A TRIAL FIBRILLATION (AF) is one of the main risk factors for stroke. In unselected materials 21–25% of the patients with stroke have this arrhythmia. The Framingham study showed a nearly six times higher incidence of stroke in patients with AF due to ischaemic heart disease than in an age-, sex-, and blood pressure-matched control group without AF. The reason for an increased stroke risk in AF has always been claimed to be the occurrence of left atrial thrombosis causing arterial embolism. In patients with rheumatic heart disease, especially mitral stenosis with AF, the frequency of atrial thrombi has been found to be 30–42%. However, this aetiology of AF is nowadays uncommon in industrialized countries and the prevalence of left atrial thrombi in non-rheumatic AF, although higher than in controls, is not more than 13–27%. Also the actual existence of embolism in stroke cases can rarely be proved either in vivo or post mortem. Consequently it seems that other explanations than embolism for the association between AF and stroke must be considered. For example, generalized arteriosclerosis might be the common cause of both conditions. If this was the case, one would expect brain infarction in AF to resemble other atherothrombotic infarctions. Differences in patient or brain lesion characteristics, on the other hand, might suggest different pathogenetic mechanisms. To elucidate this problem the following questions were studied in an unselected stroke material: What is the prevalence of AF in different types of stroke? Are there any differences in risk factors or arteriosclerotic manifestations between brain infarction patients with and without AF? Are the brain lesion characteristics in these patients similar? Within the AF group, are there any differences between a chronic and paroxysmal type of arrhythmia?

SUMMARY The association between non-rheumatic atrial fibrillation (AF) and stroke has been studied in 402 patients consecutively admitted to a stroke unit. Brain infarction patients with sinus rhythm (n = 196) and non-rheumatic AF (n = 92) were further compared. Some findings supported an embolic origin of the stroke: half of the deceased AF patients (n = 24) at autopsy either had left atrial thrombosis or arterial embolism compared to none of the ten with sinus rhythm. Patients with AF also had a higher mortality and more severe brain lesions, findings compatible with a sudden occlusion of blood flow. However, these differences might also be explained by an atherothrombotic occlusion with impaired autoregulation in the ischaemic region in conjunction with heart failure, which was more common in the AF patients. Other findings supporting an atherothrombotic mechanism were: the prevalence of AF was higher (19–29%) in all kinds of stroke, including haemorrhage, than in age-matched controls (3–9%). Also patients with previous AF and no present embolic source resembled the whole AF group and differed from patients with sinus rhythm. Thus embolism is a plausible cause of stroke in many AF patients, whereas an atherothrombotic origin is more likely in others. Characteristics identifying the mechanism in an individual case were not found.

PATIENTS AND METHODS

Between October 1976 and December 1979, 402 patients with cerebrovascular diseases were treated in the Stroke Unit at the Medical Department of Serafimerlasarettet, Stockholm. Criteria for admission, organization of the unit and diagnostic definitions have been described elsewhere. The material comprised 200 men and 202 women of mean age 73 years (men 71, women 75 years). As a basis for diagnosis, the results of the following investigations were used: ordinary clinical procedure, routine laboratory tests, ECG and chest X-ray. Lumbar puncture with CSF analysis including spectrophotometry was performed in 95% of the cases and brain scan in 82%. From May 1978 computerized axial tomography (CT) was also included and 45% of the patients were examined by this method as well. Autopsy was performed in 90% of deceased patients. Totally, CT or autopsy were performed in 55% of the material. In the remainder 180 cases CSF and brain scan constituted the basis. Hereby haemorrhages might be missed in 2–3 out of 100 stroke patients and overdiagnosed in as many. Possible diagnostic errors in the study material were therefore few and similar in the AF and SR groups.

