Background and Purpose—Many countries observed rapidly declining stroke mortality rates during 1970–1990, followed by a slowing or a cessation of this decline. This slowing was seen for both sexes and all ages. Here we test the hypothesis that improvements in coronary heart disease (CHD) survival can explain this slowing through an increase in the number of CHD survivors at an increased risk for stroke.

Methods—We created multistate life-table models based on the survival experience of 46 years of follow-up of the Framingham Heart Study cohort. Improvements in survival after CHD were modeled by decreasing mortality rates for those with CHD. We analyzed whether improved CHD survival could result in a >3% increase in annual stroke mortality rates, which would be enough to eliminate the previously observed decline.

Results—CHD survival improvements led to an increase in the number of stroke deaths but also a concomitant increase in the total population size. Under no circumstances was there an annual increase in stroke mortality rates approaching 3% for both sexes and for younger and older age groups.

Conclusions—The hypothesis that increases in the numbers of people with CHD, as a consequence of improvements in CHD survival, explain the observed slowing of the stroke mortality rate decline must be rejected. The true explanation is also likely to be a factor that changed markedly around 1990, but with more direct effects on stroke mortality. (Stroke. 2003;34:1610-1616.)

Key Words: coronary heart disease ■ incidence ■ models, theoretical ■ stroke
model was similar to the aforementioned model with the addition of an age dimension and transitions between health states. The model has the states “no CHD,” “CHD,” and “death,” and transitions to death can be stroke or nonstroke mortality. We constructed the multistate life table using age-specific transition rates derived from 46 years of follow-up of the Framingham Heart Study.6,7 We estimated each set of age-specific transition rates (“no CHD to CHD,” “no CHD to nonstroke death,” “no CHD to stroke death,” “CHD to nonstroke death,” and “CHD to stroke death”) for ages 40 to 90 years using Poisson regression with age entered continuously, using the best-fitting polynomial representation (STATA 7) (for further explanation, see the Appendix, which can be found online at http://stroke.ahajournals.org). During follow-up of the 5209 original participants, there were 3678 (1812 men and 1866 women) non-stroke deaths, 281 (114 men and 167 women) stroke deaths, and 1907 (1055 men and 852 women) cases of incident CHD. Life-table models were constructed separately for each sex and represent ages 40 to 85 years, starting with a cohort free of known CHD at age 40 years. To analyze the consequences of improved survival after CHD, we modeled a constant 30% decrease in the risk of nonstroke mortality from the state of CHD for all ages. This was the relative mortality risk (at both 28 days and up to 4 years [95% CI, 0.54 to 0.98 in men and 0.54 to 0.98 in women]) after myocardial infarction in patients in 1980 compared with 1970 in the United States.8 We analyzed the change in stroke mortality in the year after the event stroke mortality rate between ages 50 to 74 years and ages 75 to 84 years for comparison with the literature. Age standardization was performed with the use of the 1990 US population.9 All outcomes were estimated separately for men and women.

The sensitivity of the results to the underlying transition rates was tested through analyses with the use of transition rates representing the 95% confidence limits. We estimated the outcomes of a “worst case scenario” using the combination of the transition rate limits giving the greatest increase in the stroke mortality rate. We also performed sensitivity analyses on the degree of improvement in survival after CHD using a mortality decrease of 50% (based on the limit of the described 95% CI). We estimated the maximum theoretical change between ages 40 and 85 years associated with improvements in survival after CHD by analyzing a life table in which the effects have flowed through all the ages and the population has reached equilibrium. For example, changes in the stroke mortality rate for 85-year-old persons are derived from changes at all ages before and including age 85 years. Theoretically, it would take 45 years for the full effect derived by this life table to be seen in 85-year-old persons because it depends partly on changes flowing through from those aged 40 years.

