The Decline of Stroke*

Jack P. Whisnant, M.D.

SUMMARY Stroke mortality in the United States has declined since 1900. The mortality rates from stroke in Rochester, Minnesota, have declined 76% since 1950, and the primary cause for the declines is a decrease in the incidence of new cases of cerebral infarction and cerebral hemorrhage. There are no data on the incidence of stroke prior to 1945 to confirm the US mortality trend. The decline in incidence was noted in women 10–15 years before it was seen in men. Hypertension is the most important risk factor for stroke. Hypertension surveys have shown an increasing effectiveness of antihypertensive medication in lowering blood pressure, and the effect was noted earlier in women. Available evidence is consistent with the idea that treatment of hypertension is the only significant contributor to the decline of stroke.

Mortality From Stroke

For a number of years, the observation has been made that there has been a decline in the mortality from stroke for several decades. It is difficult, however, to be confident of the reliability of these data. Problems that can be identified include revisions in codes for the cause of death, changes in terminology and fashions of diagnosis, low rates of autopsy for confirmation, and low accuracy of diagnosis, particularly in differentiating the various categories of stroke. When a person is found dead or dies soon after the onset of altered consciousness without focal signs, death is often attributed to stroke. Although stroke may be the cause, it is much more likely to be a cardiac disorder.

Mortality from stroke in the United States actually has been steadily declining since 1900 (as far back as records are available) (fig. 1). This contrasts greatly with all cardiovascular mortality since 1900 (fig. 1). It should be noted that all of the codes for stroke in the International Classification of Disease (ICD) are also included in all cardiovascular mortality. The rates for all cardiovascular mortality were about the same in 1970 as in 1900, after having gone up and come back down. Since 1950, there has been a 33% decline for all races (both sexes). In 1900, stroke represented about 40% of the cardiovascular mortality. This percentage gradually declined until about 1950; since then it has been about 20%, and the relative vascular mortality has been about 80%.

It might be argued that strokes were overdiagnosed in contrast to other vascular events in the early part of the century, but that argument can neither be confirmed nor denied.

The decline in stroke mortality could be due to a decline in case fatality because of more effective early treatment or less severe cases or to a decline in the incidence of new cases of stroke or to both of these. It is necessary to have knowledge of the trends in mortality, in the incidence of new cases, and in case fatality during the same time and in the same population to make a judgment about the relative contribution of each to a change in mortality. If there is a decline in incidence or case fatality, then one has to examine what factors might have been altered to produce that effect.

We have used the population of Rochester, Minnesota, as a resource for the chronicologic study of stroke and other diseases. Medical practice for residents of Rochester and the surrounding area has been centered at the Mayo Clinic since the beginning of this century. Diagnoses made by physicians at the Mayo Clinic for patients seen in hospitals, as outpatients, or at home visits or diagnoses made at autopsies are all entered on a diagnostic master sheet in the patient's record. Diagnoses made for Rochester residents at other medical facilities in and around Olmsted County are also entered into a centralized population-based diagnostic index maintained at the Mayo Clinic. This file is continuously updated, and all diagnoses made for persons who are residents of Rochester can be identified through an automated record-retrieval system; this file ensures virtually complete case ascertainment for serious medical problems. From the perspective of stroke, the community has had a high degree of neurologic expertise since about 1915. The autopsy rate has been close to 60%, and there is a uniform method of assignment of cause of death because almost all death certificates are completed by a pathologist at the Mayo Clinic whether or not an autopsy has been performed.

We compared the mortality rates from stroke for the US white population with those for the population of Rochester. The mortality rate in each time period in Rochester was lower, and there was a more rapid decline in rates compared with the rates for the US white population (fig. 2). The difference between the changes in rates for the US white population and for Rochester was greatest in the oldest age group for example, for persons older than age 75, the stroke mortality rate for Rochester declined 54% from 1945–1954 to 1965–1974, whereas the rates for the US white
population increased by 3%. Whether these differences are due to the assignment of nonstroke deaths to stroke in the United States to a greater extent than in Rochester, particularly in older patients, is not known. Stroke mortality in Rochester in fact may be lower. There probably are no comparable communities in the country in which essentially the whole population receives care at a single medical center. However, what we consider better medical care does not always result in lower mortality.

