Comparison of Clinical and Neuroradiological Findings in First-Ever Stroke
A Population-Based Study

Arne Lindgren, MD; Bo Norrving, MD, PhD; Olof Rudling, MD; Barbro B. Johansson, MD, PhD

Background and Purpose To determine how a recently proposed clinical stroke subclassification corresponds to specific findings on computed tomography (CT) and magnetic resonance imaging (MRI) of the brain.

Methods Two hundred twenty-eight patients with first-ever stroke were divided into four clinical subgroups: (1) total anterior circulation syndrome; (2) partial anterior circulation syndrome; (3) lacunar syndrome; and (4) posterior circulation syndrome. The imaging protocol included CT of the brain on day 0 through 15 and a second CT and an MRI of the brain on day 0 through 180 after acute stroke onset.

Results There were 200 patients with cerebral infarction and 28 patients with intracerebral hemorrhage. Intracerebral hemorrhage was found in 19% of patients with total anterior circulation syndrome and in no patients with lacunar syndrome (x² test; P<.01 for the difference between the four clinical subgroups). Of the 200 patients with cerebral infarction, 27% had total anterior circulation, 30% partial anterior circulation, 26% lacunar, and 16% posterior circulation syndromes. CT within 2 days revealed a visible lesion in about two thirds of patients with infarctions of total or partial anterior circulation syndrome type, compared with only 22% of patients with lacunar infarction (x² test; P=.02 for the difference between the four subgroups). The mean volume of the symptomatic infarction on CT within 15 days was 95 mL for total anterior circulation, 20 mL for partial anterior circulation, and 2.5 mL for lacunar syndrome (one-factor ANOVA; P=.0001). A cortical involvement of the infarction on CT day 16 through 180 was seen in 81% of patients with total anterior circulation syndrome and 58% of those with partial anterior circulation syndrome, compared with only 8% of patients with lacunar syndrome (x² test; P=.0001). MRI more often than CT showed a cortical involvement of lacunar infarctions and also revealed more silent lesions.

Conclusions The described clinical subgroups significantly differed in frequencies of intracerebral hemorrhage, cortical involvement, and lesion volume on CT and MRI. (Stroke. 1994;25:1371-1377.)

Key Words • cerebral hemorrhage • cerebral infarction • magnetic resonance imaging • stroke classification • tomography, x-ray computed

Subjects and Methods

Patients
From February 1, 1991, to January 31, 1992, we registered all patients living in the local catchment area of Lund University Hospital (158 000 inhabitants) with clinical first-ever stroke, defined as acute focal neurological deficit lasting for more than 24 hours or leading to death, with no other cause than cerebrovascular disease.

Acute stroke patients from the local catchment area are primarily admitted to the Department of Neurology. We registered these patients and at regular intervals contacted other hospital departments, all common and private nursing homes, and primary-care units to detect stroke patients who had not been referred to the Department of Neurology. Data on patients with stroke dying out of hospital were requested from the Departments of Forensic Medicine and Clinical Pathology. Patients with subarachnoidal bleeding, bleeding in tumors, and iatrogenic stroke were not included.

Of 241 stroke patients registered, 2 were not examined clinically before death and 11 were not examined with CT, MRI, or autopsy. After exclusion of these 13 patients, 228 patients (mean age, 73.3 years; range, 38 to 103 years; 121 men) remained and were included in the study. Two hundred two patients were inpatients at the Department of Neurology, 6 at the Department of Neurosurgery, and 14 at other hospital departments; 6 were examined at the emergency ward and were not hospitalized.

The study was approved by the ethics committee of the University of Lund. Informed consent to participate was given.
by all patients included (or relatives if the patients were not able to communicate).

