Decreased Corticospinal Tract Fractional Anisotropy Predicts Long-term Motor Outcome After Stroke

Josep Puig, MD; Gerard Blasco, BSc; Josep Daunis-I-Estadella, PhD; Götz Thomalla, MD, PhD; Mar Castellanos, MD, PhD; Jaume Figueras, BSc; Sebastián Remollo, MD; Cecile van Eendenburg, MD; Javier Sánchez-González, PhD; Joaquín Serena, MD, PhD; Salvador Pedraza, MD

Background and Purpose—Nearly 50% of patients have residual motor deficits after stroke, and long-term motor outcome is difficult to predict. We assessed the predictive value of axonal damage to the corticospinal tract indexed by diffusion tensor imaging fractional anisotropy for long-term motor outcome.

Methods—Consecutive patients with middle cerebral artery stroke underwent multimodal MRI, including diffusion tensor imaging imaging ≤12 hours, 3 days, and 30 days after onset. Clinical severity, infarct volume, location of corticospinal tract damage on diffusion tensor tractography, and ratios of fractional anisotropy (rFA) between affected and unaffected sides of the corticospinal tract at the pons were evaluated. Severity of motor deficit at 2 years was categorized using the Motricity Index as no deficit (Motricity Index, 100), slight-moderate deficit (Motricity Index, 99–50), or severe deficit (Motricity Index, <50).

Results—We evaluated 70 patients (28 women; 72±12 years). rFA values at day 30 correlated with the degree of motor deficit at 2 years (P<0.001). rFA at day 30 was the only independent predictor of long-term motor outcome (odds ratio, 1.60; 95% confidence interval, 1.26–2.03; P<0.001). The sensitivity, specificity, and positive and negative predictive values of the cutoffs rFA<0.982 for predicting slight-moderate deficit and rFA<0.689 for severe deficit were 94.4%, 84.6%, 73.9%, and 97.1%, respectively, and 100%, 83.3%, 81.3%, and 100%, respectively.

Conclusions—rFA at day 30 is an independent predictor of long-term motor outcome after stroke. (Stroke. 2013;44:2016-2018.)

Key Words: cerebral ischemia diffusion tensor imaging fractional anisotropy

Motor deficit after stroke is common and its accurate prediction is important for management and rehabilitation.1 Age, sex, stroke type, lesion site, infarct volume, and initial motor impairment influence motor outcome.1,2 Neuroimaging studies suggest corticospinal tract (CST) integrity is important for motor outcome.3–8 Diffusion tensor imaging assesses the microstructural status of white matter objectively and quantitatively.9 Cross-sectional studies have established diffusion tensor imaging regional fractional anisotropy (FA) values as surrogate markers of motor deficit after stroke: lower values in affected CST correlate with worse motor function.5,10–12 To our knowledge, no prospective studies have assessed diffusion tensor imaging’s ability to predict long-term motor outcome after stroke. We assessed whether FA values in the CST predict motor outcome 2 years after stroke.

Methods

We scanned 89 consecutive patients with first-ever middle cerebral artery stroke within 12 hours of onset; data from 19 patients were incomplete at 2 years because of stroke recurrence (n=6), death (n=9), or motion artifacts (n=4). Analyses were, therefore, based on 70 subjects (28 women; aged 72±12 years). Our ethics committee approved the study, and all patients provided informed consent.

Severity of limb weakness was categorized by m-National Institute of Health Stroke Scale (m-NIHSS) subindex (5a, 5b, 6a, 6b): no weakness (grade I; total m-NIHSS score, 0), slight-moderate weakness (grade II; m-NIHSS score, 1–4), or severe weakness (grade III; m-NIHSS score, 5–8). Severity of motor deficit at 2 years was categorized by Motricity Index (MI) score: no deficit (total MI score, 100), slight-moderate deficit (MI, 99–50), or severe deficit (MI, <50). Clinical assessment was blind to MRI findings.

We used a 1.5-Tesla MRI system (Gyroscan Intera; Philips Medical Systems, Best, The Netherlands) and NeuroScape 2.0 MR Stroke Edition (Olea Medical, La Ciotat, France) for postprocessing. Details of FA measurement, diffusion tensor tractography reconstruction, diffusion tensor tractography assessment of CST damage, and infarct volume measurement are reported elsewhere.8,10 We calculated the FA ratio (rFA) between affected and unaffected sides of the CST at the pons.