Not many observations are concerned with the chronologic trends of the incidence of new cases of stroke, and no other population has been studied as long as that of Rochester; the first observations in Rochester were for 1945, and 1.5 million person-years of observation were recorded through 1979.5,6

Incidence of Stroke

The average annual incidence of new cases of stroke in Rochester has declined in every 5-year period since 1950. The decline for women has been rather gradual since 1955 (fig. 3). There was little change in the rate for men from 1945 through 1969, but since that time there has been a sharp decline — 44% during the last 10 years of observation. No other similar population samples include enough cases over a long enough period of time to permit a comparison. However, other population studies, such as the one in Hisayama, Japan,7 also have shown a declining incidence of stroke in more recent years over a relatively short period of time.

These points make it clear that, in Rochester, the factor of greatest importance in the declining mortality from stroke is the declining incidence of new cases. The decline in incidence and the decline in mortality in this population have both been over 50% since 1950. During this same period, the 30-day case fatality rate for cerebral infarction has been about 18–24% (except for the 5-year period 1950–1954, when it was 28%). The case fatality rate for cerebral infarction was only a little lower in 1975–1979 than it was in 1945–1949.8

This documentation of the primary factor in the decline of stroke mortality can be contrasted with that of myocardial infarction (MI) mortality in the Rochester population, as reported by Garraway et al9. There has been a sharp decline in mortality from coronary heart disease in the Rochester population since 1970, but this was observed to be associated with only minor changes in the incidence of MI. The decline seems to have been related more closely to a sharp decline in the 30-day case fatality, presumably due to better care immediately after the onset of MI.

Some observers have suggested that the decline in the incidence of stroke may simply be due to increasingly better diagnostic capability over time. We can reject that idea completely for the decline in Rochester, at least up until the mid-1970s, when computed tomography (CT) became readily available. On the basis of observations and conclusions in the medical record, the clinical diagnostic skills of earlier neurologists seem to have been as good as those of current neurologists. Regardless of any possible differences in clinical skills over time, the criteria for the selection of stroke diagnosis from information in the patient’s record were the same throughout the periods of observation. The only diagnostic procedure that might have made a difference was cervical-cerebral arteriography, and this

---

**Figure 1.** Annual rates per 100,000 population in the United States for all cardiovascular and stroke mortalities, age adjusted to 1940 US population, for 1900 through 1976. (From Whisnant JP: The role of the neurologist in the decline of stroke. Ann Neurol [in press]. By permission of the American Neurological Association. 14: 1–7, 1983)

**Figure 2.** Five-year average annual rates per 100,000 population for all stroke mortality, age and sex adjusted to 1960 US white population, for US white and Rochester, Minnesota, populations. (Modified from Anderson GL, Whisnant JP: A comparison of trends in mortality from stroke in the United States and Rochester, Minnesota. Stroke 13: 804–809, 1982. By permission of the American Heart Association.)

**Figure 3.** Five-year average annual incidence rates per 100,000 population for all first episodes of stroke in men and women in Rochester, Minnesota, age adjusted to 1960 US white population.
was performed too few times in any relationship to stroke to have made any difference. Further monitoring of the validity of diagnoses was also accomplished by an autopsy rate of nearly 60%. Even CT has had only a small effect in relation to the selection of stroke diagnosis; but it has had an important effect on the selection of the type of stroke.

Cerebral Hemorrhage

The points that have been made in reference to all stroke apply as well to cerebral infarction because that diagnosis accounts for 75–80% of strokes. The vital statistics for the United States do not allow us to make a judgment about trends of mortality from individual categories of stroke — for example, the term “cerebrovascular accident” has been commonly used as a cause of death and was coded as cerebral hemorrhage from 1950–1968; thereafter, it was coded as an ill-defined stroke. The change in coding results in a considerable distortion of the mortality trend for cerebral hemorrhage (fig. 4). With the use of Rochester 5-year average annual mortality rates after 1950, coded for all years to the 8th ICD revision for uniformity, the rates are much lower, but there is a considerable decrease in the mortality rates from 21/100,000 per year from 1950–1954 to 5 in 1970–1974 (76%) (fig. 5).

