Size Matters
Hemorrhage Volume as an Objective Measure to Define Significant Intracranial Hemorrhage Associated With Thrombolysis

Gregory A. Christoforidis, MD; Andrew Slivka, MD; Yousef Mohammad, MD; Christopher Karakasis, BS; Bindu Avutu, MPH; Ming Yang, MD

Background and Purpose—This study defines significant thrombolysis associated intracranial hemorrhage (ICH) by identifying an objective threshold volume that predicts clinical deterioration attributable to ICH.

Methods—Prospectively collected clinical and radiographic information, from 103 consecutive patients who underwent intraarterial thrombolysis for acute ischemic stroke, was reviewed. Multiple paired comparisons between stratified hematoma volume and change in National Institutes of Health Stroke Scale (NIHSS) score by 24 to 36 hours and by time of hospital discharge was used to identify significant differences. Associations between hemorrhage volume and infarct volume in relation to clinical outcomes were examined. Rates of hemorrhagic transformation (HT), symptomatic hemorrhage, and parenchymal hematoma involving over 30% of the infarct were compared with hemorrhage volume. Multivariate regression analysis was used to determine the relationship between change in discharge NIHSS score and hemorrhage volume adjusting for known predictors of clinical outcomes.

Results—Multiple paired comparisons indicate that hemorrhage greater than 25 mL (HV25) had a more distinct impact on NIHSS score by time of hospital discharge than at 24 to 36 hours. Twenty-seven (26.2%) patients had HT and 12 (11.7%) had HV25. Among symptomatic hemorrhage, parenchymal hematoma involving over 30% of the infarct, and HV25, HV25 appeared more reflective of clinical deterioration from ICH. Hemorrhage volume increased with infarct volume but they were independently associated with change in NIHSS score on regression analysis.

Conclusion—Clinical deterioration from ICH and ischemic injury are more effectively distinguished at time of hospital discharge. The authors propose to define significant hemorrhage associated with thrombolysis as hemorrhage volume greater than 25 mL. (Stroke. 2007;38:1799-1804.)

Key Words: acute stroke ■ interventional neuroradiology ■ intracerebral hemorrhage ■ thrombolysis ■ thrombolytic RX

Symptomatic intracerebral hemorrhage (ICH) is considered the most devastating complication of thrombolytic treatment for acute ischemic stroke. Definitions for symptomatic ICH in randomized clinical trials such as the National Institute of Neurological Disorders and Stroke tissue plasminogen activator trial, European Cooperative Acute Stroke Study (ECASS), Alteplase Thrombolysis for Acute Noninterventional Therapy in Ischemic Stroke, and Prolyse in Acute Cerebral Thromboembolism differ.1-5 Their rates vary and may be affected by changes in clinical outcome attributable to progression of ischemic injury or recovery from reperfusion. Because there are obvious implications when evaluating the risk–benefit ratio and ultimate outcome of thrombolysis, it stands to reason that the significance of ICH in the setting of thrombolysis for acute ischemic stroke needs further investigation. With these issues in mind, this study examines the experience of patients treated with intraarterial thrombolysis (IAT) at a single center to: (1) determine how clinical outcomes 24 to 36 hours after stroke onset at the time of hospital discharge and at 3 months are influenced by the presence and extent of ICH, clinical features at presentation, and ischemic injury measured as infarct volume; (2) identify a threshold volume for significant hemorrhage; and (3) compare this threshold volume with known methods for identifying significant hemorrhage in the setting of acute ischemic stroke.

Materials and Methods
This study includes consecutive patients who underwent IAT using tissue plasminogen activator (up to 100 mg), urokinase (up to 1 million units), and prourokinase (up to 9 mg) from May 1995 until April 2006. Data were collected using an Institutional Review Board-approved protocol. Patients seen within 6 hours of symptoms who were considered thrombolytic candidates using clinical, laboratory, CT, and angiographic criteria derived from Prolyse in Acute
Cerebral Thromboembolism underwent screening cerebral angiography. Unlike the Prolyse in Acute Cerebral Thromboembolism study, this study also included patients with ischemic stroke involving territories other than the middle cerebral artery and patients over the age of 85. The microcatheter was positioned within the thrombus for delivery of thrombolytic agents. Patients who underwent embolectomy were excluded. Most patients receiving tissue plasminogen activator were not anticoagulated after treatment, whereas patients receiving urokinase or prourokinase were. Presentation National Institutes of Health Stroke Scale (NIHSS) score (NIHSS), NIHSS of 24 to 36 hours after ictus (24hrNIHSS), NIHSS at the time of hospital discharge (dcNIHSS), modified Rankin scale score (mRankin) at 3 months, presentation laboratory values (platelet count, glucose level), systolic blood pressure, length of hospital stay, time to treatment, age, and sex were all recorded prospectively. Change in NIHSS (ΔNIHSS) from the time of ictus was calculated at 24 to 36 hours (Δ24hrNIHSS) and at the time of hospital discharge (ΔdcNIHSS). NIHSS and mRankin were determined by a stroke neurologist (A.S., Y.M.).

