

## Metrics for Measuring Quality of Care in Comprehensive Stroke Centers: Detailed Follow-Up to Brain Attack Coalition Comprehensive Stroke Center Recommendations

### A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

*Endorsed by the Society of Vascular and Interventional Neurology*

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**Background**—Stroke is a major cause of disability and death. The Brain Attack Coalition has proposed establishment of primary and comprehensive stroke centers to provide appropriate care to stroke patients who require basic and more advanced interventions, respectively. Primary stroke centers have been designated by The Joint Commission since 2003, as well as by various states. The designation of comprehensive stroke centers (CSCs) is now being considered. To assist in this process, we propose a set of metrics and related data that CSCs should track to monitor the quality of care that they provide and to facilitate quality improvement.

**Methods and Results**—We analyzed available guideline statements, reviews, and other literature to identify the major features that distinguish CSCs from primary stroke centers, drafted a set of metrics and related data elements to measure the key components of these aspects of stroke care, and then revised these through an iterative process to reach a consensus. We propose a set of metrics and related data elements that cover the major aspects of specialized care for patients with ischemic cerebrovascular disease and nontraumatic subarachnoid and intracerebral hemorrhages at CSCs.

**Conclusions**—The metrics that we propose are intended to provide a framework for standardized data collection at CSCs to facilitate local quality improvement efforts and to allow for analysis of pooled data from different CSCs that may lead to development of national performance standards for CSCs in the future. (*Stroke*. 2011;42:849-877.)

**Key Words:** AHA Scientific Statements ■ cerebrovascular disorders ■ cerebral hemorrhage ■ ischemic stroke ■ healthcare systems ■ patient care ■ university medical centers

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Stroke is the third-leading cause of death in the United States and a leading cause of disability. Its manifestations are extremely variable and often profoundly and permanently change a patient's quality of life or even lead to death. Each year,  $\approx 795\,000$  people experience a new or recurrent stroke. Approximately 610 000 of these are first attacks, and 185 000 are recurrent attacks.<sup>1</sup> To assist in ensuring adequate care for stroke patients, the Brain Attack Coalition (BAC) has proposed 2 levels of hospitals for the treatment of stroke patients: Primary stroke centers (PSCs) and comprehensive stroke centers (CSCs).<sup>2,3</sup> In addition, the concept of stroke systems of care that facilitate treatment of stroke patients at the most appropriate type of hospital has been suggested.<sup>4,5</sup> Most stroke patients can be treated appropriately at PSCs. Some patients, however, require intensive care and specialized techniques that are not available at most PSCs but constitute the key features of CSCs.<sup>3</sup> Given this background, the success of the multilevel system of care for trauma patients<sup>6</sup> suggests that there may be similar benefits to establishing such a system for stroke patients.

After the PSC concept was proposed, The Joint Commission and several states independently<sup>6–13</sup> established programs for certification of PSCs. Metrics for measuring performance of PSCs were defined and underwent several cycles of modification as experience in implementing them was obtained.<sup>10,11</sup> Analysis of PSC performance has indicated that establishment of PSCs with concomitant development of formal protocols for stroke care and measurement of adherence to the metrics has been associated with improvement in stroke care.<sup>12–15</sup> Several states have now moved ahead with plans to certify CSCs.<sup>7–9</sup>

In this setting, to help in the development of CSCs, we now propose a set of metrics and associated data elements that cover the major types of care that distinguish CSCs from PSCs. We intend that these metrics will assist in the establishment of CSCs, facilitate quality improvement activities at individual centers, and ultimately permit comparison of practices at different CSCs and ensure that hospitals designated as CSCs provide high levels of care. Quality improvement is an essential element of all medical care<sup>16</sup> but is especially important in a CSC where new techniques are being developed and refined for the care of critically ill patients with complex diseases.<sup>3</sup>

We anticipate that these metrics will undergo modification as experience is gained at CSCs. We want to emphasize that the proposed metrics are not performance measures in the sense that performance measures can be used for direct comparison of the quality of care at different institutions, but rather the proposed metrics should be viewed as quality measures that can facilitate improvement of care and may ultimately lead to the adoption of formal performance measures.<sup>17</sup> Some of the metrics have stronger evidence to support them or have greater clinical significance, and we have designated these as core metrics and the others as supplemental metrics. We anticipate that as the CSC concept is implemented, it may be appropriate to expect CSCs to measure the core metrics and a subset of supplemental metrics. This approach of requiring initial monitoring of only a partial set of metrics was used successfully by The Joint Commission when its PSC program was started.

## Methods

We began by reviewing the key components of CSCs as set forth in the BAC report.<sup>3</sup> We conducted an extensive literature review related to these elements and then drafted a set of metrics and related additional data elements to cover the distinguishing features of CSCs. In designing the metrics, our underlying assumption was that CSCs will be expected to meet all standards required for PSCs by the organization or authority certifying them. The initial draft underwent repeated revisions through cycles of conference calls and e-mail correspondence. Members of the writing committee specifically provided input in drafting metrics related to their areas of expertise and had opportunities to comment on all the metrics repeatedly until consensus was reached. We also obtained opinions from outside stroke experts during this process (see Acknowledgments). In drafting our recommendations, we sought to rely most strongly on formal guideline statements prepared under the auspices of the American Heart Association (AHA) and, when appropriate, other organizations. We particularly relied on items that the guideline statements ranked highest (Tables 1 and 2). We supplemented these with data from other reports and also considered the types of outcomes and complications that were monitored in important clinical trials in our efforts to define metrics related to individual processes of care. We sought to develop a consensus to achieve metrics that are evidence based and in keeping with clinical practice. We also sought to develop metrics that would be feasible to collect in a reproducible way and recognized that in some cases, the data that would be the most desirable might not be reliably collected in routine clinical practice.

In the course of our work, it became clear that some of the metrics had stronger support in the literature or more significant implications for quality of care. On the basis of these considerations, we reached a consensus about designating some of the metrics as core metrics, and we have designated the others as supplemental metrics. We have also identified additional data elements that we encourage CSCs to collect to facilitate interpretation of the data collected for the metrics and to provide additional information for quality improvement. We do not want to imply that this is a complete list of data elements, nor do we intend to suggest that CSCs should be required to collect all of these elements, at least not initially. We intend that the core metrics will be required initially and that CSCs may have some choice about which of the other metrics to record initially, and that over time, this requirement may be revised.

In formulating the metrics, we have divided the patients who will be cared for at a CSC into 3 categories that include the major stroke-related diseases: (1) ischemic cerebrovascular disease; (2) aneurysmal subarachnoid hemorrhage (SAH) and unruptured aneurysms; and (3) nontraumatic intracerebral hemorrhage (ICH; including hemorrhages from arteriovenous malformations [AVMs] and nonhemorrhagic AVMs). We have proposed process metrics and metrics that monitor complication rates and outcomes.

We have also proposed metrics that pertain to the documentation of the initial severity of stroke, aspects of intensive care unit (ICU) treatment and of rehabilitation care, transfer of patients from outside hospitals to a CSC, and participation in research. We also considered the risk adjustment that will be necessary to fully interpret data collected for some of the

**Table 1. Applying Classification of Recommendations and Level of Evidence**

		SIZE OF TREATMENT EFFECT <span style="float: right;">➔</span>			
		CLASS I <i>Benefit &gt;&gt;&gt; Risk</i> Procedure/Treatment <b>SHOULD</b> be performed/administered	CLASS IIa <i>Benefit &gt;&gt; Risk</i> <i>Additional studies with focused objectives needed</i> <b>IT IS REASONABLE</b> to perform procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> <i>Additional studies with broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment <b>MAY BE CONSIDERED</b>	CLASS III <i>Risk ≥ Benefit</i> Procedure/Treatment should <b>NOT</b> be performed/administered <b>SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</b>
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is useful/effective</li> <li>Sufficient evidence from multiple randomized trials or meta-analyses</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation in favor of treatment or procedure being useful/effective</li> <li>Some conflicting evidence from multiple randomized trials or meta-analyses</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation's usefulness/efficacy less well established</li> <li>Greater conflicting evidence from multiple randomized trials or meta-analyses</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is not useful/effective and may be harmful</li> <li>Sufficient evidence from multiple randomized trials or meta-analyses</li> </ul>
	LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is useful/effective</li> <li>Evidence from single randomized trial or nonrandomized studies</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation in favor of treatment or procedure being useful/effective</li> <li>Some conflicting evidence from single randomized trial or nonrandomized studies</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation's usefulness/efficacy less well established</li> <li>Greater conflicting evidence from single randomized trial or nonrandomized studies</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is not useful/effective and may be harmful</li> <li>Evidence from single randomized trial or nonrandomized studies</li> </ul>
	LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is useful/effective</li> <li>Only expert opinion, case studies, or standard of care</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation in favor of treatment or procedure being useful/effective</li> <li>Only diverging expert opinion, case studies, or standard of care</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation's usefulness/efficacy less well established</li> <li>Only diverging expert opinion, case studies, or standard of care</li> </ul>	<ul style="list-style-type: none"> <li>Recommendation that procedure or treatment is not useful/effective and may be harmful</li> <li>Only expert opinion, case studies, or standard of care</li> </ul>
Suggested phrases for writing recommendations†		should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	is not recommended is not indicated should not is not useful/effective/beneficial may be harmful

\*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use. A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Even though randomized trials are not available, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

†For recommendations (Class I and IIa; Level of Evidence A and B only) regarding the comparative effectiveness of one treatment with respect to another, these words or phrases may be accompanied by the additional terms “in preference to” or “to choose” to indicate the favored intervention. For example, “Treatment A is recommended in preference to Treatment B for . . .” or “It is reasonable to choose Treatment A over Treatment B for . . .” Studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

metrics, additional data collection related to in-hospital complications, and participation in registries that may facilitate standardized data collection.

We have also noted additional optional data elements that are related to the metrics and that we encourage CSCs to collect to assist in interpretation of the metric, to help in performance improvement, and to provide additional information for quality improvement for the metrics. Because of the extra resources that would be required to collect these additional data elements, we want to emphasize that it would be reasonable for centers to collect and analyze some of them as part of focused, time-limited quality improvement projects rather than collecting all of them on a continuous basis. We anticipate that centers might decide to conduct such specific projects on the basis of analysis of their performance on the metrics that we have proposed.

In formulating the metrics, we have not set specific performance benchmarks. There is not enough evidence to set

such quantitative standards; nevertheless, it is essential for CSCs to collect data about how quickly or how often they perform certain diagnostic and therapeutic procedures and what their complication rates are, so that they can use this information for quality improvement and so that quantitative standards that CSCs should meet can eventually be set.

## Results

Table 3 summarizes the metrics that we propose for CSCs. Table 3 indicates whether individual metrics are classified as core metrics that we recommend that all CSCs should collect initially and whether the metrics apply only to specific types of patients.

### Ischemic Stroke

#### Metric 1

**Percentage of patients who have an ischemic stroke or who have a transient ischemic attack (TIA) with a**

**Table 2. Definition of Classes and Levels of Evidence Used in AHA Stroke Council Recommendations**

Class I	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
Class IIa	The weight of evidence or opinion is in favor of the procedure or treatment
Class IIb	Usefulness/efficacy is less well established by evidence or opinion
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful
<i>Therapeutic recommendations</i>	
Level of Evidence A	Data derived from multiple randomized clinical trials or meta-analyses
Level of Evidence B	Data derived from a single randomized trial or nonrandomized studies
Level of Evidence C	Consensus opinion of experts, case studies, or standard of care
<i>Diagnostic recommendations</i>	
Level of Evidence A	Data derived from multiple prospective cohort studies using a reference standard applied by a masked evaluator
Level of Evidence B	Data derived from a single grade A study, or $\geq 1$ case-control studies, or studies using a reference standard applied by an unmasked evaluator
Level of Evidence C	Consensus opinion of experts

**deficit at the time of the initial admitting note or neurology consultation note for whom a National Institutes of Health Stroke Scale (NIHSS) score is documented. (Core metric)**

**Numerator:** Number of patients with ischemic stroke or TIA with a deficit at the time of the initial admitting or neurological consultation note for whom an NIHSS is documented. Patients are to be included in the numerator if the NIHSS is recorded in the first admitting note or in the first neurology consultation note, whichever comes first, or in a separate earlier note. Patients with acute ischemic stroke treated with intravenous tissue-type plasminogen activator (tPA) or with an acute endovascular procedure should be included in the numerator only if the NIHSS is performed before the start of these treatments.

**Denominator:** All patients who have an ischemic stroke or TIA with a deficit at the time of the initial admitting or neurology consultation note or who undergo intravenous tPA or acute endovascular treatment with complete resolution of their deficit.

For the purposes of this metric, patients with a TIA should be included if they still have a deficit at the time of the initial admitting or consultation note. In addition, the NIHSS should be performed by a certified examiner.

### *Justification*

The NIHSS is now well established as a reliable and reproducible indicator of ischemic stroke severity. The AHA/American Stroke Association (AHA/ASA) "Guidelines for the Early Management of Adults with Ischemic Stroke" advise the use of a standardized clinical examination for assessing stroke patients, "preferably the NIHSS," as a **Class I, Level of Evidence B** recommendation.<sup>18</sup> The NIHSS has been used in multiple major clinical trials.<sup>19–30</sup> It has been used in all of these trials and others for patient selection. It has been shown to be a strong predictor of outcome<sup>18,31</sup> and therefore is a fundamental factor in risk adjustment.

Ideally, the NIHSS should be administered before initial imaging is performed to establish a baseline and should therefore be administered to both ischemic and hemorrhagic stroke patients, but we recognize that often, this is not done before imaging in clinical practice. We also want to emphasize that imaging should not be delayed to perform the NIHSS. Because the NIHSS is not well established for use in hemorrhagic stroke patients, and because other clinical scales are better established for patients with SAH and ICH, as discussed below under Metric 12, we have not proposed requiring the NIHSS for hemorrhagic stroke patients. Because of the widespread acceptance of the NIHSS, its use in making clinical decisions and in determining eligibility for clinical trials, and the need for quantified measures of initial severity to interpret data about outcomes and other metrics, we have classified this metric as a core metric.

### *Additional Data Elements*

CSCs should also consider recording the size and location of ischemic strokes, the location of any related stenoses or occlusions, and the severity of any related stenoses. In addition, because the NIHSS is not a substitute for a complete neurological assessment by a vascular neurologist, CSCs should consider recording whether each stroke patient is evaluated by a vascular neurologist and the time from admission to evaluation.

### *Metric 2*

**Percentage of ischemic stroke patients eligible for intravenous thrombolysis who receive it within the appropriate time window. (Core metric)**

**Numerator:** Patients who arrive within 3.5 hours of last being known to be at baseline, are candidates for intravenous thrombolysis up to 4.5 hours since last being known to be at baseline, and are treated with intravenous thrombolysis within this time are to be included in the numerator. In addition, the subset of patients who arrive within 2.0 hours of last being known to be at baseline and are candidates for intravenous thrombolysis only up to 3.0 hours of last being known to be at baseline are also to be included in the numerator if they are treated within 3.0 hours.

**Denominator:** Patients who arrive within 3.5 hours of last being known to be at baseline and are candidates for intravenous thrombolysis up to 4.5 hours after last being known to be at baseline are included in the denominator. The subset of patients who arrive within 2.0 hours of last being known to be at baseline and are candidates for intravenous thrombolysis only up to 3.0 hours after last being known to be at baseline are also to be included in the denominator.

