Do Acute Diffusion- and Perfusion-Weighted MRI Lesions Identify Final Infarct Volume in Ischemic Stroke?

C.S. Rivers, MSc; J.M. Wardlaw, MBChB, MD, FRCR, FRCP, FmedSci; P.A. Armitage, PhD; M.E. Bastin, Dphil; T.K. Carpenter, PhD; V. Cvro, MBChB, MD, MRCP; P.J. Hand, MBChB, MD, MRCP; M.S. Dennis, MD, FRCP

Background and Purpose—An acute mismatch on diffusion-weighted MRI (DWI) and perfusion-weighted MRI (PWI) may represent the “tissue-at-risk.” It is unclear which “semiquantitative” perfusion parameter most closely identifies final infarct volume.

Methods—Acute stroke patients underwent DWI and PWI (dynamic-susceptibility contrast imaging) on admission (baseline), and T₂-weighted imaging (T₂WI) at 1 or 3 months after stroke. “Semiquantitative” mean transit time ($MTT_{sq}$, first moment of concentration/time curve), cerebral blood volume ($CBV_{sq}$, area under concentration/time curve), and cerebral blood flow ($CBF_{sq}$, $CBV_{sq}/MTT_{sq}$) were calculated. DWI and PWI lesions were measured at baseline and final infarct volume on T₂WI acquired ≥1 month after stroke. Baseline DWI, $CBF_{sq}$, and $MTT_{sq}$ lesion volumes were compared with final T₂WI lesion volume.

Results—Among 46 patients, baseline DWI and $CBF_{sq}$ lesions were not significantly different from final T₂WI lesion volume, but baseline $MTT_{sq}$ lesions were significantly larger. The correlation with final T₂WI lesion volume was strongest for DWI (Spearman rank correlation coefficient $\rho=0.68$), intermediate for $CBF_{sq}$ ($\rho=0.55$), and weakest for $MTT_{sq}$ ($\rho=0.49$) baseline lesion volumes. Neither DWI/$CBF_{sq}$ nor DWI/$MTT_{sq}$ mismatch predicted lesion growth; lesion growth was equally common in those with and without mismatch.

Conclusions—Of the 2 PWI parameters, $CBF_{sq}$ lesions most closely identifies, and $MTT_{sq}$ overestimates, final T₂WI lesion volume. “DWI/PWI mismatch” does not identify lesion growth. Patients without “DWI/PWI mismatch” are equally likely to have lesion growth as those with mismatch and should not be excluded from acute stroke treatment. (Stroke. 2006;37:98-104.)

Key Words: cerebrovascular disorders imaging, diffusion-weighted imaging, perfusion-weighted magnetic resonance imaging stroke

In acute ischemic stroke, the mismatch between the acute lesion on magnetic resonance (MR) diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) may represent the “ischemic penumbra” or “tissue-at-risk” of infarction and may provide a target of salvageable tissue within and possibly beyond 6 hours. In several studies, the presence of an acute PWI lesion larger than the DWI lesion at baseline predicted ischemic lesion growth on final (≥1 month) T₂-weighted imaging (T₂WI). One month is the minimum time at which final infarct extent should be assessed as “fogging” may obscure, and edema may exaggerate T₂WI infarct extent measured at <1 month.

Perfusion information from MR dynamic-susceptibility contrast (DSC) PWI uses “semiquantitative” or “quantitative” analyses. We used “semiquantitative” (also called “summary” or “relative”) to describe analyses of the concentration/time curve without correction with the arterial input function (AIF). In general, “semiquantitative” measures require less intensive processing than “quantitative” measures and include parameters such as: time-to-peak (TTP), which may be a good estimate of mean transit time (MTT) but is difficult to compare between imaging episodes; cerebral blood volume ($CBV_{sq}$), calculated from the area under, and MTT, calculated from the first moment of, the concentration/time curve; and cerebral blood flow ($CBF_{sq}$), calculated as $CBV_{sq}/MTT_{sq}$. “Quantitative” perfusion (also called “absolute”) requires deconvolution of the concentration/time curve with an AIF: $CBF_{q}$ may be calculated as the height, and $CBV_{q}$ as the area under, the deconvolved concentration/time curve; and $MTT_{q}$ as $CBV_{q}/CBF_{q}$. $CBV$ is not considered a good predictor of “tissue-at-risk” because of its bimodal nature and insensitivity to bolus delay and dispersion. Thus, parameters reflecting CBF and MTT have emerged as the most likely candidates for predicting final infarct extent.
Previous studies of PWI data (>24 hours after stroke) and final lesion extent (Table 1) have produced rather confusing results. Of the 13 studies, only 3 compared CBF and MTT (total n = 53):11–13 2 found that CBF related best to final infarct volume. None directly compared CBFq and MTTq. In the remainder, TTP correlated strongly with final infarct volume14–20 and MTTq and MTT underesti-

