Diffusion- and Perfusion-Weighted MRI
Influence of Severe Carotid Artery Stenosis on the DWI/PWI Mismatch in Acute Stroke

Tobias Neumann-Haefelin, MD; Hans-Jörg Wittsack, PhD; Gereon R. Fink, MD; Frank Wenserski, MD; Tie-Qiang Li, PhD; Rüdiger J. Seitz, MD; Mario Siebler, MD; Ulrich Mödder, MD; Hans-Joachim Freund, MD

Background and Purpose—Diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) have been used increasingly in recent years to evaluate acute stroke in the emergency setting. In the present study, we compared DWI and PWI findings in acute stroke patients with and without severe extracranial internal carotid artery (ICA) disease.

Methods—Twenty-seven patients with nonlacunar ischemic stroke were selected for this analysis. DWI, PWI, and conventional MRI were performed in all patients within 24 hours of symptom onset and after 1 week. To exclude patients with partial or complete reperfusion, we included only patients with a PWI deficit larger than the DWI lesion. Severe ICA disease (>70% stenosis) was present unilaterally in 9 and bilaterally in 2 patients. Acute DWI lesion volume, the size of the acute PWI/DWI mismatch, and final infarct size (on T2-weighted images) were determined.

Results—The PWI/DWI mismatch was significantly larger in patients with severe ICA disease than in patients without extracranial carotid stenosis, both when time-to-peak and mean transit time maps \( P < 0.01 \) were used to calculate the mismatch. Quantitative analysis of the time-to-peak delay in the mismatch indicated that a relatively smaller fraction of the total mismatch was critically ischemic in patients with carotid stenosis than in those without. Average lesion volume increased less in the stenosis group \( P = 0.14 \), despite the larger PWI/DWI mismatch, and final infarct size was smaller in the stenosis group \( P < 0.05 \). In the 2 patients with bilateral ICA disease, variable hemodynamic involvement of the contralateral hemisphere was found in addition to the ipsilateral PWI deficit.

Conclusions—In most acute stroke patients with severe ICA stenosis, a considerably smaller fraction of the total PWI/DWI mismatch is at risk than in patients without carotid disease. (Stroke. 2000;31:1311-1317.)

Key Words: diffusion ■ magnetic resonance imaging ■ perfusion ■ stroke, acute ■ carotid stenosis

Diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) have been used increasingly in recent years to evaluate acute stroke patients in the emergency setting. Acute DWI abnormalities are markers of critical ischemia1 and typically evolve into infarction, at least in human stroke.2–5 In the first few hours after stroke onset, PWI abnormalities are often larger than the DWI lesions. This pattern (PWI deficit>DWI lesion) is frequently associated with subsequent lesion growth into the PWI/DWI mismatch region, indicating that the mismatch may represent tissue at risk.6–9 For this reason, it has been suggested that DWI and PWI (when used in combination) may allow the identification of optimal candidates for therapeutic interventions including thrombolysis, ie, those patients with a sizeable volume of potentially salvageable tissue at risk.10,11

Although several studies have now evaluated the utility of DWI and PWI in acute stroke, relatively little is known about differences between various subpopulations of stroke patients, such as those with and without severe internal carotid artery (ICA) disease. Severe ICA stenosis or occlusion is one of the important causes of stroke12 and may be associated with various degrees of hemodynamic impairment.13,14 In patients with ICA disease, stroke may be caused by either acute thromboembolism (artery-to-artery embolism) or by an exacerbation of hemodynamic impairment. It is now well established that impaired hemodynamics can be demonstrated with PWI in patients with hemodynamically relevant ICA disease (without prior stroke).15–19 However, previous studies have not addressed the question whether DWI and PWI findings differ between acute stroke patients with and without severe carotid disease and whether this relates to differences in stroke evolution.

The intent of this study was therefore to determine acute DWI lesion volume, the size of the acute PWI/DWI mis-
match, and the change in lesion size in acute stroke patients with and without severe extracranial ICA disease.