**Table 3. Summary of Metrics for Comprehensive Stroke Centers**

Metric	Core Metric	Ischemic Stroke, TIA, or Asymptomatic Cerebrovascular Stenosis	SAH and Nonruptured Aneurysm	ICH and AVM (With or Without Hemorrhage)
Metric 1: Percentage of patients who have an ischemic stroke or who have a TIA with a deficit at the time of the initial admitting note or neurology consultation note for whom an NIHSS score is documented.	Yes	Ischemic stroke, TIA	...	...
Metric 2: Percentage of ischemic stroke patients eligible for intravenous thrombolysis who receive it within the appropriate time window.	Yes	Ischemic stroke seen within 4.5 h of patient last being seen at baseline	...	...
Metric 3: Percentage of patients who are treated for acute ischemic stroke with intravenous thrombolysis whose treatment is started ≤60 minutes after arrival.	Yes	Ischemic stroke treated with intravenous thrombolysis	...	...
Metric 4: Median time from arrival to start of multimodal CT or MR brain and vascular imaging (MRI/MRA or CT/CTA) for ischemic stroke patients arriving within 6 hours of the time that they were last known to be at baseline, if 1 of these studies is ordered.	Yes	Ischemic stroke seen within 6 h of patient last being known to be at baseline	...	...
Metric 5: Percentage of ischemic stroke patients seen within 6 hours of the time they were last known to be at baseline who have documentation that an endovascular recanalization procedure either was performed or was considered and deemed not to be appropriate or possible. A reason should be documented if an endovascular procedure was not performed.	No	Ischemic stroke seen within 6 h of patient last being known to be at baseline	...	...
Metric 6: Median time from arrival to start of treatment for acute ischemic stroke patients undergoing an endovascular intervention.	No	Ischemic stroke treated with endovascular intervention	...	...
Metric 7: Percentage of patients treated with intravenous thrombolysis who have a symptomatic intracranial hemorrhage within 36 hours of treatment.	Yes	Ischemic stroke treated with intravenous thrombolysis	...	...
Metric 8: Percentage of acute ischemic stroke patients treated with endovascular interventions who develop significant intracranial hemorrhage within 36 hours of treatment.	Yes	Ischemic stroke treated with endovascular intervention	...	...
Metric 9: Percentage of acute ischemic stroke patients who are treated with intravenous thrombolysis or who undergo endovascular interventions for whom there is documentation of a 90-day mRS score.	Yes	Ischemic stroke treated with intravenous thrombolysis or endovascular procedure	...	...
Metric 10: Percentage of patients undergoing CEA, or carotid angioplasty or stenting, with stroke or death within 30 days of the procedure.	No	CEA or stenting	...	...
Metric 11: Percentage of patients undergoing intracranial angioplasty and/or stenting for atherosclerotic disease with stroke or death within 30 days of the procedure.	No	Intracranial angioplasty and/or stenting	...	...
Metric 12: Percentage of SAH, ICH, and AVM patients for whom initial severity measures are documented.	Yes	...	Hunt and Hess scale if SAH	ICH score if ICH (whether or not AVM); Spetzler-Martin for all AVM
Metric 13: Median time from admission to start of procedure intended to obliterate a ruptured aneurysm by surgical clipping or endovascular coiling for patients who arrive within 48 hours of the hemorrhage that led directly to admission.	Yes	...	SAH	...
Metric 14: Percentage of patients with aneurysmal SAH arriving within 48 hours of hemorrhage for whom a coiling or clipping procedure was not started within 36 hours of arrival who have a documented reason for not having undergone coiling or clipping within 36 hours of arrival.	No	...	SAH	...
Metric 15: Percentage of patients with documented aneurysmal SAH for whom nimodipine treatment (60 mg every 4 hours or 30 mg every 2 hours) is started within 24 hours of diagnosis and for whom such treatment is continued until 21 days after the hemorrhage or until discharge if they are discharged <21 days after the SAH.	Yes	...	SAH	...
Metric 16: Percentage of SAH patients with diminished level of consciousness and ventriculomegaly who are treated with EVD.	No	...	SAH	...
Metric 17: Median frequency of noninvasive monitoring performed for surveillance for vasospasm in patients with aneurysmal SAH during the period between 3 and 14 days after SAH.	No	...	...	...

(Continued)

Table 3. Continued

Metric	Core Metric	Ischemic Stroke, TIA, or Asymptomatic Cerebrovascular Stenosis	SAH and Nonruptured Aneurysm	ICH and AVM (With or Without Hemorrhage)
Metric 18: Complication rates for aneurysm coiling and clipping.	No	...	All	...
Metric 19: Median time from arrival to start of treatment to reverse the INR with a procoagulant preparation (eg, fresh frozen plasma, recombinant factor VIIa, prothrombin complex concentrates) for patients with warfarin-associated ICH and an elevated INR (INR >1.4).	Yes	...	...	ICH (if warfarin-associated)
Metric 20: Percentage of patients undergoing surgical or endovascular treatment of an AVM with stroke or death within 30 days of the procedure.	No	...	...	AVM
Metric 21: Percentage of patients with ischemic or hemorrhagic stroke or TIA transferred from another hospital to the CSC with documentation of the time from the first call from the transferring hospital to the CSC (to a member of a stroke program or to a centralized transfer center) to arrival time at the CSC.	No	All patients transferred from another hospital	All patients transferred from another hospital	All patients transferred from another hospital
Metric 22: Percentage of patients admitted to each type of unit to which patients with ischemic or hemorrhagic stroke or TIA are initially admitted (eg, neurological/neurosurgical ICU, medical ICU, surgical ICU, general ICU, coronary care unit, burn ICU, stroke unit, other intermediate-level-of-care unit, neurology floor, or other floor). A separate percentage should be calculated for each type of unit.	No	All	All	All
Metric 23: Percentage of patients with stroke or death within 24 hours of diagnostic neuroangiography.	Yes	If diagnostic angiogram is performed	If diagnostic angiogram is performed	If diagnostic angiogram is performed
Metric 24: Percentage of patients who have a diagnosis of ischemic or hemorrhagic stroke who undergo EVD and then develop ventriculitis.	No	If patient has EVD	If patient has EVD	If patient has EVD
Metric 25: Median number of days from admission to completion of evaluations for physical therapy, occupational therapy, speech-language pathology, and rehabilitation medicine, unless there is documentation on admission that some or all of these evaluations are not needed or that the patient cannot tolerate them because of medical instability.	No	All	All	All
Metric 26: Percentage of patients admitted with diagnoses of ischemic stroke, SAH, AVM, intracranial hemorrhage, extracranial cervical stenosis, intracranial stenosis, or TIA who are enrolled in a clinical research study.	No	All	All	All

TIA indicates transient ischemic attack; SAH, subarachnoid hemorrhage; ICH, intracerebral hemorrhage; AVM, arteriovenous malformation; NIHSS, National Institutes of Health Stroke Scale; CT, computed tomography; MR, magnetic resonance; MRI/MRA, magnetic resonance imaging/angiography; CT/CTA, computed tomography/computed tomography angiography; mRS, modified Rankin Scale; CEA, carotid endarterectomy; EVD, external ventricular drainage; INR, international normalized ratio; CSC, comprehensive stroke center; and ICU, intensive care unit.

For patients with stroke in the hospital, the time of arrival should be taken to be the time that the deficit was first discovered.

Patients who are transferred to the CSC after intravenous thrombolysis is started at another hospital should be excluded from this metric for the CSC.

A reason should be documented if intravenous thrombolysis is not administered. The acceptable reasons for not treating patients with intravenous thrombolysis within 3 hours are listed in the AHA/ASA "Guidelines for the Early Management of Adults With Ischemic Stroke."<sup>18</sup> The acceptable reasons for not treating patients with intravenous thrombolysis within 4.5 hours are the same, with several additional reasons.<sup>32</sup> In addition, in view of the accepted target of a 60-minute door-to-needle time,<sup>33–37</sup> patients are excluded from this metric if they arrive <1 hour before the end of the appropriate time window. Finally, we also think that it is acceptable for centers to consider that patients who meet the published criteria for treatment in the 3- to 4.5-hour window are not eligible for intravenous thrombolysis for the purposes of this metric if there is documentation in the

patient's chart that the risks outweigh the potential benefit or that the patient is to be enrolled in a clinical trial. The reasons for allowing patients who could receive intravenous thrombolysis in the 3- to 4.5-hour window to be dealt with in this way are that intravenous tPA is currently approved by the US Food and Drug Administration only for patients treated within 3 hours, and treatment in the 3- to 4.5-hour window is only a **Class I, Level of Evidence B** recommendation,<sup>32</sup> whereas treatment in the less than 3-hour period is a **Class I, Level of Evidence A** recommendation.<sup>18</sup>

In addition to computing this metric for all eligible patients seen within 3.5 hours, centers should also compute it separately for patients seen within 2 hours and those seen between 2 and 3.5 hours of the time when they were last known to be at baseline.

#### Justification

Intravenous thrombolysis with tPA is still the only intervention that has been approved by the US Food and Drug Administration as a treatment to improve the clinical outcome of acute ischemic stroke patients, and it is a cornerstone of acute stroke treat-

ment.<sup>2,20</sup> Meta-analysis of trials of intravenous thrombolysis has suggested that earlier treatment with intravenous tPA leads to better outcomes,<sup>27,38</sup> so the faster that intravenous tPA is given, the better. Initial analysis of the National Institute of Neurological Disorders and Stroke tPA trial also supported this conclusion,<sup>39</sup> but several subsequent reanalyses of the trial itself have not found a statistically significant effect for earlier treatment.<sup>40–42</sup> Despite the uncertainty raised by these reanalyses, we conclude that the time from arrival to start of treatment should be kept to a minimum. A goal of  $\leq 60$  minutes for the period from time to arrival to initiation of treatment has been proposed to allow adequate time for appropriate clinical and imaging evaluation<sup>33–35</sup> and appears to be feasible.<sup>36,37</sup>

Because of ECASS (European Cooperative Acute Stroke Study) III<sup>28</sup> and the subsequent AHA science advisory that expanded the window for treatment from 3 to 4.5 hours,<sup>32</sup> we propose the inclusion of patients seen at up to 3.5 hours from the time that they were last known to be at baseline.

### **Metric 3**

**Percentage of patients who are treated for acute ischemic stroke with intravenous thrombolysis whose treatment is started  $\leq 60$  minutes after arrival. (Core metric)**

**Numerator:** Patients treated with intravenous thrombolysis for acute ischemic stroke whose treatment is started within 60 minutes of arrival.

**Denominator:** All patients treated with intravenous thrombolysis for acute ischemic stroke.

For patients with stroke in the hospital, the time of arrival should be taken to be the time that the deficit was first discovered.

Patients who are transferred to the CSC after intravenous thrombolysis is started at another hospital should be excluded from this metric for the CSC.

### *Justification*

As noted above in the justification of Metric 2, more rapid initiation of intravenous thrombolysis appears to be beneficial, so we propose that CSCs also track door-to-needle time to aid in quality improvement efforts to reduce this time as much as possible and that they review all intravenous thrombolysis cases to look for ways to reduce this time further. We anticipate that CSCs will record the door-to-needle time itself for Metric 2 (and not just whether or not it was  $< 60$  minutes) to aid in quality improvement efforts.

Although many PSCs track arrival to intravenous thrombolysis times with databases such as Get With The Guidelines,<sup>43</sup> and some states require stroke centers to report door-to-needle time for patients treated with intravenous tPA,<sup>12</sup> door-to-needle time itself is not currently one of the harmonized PSC performance measures established by The Joint Commission, the Centers for Disease Control and Prevention, and the AHA.<sup>44,45</sup> The current harmonized PSC performance measure looks at the percentage of patients arriving within 2 hours and treated within 3 hours. CSCs should be especially vigilant in monitoring the efficiency of delivering care for acute stroke patients, so we recommend that this be a core metric.

The goal of starting intravenous thrombolysis within 60 minutes of arrival has become an accepted target for stroke centers, as noted in the discussion of Metric 2,<sup>12,33–37</sup> although it

remains difficult to achieve in all cases. In addition, the AHA has recently launched a national campaign focused on reducing door-to-needle times to  $< 60$  minutes in 50% of all treated patients.<sup>47</sup>

### *Additional Data Elements*

In view of the ECASS III study,<sup>32</sup> which found a benefit to the use of intravenous tPA for patients treated within 3 to 4.5 hours of stroke onset, we would encourage CSCs to analyze data about such patients separately from data for patients treated within 3 hours of onset.

Because of concern that performance of multimodal computed tomography (CT) and magnetic resonance imaging (MRI) could delay treatment with intravenous tPA, centers should analyze the data collected under this metric and Metrics 2 and 4 to determine whether performance of multimodal imaging before intravenous tPA administration prolongs the door-to-needle time and whether the information obtained was needed to make a decision about the use of intravenous tPA (as might be the case, for example, if there was uncertainty about the diagnosis of stroke in a patient with seizure at onset of symptoms or hypoglycemia at onset).

### *Metric 4*

**Median time from arrival to start of multimodal CT or magnetic resonance (MR) brain and vascular imaging (MRI/MR angiography or CT/CT angiography [CTA]) for ischemic stroke patients arriving within 6 hours of the time that they were last known to be at baseline, if 1 of these studies is ordered. (Core metric)**

Multimodal CT may include noncontrast CT, CT perfusion, and CTA studies.<sup>18</sup> Multimodal MRI may include diffusion-weighted imaging, perfusion-weighted imaging, MR angiography, gradient echo, and fluid-attenuated inversion recovery or T2-weighted sequences.<sup>18</sup>

The start time for multimodal imaging should be the time recorded on the first vascular or perfusion imaging sequence. If multimodal imaging was performed as part of the same study that included routine anatomic imaging and a separate time cannot be identified for the first vascular or perfusion sequence, then the start time for the entire study should be used.

Patients should be excluded from this metric if there is a documented reason for not performing multimodal imaging quickly (eg, a patient with a mild clinical deficit judged too small to warrant endovascular intervention, a patient enrolled in a clinical trial that precludes emergency vascular imaging, a patient deemed medically too unstable, a patient deemed to have a deficit that is too large or too long established for these procedures, or a patient who proceeds directly to angiography without having any of these studies performed). This metric does not include patients who are only undergoing simple noncontrast CT scans to assess eligibility for intravenous tPA without performance of multimodal imaging (which may include CTA or perfusion studies). Noncontrast CT, when appropriate, should be completed within 25 minutes, as expected for PSCs,<sup>2</sup> whether or not other imaging is also performed emergently.

We also note that these brain and vascular imaging studies may be delayed in some centers in some patients who receive intravenous tPA if the intravenous tPA is given before the studies are performed, so it will be important for centers to

monitor their performance on this measure separately for patients who receive intravenous tPA before the studies are performed.

For patients with stroke in the hospital, the time of arrival should be the time that the deficit was first discovered.

The intent of this metric is to monitor the time that it takes to perform these studies when needed emergently for the care of acute ischemic stroke patients and not to imply that they should be performed on all such patients.

For purposes of this metric, if vascular imaging is performed immediately after noncontrast imaging without removing the patient from the scanner, the time the multimodal procedure was started should be used as the time the noncontrast brain study was begun.

#### *Justification*

The AHA/ASA "Guidelines for the Early Management of Adults With Ischemic Stroke" state that multimodal CT and MRI "may provide additional information that will improve the diagnosis of ischemic stroke" (*Class I; Level of Evidence A*).<sup>18</sup> This additional information includes location and severity of ischemia and location of vascular occlusions and stenoses. Potentially, the additional information may lead to refinement of care for some patients with acute stroke. The new AHA Recommendations for "Imaging of Acute Ischemic Stroke" note that vascular imaging may help in deciding between intravenous and intra-arterial thrombolytic therapies (*Level of Evidence B*) but should not delay intravenous thrombolysis. Vascular imaging should be performed if intra-arterial therapy is being considered in patients >3 hours after onset of stroke (*Level of Evidence A*).<sup>48</sup> Because of the strengths of these recommendations and the need for rapid evaluation of acute ischemic stroke patients, we propose that the time to multimodal imaging when it is ordered for acute stroke patients should be a core metric for CSCs.