Patients with brain infarction (n = 318) were further studied (fig. 1). Seven patients were excluded due to missing ECG and 23 because of possible rheumatic heart disease. Such was earlier diagnosed in 4 subjects and compatible with the history and physical findings in another 19. RHD was verified in two of these patients who came to autopsy. No clinically defined non-rheumatic AF were disproved post mortem. The remaining 288 patients were divided into three groups according to the occurrence of ECG-verified AF: chronic AF (n = 61) — constant AF during hospital stay, with or without earlier known AF, paroxysmal AF (n = 31) — inconstant occurrence of AF during hospital stay or earlier diagnosed episodes of AF, sinus rhythm (SR) — no known episodes of AF during hospital stay or earlier. The mean ages in the two AF groups were 77 and 76 years respectively and signifi-
ATRIAL FIBRILLATION IN STROKE/Britton and Gustafsson

STROKE PATIENTS 402

- TIA 45
- CEREBRAL HAEMORRHAGE 32
- ECG MISSING 5
- UNSPECIFIED 7

BRAIN INFARCTION 318

- ECG MISSING 7
- RHD 23

CHRONIC AF 61
PAROXYSMAL AF 31
SINUS RHYTHM 196

MEAN AGE 77 YEARS 76 YEARS 71 YEARS

Figure 1. Diagnostic distribution in 402 consecutive stroke patients. Brain infarction patients are divided into three groups according to their heart rhythm. For each group sex distributions and mean age are given. RHD = rheumatic heart disease.

The aetiology of AF was looked for. All patients with known or suspected RHD were, as earlier mentioned, excluded. All the remainder, accordingly, suffered from non-rheumatic AF. In all but 17% of patients one or more factors of possible aetiological importance for AF were present, as can be seen from the patient history part of table 1. The corresponding figure in the SR group was 23%.

Patient history, symptoms and the circumstances at onset of stroke were noted according to a special form. Physical examination was performed and registered at arrival, on day 1, on day 4 and at discharge. At the time, this study was not planned wherefore the observations were unbiased as regards differences between AF and SR patients. A neurological score modified after Mathew,11 as earlier described, was also included. The ECG was appraised according to the Minnesota code.12 On the chest X-ray the size of the heart was measured and signs of congestion noted. A heart of >500 ml/m² body area in men and >450 ml/m² in women was considered enlarged.

Blood samples were drawn in fasting stage on the first and fourth day after arrival. Autopsies were performed according to a schedule, with interest focused on the heart and brain. The degree of arteriosclerosis, the size, age and location of heart and brain infarcts and the presence of thrombosis or embolism were noted. Thrombosis was defined as an obstruction caused by an intravascular plug adherent to the vessel wall and embolism as a sharply delimited plug lying loose in the vessel. Sometimes the two entities could not be separated. An infarction was named haemorrhagic when it was found stripped with small haemorrhages at autopsy or when spotted haemorrhages were seen at CT within the infarcted area.

During a certain period of the study all patients (n = 209) participated in further studies. They were questioned about alcohol consumption and classified in four groups; total abstainer, <133 g, 133–667 g, >667 g ethanol per month. The blood pressure was measured in both great toes and compared to that in the arms according to methods described earlier.13 A systolic toe blood pressure of less than 65% of that in the arm was considered a sign of obstructed arterial flow. These 209 stroke patients were matched by age (± one year) and by sex to patients admitted via the Casualty Department to a general surgery ward for acute surgical problems. The frequency of AF in this group was compared with that in the study material.

Statistics

The chi-square test was used for testing the significance of differences of proportions. A two-sample T-test was used to test differences between independent means. Degrees of significance considered were 5%, 1% and 0.1%.

Results

Prevalence of AF in Different Types of Stroke

Table 2 shows the AF prevalence in the whole material in relation to diagnostic categories and age of patients. For comparison, the matched group of patients was added. The prevalence of AF increased with increasing age of patients. It was higher in all the cerebrovascular groups than in the surgical but was especially high among patients with cerebral infarction.

Risk Factors and Arteriosclerotic Manifestations in Brain Infarction Patients With and Without AF

As shown in table 1, there were more smokers in the SR group. Most previous circulatory diseases were equally distributed. However, a history of heart failure was more than twice as common among AF patients. This was confirmed by more frequent X-ray findings of heart enlargement and pulmonary congestion. The AF patients also had lowered toe blood pressure more often and slightly lower blood pressure at admission and on day 1 but not later on. No other differences in arteriosclerotic or hypertensive manifestations were noted.

