

The Incidence of Stroke in
The Kuopio Area of East Finland
Juhani Sivenius, M.D.,* Olli P. Heinonen, M.D.,† Kalevi Pyörälä, M.D.,‡ Jukka Salonen, M.D.,§ and Paavo Riekkinen, M.D.*

SUMMARY During a 20-month study period there were 373 strokes in a geographically defined population (235,100/100,000/year). When age and sex were adjusted to the mean population of Finland in 1979, the annual incidence of stroke was 270/100,000 persons. The distribution of incidence cases by diagnostic category was as follows: cerebral infarction 80%, ICH 9%, SAH 8% and NOS 3%. Case fatality of stroke within one year was 37%. The recurrence rate was 6% during the first year after any stroke.

IN THE COLLABORATIVE STUDY coordinated by WHO from 1971 to 1974 the incidence of stroke was investigated in 17 populations.† The annual age and sex adjusted incidence rates for first stroke varied from 0.24 to 2.87/1000 persons. The lowest rate was observed in Sri Lanka and the highest rate in Japan. Two Finnish populations were included in this study. The population of North Karelia county had an annual incidence rate of 1.71/1000 persons and it was fourth highest; and the population of Espoo and Kauniainen towns in Southern Finland had an incidence rate of 1.17/1000 persons, which was seventh highest among the WHO study populations.

The purpose of the present study was to determine the incidence and prognosis of stroke in four communities of the Kuopio county in East Finland.

Study Population and Methods
The Department of Neurology at the University Hospital of Kuopio started a Stroke Register for the Kuopio area on October 1st, 1978. The register functioned for twenty months, up to May 31, 1980. The study area consisted of one town, Kuopio, and three rural communities (fig. 1). The study was based on the population of this area. The distribution of the total population was: Kuopio 73,733 and the rural communities 21,687. Thus the majority of the study population consisted of urban inhabitants.

All new cases of stroke in the study population were registered during the study period. The clinical examination was scheduled to take place as soon as possible after the onset of symptoms. Most of the patients included in the register were examined by one of the authors (JS) (74%), some examinations were performed by a consulting neurologist or by the neurologist on duty (17%). The remaining 9% of the patients were cases, who died in the very early phase of the stroke, so that neurologist’s examination was not possible. In such occurrences the patients’ hospital files and possible autopsy documents were decisive in taking the patient to the study.

Prior to the study all general practitioners (25) in the area were personally asked to send all new cases of stroke either to the emergency unit or to the outpatient department of Kuopio University Hospital. During the study physicians repeatedly stated, when contacted, that only mild cases of stroke were treated at home in extremely exceptional circumstances.

All death certificates of the study population were
reviewed every two weeks in the Coroner’s Office of the Provincial Government.

Definition of Stroke and Diagnostic Categories

Stroke was defined as rapidly developing clinical signs of a focal or global disturbance in cerebral function, lasting longer than 24 hours or leading to death, with no apparent origin other than a vascular source. Included were: subarachnoid haemorrhage (SAH, ICD 430), intracerebral haemorrhage (ICH, ICD 431), ischaemic brain infarction — both embolic and nonembolic (INF, ICD 432-434) — and unspecified acute CVD (NOS, ICD 436). TIA (ICD 435) was excluded by the definition.

Diagnostic Criteria

SAH: Angiographic identification of an aneurysm or arteriovenous malformation as the source of haemorrhage or demonstration at autopsy of recent bleeding or demonstration at autopsy of recent bleeding of a saccular aneurysm or arteriovenous malformation. When angiograms were not performed, the clinical diagnosis of SAH was made by applying the criteria of Pakarinen.2

ICH: The minimum criterion for an intracerebral haemorrhage was bloody cerebrospinal fluid (CSF), demonstration of an intracerebral hematoma by computerized tomography or an avascular mass effect in cerebral angiography (without evidence of aneurysm or arteriovenous malformation), or autopsy evidence.

