those from the rural South may not be representative. The 5 year follow-up data of the Multiple Risk Factor Intervention Trial screenees demonstrate lower than expected CAD rates among blacks, with a lower risk associated with hypertension. Although based on a very large sample, the representative character of these findings remains open to question. More recent data from the pilot phase of the Community Cardiovascular Surveillance Program (CCSP) demonstrate higher age-adjusted death rates from myocardial infarction (ICD Code 410) for blacks of both sexes, and this differential is even more pronounced in the younger age groups, while age-adjusted rates of nonfatal events are higher in whites than blacks. Interestingly, however, incidence rates are consistently higher for blacks in the age groups under 45, thus confirming the crossover pattern seen in our data. Since all these estimates of incidence rely on hospitalization rates they must be viewed as an inexact reflection of the true population rate. The findings from the large data base reported here suggest that over the last decade there have been comparable rates of admission for myocardial infarction in blacks and whites. The death rate from CAD has declined in both
blacks and whites since the late 1960s, so it is unlikely that lower incidence rates from the earlier studies reflect secular trends. At the present time overall health care utilization is similar for the two races, although there may be some residual inequality if one corrects for increased need, i.e., disease prevalence, among blacks. At any rate, the present study and the pilot phase of CCSP represent the largest and most recent studies since the early 1970s. Despite differences in methods, the rates reported here are in a very similar range.

In summary, based on the different age patterns of admission to the hospital for myocardial infarction and the case-fatality rates, we conclude that CAD onset is at an earlier average age in blacks than whites. The most likely explanation of findings presented here would thus be the presence of more severe atherosclerosis in the age groups under 70 years. Lower rates in the elderly could be explained by selection. Consideration of the relative rates of CAD in the black and white populations as they relate to cause or force of mortality should thus rely on age-specific disease rates, not those that are age-adjusted. Further data from well-designed surveys that apply standardized diagnostic criteria will be needed to confirm the findings of the present study and further clarify questions related to out-of-hospital mortality.

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References
5. Neaton JD, Kuller LH, Wentworth D, Borhani NO: Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. Am Heart J 108: 759, 1984
10. Weisse AB, Abisu PD, Thind IS: Acute myocardial infarction in Newark, NJ. Arch Intern Med 137: 1402, 1985
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Community Cardiovascular Surveillance Program: Final report to the National Heart, Lung and Blood Institute, 1984, CCSP Coordinating Center, Department of Epidemiology and Preventive Medicine, University of Maryland, Baltimore


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