Diffusion- and Perfusion-Weighted Magnetic Resonance Imaging of the Brain Before and After Coronary Artery Bypass Grafting Surgery

Lucas Restrepo, MD; Robert J. Wityk, MD; Maura A. Grega, RN; Lou Borowicz, Jr, MS; Peter B. Barker, DPhil; Michael A. Jacobs, PhD; Norman J. Beauchamp, MD, MHS; Argye E. Hillis, MD, MS; Guy M. McKhann, MD

Background and Purpose—Coronary artery bypass grafting (CABG) is a frequently performed surgical procedure that can be associated with neurological complications. Some studies have demonstrated that new focal brain lesions, detected by MRI, can develop after CABG. Furthermore, it has been suggested that the presence of such new lesions is associated with a decline in neurocognitive test scores. Advanced MRI techniques, including diffusion- (DWI) and perfusion-weighted imaging (PWI), offer important diagnostic advantages over conventional imaging in the assessment of patients undergoing CABG. We sought to determine whether focal PWI and DWI abnormalities could occur after CABG, particularly in patients without any measurable neurological deterioration.

Methods—Thirteen patients prospectively underwent MRI with DWI and PWI before and after CABG. A battery of neurocognitive tests was administered before and after surgery. Demographic, clinical, and radiographic characteristics of the patients were collected and compared.

Results—Four patients developed new DWI defects after CABG. The lesions were small, rounded, and multiple (3 of 4 patients). One of these patients was diagnosed with stroke on clinical grounds. The patients with new lesions had a larger neurocognitive decline than their counterparts with stable MRI. Other clinical characteristics of patients with new DWI lesions, including stroke risk factors, were similar to those of patients without MRI changes. No focal perfusion abnormalities were observed on preoperative or postoperative scans.

Conclusions—Postoperative DWI abnormalities can occur after CABG, even in patients without overt neurological defects. The PWI scans remained unchanged. Larger prospective studies are required to determine whether the new lesions are clearly associated with neurocognitive decline or with specific perioperative stroke risk factors. (Stroke. 2002;33:2909-2915.)

Key Words: bypass surgery • magnetic resonance imaging, diffusion-weighted • magnetic resonance imaging, perfusion-weighted

Coronary artery bypass grafting (CABG) is a frequently performed surgical procedure, with more than half a million operations performed every year in the United States.1 Although CABG provides definite clinical advantages in patients with coronary artery disease, the surgery may be associated with neurological complications. The incidence of such complications is 0.4% to 5.7% for stroke, 10% to 28% for delirium, and 33% to 83% for persistent cognitive dysfunction and behavioral change.1 The mortality rate of patients with such complications is considerably higher than for the remainder of patients.2 Furthermore, the length of stay in the intensive care unit and hospital ward is significantly greater for patients with neurological complications, resulting in a 2-fold increase in the total cost of care.1,2 Several risk factors for postoperative stroke have been postulated, including hypertension (HTN), diabetes mellitus (DM), carotid stenosis, previous stroke, and age ≥65 years.3 However, current predictive models of postoperative stroke may not necessarily identify all patients who eventually experience other neurological complications such as delirium.1,3

The application of the MRI techniques of diffusion- (DWI) and perfusion-weighted imaging (PWI) to the study of the neurological complications of CABG is of particular interest because cerebral ischemia is thought to be a central pathophysiological factor underlying postoperative delirium and more persistent neurocognitive changes.1 Cerebral ischemia during CABG can be induced by hypoperfusion,
intraoperative aortoarterial embolization, coexistent cerebral vessel atherosclerosis, or a combination of such factors.

