Hemodynamic Assessment of Acute Stroke Using Dynamic Single-Slice Computed Tomographic Perfusion Imaging

Joachim Röther, MD; Lars Jonetz-Mentzel, MD; Anke Fiala, MS; Jürgen R. Reichenbach, PhD; Michael Herzau, MD; Werner A. Kaiser, MD; Cornelius Weiller, MD

Background: Stroke management would benefit from a broadly available imaging tool that detects perfusion deficits in patients with acute stroke.

Objective: To determine the role of dynamic, single-slice computed tomographic (CT) perfusion imaging (CTP) in the assessment of acute middle cerebral artery stroke.

Design and Patients: Imaging with CTP and CT within the first 6 hours of symptom onset and before the start of treatment in a consecutive clinical series of 22 patients (mean age, 68.3 years; 14 women; studied within 143±96 minutes of stroke onset).

Setting: A stroke unit in a university hospital.

Main Outcome Measures: Area of the perfusion deficit (nAP₀) from time-to-peak maps, hemispheric lesion area from follow-up CT (HLAF), final infarct volume, and stroke recovery (National Institutes of Health Stroke Scale scores).

Results: Eighteen patients had perfusion deficits in the middle cerebral artery territory and corresponding hypodensity in follow-up CT. Three patients with normal CTP findings showed lacunar infarctions or normal findings on follow-up CT. In 1 patient, CTP did not reveal a territorial deficit above the imaging slice. The overall sensitivity and specificity of CTP for the detection of perfusion deficits in patients with proven territorial infarction (n=18) on follow-up CT were 95% and 100%, respectively. The nAP₀ was significantly correlated with the National Institutes of Health Stroke Scale score at admission (P<.003) and the HLAF (P<.001). Different stroke patterns were identified in patients with follow-up CTP (n=10): (1) initial perfusion deficit and partial nutritional reperfusion (nAP₀>HLAF; n=6), (2) initial perfusion deficit and nonnutritional reperfusion (nAP₀≥HLAF; n=2), and (3) initial perfusion deficit without reperfusion (nAP₀≥HLAF; n=2).

Conclusions: Computed tomographic perfusion imaging detects major perfusion deficits in the middle cerebral artery territory. Because CTP is broadly available, it may play a role in acute stroke management.

Arch Neurol. 2000;57:1161-1166

Although the efficacy of intravenous thrombolytic therapy with recombinant tissue-type plasminogen activator for acute stroke was recently shown,¹ the beneficial effect of this potentially hazardous treatment is still under discussion.²⁻⁵ One critical point that remains is the poorly defined stroke mechanism of patients enrolled in acute thrombolytic trials. The detection of malperfused brain tissue in the acute stage would help to limit thrombolysis to patients who are likely to benefit² and might set the rationale for extending the therapeutic window to later time points. Different imaging strategies are available for the definition of perfusion deficits within the acute time window: (1) positron emission tomography,⁶⁻⁷ (2) single photon emission computed tomography (CT),⁸⁻¹² (3) xenon-CT,¹³ and (4) perfusion- or diffusion-weighted magnetic resonance imaging (MRI)¹⁴⁻¹⁸ all provide information on ischemic tissue; however, the application of these techniques is limited to specialized stroke centers. Especially modern MRI protocols that include diffusion- and perfusion-weighted sequences are promising tools that might provide the information necessary for acute stroke management. The combination of diffusion- and perfusion-weighted MRI might help define tissue at risk of infarction. However, at present, MRI has to prove that hemorrhages can be safely excluded and has to answer the question, “What is the therapeutic and prognostic value of deficit mismatches of diffusion- and perfusion-weighted images?”

From the Department of Neurology (Drs Röther and Weiller and Ms Fiala) and the Institute of Diagnostic and Interventional Radiology (Drs Jonetz-Mentzel, Reichenbach, Herzau, and Kaiser), Friedrich-Schiller-University Jena, Jena, Germany.
PATIENTS AND METHODS

Patients with symptoms of acute middle cerebral artery ischemia were prospectively studied after informed consent was obtained. Patients were included if they presented with acute onset of stroke symptoms within 6 hours before CTP. Patients were excluded if they had preexisting neurologic disease or previous stroke that would hamper interpretation of imaging data, a history of hyperthyroidism, or known allergy against iodinated contrast agent. Patients with spontaneous improvement of symptoms before CTP and with abnormal CT findings not consistent with acute cerebral ischemia were excluded from further evaluation. Only patients with technically adequate CT and CTP studies were further evaluated.