Data were analyzed using Minitab version 16.1.1 (Minitab Inc, State College, PA). To determine which variables were associated with motor deficit severity, we used χ² tests for categorical and...
ANOVA for quantitative variables. Receiver operating characteristic curves were used to determine rFA cutoffs. Multivariate ordinal logistic regression was used to predict long-term motor outcome.

**Results**

Table I in the online-only Data Supplement reports demographic, clinical, and imaging data. At admission, 53/70 patients (75.6%) had motor deficits; 38/70 (54.1%) had severe motor deficit (m-NIHSS III). All patients were mobilized within 72 hours after onset and underwent standard physiotherapy during the next 6 months. At day 30, a total of 35 patients (50%) presented motor deficits; 15 (21.3%) were classified as m-NIHSS III. At 2-year follow-up, 31 patients (44.3%) presented motor deficits and 13 (18.6%) presented MI<50.

On admission, 14 patients (20%) had involvement of the posterior limb of the internal capsule; all these presented some motor deficit at 2-year follow-up ($P<0.001$). Infarct volume at admission, day 3, and day 30 correlated with motor deficit severity at 2 years.

Mean FA values in affected CST at the pons at day 30 decreased progressively in line with increasing motor deficit at 2-year follow-up ($P<0.001$). Mean FA values in unaffected CST increased in line with increasing motor deficit ($P=0.015$). rFA values at day 30 decreased in line with motor deficit at 2 years ($P<0.001$; Figure).

The rFA cutoff points distinguish patients without motor deficit at 2-year follow-up from those with slight-moderate deficit and even differentiate the latter from patients with severe motor deficits (Figure). The rFA cutoffs <0.982 for slight-moderate and <0.689 for severe motor deficit at 2 years yielded sensitivity 94.4% and 84.6%, specificity 73.9% and 97.1%, positive predictive value 100% and 83.3%, and negative predictive value 81.3% and 100%, respectively. rFA at day 30 was the only predictor of motor outcome at 2 years (odds ratio, 1.60; 95% confidence interval, 1.26–2.03; $P<0.001$; Table II in the online-only Data Supplement).

**Discussion**

We investigated whether distal CST axonal damage marked by decreased FA can predict long-term motor outcome. rFA was the only independent predictor of motor outcome 2 years after stroke: lower rFA at day 30 was strongly associated with poor outcome and 2 cutoffs predicted motor deficit severity.

Proposed predictors of motor outcome include motor deficit on admission, infarct volume and location, and CST involvement. Although greater initial motor impairment predicts worse motor recovery, the relation varies widely among subjects, and accurate prognosis remains difficult.13 Large infarct volumes do not necessarily predict poor outcome because motor deficit occurs only when critical motor regions are involved.4,6,8,13,14 Our findings corroborate the findings that lower FA values in affected CST were associated with greater motor deficit in the first week after stroke and worse motor outcome at 3 months, indicating motor outcome strongly depends on CST integrity.5,10 Regression analysis demonstrated that rFA at day 30 predicted long-term motor outcome better than well-established outcome measures like NIHSS score. Slightly but significantly increased anisotropy in contralesional CST in our sample at day 30 could represent adaptive structural remodeling.14

More than 50% of patients who survive the first month after stroke require specialized rehabilitation. However, rehabilitation is expensive, and its success depends on careful patient selection.13 Early information from rFA analysis might help tailor rehabilitation. Patients with lower rFA are less likely to benefit from active rehabilitation. Future rehabilitation trials might enable better selection of strategies for individual patients on the basis of the motor system integrity.

