Incidence of Transient Ischemic Attack in Rochester, Minnesota, 1985–1989

Robert D. Brown, Jr, MD; George W. Petty, MD; W. Michael O’Fallon, PhD; David O. Wiebers, MD; Jack P. Whisnant, MD

Background and Purpose—There is scant information available on the incidence of transient ischemic attack (TIA) in a defined population. This study defines incidence rates of first TIA and subtypes of TIA during 1985–1989 and compares the incidence to that obtained from a 1960–1972 cohort study.

Methods—Medical records of all residents of Rochester with potential diagnosis of TIA during 1985–1989 were screened to determine whether the case met the criteria for TIA. All available data were used to determine the vascular distribution of the TIA. Average annual age- and sex-adjusted incidence rates were calculated for 1985–1989, and results were compared with incidence rates determined in a Rochester-based 1960–1972 cohort study.

Results—Two hundred two cases of first TIA or amaurosis fugax occurred among Rochester residents during 1985–1989. The age- and sex-adjusted incidence rate for any TIA was 68/100,000 population. Incidence of amaurosis fugax was 13/100,000; anterior circulation (cerebral) TIA, 38/100,000; and vertebrobasilar distribution TIA, 14/100,000. Rates were similar to those determined from a 1960–1972 cohort study.

Conclusions—The incidence rate of TIA is 41% that of stroke incidence. TIA incidence in Rochester, Minn, is higher than has been previously reported for other sites throughout the world. Although comparison with prior time periods is difficult because of ascertainment issues, it appears that there has been no significant change in TIA incidence since the decade of the 1960s or earlier. This suggests that the most common mechanism for TIA (atherosclerosis) has not changed in prevalence, nor have risk factors leading to this mechanism. (Stroke. 1998;29:2109-2113.)

Key Words: cerebral ischemia, transient ■ cerebrovascular disorders ■ epidemiology ■ incidence

Subjects and Methods

The Rochester Epidemiology Project Medical Records Linkage System provides resources to identify nearly all new cases of stroke and TIA in a community.14 Virtually all medical care in the community is supplied by Mayo Clinic and its 2 affiliated hospitals or by Olmsted Medical Center, a smaller group practice, and its hospital. All medical diagnoses made for a resident of Rochester are entered on a master sheet in the patient’s medical record, which is then entered into a central computer index. The index includes care for Rochester residents provided by other medical practices in surrounding communities, the University of Minnesota, and the Veterans’ Administration Hospital in Minneapolis. The medical record includes all inpatient and outpatient data, emergency department visits, nursing home care, and autopsy or death certificate information.

The medical records of all residents of Rochester who had a diagnosis of stroke, TIA, or a diagnosis that could be mistaken for stroke or TIA during the 5-year period from January 1, 1985, through December 31, 1989, were screened by a neurologist or trained nurse abstractor under the supervision of a neurologist to determine whether the case met the criteria for TIA. Case ascertainment through the medical record linkage system was similar to that from a cohort study for the diagnosis of stroke.1 For the diagnosis of TIA, the addition of several diagnostic rubrics that increased the number of patients screened, as well as the ascertainment of TIA cases simultaneously with stroke, should have resulted in a high level of completeness of case ascertainment.

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From the Division of Cerebrovascular Diseases and Department of Neurology (R.D.B., G.W.P., D.O.W., J.P.W.), and Department of Health Sciences Research (W.M.O’F., D.O.W., J.P.W.), Mayo Clinic and Mayo Foundation, Rochester, Minn.

Reprint requests to Robert D. Brown, Jr, MD, Division of Cerebrovascular Diseases and Department of Neurology, Mayo Clinic, Rochester, MN 55905.

E-mail brown.robert@mayo.edu

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All determined cases had verification of residence based on information from city and county directories and early medical records. The study excluded patients who had stable ischemic stroke before the first TIA occurred. The following definitions were used for TIA and TIA arterial territory designation:

**Transient ischemic attack (brain).**
A TIA was defined as an episode of focal neurological symptoms with abrupt onset and rapid resolution, lasting <24 hours and due to altered circulation to a limited region of the brain. Transient visual disturbances associated with retinal ischemia were defined as transient monocular blindness (TMB) (see below). Transient symptoms such as syncope, unexplained unconsciousness, and dizziness or wooziness were excluded unless associated with other symptoms of brain stem ischemia. Symptoms such as vertigo, dysarthria, or diplopia, which occurred in isolation without other symptoms of brain stem ischemia, were not considered TIA. Focal symptoms associated with migraine were also excluded. Patients with clinical symptoms consistent with TIA but with CT or MRI evidence of a cerebral infarct in an appropriate distribution were considered to have had TIA.