Angiograms of all patients were reviewed for occlusion site, pial collateral formation, and reperfusion by an interventional neuroradiologist (G.A.C.) who was blind to all clinical information during this review. Pial collaterals were graded as good or poor based on anatomic extent as defined elsewhere. Reperfusion was assessed as a percentage of the affected vascular territory that was revascularized. Both pial collaterals and reperfusion have been shown to be associated with improved outcomes after thrombotic treatment. Acute infarction on 24- to 48-hour CT was defined as a new hypodense region relative to pretreatment CT. Hemorrhagic transformation (HT) was defined as a new hyperdense region identified on any follow-up CT scan before patient discharge that did not display rapid clearance to suggest contrast extravasation. Infarcted regions and foci of hemorrhage were traced out on axial cross-sectional images by a certificate of added qualification (CAQ)-certified neuroradiologist (G.A.C.) and areas were calculated using IMPAX image analysis software (Agfa Corp., Ridgefield Park, NJ). Infarct and hemorrhage volumes were then determined by multiplying cross-sectional areas by slice thickness and adding them up. The clinical categories of symptomatic hemorrhagic transformation similar to the ECASS definition (SHT) and symptomatic hemorrhagic transformation similar to the Alteplase Thrombolysis for Acute Noninterventional Therapy in Ischemic Stroke definition (SHTA) were defined as any HT associated with a positive Δ24hrNIHSS of 4 or more and 2 or more, respectively. The category of parenchymal hematoma involving over 30% of the infarct (PH2) was defined as a HT whose volume was more than 30% of the volume of the area of infarction using the previously described volumetric measurements rather than estimates as done previously.

Shapiro-Wilk W test was used to reject the supposition of normality for ΔNIHSS, if appropriate. Significant mean difference test was performed between stratified hematoma volume (0 to 10, 10 to 25, 25 to 50, and 50 plus mL) and both Δ24hrNIHSS and ΔdcNIHSS to determine significant mean differences. ΔNIHSS was treated as an interval variable. Once a significant difference was identified, it was used to reclassify hemorrhage volume into 3 categories (no hemorrhage, hemorrhage volume below the threshold, and hemorrhage volume above the threshold). Multivariate analyses were used to determine significance of hemorrhage volume, clinical outcome, and infarct volume. ΔNIHSS was treated as an interval variable; however, patients who died were reported separately for multivariate analysis. Median infarct volume and mean Δ24hrNIHSS and mean ΔdcNIHSS were then calculated for each hemorrhage volume and compared. Infarct volume was stratified and compared with ΔNIHSS and stratified hemorrhage volume. Bonferroni correction was adapted as needed. Statistical analysis was done using JMP statistical software. The data were reviewed to assess the impact of infarct volume and hemorrhage volume on clinical outcomes.

Linear regression analysis was performed to determine the relationship between normally distributed ΔdcNIHSS and hemorrhage volume while adjusting for age, diabetes, platelet counts, glucose level, presenting NIHSS, time to treatment, thrombolytic agent, pial collateral formation, extent of reperfusion, and infarct volume and to determine any interaction between these variables. All of these factors have been previously shown to be associated with either cerebral hemorrhage after thrombolytic treatment or outcome. Like the Prolyse in Acute Cerebral Thromboembolism study, ΔΔ NIHSS was not normally distributed (P<0.001), separating those patients who died did not reject normality for either surviving patients (W=0.99; P<0.89) or patients who died by time of hospital discharge (W=0.96; P<0.71). Normality was not rejected for Δ24hrNIHSS (W=0.97; P<0.19). To fulfill the assumption for normality for analysis of variance and linear regression analysis for ΔNIHSS, patients who died were analyzed separately.