The therapeutic decision was based on CT and CTP results. Patients who met National Institute of Neurological Disorders and Stroke inclusion criteria and displayed a percent time delay of >6 seconds in the affected hemisphere were prospectively studied after informed consent. Patients who met stroke inclusion criteria and displayed a percent time delay of >6 seconds in the affected hemisphere were prospectively studied after informed consent. Patients who had a percent time delay of >6 seconds in the affected hemisphere were prospectively studied after informed consent.

Patients who were eligible for participation in clinical acute stroke trials were enrolled in Therapy of Patients With Acute Stroke (n = 1), the European Stroke Treatment With An-crod Trial (n = 1), or the GLYB3001-GAIN (n = 1). Some patients were included in a previous study that validated the CTP methods.

CLINICAL EVALUATION

All patients were evaluated according to the National Institutes of Health Stroke Scale (NIHSS) score immediately before CT and CTP (NIHSS0) and after 3 weeks (NIHSS3wks). Neurologic recovery was assessed as the difference between the NIHSS0 and NIHSS3wks scores (NIHSS0−NIHSS3wks). All clinical assessments were performed by a neurologist (J.R.) or a neurologic resident.

CT IMAGING

Eighteen axial images (120 kV, 500 mA) were acquired in the orbitomeatal orientation using a slip-ring CT scanner (Somatom Plus S; Siemens Medical System, Erlangen, Germany). Five-millimeter-thick slices with 8-mm table feed rate were used. Initial CT images were evaluated for early ischemic CT signs as defined by von Kummer et al.

For dynamic CT perfusion studies, a series of 25 (the first 10 studies) or 40 CT scans were acquired in a single slice (120 kV, 165 mA, 10-mm slice thickness, 512 x 512 image matrix) during injection of 60 mL of nonionic contrast agent (Ultravist 300; Schering, Berlin, Germany) using a power injector (10 mL/s). The contrast agent was injected into the antecubital vein via an 18-gauge intravenous catheter. The injection was started simultaneously to the acquisition of the serial CT data. The total acquisition time was 40 seconds. A standardized slice position covering the basal ganglia; the thalamus; and parts of the anterior, middle, and posterior arterial territories was assessed (Figure).

PARAMETER MAPS AND SEMIQUANTITATIVE ANALYSIS

All CT and CTP scans were independently evaluated by a trained stroke neurologist and a radiologist (J.R. and L.J.M.) who were informed only that the CT scans were those of patients with clinically suggested stroke.

The CTP data were transferred to a SUN workstation (SUN Microsystems, Mountain View, Calif) for semiquantitative image analysis using custom software (MRVision Co, Menlo Park, Calif). Assuming that no contrast agent leaks into the parenchyma, tracer kinetics can be applied and the attenuation time curve can be transformed to a concentration time curve, since the attenuation change is linearly dependent on the amount of contrast agent in the tissue. Parameter maps of TTP, the time from the start of the contrast agent injection to the maximum bolus peak, were calculated. A TTP delay of 6 seconds or more was considered indicative of a perfusion deficit. Maps of TTP were evaluated because they proved to be a robust parameter in previous experimental and human perfusion-weighted MRI studies.

The area with a perfusion deficit was manually defined on color-coded TTP maps. Areas with perfusion deficits were then normalized to the ipsilateral hemisphere (normalized area of perfusion deficit [nAP = area of perfusion deficit/area of ipsilateral hemisphere x 100]). The area of hypodensity in the follow-up CT was calculated as a percentage of the hemispheric lesion area (HLA = area of hypodensity/hemispheric area x 100). The percentage of saved or lost tissue was calculated as (nAP0−HLA0)/nAP0 x 100. In 10 patients with a follow-up CT study 24 hours after stroke onset (nAP1), the reperfusion area was determined in percentage as (nAP0−nAP1)/nAP0 x 100.