**Figure.** Relation between fractional anisotropy ratio (rFA) at day 30 and degree of motor deficit at 2-year follow-up. Prespecified cutoff points for predicting long-term motor outcome on basis of rFA are represented. The graphs show medians and quartiles. MI indicates Motricity Index.
Certain limitations merit comment. The m-NIHSS is not especially sensitive for limb impairment, and a more specific score administered at 30 days might better predict long-term motor outcome. Few patients had motor deficits at 2-year follow-up. Determining other anisotropic parameters like diffusion tensor, eigenvalues might be useful, and analyzing whether changes in the affected tract can be detected earlier (before day 30) might enable earlier prognosis.未来 studies should include more patients with various stroke types and locations, compare MRI outcome markers to others like NIHSS scale, and analyze other diffusion tensor imaging indices.

In conclusion, rFA at day 30 is an independent predictor of long-term motor outcome after stroke, although its usefulness as a surrogate must be validated.

Sources of Funding
This work was partially supported by a grant from the Spanish Ministry of Health (Fondo Investigaciones Sanitarias, reference PI060745).

Disclosures
None.

References
8. Puig J, Pedraza S, Blasco G, Daunis-I-Estadella J, Prados F, Rernello S, et al. Acute damage to the posterior limb of the internal capsule on diffusion tensor tractography by voxel-based analysis might help predict motor outcome. Future studies should include more patients with various stroke types and locations, compare MRI outcome markers to others like NIHSS scale, and analyze other diffusion tensor imaging indices.

In conclusion, rFA at day 30 is an independent predictor of long-term motor outcome after stroke, although its usefulness as a surrogate must be validated.

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References
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Stroke. 2013;44:2016-2018; originally published online May 7, 2013;
doi: 10.1161/STROKEAHA.111.000382

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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/44/7/2016

Data Supplement (unedited) at:
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On-line Table 1. Demographical, Clinical, Imaging and Outcome Data According to Motor Outcome at 2 Years Follow-up