This decline in mortality is also related largely to a decrease in the incidence rates of new cases of primary intracerebral hemorrhage (PIH) (that is, hemorrhage not due to another systemic disease process). These rates are more difficult to interpret than are those for infarction because of much smaller numbers (thus more variability for 5-year periods) and because after the late 1950s the use of long-term anticoagulant treatment was common, which seems to have increased the overall rate by about 20% and, of course, by much more than that in the older age groups (in which intracerebral hemorrhage is more common).

If we exclude the patients who were receiving anticoagulant treatment at the time of their PIH, these data are compatible with a decrease from about 18/100,000 per year from 1945–1954 to about 6 from 1955–1959 through 1970–1974 (about a 65% decrease) (fig. 6).

We should appropriately consider the previous observations through 1974 to apply to clinically diagnosable PIH. CT head scans have changed that picture, more so than I would have predicted for a population sample. During 1975–1979, 88% of Rochester residents who had a diagnosis of PIH had a CT head scan or an autopsy or both. Most others were moribund early, so it is likely that we identified almost all of the patients with PIH during that period. We found, though, that we could not make comparisons with prior time periods on the basis of examination of cerebrospinal fluid for blood because the reliability of the CT scan for this diagnosis has resulted in this examination not being done at all in many such patients. The presence of headache or stiff neck was not noted consistently enough in patients who were frequently unconscious to be used for comparison. Level of alertness at the time of first medical attention was the only reliable
factor from which a judgment could be made. From 1945-1974, 4.5% of Rochester residents diagnosed as having PIH were alert (range, 0% to 8% for the 6 quinquennia). From 1975-1979, 23% were alert. This undoubtedly means that before the development of CT, we mislabeled the small intracerebral hemorrhages that did not cause altered consciousness and did not produce blood in the cerebrospinal fluid. If we assume that we should have had 23% alert from 1945-1974, a 24% increase in the number of new cases of PIH in this 30-year period would have resulted.10

The identification of all the smaller hemorrhages that were previously called infarcts dramatically alters the probability of survival following the diagnosis of intracerebral hemorrhage. The 30-day survival was only 8% before 1974 and was 44% after 1974, and the 1-year survivals were 5% and 31%, respectively. There were even larger differences if we consider only those persons who were not receiving anticoagulant treatment at the time of the PIH.10

Subarachnoid Hemorrhage
Subarachnoid hemorrhage, contrary to other categories of stroke, showed an increasing mortality in the United States from 1950-1970.9 Since then, there has been a slight decline. We cannot see these trends in the Rochester population (fig. 7). The morality rates in Rochester seem to be the same for 1950-1974, and the incidence rates of new cases show no significant change during this time.11,12 Thus, subarachnoid hemorrhage does not contribute to the decline of stroke, so it will not be considered further.

Risk Factors
The important question now is what has caused the decline in the incidence of stroke in Rochester. The magnitude of decline is such that it would be comparable to changes in disease patterns affected in the past by factors such as improved sanitation, but that concept does not apply here. We may consider the possible effect of influencing risk factors that have been shown by the Framingham study and other studies to be associated with an increased incidence of stroke.13,14 We do not have sampling of the Rochester population over time to determine whether the trends of any of the risk factors in the population are compatible with having had an effect on stroke occurrence. The risk factors I should like to consider are transient ischemic attacks, carotid stenosis, serum lipids, cigarette smoking, cardiac disease, diabetes mellitus, and hypertension.

Transient Ischemic Attacks (TIA)
Some observers would think that it is more reasonable to consider TIA as the onset of the disease rather than as a risk factor for stroke, but it is clear that the risk of stroke in patients with TIA is high, perhaps 6 or more times the rate in a normal population.15 However, in the Rochester population, less than 10% of the patients had TIA prior to their first cerebral infarction.16 A similar percentage has been observed in other population studies.17 This is in contrast to patients with cerebral infarction referred to the Mayo Clinic from outside Rochester, in whom nearly 35% have had a prior history of TIA, a figure comparable to that from other medical centers.18 This points out the extent of sampling bias that may exist among referred patients.

Even though the risk of stroke in patients with TIA is relatively high, the prevalence of TIA in the population is rather low. In a 1960 cohort study of the Rochester population involving nearly 1,900 persons older than age 50 who were followed for 13 years, the calculated percentage of strokes that could be attributed to TIA was 9% (Schoenberg BS, Whisnant JP, Lillenfeld A: Unpublished data).