Subjects and Methods

Patients
From a larger prospective study (approved by the local ethics committee) investigating the utility of MRI in acute stroke, 27 patients with nonlacunar ischemic stroke (symptom onset <24 hours) were selected for this analysis. Of all 44 patients with nonlacunar ischemic stroke recruited between January 1998 and November 1999, only those with anterior circulation ischemia and a positive PWI/DWI mismatch (n=33) were selected, to exclude patients with partial or complete reperfusion before the acute MR study. In 5 patients, either the acute DWI or PWI lesion volumes could not be determined owing to gross patient motion during DWI or technical problems. One patient was excluded because the result of the cervical ultrasound examination was not available. DWI and time-to-peak (TTP) results for 10 of these patients were reported previously.9

MRI Protocol
All patients included in this analysis were imaged within 24 hours of symptom onset, and follow-up scans were obtained at ~7 days (range 5 to 12 days). The MR measurements were performed on a 1.5-T echo-planar imaging (EPI)–capable whole-body MR scanner (Siemens Magnetom Vision) equipped with a gradient overdrive with the standard head coil. In each patient, the acute study included DWI, PWI, and conventional imaging. The follow-up study consisted of DWI and conventional imaging.

DWI was performed with a single-shot EPI spin-echo sequence (TE 103 ms; field of view 240 mm; 96×128). Each of the 20 axial slices (slice thickness 5 mm; gap 1.5 mm) obtained with DWI was acquired with b-values of 0 and 1000 s/mm²; the high b-value DWI measurements were done with diffusions gradients in the 3 orthogonal (x, y, z) directions in space.

For bolus-tracking PWI, we used a gradient-echo EPI sequence (TE 54 ms; field of view 240 mm; 128×128). We obtained 40 T2*–weighted images for each of the 12 slices (slice thickness 5 mm; gap 1.5 mm) at intervals of 2 seconds. The contrast agent (Magnevist, Schering; 15 mL) was injected into the antecubital vein with an MR-compatible power injector at a rate of 5 mL/s.

Cervical Ultrasound
In all subjects, continuous-wave Doppler sonography and color Doppler–assisted duplex imaging (Acuson XP125) was performed by a neurologist (working on our stroke unit) or a technician with extensive experience in cervical ultrasound. Quantification of ICA stenosis followed the consensus guidelines published by de Bray and Glatt.20 The examinations were done either on the same day or the day after the initial MRI scan.

For analysis of the MR data, we stratified the patients into 3 groups: (1) patients with unilateral severe (>70% to 100%) ICA stenosis (or occlusion) ipsilateral to the symptomatic hemisphere, (2) patients with bilateral severe ICA disease, and (3) patients without severe carotid disease (<50%). In all patients with severe ICA stenosis or occlusion, the diagnosis was confirmed by conventional intra-arterial digital subtraction angiography of the neck vessels.

Postprocessing and Image Analysis
For postprocessing, all MR data were transferred to a SPARC workstation (Sun Microsystems). Images were displayed and processed with the software package MRVision (Menlo Park). To minimize the effects of diffusion anisotropy, trace diffusion-weighted images were generated by averaging the images obtained with DWI in the x, y, and z directions. In addition, apparent diffusion coefficient maps were calculated from the DWI data (mainly to exclude T2 shine-through effects).

PW1 raw images were processed on a pixel-by-pixel basis to generate maps of the TTP, relative mean transit time (rMTT), relative cerebral blood flow (rCBF), and relative cerebral blood volume (rCBV). TTP maps were generated without the use of an arterial input function (AIF) or curve fitting, and the maps were displayed both with arbitrarily adjusted contrast and with thresholds indicating TTP delays of at least 4, 6, or 8 seconds compared with the unaffected hemisphere, as reported previously.9

For the calculation of rMTT, rCBF, and rCBV maps, we used the model-independent SVD method (nonparametric singular value decomposition deconvolution) described by Ostergaard et al.21,22 With this method, rCBF is determined by deconvolution of the tissue concentration time curve with an AIF. We determined the shape of the AIF by manually choosing 5 to 10 pixels over the first 2 segments of the middle cerebral artery (MCA) of the unaffected hemisphere. In the 2 patients with bilateral severe carotid artery disease, pixels over the posterior cerebral artery were chosen to obtain the AIF. To determine rCBV, the tissue concentration time curve was numerically integrated between 2 time points (t1 and t2), which were individually determined for each patient. t1 was chosen from the AIF as a time point before the arterial arrival of the contrast agent, and t2 was chosen from the tissue concentration time curve in ischemic tissue as a time point at which the signal had again completely or almost completely returned to baseline. MTT was then calculated from these measurements as rCBV/rCBF.