Figures 1 and 2 demonstrate that the apparent effect of hemorrhage on Δ24hrNIHSS versus ΔdcNIHSS differs. A dichotomized effect of hemorrhage volume becomes apparent at time of hospital discharge based on multiple comparison tests. NIHSS improved in patients with HT with hemorrhage volumes of 0 to 10 mL and 10 to 25 mL in a statistically significant fashion based on the multiple comparisons test, whereas patients with HT volumes of 25 to 50 mL and greater than 50 mL were associated with increases in NIHSS. Therefore, 25 mL was used as a threshold volume in defining significant hemorrhage volume.

Table 1 compares clinical improvement, based on ΔNIHSS, with hemorrhage volume. Patients with hemorrhage volumes less than 25 mL had similar outcomes as those
Table 1. Clinical Change and Infarct Volumes Relative to Stratified Hemorrhage Volume

<table>
<thead>
<tr>
<th>Hemorrhage Volume</th>
<th>0</th>
<th>&lt;25 mL</th>
<th>&gt;25 mL</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>76</td>
<td>15</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Change in NIHSSS between ictus and time of hospital discharge†</td>
<td>−6.08 (0.73)</td>
<td>−6.36 (1.67)</td>
<td>+6.25 (3.12)</td>
<td>0.0009‡</td>
</tr>
<tr>
<td>Change in NIHSSS 24–36 hours following ictus§</td>
<td>−4.1 (0.725)</td>
<td>−1.73 (1.63)</td>
<td>+5.5 (1.99)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>No. of deaths by the time of hospital discharge (column percentage in parentheses)</td>
<td>2 (2.6%)</td>
<td>1 (7.1%)</td>
<td>8 (61.5%)</td>
<td>&lt;0.0001Ⅰ</td>
</tr>
<tr>
<td>Median infarct volume in 92 patients who did not die in milliliters with quartile range</td>
<td>33.4 (8.93–89.8)</td>
<td>96.4 (41.2–180)</td>
<td>90.2 (77.0–242)</td>
<td>0.0075▌</td>
</tr>
<tr>
<td>Median infarct volume in 11 patients who died in milliliters with quartile range</td>
<td>150 (53.4–247)</td>
<td>272</td>
<td>333 (234–490)</td>
<td>0.218▌</td>
</tr>
<tr>
<td>Median infarct volume in all 103 patients in milliliters with quartile range</td>
<td>35.3 (9.1–91.1)</td>
<td>98.8 (45.9–191)</td>
<td>282 (113–372)</td>
<td>&lt;0.0001▌</td>
</tr>
</tbody>
</table>

Standard error values or quartile range are in parentheses.
*Bonferroni correction requires P≤0.0083 for significance.
†Does not include patients who died during hospitalization.
§Analysis of variance.
¥Does not include 2 patients who died within 24 hours of treatment.
¶Pearson.
▌Wilcoxon rank sums.

Discussion

Definitions for symptomatic ICH suggest a cause–effect relationship between ICH and clinical deterioration.1–4 However, experience shows that deterioration can also occur in patients with infarction and no ICH, and improvement can occur as a result of reperfusion. In an attempt to address these concerns, this study was undertaken to determine whether a threshold ICH volume could serve as an objective method, which defines clinically significant hemorraghes. To accomplish this, the time point at which clinical deterioration from hemorrhage and clinical deterioration from ischemic injury after acute ischemic stroke onset differed the most was identified and the threshold volume was subsequently identified (Figures 1 and 2; Table 1). The association of hemorrhage volume to ischemic injury measured as infarct volume relative to clinical outcome was then assessed (Table 2). Methods currently used to identify significant ICH were then compared with HV25 (Table 3). Finally, a multivariate regression analysis was performed to determine the relative

without hemorrhage. Among patients who did not die during their hospitalization, those with hemorrhage volumes greater than 25 mL (HV25) had worse outcomes. A substantially higher death rate (66.7% versus 3.3%) occurred in those patients with HV25. Hemorrhage volume was shown to increase with infarct volume.