**Amaurosis fugax (transient monocular blindness).**
An episode of transient monocular visual disturbance with abrupt onset and rapid resolution, lasting <24 hours and due to altered circulation to the retina was considered an episode of AF (TMB). The patient may have total or partial loss of visual acuity affecting all or part of the visual field of the affected eye. Visual symptoms associated with migraine were excluded.

**Carotid system.**
The following symptoms were considered to represent transient cerebral ischemia in the carotid system: motor or sensory symptoms limited to 1 side of the body, aphasia or dysphasia, retinal ischemia, or any combination of these symptoms.

**Vertebrobasilar system.**
The vertebrobasilar system was considered the source of transient cerebral ischemia when the following symptoms occurred: motor or sensory symptoms or both on both sides in the same attack; ataxia of gait or clumsiness of the extremities on both sides; diplopia; dysphasia; bilateral homonymous hemianopsia; or any combination of the symptoms. Unilateral motor or sensory symptoms were not defined as vertebrobasilar system unless there were associated symptoms indicating brain stem or cerebellar ischemia or unless the symptoms were bilateral. Vertigo and diplopia occurring with other appropriate symptoms were considered vertebrobasilar distribution ischemia. Dysarthria occurring alone was considered uncertain location. Homonymous hemianopsia occurring alone was considered vertebrobasilar system. Symptoms occurring in both carotid and vertebral distributions were combined with the “uncertain” distribution.

To exclude persons who may have moved to Rochester to facilitate treatment or diagnosis of an existing disorder, cases were eligible only if the person was a resident of Rochester for ≥1 year before TIA.

**Statistical Methods**
In the calculation of incidence rates, the entire population of Rochester was considered to be at risk. Denominator age- and sex-specific person-years were estimated from decennial census data for Rochester with linear interpolation between census years.

To obtain some sense of variability, it was assumed that, given a fixed number of person-years, the number of cases follows a Poisson distribution. This allowed for the estimation of SEs and the calculation of 95% CIs for the incidence rates. Overall rates were directly age- and/or age/sex-adjusted to the population distribution of US whites in 1980. The SEs and CIs for the adjusted rates were based on the same assumption as above.

The relationships of crude incidence rates to age and sex were assessed with the use of generalized linear models with a log-link function that assume a Poisson error structure. Such models fit the natural logarithms of the crude incidence rates as linear combinations of sex and age group. Model fit was assessed with the use of the model deviance, which is a measure of how well the observed and predicted incidence rates agree. The model fits the data reasonably well if the expected value of the deviance is approximately equal to its degrees of freedom.

The incidence rates for TIA determined for 1985–1989 were compared with rates obtained from a similar study covering 1955–1979 and with a cohort study performed in Rochester, Minn, for 1960–1972. For 1955–1979, the ascertainment of TIAs may not have been complete for several reasons, leading to incidence rates reported for 1955–1979 and 1960–1972 using the medical record linkage system being lower than those detected for the cohort study for 1960–1972.

During 1955–1979, TIA cases were ascertained separately from the ascertainment effort for the stroke incidence studies. This led to some cases of TIA being missed, particularly those with stroke after TIA with the stroke occurring before diagnosis of the TIA, or when the case indexed stroke was actually TIA. Ascertainment of TIAs performed simultaneously with ascertainment of stroke in 1985–1989 should have eliminated this problem. A previous study demonstrated that when the cases detected in the cohort study were compared with the cases determined by medical record linkage for the same time period (1960–1972), some of the TIA cases were missed when the medical record linkage system was used. An additional diagnostic coding rubric that would have led to detection of some of these cases missed by medical record linkage was used for cases reviewed during the 1985–1989 quinquennium. These 2 practices (ascertaining TIA cases concurrently with cerebral infarct cases and adding an appropriate rubric) should have eliminated essentially all of the cases previously “missed” by the medical record linkage method. The diagnostic coding rubrics now used to ascertain TIA/AF cases are recorded in Table 1.