Oclusion of precerebral arteries: Of the non-haemorrhagic strokes, those with angiographic or autopsy evidence of neck artery occlusion.

Brain infarction: Of the cases without CSF examination or autopsy, those with a history of TIA and without disturbance of consciousness.

Embolic brain infarction: Accepted as embolic were those cases of infarction which featured an abrupt onset and had an identified source of embolism, such as a recurrent myocardial infarction or atrial fibrillation.

Unspecified stroke: Cases with insufficient data.

Embolic brain infarction and occlusion of the precerebral arteries are here combined under the diagnostic category of brain infarction.

Incidence Rates

The number of persons falling ill with stroke was expressed as an annual rate per 100,000 of the total population. Persons with previous stroke who experienced a new stroke during the study period were included in the incidence figures. However, if a registered patient had a recurrence during the first study year, this incidence was not included. Thus the incidence figures refer to the number of persons experiencing a stroke in a year. Age and sex adjustment of the rates was made by a direct method using the 1979 mean population of Finland as a standard.

Results

Incidence of Stroke

During the 20 month study period 373 people suffered a stroke, an annual incidence of 235/100,000 for the entire population. There was no difference in the incidence rates between men and women (table 1). The incidence of stroke as a whole increased steeply with

<table>
<thead>
<tr>
<th>Type of stroke</th>
<th>Study population</th>
<th>Age-adjusted</th>
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<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
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<tr>
<td>INF.</td>
<td>190</td>
<td>188</td>
</tr>
<tr>
<td>ICH</td>
<td>17</td>
<td>24</td>
</tr>
<tr>
<td>SAH</td>
<td>21</td>
<td>14</td>
</tr>
<tr>
<td>NOS</td>
<td>8</td>
<td>7</td>
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<tr>
<td>Total</td>
<td>236</td>
<td>233</td>
</tr>
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</table>

or age (years)

15–24
25–34
35–44
45–54
55–64
65–74
75–
Total

<table>
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<tr>
<th>Age (years)</th>
<th>Male</th>
<th>Female</th>
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<td>7</td>
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<tr>
<td>25–34</td>
<td>27</td>
<td>34</td>
<td>30</td>
</tr>
<tr>
<td>35–44</td>
<td>92</td>
<td>29</td>
<td>60</td>
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<tr>
<td>45–54</td>
<td>383</td>
<td>108</td>
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<tr>
<td>55–64</td>
<td>701</td>
<td>350</td>
<td>494</td>
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<tr>
<td>65–74</td>
<td>1471</td>
<td>902</td>
<td>1107</td>
</tr>
<tr>
<td>75–</td>
<td>2301</td>
<td>2363</td>
<td>2345</td>
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<tr>
<td>Total</td>
<td>236</td>
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or age (years)

15–24
25–34
35–44
45–54
55–64
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Total

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<th>Age (years)</th>
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<th>Female</th>
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<td>75–</td>
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<td>2345</td>
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<tr>
<td>Total</td>
<td>236</td>
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</table>
Table 5. Annual Incidence per 100,000 Population of Stroke in Selected Communities, Age Groups 35-74 Years

<table>
<thead>
<tr>
<th>Community</th>
<th>Study years</th>
<th>35-44</th>
<th>45-54</th>
<th>55-64</th>
<th>65-74</th>
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<tr>
<td>*Rochester²</td>
<td>1955-69</td>
<td>35</td>
<td>110</td>
<td>364</td>
<td>791</td>
</tr>
<tr>
<td>†Frederiksdal⁴</td>
<td>1971-73</td>
<td>—</td>
<td>—</td>
<td>300</td>
<td>640</td>
</tr>
<tr>
<td>†Akita¹</td>
<td>1971-74</td>
<td>—</td>
<td>504</td>
<td>967</td>
<td>2693</td>
</tr>
<tr>
<td>†Espoo-Kauniainen⁵</td>
<td>1972-73</td>
<td>59</td>
<td>173</td>
<td>358</td>
<td>1061</td>
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<tr>
<td>*National Survey⁸</td>
<td>1975-76</td>
<td>31</td>
<td>106</td>
<td>262</td>
<td>582</td>
</tr>
<tr>
<td>U.S.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>†Tilburg⁰</td>
<td>1978-79</td>
<td>—</td>
<td>—</td>
<td>302</td>
<td>868</td>
</tr>
<tr>
<td>†Melbourne⁷</td>
<td>1978-79</td>
<td>24</td>
<td>123</td>
<td>342</td>
<td>1028</td>
</tr>
<tr>
<td>†Kuopio Area</td>
<td>1978-80</td>
<td>60</td>
<td>238</td>
<td>494</td>
<td>1107</td>
</tr>
</tbody>
</table>