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<td>Manual outline</td>
<td></td>
<td>MTT overestimates final infarct volume by 282%</td>
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</table>

*Likely includes the 18 patients reported previously in Barber et al; $may include 18 of 38 patients reported previously in Parsons et al.18
tients who did not receive thrombolyis or experimental agents. The small sample sizes (Table 1), different PWI lesion measurement methods, inappropriate statistical tests (parametric versus nonparametric), and total absence of data comparing CBF$_{sq}$ and MTT$_{sq}$ (more rapidly calculated than “quantitative” data acutely) indicate that more data are required to determine which PWI parameter identifies “tissue-at-risk.”

We aimed to determine whether CBF$_{sq}$ or MTT$_{sq}$ on acute DSC PWI most closely identified the final T$_2$WI infarct extent, the relationship of “DWI/PWI mismatch” and lesion growth, and to identify possible reasons for discrepancies in previous studies.

**Methods**

**Patients**

We prospectively recruited patients from the hospital stroke service with moderate to severe ischemic stroke. We performed imaging urgently (maximum for the study of 24 hours after stroke onset [baseline]). Baseline clinical assessment including the National Institutes of Health Stroke Scale (NIHSS) score and Oxfordshire Community Stroke Project (OCSP) classification were performed by a trained stroke physician. Functional outcome (modified Rankin scale [mRS]) was measured at 3 months. A panel of experts determined a final diagnosis of stroke. We recorded any acute treatments that the patients received (treatment did not affect inclusion in study).

**Imaging**

We used a GE Sigma LX 1.5T (General Electric) MR scanner with a self-shielding gradient set (22 mT/m maximum) and “birdcage” quadrature head coil. We included fast spin-echo T$_1$WI and gradient-echo T$_2$WI, diffusion-weighted (DWI), and CBF$_{sq}$, and MTT$_{sq}$ maps, using a Sun Ultra Sparc Station 10 (Sun Microsystems) in Analyze (Mayo Foundation) format and presented as a color-coded map. Voxels in which the data did not reach a maximum value of $>2$ times the maximum precontrast baseline signal SD and then begin to fall before the end of the imaging time (85 seconds) were assigned as error voxels, visible as white areas on the perfusion color maps (there were extremely few patients with any of these), indicating very low flow areas in the infarct.

**Baseline PWI and DWI and Final T$_2$WI Lesion Volume Analysis**

A neuroradiologist, blind to all clinical and other imaging data, guided the tracing by an experienced neuroscientist of the visible lesions on DWI, CBF$_{sq}$, and MTT$_{sq}$ maps, using a Sun Ultra Sparc Station 10 (Sun Microsystems) in Analyze (ie, a consensus of 2 experienced observers). Image brightness and contrast were optimized between areas of abnormal diffusion or perfusion and normal-appearing brain. The neuroradiologist also traced the final infarct on the latter of the 1- or 3-month T$_2$WI as described above. Lesion volumes were obtained by summing the number of outlined voxels and multiplying by the slice thickness on each slice on which the lesion was visible.