To address the question of how reliable a single-slice approach in the chosen location would mirror the infarct volume, the total infarct volume was calculated from the follow-up CT. The total infarct volume, expressed as a percentage of infarcted tissue normalized to the ipsilateral hemispheric volume, was then correlated with the nAP0 and the HLA0.

STATISTICAL ANALYSIS

Demographic data are presented as mean ± SD. The Spearman correlation coefficient was used as an estimate of the strength of association between CT, CTP variables, and clinical measures. Results were considered statistically significant at the 5% level.

Dynamic CT perfusion imaging (CTP) might overtake the role of an imaging modality that detects cerebral perfusion deficits in the acute stage and that is easily accessible to a broad community. Imaging with CTP is an easily performed add-on examination that requires a slip-ring CT scanner and takes only a few minutes. The tissue attenuation during the first pass of an intravenously applied contrast bolus through the brain tissue is tracked by serial CT acquisitions.
Semiquantitative parameter images of the time to peak (TTP), cerebral blood flow, mean transit time, and cerebral blood volume delineate the perfusion deficit. The obvious disadvantage of the technique is the restriction to a single-slice measurement.

We applied CTP to patients with acute stroke within the first 6 hours of stroke onset to test its utility and sensitivity for the detection of perfusion deficits. We hypothesized that (1) the single-slice approach is sensitive for the detection of major, clinically relevant perfusion deficits; (2) the area of the initial perfusion deficit in CTP correlates with the severity of stroke symptoms, clinical recovery, and final infarct size; and (3) the reperfusion area and areas of saved tissue (nutritional reperfusion) as detected by follow-up CT and CTP are useful indicators of therapeutic efficacy.

RESULTS

Three studies were excluded because of motion artifacts in CTP. Four patients with stroke onset within the 6-hour time window and known hyperthyroidism were not eligible. One patient with previously unknown hyperthyroidism developed atrial fibrillation. The remaining 22 consecutive patients were further evaluated. Ages ranged from 52 to 84 years (mean age, 68.3 years; 14 women), and the NIHSS0 was 13.2±5.2. The mean interval between the onset of symptoms and CT or CTP scanning was 143±96 minutes (range, 30-360 minutes).

Eighteen patients had a perfusion deficit on CTP (group A), whereas 4 patients had normal CTP results (group B). The nAP0 but not final infarct volume and HLAcf correlated significantly with the NIHSS0 (P<.003, Spearman correlation coefficient). Neurologic recovery, measured as the difference between NIHSS0 and NIHSS3wk, correlated with the reperfusion area (P<.04, Spearman correlation coefficient) but not with the percentage of lost or saved tissue.

PATIENTS WITH PERFUSION DEFICIT ON CTP (GROUP A)

Eighteen patients showed perfusion deficits on the TTP parameter maps of the initial CTP study. The mechanism of infarction according to the criteria of the Trial of Org 10172 in Acute Stroke Treatment was cardioembolism in 5 patients (28%), large-artery atherosclerosis in 8 (44%), and undetermined in 6 (33%). Six patients met the National Institute of Neurological Disorders and Stroke criteria for thrombolytic treatment and received recombinant tissue-type plasminogen activator immediately after CT within the 3-hour time window. Eight patients were included 3 to 6 hours after stroke onset and received intravenous heparin, aiming to double the partial thromboplastin time. Four patients were enrolled in acute stroke trials (Therapy of Patients With Acute Stroke, the European Stroke Treatment With Ancrod Trial, and GLYB3001-GAIN) 3 to 6 hours after stroke onset. Group data are summarized in the Table.

The nAP0 correlated with the HLAcf (P<.001, Spearman correlation coefficient). The infarct evolution as calculated from the difference between the nAP0 and the HLAcf was expressed as the lost/saved tissue index. The mean saved tissue index was 24.3%±46.9%. The saved tissue index did not correlate with neurologic recovery. In 10 patients with follow-up CTP the first day after stroke onset the mean reperfusion area was calculated as a percentage of the nAP0 (36.3%±41.4%); the reperfusion area...
correlated with neurologic recovery (P < .04, Spearman correlation coefficient).