*Incidence rates are based on first attacks of stroke. †Incidence rates include patients with previous stroke.

Discussion

Validity of Methods

The study area was chosen to insure that its population would receive care in only one hospital, the Central University Hospital of Kuopio. Hospitalized patients were detected with the highest probability in every case. Every morning the diagnoses of patients seen in the emergency room were checked. The neurologist on duty had a protocol for all stroke cases. The physicians of the departments of medicine were informed to make contact when a stroke case was found among patients treated for other reasons. In addition a computer list was generated for all patients discharged from the hospital with diagnoses of stroke or TIA.

For fatal cases the coverage of the registration must be close to 100%. Besides the careful screening of hospitalized patients all death certificates of the study population were regularly examined in the study period and three months thereafter.

It may be more difficult to prove that all mild cases were detected. The health care organisation in Finland is centralized so that nearly all of the open care is given by municipal physicians. They were personally contacted and asked to notify the register of all new strokes not referred as emergency patients to the hospital. In group, the fatality increased throughout the study period but remained the lowest. Recurrent stroke occurred in 23 out of 373 patients (6%) during the first year after the stroke.

**Case-Fatality**

One year after the stroke, the cumulative fatality rate was 37% (fig. 2); within the first three months it was 26%. Among the patients with SAH all deaths occurred during the first three weeks. In the infarct
discussions with local physicians it was frequently stated that they very rarely treated stroke patients, even mild cases, at home. Most cases that were not admitted directly to the hospital were admitted for diagnostic purposes to the neurology outpatient unit.

At autopsy (which was performed in 58% of fatal cases) only one clinical diagnosis appeared to be erroneous (a cerebral astrocytoma in an elderly male). Diagnostic procedures were frequently utilized (examined by a neurologist in 91%, lumbar puncture in 69%, brain scan in 61%, EEG in 51%, angiography in 12% and CT in 8% of patients).

Incidence

The adjusted annual incidence of stroke was 270 per 100,000. This incidence is high compared to international studies,1 where the other two Finnish studies are represented. Comparison of the total incidence with findings in other studies is not possible, but where age grouping fits with the present series, the age-specific incidence rates are comparable with those in several recent series.1,3-8 Our findings suggest a clearly lower incidence rate than in the Japanese study, but otherwise the figures are higher than in the other series (table 5). The difference is most marked in the youngest age groups.

Besides comparing of age-specific incidences previous Finnish stroke studies,5,9 are suitable for comparison of the total incidence (table 6). The adjusted incidence of stroke was higher in the Kuopio area for both females and males than elsewhere in Finland; the difference was not statistically significant.

In Finland and many other countries the mortality from cerebrovascular diseases has been decreasing. From 1967 to 1976 it declined in all age groups in both sexes, but the change was most marked in the oldest groups.10 The same trend occurred in the county of Kuopio (Valkonen, unpublished results, 1983).

It has been reported also that the incidence of stroke, including non-fatal events, is decreasing in most developed countries.5,11 The same phenomenon has occurred in Finland in North Karelia,12 where the incidence rates decreased by 34% among men and by 32% among women between 1972-73 and 1976-77.

In this study the incidence rates are higher. The best explanation may be the more intensive case-finding and perhaps the more precise diagnostic methods.