Among laboratory data (table 1) mean fasting glucose as well as the proportion of fasting glucose >8.0 mmol on day 1 was higher in the AF group. Lipid
<table>
<thead>
<tr>
<th>Patient history data, %</th>
<th>AF n = 92</th>
<th>SR n = 196</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>37</td>
<td>49</td>
<td>*p &lt; 0.001</td>
</tr>
<tr>
<td>Heart failure</td>
<td>63</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>27</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption (n = 154)*: ≥ 667 g/year</td>
<td>28</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>None of the above mentioned factors</td>
<td>17</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>34</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>Intermittent claudication</td>
<td>9</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>16</td>
<td>33</td>
<td>*p &lt; 0.01</td>
</tr>
</tbody>
</table>

Hospital findings

Mean blood pressure, mm Hg:
- On admission: 173/94 (AF) vs 180/95 (SR) *p < 0.05/NS*
- Day 1: 161/85 (AF) vs 171/90 (SR) *p < 0.01/p < 0.01*

Toe blood pressure (n = 82)*
- Pathologically lowered, %: 78 (AF) vs 55 (SR) *p < 0.05*
- Carotic bruits, %: 8 (AF) vs 10 (SR)  
- ECG, %:
  - Normal (except rhythm): 7/16 (AF) vs 16/19 (SR) *p < 0.05*
  - Signs of earlier infarction: 23/19 (AF) vs 19/19 (SR)  
  - Left ventricular hypertrophy: 3/4 (AF) vs 4/4 (SR)  
  - Bundle branch block: 19/19 (AF) vs 19/19 (SR)  

Chest X-ray: %
- Heart enlargement: 53/17 (AF) vs 17/17 (SR) *p < 0.001*
- Pulmonary congestion: 49/18 (AF) vs 18/18 (SR) *p < 0.001*

Laboratory data, mean values
- Haemoglobin, g/l: 146 (AF) vs 142 (SR)  
- Haematocrit, %: 44 (AF) vs 43 (SR)  
- Blood glucose, mmol/l: 6.8 (AF) vs 6.0 (SR) *p < 0.01*
- Triglycerides, mmol/l: 1.4 (AF) vs 1.7 (SR) *p < 0.05*  
- Cholesterol, mmol/l: 5.9 (AF) vs 6.5 (SR) *p < 0.05*  
- Prothrombin, %: 76.4 (AF) vs 84.1 (SR) *p < 0.001*  
- Platelets, × 10^9/l: 221 (AF) vs 219 (SR)  
- Leucocytes, × 10^9/l: 8.8 (AF) vs 7.5 (SR) *p < 0.01*

*No. of patients participating in investigations limited to parts of the material.

Ischaemic heart disease = myocardial infarction or angina pectoris.

concentrations, prothrombin values and leucocyte counts also differed between the groups.

No difference in causes of death could be seen between those with AF or SR. Autopsies were performed in 34 of the deceased. The AF group had left auricular thrombi in 21% of cases (5/24) and peripheral emboli (spleen, kidney, femoral or mesenterial artery) in one third (9/24). In the ten autopsied SR patients neither atrial thrombosis nor peripheral embolism was found. Occlusion of the relevant cerebral artery was found in 40% of cases, a similar proportion in both groups. It could be classified as embolic in three of the AF cases. In the remaining seven the pathogenesis of the occlusion could not be determined, nor in the four patients with occlusion in the SR group.

Brain Lesion Characteristics in Brain Infarction Patients With and Without AF

The symptoms were known to have occurred suddenly in 50–60% of cases in both groups. Paresis was the dominating symptom. Seizure at debut or in the first hours occurred in 7% of the AF group compared to 2% of the SR group (p > 0.05). No difference was seen between the groups as regards the location of the lesions.