<table>
<thead>
<tr>
<th></th>
<th>Overall (N=70)</th>
<th>MI 100 (N=39, 55.7%)</th>
<th>MI 99-50 (N=18, 25.7%)</th>
<th>MI &lt;50 (N=13, 18.6%)</th>
<th>P-value</th>
</tr>
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<td><strong>Demographics</strong></td>
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<td>Age, median (IQR)</td>
<td>72 (61-78.3)</td>
<td>71 (61-79)</td>
<td>67 (51.8-76.5)</td>
<td>75 (70.5-80)</td>
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<td>Female, n (%)</td>
<td>28 (40.0%)</td>
<td>13 (33.3%)</td>
<td>8 (44.4%)</td>
<td>7 (53.8%)</td>
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<td><strong>Risk factors</strong></td>
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<td>Hypertension, n (%)</td>
<td>34 (60.7%)</td>
<td>17 (58.6%)</td>
<td>9 (56.2%)</td>
<td>8 (72.7%)</td>
<td>0.653</td>
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<td>Diabetes, n (%)</td>
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<td>7 (12.5%)</td>
<td>2 (13.6%)</td>
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<td>Smoking, n (%)</td>
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<td>7 (43.8%)</td>
<td>3 (27.3%)</td>
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<td>Atrial fibrillation, n (%)</td>
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<td>4 (25%)</td>
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<td><strong>Stroke etiology (TOAST)</strong></td>
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<td>Large artery, n (%)</td>
<td>14 (25%)</td>
<td>7 (24.1%)</td>
<td>3 (18.8%)</td>
<td>4 (36.4%)</td>
<td></td>
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<td>Cardioembolic, n (%)</td>
<td>26 (46.4%)</td>
<td>13 (44.8%)</td>
<td>6 (37.5%)</td>
<td>7 (63.6%)</td>
<td></td>
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<tr>
<td>Indeterminate/other, n (%)</td>
<td>16 (28.6%)</td>
<td>9 (31%)</td>
<td>7 (43.8%)</td>
<td>0 (0%)</td>
<td></td>
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<td><strong>Clinical and Imaging Data</strong></td>
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<tr>
<td>Infarct side, right n (%)</td>
<td>29 (41.4%)</td>
<td>14 (35.9%)</td>
<td>6 (33.3%)</td>
<td>9 (69.2%)</td>
<td>0.077</td>
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<td><strong>Total NIHSS scores, median (IQR)</strong></td>
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<tr>
<td>Admission</td>
<td>12 (6-18)</td>
<td>9 (5-12)</td>
<td>15.5 (10.3-20)</td>
<td>19 (16-20)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Day 3</td>
<td>5.5 (1.8-11.3)</td>
<td>2 (1-4)</td>
<td>8 (6-13.5)</td>
<td>17 (12-19)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Day 30</td>
<td>2 (0-9)</td>
<td>1 (0-2)</td>
<td>5 (2-10)</td>
<td>16 (10-19)</td>
<td>&lt;0.001</td>
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<tr>
<td>Day 90</td>
<td>2 (0.8-4)</td>
<td>1 (0-2)</td>
<td>2.5 (1-4)</td>
<td>14 (4-4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Year 2</td>
<td>0 (0-6.3)</td>
<td>0 (0-0)</td>
<td>1 (0.8-6.5)</td>
<td>13 (10-14)</td>
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<td><strong>m-NIHSS scores, median (IQR)</strong></td>
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<tr>
<td>Admission</td>
<td>4.5 (1-8)</td>
<td>2 (0-5)</td>
<td>7 (1.8-8)</td>
<td>8 (7-8)</td>
<td>&lt;0.001</td>
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<tr>
<td>Day 3</td>
<td>0 (0-5)</td>
<td>0 (0-0)</td>
<td>3 (0.8-5.5)</td>
<td>8 (6-8)</td>
<td>&lt;0.001</td>
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<tr>
<td>Day 30</td>
<td>0 (0-4)</td>
<td>0 (0-0)</td>
<td>3 (0-4.3)</td>
<td>7 (5.5-8)</td>
<td>&lt;0.001</td>
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<tr>
<td>Day 90</td>
<td>0 (0-4)</td>
<td>0 (0-0)</td>
<td>1 (0-4.3)</td>
<td>7 (5.5-7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Year 2</td>
<td>0 (0-3)</td>
<td>0 (0-0)</td>
<td>1 (0-3)</td>
<td>7 (6-7.5)</td>
<td>&lt;0.001</td>
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<td><strong>FA at admission, median (IQR)</strong></td>
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<tr>
<td>Affected side</td>
<td>0.61 (0.65-0.61)</td>
<td>0.6 (0.64-0.6)</td>
<td>0.64 (0.66-0.64)</td>
<td>0.63 (0.67-0.63)</td>
<td>0.967</td>
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<td>Unaffected side</td>
<td>0.61 (0.65-0.61)</td>
<td>0.61 (0.64-0.61)</td>
<td>0.61 (0.68-0.61)</td>
<td>0.61 (0.65-0.61)</td>
<td>0.935</td>
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<td><strong>FA at day 3, median (IQR)</strong></td>
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<tr>
<td>Affected side</td>
<td>0.62 (0.65-0.62)</td>
<td>0.62 (0.65-0.62)</td>
<td>0.63 (0.69-0.63)</td>
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<td>0.61 (0.64-0.61)</td>
<td>0.63 (0.67-0.63)</td>
<td>0.61 (0.64-0.61)</td>
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### FA at day 30, median (IQR)