DWI is more sensitive than CT and conventional MRI sequences for the detection of early signs of ischemia, disclosing areas of brain infarction within a few hours after symptom onset in ≈95% of acute stroke patients.4–9 PWI, on the other hand, uses a bolus of gadolinium to generate changes in signal intensity throughout the brain in a time-dependent fashion, paralleling cerebral blood flow.6 PWI, however, does not provide an absolute estimate of cerebral blood flow but rather uncovers regions of relative focal cerebral hypoperfusion compared with equivalent contralateral areas.6 Nevertheless, the localization of perfusion defects thus detected correlates well with focal neurological dysfunction, which in some cases may be reversible.10,11 Some patients may exhibit a perfusion-diffusion “mismatch,” which suggests the presence of salvageable brain tissue.6 Therefore, the information provided by the combination of DWI and PWI has relevant therapeutic implications.12

Diffusion abnormalities can be demonstrated in almost all patients with postoperative stroke and even in some patients with postoperative delirium.13 Using conventional MRI sequences, some investigators have observed new focal brain abnormalities developing after CABG in patients without apparent neurological impairment.14,15 However, the pathophysiological basis of such changes and their clinical significance remain speculative. It is unknown at present whether focal DWI or PWI abnormalities can also occur in patients without postoperative neurological symptoms or in those patients who develop postoperative neurocognitive decline. The purpose of this study was to determine whether brain diffusion and perfusion abnormalities can occur after CABG and, if so, their clinical relevance.

Materials and Methods

Patients

This study was reviewed and approved by the Institutional Review Board of the Johns Hopkins Hospital. Twenty-three patients undergoing elective CABG from January 2000 to March 2001 were prospectively asked to participate in our study. The study included any patient undergoing elective CABG with no contraindications for MRI (including allergy to gadolinium-DTPA, presence of pacemaker, aneurysm clips, and/or a history of shrapnel injury); no concomitant cardiovascular surgical procedures were contemplated, including carotid endarterectomy and valve replacement; and the patients had to be able to provide written consent. Informed consent was obtained from all subjects before the first MRI. Our protocol consisted of obtaining the brain MRIs within 8 days before and after the surgery.

Twenty patients underwent MRI before CABG; 13 completed both the preoperative and postoperative scans. Two patients could not complete the preoperative MRI because of scheduling problems, and 1 patient could not fit within the scanner. Seven patients did not complete the postoperative MRI: 1 patient did not have surgery, 4 patients refused the follow-up scan, and 2 patients were missed because of MRI scheduling problems.

Neurocognitive Testing

A battery of standardized neurocognitive tests was administered by 1 of 3 investigators (M.A.G., L.M.B., A.E.H.) within 1 week before and after CABG. The trail making test B was used to assess psychomotor speed. Language function was measured through the oral and written naming test and oral reading tests.16,17 Visuospatial functions were evaluated with the line cancellation and Bells tests.16 All neuropsychological test scores were converted into z scores, as follows: z score equals individual test score minus mean baseline test score divided by the SD of the baseline test score (individual test score minus mean baseline test score divided by baseline test score).

The postoperative z scores were subtracted from the preoperative z scores to calculate the absolute change in the neurocognitive test scores. In addition to calculating z scores for each individual patient, we also summed up and averaged all z scores of different tests in every particular patient to determine the overall cognitive performance of each patient (“composite” cognitive performance score). We defined short-term neurocognitive decline as a drop of >0.5 SD of the baseline mean z score of each individual test.

Neuroimaging Studies

All except 2 patients underwent preoperative carotid Duplex ultrasound examination of the extracranial carotid arteries. Brain MRI examination consisted of conventional T1 (time to repeat [TR], 800 ms; echo time [TE], minimum; field of view [FOV], 24 cm; number of excitations [NEX], 1; matrix, 256×192; slice thickness, 5 mm), axial fast-spin echo T2-weighted (TR, 4000 ms; effective TE, 85 ms; FOV, 24 cm; NEX, 2; matrix, 256×192; slice thickness, 5 mm), and axial fluid-attenuated inversion-recovery (TR, 8800 ms; TE, 133 ms; inversion time, 2200 ms; FOV, 24 cm; matrix, 256×192; slice thickness, 5 mm) sequences. Three-dimensional time of flight MR angiography of the circle of Willis was performed during the preoperative MRI scan with the following parameters: flip angle, 20°; TR, 43 ms; TE, 6 ms; slice thickness, 1 mm; FOV, 220 cm (3 of 4); matrix size, 512×192; 2 slabs of 54 slices each; scan time, 7.5 minutes.