In a multivariate analysis of the patients with TIA in Rochester over a 15-year period with the use of the variables noted in table 1, the factors noted were determined to be present or absent at the time of or prior to the onset of the first TIA. None of these significantly affected survival. Only the level of diastolic blood pressure significantly affected stroke occurrence—that is, patients with lower blood pressures had fewer strokes.19

It is not the purpose of this paper to consider management of TIA. It is sufficient to state that there is no consensus concerning the most effective treatment, and some physicians would hold that there is no effective treatment. Even the most therapeutically optimistic among us would not suggest that any medical or surgical treatment has reduced the frequency of stroke occurrence.

---

**Table 1 Variables Used in Multivariate Analysis of Patients With TIA**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Coefficient</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anticoagulants (administered &lt; 60 days after onset of TIA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic blood pressure (continuous variable)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina pectoris</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac arrhythmia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

following TIA by more than 50%, even among those patients who sought medical care promptly. It is unlikely then that the treatment of TIA has played a substantial role in the decline of stroke.

**Carotid Stenosis**

Could we have prevented a large percentage of strokes by identifying carotid stenosis, the anatomic prelude to TIA in some patients? Such patients certainly can be safely detected by screening for asymptomatic carotid bruits, by noninvasive studies such as oculopneumoplethysmography and ultrasound, or by intravenous digital subtraction angiography. A stroke rate of about 2½% per year in patients with asymptomatic carotid bruits was observed in a prospective study (Sandok BA, Whisnant JP: Unpublished data). If we assume that the long-term risk of stroke after endarterectomy in patients with asymptomatic carotid stenosis would be similar to that for patients with TIA after endarterectomy, we then could be comparing a 2½% rate per year for untreated patients with a 2% rate per year for patients treated with endarterectomy, in addition to the initial surgical morbidity and mortality. These considerations make it seem unlikely that treatment of asymptomatic carotid stenosis has had any impact on stroke incidence, and it is not likely to have much impact in the future.

**Serum Lipids**

The relationship of elevated levels of serum lipids to ischemic stroke has not been consistent. In fact, a negative relationship of stroke to low-density lipoprotein cholesterol was noted in the Framingham study. Even with special efforts, only modest decreases in the level of serum cholesterol have been demonstrated in population samples over recent years. Because of the inconsistent relationship of serum lipids to stroke, it is doubtful that a decrease in cholesterol levels has had any significant effect on stroke incidence.

**Cigarette Smoking**

Although cigarette smoking is clearly related to the rate of development of coronary heart disease, it has only a weak association with stroke. The peak year for overall consumption of cigarettes per person in the United States was 1963 (fig. 8). The percentage of men smokers has sharply declined since then, and the percentage of women smokers has remained about the same (fig. 8); but a fairly sharp increase has occurred among young women. Also, the Framingham study showed that there were more women smokers aged 45-69 in 1968 than in 1956. These trends are not consistent with the idea that changing smoking habits had a significant effect on the declining incidence of stroke, which occurred much earlier in women than in men.

Pathologically, carotid artery atherosclerosis is parallel in severity to coronary artery disease. It is certainly possible that strokes that are directly or indirectly related to atherosclerotic carotid stenosis or occlusion are affected by cigarette smoking, but such an effect is not apparent in the overall picture.
son-years of observation; the relative risk was 1.1.24
There are no data concerning the temporal trends of
control of diabetes mellitus, but with such a low rela-
tive risk in nonhypertensive persons with diabetes it
does not seem likely that treatment of diabetes has had
much overall effect on stroke occurrence.

Hypertension

Hypertension has long been recognized as the most
important risk factor for stroke (both ischemic stroke
and PIH). This is not simply an increased risk of stroke
over a certain level of blood pressure, but it is an ever
increasing risk from the lowest to the highest levels of
blood pressure (fig. 9) — this is true in both sexes and
at all ages.14

Not only does the presence of elevated blood pres-
sure represent a risk of stroke, but elevated blood pres-
sure is a highly prevalent condition (perhaps as preva-
lent as 30% or more in the population over the age of
40). The 1960 cohort study by Schoenberg et al. in the
Rochester population indicated that the risk of stroke
attributable to hypertension was about 70% (Schoen-
berg BS, Whisnant JP, Lillenfeld A: Unpublished
data). This rate involved overlapping risks and so is
not as precise as one would like, but it is far higher than
any other known risk (for example, when compared with
the 9% that was noted for transient ischemic attacks).