Results

The crude annual rates of stroke mortality decline during 1981–1991 in the United States were 4.1% (95% CI, 4.0 to 4.2) and 3.8% (95% CI, 3.7 to 3.9) for men and women, respectively. The age-standardized rate of stroke mortality for 40- to 85-year-old persons was 35.4 per 100,000 population in 1981 and 29.7 per 100,000 population in 1991 (95% CI, 29.2 to 30.2). The age-standardized rate of stroke mortality for those aged 85 years or older was 45.4 per 100,000 population in 1981 and 26.8 per 100,000 population in 1991 (95% CI, 15.0 to 48.7). The age-standardized rate of stroke mortality for those aged 40 years or older was 30.1 per 100,000 population in 1981 and 26.3 per 100,000 population in 1991 (95% CI, 25.8 to 26.7) (Table 1).
The CHD prevalence and the relative risk for stroke associated with CHD (Table 1). To see a ≥3% increase in the stroke mortality risk, a combination such as a 25% increase in the population with CHD, relative risks of stroke after CHD of ≥2, and an initial CHD prevalence of ≥20% is needed (Table 1). These results suggest that increases in the stroke mortality rate of ≥3% could only be expected in a very limited range of age and sex groups. High CHD prevalence (≥20%) and a large increase in the population with CHD (≥25%) are more likely in older, male populations because they require both high absolute risks of CHD and an accumulation of survival benefits. In contrast, relative risks for stroke associated with prior CHD of ≥2 are restricted to younger age groups (data not shown). Finally, we would not expect such large increases in the population with CHD to be likely in a single year, suggesting that if stroke mortality rate increases of 3% due to improved CHD survival were possible, they could only occur after a number of years of accumulation rather than consecutively each year.

To take into account the accumulation of survivors with CHD across ages, we created a multistate life table representing the situation before improvements in post-CHD survival. The age-standardized population stroke mortality risks derived from this model were 81/100 000 (ages 40 to 74 years) and 534/100 000 (ages 75 to 84 years) for men and 67/100 000 (ages 40 to 74 years) and 438/100 000 (ages 75 to 84 years) for women. These figures can be compared with the US national stroke mortality rates during 1989–1993, which were 42/100 000, 526/100 000, 33/100 000, and 436/100 000, respectively.3 The total mortality rates were also comparable to the US white population rates for 1989–199110 (data not shown). For men, the CHD prevalence was 20% at age 65 years and 29% at age 75 years. The relative risk of stroke mortality after CHD was 2.0 at age 65 years and 1.6 at age 75 years.

For men, a 30% reduction in nonstroke mortality from the state of “CHD” resulted in a 1-year increase in the number of stroke deaths of 1% at age 75 (Table 2). The stroke mortality rate increased by only 0.3%. This discrepancy arises as a result of the simultaneous increase in the number of stroke deaths and the decrease in nonstroke deaths. In a population homogeneous except for CHD status (Figure 2), the stroke mortality risk increases as the proportion of those with CHD increases (Table 1) (as long as the stroke mortality risk is greater in those with CHD). The degree of increase is independent of the underlying absolute stroke risk in the population free of CHD. It is dependent on

The age-standardized population stroke mortality rates decreased by 3% to 4% annually (Figure 1), the objective of our analysis was to determine whether improvements in survival after CHD could plausibly lead to the 3% to 4% annual increase in stroke mortality rates required to eliminate the observed declines.

In a population homogeneous except for CHD status (Figure 2), the stroke mortality risk increases as the proportion of those with CHD increases (Table 1) (as long as the stroke mortality risk is greater in those with CHD). The degree of increase is independent of the underlying absolute stroke risk in the population free of CHD. It is dependent on

4.3) for men aged 35 to 74 years, 3.6% (95% CI, 3.5 to 3.7) for men aged ≥75 years, 4.1% (95% CI, 4.0 to 4.2) for women aged 35 to 74 years, and 3.1% (95% CI, 3.0 to 3.2) for women aged ≥75 years. Because the stroke mortality rates did not change (or slightly increased) in these groups during 1992–1995 (Figure 1), the objective of our analysis was to determine whether improvements in survival after CHD could plausibly lead to the 3% to 4% annual increase in stroke mortality rates required to eliminate the observed declines.

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### TABLE 1. Percentage Increase in Population Stroke Mortality Risk After an Increase in the Population With CHD Within a Single, Otherwise Homogeneous Population*

<table>
<thead>
<tr>
<th>Initial CHD prevalence</th>
<th>Relative Risk of Stroke Mortality Post-CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1.5</td>
</tr>
<tr>
<td>0.02</td>
<td>0.24%</td>
</tr>
<tr>
<td>0.05</td>
<td>0.57%</td>
</tr>
<tr>
<td>0.10</td>
<td>1.05%</td>
</tr>
<tr>
<td>0.20</td>
<td>1.73%</td>
</tr>
<tr>
<td>0.30</td>
<td>2.12%</td>
</tr>
</tbody>
</table>

Increase in the number with CHD: 50%

<table>
<thead>
<tr>
<th>Initial CHD prevalence</th>
<th>Relative Risk of Stroke Mortality Post-CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1.5</td>
</tr>
<tr>
<td>0.02</td>
<td>0.48%</td>
</tr>
<tr>
<td>0.05</td>
<td>1.13%</td>
</tr>
<tr>
<td>0.10</td>
<td>2.04%</td>
</tr>
<tr>
<td>0.20</td>
<td>3.31%</td>
</tr>
<tr>
<td>0.30</td>
<td>3.97%</td>
</tr>
</tbody>
</table>

*See Figure 2.