Table 2 compares clinical improvement with infarct volume taking into account hematoma volume. Hemorrhage rate and hemorrhage volume both increased with infarct volume. ΔNIHSSS and death rate worsened with larger infarct volumes. HV25 appeared to be associated with higher death rates in the presence of larger infarct volumes. Small hematomas appear to have a smaller impact on clinical outcome. Patients with infarct volumes less than 50 mL had hematoma volumes less than 25 mL and significantly improved by the time of hospital discharge based on matched pairs analysis (P=0.0032). Lack of statistical significance does not obviate type II error. With statistical power of 90%, ΔNIHSSS should be no greater than 11 for large infarct volumes (>200 mL) and no greater than 6 in smaller infarct volume subgroups (0 to 50 mL).

Table 3 compares patients with no hemorrhage, HT, PH2, SHT, and HV25 for outcomes at 24 to 36 hours, time of discharge, and at 3 months. A total of 19 patients with HT did not qualify as SHT; 6 (31.6%) of these patients had HV25 (3 of whom died during their hospital stay and one went on to deteriorate by a total of 5 points by the time of hospital discharge). The other 2 patients had infarct volumes that were relatively smaller. Twelve of the other 13 patients improved. One patient with hemorrhage volume 3 mL and an infarct volume of 388 mL deteriorated by 2 points on the NIHSS.

Multivariate regression analysis for ΔNIHSSS is shown in Table 4. Presenting NIHSSS, infarct volume, and hemorrhage volume were independently associated with increases in NIHSSS, whereas extent of reperfusion was associated with decreases in NIHSSS.
Impact of hemorrhage volume on clinical outcomes while adjusting for other variables (Table 4). Because thrombolytic agents are generally cleared within 24 to 36 hours after administration, identifying ICH during this timeframe establishes a cause–effect relationship. Defining the clinical impact at that limited timeframe may falsely estimate the long-term consequences. The pathophysiology of edema attributable to cerebral infarction and ICH may help

**TABLE 3. Comparison of Methods for Assessing Hemorrhage to Clinical Outcomes**

<table>
<thead>
<tr>
<th></th>
<th>0–50</th>
<th>50–200</th>
<th>&gt;200</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. (percent per IV subgroup)</td>
<td>48 (92.3%)</td>
<td>7 (38.9%)</td>
<td>3 (16.7%)</td>
</tr>
<tr>
<td>ΔNIHSSS by the time of hospital discharge†</td>
<td>−6.90 (1.01%)</td>
<td>−1.83 (1.12%)</td>
<td>−5.00 (1.94%)</td>
</tr>
<tr>
<td>P value</td>
<td>0.663‡</td>
<td>0.0013‡</td>
<td>0.149‡</td>
</tr>
<tr>
<td>ΔNIHSSS 24–36 hours after ictus§</td>
<td>−5.52 (1.06)</td>
<td>−0.286 (1.96)</td>
<td>+3.33 (2.99)</td>
</tr>
<tr>
<td>P value</td>
<td>0.598‡</td>
<td>0.0001‡</td>
<td>0.345‡</td>
</tr>
<tr>
<td>Deaths while hospitalized (percent column)</td>
<td>0 0</td>
<td>1 (4.76%)</td>
<td>1 (14.3%)</td>
</tr>
<tr>
<td>Deaths by 24 hours (percent column)</td>
<td>0 0</td>
<td>0 0 0 0</td>
<td>0 0 2 (25%)</td>
</tr>
</tbody>
</table>

Standard error values or percentages per infarct volume subgroups/column are in parentheses.
*Bonferroni correction requires P≤0.0083 for significance.
†Does not include patients who died during hospitalization.
‡Analysis of variance.
§Does not include patients who died within 24 hours of treatment.
IV indicates infarct volume.

Impact of hemorrhage volume on clinical outcomes while adjusting for other variables (Table 4).

Because thrombolytic agents are generally cleared within 24 to 36 hours after administration, identifying ICH during this timeframe establishes a cause–effect relationship. Defining the clinical impact at that limited timeframe may falsely estimate the long-term consequences. The pathophysiology of edema attributable to cerebral infarction and ICH may help

**TABLE 2. Subgroup Analysis Comparing Clinical Improvement After Treatment Relative to Infarct and Hemorrhage Volumes***

<table>
<thead>
<tr>
<th>Infarct Volume (mL)</th>
<th>0–50</th>
<th>50–200</th>
<th>&gt;200</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage Volume (mL)</td>
<td>0</td>
<td>&lt;25</td>
<td>&gt;25</td>
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<tr>
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<td>0 0</td>
<td>0 0 0 0</td>
<td>0 0 2 (25%)</td>
</tr>
</tbody>
</table>