Even though systolic and diastolic pressures usually
are closely correlated, the risk of stroke is at least as
high in systolic hypertension as in diastolic hyperten-
sion. The Framingham study showed this effect of
systolic hypertension in the absence of diastolic hyper-
tension.25 A study of eight different communities in
Japan also showed a large and significant correlation
between mean levels of systolic blood pressure and
incidence rates for stroke in all age groups studied;26
but this was not true for diastolic pressures in that
study. Systolic hypertension is increasingly prevalent
with age and more so than is diastolic pressure. It thus
has an ever-increasing opportunity to contribute to
stroke occurrence.

It is difficult to compare studies from any two or
more sources to determine the prevalence of hyperten-
sion. It is even more difficult to find data from the era
preceding the use of effective antihypertensive drugs
for comparison with more recent surveys. A study of
nearly 11,000 persons older than age 15, published in
1939, showed a prevalence of hypertension (greater
than 140/90 mm Hg) of only 7–8% in men and wom-
en.27 This may be compared with 15% in a national
health survey in 1960–196228 and with 12.5% in a
study in Charlottesville, Virginia, in 197429 (blood
pressure greater than 160/95 mm Hg was the cutoff) —
a time when many of those surveyed were being treat-
ed for hypertension. It is not possible to determine
whether the prevalence really increased over this inter-
val because of the lack of opportunity for satisfactory
comparisons.

It is clear that the prevalence of hypertension has
decreased during the period of effective antihyperten-
sive treatment. In the Framingham study, the percent-
age of persons who were hypertensive decreased from
1954–1968 in women of all age groups, but there was
relatively little change in men except in the oldest age
group.22 Also, the percentage of persons taking antihy-
pertensive drugs increased markedly from 1956–1968,
and the increase was much greater in women. More
recent studies in Chicago, with observations as recent
as 1977, also showed a decreasing prevalence of hy-
pertension.30

It is also clear that the proportion of persons with
hypertension being identified and effectively treated is
increasing (table 2). The earliest observations were in
1960–1962 when 16% of patients with hypertension
were treated and controlled.30 Only 11% were treated
and controlled in Chicago in a 1967–1971 survey of an
industrial population.31 Most impressive was a com-
unity-wide screening and education effort in Chicago
during which the proportion of persons with hyperten-
sion treated and controlled went from 59% to 73% in 1
year.30 The percentage of persons with unidentified
hypertension or with known but untreated hyperten-
sion decreased during the same years of observation
(table 2). The surveys indicated that more women than
men took antihypertensive medication.

What is the evidence that increasing treatment and
control of hypertension has had an effect on stroke
occurrence? Randomized treatment trials were done
with two different levels of blood pressure in a Veter-
ans Administration cooperative study.33,34 There was a
Public Health Service hospitals cooperative trial35 and
there were trials in Norway,36 Sweden,37 and Austra-
lia,38 and one is in progress in the United Kingdom.39
None of these has shown a significant difference in
stroke occurrences between treated patients and con-
controls, but some did show significant differences in the occurrence of all vascular events. In each trial, there were small numbers of patients with stroke (predominantly in untreated patients).

It was not until the results of the Hypertension Detection and Follow-Up Program were published for stroke occurrence in 1982 that it was clear that systematic treatment of patients with hypertension resulted in significantly fewer strokes than in patients referred to their own physicians for routine care. This required nearly 11,000 persons in the study for 5 years to detect an overall stroke incidence of 1.9% in stepped-care patients compared with 2.9% in referred-care patients. This represented a 35% reduction in all strokes and a 44% reduction in fatal strokes. Total stroke rates were lower in the stepped-care group than in the referred group in all race and sex categories. The greatest reductions were in black women and in white men. An observation of particular interest to us was that the reduction of stroke was greatest in the oldest age group (60–69 years), in which there was a 45% reduction.