Clinical Evaluation

Patients admitted to the Department of Neurology were examined by a neurologist at the emergency ward. Within 1 week after the acute stroke onset the patients were also clinically examined by one of the authors (A.L. or B.N.). The results of the examinations were registered in a protocol. Based on the clinical features of the initial neurologists' and the authors' examinations, patients were grouped into four clinical subgroups according to the same criteria as described earlier by Bamford et al: (1) total anterior circulation syndrome (TACS); both cortical and subcortical symptoms from anterior and middle cerebral artery territory; (2) partial anterior circulation syndrome (PACS); more restricted and predominantly cortical symptoms from the same arterial territories; (3) lacunar syndrome (LACS); lacunar syndromes in anterior, middle, or posterior cerebral or vertebrobasilar artery territories, including sensorimotor lacunar syndrome; and (4) posterior circulation syndrome (POCS); vertebrobasilar or posterior cerebral artery symptoms.

Subclassification of patients as described above was independently made by two of the authors (A.L. and B.N.), using the protocol with the clinical findings and without knowledge of the neuroradiological findings (thus both examinations of patients with cerebral infarction and hemorrhage were assessed). We measured interobserver agreement with kappa statistics. In cases of disagreement, a joint reevaluation of the clinical diagnosis was made. The side of the lesion, if supratentorial, was clinically evaluated. Patients not immediately admitted to the Department of Neurology were evaluated from patient reports in the same way as described above. Ten of the patients were not examined within 7 days after acute stroke onset, but the symptoms at examination were reported as similar to those in the acute phase.

Brain Imaging

Patients underwent a CT examination of the brain as soon as possible after acute stroke onset. Because CT findings in patients with intracerebral hemorrhage may resemble those of cerebral infarction after 2 weeks, we considered examinations within 15 days as "early." CT scans from day 0 through 2 and day 3 through 15 were assessed separately in some of the analyses. The CT scan was repeated on day 16 through 180 (when possible on day 30 through 60), when an MRI of the brain was also carried out. Although the study protocol allowed CT or MRI follow-up to be performed up to 180 days, if no symptomatic lesion was found, the results of the later-performed CT and MRI were used to identify a symptomatic lesion.

We judged a lesion found on any of the neuroradiological examinations to be the cause of the acute stroke if it was consistent with the clinical symptoms. If the patient had several lesions, the largest one appropriate to the symptoms was presumed to be the acute lesion. To define a symptomatic lesion in each patient we first considered the CT examination performed within 15 days. If no symptomatic lesion was found, the results of the later-performed CT and MRI were used to identify a symptomatic lesion.

The neuroradiological characteristics of the largest symptomatic lesion were compared between patients in the four clinical subgroups. Diffuse, nonfocal white matter changes (leukoaraiosis) were not regarded as focal lesions.

Statistics

The number of patients and relative frequencies of different findings are given. The chi-square test was used to examine differences between clinical subgroups of stroke for nominal scale variables, whereas one-factor ANOVA was used for continuous variables. Means with 95% confidence intervals are given where appropriate. Kappa statistics were used to compare clinical interobserver agreement.

Results

The clinical subtypes of stroke are shown in Table 1, together with details of the number of patients examined with CT, MRI, or autopsy. The median time for "early" CT examination was 3 days, and for "late" CT and MRI it was 49 days after stroke onset. Only 4 patients had a new stroke before CT or MRI was performed on day 16 through 180. They were not included in the follow-up analysis.

The overall interobserver agreement in the clinical assessment of the 228 patients regarding clinical stroke subtypes was 92%. The kappa value for this agreement was 0.89 (P < .001).

Intracerebral Hemorrhage

Intracerebral hemorrhages were found in 28 (12%) of the 228 patients (Table 2). The proportion of patients with hematomas differed significantly between the four clinical subgroups (chi-square test; P < .01) and was highest for patients with TACS (19%). By contrast, no patient with LACS was found to have intracerebral hemorrhage.

Cerebral Infarctions

The clinical stroke subtypes of the 200 patients with cerebral infarction are shown in Table 2.
2 the majority of patients with TACS and PACS had a visible symptomatic infarction (Table 3). Fig 2 shows the cumulative proportions of patients with a detectable symptomatic infarction, when results from CT after 16 through 180 days and MRI are added to findings on CT day 0 through 15.