This study was approved by the Mayo Clinic Institutional Review Board.

### Results
We identified 202 residents of Rochester who had their first episode of transient cerebral or retinal ischemia during 1985–1989. The age- and sex-adjusted (to the 1980 US white population) incidence rate was 68/100 000 population. The age-adjusted rate was somewhat higher in men (76/100 000; 95% CI, 59.5 to 92.6) than in women (62/100 000; 95% CI, 50.1 to 73.7), although the difference was not statistically significant.

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**TABLE 1. ICD-9-CM Rubrics That Yield Patients With TIA**

<table>
<thead>
<tr>
<th>Primary rubric</th>
<th>Secondary rubrics</th>
</tr>
</thead>
<tbody>
<tr>
<td>435—Transient cerebral ischemia</td>
<td>Nonhemorrhagic stroke</td>
</tr>
<tr>
<td></td>
<td>433—Oclusion and stenosis of precerebral arteries</td>
</tr>
<tr>
<td></td>
<td>434—Oclusion of cerebral arteries</td>
</tr>
<tr>
<td></td>
<td>436—Acute but ill-defined cerebrovascular disease</td>
</tr>
<tr>
<td></td>
<td>437—Other and ill-defined cerebrovascular disease</td>
</tr>
<tr>
<td></td>
<td>438—Late effects of cerebrovascular disease</td>
</tr>
<tr>
<td></td>
<td>Other</td>
</tr>
<tr>
<td></td>
<td>368.12—Transient vision loss</td>
</tr>
<tr>
<td></td>
<td>781.4—Transient limb paralysis</td>
</tr>
<tr>
<td></td>
<td>784.3—Aphasia</td>
</tr>
</tbody>
</table>

ICD-9-CM indicates International Classification of Diseases, Ninth Revision, Clinical Modification.
Age- and sex-specific incidence rates for TIA are displayed in Table 2. In general, rates increased with age until age >85 years, when rates were slightly lower in men and women.

The age-adjusted incidence rates of TIA by arterial territory are reported in Table 3. The age- and sex-adjusted incidence rate for TMB was 13/100 000 population, 38/100 000 for other anterior circulation distribution TIA, and 14/100 000 for vertebrobasilar distribution TIA. There was no sex-related difference detected within TIA subtypes, although vertebrobasilar rates were somewhat higher in men.

Incidence rates of TIA for 1985–1989 were compared with rates obtained by the medical record linkage system with first TIA in 1955–1979. Rates were also compared with a cohort study of Rochester residents performed for 1960–1972 and with medical records linkage system data limited to 1960–1972. The average annual age- and sex-adjusted incidence rate for TIA during 1985–1989, among persons aged >50 years, was 231/100 000 population. This is similar to the rate of 237/100 000 determined from the 1960–1972 cohort study. TIA incidence rates were lower when medical record linkage system methods were used during 1955–1979 (138/100 000) and during 1960–1972 (134/100 000). However, evaluation of cases of TIA detected in the cohort study identified logistic reasons for failure of ascertainment with the use of the medical record linkage system. The present study, with ascertainment of cases simultaneously with stroke cases and with the use of a diagnosis index rubric that had been shown to detect additional cases that would have previously been missed, led to higher and more accurate incidence rates.

TABLE 2. Average Annual Age- and Sex-Specific Incidence Rates* of TIA in Rochester, Minn, 1985–1989

<table>
<thead>
<tr>
<th>Age Group, y</th>
<th>Incidence Population</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate No.</td>
<td>Rate No.</td>
<td>Rate No.</td>
<td></td>
</tr>
<tr>
<td>0–34</td>
<td>93 650</td>
<td>99 226</td>
<td>192 876</td>
<td>1</td>
</tr>
<tr>
<td>35–44</td>
<td>22 434</td>
<td>24 103</td>
<td>46 537</td>
<td>18</td>
</tr>
<tr>
<td>45–54</td>
<td>14 788</td>
<td>15 825</td>
<td>30 613</td>
<td>54</td>
</tr>
<tr>
<td>55–64</td>
<td>10 962</td>
<td>12 446</td>
<td>23 408</td>
<td>128</td>
</tr>
<tr>
<td>65–74</td>
<td>7422</td>
<td>10 815</td>
<td>18 237</td>
<td>323</td>
</tr>
<tr>
<td>75–84</td>
<td>3785</td>
<td>8715</td>
<td>12 500</td>
<td>687</td>
</tr>
<tr>
<td>=&gt;85</td>
<td>1206</td>
<td>4285</td>
<td>5491</td>
<td>498</td>
</tr>
<tr>
<td>ALL</td>
<td>154 247</td>
<td>175 415</td>
<td>329 662</td>
<td>76</td>
</tr>
</tbody>
</table>