The BAC CSC report emphasized the importance of rapid performance of brain and vascular imaging studies in acute ischemic stroke patients. The report proposed a 2-hour standard for time from ordering MR studies to their completion, but we have chosen the time from arrival to start of imaging as the primary measure because of concerns that the time of ordering often cannot be determined reliably and concerns that the time the studies were begun can be obtained more consistently from currently available scanners than the time of completion. In addition, we believe that a 2-hour delay to imaging is unacceptably long for many acute stroke patients, so rather than proposing a specific benchmark, we propose that centers should document the time that it takes to complete imaging and use these data as part of a continuous quality improvement effort to improve and, when appropriate, speed up care of acute stroke patients. We have expanded this metric to include multimodal CT because we recognize that in many centers, CTA and sometimes CT perfusion studies are performed in preference to MR, because CT can be performed more quickly and on more patients. Despite concerns about contrast-induced renal failure with CTA and CT perfusion, the incidence of this complication is extremely low.<sup>49</sup> We recognize that in many centers, MR or CT perfusion studies are performed, but we do not require them for studies performed under this metric, because the evidence

supporting their use is weaker than that for the other studies that are included in this measure.<sup>3</sup>

The additional information obtained by multimodal imaging with regard to prognosis, severity, cause of stroke, and possible therapeutic options justifies its use for evaluation of acute stroke patients (typically those within a 6-hour window) in a CSC. Although it is not yet certain how this information may be applied, many reports have proposed using these types of data to choose patients for endovascular reperfusion procedures.<sup>50–53</sup> If these studies are performed for this purpose, they should be performed quickly. Although multimodal CT currently can be performed more quickly than MR, some investigators report that multimodal MR can also be performed in some circumstances without preventing rapid administration of intravenous tPA.<sup>54</sup> However, multimodal brain and vascular imaging studies should not delay emergency treatment of stroke (ie, treatment with intravenous tPA), as noted in a *Class I; Level of Evidence C* recommendation in the AHA/ASA "Guidelines for the Early Management of Adults With Ischemic Stroke."<sup>18</sup>

#### *Additional Data Elements*

CSCs should consider tracking the time required for ordering MRI/MR angiography or CTA, times of completion, and time to initial interpretation of the studies as part of their quality improvement efforts.

The BAC PSC and CSC reports both emphasize the importance of having a stroke team with specialized expertise assist in the rapid evaluation and ongoing care of stroke patients. Clinical experience suggests that early involvement of the stroke team is important in starting treatment rapidly, so we also suggest that CSCs should consider carefully monitoring the time from arrival of the patient (or from recognition of a possible acute stroke for strokes in inpatients) to the first call to the stroke team and to arrival of the initial responding member of the team at the patient's bedside.

#### *Metric 5*

**Percentage of ischemic stroke patients seen within 6 hours of the time they were last known to be at baseline who have documentation that an endovascular recanalization procedure either was performed or was considered and deemed not to be appropriate or possible. A reason should be documented if an endovascular procedure was not performed.**

**Numerator:** Number of ischemic stroke patients seen within 6 hours of the time when they were last known to be at baseline who undergo an endovascular revascularization procedure or are documented not to be a candidate for such a procedure.

**Denominator:** Number of ischemic stroke patients seen within 6 hours of the time when they were last known to be at baseline.

Allowable reasons for not performing an endovascular procedure but still including a patient in the numerator may include the following: Enrollment in a clinical trial, arrival time that is too late for treatment, deficits that are too severe or too mild, elevated creatinine, comorbidities, advanced age, lack of major vessel occlusion, rapid improvement, large volume of advanced ischemic injury, refusal of a procedure by the patient or the



patient's family, lack of an appropriate proxy for a patient unable to consent to the procedure, and insufficient evidence to support intervention in the judgment of the treating physicians.

Unavailability of endovascular services is also an allowable reason for including a patient in the numerator for this metric, because even though coverage by endovascular interventionalists 24 hours a day, 7 days a week is expected to be an integral part of CSCs,<sup>3</sup> we recognize that there are some occasions when endovascular intervention is legitimately not available. Reasons for such unavailability include simultaneous emergencies, equipment failure, and unavailability of key staff, for example, for medical- or weather-related reasons. These reasons and their frequency should be tracked carefully and reported separately. Moreover, CSCs must have contingency plans to minimize the chance that services are unavailable. Quality improvement efforts should be directed toward decreasing such unavailability over time. For example, CSCs should develop plans to transfer patients to another center when time permits if there are short periods during which endovascular procedures are not available (or to divert them to another center initially). CSCs should not accept transfers of patients who may be candidates for such procedures during these periods unless there are other overriding reasons for transfer, such as a need for intensive care not available at another center that is within an appropriate distance. Quality improvement efforts should be directed toward decreasing such periods when services are unavailable.

In addition, we want to emphasize that the intent of this metric is to ensure that CSCs are actively considering the endovascular procedures that they are capable of performing for acute ischemic stroke patients, not to encourage indiscriminate use of such procedures, whose benefit for stroke treatment has not yet been established. We also want to emphasize that CSCs should consider active participation in clinical trials for patients who are seen within 8 hours of stroke onset and are monitored under this metric because of the many unanswered questions about the optimum treatment for such patients and the large potential benefits if the damage from their strokes can be limited.

#### *Justification*

The AHA/ASA "Guidelines for the Early Management of Adults With Ischemic Stroke" state that endovascular thrombolysis (**Class I; Level of Evidence B**) and mechanical clot retrieval with Merci or Penumbra devices (**Class IIb; Level of Evidence B**) are options for treatment of some ischemic stroke patients,<sup>18</sup> and endovascular procedures should be available at a CSC.<sup>3</sup> Both the American College of Chest Physicians Evidence-Based Clinical Practice Guidelines and the AHA *ACLS Provider Manual* have also stated that endovascular thrombolysis has a role in the treatment of some acute ischemic stroke patients.<sup>33,35</sup> In this setting, we recommend that CSCs should consider endovascular interventions for acute stroke, but because of limited data about the actual efficacy of endovascular procedures,<sup>55–57</sup> we do not think that a metric that specifically prescribes endovascular interventions for any group of patients is appropriate yet, nor do we think that there is enough evidence to limit this metric to patients with strokes of any specific severity. In addition, because of these issues, we have not classified this metric as a core metric. Finally, because mechanical clot-

retrieval devices have been approved for use up to 8 hours from the time of stroke onset, we propose the inclusion of patients seen within 6 hours, to allow 2 hours for noninvasive evaluation, diagnostic angiography, and endovascular treatment.

#### *Additional Data Element*

CSCs should consider tracking whether patients who present with posterior circulation strokes up to 24 hours from last being known to be at baseline are considered for endovascular treatment and whether a reason for not treating them is documented if they do not undergo a procedure. The window for treating such patients may be considerably longer than 6 hours.<sup>18,58</sup>

#### **Metric 6**

#### **Median time from arrival to start of treatment for acute ischemic stroke patients undergoing an endovascular intervention.**

The start of treatment is defined here as the start of intra-arterial infusion of a thrombolytic drug or the first pass with a device such as a Merci or Penumbra catheter.<sup>26,55</sup> If the time that treatment was started cannot be determined accurately, centers may use the time halfway between groin puncture and completion of the procedure. For patients who have a stroke in the hospital, the time of arrival should be taken to be the time that the deficit was first discovered.

#### *Justification*

Although a consensus does not exist about the efficacy of endovascular interventions for acute ischemic stroke, it is clear that the sooner reperfusion is achieved, the more likely it is to be beneficial.<sup>59,60</sup> We have chosen this metric as the best way to measure the overall speed of the process. It is closer to the physiologically relevant time of reperfusion than the time of puncture. Time of reperfusion, however, may be difficult to determine, because partial recanalization is often achieved before maximal recanalization, and different branches may be opened at different times.

If and when additional evidence becomes available to support the efficacy of endovascular treatment for ischemic stroke, a metric related to the speed of performing endovascular procedures should be considered for inclusion in the set of core metrics. For now, however, we have classified this measure as a supplemental metric. In addition, as experience is gained with this metric, establishment of a benchmark time from arrival to start of treatment should be considered. In view of current cardiology guidelines for door-to-angioplasty time for acute myocardial infarction, for which the goal is 90 minutes,<sup>61</sup> we encourage CSCs to at least aim for a goal of 2 hours, given that stroke patients need to undergo imaging before any endovascular procedure.

#### *Additional Data Elements*

To assist in quality improvement, centers should also consider collecting information about time of arrival in the angiography laboratory, time of groin puncture, time of diagnostic angiographic injection in the vessel supplying the region of the stroke, and time of initial reperfusion and final attempts at reperfusion. The effects of intravenous thrombolysis and of the performance of CTA/CT perfusion or MRI/MR angiography on times of angiography and on outcome should be analyzed as appropriate for quality improvement efforts. Centers should also consider

monitoring the time of arrival in laboratory, the time of puncture, and the time of diagnostic injection for acute stroke patients who do not undergo an intervention.

Centers should also consider monitoring the extent of initial perfusion and of reperfusion at the end of the procedure, as well as degree of reperfusion, using a validated metric. Metrics could include the Thrombolysis in Cerebral Ischemia scale, the Thrombolysis in Myocardial Infarction scale adapted for the cerebral circulation, the Arterial Occlusive Lesion scale, and/or the Qureshi Site of Occlusion Scale.<sup>29,30,62–65</sup>

### **Metric 7**

#### **Percentage of patients treated with intravenous thrombolysis who have a symptomatic intracranial hemorrhage within 36 hours of treatment. (Core metric)**

**Numerator:** Patients treated with intravenous thrombolysis who have a symptomatic intracranial hemorrhage in the first 36 hours after treatment.

For the purposes of this metric, symptomatic intracranial hemorrhage is defined by the presence of a new intracranial hemorrhage on a CT or MRI that is performed within 36 hours of the end of treatment, with documentation in the medical record that there has been a clinical deterioration, in the absence of documentation that an alternate mechanism caused the deterioration. Acceptable alternate mechanisms may include infection, new stroke or increased swelling of ischemic tissue, seizures, and metabolic and toxic encephalopathy.

Any hemorrhage seen on CT and not identified on a pretreatment scan should be considered new. For patients who have a posttreatment MRI that shows a hemorrhage and only pretreatment CT imaging that did not show the hemorrhage, the hemorrhage should be considered old if its imaging characteristics suggest that it occurred before treatment. If clinically appropriate, a posttreatment CT in addition to the MRI may help determine the age and significance of the hemorrhage. Evidence from older scans performed before the acute presentation, a history of intracranial hemorrhage in the appropriate location, or other relevant clinical information may be used to help determine whether a hemorrhage seen on MRI is new or old. We also note that in general, hemorrhagic transformation with parenchymal hematoma, rather than petechial hemorrhage,<sup>28,66</sup> is expected when brain hemorrhage produces neurological deterioration.

Neurological deterioration is defined as any increase in the patient's NIHSS score, if the score was documented. If it was not documented, neurological deterioration is defined as any documentation of neurological worsening. In addition, even if the NIHSS does not increase, a patient should be included in this metric if the clinicians caring for the patient document that there is worsening that they believe was caused by the hemorrhage. Isolated headache, however, should not be considered evidence of neurological worsening. To determine whether a hemorrhage occurred within 36 hours of treatment, the time to be calculated is the interval from the conclusion of treatment to the time of the last assessment before the one in which the clinical deterioration associated with the hemorrhage was first noted.

**Denominator:** All patients treated with intravenous thrombolysis. If a center uses a "bridging" protocol and treats

some patients with intravenous thrombolysis followed by an endovascular procedure, these patients should be included in the primary metric calculated here, but centers should also track symptomatic intracranial hemorrhage rates separately for patients treated only with intravenous thrombolysis and those treated with a bridging protocol and should consider whether particular types of endovascular therapy are associated with higher rates of intracranial hemorrhage.

### *Justification*

The risk of intracranial hemorrhage is one of the most serious complications associated with the use of intravenous tPA. In the National Institute of Neurological Disorders and Stroke trial,<sup>20</sup> there was a 6.4% risk of symptomatic hemorrhage among those treated with intravenous tPA compared with a 0.6% risk for control patients ( $P < 0.001$ ). As stated in the AHA/ASA "Guidelines for the Early Management of Adults With Ischemic Stroke," "It is now clear that the risk of hemorrhage is proportional to the degree to which the NINDS protocol is not followed."<sup>18,67–69</sup> Given this background, we recommend that the rate of hemorrhage be tracked as a metric by CSCs and that quality improvement efforts be directed to analyze any protocol deviations in patients who have a symptomatic intracranial hemorrhage after intravenous thrombolysis and to identify other factors that may have contributed to intracranial hemorrhage.<sup>70,71</sup> Given the potentially devastating consequences of intracranial hemorrhage after thrombolysis, this should be a core metric, because it is essential for the assessment of CSC performance.

We recognize that the use of a 36-hour window for inclusion of hemorrhages is somewhat arbitrary, but we have chosen this in keeping with the ECASS III trial, in which routine follow-up scans were performed up to 36 hours after treatment.<sup>28</sup> We have defined the window in terms of the time of clinical deterioration rather than the time of scanning, however, so that hemorrhages are included even if the scan is delayed because a patient is too unstable to be scanned.

We have not specifically excluded petechial hemorrhages from the numerator because we are concerned that hemorrhages may not be classified reliably in clinical practice. However, minor hemorrhages that are not associated with any worsening will not be included in the numerator whether they are only petechial or not.

We recognize that the hemorrhage rates from this metric may differ from those obtained in previous clinical trials, but we think that the definition we are proposing is a more feasible one to implement in clinical practice, and it will be possible to develop expected norms for this metric by comparing data from different centers.

### *Additional Data Elements*

CSCs should also consider tracking asymptomatic hemorrhages and delayed symptomatic hemorrhages, the size and location of any postthrombolysis hemorrhages, and the type of hemorrhage (eg, hemorrhagic infarction or parenchymal hematoma as defined in the ECASS trials).<sup>28,66</sup> We also encourage CSCs to record the NIHSS score whenever a deterioration occurs after thrombolysis and to consider the effects of the time from onset to treatment on the rate of hemorrhage.

**Metric 8****Percentage of acute ischemic stroke patients treated with endovascular interventions who develop symptomatic intracranial hemorrhage within 36 hours of treatment. (Core metric)**

**Numerator:** Patients who undergo endovascular intervention for acute ischemic stroke and have a symptomatic intracranial hemorrhage in the first 36 hours after treatment. Symptomatic intracranial hemorrhage should be defined as in Metric 7.

**Denominator:** All patients who undergo endovascular intervention for acute ischemic stroke. If a center uses a “bridging” protocol and treats some patients with intravenous thrombolysis followed by an endovascular procedure, these patients should be included in the primary metric calculated here, but as noted under Metric 7, centers should also track symptomatic intracranial hemorrhage rates separately for patients treated only with endovascular thrombolysis and those treated with a bridging protocol.

*Justification*

The risk of ICH is the most serious problem associated with endovascular procedures for treatment of acute ischemic stroke, just as it is with intravenous tPA. Especially in view of the lack of proof for the efficacy of endovascular procedures, it is important for CSCs to monitor patients who undergo these procedures carefully for the development of hemorrhage. CSCs should track the development of postprocedural hemorrhages and subject these to rigorous quality improvement analysis to identify potential contributing factors.<sup>70,71</sup> Given the potentially devastating consequences of intracranial hemorrhage after endovascular interventions, this should be a core metric, because it is essential for assessment of CSC performance.

*Additional Data Elements*

CSCs should also consider tracking the same additional data elements suggested for treatment with intravenous thrombolysis under Metric 7.

**Metric 9****Percentage of acute ischemic stroke patients who are treated with intravenous thrombolysis or who undergo endovascular interventions for whom there is documentation of a 90-day Modified Rankin Scale (mRS) score. (Core metric)**

**Numerator:** All patients with ischemic stroke acutely treated with intravenous thrombolysis or with an endovascular recanalization procedure who had an mRS performed at approximately 90 days after the stroke, either in person or by telephone if it was not possible to perform in person. The mRS should be conducted by an appropriately trained individual using a standardized interview. The mRS may be based on information obtained from the patient or from an appropriate family member or caregiver. The mRS should be performed within 2 weeks of the date at which it has been 90 days since stroke onset; it may be performed over the telephone if necessary.

**Denominator:** All patients admitted with ischemic stroke acutely treated with intravenous thrombolysis or with an endovascular recanalization procedure.

*Justification*

The mRS has been shown to be a reliable and reproducible measure of stroke outcome. Because the mRS considers impairments, functional ability, and role outcomes, it serves as a global functional health index with strong emphasis on physical disability.<sup>72</sup> Its popularity derives both from its face validity, its relative efficiency, and its generally well-accepted dichotomization in categorizing dependent versus independent stroke survivors (mRS score 0 to 2 versus 3 to 6). The mRS at 3 months after stroke has become the accepted standard for assessing recovery from ischemic stroke and has been used in numerous recent large randomized clinical trials.<sup>20–24,73</sup>

Because the mRS categories can be assessed somewhat subjectively, as shown by multiple studies demonstrating lower than desirable interrater reliability, the use of a structured interview by a trained evaluator to assign mRS scores should be standard; this leads to good reproducibility of results.<sup>74–76</sup> The use of a standardized interview to assess the mRS score at 3 months should therefore be a key outcome measure in acute ischemic stroke patients treated with intravenous thrombolysis or acute endovascular recanalization at CSCs. This interview could be conducted over the telephone, if necessary (a **Class I; Level of Evidence B** recommendation of the AHA review of telemedicine for stroke<sup>77</sup>). A family member or caregiver may be interviewed if appropriate.