### Volume of Infarctions

The mean volumes of infarctions in the clinical subtypes of ischemic stroke are shown in Table 4 for patients examined with both early and late CT and MRI (n=103). In patients with a cerebral infarction visible on all three examinations, the mean volumes of the infarctions differed significantly between the clinical subgroups TACS, PACS, and LACS (Fig 3). However, between the three different neuroradiological examinations, there were no significant changes in mean volumes (Fig 3).

We also calculated the mean volume of all symptomatic infarctions detected on any of the neuroradiological examinations, beginning the algorithm with the findings on MRI, followed by results from late CT and thereafter early CT, had MRI not been performed or not disclosed any lesion. Similar findings were seen in this analysis with mean volumes ranging from 91.8 mL (TACS) to 4.2 mL (LACS), a more than 20-fold difference in size (ANOVA for comparison between subgroups; \( P=.0001 \)) (Table 5).

### Cortical Involvement

On CT within 15 days, cortical involvement (ie, cortical or cortical and subcortical location) of the symptomatic infarction was present in the different subgroups in the following proportions: TACS, 79%; PACS, 57%; LACS, 9%; POCS, 33%; and all subgroups combined, 53%. The \( \chi^2 \) test showed that this difference between the subgroups was highly significant (\( P=.0001 \)). On CT after 16 through 180 days, the findings were similar with cortical involvement as follows: TACS, 81%; PACS, 58%; LACS, 8%; POCS, 45%; and all subgroups combined, 49% (\( \chi^2 \) test; \( P=.0001 \) for difference between subgroups).

MRI more often (24%) than CT (8% to 9%) disclosed a cortical involvement of the visible infarction in patients with LACS. In patients with TACS or PACS, cortical involvement was seen in similar proportions on MRI and CT examinations. If the 16 patients that were not examined with autopsy or CT within 15 days were removed from the analysis, cortical involvement on CT day 16 through 180 occurred in 4% and on MRI in 21% of patients with LACS.

### Location in Carotid or Vertebrobasilar Artery Territory in Patients With Cerebral Infarction

We registered the presence of any lesion (without defining the lesions as symptomatic) within the posterior circulation territory in patients with at least one visible lesion on neuroradiological examination. On CT on day 0 through 15, such findings were seen in patients with TACS, 12%; PACS, 29%; LACS, 21%; POCS, 86%; and all subgroups combined, 28% (\( \chi^2 \) test; \( P=.0001 \) for difference between clinical subgroups). The corresponding findings on MRI were: in patients with TACS, 38%; PACS, 17%; LACS, 34%; POCS,
89%; and all subgroups combined, 36% ($\chi^2$ test; $P=.003$ for difference between subgroups).

Silent Lesions

A total of 70 "silent" lesions were found in 43 (24%) of 179 patients with cerebral infarction on CT day 0 through 15. Of the 70 silent lesions, 61 (87%) were infarctions and 9 (13%) were other lesions (meningioma, local calcification, arachnoidal cyst, and lipoma of plexus choroideus). Among the 61 silent infarctions seen in 35 patients, 54 were supratentorial (7 cortical, 45 subcortical, 2 both cortical and subcortical), 2 were located in the brain stem, and 5 in the cerebellum. On late CT, a middle cerebral artery aneurysm not seen on early CT was observed in 1 patient. On MRI, 52 (48%) of 108 patients had at least 1 silent lesion (diffuse, nonfocal white matter abnormalities not included). The occurrence of silent lesions did not differ significantly between the clinical subgroups of cerebral infarction (data not shown).

Discussion

Our results show that the four clinical stroke subgroups introduced by Bamford et al. have several distinctive features on CT and MRI of the brain. Between the subgroups, we found significant differences for proportions of patients with intracerebral hemorrhage and symptomatic infarctions visible on CT and MRI, as well as mean volume and location of the infarction causing the clinical symptoms. This bedside stroke classification is simple, easily reproduced, and has earlier been shown to be related to prognosis. The proportions of patients in subgroups of cerebral infarction differed somewhat from the findings by Bamford et al. Both our group and Lindley et al. more often found patients with TACS and more seldom patients with POCS. The observed variation may be due to population differences (age, geographical variations). The interobserver reliability, as measured with kappa statistics was higher in our study than earlier reported. This is probably due to the fact that, in our study, protocols based on the same clinical examinations were used by both observers, whereas in the study by Lindley et al., each patient was examined individually by both observers. Bamford's classification depends only on clinical observations, in contrast to another recent classification in which neuroimaging is necessary for the categorization of stroke patients.