*Per 100 000 population.
†Age-adjusted or ‡age- and sex-adjusted to 1980 US white population.

TABLE 3. Average Annual Incidence Rates of TIA Subtypes, 1985–1989

<table>
<thead>
<tr>
<th>Subtype</th>
<th>Men* Rate No.</th>
<th>Women* Rate No.</th>
<th>Total† Rate No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid distribution (excluding TMB)</td>
<td>41</td>
<td>45</td>
<td>37</td>
</tr>
<tr>
<td>TMB</td>
<td>12</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>All carotid</td>
<td>53</td>
<td>58</td>
<td>51</td>
</tr>
<tr>
<td>Vertebrobasilar distribution</td>
<td>20</td>
<td>22</td>
<td>10</td>
</tr>
<tr>
<td>Uncertain location</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*Age-adjusted to 1980 US white population, per 100 000 population.
†Age- and sex-adjusted to 1980 US white population, per 100 000 population.

Discussion

Incidence of TIA has been reported for numerous sites worldwide. Despite considerable differences in study type, method of case ascertainment, and TIA definition, comparison of age- and sex-adjusted rates has revealed little difference in incidence rates (Table 4) between earlier Rochester, Minn, studies1–3 and Oxfordshire, England,5 and Estonia, USSR.9 The incidence rates reported for Lehigh Valley, Pa,10 were somewhat lower. The incidence rates for Rochester used in prior comparisons were an underestimate of the true TIA incidence because of failure to detect some TIAs with the use of the medical record linkage system.2,3,16 The present study, with ascertainment of cases simultaneously with stroke cases and with the use of a diagnosis index rubric that had been shown to detect additional cases that would have previously been missed, led to higher and more accurate incidence rates. Comparison of age- and sex-adjusted incidence rates with other sites from throughout the world (Table 4) shows much higher rates in Rochester. It is possible that this demonstrates a true difference in rates. However, cerebral infarction rates are not markedly higher in Rochester23 than in the sites in Table 4 that have also reported cerebral infarction incidence. This makes it more likely that incomplete case ascertainment in the other studies is contributing to the large differences. Given the transient nature of the symptoms and other complexities in comprehensively detecting TIA cases, this would be a plausible explanation.

The age/sex-adjusted annual incidence rate of TIA (68/100 000 population per year) is 41% of the annual stroke incidence rate (145/100 000 population per year) reported for...
The incidence of TIA and cerebral infarction increases with age. In the present study, the TIA incidence was slightly lower in those aged ≥85 years compared with those aged 75 to 84 years. Because of the relatively small population aged ≥85 years, ascertainment of a small number of additional cases would have markedly altered the incidence rate. It is possible that case ascertainment for these transient events was incomplete in this oldest age group, especially with a high proportion in nursing homes. While all nursing home diagnoses were reviewed, transient events may be less likely to be recorded. In addition, transient events may be viewed as relatively minor in older patients with numerous other medical disorders and may be less likely to be comprehensively recorded in a medical diagnosis indexing system.

Carotid distribution TIAs (cerebral or retinal) constitute 80% of all TIAs for which the distribution could be defined. The percentage of carotid cases is considerably higher than that reported in Japan but is similar to that reported in Oxfordshire. While a true difference in the ratio of TIA to stroke is possible, methodological considerations such as differences in population type, method of case ascertainment, incomplete TIA ascertainment, or incomplete stroke ascertainment are all possible explanations. However, the completeness of stroke ascertainment in Rochester by the medical record linkage method has been shown to be comparable to a cohort study in the same population, making incomplete stroke ascertainment in Rochester an unlikely explanation.