Three different patterns of stroke evolution were observed: (1) Areas of hypoattenuation on the follow-up CT scan that were smaller than the initial perfusion deficit and showed reperfusion on the follow-up CTP scan were classified as (partial) nutritional reperfusion (nAP0

HLAF = saved tissue; n = 6). (2) Areas of hypoattenuation on the follow-up CT scan equal to the area of the initial perfusion deficit and reperfusion in the follow-up CTP scan were classified as nonnutritional reperfusion (nAP0

HLAF = lost tissue; n = 2). (3) Areas of hypoattenuation on the follow-up CT scan equal to the area of the initial perfusion deficit without reperfusion on the follow-up CTP scan (nAP0

HLAF = lost tissue; n = 2).

We addressed the question of how reliably a single-slice approach in the chosen location would mirror the total infarct volume and found that the nAP0 and the HLAF significantly correlated with the final infarct volume (nAP0: P < .008; HLAF: P < .001, Spearman correlation coefficient).

PATIENTS WITHOUT PERFUSION DEFICIT ON CTP (GROUP B)

In 4 patients (group B) with normal initial CT and CTP results, the follow-up CT scan was either normal (n=2) or showed lacunar stroke (n=1). In 1 patient with an infarction of the prefrontal artery, the perfusion deficit was not revealed. The mechanisms of infarction were small-vessel occlusion (n=3) and cardioembolism (n=1). Patients were treated with aspirin (n=1) or heparin (n=2) or were enrolled in the Therapy of Patients With Acute Stroke trial (n=1) (Table).

The results of this study show that CTP is sensitive for the detection of territorial perfusion deficits in the middle cerebral artery territory and is easily applicable with minimal additional scan time. The information from CTP is readily available from TTP parameter maps.