**Statistical Analyses**

Baseline DWI, CBF$_{sq}$, MTT$_{sq}$, and final T$_2$WI lesion volumes were not normally distributed (Kolmogorov–Smirnov test; $P<0.01$), so median lesion volumes were compared using Wilcoxon signed rank sum tests, and correlations between baseline DWI, CBF$_{sq}$, and MTT$_{sq}$ volumes with final T$_2$WI lesion volume were assessed with Spearman rank correlation coefficients ($r$). We also calculated mean volumes ($\pm$SD) for comparison with previous studies (Table 1), and Pearson product moment correlation coefficients ($r$, appropriate for parametric data) to compare baseline DWI, CBF$_{sq}$, and MTT$_{sq}$ volumes with final T$_2$WI lesion volume, to determine whether use of Pearson $r$ might explain differences between previous studies. “Lesion growth” was defined as final T$_2$WI lesion volume–baseline DWI lesion volume, and “DWI/PWI mismatch” as PWI lesion volumes–DWI lesion volumes. $\chi^2$ tests were used to test whether DWI/CBF$_{sq}$ or DWI/MMT$_{sq}$ “mismatch” predicted “lesion growth.”

**Results**

**Patients**

A total of 46 patients were included; 28 of 46 (61%) were male, with an average age of 72 years (range 37 to 94 years). Mean NIHSS score on admission was 8 (median 8; range 0 to 25). For OCSP classifications, 12 of 46 (26%) patients had a total anterior circulation infarct, 29 of 46 (63%) had a partial anterior circulation infarct, 4 of 46 (9%) had a lacunar infarct, 1 of 46 (2%) had a posterior circulation infarct. All patients were treated with aspirin, but none received thrombolyis or investigational drugs. Mean mRS score at 3 months was 2 (median 2); 2 of 46 (4%) patients were dead (mRS=6), and 13 of 46 (28%) patients were dependent (mRS=3 to 5). Mean ($\pm$SD) time from stroke onset to baseline MTT$_{sq}$ was defined as final T$_2$WI lesion volume–baseline DWI lesion volume, and “DWI/PWI mismatch” as PWI lesion volumes–DWI lesion volumes. $\chi^2$ tests were used to test whether DWI/CBF$_{sq}$ or DWI/MMT$_{sq}$ “mismatch” predicted “lesion growth.”

**Results**

**Table 2. Baseline DWI, CBF$_{sq}$, and MTT$_{sq}$ Lesion Volumes Compared With Final T$_2$WI Volume**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Median Volume (cm$^3$)</th>
<th>% of Median Volume</th>
<th>Minimum Volume (cm$^3$)</th>
<th>Maximum Volume (cm$^3$)</th>
<th>Mean Volume (cm$^3$)</th>
<th>SD (cm$^3$)</th>
<th>% of Mean Volume</th>
<th>Median Final T$_2$WI Volume</th>
<th>n$\geq$T$_2$WI Volume</th>
<th>%$\geq$T$_2$WI Volume</th>
<th>P</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Final T$_2$WI</td>
<td>18.47</td>
<td>...</td>
<td>0.24</td>
<td>304.80</td>
<td>39.61</td>
<td>57.84</td>
<td>...</td>
<td>304.80</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>(1 or 3 months)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline DWI</td>
<td>13.28</td>
<td>72%</td>
<td>0.33</td>
<td>154.49</td>
<td>25.61</td>
<td>32.78</td>
<td>65%</td>
<td>25.61</td>
<td>0.14</td>
<td>20/46</td>
<td>44%</td>
<td>0.68</td>
</tr>
<tr>
<td>Baseline CBF$_{sq}$</td>
<td>23.74</td>
<td>129%</td>
<td>0</td>
<td>287.60</td>
<td>44.14</td>
<td>58.62</td>
<td>111%</td>
<td>44.14</td>
<td>0.82</td>
<td>18/46</td>
<td>39%</td>
<td>0.55</td>
</tr>
<tr>
<td>Baseline MTT$_{sq}$</td>
<td>50.60</td>
<td>274%</td>
<td>0</td>
<td>582.02</td>
<td>96.41</td>
<td>118.16</td>
<td>243%</td>
<td>96.41</td>
<td>0.00</td>
<td>30/46</td>
<td>65%</td>
<td>0.49</td>
</tr>
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</table>

$P$=exact significance result of Wilcoxon signed rank sum tests; $\rho$=Spearman rank, $r$=Pearson product moment, correlation coefficients between mean baseline DWI, CBF$_{sq}$, and MTT$_{sq}$ and final T$_2$WI lesion volumes; n$\geq$T$_2$WI volume=No. of DWI/CBF$_{sq}$/MTT$_{sq}$ lesions $\geq$final T$_2$WI volume.
baseline MRI was 10±7 hours; 18 of 46 (39%) patients were imaged within 6 hours, 11 of 46 (24%) at 6 to 12 hours, and 17 of 46 (37%) at 12 to 24 hours. Final T2WI was performed at 1 month (mean 32±4 days) in 6 of 46 (13%) patients and at 3 months (mean 96±7 days) in 40 of 46 (87%) patients.