At admission to hospital the AF group had several signs of more serious brain damage than the SR group (table 3). The condition of AF patients was worse as evaluated by level of consciousness, ability to walk and total neurological score. Mortality was higher (in
Atrial Fibrillation in Stroke/Britton and Gustafsson

Table 2 Prevalence of Atrial Fibrillation (AF) in Relation to Age of Patient and Diagnosis

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Cerebral Haemorrhage</th>
<th>TIA</th>
<th>Cerebral Infarction</th>
<th>Surgical Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of pat.</td>
<td>AF %</td>
<td>No. of pat.</td>
<td>AF %</td>
</tr>
<tr>
<td>&lt;59</td>
<td>1</td>
<td>0</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>60-69</td>
<td>9</td>
<td>0</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>70-79</td>
<td>9</td>
<td>33</td>
<td>16</td>
<td>31</td>
</tr>
<tr>
<td>80+</td>
<td>8</td>
<td>25</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
<td>19</td>
<td>45</td>
<td>20</td>
</tr>
</tbody>
</table>

Table 3 Brain Lesion Characteristics in 288 Patients with AF and SR

<table>
<thead>
<tr>
<th>Condition on admission</th>
<th>AF</th>
<th>SR</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological score, mean points</td>
<td>53</td>
<td>67</td>
<td>( p &lt; 0.001 )</td>
</tr>
<tr>
<td>Impaired consciousness, %</td>
<td>33</td>
<td>10</td>
<td>( p &lt; 0.001 )</td>
</tr>
<tr>
<td>Cannot walk independ-</td>
<td>77</td>
<td>64</td>
<td>( p &lt; 0.05 )</td>
</tr>
<tr>
<td>Mortality, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;70 years</td>
<td>26</td>
<td>5</td>
<td>( p &lt; 0.01 )</td>
</tr>
<tr>
<td>70-77 years</td>
<td>26</td>
<td>8</td>
<td>( p &lt; 0.05 )</td>
</tr>
<tr>
<td>&gt;77 years</td>
<td>26</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>7</td>
<td>( p &lt; 0.001 )</td>
</tr>
</tbody>
</table>

Brain scan and CT findings, %

<table>
<thead>
<tr>
<th>Brain scan (n = 233)*:</th>
<th>AF</th>
<th>SR</th>
<th>( p &lt; 0.001 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT (n = 127)*: infusion visible, %</td>
<td>36</td>
<td>35</td>
<td>( p &lt; 0.05 )</td>
</tr>
<tr>
<td>Brain scan or CT (n = 247)*:</td>
<td>68</td>
<td>42</td>
<td>( p &lt; 0.001 )</td>
</tr>
</tbody>
</table>

Haemorrhagic infarction, %

| Visible at CT (n = 127)* | 11 | 2  |
| Revealed at autopsy (n = 34)* | 42 | 50 |
| CSF: Haemorrhagic component in cerebral infarction verified at CT or autopsy (n = 156) | 6  | 12 |

No. of patients investigated.

Neurological score as modified after Mathew, 100 points = normal function, CSF findings of a haemorrhagic component = protein conc. > 1 g/l and/or spectrophotometric absorbance \( \geq 0.040 \) at 415 nm.

Differences Between Patients with Chronic and Paroxysmal AF

The paroxysmal AF group was rather heterogeneous. In one third of the patients AF had been recorded earlier but never during the present hospital stay. In spite of this, the blood pressure tended to be lower, as did the frequency of heart enlargement. The higher blood glucose level in AF than SR patients was mainly confined to the paroxysmal group. Thus the two AF groups turned out to be similar in most respects and with common differences when compared to the SR patients (table 4).

Analysis of the Present Material According to Suggestions by Hart et al.¹¹

When our study was finished this journal published a paper by Hart et al.¹⁵ who were also questioning embolism as the sole pathogenetic mechanism of stroke in patients with nonvalvular AF. Fifty-six cases were classified as probably embolic, nonembolic and indeterminate on the basis of clinical characteristics. Abrupt onset while awake, no further progress, history of embolic episodes, young age, absence of carotid artery stenosis, bilateral CT infarcts i.e., were considered signs of embolism. We divided our material accordingly (table 4). The proportion of embolic strokes was 19% in our material compared to 63% in that of Hart. Possible reasons for the discrepancy were: Our patients were six years older, frequent regular controls were done to detect progression of symptoms. As a patient could have characteristics fitting into more than one group the classification was open for varying interpretations. Also, we did not alter the primary group division according to autopsy results. Instead we used them to evaluate correctness of the clinically based classification. Thus it was evident that embolism was revealed in a high extent of deceased patients even when clinically classified as nonembolic. Except as regards factors included in the definition the three Hart groups turned out similar to our original groups of chronic and paroxysmal AF with common differences as compared to the brain infarction patients with sinus rhythm (table 4).