DWI was performed by use of single-shot, multislice, spin-echo, diffusion-weighted echo planar imaging of the whole brain (TR, 10 000 ms; FOV, 24 cm; NEX, 1; matrix, 128×128; slice thickness, 5 mm). Apparent diffusion coefficient maps showing restricted diffusion were used to confirm that regions of DWI hyperintensity were due to decreased diffusion instead of T2 “shine through.” Whole-brain DWI was performed with a multislice gradient-echo scan with the sequence repeated every 2 seconds for 60 seconds during an intravenous bolus injection of 20 mL gadolinium-DTPA at 5 cm3/s (Magnevist, Schering-Plough AG) injected at a rate of 5 mL/s (TR, 2000 ms; TE, 60 ms; flip angle, 90°; FOV, 24 cm; NEX, 1; matrix, 128×64; slice thickness, 5 mm; axial slices, 17). Injection of gadolinium was followed by 20 cm3 of normal saline at 5 cm3/s with an MRI-compatible power injector (Spectris, Medrad Inc.). All volumes were measured by a trained technician under the supervision of a neuroradiologist (N.J.B.). Both were blinded to the outcome and the identity of all patients. Volumes of any DWI and PWI abnormalities were manually delineated by region of interest with the Scion Imaging program (Scion Corp, 1998). A perfusion abnormality was defined as a region with >2.5-second delay compared with the contralateral side on the time-to-peak (TTP) map. We chose this threshold on the basis of our previously published data showing that detailed language and neurocognitive test scores strongly correlate with the PWI volume of regions exhibiting a TTP delay of 2.5 seconds.11–13 Nevertheless, we also calculated regional cerebral blood volume (rCBV), regional cerebral blood flow (rCBF), and mean transit time (MTT) for all available PWI scans (11 patients) to compare them qualitatively with the TTP map. The PWI scans of the 2 remaining patients were not available to generate rCBV, rCBF, and MTT because of archival problems and therefore had only TTP sequences. MTT images were generated from the following equation16,21:

\[ MTT = \frac{1}{\text{flow}} + \frac{v}{\text{CBF}} \]
Fisher’s neurologist from our institution through consultation. We used 2 investigators (M.A.G., L.M.B.), who evaluated all patients after possible postoperative neurological complication was monitored by medium (2% to 5%), or high (>5%). The development of any medium (2% to 5%), or high (>5%). The development of any neurological abnormalities, including delirium. This patient with postoperative stroke had multiple stroke risk factors and had subclinical (old) infarcts on the preoperative MRI. All patients with postoperative DWI abnormalities had a history of HTN and postoperative atrial fibrillation. However, this was not statistically different from the rest of the patients (Table 2). Other variables, including age, risk factors for preoperative stroke, and preoperative risk classification, did not differ between the patients with new DWI lesions and those without changes (Table 2).

Most patients (10 of 13) experienced a decline in the score of at least 1 neurocognitive test. Usually only 1 test caused a drop in the postoperative scores (7 of 13 patients had postoperative decline in 1 test, 2 patients had a decline of >1 test, and 1 patient had a decline in 5 different tests). When the $z$ scores of each individual patient were averaged across tests, it was noted that 3 of 13 patients had a decline in their composite $z$ score calculation. Two of these patients had new DWI lesions. Patients with new focal diffusion lesions had a larger postoperative $z$ score change than their counterparts without new DWI abnormalities (mean difference, 0.31 versus −0.44; $P = 0.04$; 95% confidence interval, 0.017 to 0.6). The number of multiple new DWI lesions was associated with a larger decline in composite $z$ score (Pearson’s $\chi^2$, 7.993; $P = 0.046$). Furthermore, the number of new postoperative DWI lesions was significantly associated with a decline in $>1$ neurocognitive test (Pearson’s $\chi^2$, 26.894; $P = 0.008$; Pearson’s $r$, 0.467; $P = 0.107$). The most affected tests were the oral naming (Pearson’s $\chi^2$, 7.993; $P = 0.005$), written naming (Pearson’s $\chi^2$, 3.704; $P = 0.054$), and line cancellation (Pearson’s $\chi^2$, 5.318; $P = 0.021$; Pearson’s $r$, 0.64; $P = 0.019$). Nevertheless, 1 patient with new focal postoperative DWI changes (patient 9) had no discernible decline in any of the neurocognitive tests.