**Summary of Individual Clinical and CT Data**

<table>
<thead>
<tr>
<th>Patient No./ Sex/Age, y</th>
<th>Delay Time, min</th>
<th>NIHSS2</th>
<th>NIHSS-Diff</th>
<th>EIS</th>
<th>nAP0†</th>
<th>nAP1†</th>
<th>HLAF†</th>
<th>HLV†</th>
<th>Saved/Lost Tissue, %‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/60</td>
<td>100</td>
<td>18</td>
<td>9</td>
<td>9</td>
<td>No</td>
<td>36.7</td>
<td>6.9</td>
<td>11.9</td>
<td>21.7</td>
</tr>
<tr>
<td>2/F/74</td>
<td>166</td>
<td>11</td>
<td>10</td>
<td>1</td>
<td>Yes</td>
<td>25.2</td>
<td>19.8</td>
<td>8.5</td>
<td>9.6</td>
</tr>
<tr>
<td>3/F/67</td>
<td>120</td>
<td>20</td>
<td>15</td>
<td>5</td>
<td>Yes</td>
<td>50.5</td>
<td>48.0</td>
<td>60.4</td>
<td>61.8</td>
</tr>
<tr>
<td>4/F/69</td>
<td>120</td>
<td>14</td>
<td>5</td>
<td>9</td>
<td>No</td>
<td>23.2</td>
<td>NA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5/F/66</td>
<td>75</td>
<td>12</td>
<td>18</td>
<td>−6</td>
<td>Yes</td>
<td>28.7</td>
<td>34.8</td>
<td>33.2</td>
<td>35.1</td>
</tr>
<tr>
<td>6/F/65</td>
<td>60</td>
<td>17</td>
<td>7</td>
<td>10</td>
<td>No</td>
<td>46.2</td>
<td>0.4</td>
<td>13.7</td>
<td>23.0</td>
</tr>
<tr>
<td>7/F/62</td>
<td>60</td>
<td>21</td>
<td>34</td>
<td>−13</td>
<td>No</td>
<td>53.7</td>
<td>61.6</td>
<td>52.4</td>
<td>60.7</td>
</tr>
<tr>
<td>8/F/57</td>
<td>240</td>
<td>11</td>
<td>15</td>
<td>6</td>
<td>No</td>
<td>33.6</td>
<td>0</td>
<td>35.1</td>
<td>34.2</td>
</tr>
<tr>
<td>9/F/77</td>
<td>30</td>
<td>14</td>
<td>13</td>
<td>1</td>
<td>No</td>
<td>39.6</td>
<td>NA</td>
<td>43.9</td>
<td>39.6</td>
</tr>
<tr>
<td>10/F/67</td>
<td>70</td>
<td>14</td>
<td>34</td>
<td>−20</td>
<td>No</td>
<td>26.1</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>11/M/77</td>
<td>117</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>No</td>
<td>16.9</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>12/M/78</td>
<td>90</td>
<td>12</td>
<td>34</td>
<td>−12</td>
<td>No</td>
<td>51.7</td>
<td>NA</td>
<td>69.5</td>
<td>60.1</td>
</tr>
<tr>
<td>13/M/68</td>
<td>120</td>
<td>20</td>
<td>34</td>
<td>−14</td>
<td>Yes</td>
<td>31.2</td>
<td>18.9</td>
<td>5.2</td>
<td>6.7</td>
</tr>
<tr>
<td>14/F/59</td>
<td>285</td>
<td>19</td>
<td>34</td>
<td>−15</td>
<td>Yes</td>
<td>48.2</td>
<td>NA</td>
<td>41.8</td>
<td>63.3</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>69.2 ± 8.8</td>
<td>142.4 ± 98.9</td>
<td>14.6 ± 4.7</td>
<td>17.0 ± 13.0</td>
<td>−1.8 ± 10.2</td>
<td>34.1 ± 14.4</td>
<td>23.8 ± 20.2</td>
<td>28.1 ± 22.1</td>
<td>30.7 ± 22.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient No./ Sex/Age, y</th>
<th>Delay Time, min</th>
<th>NIHSS2</th>
<th>NIHSS-Diff</th>
<th>EIS</th>
<th>nAP0†</th>
<th>nAP1†</th>
<th>HLAF†</th>
<th>HLV†</th>
<th>Saved/Lost Tissue, %‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>19/F/72</td>
<td>240</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>No</td>
<td>0</td>
<td>NA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>20/M/68</td>
<td>105</td>
<td>5</td>
<td>0</td>
<td>5</td>
<td>No</td>
<td>0</td>
<td>NA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>21/M/64</td>
<td>254</td>
<td>9</td>
<td>5</td>
<td>4</td>
<td>No</td>
<td>0</td>
<td>NA</td>
<td>1.7</td>
<td>0</td>
</tr>
<tr>
<td>22/M/52</td>
<td>165</td>
<td>9</td>
<td>14</td>
<td>−5</td>
<td>No</td>
<td>3.0</td>
<td>16.5</td>
<td>26.7</td>
<td>NA</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>64 ± 7.5</td>
<td>191 ± 60.1</td>
<td>6.75 ± 2.3</td>
<td>5.5 ± 5.2</td>
<td>1.3 ± 3.9</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

*CT indicates computed tomography; CTP, computed tomographic perfusion imaging; NIHSS0, National Institutes of Health Stroke Scale (NIHSS) score; before CT and CTP; NIHSS-Diff, neurologic recovery assessed as the difference between NIHSS0 and NIHSS3wk scores; EIS, early ischemic CT signs; nAP0, area of perfusion deficit at baseline; nAP1, area of perfusion deficit with a follow-up CTP; HLAF, hemispheric lesion area from follow-up CT; HLV, final infarct volume; NA, not applicable; ACAO, anterior cerebral artery occlusion; CE, cardioembolism; dCAO, distal internal carotid artery occlusion; L, left; LA, large-artery atherosclerosis; MCAO, middle cerebral artery occlusion; dMCAO, distal middle cerebral artery occlusion; DMCA, distal middle cerebral artery; LMWH, low-molecular-weight heparin; R, right; sten, stenosis; SVO, small-vessel occlusion; rtPA, recombinant tissue-type plasminogen activator; GAIN, GLYB3001-GAIN; TOPAS, Therapy of Patients with Acute Stroke; and Ancrod, European Stroke Treatment with Ancrod Trial (ESTAT).
†Numbers indicate the perfusion deficit (nAP0,1) or lesion area (HLAF,V) as a percentage of the ipsilateral hemisphere.
‡Numbers indicate the percentage of saved, lost, or reperfused tissue as described in the “Patients and Methods” section.
The neurologic symptoms at stroke onset (NIHSS0). The farct volume of follow-up CT studies and the severity of studies, correlated with neurologic recovery. These findings are in line with those of previous acute stroke studies that support a correlation between reperfusion areas, the tissue volume saved from infarction, and stroke outcome, although others were not able to reproduce these findings.