*Three months follow-up mRankin was missing in 5 patients; 3 months indicates 3 months follow up.
explain why differences in clinical outcome differ more at the time of hospital discharge than at 24 to 36 hours. Edema from acute ischemic stroke occurs at maximum 3 to 5 days after ictus, whereas edema from hemorrhage occurs at maximum up to 2 to 3 weeks with ICH and is strongly predictive of functional outcomes.\(^{14,15}\) Although hematoma volumes tend to be larger in thrombolysis-related ICH, associated edema tends to be less than in spontaneous ICH.\(^{16}\) Assuming that edema attributable to ischemia transiently impacts clinical deterioration in the first few days, waiting until edema attributable to ischemia has receded may help clarify the true impact of hemorrhage that persists longer. Indeed, Tables 1, 2, and 3 indicate that patients without hemorrhage tended to improve during their hospital course, unlike patients with HV25 who deteriorated.

Data presented in this study support the theoretical rationale for evaluating clinical deterioration attributable to hemorrhage at hospital discharge rather than at 24 to 36 hours. In this study, 24 to 36 hours after presentation, 16 patients deteriorated by 4 or more points on the NIHSS; only 8 (50\%) had an associated hemorrhage. This implies that clinical deterioration may occur regardless of presence of hemorrhage and may confound evaluation of the impact of the hemorrhage. Among 19 patients with HT but not SHT, 6 patients had HV25, 3 of whom died and one went on to deteriorate by a total of 5 points by the time of hospital discharge; in addition, Tables 1 and 2 show that patients with larger hemorrhages tended to die during their hospital stay, especially when associated with large infarct volumes. Therefore, characterizing symptomatic hemorrhage within the first 24 or 36 hours after thrombolysis would not be representative of potential consequences of the hemorrhage. Furthermore, patients who developed small hemorrhages (<25 mL) initially did not do as well as those who did not hemorrhage within the first 24 to 36 hours but by the time of hospital discharge tended to do as well. Clinical improvement in patients with smaller hemorrhages by the time of hospital discharge and clinical deterioration in patients with larger hemorrhages by the time of hospital discharge suggest that it may be more effective to use the ΔcNIHSSS rather than the Δ24hrNIHSSS as a more comprehensive indicator of clinical significance of such hemorrhages.

Furthermore, in Table 3, comparison of clinical outcomes at various timeframes supports the use of clinical outcome at the time of hospital discharge rather than at 24 to 36 hours or at 3 months. The rates at which patients with hemorrhage at 24 to 36 hours had either a Δ24hrNIHSSS more than 4 were approximately 2- to 3-fold more than in those patients without hemorrhage. At the time of hospital discharge, these rates differed by approximately 5- to 7-fold. Three-month mRankin was only 2-fold larger in patients with HT. Three-month outcomes measured by mRankin are more likely influenced by factors not directly related to the stroke and require large patient cohorts to determine clinically significant effects from acute ICH.\(^{17}\) The most striking difference was with death rates, which had an almost 13-fold difference while in-house and 4.5-fold by 3 months. It is thus evident that clinical deterioration by the time of hospital discharge reflects the effect of ICH more clearly than deterioration by 24 to 36 hours or at 3 months. This helps explain why the impact of hemorrhage volume becomes more apparent by the time of hospital discharge in Figures 1 and 2 and helps establish a threshold ICH volume of 25 mL.

Results in this study confirm data derived from ECASS\(^{3}\) indicating that clinical deterioration in patients after stroke can occur with or without hemorrhagic transformation. Tables 1 through 4 demonstrate that increasing infarct volume (a measure of ischemic injury) is associated with clinical deterioration as well as larger and more frequent hemorrhage. Table 2 indicates that the risk of death during hospitalization is substantially higher when a large infarction (>200 mL) is associated with a large hemorrhage (>25 mL). Among the patients who died with infarct volumes less than 200 mL, one had a posterior fossa infarction (basilar artery thrombosis) without associated hemorrhage and the other had a hematoma volume of 115 mL. Factors such as basilar artery thrombosis and excessive hemorrhage relative to infarct volume may therefore also need to be taken into consideration when assessing clinical deterioration. However, of the 11 in-hospital deaths in this series, 8 occurred in patients with HV25 with a median infarct volume of 333 mL, which contrasts with the median infarct volume of 90.2 mL in the 5 patients who survived with HV25. It becomes clear that death predominantly occurs in patients with large infarctions who also develop a large hemorrhage.