Discussion

This study demonstrates that focal brain diffusion abnormalities can occur after CABG, even in patients without clinically identified stroke or delirium. Nevertheless, the development of focal postoperative DWI abnormalities was often associated with objective evidence of neurocognitive decline and/or a definite stroke syndrome in our cohort. Another finding of particular relevance was the absence of perfusion defects in our patients. A caveat is that PWI estimates relative brain perfusion compared with homolo-
gous regions of the contralateral hemisphere rather than providing a direct measurement of cerebral blood flow. Homogeneous bilateral perfusion changes are conceivable after CABG; for instance, a global augmentation of (ie, resulting from anemia) or reduction in (ie, secondary to heart failure) cerebral perfusion could occur after heart surgery. Therefore, relative perfusion maps using rCBV, rCBF, MTT, and TTP will not reveal abnormalities in the absence of perfusion asymmetry, spuriously resulting in a normal scan. Quantitative measures of cerebral blood perfusion with techniques such as PET or dynamic arterial spin-labeling MRI would be of interest but were not performed in the present cohort. Nevertheless, we have previously described regional hypoperfusion on TTP in other patients with clinical stroke after CABG.

In addition, hypoperfused brain regions may resolve over time as sufficient blood flow is reestablished. Because we obtained our follow-up scans an average of 4 ± 1.6 days after CABG, it is conceivable that some of the observed DWI abnormalities were associated at some point with rCBF changes, which resolved by the time of the follow-up scan. On the other hand, the intrinsic technical constraints of PWI mapping may not permit the appropriate spatial resolution of hypoperfused brain regions caused by diminutive embolic debris, although color contrast manipulation can be used to improve the conspicuity of small perfusion defects. Another issue is the diagnostic accuracy of the PWI techniques used in the present study. Nevertheless, a recent study suggests that a combination of PWI parameters such as relative contrast bolus peak height and TTP are excellent

TABLE 1. Clinical and Radiologic Characteristics of the Patients

<table>
<thead>
<tr>
<th>Pt/Sex/Age</th>
<th>Risk Factors</th>
<th>Preop Radiologic Findings</th>
<th>Postop Stroke</th>
<th>New DWI Lesion</th>
<th>Composite Cognitive Score Decline</th>
<th>No. of Lesions</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/52</td>
<td>None</td>
<td>Normal</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>2/F/64</td>
<td>HTN, DM, postop Afib</td>
<td>Had small R corona radiata diffusion abnormality on preop DWI; normal MRA</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>2</td>
<td>R occipital lobe (watershed)</td>
</tr>
<tr>
<td>3/M/51</td>
<td>Past stroke, unilateral carotid stenosis, tobacco</td>
<td>MRI: mild PVWMD; MRA: mild R ICA stenosis; carotid duplex: L carotid stenosis, 40%–60%</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>4/M/52</td>
<td>None</td>
<td>Normal MRI; MRA not performed</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>5/F/59</td>
<td>HTN, DM, bilateral carotid stenosis, postop Afib</td>
<td>MRI: L frontal lobe wedge-shaped infarct; normal MRA; carotid duplex: bilateral carotid stenosis, 40%–60% (L), 61%–80% (R)</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>6/M/67</td>
<td>HTN, bilateral carotid stenosis, carotid bruit, postop Afib</td>
<td>MRI: mild PVWMD; normal MRA; carotid duplex: carotid stenosis, 40%–60% bilaterally</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>7/F/72</td>
<td>Past TIA, unilateral carotid stenosis, HTN, PVD, postop Afib</td>
<td>R anterior corona radiata lacune; moderate PVWMD</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>8/M/69</td>
<td>HTN, DM, unilateral carotid stenosis, tobacco</td>
<td>MRI: moderate PVWMD; normal MRA</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>9/M/65</td>
<td>HTN, postop Afib</td>
<td>MRI: moderate PVWMD; normal MRA</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>2</td>
<td>R occipital lobe and L thalamus</td>
</tr>
<tr>
<td>10/M/74</td>
<td>HTN, DM, bilateral carotid stenosis, carotid bruit, tobacco, postop Afib</td>
<td>MRI: 2 lacunes, mild PVWMD; MRA: L cavernous ICA stenosis (90%); carotid duplex: carotid stenosis, 40%–60% bilaterally</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>1</td>
<td>R corona radiata</td>
</tr>
<tr>
<td>11/M/70</td>
<td>HTN, tobacco</td>
<td>MRI: moderate PVWMD; normal MRA</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>12/M/81</td>
<td>HTN, DM, chronic Afib, PVD, tobacco</td>
<td>MRI: moderate PVWMD; normal MRA</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>13/M/72</td>
<td>HTN, tobacco, postop Afib</td>
<td>MRI: moderate PVWMD; normal MRA</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>4</td>
<td>R cerebellum, R hippocampus (PCA), L postlimb internal capsule, L MCA watershed</td>
</tr>
</tbody>
</table>