Bolded figures represent scenarios in which the population stroke mortality risk increased by ≥3%.

TABLE 2. Percentage Increase in Stroke Death Rates and Numbers After a 30% Decrease in the Nonstroke Mortality Rate Post-CHD

<table>
<thead>
<tr>
<th>Period of Change</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 year</td>
<td>1 year</td>
</tr>
<tr>
<td>Scenario†</td>
<td>Base</td>
<td>Worst Case</td>
</tr>
<tr>
<td>Percentage increase in:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of stroke deaths, age 75</td>
<td>1.0%</td>
<td>1.5%</td>
</tr>
<tr>
<td>Stroke mortality rate, age 75</td>
<td>0.3%</td>
<td>0.7%</td>
</tr>
<tr>
<td>Age-standardized stroke mortality rate, ages 50–74</td>
<td>0.2%</td>
<td>0.6%</td>
</tr>
<tr>
<td>Age-standardized stroke mortality rate, ages 75–84</td>
<td>0.3%</td>
<td>0.9%</td>
</tr>
</tbody>
</table>

* A 45-year period of change is the maximum change possible in this population, as the effects have flowed through all the ages analyzed, from 40 to 85.

†The “worst case scenario” is when the combination of upper and lower 95% CI transition rate estimates is chosen to produce the highest possible increase in the stroke mortality rate.
deaths and the number of person-years lived (Figure 3). The age-standardized stroke mortality rates increased by 0.2% for ages 40 to 74 years and by 0.3% for ages 75 to 84 years. For women, the increases were even less (Table 2). Lesser rate increases were seen for the second and third years after the introduction of the change.

These results were not very sensitive to the nonstroke mortality rate or the CHD incidence rate. A survival improvement of a 50% decrease in the post-CHD nonstroke mortality rate led to annual increases in the age-standardized stroke mortality rates of 0.4% for men and 0.2% for women. A worst case scenario using the upper 95% CI limits for the transitions “no CHD to CHD,” “no CHD to nonstroke death,” “CHD to nonstroke death,” and “CHD to stroke death” and the lower 95% CI limits for the transition “no CHD to stroke death” was still associated with less than a 1% increase in the annual stroke mortality rate for men and women at all ages (Table 2).

Varying the type of polynomial representation for age within the Poisson models predicting the transition rates had very little effect on the outcome. Analysis of the maximum hypothetical effect after a 30% reduction in nonstroke mortality from the state of “CHD” for 45 years resulted in increases in the male age-standardized stroke mortality rates of only 2.0% for ages 40 to 74 years and 2.6% for ages 75 to 84 years (Table 2).

**Discussion**

We have shown, using models, that changes in survival after CHD are unable to cause the 3% to 4% annual increase in stroke mortality rates required to eliminate the stroke mortality rate decline observed in the United States before 1991. We found that a 30% to 50% decrease in nonstroke mortality rates in those with CHD would lead to a small increase in stroke mortality rates and that a variety of factors affect the degree of this effect. However, we found no reasonable scenarios in which the increase in stroke mortality rate was >1% per year. This is because while increased numbers of CHD survivors increase the number of stroke deaths in the population, they also inflate the population size.

This is the first study to test this hypothesis. To examine the effect of improved CHD survival independently from the many other population changes, it was necessary to use models. To evaluate the robustness of our conclusions requires consideration of the sources of variance in the estimates, the limitations of this model type, and the degree to which the base model represents the situation in the United States before 1991.

Unfortunately, it is not straightforward to produce CIs for our outcomes because the variance needs to be combined from the 5 (dependent) regression analyses and the life tables, which also have internal dependence between transition types and across ages. Uncertainty is also found in the estimation of the degree of CHD survival improvement. Therefore, we chose to explore the impact of possible variance through a range of uncertainty analyses. We found that reasonable (and extreme) variations (based on estimated CIs) in the transition rates and in the degree of survival improvement did not alter the conclusions.