Baseline PWI Lesion Volumes (Table 2)
Baseline CBFsi lesion volumes (median 23.74 cm³) were significantly smaller than baseline MTTsi lesion volumes (median 50.60 cm³; \( P<0.01 \)). Eleven patients (24%) had no visible lesion on baseline CBFsi, and 10 patients (22%) had no visible lesion on baseline MTTsi; 6 (13%) had no visible lesion on either baseline CBFsi or MTTsi.

Baseline DWI Lesion Volumes (Table 2 and Figure 1)
All patients had a visible baseline DWI lesion. Baseline DWI lesion volumes (median 13.28 cm³) were significantly smaller than baseline CBFsi (\( P=0.01 \)) and MTTsi volumes (\( P<0.01 \)) but were not significantly different from final T2WI lesion volume (\( P=0.14 \)).
Baseline PWI Versus Final T2WI Lesion Volumes (Table 2 and Figure 1)

All patients had a visible lesion on final T2WI (median 18.47 cm³) corresponding with the lesion indicated on baseline DWI. Overall, baseline CBFₚq lesion volume was not significantly different from final T2WI lesion volume ($P=0.82$). Baseline MTTₚq volume was significantly larger ($P=0.01$) than final T2WI lesion volume.

Correlation Between Baseline DWI, PWI, and Final T2WI (Table 2 and Figure 1)

Final T2WI lesion volume most strongly correlated with baseline DWI ($\rho=0.68$), followed by baseline CBFₚq ($\rho=0.55$) and baseline MTTₚq ($\rho=0.49$) lesion volumes (all $P<0.01$). Using Pearson’s $r$, final T2WI lesion volume was most strongly correlated with baseline MTTₚq ($r=0.66$), followed by baseline DWI ($r=0.53$) and baseline CBFₚq ($r=0.41$) lesion volumes (all $P<0.01$).

“DWI/PWI Mismatch” and Final T2WI Lesion Volume

There was no significant association between “lesion growth” (final T2WI lesion > baseline DWI lesion) and the presence or absence of “DWI/PWI mismatch” (Table 3). On CBFₚq, 15 of 25 patients with “mismatch” had “lesion growth,” and 10 of 25 patients did not; 11 of 21 without “mismatch” had “lesion growth,” and 10 of 21 did not ($\chi^2=0.27; P=0.60$; Table 3). On MTTₚq, 21 of 33 with “mismatch” had “lesion growth,” and 12 of 33 did not; 5 of 13 without “mismatch” had “lesion growth,” and 8 of 13 did not ($\chi^2=2.40; P=0.12$; Table 3). Thus, about half of patients without “mismatch” had lesions that grew, and “mismatch” was as common among patients with lesions that grew as among those with lesions that did not grow (Figure 2).

Discussion

Because there were no previous studies testing the relationship between acute CBFₚq and MTTₚq and final infarct volume, we asked: which “semiquantitative” PWI parameter, if any, best predicts final infarct extent? In this large systematic study, we found that acute CBFₚq lesions more closely identified final T2WI lesion extent than acute MTTₚq lesions, which clearly overestimate final lesion extent. We also demonstrate that “lesion growth” was equally common among patients with lesions that grew as among those with lesions that did not grow (Figure 2).
the patients without a “mismatch” had “lesion growth”; half of those with “mismatch” did not. Although “DWI/PWI mismatch” is being used to identify patients for inclusion in trials of stroke treatments, our results imply that patients without “mismatch” may also benefit and should not be denied that possibility by being excluded.