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<th>Location</th>
<th>FA at day 30, median (IQR)</th>
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<tbody>
<tr>
<td>Affected side</td>
<td>0.57 (0.63-0.57)</td>
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<tr>
<td>Unaffected side</td>
<td>0.62 (0.65-0.62)</td>
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<tr>
<td>rFA</td>
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<tr>
<td>Admission</td>
<td>1.01 (1.05-1.01)</td>
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<tr>
<td>Day 3</td>
<td>1.01 (1.05-1.01)</td>
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<tr>
<td>Day 30</td>
<td>0.98 (1.02-0.98)</td>
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<td>Location of CST damage, admission, n (%)</td>
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<tr>
<td>Motor cortex</td>
<td>18 (25.7%)</td>
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<tr>
<td>Premotor cortex</td>
<td>15 (21.4%)</td>
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<tr>
<td>Centrum semiovale</td>
<td>12 (17.1%)</td>
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<tr>
<td>Corona radiata</td>
<td>35 (50.0%)</td>
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<tr>
<td>PLIC</td>
<td>14 (20.0%)</td>
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<td>Location of CST damage day 3, n (%)</td>
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<tr>
<td>Motor cortex</td>
<td>16 (22.9%)</td>
</tr>
<tr>
<td>Premotor cortex</td>
<td>17 (24.3%)</td>
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<tr>
<td>Centrum semiovale</td>
<td>18 (25.7%)</td>
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<td>Corona radiata</td>
<td>39 (55.7%)</td>
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<td>PLIC</td>
<td>17 (24.3%)</td>
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<td>Infarct volume, median (IQR)</td>
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<tr>
<td>Admission</td>
<td>9.2 (4.8-24.7)</td>
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<td>Day 3</td>
<td>19.1 (9.2-53.5)</td>
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<td>Day 30</td>
<td>10.1 (4.1-40.3)</td>
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<td>Treatment process and outcomes</td>
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<td>IV rt-PA treatment, n (%)</td>
<td>36 (41.4%)</td>
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<td>Time onset to IV rt-PA, min (IQR)</td>
<td>170.5 (150-191.3)</td>
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<td>Recanalization after IV rt-PA, n (%)</td>
<td>14 (38.9%)</td>
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<td>Hemorrhagic transformation, n (%)</td>
<td>25 (44.6%)</td>
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<td>mRS, median (IQR)</td>
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<td>Day 30</td>
<td>2 (1-4)</td>
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<td>Day 90</td>
<td>1 (1-2)</td>
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<tr>
<td>Year 2</td>
<td>0 (0-2.3)</td>
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<td>BI Score, median (IQR)</td>
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<tr>
<td>Day 30</td>
<td>100 (48.8-100)</td>
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<td>Day 90</td>
<td>100 (63.8-100)</td>
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<td>Year 2</td>
<td>100 (77.5-100)</td>
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<td>Global</td>
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<td>--------------------------</td>
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<tr>
<td>MI Score at 2 years, median (IQR)</td>
<td>100 (61-100)</td>
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<td>84 (61-91)</td>
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<tr>
<td></td>
<td>25 (4.5-28.5)</td>
</tr>
<tr>
<td></td>
<td>&lt;0.001</td>
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</table>

MI indicates Motricity Index score; NIHSS, National Institutes of Health Stroke Scale; m-NIHSS, motor subindex score of the NIHSS; FA, fractional anisotropy; rFA, FA ratio; CST, corticospinal tract; PLIC, posterior limb of internal capsule; rt-PA, recombinant tissue plasminogen activator; mRS, modified Rankin Scale; BI, Barthel Index
**SUPPLEMENTAL MATERIAL**

On-line Table 2. Model Selected from Ordinal Logistic Regression Analysis for Predicting Long-term Motor outcome 2 years After Stroke

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Regression Coefficient</th>
<th>Z</th>
<th>P-value</th>
<th>Odds Ratio</th>
<th>95% CI</th>
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<tr>
<td>rFA day 30</td>
<td>0.472</td>
<td>3.90</td>
<td>&lt;0.001</td>
<td>1.60</td>
<td>1.26-2.03</td>
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<td>Infarct volume day 3</td>
<td>-0.023</td>
<td>-1.47</td>
<td>0.142</td>
<td>0.98</td>
<td>0.97-1.01</td>
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<tr>
<td>m-NIHSS II day 30</td>
<td>-0.240</td>
<td>-0.14</td>
<td>0.887</td>
<td>0.79</td>
<td>0.03-21.33</td>
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<td>m-NIHSS III day 30</td>
<td>2.342</td>
<td>0.85</td>
<td>0.394</td>
<td>10.40</td>
<td>0.05-22.60</td>
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<td>PLIC damage at admission</td>
<td>-2.808</td>
<td>-1.35</td>
<td>0.177</td>
<td>0.06</td>
<td>0.00-3.57</td>
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<tr>
<td>Constant (1)</td>
<td>-44.872</td>
<td>-3.83</td>
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<td>NA</td>
<td>NA</td>
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<td>Constant (2)</td>
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<td>-3.89</td>
<td>&lt;0.001</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

m-NIHSS  indicates motor subindex score of the National Institutes of Health Stroke Scale; rFA, fractional anisotropy ratio; PLIC, posterior limb of internal capsule; rt-PA.