Discussion

The most striking findings in this study were that AF was more common in patients with stroke than among controls. The AF patients were older and had more serious brain damage than those with SR. Some factors
### Table 4 Characteristics of Patients with Sinusrhythm, Chronic and Paroxysmal AF

<table>
<thead>
<tr>
<th></th>
<th>Sinus rhythm</th>
<th>Chron AF</th>
<th>Parox AF</th>
<th>AF in patients</th>
<th>Autopsy findings of embolism or cardiac embolic source in patients</th>
<th>Division according to Hart et al.*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>196</td>
<td>61</td>
<td>31</td>
<td>14</td>
<td></td>
<td>Embolic</td>
</tr>
<tr>
<td>Mean age</td>
<td>71</td>
<td>77</td>
<td>76</td>
<td>76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous systemic emboli, %</td>
<td>0</td>
<td>5</td>
<td>3</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sudden onset of maximal symptoms, %</td>
<td>51</td>
<td>57</td>
<td>55</td>
<td>50</td>
<td></td>
<td>100</td>
</tr>
<tr>
<td>Early recurrence†, stroke %</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>14</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>other arterial occlusion, %</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>7</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Brain scan or CT (n = 247); positive findings, %</td>
<td>42</td>
<td>67</td>
<td>71</td>
<td>86</td>
<td></td>
<td>77</td>
</tr>
<tr>
<td>Mortality, %</td>
<td>7</td>
<td>25</td>
<td>29</td>
<td>100</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>Autopsies no.</td>
<td>10</td>
<td>15</td>
<td>9</td>
<td>14</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Autopsy findings of cerebral emboli or cardiac embolic source, %</td>
<td>30</td>
<td>67</td>
<td>44</td>
<td>100</td>
<td></td>
<td>67</td>
</tr>
<tr>
<td>Classification according to Hart</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>embolic, %</td>
<td></td>
<td>21</td>
<td>13</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>nonembolic, %</td>
<td></td>
<td>67</td>
<td>65</td>
<td>79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>indeterminate, %</td>
<td>12</td>
<td>22</td>
<td>7</td>
<td>7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Only 81 cases are included since 11 with paroxysmal AF, not present during hospital stay, did not fit into the criteria.
†Early recurrences: new stroke or acute onset of peripheral arterial occlusion within 10 days of initial stroke.

For comparison the 14 cases with embolism or cardiac embolic source verified at autopsy are separately presented. The AF material was also divided according to criteria given by Hart et al.15 for identifying stroke in AF patients as embolic, nonembolic and indeterminate.

have to be dealt with before discussing whether these and other differences between the groups favour an athero-thrombotic or embolic origin of stroke in AF patients.

We chose to compare all patients with AF with those with SR. It has been claimed that a sudden onset of maximum symptoms in a patient with an embolic source, like AF, is typical of embolism.16,17 On the other hand a stepwise or gradual debut has been shown to occur in some patients.16,18 We found progression of symptoms after arrival at hospital to be as common among AF patients as among others.16,18 Therefore it was not possible to define a subsample within the non-rheumatic AF group in which embolism would be more likely. Furthermore, we divided our AF patients in the same way as Hart et al have presented.15 Still, the same main differences appeared as in the whole material in comparison to the SR group.

Then what do the present findings suggest as regards the pathogenetic mechanism underlying the increased incidence of stroke in AF? We found considerable evidence arguing against embolism. The prevalence of AF in stroke patients was much higher than in the matched surgical controls and also as compared to the 3–7% found in a Swedish population study of 75 year old persons.39 However, AF was also common among patients with haemorrhage which is difficult to explain by the embolism theory.

The AF patients were moreover older, reflecting the increasing prevalence of AF with increasing age. More extensive arteriosclerosis was also noted through the finding of a higher proportion of pathologically lowered toe blood pressure and fewer, normal ECGs in AF patients. Arteriosclerotic macro- and microangiopathy progressing with age, might instead be the cause of the stroke as well as the heart disease. The latter can either reveal itself by AF leading to heart failure, or as heart failure which in turn provokes AF.