We recognize that acquiring data after hospital discharge may be difficult, but we believe that such data are essential if CSCs are to monitor the outcomes of their care of acute stroke patients in a meaningful way. Such monitoring is necessary for quality assessment and improvement, which are especially important for patients treated with acute interventions because of the risks associated with these interventions. It is important to track whether success rates with intravenous thrombolysis in clinical practice at each CSC match those reached in clinical trials,<sup>18,67–69,78–82</sup> and close monitoring of the outcomes of endovascular procedures is also crucial, especially in view of the lack of definitive evidence for the efficacy of these treatments. The importance of these issues to care of acute ischemic stroke patients justifies the classification of this metric as a core metric for CSCs.

In addition, collection of follow-up information provides a mechanism for contact between the CSC and patients that may facilitate long-term follow-up care and compliance with therapy for secondary prevention of stroke. In this regard, collection of follow-up data at 30 days after other procedures, as noted below, may similarly facilitate long-term care.

Because outcome at 90 days will depend heavily on initial stroke severity and may also depend on the quality of care received after discharge from the CSC, and because collection of the score at 90 days may be difficult in some cases, we have proposed that the primary metric involving the mRS simply be whether it is collected. We expect, however, that CSCs will track the mRS data and use these data for quality improvement, as noted above.

*Additional Data Elements*

If the mRS is not obtained, CSCs should record whether attempts were made to obtain it in accordance with an institutionally established protocol. When collecting informa-

tion about the 90-day mRS, CSCs should also consider whether to collect information about compliance with medications prescribed at discharge, such as antithrombotic drugs, statins, and antihypertensive drugs. Outpatient compliance with therapy for secondary prevention is an essential element of care both at CSCs and at PSCs; we raise this issue here because there is as yet no requirement for monitoring outpatient compliance at PSCs, and because part of the overall mission of CSCs is to provide comprehensive high-quality care, they should also consider addressing outpatient compliance. In this regard, the BAC CSC report notes that an outpatient stroke clinic is an optional component of a CSC.

Although we have recommended collecting the mRS only on ischemic stroke patients undergoing intravenous thrombolysis or endovascular procedures, CSCs should also consider collecting the score on all ischemic and hemorrhagic stroke patients. In this regard, the importance of 90-day follow-up data was previously recognized in the proposed design of the Coverdell Stroke Registry.<sup>83</sup> If collecting the 90-day data on the limited set of stroke patients treated acutely proves feasible and useful, we would favor expanding this metric to include all stroke patients in the future.

We also recognize that recovery may continue long beyond 3 months for ischemic stroke patients and may be slower for patients with hemorrhagic stroke (especially SAH). In this regard, the primary end point of the International Subarachnoid Aneurysm Trial (ISAT) was the mRS at 1 year.<sup>84</sup> Given the difficulties inherent in obtaining lengthy follow-up data, however, we suggest a 90-day follow-up assessment as a reasonable outcome. CSCs should consider collecting data on 1-year follow-up as an additional data element, especially for SAH patients.

CSCs should also consider collecting other functional measures of patient status, such as the Barthel Index. In addition, they should consider collecting the NIHSS at selected times after admission, such as 24 hours after acute interventions and at discharge. Changes in the NIHSS score appear to be a useful stroke outcome measure.<sup>85</sup>

### **Metric 10**

#### **Percentage of patients undergoing carotid endarterectomy (CEA), or carotid angioplasty or stenting, with stroke or death within 30 days of the procedure.**

**Numerator:** Number of patients who have a stroke or die within 30 days of CEA, or who have carotid angioplasty or stenting performed because of atherosclerotic disease.

**Denominator:** Total number of patients who undergo CEA or who undergo carotid angioplasty or stenting because of atherosclerotic disease.

The metric should be calculated for all procedures taken together and separately for the following groups of patients: (1) Symptomatic patients undergoing endarterectomy; (2) symptomatic patients undergoing carotid angioplasty or stenting; (3) asymptomatic patients undergoing endarterectomy; and (4) asymptomatic patients undergoing carotid angioplasty or stenting. Patients should be excluded from this metric if it is not possible to obtain information about their 30-day stroke and mortality status after reasonable efforts in accordance with a locally established protocol have been made. The number of

such failures to obtain follow-up information should be tracked by CSCs and must be documented formally, along with the complication rates whenever these are reported. This proviso will also apply to subsequent metrics about outcomes for other procedures.

Strokes should be included if they meet the clinical definition of a focal neurological deficit that persists for  $\geq 24$  hours without other cause or if there is a focal deficit that lasts for a shorter period of time but is associated with an appropriately located acute ischemic lesion on MRI. Clinically silent acute lesions detected on diffusion-weighted MRI should not be included as complications, because they are likely to be common when MRI is performed, although their incidence and clinical significance are uncertain. Patients with confusion or encephalopathy who have multiple punctate lesions that together may explain their clinical findings should also be included as having had a stroke. Published clinical trials about complications after carotid procedures and other interventions have typically used clinical stroke as the end point,<sup>86,87</sup> and other ongoing trials also are using clinical end points.<sup>88,89</sup> This definition of stroke will apply to this metric and subsequent ones.

This metric is limited to patients with atherosclerotic disease to ensure that the metric encompasses a uniform population of patients.

#### *Justification*

The AHA/ASA guidelines for patients with recent TIA or ischemic stroke within the past 6 months and ipsilateral severe (70% to 99%) carotid artery stenosis recommend endarterectomy by a surgeon with a perioperative morbidity and mortality rate of  $<6\%$  (**Class I; Level of Evidence A**).<sup>90</sup> For patients with recent TIA or ischemic stroke and ipsilateral moderate (50% to 69%) carotid stenosis, CEA is recommended, depending on patient-specific factors such as age, sex, comorbidities, and severity of initial symptoms if the perioperative morbidity and mortality risk is estimated to be  $<6\%$  (**Class I; Level of Evidence B**).<sup>90</sup>

Among patients with symptomatic severe stenosis ( $>70\%$ ) in whom either the stenosis is difficult to access surgically, medical conditions are present that greatly increase the risk for surgery, or other specific circumstances exist such as radiation-induced stenosis or restenosis after CEA, the use of carotid angioplasty and stent placement is not inferior to endarterectomy and may be considered (**Class IIb; Level of Evidence B**).<sup>90</sup> The procedure is reasonable when performed by operators with established periprocedural morbidity and mortality rates of 4% to 6% (**Class IIa; Level of Evidence B**).<sup>90</sup>

The AHA/ASA "Guidelines for the Primary Prevention of Stroke" state that prophylactic CEA can be useful in highly selected patients with high-grade asymptomatic carotid stenosis if performed by surgeons with morbidity/mortality rates  $<3\%$  (**Class IIa; Level of Evidence A**).<sup>91</sup> The threshold of 3% is defined on the basis of the results of the Asymptomatic Carotid Artery Surgery (ACAS) trial,<sup>87,92</sup> which ascertained the rate of any perioperative stroke or death within 30 days. The combined arteriographic and perioperative surgery-related mortality and stroke rates achieved by the carefully

selected surgical teams was low (2.3%). A low rate of any stroke or death is considered necessary to confer benefit of the procedure in patients with asymptomatic carotid artery stenosis.<sup>93</sup>

The role of carotid angioplasty/stenting in asymptomatic patients has not been established. The AHA/ASA “Guidelines for the Primary Prevention of Stroke” state, “The usefulness of CAS [carotid angioplasty/stenting] as an alternative to CEA in asymptomatic patients at high risk for the surgical procedure is uncertain (*Class IIb; Level of Evidence C*).”<sup>91</sup> In this setting, if centers choose to perform carotid angioplasty/stenting on asymptomatic patients, the 30-day rate of stroke and death should be tracked separately for such patients and monitored carefully.

The recommended end point to be ascertained after carotid angioplasty and stent placement is any stroke or death within 30 days, to remain consistent with the data collected for CEA. This end point has been used in trials of carotid angioplasty and stenting. For comparable patients, the complication rate for stenting should be similar to that for endarterectomy if stenting is to be a reasonable option. In particular, the complication rate should be expected to be between 4% and 6% for symptomatic >70% stenoses.<sup>90</sup> If carotid angioplasty and stenting are performed, therefore, careful attention must be paid to complication rates, so it is important for CSCs to monitor these rates.

We have not included myocardial infarction among the complications to be tracked for this metric because data from the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) indicated that quality of life was worse for patients whose end point was stroke than for those whose end point was myocardial infarction.<sup>94</sup>

We have not proposed this metric as a core metric because of concerns about how appropriate it is to apply the known thresholds from clinical trials to clinical practice and concerns about how to adjust for baseline risks, but after some initial experience with its use, we anticipate that it may become a required metric.

#### *Additional Data Elements*

In addition to calculating the metric, centers should consider tracking the rates of stroke and death separately and recording the location and other characteristics of any stroke that occurs after endovascular or surgical carotid procedures, as well as the cause of death in patients who died. Centers should also consider monitoring the degree of residual stenosis on an angiogram at the end of the procedure for stenting (or on any postprocedure study if one is performed before discharge or within 30 days of the procedure on CEA patients). In addition, they should consider monitoring other complications such as myocardial infarction and recording whether strokes are ipsilateral or contralateral to the procedure. For CEA, lower cranial nerve palsies could also be tracked. For angioplasty and stenting, centers should also consider tracking the major nonneurological angiographic complications, specifically renal failure, retroperitoneal or thigh hematoma requiring transfusion or surgical evacuation, arterial occlusions requiring thrombectomy or thrombolysis, arteriovenous fistula, and pseudoaneurysm, as detailed by the Joint Standards of Practice Task Force of the Society of

Interventional Radiology, the American Society of Interventional and Therapeutic Neuroradiology, and the American Society of Neuroradiology.<sup>95</sup>

Centers should also track whether patients who undergo stenting are at high risk for complications of endarterectomy, because the available data primarily support stenting in such patients. High risk is defined as (1) patients with severe comorbidities (class III/IV congestive heart failure, class III/IV angina, left main coronary artery disease,  $\geq 2$ -vessel coronary artery disease, left ventricular ejection fraction  $\leq 30\%$ , recent myocardial infarction, severe lung disease, or severe renal disease) or (2) patients with technical or anatomic factors such as prior neck operation or neck irradiation, postendarterectomy restenosis, surgically inaccessible lesions (ie, above C2, below the clavicle), contralateral carotid occlusion, contralateral vocal cord palsy, or the presence of a tracheostomy.<sup>90</sup>

Centers that perform angioplasty without stenting may consider tracking these patients separately from patients who are stented.

Centers should consider monitoring whether patients were pretreated with antiplatelet therapy and whether they were prescribed antiplatelet therapy at discharge, and if so, which drug or drugs. Rapid thrombus formation after endothelial damage is a well-known and understood complication of endovascular stenting.<sup>96,97</sup> Early carotid stenting was associated with high rates of distal embolic events, as well as in-stent thrombosis. Subsequently, studies have demonstrated that dual-antiplatelet therapy with aspirin and a thienopyridine (ticlopidine or clopidogrel) significantly reduces 30-day morbidity.<sup>98,99</sup>

Finally, centers should consider tracking patients who undergo interventions for nonatherosclerotic disease processes such as arterial dissection or fibromuscular dysplasia.

#### **Metric 11**

#### **Percentage of patients undergoing intracranial angioplasty and/or stenting for atherosclerotic disease with stroke or death within 30 days of the procedure.**

**Numerator:** Patients who undergo intracranial angioplasty and/or stenting for atherosclerotic stenosis who die or have a stroke within 30 days of the procedure.

**Denominator:** All patients undergoing intracranial angioplasty and/or stenting because of atherosclerosis.

Patients who undergo these procedures of stenosis with other causes such as vasospasm, arterial dissection, or fibromuscular dysplasia should be excluded from this metric. Centers should consider tracking these patients separately. Patients who are stented as part of a procedure for coiling of an aneurysm should also be excluded. Outcomes of such patients will be recorded under the metric that tracks outcomes of aneurysm treatment. Stroke will be defined as in Metric 10, and patients for whom outcome data cannot be obtained should also be excluded and documented as detailed in Metric 10.

#### *Justification*

Angioplasty and stenting have become options for treatment of intracranial stenosis both within the context of clinical trials and in clinical practice. Because of the lack of definitive data about the efficacy of intracranial angioplasty stenting, it

is especially important to monitor patients for complications of the procedure. For secondary stroke prevention, angioplasty and stenting have been classified as investigational, with a **Class IIb; Level of Evidence C** rating for patients with >50% symptomatic stenosis of a major intracranial artery.<sup>90</sup> For acute ischemic stroke, angioplasty and stenting have also been classified as investigational, again with a **Class IIb; Level of Evidence C** rating.<sup>18</sup>

The end point of any stroke or death within 30 days of the procedure was chosen on the basis of both direct relevance to the procedure and reproducible ascertainment across studies.<sup>100,101</sup> The end point has been used in 2 major prospective registry studies of intracranial stenting, Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVA) and the Wingspan stent study, which resulted in approval of endovascular devices by the US Food and Drug Administration. Multiple observational studies<sup>102,103</sup> have demonstrated that the end point of any stroke or death within 30 days can be ascertained reliably in multiple centers that perform intracranial angioplasty and/or stent placement.

#### *Additional Data Elements*

In addition to calculating this metric, centers should consider recording the location and other characteristics of any stroke that occurs after angioplasty and/or stenting, as well as the cause of death in patients who die. Centers should consider tracking the same elements that we proposed as additional elements for carotid stenting. In particular, they should consider tracking the degree of residual stenosis on angiogram at the end of the procedure, pretreatment with antiplatelet therapy, discharge with a prescription for antiplatelet therapy, and major nonneurological angiographic complications.<sup>95</sup>

## **Intracranial Hemorrhage**

### **Metric 12**

#### **Percentage of SAH, ICH, and AVM patients for whom initial severity measures are documented. (Core metric)**

The severity of SAHs should be documented with the Hunt and Hess scale,<sup>104</sup> and the severity of ICHs should be documented with the ICH score, which incorporates the Glasgow Coma Scale (GCS) and the size and location of the hemorrhage.<sup>105</sup> AVMs should be graded according to the Spetzler-Martin scale.<sup>106</sup> A combined ratio should be calculated as the primary metric, but separate ratios should also be calculated for each scale.

**Numerator:** The sum of the number of SAH patients for whom the Hunt and Hess scale is documented, the number of ICH patients without an AVM for whom the ICH score is documented, the number of AVM patients with hemorrhage for whom the ICH score and Spetzler-Martin score are documented, and the number of AVM patients without hemorrhage for whom the Spetzler-Martin score is documented. For a patient to be counted in the numerator, the Hunt and Hess and GCS scores should be documented in the initial neurological or neurosurgical admitting or consultation note or in a separate earlier note and should be evaluated before the start of any endovascular or surgical procedure. The ICH

score and Spetzler-Martin score may be determined later after analysis of imaging.

**Denominator:** Sum of the number of SAH patients, the number of ICH patients without an AVM, and the number of AVM patients.

#### *Justification*

There is less consensus about what scales to use to assess SAH and ICH patients than to assess ischemic stroke patients. For SAH, the AHA/ASA "Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage"<sup>107</sup> state that scales that rely heavily on the severity of the initial hemorrhage are helpful in planning future care (**Class I; Level of Evidence B**), and the degree of neurological impairment according to an accepted SAH grading system can be useful for prognosis and triage (**Class IIa; Level of Evidence B**).<sup>108</sup> Thus, to capture the clinical state of the patient, we recommend using the Hunt and Hess scale for SAH patients.