In the international surveys the incidence of SAH varies widely, the highest rates being in Finland. In three Finnish studies the rates were 15.7,2 23.99 and 19.4/100,000/year.13 In this study the incidence was of the same high level, 18/100,000/year.

Case Fatality

The cumulative fatality within one year after the stroke was 37% which is fairly low compared with many previous studies.14-16 Likewise in the National Survey of Stroke4 and in the cooperative WHO coordinated study1 48% of patients died during one year. In the Finnish study of Espoo-Kauniainen7 the three month’s mortality of 45.8% was higher than in the population of the present study during the whole year. In the extension of this study to the years 1978–80 the case fatality was decreased to 40% per year.14

The relatively small case fatality in the present study has several explanations. It is possible that a larger proportion of mild cases was detected than in the other studies. The proportion of cerebral infarcts among all incident cases, 80%, was higher than in the other studies in Finland. Since the prognosis is better after a cerebral infarct than after cerebral haemorrhage, this might be an explanation of the better prognosis for patients in our study.

Improved control of hypertension might be one factor contributing to better survival of stroke patients. It is possible that the favorable one year survival in our study population partly reflects improving medical care.

References


<table>
<thead>
<tr>
<th>TABLE 6</th>
<th>Total Crude Sex-specific and Age-adjusted Annual Incidence (per 100,000 persons) in the Three Populations in Finland</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence Rate</td>
<td>Kuopio</td>
</tr>
<tr>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Crude</td>
<td></td>
</tr>
<tr>
<td>236</td>
<td>233</td>
</tr>
<tr>
<td>Age-adjusted*</td>
<td></td>
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<tr>
<td>274</td>
<td>266</td>
</tr>
</tbody>
</table>

*Age-adjusted by direct method to the 1979 population of Finland.
MANY POSSIBLE THERAPIES for acute cerebral ischemia alter blood viscosity, by hemodilution or phlebotomy,1-5 alteration of red blood cell deformability6 or reduction of fibrinogen concentration.7-9 The present study focuses on the effect of fibrinogen reduction alone on cerebral blood flow and cellular injury in an animal model of severe cerebral ischemia.

The viscosity of blood involves a complex relationship between the hematocrit and the concentration of fibrinogen, which is a significant component of plasma viscosity.10 The viscosity of blood is a function of both the number of red blood cells and the concentration of plasma proteins, particularly fibrinogen.11

CBF = \frac{\text{perfusion pressure}}{\text{cerebral resistance}}

Increased CBF and clinical improvement have been documented following viscosity reduction by phlebotomy and hemodilution, but these maneuvers effect viscosity primarily by decreasing hematocrit. Less attention has been focused on the effect of reducing fibrinogen, even though the concentration of this molecule is increased in cerebral ischemia.12

**Summary**

This study examines the effect of fibrinogen and consequent blood viscosity reduction on cerebral blood flow and cellular injury following severe cerebral ischemia for 30 minutes in 78 Wistar rats. In half of these rats 10 to 15 cc's of blood was removed and replaced with a mixture of 5% albumin and autologous red blood cells maintaining a constant hematocrit but resulting in a 30% decrease in fibrinogen and corresponding reduction in viscosity. Fibrinogen reduction resulted in a slight increase in baseline CBF and the elimination of post-ischemic hyperemia at 24 hours. Both study and control animals showed a similar decrease in CBF at 30 minutes and 2 hours. There was no significant difference in the severity of ischemic cellular change between the fibrinogen reduction group and controls, although there was a significant inverse relationship between the amount of viscosity change and severity of cellular injury within the treatment group. Fibrinogen reduction alone cannot significantly ameliorate ischemic injury in this model. Viscosity reduction therapy should include reduction of hematocrit and alteration of red cell deformability.

**Acknowledgments**

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12. National Public Health Laboratory of Finland: Community Control of cerebrovascular diseases. World Health Organization, Regional Office for Europe, Copenhagen, 190-191, 1981

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