Do these observations concerning the effect of treatment of hypertension square satisfactorily with US stroke mortality trends? Not very well! There was little effective treatment available for hypertension prior to 1955; yet the rate of decline in mortality from stroke in the United States was about the same from 1920 through 1970, and a greater rate of decline occurred after 1970 (fig. 1). The most one could suggest from these trends would be a contribution to the increased rate of decline.

It has been suggested that decreased salt intake and the effect that would have had on the prevalence of hypertension may have been an important factor in the decline of mortality in earlier years. Salt intake in western countries certainly declined because the introduction of refrigerators and freezers made it unnecessary to preserve food with salt. Data concerning salt intake and hypertension are not uniform, but some studies do show a relationship between the amount of salt consumed and the level of systolic blood pressure (that is, the more salt consumed, the higher the level of systolic blood pressure). Whether there is a genetically determined sensitivity to salt in regard to the effect on blood pressure, as has been demonstrated in rats, is not known.

Although there could have been some other effect on hypertension prior to effective medical treatment, we do not have data to indicate that hypertension was more prevalent in the several decades before antihypertensive treatment. In fact, what evidence there is would indicate that it may have been less prevalent.

There may have been some factor other than changes in blood pressure to account for the earlier decrease in stroke mortality or, just as likely, the US stroke mortality trends could be wrong — that is, there may have been more mixing of types of cardiovascular mortality, and stroke or apoplexy may have been used more frequently than was appropriate. It is fairly clear that a similar practice occurred in Japan in more recent years.

The observations on the effect of treatment of hypertension fit rather well with the trend of incidence of stroke in Rochester from 1945. It would be easy to interpret that the rate in women (fig. 3) were stable from 1945–1954 and have gradually declined since then through 1979 and that the rates in men have shown only a slight decline through 1969 and a sharp rate of decline after that. The trend in rates for PIH fits very well with the much higher and stable rates from 1945 through 1954 and significantly lower rates after that (fig. 6). It is of interest to note here that among Rochester residents older than age 65 who died after PIH in the latest 5-year period of our observations, in about half of the patients the cause for the hemorrhage was amyloid angiopathy. Thus, pathologically, hypertensive vascular disease is now playing a lesser role in cerebral hemorrhage in older patients compared with earlier periods.

Do not have incidence rates for strokes prior to 1945, nor am I aware of such information from other studies. Such data would provide more years of observation prior to the availability of antihypertensive medication and might provide a better base of information to judge the effect of treatment of hypertension in later years.

Antihypertensive treatment was used rather frequently in Rochester after 1955, and we have shown that it was used with increasing frequency from that time through 1974 in patients with stroke. We do not yet have data to indicate a decreasing prevalence of hypertension or an increase in use of antihypertensive medication in Rochester as a whole, and we have not yet demonstrated that the identification and treatment of hypertension is improving.

---

### Table 2: Hypertension Surveys (Blood Pressure ≥ 160/95 mm Hg)

<table>
<thead>
<tr>
<th>Survey</th>
<th>No. with hypertension</th>
<th>Previously unknown, %</th>
<th>Known but untreated, %</th>
<th>Treated, but diastolic pressure ≥ 95 mm Hg, %</th>
<th>Treated and controlled, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Health Survey, 1960–1962</td>
<td>1,214</td>
<td>43</td>
<td>22</td>
<td>19</td>
<td>16</td>
</tr>
<tr>
<td>Chicago, 1967–1971</td>
<td>4,625</td>
<td>59</td>
<td>16</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>CHEC, 1973–1975</td>
<td>220,217</td>
<td>28</td>
<td>17</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>Chicago, 1976</td>
<td>35,238</td>
<td>12</td>
<td>17</td>
<td>17</td>
<td>12</td>
</tr>
<tr>
<td>Chicago, 1977</td>
<td>52,911</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
</tbody>
</table>

*Community hypertension evaluation clinic program.*
of hypertension in men have lagged behind those in women, as I have indicated from other studies. We should have had community surveys to examine that.

I would like to call attention to the following points that have been made in regard to the effect of treatment of hypertension.