The Hunt and Hess scale was developed 40 years ago and is probably still the most commonly used scale in the United States, if not the world, for assessment of risk of repair of ruptured intracranial aneurysms. It remains as accurate as other scales that have been proposed since its development, if not more so. Although the initial Hunt and Hess score should be documented, the score immediately before surgery may be the most accurate. The Hunt and Hess score has been used in clinical decision making and is useful as a predictor of outcome.<sup>104,108–112</sup>

To help stratify severity of illness for ICH patients, an easy-to-use, common scale developed for this purpose should be used and documented. Although there have been many attempts to develop prognostic tools for outcome after ICH, the only clinical scale for this purpose is the ICH score.<sup>105</sup> The ICH score combines the ubiquitously used GCS, the patient's age, the presence of intraventricular hemorrhage, location (infratentorial or supratentorial) of the hemorrhage, and the volume calculated by the ABC method.<sup>105,113</sup> The GCS score, age, hematoma location and volume, and presence of intraventricular hemorrhage have all been shown repeatedly to be predictors of outcome after ICH, as noted in the AHA/ASA "Guidelines for the Management of Spontaneous Intracerebral Hemorrhage."<sup>114</sup> The ICH score, which has been validated in several populations, reliably predicts outcome and 30-day mortality for patients with spontaneous ICH.<sup>105,115–117</sup>

To help stratify the risk of surgery for the AVM patient, an easy-to-use, common scale developed for this purpose should be used and documented. Although there have been many changes in the treatment of AVMs, the only clinical scale developed for the purpose of predicting outcome after AVM surgery is the Spetzler-Martin score. This scale, which is based on characteristics of the AVM, was also validated in surgical populations and in radiosurgery-treated patients.<sup>106,118,119</sup>

Because of the use of these scores in making clinical decisions and because of the need for quantified measures of initial severity to interpret data about outcomes and other metrics, we have classified this metric as a core metric.

*Additional Data Elements*

CSCs should also consider tracking the individual components that lead to the grades for the different scores. For example, for Spetzler-Martin grading of AVMs, AVM size, the presence of deep or superficial drainage, and involvement of eloquent brain areas should be recorded. For the ICH score, hemorrhage dimensions and GCS score should be recorded. Centers may also want to track the Fisher scale,<sup>120</sup> the World Federation of Neurological Surgeons scale,<sup>121</sup> or the GCS for SAH patients.<sup>108</sup> CSCs should also consider recording the location of aneurysms, ICH, and AVM; the size of aneurysms; the procedures used for aneurysm and AVM treatment; the presence of any residual aneurysm or AVM; and rebleeding during admission.

**Subarachnoid Hemorrhage***Metric 13*

**Median time from admission to start of procedure intended to obliterate a ruptured aneurysm by surgical clipping or endovascular coiling for patients who arrive within 48 hours of the hemorrhage that led directly to admission. (Core metric)**

Patients who are not treated should be excluded from this metric, but the reason they were not treated should be recorded, as discussed in Metric 14. Times for this metric should be recorded to the nearest hour, in contrast to the measures for acute ischemic stroke, which should be recorded in hours and minutes.

Patients with sentinel hemorrhage >48 hours before admission and a second hemorrhage within the 48 hours before admission should be included in this metric.

*Justification*

Securing an aneurysm by endovascular coiling or surgical clipping is an urgent goal to prevent rerupture of the aneurysm and to facilitate treatment for vasospasm, if it occurs. Later treatment times are associated with increased rates of preoperative bleeding.<sup>122–124</sup> There is a 3% to 4% or higher risk of rebleeding in the first 24 hours and a 1% to 2% risk per day in the first month,<sup>107</sup> so the AHA/ASA “Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage” recommend urgent evaluation and treatment of patients with suspected SAH (*Class I; Level of Evidence B*). Given the strength of the evidence in favor of urgent treatment of aneurysms and the catastrophic consequences of rebleeding before treatment, we propose that time to aneurysm treatment should be a core metric.

Because the risk of rebleeding decreases with each day after hemorrhage and the urgency of securing an aneurysm therefore decreases, we have limited this metric to patients who arrive within 48 hours of hemorrhage.

We have not specified a benchmark time from arrival for securing aneurysms because the available evidence does not clearly identify such a time.

*Additional Data Element*

Centers should also consider recording the type of procedure (coiling or clipping) and at least basic procedural details (eg, the specific coils and clips that were used) that are needed for quality improvement purposes.

*Metric 14*

**Percentage of patients with aneurysmal SAH arriving within 48 hours of hemorrhage for whom a coiling or clipping procedure was not started within 36 hours of arrival who have a documented reason for not having undergone coiling or clipping within 36 hours of arrival.**

**Numerator:** Number of patients with aneurysmal SAH who arrive within 48 hours of hemorrhage and whose ruptured aneurysm is not coiled or clipped within 36 hours of arrival for whom the reason for not treating is documented.

**Denominator:** Total number of patients with aneurysmal SAH who arrive within 48 hours of hemorrhage and whose ruptured aneurysm is not coiled or clipped within 36 hours of arrival.

Reasons for not treating may include but are not limited to futility, medical instability, patient or family wishes, and delayed arrival of the patient to the CSC. Problems leading to medical instability may include but are not limited to stunned myocardium with shock, neurogenic pulmonary edema, status epilepticus, septic shock, hypoxemic respiratory failure, uncontrolled or refractory intracranial pressure, poor neurological status, and repeat hemorrhage before treatment.

Unavailability of neurosurgical or endovascular services is also an acceptable reason for this metric, because although coverage by neurosurgeons and endovascular interventionalists 24 hours a day, 7 days a week is expected to be an integral part of CSCs,<sup>3</sup> we recognize that there are some occasions when angiography or surgical clipping is legitimately not available. Reasons for such unavailability include simultaneous emergencies, equipment failure, and unavailability of key staff, for example, for medical- or weather-related reasons. Reasons and their frequency should be tracked carefully and reported separately. Moreover, CSCs must have contingency plans to minimize the chance that services are unavailable. In particular, CSCs should develop contingency plans to transfer patients to another center (or to divert them to another center initially) if there are short periods when endovascular or neurosurgical procedures are not available. CSCs should not accept transfers of patients who may be candidates for such procedures during these periods unless there are other overriding reasons for transfer, such as a need for intensive care not available at another center that is within an appropriate distance. Quality improvement efforts should be directed toward decreasing such periods when services are unavailable.

*Justification*

Because of the risks of rebleeding<sup>107,122–124</sup> and the difficulty in treating vasospasm when a ruptured unsecured aneurysm is present, it is important to document the reason why a ruptured aneurysm is not treated quickly. The reasons why patients are not treated or why treatment is delayed should be reviewed as part of quality improvement efforts.

*Metric 15*

**Percentage of patients with documented aneurysmal SAH for whom nimodipine treatment (60 mg every 4 hours or 30 mg every 2 hours) is started within 24 hours of diagnosis and for whom such treatment is continued until**

**21 days after the hemorrhage or until discharge if they are discharged <21 days after the SAH. (Core metric)**

**Numerator:** Patients with documented aneurysmal SAH treated with nimodipine 60 mg every 4 hours (or 30 mg every 2 hours) within 24 hours of diagnosis and who continue this treatment until 21 days after their hemorrhage, or until discharge if they are discharged <21 days after the SAH, or until they develop a contraindication to nimodipine. Acceptable contraindications include documentation of intractable hypotension or allergy to nimodipine.

**Denominator:** All patients with a diagnosis of aneurysmal SAH.

Patients whose dose of nimodipine is reduced because of hypotension will be considered to be in compliance with this metric. Patients who have a known contraindication to nimodipine and are therefore not treated with it will also be considered to be in compliance with this metric. Patients who arrive at a CSC with documented aneurysmal SAH should receive nimodipine within 24 hours of admission.

*Justification*

The AHA/ASA “Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage” recommend nimodipine to reduce the risk of poor outcomes after aneurysmal SAH (**Class I; Level of Evidence A**).<sup>107,125</sup> Nimodipine has been shown to be beneficial in randomized controlled trials<sup>126,127</sup> and has been approved by the US Food and Drug Administration for “improvement of neurological outcome by reducing the incidence and severity of ischemic deficits in patients with subarachnoid hemorrhage from ruptured intracranial berry aneurysms.”<sup>128</sup> Given the strength of the evidence in favor of the use of nimodipine, we have classified this metric as a core metric.

*Additional Data Element*

As part of their quality improvement efforts, CSCs should consider monitoring whether patients who are not treated with nimodipine as described above are not treated with it all, have it stopped prematurely, or are treated with an incorrect dose.

**Metric 16****Percentage of SAH patients with diminished level of consciousness and ventriculomegaly who are treated with external ventricular drainage (EVD).**

**Numerator:** SAH patients with diminished level of consciousness and ventriculomegaly who are treated with EVD.

**Denominator:** SAH patients with diminished level of consciousness and ventriculomegaly.

Patients for whom EVD is recommended but not performed because the patients or their families or proxies refuse permission for it should be excluded from this metric.

*Justification*

The AHA/ASA “Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage” note that EVD can be beneficial in patients with ventriculomegaly and a diminished level of consciousness after acute SAH (**Class IIa; Level of Evidence B**).<sup>107</sup> Several reports support this conclusion about the value of EVD.<sup>129–132</sup>

**Metric 17****Median frequency of noninvasive monitoring performed for surveillance for vasospasm in patients with aneurysmal SAH during the period between 3 and 14 days after SAH.**

Most studies performed for this purpose will be transcranial Doppler (TCD) studies, but centers may count a single study by another modality (eg, CTA, CT perfusion, MR angiography, MR perfusion, catheter angiography, or electroencephalography) on days when TCD is not performed. Centers that perform surveillance TCD (or another type of study for surveillance) more than once daily would count each of the studies for the purposes of calculating the frequency of monitoring. Additional studies performed on a given day because the first study performed raised suspicion of vasospasm or because of clinical changes that suggest vasospasm should not be included in calculating the frequency of monitoring. Patients will be excluded on days on which there is documentation of a reason not to monitor for vasospasm (eg, patients being treated with comfort care only), on days after discharge or death, and on days before transfer to the CSC.

*Justification*

Cerebral vasospasm after aneurysmal SAH is seen in 30% to 70% of patients.<sup>120,133</sup> Vasospasm can lead to devastating ischemic infarcts, and monitoring for vasospasm is important so that it can be diagnosed early and treated to prevent ischemic injury. Vasospasm usually develops 3 to 5 days after SAH and peaks between 5 and 14 days after SAH.<sup>107</sup> Typically, TCD ultrasound has been the monitoring modality of choice, because it is noninvasive, relatively inexpensive, and portable. There are variable reports on its sensitivity and specificity for detecting vasospasm in general, but it reliably detects severe spasm, and the American Academy of Neurology Expert Committee concluded that the literature provides evidence that establishes TCD as useful for diagnosis of vasospasm.<sup>134–137</sup> In addition to TCD and traditional angiography, there are newer imaging modalities that can also detect vasospasm, such as perfusion imaging and angiography with MR or CT. Because of the continual changes in technology, we recommend monitoring the use of any of these modalities.

The intent of this metric is to track the number of studies performed for quality improvement purposes. We do not intend to imply more studies are necessarily better. We recognize that the available evidence does not permit the establishment of a specific benchmark for the minimum frequency of studies needed. Indeed, TCD may only have a limited ability to predict development of delayed cerebral ischemia, although this may be a result of therapies initiated because of the results of TCD.<sup>44</sup> Because there is a wide range of opinion about the optimum frequency of surveillance for vasospasm, we have not recommended this as a core metric. CSCs that choose not to track this metric should consider developing their own alternative metrics based on the protocols that they use to detect and treat vasospasm. In this regard, it is worth noting that The Joint Commission (for the Accreditation of Hospitals) has allowed hospitals to use “nonstandardized” measures of their own choosing for some



aspects of care in the early stages of developing criteria for disease-specific centers that treat diseases other than stroke.<sup>138</sup>

#### *Additional Data Elements*

In addition to recording the frequency of studies that were performed for detection of vasospasm, CSCs should consider tracking the types of studies performed and the days on which they were performed, as well as the types of therapy that were used for treatment of vasospasm and whether vasospasm was detected and treated before development of symptoms of delayed cerebral ischemia.

#### **Metric 18**

##### **Complication rates for aneurysm coiling and clipping.**

**Numerator:** Patients undergoing coiling or clipping of a ruptured or a nonruptured cerebral aneurysm who have complications as detailed below.

The complications to be monitored for endovascular or surgical treatment of ruptured intracranial aneurysms are ischemic stroke or death within 24 hours of the procedure or any rebleeding or second treatment for residual aneurysm within 30 days of the procedure. The complications to be monitored for endovascular or surgical treatment of unruptured intracranial aneurysms are any ischemic stroke, death, subarachnoid bleeding, or second treatment for residual aneurysm within 30 days of the procedure.

**Denominator:** Patients undergoing coiling or clipping of a ruptured or a nonruptured cerebral aneurysm.

The metric should be calculated separately for the following groups of patients: (1) Unruptured aneurysms undergoing coiling; (2) unruptured aneurysms undergoing clipping; (3) ruptured aneurysms undergoing coiling; and (4) ruptured aneurysms undergoing clipping. Death and stroke should be recorded separately. Stroke is defined as in Metric 10, and patients for whom 30-day outcome data are not available must be documented as noted for Metric 10.

#### *Justification*

The end points were chosen on the basis of both direct relevance to the procedure and reproducible ascertainment across studies.<sup>84,96,97,139–145</sup>

Bleeding is classified into 3 broad categories, and all of these should be included in calculating the metric: (1) Preprocedural if it occurred after admission but before the first attempt at aneurysm obliteration; (2) procedural if it occurred during the first (or subsequent) procedures; and (3) postprocedural if it occurred in the first 30 days after the procedure. CSCs should also track each of the 3 types of rebleeding separately.

Preprocedural rebleeding is included because the measure may be related to time interval between onset and treatment. In this regard, in ISAT, there was a small but significant difference for those patients allocated to endovascular treatment (mean interval 1.1 day) and those allocated to neurosurgery (mean interval 1.7 day), and the rate of rebleeding before the procedure was higher among those randomized to surgery than among those randomized to endovascular treatment (1.6% versus 0.7%).<sup>84,141</sup>

Postprocedural rebleeding is included because it reflects the adequacy of obliteration. In ISAT, incomplete obliteration of aneurysms, whether by coiling or by clipping, was associated with an increased risk of rebleeding.<sup>84,141</sup>

The requirement for a second procedure within 30 days for the target aneurysm is included because it is a measure of incomplete obliteration of the target aneurysm in the initial procedure. Subtotal occlusion of aneurysms (including neck remnants, incomplete occlusions, and failed occlusions) was another end point that has been reported in previous studies but was not recommended because of the lack of a clinically meaningful and reproducible definition.

For ruptured aneurysms, we recommend considering only ischemic strokes and death within 24 hours of the procedure. Later strokes may be a result of vasospasm and therefore may be an effect of the initial hemorrhage. Similarly, although deaths shortly after a procedure should be presumed to be a result of the procedure, deaths at longer intervals are increasingly likely to be related to the severity of the underlying hemorrhage or other causes. In this regard, the consensus among radiologists is that deaths should be considered related to diagnostic angiograms only if the cause of death was present within 24 hours of the procedure.<sup>95</sup> To avoid the difficulty of determining when the cause of death developed, we propose to simply include all deaths within 24 hours.

Factors such as the patient's age, clinical grade and location of aneurysm, and the need for stenting should be taken into account before interpretation of the observed rate of these end points.<sup>84,141,142,146</sup>

#### *Additional Data Elements*

CSCs should consider tracking other variables involving aneurysm patients, including the location and size of their aneurysms, development of vasospasm, delayed cerebral ischemia, presence of residual aneurysm after treatment, and for coiling, major nonneurological angiographic complications.<sup>95</sup> They should also record whether aneurysm patients were seen in consultation by a vascular neurologist before and/or after coiling or clipping.

### **Intracerebral Hemorrhage**

#### **Metric 19**

**Median time from arrival to start of treatment to reverse the international normalized ratio (INR) with a procoagulant preparation (eg, fresh frozen plasma, recombinant factor VIIa, prothrombin complex concentrates) for patients with warfarin-associated ICH and an elevated INR (INR >1.4). (Core metric)**

Patients with an elevated INR should be excluded from this metric if a reason is documented for not treating them, for example, if there is a decision to treat the patient with comfort measures only or if the risks of reversing anticoagulation are judged to outweigh the benefits. Times for this metric should be recorded in minutes.