Main Neuroradiological Findings in Stroke Subgroups

Total Anterior Circulation Syndrome

Cerebral hemorrhage was found in no less than 19% of patients with TACS. Thus, approximately 1 of 5 patients with TACS can be expected to have an intracerebral hematoma, which is of importance in selection of patients for clinical trials of acute phase treatment of cerebral infarctions. A CT scan on day 0 through 2

![Graph showing patients with cerebral infarctions classified into clinical subgroups](image1.png)

![Graph showing patients with cerebral infarction examined with computed tomography (CT) on day 0 through 15 and day 16 through 180 and with magnetic resonance imaging (MRI) (n=103)](image2.png)

Table 3. Number of Patients With a Symptomatic Infarction and All Patients Examined in Clinical Subgroups of Cerebral Infarction

<table>
<thead>
<tr>
<th>Examination</th>
<th>TACS</th>
<th></th>
<th></th>
<th>PACS</th>
<th></th>
<th></th>
<th>LACS</th>
<th></th>
<th></th>
<th>POCS</th>
<th></th>
<th></th>
<th>All</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>CT day 0-2*</td>
<td>15/22</td>
<td>68</td>
<td>13/33</td>
<td>57</td>
<td>5/23</td>
<td>22</td>
<td>5/13</td>
<td>38</td>
<td>38/81</td>
<td>47</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>CT day 3-15*</td>
<td>24/29</td>
<td>83</td>
<td>24/32</td>
<td>75</td>
<td>17/24</td>
<td>71</td>
<td>7/13</td>
<td>54</td>
<td>72/98</td>
<td>73</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CT day 16-180</td>
<td>27/30</td>
<td>90</td>
<td>26/38</td>
<td>68</td>
<td>26/40</td>
<td>65</td>
<td>11/18</td>
<td>61</td>
<td>90/126</td>
<td>71</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRI day 16-180</td>
<td>21/23</td>
<td>91</td>
<td>22/32</td>
<td>69</td>
<td>29/38</td>
<td>76</td>
<td>8/15</td>
<td>53</td>
<td>80/108</td>
<td>74</td>
<td></td>
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</tr>
</tbody>
</table>

TACS indicates total anterior circulation syndrome; PACS, partial anterior circulation syndrome; LACS, lacunar syndrome; POCS, posterior circulation syndrome; Sym, symptomatic infarction; All, all patients examined; CT, computed tomography; and MRI, magnetic resonance imaging.

*Patients were examined either on day 0-2 or day 3-15.
visualized the majority (68%) of cerebral infarctions in patients with TACS. Cerebral infarctions in patients with TACS involved the cerebral cortex in approximately 80% of cases and had larger volumes than infarctions in the other clinical subgroups. The mean infarction volume in patients with TACS was approximately four times larger than in patients with PACS. Thus, in patients with TACS, CT of the brain early after stroke onset is useful in identification of subjects with hemorrhage, and in case of infarction, the early CT scan provides data about location and size of the lesion in most patients.

**Partial Anterior Circulation Syndrome**

Compared with patients with TACS, the symptomatic infarctions in patients with PACS were about four times smaller, less frequently involved the cerebral cortex, and were visible in 57% of CT scans on day 0 through 2. An additional 15% to 20% of the infarctions in patients with PACS were visible on CT scans after day 2 or on MRI. It may therefore be useful to repeat the examination later on, if visualization of the infarction is required. Supratentorial symptomatic infarcts were subcortical in no less than 43% of patients with PACS. Recent classifications have stressed the importance of separating different types of subcortical infarctions.8 Small-vessel disease may be the most common cause of lacunar infarctions associated with LACS. However, several other types of subcortical infarctions are associated with cortical symptoms (and thus would be classified as PACS or TACS) and mechanisms other than penetrating artery disease.