Follow-up studies using CTP discriminated different patterns of stroke evolution: (1) Patients with nutritional reperfusion that depicted infarctions smaller than the nAP0, thus indicating the salvage of tissue at risk. In these patients, saved tissue correlated with neurologic recovery. (2) Patients with nonnutritional reperfusion and final infarct size corresponding with the nAP0. (3) Patients without reperfusion and final infarct size corresponding with the nAP0. Similar patterns of stroke evolution previously have been described in a single photon emission CT study and are valuable indicators of stroke evolution.

The drawback of CTP is the restriction to a single-slice approach. However, if applied on the suggested level through the basal ganglia, the sensitivity for the detection of territorial perfusion deficits is high (95%). The population being studied is biased toward patients with perfusion deficits and severe neurologic deficits because patients with acute stroke with severe neurologic deficits are admitted to the hospital faster. Despite this limitation, with the exception of one false-negative study, small-vessel disease was the presumable stroke mechanism in cases of normal CTP findings.

One might argue that CT angiography performed instead of CTP provides useful information about intracranial artery disease. We find that CTP delivers functional information about tissue perfusion that is not available from CT angiography and that artery disease and vessel patency can be likewise assessed by transcranial Doppler sonography and transcranial color-coded duplex sonography. The major drawback of CTP, namely, the limitation to a single slice, will be overcome by the new generation of multi-ring CT systems.

In conclusion, future acute stroke trials might use an imaging protocol applicable to a multicenter setting that identifies patients who are eligible for thrombolysis and yields basic parameters of treatment efficacy. Because CTP is ubiquitously available, it might well overtake this role. The diagnostic value of CTP may be enhanced by combination with transcranial Doppler sonography and transcranial color-coded duplex sonography. The clinical value of CTP in patient selection for thrombolysis during the first hours of acute ischemic stroke remains to be determined within a larger prospective clinical trial.

Accepted for publication January 25, 2000.

Reprints: Joachim Röther, MD, University Hospital Hamburg Eppendorf, Department of Neurology, Martinistr. 52, 20246 Hamburg, Germany (e-mail: rother@uke.uni-hamburg.de).

REFERENCES

2. Grotta J. Should thrombolytic therapy be the first-line treatment for acute ische-

3. Wardlaw JM, Warlow CP, Counsell C. Systematic review of evidence on throm-


11. Baird AE, Austin MC, McKay WJ, Donnan GA. Changes in cerebral tissue perfu-

12. Grotta JC, Alexandrov AV. tPA-associated reperfusion after acute stroke demon-
strated by SPECT [see comments]. Stroke. 1998;29:429-432.

13. Firlik AD, Kaufmann AM, Wechsler LR, Firlik KS, Fukui MB, Yonas H. Quantita-


18. Tong DC, Yenari MA, Albers GW, O’Brien M, Marks MP, Moseley ME. Correla-
tion of perfusion- and diffusion-weighted MRI with NIHSS score in acute (<6.5 hour) ischemic stroke [see comments]. Neurology. 1998;50:864-870.


22. Taniyama T, Tanizaki Y, Hongo K, Otsawa M, Kobayashi S. Functional image of dynamic computed tomography in diagnostic and prognostic evaluation of is-
chemic stroke within the first 6 hours. Stroke. 1993;24:1933-1944.


26. Axel L. Cerebral blood flow determination by rapid-sequence computed tomog-


32. Shrier DA, Tanaka H, Numaguchi Y, Konno S, Patel U, Shibata D. CT angiogra-


34. Postert T, Braun B, Federman J, Przuntek H, Koster O, Buttnner T. Diagnosis and monitoring of middle cerebral artery occlusion with contrast-enhanced trans-

35. Goertler M, Kross R, Bauemer M, et al. Diagnostic impact and prognostic rel-