Preop indicates preoperative; postop, postoperative; DWI, diffusion-weighted imaging; HTN, chronic hypertension; DM, diabetes mellitus; Afib, atrial fibrillation; PVWMD, periventricular white matter disease; TIA, transient ischemic attack; PVD, peripheral vascular disease; ICA, internal carotid artery; PCA, posterior cerebral artery; MCA, middle cerebral artery.
predictors of infarct growth in acute stroke cases diffusion-perfusion mismatch. TTP has been shown to highly correlate with MTT, whereas rCBV and rCBF are needed to generate the MTT maps.

We hypothesize that the observed DWI abnormalities correspond to small areas of brain ischemia caused by emboli arising from the heart or aorta during surgical intervention. We base this opinion on the radiographic appearance of the lesions, particularly because of their multiplicity (3 of 4 patients had multiple areas of restricted diffusion). In addition, most patients did not have significant intracranial or extracranial arterial stenosis ipsilaterally that could serve as additional embolic source.

The possible embolic nature of the observed MRI lesions is suggested by prior pathological and neuroimaging studies. Pathological brain examination of patients who died after cardiac surgery has revealed the presence of embolic material diffusely lodged in the cerebral microcirculation. However, such material appears to be made up mainly of microscopic lipid fragments. Therefore, the terms “microemboli” and “macroemboli” have been proposed to differentiate the smaller embolic debris from larger particles, which have the potential to produce focal neurological and/or radiological abnormalities. In addition, studies using intraoperative transcranial Doppler monitoring have demonstrated frequent microembolic signals detected at different stages during cardiac surgery. Although high counts of microembolic signals by transcranial Doppler correlate with postoperative neurological complications (including stroke), most patients with microembolic signals do not develop neurological deficits after surgery.

Several studies have employed perioperative brain MRI to evaluate the neurological complications of CABG, some of which report asymptomatic focal brain lesions developing after surgery. The acuity and radiographic appearance of the new lesions suggest that such abnormalities constituted small infarcts, although the finding of discrete areas of increased signal on T2-weighted images is nonspecific. Some investigations report that the new postoperative MRI lesions were associated with a neurocognitive decline, although this claim was not confirmed by a subsequent study. Nevertheless, other investigations, including one using DWI, did not find focal brain postoperative changes.

Most investigations using MRI, including our own, have intrinsic methodological limitations, particularly small sample size (the largest study includes 38 patients). Only 2 previous studies have examined control subjects who did not undergo CABG. Other limitations of previous reports include the use of low-field-strength scanners (0.15

| TABLE 2. Differences Between Patients With New Postoperative DWI Abnormalities on Brain MRI and Their Counterparts Without New Lesions |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Patients With New DWI Lesions (n=4) | Patients With Normal DWI (n=9) | P Value |
| HTN | 4 | 6 | 0.497 |
| DM | 2 | 3 | 1.00 |
| Carotid bruit | 1 | 1 | 1.00 |
| Carotid stenosis >60%$^*$ | 0 | 1 | 1.00 |
| Age >65 years | 1 | 3 | 1.00 |
| History of past stroke | 0 | 1 | 1.00 |
| Postop Afib | 4 | 3 | 0.07 |
| Preop stroke risk assessment† | | | |
| Low risk | 0 | 3 | 0.497 |
| Medium risk | 1 | 1 | 1.00 |
| High risk | 3 | 5 | 1.00 |
| Old infarcts on MRT‡ | 2 | 2 | 0.53 |

HTN indicates chronic hypertension; DM, diabetes mellitus; Afib, atrial fibrillation; postop, postoperative; preop, preoperative.