#### *Justification*

Hemorrhage expansion appears to occur primarily in the first hours after a hemorrhage, so treatment should be initiated as quickly as possible, with a goal of rapid correction of the INR. Warfarin use is a significant risk factor for hemorrhage

expansion, with an odds ratio of 6.2.<sup>114,147</sup> It is therefore imperative to correct the INR rapidly to prevent hemorrhage expansion.<sup>148,149</sup> The AHA/ASA “Guidelines for the Management of Spontaneous Intracerebral Hemorrhage”<sup>114</sup> recommend treatment with intravenous vitamin K and with replacement of clotting factors (*Class I; Level of Evidence B*), although clinical trials have not established the superiority of any specific treatment strategy.<sup>150</sup> Given the strength of the evidence in favor of prompt correction of the INR, we propose that time to initiation of treatment to correct the INR should be a core metric. There are no definitive trials establishing the best treatment; however, procoagulant preparations such as fresh frozen plasma and newer options including recombinant factor VIIa and prothrombin complex concentrate work faster than vitamin K, so the time to start of treatment with a procoagulant preparation should be recorded for this metric rather than the time of treatment with vitamin K.<sup>151–158</sup>

#### *Additional Data Element*

CSCs should also consider tracking the time from arrival to achievement of an INR <1.4 for patients who present with ICH and an INR of  $\geq 1.4$ .

#### *Metric 20*

**Percentage of patients undergoing surgical or endovascular treatment of an AVM with stroke or death within 30 days of the procedure.**

**Numerator:** Patients undergoing surgical or endovascular treatment of an AVM with new intracranial hemorrhage or ischemic stroke or death within 30 days of the procedure.

**Denominator:** All patients undergoing surgical or endovascular treatment of an AVM.

The metric should be calculated for AVMs treated after hemorrhage separately for embolization and for surgical resection and for AVMs treated with no history of hemorrhage separately for each type of procedure, with the 30-day assessment periods depending on the date of the given procedure. Stroke is defined as in Metric 10, and patients for whom 30-day outcome data are not available must be documented as noted for Metric 10.

#### *Justification*

The recommended end points to monitor for endovascular or surgical treatment of an AVM are new intracranial hemorrhage or ischemic stroke or death within 30 days of the procedure. The end point is derived from the ARUBA trial (A Randomized Multicenter Clinical Trial of Unruptured Brain AVMs).<sup>159</sup> This trial, which is currently in progress and is the first major randomized trial of AVM treatment, compares medical management of unruptured AVMs to interventional treatment with any combination of endovascular, surgical, and radiosurgical approaches. Its primary end point is stroke or death. To examine procedure-related complications, we propose examining these end points within 30 days. Because of the variations in morphological features, clinical presentations, and the multitude of modalities used for treatment, adjustment for known predictors is strongly recommended before the complication rates of the procedure are interpreted.<sup>160</sup> It is particularly important to track the complications of treating

unruptured AVMs, because the benefit of treating such lesions is unproven.

#### *Additional Data Elements*

In addition to recording whether or not there was any complication, centers should consider tracking the rates of each of the complications and should analyze them for quality improvement purposes. CSCs should also consider tracking details of AVM location and vascular characteristics, as well as features of the clinical presentation, including the presence of seizures, progressive deficit, headaches, and hemorrhage, and the use of stereotactic radiosurgery.

CSCs should also consider tracking other major complications of AVM treatment that are being monitored in the ARUBA trial and have been recorded in other studies of AVM treatment.<sup>159,161</sup> These include neurological events such as new seizures, focal neurological deficits (unrelated to stroke), and new-onset headache, as well as nonneurological complications including acute renal failure, procedure-related nephropathy, contrast reaction, infection related to invasive therapy, periprocedural bleeding (other than intracranial), systemic (nonbrain) embolization, vascular injury related to invasive therapy, and catheter adherence to embolization material.

### **Stroke Systems of Care**

#### *Metric 21*

**Percentage of patients with ischemic or hemorrhagic stroke or TIA transferred from another hospital to the CSC with documentation of the time from the first call from the transferring hospital to the CSC (to a member of a stroke program or to a centralized transfer center) to arrival time at the CSC.**

**Numerator:** Patients with ischemic or hemorrhagic stroke or TIA transferred from another hospital for whom time from initial call to arrival is documented.

**Denominator:** All patients with ischemic or hemorrhagic stroke or TIA transferred from another hospital to the CSC.

Times should be recorded in hours and minutes. The percentage of transferred patients for whom the time of the initial contact cannot be identified should also be tracked.

#### *Justification*

CSCs need to demonstrate the existence of a functioning network and effective transfer protocols for timely transfer to the CSC from regional referring hospitals. Within a region, CSCs serve as the foundation of stroke care. Although most patients will initially present to either PSC or nonstroke centers, many patients may require more advanced interventions and management, including endovascular or neurosurgical procedures, specialized ICU care, and specialized diagnostic procedures. Data clearly demonstrate the important association of time to initiation of both intravenous and endovascular treatment of ischemic stroke. Good clinical outcome and rapid treatment are also important for patients with SAH and ICH. Thus, a CSC must document and monitor the details of transfers to ensure timely and efficient transport. The time of the call from the originating hospital and the time of arrival at the CSC should be collected and reviewed to eliminate systematic delays.<sup>5</sup>

Given variability in local conditions, the lack of data about what is a reasonable expectation, and the varying urgency of transfer for different types of patients, we do not think that it is appropriate to set a benchmark time or to compare centers on the basis of their average transfer times, and therefore, the metric is simply whether the time is recorded, so that it can be used for quality improvement and to look for improvements as the CSC and its surrounding hospitals gain more experience. We recognize that it may be difficult to identify the time of the initial contact from the referring hospital, but because of the importance of an efficient transfer process, we encourage CSCs to develop procedures to make it possible to track the time that the transfer process takes.

#### *Additional Data Elements*

Centers should also consider tracking additional data points for purposes of quality improvement (eg, time from onset to initial call, transfer ambulance dispatch time, departure time, source of ambulance, reason for transfer, transport delay for administration of intravenous thrombolysis, or transport during intravenous thrombolysis) depending on local procedures, availability of air transportation, and weather conditions.<sup>5</sup> Analysis of the times for patients for whom the initial call is within 6 hours of stroke onset should be a priority, because these are the patients for whom rapid transfer is generally most important. We anticipate that the actual transfer times for such patients may eventually become a metric for stroke systems of care that include both CSCs and PSCs.

CSCs should consider documenting all requests for patient transfers and should document reasons for accepting or not accepting the transfer to ensure the CSC provides necessary services as appropriate. Hospitals that make the commitment to serve as a CSC must be available to the regional hospitals without exception. Overcrowding issues often cause hospitals to divert patients during these times. Because most CSCs will be geographically distant from other CSCs, every CSC must make the necessary accommodations to accept appropriate patients who require the unique services of the CSC.

### **ICU and Stroke Unit**

#### *Metric 22*

**Percentage of patients admitted to each type of unit to which patients with ischemic or hemorrhagic stroke or TIA are initially admitted (eg, neurological/neurosurgical ICU, medical ICU, surgical ICU, general ICU, coronary care unit, burn ICU, stroke unit, other intermediate-level-of-care unit, neurology floor, or other floor). A separate percentage should be calculated for each type of unit.**

**Numerator:** Patients admitted to each type of unit to which all patients with ischemic or hemorrhagic stroke or TIA could be admitted initially.

**Denominator:** All patients with ischemic or hemorrhagic stroke or TIA.

Patients with inpatient strokes are excluded from this measure.

#### *Justification*

The BAC PSC report emphasizes that all PSCs must have a stroke unit (**Class I; Level of Evidence A**), and the BAC CSC report reaffirms this for CSCs.<sup>2,3</sup> The BAC CSC report<sup>3</sup> adds

that a CSC must have a full ICU and that a dedicated neurosciences ICU is desirable but not required for a CSC. There is good evidence that clinical outcomes are better and resource utilization is lower for neurocritically ill patients treated in a neurosciences ICU,<sup>162–167</sup> so documenting the location of treatment is warranted. The primary argument against requiring a specialized neurosciences ICU is resource availability: The number of specialty ICUs in any hospital may be limited, and the number of neurointensivists may be equally limited. In this setting, we believe that it is essential to track the unit to which patients are admitted so that quality improvement efforts can identify and, if necessary, correct variations in care between different units in a hospital or improve triage of patients to the most appropriate unit.

#### *Additional Data Element*

We recognize that patients move from one unit to another during an admission and that tracking these transfers may be important to understanding variations in care, but because collecting the necessary data may be time consuming, we have not included it in Metric 22. We do recommend, for quality improvement purposes, that CSCs should consider tracking the units to which a patient is transferred after admission. In addition, if and when the CSC guidelines are modified and a neurosciences ICU is required, we would favor consideration of a metric that examines whether or not each stroke patient who requires ICU admission is admitted to the neurosciences ICU and whether or not each stroke patient not admitted to an ICU is admitted to a stroke unit or to another intermediate-care unit or regular floor bed.

### **Outcomes and Complications**

#### *Metric 23*

**Percentage of patients with stroke or death within 24 hours of diagnostic neuroangiography. (Core metric)**

**Numerator:** Patients with death or stroke after diagnostic neuroangiography within 24 hours of the procedure or before discharge, whichever comes first.

**Denominator:** All patients who undergo a diagnostic neuroangiographic procedure.

Patients should be excluded if they undergo a therapeutic angiographic intervention as part of the same procedure or within the first 24 hours after the diagnostic procedure unless the complication is identified before the therapeutic intervention begins. Stroke is defined as in Metric 10, and patients for whom 24-hour outcome data are not available must be documented as noted for Metric 10.

#### *Justification*

The Joint Standards of Practice Task Force of the Society of Interventional Radiology, the American Society of Interventional and Therapeutic Neuroradiology, and the American Society of Neuroradiology<sup>95</sup> reviewed the complications of diagnostic neuroangiography. The Task Force stated that neurological complications that occurred within 24 hours of the angiogram should be attributed to the angiogram, as should all deaths for which the onset of the cause is within 24 hours of the angiogram. The Task Force suggested that the rate of reversible neurological deficits (including TIA and stroke) should be <2.5% and that of permanent neurological

deficits <1% but acknowledged that it is difficult to set universal thresholds, and the Task Force advised institutions to alter the thresholds as needed to higher or lower values to meet their own quality improvement program needs. To define a metric in a way that minimizes the need for subjective interpretation and simplifies data collection, we propose the inclusion of only those strokes or deaths that occur within 24 hours of the diagnostic angiogram. Because of the consensus that diagnostic angiography should be a low-risk procedure, and because the end point should be straightforward to collect, this is a core metric.

#### *Additional Data Elements*

Centers should consider monitoring the nature of individual complications to assist in quality improvement efforts. Centers should also consider tracking the major nonneurological angiographic complications, specifically renal failure, retroperitoneal or thigh hematoma requiring transfusion or surgical evacuation, arterial occlusions requiring thrombectomy or thrombolysis, arteriovenous fistula, and pseudoaneurysm, as detailed by the Joint Standards of Practice Task Force.<sup>95</sup> We have not included these complications as part of this or other metrics because of concerns about the number of patients who would require follow-up and because of difficulty in the identification of complications, because they may occur after discharge and patients may not necessarily return for treatment to the center where the procedure was performed. Centers should also consider tracking the use of measures to prevent acute renal injury, such as treatment with N-acetylcysteine and prehydration.

#### *Metric 24*

##### **Percentage of patients who have a diagnosis of ischemic or hemorrhagic stroke who undergo EVD and then develop ventriculitis.**

For this metric, ventriculitis is defined as the presence of positive cerebrospinal fluid cultures in a patient with EVD if there is no documentation in the medical record stating that the culture results are thought to be the result of a contaminant or of some other process (eg, preexisting infection or infection resulting from another surgical procedure).

**Numerator:** All patients with ventriculitis after EVD, as defined above, and a diagnosis of ischemic or hemorrhagic stroke.

**Denominator:** All patients who undergo ventriculostomy because of problems related to ischemic or hemorrhagic stroke.

#### *Justification*

Ventriculitis is a dangerous and potentially avoidable nosocomial infection that can lead to serious morbidity or mortality and significantly prolong hospitalization. For these reasons, it is important for hospitals to identify all cases of ventriculitis for purposes of quality improvement. Although there are no guideline statements that specifically address ventriculitis, because the consequences are so significant and because it is a nosocomial problem, we recommend that this be a core metric.<sup>168,169</sup>

#### *Additional Data Element*

For patients with ventriculitis, CSCs should consider tracking the unit (or units) to which a patient who developed ventriculitis was admitted while the EVD was in place. CSCs should also consider tracking the number of days that an EVD was in place before the development of ventriculitis; the total number of days that an EVD was in place, whether or not an infection developed; the frequency with which the EVD was changed; and other measures taken to prevent ventriculitis. CSCs should also consider tracking whether ventriculitis developed in the setting of systemic sepsis, whether patients were treated with prophylactic antibiotics, and where the EVD was placed (eg, operating room, ICU, or emergency department).

### **Poststroke Rehabilitation**

#### *Metric 25*

##### **Median number of days from admission to completion of evaluations for physical therapy, occupational therapy, speech-language pathology, and rehabilitation medicine, unless there is documentation on admission that some or all of these evaluations are not needed or that the patient cannot tolerate them because of medical instability.**

The center should track its record for each discipline separately, but the overall metric for completion of all of the rehabilitation-related consultations that are deemed appropriate for an individual patient should be the primary statistic to be monitored. In other words, the primary time recorded for each patient would be the time when the last of the consultations that were deemed necessary on admission was completed.

#### *Justification*

There is limited evidence that early initiation of stroke rehabilitation is associated with improved functional outcomes, on the basis of nonrandomized trials and 1 meta-analysis<sup>170</sup> (**Class I; Level of Evidence B**). In their review of 38 randomized controlled trials dating back to 1965, Cifu and Stewart<sup>171</sup> concluded that early stroke rehabilitation “appears to have a strong relationship” with improved functional outcome at hospital discharge and follow-up. However, as with many reviews of the topic, the studies did not delineate a specific amount of time at which rehabilitation began. They did not describe the association of the provision and timing of specific therapies with functional gain. None of the studies compared early therapy with either delayed therapy or standard care.

There is evidence from 2 randomized controlled trials that early mobilization is associated with improved outcome (**Class I; Level of Evidence B**). There are, however, no randomized trials that directly examined the intensity, duration, frequency, and risks and benefits of early rehabilitation therapy<sup>172</sup> (**Class IIa; Level of Evidence B**). Early mobilization in acute stroke care is recommended in a range of European, US, and United Kingdom policy guidelines as a strategy to minimize or prevent complications.<sup>173,174</sup>

Despite the advent of tPA and other therapies for the hyperacute treatment of stroke, rehabilitation remains the primary treatment modality for patients recovering from

stroke. Fifty percent to 70% of stroke survivors regain functional independence, but 15% to 30% are permanently disabled, and 20% require institutional care at 3 months after onset.<sup>175</sup> Published studies have demonstrated that organized multidisciplinary stroke rehabilitation reduces death, death or disability, and death or institutionalization<sup>176–184</sup> (**Class I; Level of Evidence B**). Rehabilitation may increase the stroke patient's quality of life and reduce the financial and physical burden on society.<sup>185–187</sup> In addition to inpatient rehabilitation, outpatient rehabilitation programs can improve outcomes and prevent functional deterioration.<sup>188</sup>

Stroke rehabilitation begins during the acute hospitalization, as soon as the diagnosis of stroke is established and the stroke survivor is deemed medically stable. During the acute phase, the primary goals of rehabilitation are to ensure proper management of general health functions, mobilize the patient, encourage resumption of self-care activities, and provide emotional support to the patient and family. The evidence for acute stroke rehabilitation care suggests that organized care for poststroke patients achieves substantial and optimal outcomes, such as decreased mortality and dependency and a return to community living.<sup>186</sup>

The Joint Commission PSC performance standard for rehabilitation states, “A rehabilitation plan must be considered.”<sup>7</sup> The standard reminds PSCs that they should assess stroke survivors for postacute rehabilitation services but only requires documentation of the necessity of a postacute rehabilitation program. On the other hand, the PSC is not accountable for the infrastructure of the rehabilitation team, the timing of mobilizing the patient, or the process of synthesizing a rehabilitation plan.