The incidence rates for cerebral infarction and TIA lead to an estimation of the number of people in a defined population that may require diagnostic evaluation to characterize the mechanism of an ischemic event. These data indicate that if all patients with TIA were to present before a stroke, the total number requiring diagnostic evaluation would be nearly half again the number presenting with first ischemic stroke. These considerations are particularly important since recent studies have documented the efficacy of surgery in stroke prevention for patients with high-grade carotid stenosis, and others are evaluating the use of warfarin in symptomatic intracranial arterial stenoses.

The data reported here suggest that the incidence of TIA has not changed since 1960–1972. Although these data may be best compared with cohort data reported from 1960–1972 with different methods of case ascertainment, a prior study indicates that methods of case ascertainment using the medical records linkage system utilized during 1985–1989 should provide comprehensive case detection equivalent to the cohort study results.

The potential impact of carotid endarterectomy and medical therapies on incidence of TIA during 1960–1989 must be considered. It is unlikely that use of carotid endarterectomy has a significant impact on TIA incidence in our population. In a population-based study of the prevalence of cardiovascular risk factors in Rochester, Minn, in 1986, the prevalence of carotid endarterectomy was between 0.5% (women aged 65 to 74 years) and 3.7% (men aged 65 to 74 years) among those aged >55 years, with a total prevalence of 17 among 2122 randomly selected residents surveyed. The study did not differentiate which of the people had prior cerebral ischemic symptoms, making it likely that the prevalence for asymptomatic patients only would be even lower than the prevalence noted.

Few data are available regarding the frequency of use of antiplatelet agents or warfarin among people without prior symptoms of cerebral ischemia. Information from a previously reported Rochester, Minn, based case-control study of stroke risk factors provides some data. Residents of Rochester, Minn, without prior cerebral infarction were identified, with age and sex matching those of the cerebral infarction cases. Medication use at the time of a defined “index date” was determined. The frequency of use of antiplatelet agents among these cerebral infarction controls increased from 0.8% during 1960–1964, to 5.7% during 1975–1979, to 21.3% during 1985–1989. Warfarin use in the controls remained stable at approximately 0.5% during the 30-year period. The data on the cerebral infarction controls are similar to those regarding the frequency of use among the TIA/AF cases during 1985–1989. Twenty-four percent were on antiplatelet agents, and 1.5% were on warfarin at the time of occurrence.

### TABLE 4. Incidence of TIA Reported From Sites Throughout the World

<table>
<thead>
<tr>
<th>Location</th>
<th>Time Period</th>
<th>Study Type</th>
<th>No. of Cases</th>
<th>Incidence Rate (Crude, Unadjusted by Age, per 100 000)</th>
<th>Age/Sex-Adjusted Incidence Rate*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Total</td>
</tr>
<tr>
<td>Rochester, Minn</td>
<td>1985–1989</td>
<td>Population-based, retrospective registry</td>
<td>202</td>
<td>68</td>
<td>54</td>
</tr>
<tr>
<td></td>
<td>1983–1986</td>
<td>Population-based, prospective registry</td>
<td>53</td>
<td>56</td>
<td>45</td>
</tr>
<tr>
<td>Hisayma, Japan</td>
<td>1961–1982</td>
<td>Population-based, prospective cohort study</td>
<td>18</td>
<td>78</td>
<td>38</td>
</tr>
</tbody>
</table>

*Age- and sex-adjusted to 1980 US white population, per 100 000 population. NR indicates not reported.
of this first TIA/AF episode. These data suggest that the frequency of aspirin use among people without prior cerebral infarct may be increasing, but there has not been a simultaneous reduction in TIA incidence. Warfarin use in the population has not changed over 30 years and likely does not affect TIA incidence. Although the mechanism for these TIAs cannot be defined with certainty, it is assumed that the most common definable cause is carotid and vertebral-basilar atherosclerosis. It had previously been suggested that a lack of change in TIA incidence during 1955–1979 implied unchanging ulcerative atherosclerotic cerebrovascular arterial disease. The present data indicate that there continues to be little change in TIA incidence and thus in occurrence of the most common mechanism for TIA, atherosclerosis of the cerebrovascular system.

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References
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