Lesion-volume measurements were performed on acute DWI images and TTP and MTT maps by a semiautomated segmentation technique based on seed growing and local thresholding. The images were edited by an independent observer to confine lesion measurements to brain parenchyma (if necessary). This was needed primarily to prevent inclusion of the lateral ventricles. For lesion measurements, we first determined a mean value (±SD) for unaffected contralateral tissue, which was defined by manually outlining a large part of the contralateral MCA territory in 3 central slices. In the 2 patients with bilateral severe carotid stenosis, we used a region in the posterior circulation as reference tissue. For lesion segmentation, we then used the mean±2SD for DWI and TTP maps, and the mean±3SD for MTT maps as thresholds. The higher threshold for MTT maps was used because there was more variation between different slice levels than with DWI and TTP maps. For the TTP maps, we additionally used TTP delays of 4, 6, and 8 seconds as thresholds. We did not measure lesion volumes on rCBF or rCBV maps, because it is difficult to delineate lesion borders on these maps. On the chronic scans, we determined lesion volume on the T2-weighted images by outlining the lesions manually. Finally, the abnormal areas on the images were summed and multiplied with the slice thickness plus interslice gap to calculate lesion volume.

In addition to measuring lesion volumes, a region-of-interest (ROI) analysis was performed to determine relative values of PWI parameters (TTP, rMTT, rCBF, and rCBV) in the DWI lesion center, the PWI/DWI mismatch close to the DWI lesion border, and the periphery of the mismatch (outer third of the mismatch). In each of these 3 regions, as well as in symmetrical contralateral regions, 3 ROIs (4 voxels each) were chosen. The TTP delay and the increase in rMTT in the 3 respective regions were calculated as ipsilateral minus contralateral values. For rCBF and rCBV, hemispheric ratios (ipsilateral divided by contralateral values) were calculated.

To obtain an estimate of the extracranial bolus delay due to severe carotid stenosis or occlusion, we determined the MR signal time course in 5 to 10 pixels over the ipsilateral and contralateral MCA. This was done by the same procedure as for the definition of the AIF. Both bolus arrival time and TTP (TTParrival) were calculated for both sides.

Statistical Analysis
All results are presented as mean±SD. The nonparametric Kruskal-Wallis test was used to test for differences in MRI parameters between patients with and without severe carotid disease. Results were considered statistically significant at the 5% level.
ipsilateral stenosis. Although DWI lesion size was nonsignificantly smaller in the stenosis group (18 versus 41 mL), both the size of the total perfusion deficit (on TTP maps) and the size of the mismatch (on TTP and MTT maps; Figure 2) were significantly larger. When TTP thresholds (TTP delay of 4, 6, or 8 seconds) were used for the mismatch calculation, there was still a trend toward significance when a threshold of 4 seconds was applied (P=0.19) but not with 6- (P=0.95) or 8-second thresholds (P=0.82).

Although the total PWI/DWI mismatch was larger in patients with ipsilateral severe carotid disease, average lesion size increased (nonsignificantly) less in these patients than in patients without stenoses (5 versus 26 mL; P=0.14; see Table 2). The average increase in lesion size (T2WI chronic−DWI acute), expressed as a percentage of the total mismatch (calculated from TTP maps), was 7±8% and 40±50% for patients with and without severe carotid disease, respectively. Final infarct size was smaller in patients with severe stenosis than in those without carotid stenosis (23 versus 67 mL; P<0.05). Scatterplots showing the relationship between the increase in lesion size and the size of the acute PWI/DWI mismatch are presented in Figure 3.

The results of the ROI analysis of all measured PWI parameters (TTP, rMTT, rCBF, and rCBV) are presented in Table 3. Differences between the stenosis group and the patients without carotid stenosis were found particularly in the periphery of the PWI/DWI mismatch and partially also in the vicinity of the DWI lesions, but not in the DWI lesion center. Perfusion abnormalities (TTP delay, rMTT increase, and rCBF reduction) were less severe in the mismatch in the stenosis group than in the group without stenosis, and there was a trend toward higher rCBV values in the mismatch (Table 3).

As expected, bolus characteristics measured over the ipsilateral and contralateral MCA differed between patients with and without severe ICA stenosis. Compared with the contralateral side, bolus arrival on the affected side was delayed by 1.5±2.1 seconds and TTP arterial by 3.1±2.0 seconds in the stenosis group versus 0.5±1.7 and 0.7±2.4 seconds in the group without severe carotid disease (only TTP arterial significantly differed between groups at P<0.05). There was considerable variation between subjects with ICA disease, probably reflecting the quality of collateral flow (Figure 4).