Our findings concerning “semiquantitative” PWI parameters agree overall with the few previous “quantitative” studies directly comparing CBF\(_q\) and MTT\(_q\).\(^{11,13,16,19,20}\) Some disparities among previous studies may result from different statistical analyses; use of Pearson \(r\) instead of Spearman \(\rho\) completely changed the strength and order of correlation between baseline CBF\(_q\) and MTT\(_q\) lesions and final T\(_2\)WI lesion volume. Previous studies also support the conclusion that acute MTT (MTT\(_q\) or MTT\(_sq\)) lesions overestimate final lesion extent,\(^{12–21}\) although some studies found stronger correlations between MTT and final infarct volumes than we did (eg, \(r=0.77\) to 0.90\(^{12,13,19,20}\); these studies used MTT\(_q\) were small (mean \(n=21\); Table 1), and tested some very restricted perfusion thresholds (eg, highest 70% of MTT signal intensities\(^{12}\)). Furthermore, it is important to distinguish between “correlation” and “size equivalence”; a good correlation between baseline MTT volume and final T\(_2\)WI lesion volume is not incompatible with MTT significantly overestimating final infarct volume.

In contrast to some previous studies,\(^{4,11–13,16,19,20}\) we found no significant association between DWI/CFB\(_q\) or DWI/MTT\(_q\) “mismatch” and “lesion growth” (Table 3). However, some of these studies only included patients with “mismatch.”\(^{11,12}\) A recent study using “DWI/PWI mismatch” as an inclusion criteria in a thrombolyis trial\(^{22}\) failed to show an association between “DWI/PWI mismatch” and lesion expansion but did show that reperfusion was inversely associated with “lesion growth.”

The superiority of “quantitative” over “semiquantitative” PWI measures is not yet proven. “Quantitative” measures are still only estimates\(^7\) and require considerably more complex image processing than “semiquantitative” measures so are still only estimates\(^7\) and require considerably more complex image processing than “semiquantitative” measures so are less appropriate to the acute setting. Newer perfusion measures including flow heterogeneity\(^{21}\) and bolus-delay corrected maps\(^{13}\) appear promising, but further validation is required to determine their overall usefulness. There is a further question concerning the use of the term “perfusion-weighted imaging” itself, which is somewhat misleading because MR perfusion maps are actually calculated parameter maps.

The strengths of the current study include the large sample, inclusion of patients with and without “DWI/PWI mismatch,” careful clinical characterization, blinded image analysis and outcome assessment, and careful use of statistics. We included patients up to 24 hours, and interestingly most (75% to 80%) had a perfusion lesion on CBF, MTT, or both, with only just >10% not having a perfusion lesion. We used manual rather than “threshold” measurement methods because the former is currently the most rapid method to apply in the clinical setting. Thresholds might reduce observer variation, but the hemodynamics underlying ischemic stroke are complex and variable. Baseline perfusion values greatly overlap between tissue that does and does not infarct,\(^{12,17}\) and PWI parameter thresholds are time dependent; a threshold for ischemic but viable brain at 3 hours may be very different from that at 6 hours. None of our patients received thrombolysis or any investigational drugs, therefore this series represents a baseline measure of the presence or absence of spontaneous lesion expansion and could be used to estimate sample sizes for future acute stroke treatment trials using imaging markers of outcome. However, our data emphasize the need for further study to identify which, if any, alternative imaging markers might identify patients in whom treatments such as thrombolysis might prevent infarct growth. For example, it is unclear precisely how large artery patency relates to the PWI lesion. Are middle cerebral artery or internal carotid artery occlusions always accompanied by PWI lesions, and do these lesions resolve completely and rapidly as the artery reperatures, or is there a time lag between arterial reperfusion and PWI lesion disappearance?

In conclusion, acute DWI, CBF\(_q\), and MTT\(_q\) lesion volumes all correlated well with final infarct volume, but acute MTT\(_q\) lesion volume significantly overestimated final infarct volume. Of the 2 “semiquantitative” PWI measures, the acute CBF\(_q\) lesion most closely identifies the final T\(_2\)WI lesion. There was no clear association between the presence of a “DWI/PWI mismatch” and lesion expansion, questioning the value of the “DWI/PWI mismatch” as a clinical marker of “tissue-at-risk.” About half of patients without “DWI/PWI mismatch” have “lesion growth” so they may benefit from acute stroke treatments and should probably not be excluded from clinical trials.

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