$^*$As measured by preoperative carotid Duplex ultrasound.

†Probability of postoperative stroke according to McKhann et al$^3$ low (<0.02), medium (0.02–<0.05), high (>0.05).

‡Radiographic presence of infarcts. Can be asymptomatic and does not include periventricular white matter disease.

DWI of a 64-year-old woman (patient 2, Table 1) without any demonstrable neurological deficits after CABG. Preoperative scan, performed 2 days after a diagnostic cardiac catheterization, revealed a small diffusion abnormality on the right corona radiata. Postoperative scan, performed on postoperative day 4, demonstrated 2 new discrete areas of diffusion restriction in the inferior cerebellar hemisphere and posterior watershed territory on the right side, consistent with embolic cerebral infarctions. PWI (not shown) did not show any perfusion defects.
to 1.0 T), broad slice thickness (sometimes 8 mm), and limited sequences (mostly T1- and T2-weighted images). One substantial limitation of conventional MRI sequences is that white matter hyperintensities (reported in 54% to 100% of patients undergoing CABG) may “hide” new superimposed lesions. Therefore, it is plausible that some of the previous studies actually report falsely negative MRI examinations. It is also conceivable that current imaging techniques incorporating DWI may enable us to detect only the “tip of the iceberg,” with most embolic material being too small to cause an infarct of sufficient conspicuity on MRI. On the other hand, a postoperative neurocognitive decline could be explained by factors different from ischemia (ie, the effects of polypharmacy). Such factors could explain why some of our patients with neurocognitive test decline (including 1 of the 3 patients with decline in the composite neurocognitive z score) did not have new MRI abnormalities.

Certain preoperative risk factors are associated with stroke and delirium after CABG. Although most patients from our cohort had a medium or high stroke risk, this finding is expected in about half of patients undergoing CABG in a tertiary referral center such as our institution. Despite this robust figure, only 4.8% of patients who are labeled preoperatively as having a medium or high risk of stroke eventually develop clinical stroke after surgery. We found that all subjects from our cohort with new postoperative DWI abnormalities had a history of HTN and postoperative episodes of atrial fibrillation. However, several patients without new lesions on postoperative MRI also had these factors. The occurrence of postoperative atrial fibrillation was found to increase the odds of a postoperative stroke by 3-fold in a retrospective study. A history of HTN is found in 63% patients with CABG and could decrease the occurrence or severity of strokes and postoperative encephalopathy. We suggest that the MRI technique of DWI may be a valuable tool for the evaluation of preventive and therapeutic interventions in patients undergoing CABG.

We conclude that focal postoperative DWI abnormalities, consistent with small embolic infarctions, can occur after CABG, even in patients without obvious neurological defects. Larger prospective studies are required to determine whether such lesions are associated with relevant neurocognitive changes or whether any postoperative stroke risk factor, particularly postoperative atrial fibrillation and HTN, are predictors of these MRI abnormalities.

Acknowledgments

This study was supported in part by a grant of the SmithKline Beecham pharmaceutical company and a gift from the Rogers Wilbur foundation (to R.J.W.). This article is dedicated to the enduring memory of Carlos J. Abad, MD. We are indebted to all the patients who kindly participated in this study. We appreciate the technical expertise of Charles Simpson, who performed the volumetric analysis of all MRI lesions. We are equally grateful to Jennifer Heidler, who participated in the neurocognitive testing of our patients.

References

23. Grandin CB, Duprez TP, Smith AM, et al. Which MR-derived perfusion parameters are best predictors of infarct growth in hyperacute...
Diffusion- and Perfusion-Weighted Magnetic Resonance Imaging of the Brain Before and After Coronary Artery Bypass Grafting Surgery
Lucas Restrepo, Robert J. Wityk, Maura A. Grega, Lou Borowicz Jr, Peter B. Barker, Michael A. Jacobs, Norman J. Beauchamp, Argye E. Hillis and Guy M. McKhann

Stroke. 2002;33:2909-2915; originally published online November 21, 2002;
doi: 10.1161/01.STR.0000040408.75704.15
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2002 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/33/12/2909

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/