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% of the cardiovascular mortality. One might argue 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...
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 al.9 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
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 the 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 hemor-
rhages 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 cate-
gories of stroke, showed an increasing mortality in the
United States from 1950–1970.4 Since then, there has
been a slight decline. We cannot see these trends in the
Rochester population (fig. 7). The mortality rates in
Rochester seem to be the same for 1950–1974, and the
incidence rates of new cases show no significant
difference during this time.11 12 Thus, subarachnoid hem-
orrhage 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 compar-
able 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 asso-
ciated 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, car-
diac disease, diabetes mellitus, and hypertension.

Transient Ischemic Attacks (TIA)

Some observers would think that it is more reason-
able 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 pa-
tients had TIA prior to their first cerebral infarction.16
A similar percentage has been observed in other popu-
lation 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
some other medical centers.18 This points out the ex-
tent 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 deter-
mined 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 man-
agement 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 effec-
tive treatment. Even the most therapeutically optimis-
tic among us would not suggest that any medical or
surgical treatment has reduced the frequency of stroke

<table>
<thead>
<tr>
<th>TABLE 1 Variables Used in Multivariate Analysis of Patients With TIA</th>
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<td>Anticoagulants (administered &lt; 60 days after onset of TIA)</td>
</tr>
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<td>Diastolic blood pressure (continuous variable)</td>
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<td>Myocardial infarction</td>
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<td>Angina pectoris</td>
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<td>Valvular heart disease</td>
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<td>Cardiac arrhythmia</td>
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<td>Congestive heart failure</td>
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FIGURE 7. Mortality rates per 100,000 population for sub-
arachnoid hemorrhage for white men and women in the United
Age adjusted to 1960 US white population. (From Whisnant JP,
Phillips LH II, Sundt TM Jr. Aneurysmal subarachnoid hemor-
rhage: timing of surgery and mortality. Mayo Clin Proc 57:
471–475, 1982. By permission.)
following TIA by more than 50%, even among those patients who sought medical care promptly. It is unlikely 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,20 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.21 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.19 The peak year for overall consumption of cigarettes per person in the United States was 196322 (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.22 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.

**Cardiac Disease**

There is clearly an increased risk of stroke in persons with cardiac disease. The combination of the Framingham study and a 1960 study of a Rochester cohort indicate that with coronary heart disease the relative risk is 3 — that is, stroke is 3 times more likely to develop in a person with coronary heart disease than in a person without that disorder. The relative risk for persons with atrial fibrillation is 6, for those with congestive failure the risk is 5, and for those with valvular heart disease the risk is 2.23 (Schoenberg BS, Whisnant JP, Lilienfeld A: Unpublished data).

We do not have data on the prevalence of these conditions in the population, except for coronary heart disease, which is the most prevalent. I have already indicated that in Rochester there has been a relatively small decline in incidence rates of coronary heart disease. The fact that there are more survivors of acute MI might allow even more opportunity for stroke to be associated with coronary heart disease.

We do not have any data on the temporal trends of the incidence or prevalence of valvular heart disease, congestive failure, and atrial fibrillation; nor do we have data concerning the effect of treatment of these conditions over time, which might have had an influence on stroke occurrence. Except for coronary heart disease, the available data do not allow us to make a judgment about any possible effect on stroke occurrence that might have been related to the management of these other cardiac disorders.

**Diabetes Mellitus**

The fact that diabetes mellitus accelerates the atherosclerotic process is widely recognized. There is considerable variation in the incidence of ischemic stroke among persons with diabetes, depending on other risk factors that are present; but the Framingham study estimated the relative risk to be only about 2 after adjustment for differences in associated cardiovascular risk factors.21 Data from a diabetic cohort in Rochester indicated that nonhypertensive persons with diabetes had no significant increase in stroke occurrence compared with persons without diabetes in over 7,000 per-

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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 relative 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 pressure represent a risk of stroke, but elevated blood pressure is a highly prevalent condition (perhaps as prevalent 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% (Schoenberg 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 hypertension. The Framingham study showed this effect of systolic hypertension in the absence of diastolic hypertension.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 hypertension. 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 women.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 treated for hypertension. It is not possible to determine whether the prevalence really increased over this interval because of the lack of opportunity for satisfactory comparisons.

It is clear that the prevalence of hypertension has decreased during the period of effective antihypertensive treatment. In the Framingham study, the percentage 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.23 Also, the percentage of persons taking antihypertensive 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 hypertension.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.31 Only 11% were treated and controlled in Chicago in a 1967–1971 survey of an industrial population.31 Most impressive was a community-wide screening and education effort in Chicago during which the proportion of persons with hypertension treated and controlled went from 59% to 73% in 1 year.30 The percentage of persons with unidentified hypertension or with known but untreated hypertension 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 Veterans Administration cooperative study.33,34 There was a Public Health Service hospitals cooperative trial35 and there were trials in Norway,36 Sweden,37 and Australia,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-

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**Figure 9.** Average annual incidence rates for cerebral infarction in men and women aged 50-59 at entry according to systolic blood pressure (Framingham study at 22 years follow-up). (Data from Dawber TR, Wolf PA, Colton T, Nickerson RJ.)14
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 the 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 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 1945 through 1954 and significantly lower rates after that (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 rates 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.

We 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, or systolic blood pressure ≥ 160/95 mm Hg results in a lower risk of stroke.

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<td>Treated and controlled, %</td>
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<td>11</td>
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*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 than in men.

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.

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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 grantee directed or with the federally supervised contract mechanism?
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