To estimate the annual effect of changes across all ages, we used life-table models. Other, more “2-dimensional” models, such as time series analyses, suffer from the difficulty of linking changes in one age group with outcomes in all future age groups, with appropriate time intervals taken into account. The life table does this automatically. An empirical approach closer to the life table would be to examine whether improved survival from CHD in middle age was linked to increased stroke mortality rates at older ages within a given cohort. However, it would still be difficult to exclude the effect of all other population changes affecting stroke mortality rates. One principal assumption of the life table is that within each category the population is homogeneous, including the markovian assumption that transitions are independent of prior history. This is appropriate for analyzing the effect of an increase in the proportion of the population with CHD. However, it is important to recognize that such a model does not evaluate possible changes in the stroke mortality risk of those with CHD associated with improvements in treatment.

One other potential limitation of this model is that the transition rates are estimated from a long follow-up period within the Framingham Heart Study during 1948–1998. We demonstrated that important factors affecting the degree of change in stroke mortality rates were the relative risk of stroke mortality after CHD and the prevalence of CHD. Therefore, insofar as these have not increased greatly over
time, we would expect models based on other, more recent, data to produce similar results. A CHD prevalence of 25% in 70-year-old men is similar to the prevalence of 26% observed for heart disease in 65- to 74-year-old men in the United States in 1989.11 However, for women the prevalence of CHD at age 70 years was only 14%, compared with a reported prevalence of 21%.12 While this may have led to underestimation of the increase in the stroke mortality rate in women, we would still not expect the increase to be greater than that seen for men. We also showed that the age-specific total and stroke mortality rates derived from the models were similar to American mortality rates around 1991, with stroke mortality rates more similar for the older than younger age groups. The similarity of the mortality rates arises because the majority of Framingham deaths occur later in the follow-up period and because the population is a selected, relatively healthy population.7,12 Those dying young were more likely to have died in the earlier follow-up period and therefore reflect 1990 rates less closely. A further limitation of the Framingham population is that it consists of primarily white Americans. However, since the decline in stroke mortality rates in the United States has been the greatest for the white population,4 our results make the tested hypothesis even less likely.

These results suggest that improvements in CHD survival may not be accompanied by as large an increase in the burden of disease in the elderly as has been feared.1 While the absolute number of stroke deaths (and before them incident strokes) will increase, the age-specific population rates will not change substantially. Furthermore, these results suggest that we may be able to restimulate the stroke mortality rate decline if it is not an unavoidable consequence of a desired treatment improvement. This coincides well with the data showing that the stroke mortality rate in the United States is once again declining (Figure 1). Further understanding of the factors driving the changes in stroke mortality rates is required if we are to optimize this decline.

We demonstrate that increases in the population with CHD cannot alone explain the higher than expected stroke mortality rates in the 1990s. The remaining possibilities are that stroke mortality rate changes were caused by factors other than improvements in survival with CHD (such as the worsening of prevalence and control of other risk factors) or that with improvements in CHD survival the risks of stroke mortality in the population with CHD also increased. Competing theories will have to explain why the slowing of the decline occurred around 1990 and was observed to a similar extent in all sex and age groups (Figure 1) and in widely different healthcare systems such as those in the United States, the Netherlands, and Sweden.13 They will also have to explain why the stroke mortality rates appear to be declining once again. An additional hypothesis fitting these conditions is the international introduction of a new therapy with immediate effects on stroke mortality. The introduction of thrombolytic therapy for myocardial infarction, with its associated hemorrhagic side effects,13 is one possible and testable candidate.

Acknowledgments
This study was supported by grants from the Netherlands Heart Foundation and the Netherlands Organization for Scientific Research. The authors would like to acknowledge the Framingham Heart Study coordinators for access to the original data set. The Framingham Heart Study is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the Framingham Heart Study Investigators. The Netherlands Epidemiology and Demography Compression of Morbidity Research Group (NEDCOM) includes, in addition to the listed authors, the following: F. Janssen, A. Kunst, C. de Laet, A.A. Mamun, W. Nusselder, and F.J. Willekens. The authors would like to thank Professor P.J. Koudstaal for critical review of the manuscript and Dr C. Looman for helpful discussions.

References
Over the last 3 decades, it has been rewarding to observe the steep decline in stroke mortality in many countries. In part, epidemiologists have attributed this decline to improvements in treatment of hypertension. Unfortunately, during the last several years some of these countries have experienced a slowdown of the declining trend. A plethora of studies have been published since attempting to explain this phenomenon.