To differentiate itself from a PSC, a CSC should explicitly involve appropriate members of the rehabilitation team: Physical therapy, occupational therapy, speech-language pathology, and a physician specializing in physical medicine and rehabilitation or having specific expertise in stroke rehabilitation. On the basis of evidence in the literature, the CSC must mobilize the stroke survivor and begin rehabilitation as soon as possible.

#### *Additional Data Elements*

CSCs should consider tracking whether there is documented communication between rehabilitation disciplines involved in the care of stroke patients on all normal business days. Brief documentation that simply states that rehabilitation therapy is being performed by the necessary disciplines as discussed at multidisciplinary rounds would be adequate. Because of the relatively short period of time that a stroke survivor may spend in an acute-care hospital, the clinical record should document formal or informal communication among the rehabilitation disciplines on normal business days (ie, Monday through Friday, except for holidays) to (1) assess the stroke survivor's progress or problems impeding progress, (2) consider possible resolutions to such problems, and (3) assess or reassess the rehabilitation plan (including discharge plans) established by the team.<sup>7</sup> Results of formal conferences or rounds should be documented in the clinical record.

## Research

### *Metric 26*

**Percentage of patients admitted with diagnoses of ischemic stroke, SAH, AVM, intracranial hemorrhage, extracranial cervical stenosis, intracranial stenosis, or TIA who are enrolled in a clinical research study.**

**Numerator:** Patients who are admitted with diagnoses of ischemic stroke, SAH, AVM, intracranial hemorrhage, extracranial cervical stenosis, intracranial stenosis, or TIA and are enrolled in a clinical research trial studying acute ischemic or hemorrhagic stroke or TIA, prevention of ischemic or hemorrhagic stroke, rehabilitation after stroke, or other aspects of cerebrovascular disease.

**Denominator:** All patients admitted with diagnoses of ischemic stroke, SAH, AVM, intracranial hemorrhage, extracranial cervical stenosis, intracranial stenosis, or TIA.

Any protocol approved by the institutional review board of the CSC is considered a clinical research study for the purposes of this metric.

If a patient meets all criteria for enrollment in a clinical study that is active at the center and is not enrolled in that study, the reasons for this should be documented and tracked.

### *Justification*

The BAC CSC report states that research is an important but optional component of CSCs. We strongly suggest that CSCs be active participants in ongoing acute stroke research, because there is a need for coordinated multisite initiatives to improve our ability to address critical questions about stroke treatments.<sup>189</sup> We propose this metric to assess actual enrollment of patients in clinical trials. As noted previously, enrollment of patients in trials studying acute ischemic stroke is especially important. Other trials, including those studying aneurysm and AVM treatment, interventions for ICH, stenting of carotid and intracranial stenosis, medical management for secondary prevention of ischemic stroke, and rehabilitation, are also critical to improving stroke care, and CSCs should participate in such trials and actively enroll patients in them.

### *Additional Data Element*

CSCs should also consider tracking the percentage of patients who are eligible for a clinical trial that is active at the center and are actually enrolled in a clinical trial.

## Decompressive Surgery

Although the AHA/ASA “Guidelines for the Early Management of Adults With Ischemic Stroke” and the AHA/ASA “Guidelines for the Management of Spontaneous Intracerebral Hemorrhage” both state that decompressive surgery is recommended under certain circumstances,<sup>18,114,190</sup> we have not recommended metrics related to this type of surgery because of its relatively uncommon nature and because of difficulties in defining the patients to whom such metrics would apply. We do recommend that CSCs consider collecting the data elements noted below about patients who undergo decompressive surgery.

**Additional Data Elements**

The AHA/ASA “Guidelines for the Management of Spontaneous Intracerebral Hemorrhage” advise cerebellar decompression for patients with a cerebellar hemorrhage >3 cm in size who are deteriorating neurologically or have brain stem compression or hydrocephalus (**Class I; Level of Evidence B**).<sup>18,114,190</sup> The AHA/ASA “Guidelines for the Early Management of Adults With Ischemic Stroke” support decompression for patients with “space-occupying cerebellar infarction” (**Class I; Level of Evidence B**).<sup>18,114,190</sup> The AHA/ASA “Guidelines for the Early Management of Adults With Ischemic Stroke” also suggest that hemicraniectomy may be appropriate for some patients with large infarcts in the cerebral hemispheres, but this is a weaker recommendation (**Class IIa; Level of Evidence B**), and again, there is not a definition that would be easy to apply to identify patients in whom the procedure should be performed.<sup>18,114,190</sup> In view of these issues, we recommend only that CSCs should consider tracking patients who undergo decompressive procedures, with attention given to their clinical examination before surgery, the time from stroke onset to surgery, and details of the procedure, and use these additional data elements for quality improvement efforts in combination with data about initial stroke severity and follow-up data, including the mRS at 3 months.

**Other Complications**

We have not recommended routine tracking of complications not related to procedures because such complications are often difficult to define and because we want to avoid creating an excessively time-consuming burden on CSCs. However, we recognize that centers may choose to monitor neurological and medical complications that we have not mentioned explicitly in our discussion. This could be done through participation in registries or through quality improvement projects that focus on different complications for limited periods of time.

Neurological complications in this category may include extension of ischemic stroke, new strokes, or hemorrhagic conversion of stroke, as well as others. Medical complications may include myocardial infarction, pneumonia, urinary tract infections, deep venous thrombosis, pulmonary embolism, and falls, among others.

**Risk Adjustment**

Measurement of outcomes and use of outcome data to improve quality are fundamental to the efforts of CSCs to provide the best possible quality of care to stroke patients; however, we recognize that outcome measures without adjustment for severity of illness or patient characteristics and clinical situation can be misleading. Basic clinical characteristics that should be collected include age, sex, race, ethnicity, and initial disease severity (eg, NIHSS score for ischemic stroke patients). These factors can provide the rudimentary risk adjustment that will be necessary so that patient outcomes can be compared fairly across centers. Indeed, the initial NIHSS score by itself is a strong predictor of outcome.<sup>191,192</sup> Many additional factors (eg, medical comorbidities, degree of stenosis, location and size of occlusive lesion)

may also need to be tracked for proper risk adjustment. These factors vary to some extent depending on the specific type of stroke that a patient has had and on the specific procedures and therapies that are used to treat it. More detailed risk adjustment schemes in the future should be collected in registries that centers are encouraged to participate in, as discussed in the next section.

**Registries**

To facilitate data collection in a standardized way and to avoid the redundant efforts that would occur if CSCs designed their own databases, we expect that CSCs will make use of national databases or registries to collect data required for metrics and to collect additional detailed data that will assist in quality improvement, some of which we have noted in the additional data elements discussed above. Such data may include information about the baseline characteristics of patients, the location and size of their strokes and vascular abnormalities, diagnostic tests and their results, treatments that are initiated, complications that develop, discharge plans, and clinical outcomes and ongoing treatments at follow-up after discharge. Although registries do exist for some of the diseases, conditions, and procedures that CSCs will need to monitor, some may require modification to capture all of the data elements that will be needed, and other databases will need to be developed. Participation in standardized registries will permit risk adjustment and eventually allow for comparisons between different CSCs. To optimize the efficiency of data collection and analysis, unified databases with different modules covering all of the types of patients seen at CSCs may be desirable.

**Discussion**

We have proposed a set of metrics and related data elements to facilitate monitoring the quality of care delivered at CSCs. Collection of such data will be an essential part of the dedication to quality improvement that is expected of CSCs.<sup>2,3</sup> In this regard, the data that CSCs collect will be more useful if they are collected in a standardized way so that they can be pooled for analysis. The willingness of CSCs to share data for this purpose will therefore be important. We recommend that one of the initial goals of analysis of data collected by CSCs should be refinement of these proposed metrics. We expect that such analysis will lead to improved protocols for clinical care and to hypotheses that can be tested in clinical trials.

Experience with the establishment of PSCs has demonstrated that designation of hospitals as stroke centers with formalized protocols for care and with mechanisms for monitoring their performance has been associated with improved performance.<sup>12–15</sup> The metrics that we have proposed for CSCs should help provide a framework for establishing CSCs and a foundation for improving care once they are established.

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\*Modest.

†Significant.

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## References

- Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, Ford E, Furie K, Go A, Greenlund K, Haase N, Hailpern S, Ho M, Howard V, Kissela B, Kittner S, Lackland D, Lisabeth L, Marelli A, McDermott M, Meigs J, Mozaffarian D, Nichol G, O'Donnell C, Roger V, Rosamond W, Sacco R, Sorlie P, Stafford R, Steinberger J, Thom T, Wasserthiel-Smolter S, Wong N, Wylie-Rosett J, Hong Y. Heart disease and stroke statistics—2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee [published corrections appear in *Circulation*. 2009;119:e182 and *Circulation*. 2010;122:e11]. *Circulation*. 2009;119:e21–e181.
- Alberts MJ, Hademenos G, Latchaw RE, Jagoda A, Marler JR, Mayberg MR, Starke RD, Todd HW, Viste KM, Girgus M, Shephard T, Emr M, Shwayder P, Walker MD; for the Brain Attack Coalition. Recommendations for the establishment of primary stroke centers. *JAMA*. 2000;283:3102–3109.
- Alberts MJ, Latchaw RE, Selman WR, Shephard T, Hadley MN, Brass LM, Koroshetz W, Marler JR, Booss J, Zorowitz RD, Croft JB, Magnis E, Mulligan D, Jagoda A, O'Connor R, Cawley CM, Connors JJ, Rose-DeRenzy JA, Emr M, Warren M, Walker MD; Brain Attack Coalition. Recommendations for comprehensive stroke centers: a consensus statement from the Brain Attack Coalition. *Stroke*. 2005;36:1597–1616.
- Schwamm LH, Pancioli A, Acker JE 3rd, Goldstein LB, Zorowitz RD, Shephard TJ, Moyer P, Gorman M, Johnston SC, Duncan PW, Gorelick P, Frank J, Stranne SK, Smith R, Federspiel W, Horton KB, Magnis E, Adams RJ. Recommendations for the establishment of stroke systems of care: recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems. *Stroke*. 2005;36:690–703.
- Acker JE 3rd, Pancioli AM, Crocco TJ, Eckstein MK, Jauch EC, Larrabee H, Meltzer NM, Mergendahl WC, Munn JW, Prentiss SM, Sand C, Saver JL, Eigel B, Gilpin BR, Schoeberl M, Solis P, Bailey JR, Horton KB, Stranne SK. Implementation strategies for emergency medical services within stroke systems of care: a policy statement from the American Heart Association/American Stroke Association Expert Panel on Emergency Medical Services Systems and the Stroke Council. *Stroke*. 2007;38:3097–3115.
- Markovchick VJ, Moore EE. Optimal trauma outcome: trauma system design and the trauma team. *Emerg Med Clin North Am*. 2007;25:643–654, viii.
- Florida Agency for Health Care Administration. Primary stroke centers and comprehensive stroke center designation. Available at: [http://www.fdhc.state.fl.us/mchq/health\\_facility\\_regulation/Hospital\\_Outpatient/hospital.shtml#stroke](http://www.fdhc.state.fl.us/mchq/health_facility_regulation/Hospital_Outpatient/hospital.shtml#stroke). Accessed December 19, 2010.
- New Jersey Primary Stroke Centers and Comprehensive Stroke Centers. New Jersey Stroke Center Act, certificate of need and acute care licensure program. Available at: [http://www.state.nj.us/health/legal/documents/njac843g\\_stroke.pdf](http://www.state.nj.us/health/legal/documents/njac843g_stroke.pdf). Accessed December 19, 2010.
- Texas Department of State Health Services. Stroke system development. Available at: <http://www.dshs.state.tx.us/emtraumasystems/stroke.shtm>. Accessed December 19, 2010.
- Joint Commission on Accreditation of Healthcare Organizations. Updated Primary Stroke Center Certification appendix for the disease-specific care manual. *Jt Comm Perspect*. 2005;25:3–6.
- Approved: revisions to disease-specific care certification requirements for primary stroke centers: new requirements regarding IV thrombolytic therapy. *Jt Comm Perspect*. 2008;28:6–7.
- Gropen TI, Gagliano PJ, Blake CA, Sacco RL, Kwiatkowski T, Richmond NJ, Leifer D, Libman R, Azhar S, Daley MB; NYSDOH