In the 2 patients with bilateral carotid artery disease, most of the ipsilateral MCA territory appeared abnormal on TTP, rMTT, and rCBF maps. In 1 of the 2 patients (Figure 5), the contralateral hemisphere was also clearly abnormal compared with tissue supplied by the posterior cerebral artery, whereas in the other patient, contralateral PWI abnormalities were relatively inconspicuous. Both the initial DWI lesion volume in these 2 patients and the change in lesion size between the acute and the follow-up studies were within the range of values found in the other patients, but owing to the small sample size, no formal comparisons were made.

Discussion

In this study, we compared DWI and PWI findings in acute stroke patients with and without severe carotid stenosis. Our results show that the total PWI/DWI mismatch is often larger
in patients with ipsilateral carotid stenosis >70% than in patients without carotid stenosis. However, several findings of this study indicate that most of the total mismatch in these patients is not at high risk of irreversible tissue damage. First, lesion size did not increase more (between the acute and the follow-up MR studies) in the stenosis group than in the group without stenosis. Second, in the stenosis group, only 7% of the total mismatch (calculated with TTP maps) became recruited into the infarct, compared with 40% for the group without stenosis. Third, semiquantitative analysis of PWI parameters indicated that the severity of the perfusion deficit in the periphery of the mismatch was relatively mild in most patients with carotid stenosis.

Previous studies of acute stroke using DWI and PWI found that a positive PWI/DWI mismatch was associated with subsequent lesion enlargement.6–8 DWI abnormalities are markers of critical ischemia, and at least in humans, the overwhelming majority of DWI lesions evolve into infarction. The PWI/DWI mismatch region, on the other hand, is believed to represent viable tissue at risk that may become irreversible.

**TABLE 2. DWI/PWI Lesion Volume in Acute Stroke Patients With and Without ICA Disease**

<table>
<thead>
<tr>
<th></th>
<th>All* (n=25)</th>
<th>ICA Stenosis (n=9)</th>
<th>No Stenosis (n=16)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>DWI volume (acute), mL</td>
<td>34 (±44)</td>
<td>18 (±23)</td>
<td>41 (±51)</td>
<td>NS</td>
</tr>
<tr>
<td>T2WI volume (follow-up), mL</td>
<td>54 (±53)</td>
<td>23 (±28)</td>
<td>67 (±57)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Change in lesion size, %</td>
<td>56</td>
<td>22</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>PWI volume (acute), mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TTP&lt;sub&gt;total&lt;/sub&gt;</td>
<td>108 (±57)</td>
<td>142 (±47)</td>
<td>85 (±53)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;4s&lt;/sub&gt;</td>
<td>88 (±51)</td>
<td>91 (±43)</td>
<td>81 (±58)</td>
<td>NS</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;6s&lt;/sub&gt;</td>
<td>57 (±44)</td>
<td>50 (±33)</td>
<td>58 (±49)</td>
<td>NS</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;8s&lt;/sub&gt;</td>
<td>39 (±37)</td>
<td>29 (±21)</td>
<td>39 (±42)</td>
<td>NS</td>
</tr>
<tr>
<td>MTT&lt;sub&gt;total&lt;/sub&gt;</td>
<td>123 (±57)</td>
<td>147 (±56)</td>
<td>104 (±57)</td>
<td>NS</td>
</tr>
<tr>
<td>Mismatch (acute), mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TTP&lt;sub&gt;total&lt;/sub&gt;−DWI volume</td>
<td>74 (±55)</td>
<td>124 (±52)</td>
<td>44 (±32)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;4s&lt;/sub&gt;−DWI volume</td>
<td>54 (±47)</td>
<td>73 (±45)</td>
<td>40 (±47)</td>
<td>NS</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;6s&lt;/sub&gt;−DWI volume</td>
<td>23 (±45)</td>
<td>31 (±37)</td>
<td>17 (±48)</td>
<td>NS</td>
</tr>
<tr>
<td>TTP&lt;sub&gt;8s&lt;/sub&gt;−DWI volume</td>
<td>3 (±43)</td>
<td>11 (±30)</td>
<td>−2 (±47)</td>
<td>NS</td>
</tr>
<tr>
<td>MTT&lt;sub&gt;total&lt;/sub&gt;−DWI volume</td>
<td>88 (±55)</td>
<td>129 (±50)</td>
<td>63 (±44)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

T2WI indicates T2-weighted image; TTP<sub>4s</sub>, TTP<sub>6s</sub>, and TTP<sub>8s</sub>, TTP delay thresholds of 4, 6, and 8 seconds, respectively.