**Lacunar Syndrome**

Case reports and studies of small series of patients have shown that any of the lacunar syndromes may be caused by intracerebral hemorrhage, although this probability is rare in a population-based series of stroke patients like ours.10,11 A cerebral infarction causing LACS was more likely to be detected if the CT examination was performed more than 2 days after stroke onset: only 22% of infarctions were seen on day 0 through 2, compared with 65% to 71% after day 2, which is consistent with previous findings.12 Thus, late neuroimaging studies are especially useful in this clinical subtype of stroke. The infarctions were often subcortical and had small volumes. In our study, cortical involvement was seen on MRI in no less than 24% (95% confidence interval [CI], 8.6% to 39.7%). This CI overlapped that of a recent report,13 where 8 of 78 (10%; 95% CI, 3.5% to 17.0%) patients with LACS had a cortical involvement on MRI.

**Posterior Circulation Syndrome**

Only 39 patients in our study had POCS. However, almost 1 of 5 (18%) of these patients had an intracerebral hemorrhage, a proportion similar to that in patients

### Table 4. Mean Volume of Symptomatic Lesion in Patients With Cerebral Infarction Examined With All Neuroradiological Examinations

<table>
<thead>
<tr>
<th>Examination</th>
<th>TACS</th>
<th>PACS</th>
<th>LACS</th>
<th>POCS</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CT day 0-15</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean volume, mL</td>
<td>85.0</td>
<td>15.4</td>
<td>2.8</td>
<td>6.4</td>
<td>.001</td>
</tr>
<tr>
<td>Cl</td>
<td>43.0-127.1</td>
<td>5.8-25.0</td>
<td>0.1-5.5</td>
<td>0-21.3</td>
<td>17.8-50.4</td>
</tr>
<tr>
<td>n</td>
<td>19</td>
<td>19</td>
<td>16</td>
<td>4</td>
<td>58</td>
</tr>
<tr>
<td><strong>CT day 16-180</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean volume, mL</td>
<td>80.4</td>
<td>10.2</td>
<td>2.3</td>
<td>3.3</td>
<td>.001</td>
</tr>
<tr>
<td>Cl</td>
<td>44.5-116.4</td>
<td>3.6-16.8</td>
<td>0.6-4.1</td>
<td>0-6.8</td>
<td>14.4-39.9</td>
</tr>
<tr>
<td>n</td>
<td>20</td>
<td>21</td>
<td>22</td>
<td>7</td>
<td>70</td>
</tr>
<tr>
<td><strong>MRI day 16-180</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean volume, mL</td>
<td>81.8</td>
<td>11.9</td>
<td>2.7</td>
<td>3.9</td>
<td>.001</td>
</tr>
<tr>
<td>Cl</td>
<td>46.2-117.3</td>
<td>5.1-18.7</td>
<td>0.9-4.6</td>
<td>0-9.9</td>
<td>15.2-39.9</td>
</tr>
<tr>
<td>n</td>
<td>21</td>
<td>21</td>
<td>26</td>
<td>7</td>
<td>75</td>
</tr>
</tbody>
</table>

TACS indicates total anterior circulation syndrome; PACS, partial anterior circulation syndrome; LACS, lacunar syndrome; POCS, posterior circulation syndrome; CT, computed tomography; MRI, magnetic resonance imaging; and Cl, 95% confidence interval.

*ANOVA for comparison between subgroups.

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Note: only patients with viable infarction on til 3 examination included.

**Figure 3.** Graph showing mean volume (with 95% confidence intervals [CI]) of infarction in clinical subgroups of patients with a visible cerebral infarction on computed tomography (CT) on day 0 through 15 and day 16 through 180 and magnetic resonance imaging (MRI) (n=51). TACS indicates total anterior circulation syndrome; PACS, partial anterior circulation syndrome; and LACS, lacunar syndrome.
with TACS. The main use of CT in patients with POCS is to detect an intracerebral hemorrhage or large infarctions in the posterior cerebral artery territory or the cerebellum. If no lesion is found on CT, an MRI of the brain may be performed to identify small brain-stem lesions. Symptomatic and nonsymptomatic infarctions taken together were more often located in the posterior circulation territory in patients with POCS when compared with the other clinical groups. Patients with POCS often did not have a visible lesion on CT or MRI (Table 3), perhaps because of difficulty in detecting small brain-stem infarctions, as has been reported earlier.