It seems unlikely that flattening of the stroke mortality curve is an indication of a floor effect of primary and secondary prevention efforts. Rather, it seems more likely to be masking a relapse in hypertension prevention. Although screening for high blood pressure has increased in the 1980s, still only half of hypertensive patients received treatment from their physicians in the 1990s, and only 69% are even aware that they have elevated blood pressure. It is also possible that the increase in incidence of other stroke risk factors, such as diabetes, obesity, and alcohol drinking, may be contributing to the slowdown in mortality decline.

In the accompanying article, Peeters and colleagues focused on a more optimistic hypothesis. The investigators tested the hypothesis that the increase in CHD cases, secondary to improvement in survival, could lead to an increase in stroke mortality that will eliminate the previously observed decline. First, they estimated the annual rate of decline in stroke mortality in the United States during 1981–1991 on the basis of ICD-9 codes data from the Compressed Mortality Database. Probability models were used to identify whether improvements in survival after CHD could plausibly eliminate the stroke mortality rate decline observed. The results of the study showed that changes in survival after CHD are unable to cause the 3% to 4% annual increase in stroke mortality rate required to eliminate the stroke mortality rate decline observed in the United States before 1991. The investigators found no reasonable scenario in which the increase in stroke mortality rate was more than even 1% per year.

The study in the accompanying article has several methodological strengths. The multistate life-table analysis was based on age-specific transition rates derived from 46 years of follow-up of the Framingham Heart Study. The study therefore provided an accurate way to link changes in one age group with outcomes in future age groups. The strengths of this approach are in part offset by several limitations. The life-table analysis was based primarily on a white American homogeneous population and was not very reflective of changes in trends on a larger demographic scale. Interestingly, Howard et al found that the pattern of decline in stroke mortality in the United States was heterogeneous, with substantial variations among population groups, geographic areas, race, and sex.

As survival of patients with CHD improves, the pool of people at high risk of stroke is expected to increase. This hypothetically will increase stroke mortality rates. However, in the accompanying article Peeters et al provided enough evidence to reject this hypothesis; the only shortcoming in their argument was that they lumped all stroke subtypes into 1 group. Although cerebral infarct shares similar risk factors with CHD, its risk profile is different from intracerebral hemorrhage (ICH) or subarachnoid hemorrhage. Hypertension is among the few risk factors common to both ischemic stroke and ICH. In some reports, ischemic stroke risk factors were even found to have a protective effect on ICH. Such risk factors included history of cardiovascular disease, high cholesterol levels, being mildly overweight, smoking, and hormone replacement therapy.

The difference between stroke subtypes was not only limited to risk factor profiles. Lawlor et al demonstrated different mortality trends associated with different stroke subtypes. In the period 1981–1991, ischemic stroke mortality dropped from 18% to 11%, whereas ICH mortality increased only by 1% from 28%. The stroke subtype that had the largest mortality changes was the “ill defined cerebrovascular disease” subtype in which mortality increased from 53% in 1981 to 60% in 1999. This group included patients with ICD-9 codes 342, 344, and 436 to 438. The authors also demonstrated a 4-fold increase in the ratio of cerebral infarct to cerebral hemorrhage, from 0.5 in the 1930s to 2.0 by the 1990s. This ratio seemed to plateau in the 1990s. Interestingly, the combined decline in mortality rate for all stroke subtypes was 3% during 1981–1991. Similar rates have been reported by Peeters et al in the accompanying article and by others. This decline rate dropped to 0% during 1991–1999.

It appears that differences in risk factors and secular trends between stroke subtypes may be playing an important role in the flattening of stroke mortality curve. Hence, it is important to consider splitting rather than lumping the different subtypes of stroke to better understand epidemiological trends and long-term effectiveness of primary and secondary prevention efforts.

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References
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Editorial Comment
Decline in Stroke Mortality: Splitters and Lumpers

Over the last 3 decades, it has been rewarding to observe the steep decline in stroke mortality in many countries. In part, epidemiologists have attributed this decline to improvements in treatment of hypertension. Unfortunately, during the last several years some of these countries have experienced a slowdown of the declining trend. A plethora of studies have been published since attempting to explain this phenomenon.

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References
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Improvements in Treatment of Coronary Heart Disease and Cessation of Stroke Mortality Rate Decline

Anna Peeters, Luc Bonneux, Jan J. Barendregt and Johan P. Mackenbach
for the Netherlands Epidemiology and Demography Compression of Morbidity Research Group

Stroke. 2003;34:1610-1614; originally published online June 19, 2003;
doi: 10.1161/01.STR.0000078661.72578.0A

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