*Except patients with bilateral severe ICA disease.

Figure 1. Acute DWI and PWI results in 2 cases with similarly small DWI lesions, one without (A) and one with (B) ipsilateral severe ICA stenosis. The PWI/DWI mismatch is comparatively larger in the patient with carotid stenosis than in the patient without stenosis, particularly on TTP, rMTT, and rCBF maps. However, the TTP delay is only moderate (<4 seconds) in large parts of the mismatch in the patient with ICA stenosis, indicating that this tissue is probably not at risk of irreversible tissue damage.
recruited into the infarct. The present study adds to previously existing data by showing that a considerably smaller percentage of the total PWI/DWI mismatch is at risk of becoming recruited into the infarct in patients with severe carotid stenosis than in those without carotid stenosis. We suggest that this should be taken into account when PWI and DWI are used to select patients for therapeutic interventions.

In acute stroke patients with extracranial severe carotid artery disease, perfusion abnormalities on PWI may be due to flow obstruction at the level of the stenosis or to emboli lodging in downstream intracranial cerebral vessels. Collateral flow, mainly via the circle of Willis, partially compensates for the local flow obstruction at the site of stenosis, and the overall CBF reduction is often only moderate in patients with severe carotid disease (but without prior stroke). Nevertheless, moderate perfusion abnormalities may affect large regions of the ipsilateral MCA territory in patients with ICA disease and are reliably detected with PWI.15,16,18,19 Our results indicate that in most patients with severe extracranial carotid stenosis, only relatively small subregions of these larger, most likely preexisting perfusion deficits become critically ischemic at the time of stroke onset (with perfusion abnormalities in or below the “penumbral” range) because of acute artery-to-artery thromboembolism or locally impaired hemodynamics.

Currently, it is still a matter of debate which PWI parameters (TTP, rMTT, rCBF, or rCBV) are optimal for differentiating critical from only moderate perfusion abnormalities, which is of particular importance for the evaluation of the mismatch in patients with carotid stenosis. TTP maps are relatively easy to generate, and perfusion deficits are easily visualized on these maps. Quantification of the TTP delay may be used to semiquantitatively assess the severity of the perfusion deficit in the mismatch9 and is clearly superior to purely qualitative forms of evaluation, but TTP is only an indirect measure of tissue perfusion. Absolute quantification of CBF, CBV, and MTT, on the other hand, has been shown to be possible in healthy volunteers when deconvolution methods that require determination of an AIF are used.21–25 However, although absolute flow quantification would be ideal for defining tissue at risk, it is currently unknown how accurate these methods will be when applied to acute stroke patients. As shown in simulations with one of the methods, systematic errors of both CBF and MTT may occur with bolus arrival delays and bolus dispersion due to upstream vascular occlusion,26 which is particularly relevant in patients with severe carotid stenosis (Figure 4). For this reason, with the exception of 1 study reporting results in 2 stroke patients,25 only relative rCBF, rCBV, and rMTT maps have been used in acute stroke studies so far, even when deconvolution methods requiring an AIF were applied (as in our analysis).
Most investigators using rMTT, rCBF, or rCBV maps measured total lesion volumes but did not attempt to quantify the severity of the perfusion deficit. Recently, however, Schlaug et al determined rCBF and rCBV values (relative in comparison with unaffected contralateral tissue) in the ischemic core (DWI lesion) and in an operationally defined “penumbra” (tissue that became recruited into the infarct after the acute study). Among other parameters, rCBF values of 0.12 and 0.37 were reported for the ischemic core and the penumbra, respectively. These values are similar to those found in our study, particularly the rCBF value reported for the penumbra, which corresponds to the subregion of the DWI/PWI mismatch “close to the DWI lesions” in our study (rCBF 0.43; average value obtained for all patients). However, although it may seem relatively easy to use these values to obtain an estimate of the volume of tissue at risk, the application of thresholds (eg, a penumbral threshold of 0.37 to 0.43) to rCBF maps is not straightforward. Normal rCBF is almost 3 times higher in gray than in white matter, indicating that ideally gray and white matter should be evaluated separately. Hence, such approaches should control for gray/white matter differences, which will require the development of appropriate segmentation techniques.