It is surprising that the symptomatic infarction in patients with POCS was seen more often on CT day 16 through 180 than on MRI day 16 through 180 (61% versus 53%). However, the 95% CIs for these proportions overlap (38.6% to 83.6% versus 28.1% to 78.6%).

Silent Lesions in Patients With Brain Infarction

Earlier studies have reported a proportion of 10% and 11% of silent infarctions in stroke patients and 22% of autopsy-confirmed cerebral infarcts. In one recent study, 18% of patients with minor ischemic stroke had silent infarction, and in another report as many as 38% of patients with first-ever stroke had silent brain infarction. On CT day 0 through 15, 20% of our patients with cerebral infarction had at least one silent infarction. It is possible that recent CT equipment is more sensitive in detecting small lesions than the CT scanners used in earlier studies.

We recognize the difficulties in identifying a brain lesion as symptomatic. In patients with several lesions, MRI studies with gadolinium have been shown to facilitate identification of the symptomatic lesion.

No Visible Symptomatic Lesion on Neuroradiological Examination of Patients With Stroke

The sensitivity to detect a symptomatic infarction on CT increased from day 0 through 2 to day 3 through 15, but even on late CT and MRI about 25% to 30% of patients with cerebral infarction had no visible lesion. In the groups of LACS and POCS this possibly may have been due to difficulties in visualizing the small lesion (located either cortically, subcortically, or in the brain stem) responsible for the symptoms. The severity and duration of clinical symptoms may also affect the probability of detection of a symptomatic lesion on CT or MRI of the brain. Patients with stroke and neuroradiological examinations without visible focal lesions have been reported by others.

On MRI examinations, one difficulty is to delineate a symptomatic lesion from diffuse white matter abnormalities. Small focal subcortical lesions are sometimes difficult to separate from white matter changes. Some characteristics distinguish cerebral infarctions from diffuse white matter changes: Subcortical infarcts may extend into the cortex; they often involve the internal capsule, the basal ganglia, or thalamus; they are well demarcated and located in a specific vascular territory; they are asymmetrical; and they may (if they are cystic) have a low signal on proton density-weighted MRI images. It is possible that more symptomatic lesions would have been identified with high-field-strength MRI equipment.

Methodological Aspects

In the present study, we did not attempt to describe detailed anatomic features on neuroimaging because this issue needs to be addressed in the context of pathophysiological mechanisms assessed by echocardiography and ultrasonography of the carotid arteries. This important topic is of considerable complexity and will be reported separately. We cannot be sure whether the largest appropriate lesion found on CT or MRI was the symptomatic lesion in the individual patients in our study. The reason for selecting only the largest appropriate infarction was to obtain a clear definition of which lesion was analyzed and avoid confounding. For the same reason, we have not made a subanalysis of the smaller lesions in patients with multiple infarctions. The reasons for delay of the acute CT scan were: (1) patients were sometimes admitted 1 or several days after stroke onset, (2) patients were admitted to departments other than the Department of Neurology, or (3) patients sometimes had to wait for the CT examination to become available. If the 16 patients that were not examined with autopsy or CT within 15 days were removed from the analysis, the results were similar except that cortical involvement of LACS on CT and MRI on day 16 through 180 was less common (see above).

Conclusions

We conclude that in patients with first-ever stroke, the clinical stroke classification suggested by Bamford et al is significantly related to several distinctive features on CT and MRI examination of the brain, such as frequency of bleeding, lesion volume, cortical engagement, and involvement of posterior circulation territory. We believe that our study supports the assertion that the clinical classification suggested by the Oxford group is useful in the clinical evaluation of patients with stroke. However, it remains for future studies to assess...
how these clinical and neuroradiological findings correlate to heart and carotid disease and other cerebrovascular risk factors.

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