Further evidence against the embolic theory was the findings in the paroxysmal AF group. Even patients with previous AF and no present embolic source were similar to the whole AF group in all main aspects and differed from patients with SR.

High haematocrit values are associated with a risk of thrombotic disease.21,22 No difference in mean values, or in the proportion of pathologically raised ones, were seen between the two groups. Haemorrhagic infarctions, claimed to be more common after embolism,18,23 were found in half of the deceased whether AF or not. Only a few haemorrhagic infarctions were revealed by CT in both groups.

The AF group had higher blood glucose levels known to be associated with a worsened prognosis.24 Patients with normal blood glucose (<6.0 mmol/l) though, still differed from the control group. Both the raised concentrations of blood glucose and leucocytes
might also be connected with the more serious brain lesions in this group.25, 26

There was, however, also evidence favouring an embolic cause of stroke in AF patients. In 50% of those who died, atrial thrombosis or signs of arterial embolism were revealed at autopsy whereas such findings were absent in the autopsied SR patients. This is in accordance with results from previous autopsy studies.6, 7 Atrial thrombosis has also been confirmed in vivo in 27% of stroke patients with AF by pulmonary cine-angiography, whereas such findings were much more uncommon in the absence of arrhythmia.8 Jörgensen and Torvik have shown that in cases in which cerebral embolism was revealed at autopsy, 82% had suffered from AF compared to 32% of those with cerebral thrombosis.27 In a surgical material, in which peripheral embolism had been proven at surgery, 72% of the patients had AF.28

The findings in this study of more severe brain damage in AF patients may also argue in favour of embolism. A sudden interruption of blood flow caused by an embolus might give rise to larger infarctions than a gradual occlusion with opportunity for development of the collateral circulation. More severe lesions in embolic strokes have also been found at autopsy by Jörgensen and Torvik.28 In some clinical materials where this problem was analyzed, AF was noted to be associated with a worse prognosis29, 30 but this was not so in the Framingham study.30

There might be other explanations than embolism for the poor outcome of stroke in AF patients. Their blood pressure was somewhat lower. A decreased cardiac output due to the higher incidence of heart failure might also contribute to a reduced cerebral blood flow when autoregulatory mechanisms are impaired as in an ischaemic region.31

We would like to conclude that embolism is a plausible cause of stroke in many AF patients. In a large group though, an atherothrombotic origin may be more likely. We have not been able to find any characteristics helpful in defining which mechanism is involved in an individual case. Nor have we found convincing evidence in the literature for assured recognition of embolism. The most promising, but not practicable, methods so far presented might be an immediate carotid angiography32 or pulmonary cine-angiography.8

Anyhow, brain infarction in non-rheumatic AF patients, taken as a group, constitutes a special problem. The patients are worse struck than others and therefore make up a "high risk" group for which acute treatment trials would be especially indicated. Also available data point to a favourable effect of secondary prophylaxis with anticoagulants in these patients.32, 33

Another possibility would be to attack the AF problem primarily. Not only do these patients have increased incidence of stroke but also their strokes are more serious. Primary prophylaxis with anticoagulants might therefore be considered. However, this treatment is difficult, many patients are old and contraindications often exist. Due to the likely atherothrombotic pathogenesis in a considerable number of patients, there is also a rationale for a much easier prophylactic treatment with, for example, aspirin.

Acknowledgments

This study was supported by grants from the Foundations of Serafi-merasarettet, Clas Groschinsky, Loo and Hans Osterman and Fredrik and Ingrid Thuring.

References

20. Fisher CM, Adams RD: Observations on brain embolism with...
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 Paaavo Riekkinen, M.D.*

SUMMARY During a 20-month study period there were 373 strokes in a geographically defined population (235/100,000/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 incident 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.

Stroke Vol 16, No 2, 1985

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 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 making 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
Non-rheumatic atrial fibrillation as a risk factor for stroke.
M Britton and C Gustafsson

Stroke. 1985;16:182-188
doi: 10.1161/01.STR.16.2.182

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/16/2/182

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/