Interestingly, there may also be pathophysiological differences in stroke evolution between patients with and without carotid stenosis, as indicated by the trend toward smaller initial DWI lesions, a smaller increase in lesion size, and significantly smaller final infarcts \( (P<0.05) \) in the stenosis group. Theoretically, a variety of mechanisms could be responsible for this observation, such as a greater capacity for activating collaterals after acute artery-to-artery thromboembolism or increased ischemic tissue tolerance distal to chronic, hemodynamically relevant carotid occlusive disease. In addition, it is conceivable that emboli in carotid stenosis patients are (on average) smaller than in patients with a bilateral severe carotid disease. Note the shorter TTP and MTT as well as the higher rCBF in the posterior circulation compared with the anterior circulation. Hemodynamic impairment is more pronounced in the affected left hemisphere. No lesion is seen on the rCBV image. On DWI (not shown), only a small lesion was detected at a higher slice level.

### Table 3. ROI Analysis of DWI and PWI Parameters in Patients With and Without Severe Carotid Stenosis

<table>
<thead>
<tr>
<th></th>
<th>DWI Lesion Center</th>
<th>Mismatch (Close to DWI Lesion)</th>
<th>Mismatch (Periphery)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stenosis</td>
<td>No Stenosis</td>
<td>Stenosis</td>
</tr>
<tr>
<td>DWI (HR)</td>
<td>1.81±0.4</td>
<td>1.53±0.5</td>
<td>1.12±0.1</td>
</tr>
<tr>
<td>ADC (HR)</td>
<td>0.63±0.2</td>
<td>0.62±0.2</td>
<td>0.97±0.3</td>
</tr>
<tr>
<td>ΔTTP, s</td>
<td>13.9±9.0</td>
<td>14.9±9.3</td>
<td>7.20±5.3</td>
</tr>
<tr>
<td>ΔMTT, s</td>
<td>24.8±9.5</td>
<td>17.1±10.1</td>
<td>14.4±4.2</td>
</tr>
<tr>
<td>rCBF (HR)</td>
<td>0.27±0.1</td>
<td>0.29±0.2</td>
<td>0.47±0.1</td>
</tr>
<tr>
<td>rCBV (HR)</td>
<td>1.38±0.7</td>
<td>0.93±0.5</td>
<td>1.80±0.7</td>
</tr>
</tbody>
</table>

HR indicates hemispheric ratio.

\*\( P<0.05 \), †\( P<0.05 \), ‡\( P<0.01 \).

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**Figure 5.** PWI data from a patient with bilateral severe carotid disease. Note the shorter TTP and MTT as well as the higher rCBF in the posterior circulation compared with the anterior circulation. Hemodynamic impairment is more pronounced in the affected left hemisphere. No lesion is seen on the rCBV image. On DWI (not shown), only a small lesion was detected at a higher slice level.
cardiac embolic source, for example. However, more work is needed to confirm this observation in a larger patient sample and then to clarify the underlying mechanisms.

Finally, there are some limitations to our study. First, the 2 subgroups analyzed in our study (patients with and without carotid stenosis) were not perfectly matched for potentially confounding factors, such as time between symptom onset and acute MRI, treatment, age (see Table 1), and cerebral risk factors. Some of these factors may well have an influence both on the size of the initial PWI/DWI mismatch and on the change in lesion size between the acute and follow-up studies. Second, the sample size of our study was relatively small, particularly for statistical analyses. Third, we did not systematically perform MR angiography in all of our patients. Particularly in patients with extracranial carotid disease, absent MCA flow on MRA is likely to be a valuable marker indicating a relatively high risk for the often large PWI/DWI mismatch, whereas normal or close-to-normal flow probably translates into a relatively low risk.

In conclusion, the PWI/DWI mismatch region is often substantially larger in acute stroke patients with severe ICA stenosis than in patients without carotid disease. However, in these patients, a relatively smaller fraction of the total PWI/DWI mismatch appears to be at risk of irreversible damage than in patients without carotid disease. This should be kept in mind when DWI and PWI are used to select patients for therapeutic interventions. The definition of tissue at risk in these patients may be improved by assessing PWI maps semiquantitatively and probably also by using MRA in combination with DWI/PWI.

References


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Stroke. 2000;31:1311-1317
doi: 10.1161/01.STR.31.6.1311

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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