Definitions of symptomatic ICH in major clinical trials differ. Along with blood at any site on CT scan, symptomatic ICH was defined as: a decrease in NIHSSS of 4 or more points within 36 hours in ECASS II;\(^{2}\) clinical deterioration in the judgment of the clinical investigator within 36 hours of treatment in the National Institute of Neurological Disorders and Stroke;\(^{1}\) deterioration of 2 or more points on the NIHSS by 24 hours in Alteplase Thrombolysis for Acute Noninter-

| Parameter | Estimate | SE  | t Ratio | P>|t| |
|-----------|----------|-----|--------|-------|
| Intercept | -2.78    | 1.62| -1.72  | 0.0893|
| Presenting NIHSS score | 0.474 | 0.0928 | 5.11 | <0.0001 |
| Infarct volume | -0.0204 | 0.00687 | -2.97 | 0.0038 |
| Hemorrhage volume | -0.117 | 0.0508 | -2.30 | 0.0238 |
| Extent of reperfusion (percent) | 0.0578 | 0.0126 | 4.58 | <0.0001 |

\(r^2=0.47; P<0.0001.\) Variables with \(P>0.10\) were excluded from the final model.

**TABLE 4. Multivariate Regression Analysis for Change in NIHSS Score by the Time of Hospital Discharge in Patients Who Survived**

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Christoforidis et al. Hemorrhage Volume and Intraarterial Thrombolysis
ventional Therapy in Ischemic Stroke; and deterioration of 4 or more points on the NIHSS in Prolyse in Acute Cerebral Thromboembolism II. The ECASS study group reassessed hemorrhagic infarction using CT scans by applying previous definitions, which did not consider clinical outcome. They divided ICH into 4 subtypes and found only PH2 hemorrhage to be associated with clinical deterioration. HV25 appears to reflect deterioration attributable to hemorrhage more accurately than other methods. As stated earlier, the most striking differences between patients with and without HT are death and large clinical deteriorations by time of hospital discharge. Examining these 2 parameters on Table 3, it becomes apparent that among patients with HT who either died or had ΔdcNIHSSS +4 or greater, HV25 has more patients in common. As stated earlier, SHT does not take into account patients with ICH who deteriorated further during their hospital stay. One should expect a percentage of patients with HT would deteriorate predominantly attributable to ischemic injury. Accordingly, the percentage of patients who deteriorate attributable to hemorrhage should be equivalent to the total patients who deteriorated as a result of hemorrhage less the percentage of patients who deteriorated without hemorrhage. Of the methods compared in Table 3, HV25 more closely estimates deterioration attributable to hemorrhage in Table 3. Furthermore, as was the case in one patient in this study, complete reperfusion can counterbalance deterioration associated with HV25. This can also be inferred from the regression analysis (Table 4). These findings suggest that the significance of ICH after IAT may be more objectively assessed by measuring hemorrhage volume.

In the current study, there are several limitations of note. Retrospective review could result in misclassification and selection bias. These were controlled by using a consecutive series, blinding the investigator, and using preestablished definitions of SHT and PH2 to categorize patients. A larger prospective study can confirm the stated results. The reasoning that it is consistent with previous findings supports the regression model’s validity.

Conclusion
Although any hemorrhage may have a clinical impact, this study indicates that by the time of hospital discharge, patients with ICH volumes less than 25 mL fare as well as those without ICH, whereas ICH volumes greater than 25 mL are negatively associated with clinical outcome. Patients receiving IAT for acute ischemic stroke more often had large hemorrhages if the infarct volume was large. Finally, clinical deterioration related to hemorrhage volume becomes more apparent when assessing NIHSS scores at the time of hospital discharge rather than NIHSS score at 24 to 36 hours. The authors therefore propose to define significant hemorrhage associated with IAT as a hemorrhage volume greater than 25 mL within 24 hours of treatment. A volume-based definition of ICH is suggested to provide a more objective description of the impact of ICH relative to current definitions of SHT.

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Disclosures
None.

References
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