1. Hypertension has been clearly demonstrated to be a powerful risk factor for stroke.
2. The incidence rates for stroke in Rochester have declined since 1955 for women (men lag far behind women in terms of the start of the decline), and the decline in the incidence rates is the primary factor in the decline of the mortality rate for stroke.
3. Blood pressure decreased over time in age groups of the Framingham population sample, and the effect was earlier in women.
4. In hypertension surveys, treatment of hypertension has been increasingly effective in lowering blood pressure, and the effect was noted earlier in women than in men.
5. Stroke occurrence decreased in the Hypertension Detection and Follow-Up Program in 5 years in carefully treated patients compared to referred-care patients, and differences were comparable in men and women when both were managed similarly.
6. Finally, although mortality from stroke in the United States has declined since 1900, there are no data on the incidence of new cases of stroke prior to 1945 to confirm the mortality trend.

All of these points collectively support the idea that treatment of hypertension is the major contributor to the decrease in stroke incidence and mortality. The available evidence would be consistent with the idea that it is the only significant contributor to the decline. Hypertension has been prevalent enough and the effect of treatment would have been powerful enough to account for the extent of the decline.

This is not to say that management of other powerful risk factors such as transient ischemic attacks and cardiac disease, if clearly effective, could not have some effect. However, TIA is not prevalent enough, and the effect of medical and surgical treatment at present, balanced against risk of treatment, is not potent enough to have much effect on stroke incidence. Too many patients are not seen soon enough after the onset of TIA for present medical and surgical treatment to have maximal opportunity for benefit. The relatively stable incidence rates for MI make it unlikely that management of this disorder has had much of a role in the decreasing incidence of stroke, and we have too little data about temporal trends of other cardiac disorders to make a judgment. It is not likely that the prevalence of these other cardiac conditions is high enough to have had much effect on stroke occurrence.

We still have plenty of mileage left in stroke prevention in regard to recognition, treatment, and control of hypertension, probably more so in black populations; and systolic hypertension deserves more attention than it has received in terms of effective management. We also must increase our efforts toward more effective medical or surgical management of TIA and of cardiac disorders and aim at a more satisfactory demonstration of the effect of this management for prevention of stroke.

References
23. Kannel WB, Thom T. Implications of the recent decline in cardio-
adequate progress in his research. A third mechanism investigator must develop internal mechanisms to assure the responsibility of government and the principal investigator. The conduct of the investigation is not awarded to an institution or an individual on behalf of a designated goal. A second mechanism would be center programs targeted toward stroke and trauma to the nervous system. The first is the use of contracts which are task-oriented projects. The former allows an individual to pursue his own research questions, while the latter requires a group of investigators to coordinate activities with one another and proceed together toward a common goal. Do basic scientists achieve more when functioning independently (individual grantees) or in conjunction with clinical investigators (program projects)? Does research accomplishment fare better with grantees directed or with the federally supervised contract mechanism?

Federal Funding for Research in Stroke and Trauma — A Clinical Investigator’s Viewpoint

JAMES F. TOOLE, M.D., AND WILLIAM W. TOOLE

OUR FEDERAL BIOMEDICAL RESEARCH ESTABLISHMENT utilizes three mechanisms for funding research designed to increase knowledge related to stroke and trauma to the nervous system. The first mechanism would be center programs targeted toward a designated goal. A second would be individually initiated research. Both of these are conditional gifts awarded to an institution or an individual on behalf of an investigator. The conduct of the investigation is not the responsibility of government and the principal investigator must develop internal mechanisms to assure adequate progress in his research. A third mechanism is the use of contracts which are task-oriented projects with clearly defined goals and timetables supervised by federal employees. Compliance with specifications is the responsibility of government officials.

Which of these three funding mechanisms returns most for the taxpayer’s investment — a large number of small awards to individuals pursuing endeavors which spring from their own initiative, or fewer large awards made to teams which have been assembled for the purpose of answering carefully defined hypotheses?

The former allows an individual to pursue his own research questions, while the latter requires a group of investigators to coordinate activities with one another and proceed together toward a common goal. Do basic scientists achieve more when functioning independently (individual grantees) or in conjunction with clinical investigators (program projects)? Does research accomplishment fare better with grantees directed or with the federally supervised contract mechanism?
The decline of stroke.
J P Whisnant

doi: 10.1161/01.STR.15.1.160

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/15/1/160

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Stroke* is online at:
http://stroke.ahajournals.org/subscriptions/