- Stroke Center Designation Project Workgroup. Quality improvement in acute stroke: the New York State Stroke Center Designation Project. *Neurology*. 2006;67:88–93.
13. Stradling D, Yu W, Langdorf ML, Tsai F, Kostanian V, Hasso AN, Welbourne SJ, Schooley Y, Fisher MJ, Cramer SC. Stroke care delivery before vs after JCAHO stroke center certification. *Neurology*. 2007;68:469–470.
  14. LaBresh KA, Reeves MJ, Frankel MR, Albright D, Schwamm LH. Hospital treatment of patients with ischemic stroke or transient ischemic attack using the “Get With The Guidelines” program. *Arch Intern Med*. 2008;168:411–417.
  15. Stoeckle-Roberts S, Reeves MJ, Jacobs BS, Maddox K, Choate L, Wehner S, Mullard AJ. Closing gaps between evidence-based stroke care guidelines and practices with a collaborative quality improvement project. *Jt Comm J Qual Patient Saf*. 2006;32:517–527.
  16. Committee on Quality of Health Care in America and Institute of Medicine. *Crossing the Quality Chasm: A New Health System for the 21st Century*. Washington, DC: National Academies Press; 2001.
  17. Bonow RO, Masoudi FA, Rumsfeld JS, Delong E, Estes NA 3rd, Goff DC Jr, Grady K, Green LA, Loth AR, Peterson ED, Piña IL, Radford MJ, Shahian DM. ACC/AHA classification of care metrics: performance measures and quality metrics: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures. *J Am Coll Cardiol*. 2008;52:2113–2117.
  18. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, Grubb RL, Higashida RT, Jauch EC, Kidwell C, Lyden PD, Morgenstern LB, Qureshi AI, Rosenwasser RH, Scott PA, Wijndicks EF. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Stroke*. 2007;38:1655–1711.
  19. Furlan A, Higashida R, Wechsler L, Gent M, Rowley H, Kase C, Pessin M, Ahuja A, Callahan F, Clark WM, Silver F, Rivera F. Intra-arterial prourokinase for acute ischemic stroke: the PROACT II study: a randomized controlled trial: Prolyse in Acute Cerebral Thromboembolism. *JAMA*. 1999;282:2003–2011.
  20. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med*. 1995;333:1581–1587.
  21. Diener HC, Lees KR, Lyden P, Grotta J, Davalos A, Davis SM, Shuaib A, Ashwood T, Wasiewski W, Alderfer V, Hårdemark HG, Rodichok L; SAINT I and II Investigators. NXY-059 for the treatment of acute stroke: pooled analysis of the SAINT I and II Trials. *Stroke*. 2008;39:1751–1758.
  22. Davis SM, Donnan GA, Parsons MW, Levi C, Butcher KS, Peeters A, Barber PA, Bladin C, De Silva DA, Byrnes G, Chalk JB, Fink JN, Kimber TE, Schultz D, Hand PJ, Frayne J, Hankey G, Muir K, Gerraty R, Tress BM, Desmond PM; EPITHET Investigators. Effects of alteplase beyond 3 h after stroke in the Echoplanar Imaging Thrombolytic Evaluation Trial (EPITHET): a placebo-controlled randomised trial. *Lancet Neurol*. 2008;7:299–309.
  23. Furlan AJ, Eydung D, Albers GW, Al-Rawi Y, Lees KR, Rowley HA, Sachara C, Soehngen M, Warach S, Hacke W; DEDAS Investigators. Dose Escalation of Desmoteplase for Acute Ischemic Stroke (DEDAS): evidence of safety and efficacy 3 to 9 hours after stroke onset. *Stroke*. 2006;37:1227–1231.
  24. Hacke W, Albers G, Al-Rawi Y, Bogousslavsky J, Davalos A, Eliasziw M, Fischer M, Furlan A, Kaste M, Lees KR, Soehngen M, Warach S; DIAS Study Group. The Desmoteplase in Acute Ischemic Stroke Trial (DIAS): a phase II MRI-based 9-hour window acute stroke thrombolysis trial with intravenous desmoteplase. *Stroke*. 2005;36:66–73.
  25. Khatri P, Hill MD, Palesch YY, Spilker J, Jauch EC, Carrozzella JA, Demchuk AM, Martin R, Mauldin P, Dillon C, Ryckborst KJ, Janis S, Tomsick TA, Broderick JP; Interventional Management of Stroke III Investigators. Methodology of the Interventional Management of Stroke III Trial. *Int J Stroke*. 2008;3:130–137.
  26. Smith WS, Sung G, Saver J, Budzik R, Duckwiler G, Liebeskind DS, Lutsep HL, Rymer MM, Higashida RT, Starkman S, Gobin YP, Frei D, Grobelny T, Hellinger F, Huddle D, Kidwell C, Koroshetz W, Marks M, Nesbit G, Silverman IE; Multi MERCI Investigators. Mechanical thrombectomy for acute ischemic stroke: final results of the Multi MERCI trial. *Stroke*. 2008;39:1205–1212.
  27. Hacke W, Donnan G, Fieschi C, Kaste M, von Kummer R, Broderick JP, Brott T, Frankel M, Grotta JC, Haley EC Jr, Kwiatkowski T, Levine SR, Lewandowski C, Lu M, Lyden P, Marler JR, Patel S, Tilley BC, Albers G, Bluhmki E, Wilhelm M, Hamilton S; ATLANTIS Trials Investigators; ECASS Trials Investigators; NINDS rt-PA Study Group Investigators. Association of outcome with early stroke treatment: pooled analysis of ATLANTIS, ECASS, and NINDS rt-PA stroke trials. *Lancet*. 2004;363:768–774.
  28. Hacke W, Kaste M, Bluhmki E, Brozman M, Dávalos A, Guidetti D, Larrue V, Lees KR, Medeghri Z, Machnig T, Schneider D, von Kummer R, Wahlgren N, Toni D; ECASS Investigators. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. *N Engl J Med*. 2008;359:1317–1329.
  29. Khatri P, Neff J, Broderick JP, Khoury JC, Carrozzella J, Tomsick T; IMS-I Investigators. Revascularization end points in stroke interventional trials: recanalization versus reperfusion in IMS-I. *Stroke*. 2005;36:2400–2403.
  30. Tomsick T, Broderick J, Carrozzella J, Khatri P, Hill M, Palesch Y, Khoury J; Interventional Management of Stroke II Investigators. Revascularization results in the Interventional Management of Stroke II trial. *AJNR Am J Neuroradiol*. 2008;29:582–587.
  31. Schlegel DJ, Tanne D, Demchuk AM, Levine SR, Kasner SE; Multi-center rt-PA Stroke Survey Group. Prediction of hospital disposition after thrombolysis for acute ischemic stroke using the National Institutes of Health Stroke Scale. *Arch Neurol*. 2004;61:1061–1064.
  32. Del Zoppo GJ, Saver JL, Jauch EC, Adams HP Jr; American Heart Association Stroke Council. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: a science advisory from the American Heart Association/American Stroke Association [published correction appears in *Stroke*. 2010;41:e562]. *Stroke*. 2009;40:2945–2948.
  33. Albers GW, Amarencu P, Easton JD, Sacco RL, Teal P; American College of Chest Physicians. Antithrombotic and thrombolytic therapy for ischemic stroke: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*. 2008;133(suppl):630S–669S.
  34. Barsan WG. Setting new directions for stroke care: proceedings of a national symposium on rapid identification and treatment of acute stroke. Paper presented at: Emergency Department Management of Stroke; December 12–13, 1996; Bethesda, MD.
  35. Field J. The ACLS Core Cases: Acute Stroke Case. In: Field, J, ed. *ACLS Provider Manual*. Dallas, TX: American Heart Association; 2006:103–117.
  36. Audebert HJ, Kukla C, Vatankeh H, Gotzler B, Schenkel J, Hofer S, Fürst A, Haberl RL. Comparison of tissue plasminogen activator administration management between Teletstroke Network hospitals and academic stroke centers: the Telemedical Pilot Project for Integrative Stroke Care in Bavaria/Germany. *Stroke*. 2006;37:1822–1827.
  37. Grotta JC, Burgin WS, El-Mitwalli A, Long M, Campbell M, Morgenstern LB, Malkoff M, Alexandrov AV. Intravenous tissue-type plasminogen activator therapy for ischemic stroke: Houston experience 1996 to 2000. *Arch Neurol*. 2001;58:2009–2013.
  38. Lees KR, Bluhmki E, Toni D, von Kummer R, Marler J, Brott TG, Kaste M, Grotta JC, Albers GW, Hamilton S, Tilley BC, Hacke W; for the ECASS I–III, ATLANTIS and NINDS Investigators. Time dependent response to treatment with intravenous rtPA for stroke: an updated pooled analysis of ECASS, ATLANTIS and NINDS stroke trials. Presented at: International Stroke Conference 2010, San Antonio, TX; February 26, 2010. *Stroke*. 2010;41:e247. Abstract 168.
  39. Marler JR, Tilley BC, Lu M, Brott TG, Lyden PC, Grotta JC, Broderick JP, Levine SR, Frankel MP, Horowitz SH, Haley EC Jr, Lewandowski CA, Kwiatkowski TP. Early stroke treatment associated with better outcome: the NINDS rt-PA stroke study. *Neurology*. 2000;55:1649–1655.
  40. Hoffman JR, Schriger DL. A graphic reanalysis of the NINDS Trial. *Ann Emerg Med*. 2009;54:329–336. 336.e1–336.e35.
  41. Hertzberg V, Ingall T, O’Fallon W, Asplund K, Goldfrank L, Louis T, Christianson T. Methods and processes for the reanalysis of the NINDS tissue plasminogen activator for acute ischemic stroke treatment trial. *Clin Trials*. 2008;5:308–315.
  42. Mann J. NINDS Reanalysis Committee’s reanalysis of the NINDS trial. *Stroke*. 2005;36:230–231. Letter.
  43. American Heart Association. Get With The Guidelines–Stroke. Available at: <http://www.strokeassociation.org/presenter.jhtml?identifier=3002728>. Accessed June 3, 2010.

44. Carrera E, Schmidt JM, Oddo M, Fernandez L, Claassen J, Seder D, Lee K, Badjatia N, Connolly ES Jr, Mayer SA. Transcranial Doppler for predicting delayed cerebral ischemia after subarachnoid hemorrhage. *Neurosurgery*. 2009;65:316–323.
45. The Joint Commission Disease-Specific Care Certification Program. *Stroke Performance Measurement Implementation Guide*. 2nd ed, version 2.a. Available at: [http://www.jointcommission.org/assets/1/18/stroke\\_pm\\_implementation\\_guide\\_ver\\_2a.pdf](http://www.jointcommission.org/assets/1/18/stroke_pm_implementation_guide_ver_2a.pdf). Accessed December 30, 2010.
46. Deleted in proof.
47. American Heart Association Web site. Target: Stroke. Available at: [www.americanheart.org/targetstroke](http://www.americanheart.org/targetstroke). Accessed September 21, 2010.
48. Latchaw RE, Alberts MJ, Lev MH, Connors JJ, Harbaugh RE, Higashida RT, Hobson R, Kidwell CS, Koroshetz WJ, Mathews V, Villablanca P, Warach S, Walters B; on behalf of American Heart Association Council on Cardiovascular Radiology and Intervention, Stroke Council, and the Interdisciplinary Council on Peripheral Vascular Disease. Recommendations for imaging of acute ischemic stroke: a scientific statement from the American Heart Association. *Stroke*. 2009;40:3646–3678.
49. Josephson SA, Dillon WP, Smith WS. Incidence of contrast nephropathy from cerebral CT angiography and CT perfusion imaging. *Neurology*. 2005;64:1805–1806.
50. Schramm P, Schellinger PD, Klotz E, Kallenberg K, Fiebich JB, Kulkens S, Heiland S, Knauth M, Sartor K. Comparison of perfusion computed tomography and computed tomography angiography source images with perfusion-weighted imaging and diffusion-weighted imaging in patients with acute stroke of less than 6 hours' duration. *Stroke*. 2004;35:1652–1658.
51. Sylaja PN, Dzialowski I, Krol A, Roy J, Federico P, Demchuk AM; Calgary Stroke Program. Role of CT angiography in thrombolysis decision-making for patients with presumed seizure at stroke onset. *Stroke*. 2006;37:915–917.
52. Wintermark M, Albers GW, Alexandrov AV, Alger JR, Bammer R, Baron JC, Davis S, Demaerschalk BM, Derdeyn CP, Donnan GA, Eastwood JD, Fiebich JB, Fisher M, Furie KL, Goldmakher GV, Hacke W, Kidwell CS, Kloska SP, Köhrmann M, Koroshetz W, Lee TY, Lees KR, Lev MH, Liebeskind DS, Ostergaard L, Powers WJ, Provenzale J, Schellinger P, Silbergleit R, Sorensen AG, Wardlaw J, Wu O, Warach S. Acute stroke imaging research roadmap. *Stroke*. 2008;39:1621–1628.
53. Xue J, Gao P, Wang X, Liao X, Wang Y. Ischemic lesion typing on computed tomography perfusion and computed tomography angiography in hyperacute ischemic stroke: a preliminary study. *Neurol Res*. 2008;30:337–340.
54. Kang DW, Chalela JA, Dunn W, Warach S; NIH-Suburban Stroke Center Investigators. MRI screening before standard tissue plasminogen activator therapy is feasible and safe. *Stroke*. 2005;36:1939–1943.
55. Bose A, Henkes H, Alfke K, Reith W, Mayer TE, Berlis A, Branca V, Sit SP; Penumbra Phase I Stroke Trial Investigators. The Penumbra System: a mechanical device for the treatment of acute stroke due to thromboembolism. *AJNR Am J Neuroradiol*. 2008;29:1409–1413.
56. del Zoppo GJ, Higashida RT, Furlan AJ, Pessin MS, Rowley HA, Gent M; PROACT Investigators. PROACT: a phase II randomized trial of recombinant pro-urokinase by direct arterial delivery in acute middle cerebral artery stroke: Prolyse in Acute Cerebral Thromboembolism. *Stroke*. 1998;29:4–11.
57. Gobin YP, Starkman S, Duckwiler GR, Gobelny T, Kidwell CS, Jahan R, Pile-Spellman J, Segal A, Vinuela F, Saver JL. MERCI 1: a phase I study of Mechanical Embolus Removal in Cerebral Ischemia. *Stroke*. 2004;35:2848–2854.
58. Macleod MR, Davis SM, Mitchell PJ, Gerraty RP, Fitt G, Hankey GJ, Stewart-Wynne EG, Rosen D, McNeil JJ, Bladin CF, Chambers BR, Herkes GK, Young D, Donnan GA. Results of a multicentre, randomised controlled trial of intra-arterial urokinase in the treatment of acute posterior circulation ischaemic stroke. *Cerebrovasc Dis*. 2005;20:12–17.
59. Saver JL. Time is brain—quantified. *Stroke*. 2006;37:263–266.
60. Khatri P, Abruzzo T, Yeatts SD, Nichols C, Broderick JP, Tomsick TA; IMS I and II Investigators. Good clinical outcome after ischemic stroke with successful revascularization is time-dependent. *Neurology*. 2009;73:1066–1072.
61. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC Jr, Alpert JS, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Halperin JL, Hiratzka LF, Hunt SA, Jacobs AK. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction) [published correction appears in *Circulation*. 2005;111:2013]. *Circulation*. 2004;110:588–636.
62. Higashida RT, Furlan AJ, Roberts H, Tomsick T, Connors B, Barr J, Dillon W, Warach S, Broderick J, Tilley B, Sacks D; Technology Assessment Committee of the American Society of Interventional and Therapeutic Neuroradiology; Technology Assessment Committee of the Society of Interventional Radiology. Trial design and reporting standards for intra-arterial cerebral thrombolysis for acute ischemic stroke [published correction appears in *Stroke*. 2003;34:2774]. *Stroke*. 2003;34:e109–e137.
63. Qureshi AI. New grading system for angiographic evaluation of arterial occlusions and recanalization response to intra-arterial thrombolysis in acute ischemic stroke. *Neurosurgery*. 2002;50:1405–1414.
64. Qureshi AI. Endovascular treatment of cerebrovascular diseases and intracranial neoplasms. *Lancet*. 2004;363(9411):804–813.
65. Tomsick T. TIMI, TIBI, TICI: I came, I saw, I got confused. *AJNR Am J Neuroradiol*. 2007;28:382–384.
66. Hacke W, Kaste M, Fieschi C, Toni D, Lesaffre E, von Kummer R, Boysen G, Bluhmki E, Hoxter G, Mahagne MH; for the ECASS Study Group. Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke: the European Cooperative Acute Stroke Study (ECASS). *JAMA*. 1995;274:1017–1025.
67. Graham GD. Tissue plasminogen activator for acute ischemic stroke in clinical practice: a meta-analysis of safety data. *Stroke*. 2003;34:2847–2850.
68. Katzan IL, Hammer MD, Furlan AJ, Hixson ED, Nadzam DM; Cleveland Clinic Health System Stroke Quality Improvement Team. Quality improvement and tissue-type plasminogen activator for acute ischemic stroke: a Cleveland update. *Stroke*. 2003;34:799–800.
69. Katzan IL, Hammer MD, Hixson ED, Furlan AJ, Abou-Chebl A, Nadzam DM; Cleveland Clinic Health System Stroke Quality Improvement Team. Utilization of intravenous tissue plasminogen activator for acute ischemic stroke. *Arch Neurol*. 2004;61:346–350.
70. Khatri P, Wechsler LR, Broderick JP. Intracranial hemorrhage associated with revascularization therapies. *Stroke*. 2007;38:431–440.
71. Larrue V, von Kummer R, del Zoppo G, Bluhmki E. Hemorrhagic transformation in acute ischemic stroke: potential contributing factors in the European Cooperative Acute Stroke Study. *Stroke*. 1997;28:957–960.
72. de Haan R, Limburg M, Bossuyt P, van der Meulen J, Aaronson N. The clinical meaning of Rankin “handicap” grades after stroke. *Stroke*. 1995;26:2027–2030.
73. Lyden PD, Shuaib A, Lees KR, Davalos A, Davis SM, Diener HC, Grotta JC, Ashwood TJ, Hardemark HG, Svensson HH, Rodichok L, Wasiewski WW, Ahlberg G; CHANT Trial Investigators. Safety and tolerability of NXY-059 for acute intracerebral hemorrhage: the CHANT Trial. *Stroke*. 2007;38:2262–2269.
74. Banks JL, Marotta CA. Outcomes validity and reliability of the modified Rankin scale: implications for stroke clinical trials: a literature review and synthesis. *Stroke*. 2007;38:1091–1096.
75. Quinn TJ, Dawson J, Walters MR, Lees KR. Reliability of the modified Rankin Scale. *Stroke*. 2007;38:e144.
76. Wilson JT, Hareendran A, Hendry A, Potter J, Bone I, Muir KW. Reliability of the modified Rankin Scale across multiple raters: benefits of a structured interview. *Stroke*. 2005;36:777–781.
77. Schwamm LH, Holloway RG, Amarenco P, Audebert HJ, Bakas T, Chumbler NR, Handschu R, Jauch EC, Knight WA IV, Levine SR, Mayberg M, Meyer BC, Meyers PM, Skalabrinn E, Wechsler LR; American Heart Association Stroke Council; Interdisciplinary Council on Peripheral Vascular Disease. A review of the evidence for the use of telemedicine within stroke systems of care: a scientific statement from the American Heart Association/American Stroke Association. *Stroke*. 2009;40:2616–2634.
78. Derex L, Hermier M, Adeleine P, Pialat JB, Wiart M, Berthezène Y, Philippeau F, Honnorat J, Froment JC, Trouillas P, Nighoghossian N. Clinical and imaging predictors of intracerebral haemorrhage in stroke patients treated with intravenous tissue plasminogen activator. *J Neurol Neurosurg Psychiatry*. 2005;76:70–75.
79. Grond M, Stenzel C, Schmülling S, Rudolf J, Neveling M, Lechleuthner A, Schneeweis S, Heiss WD. Early intravenous thrombolysis for acute ischemic stroke in a community-based approach. *Stroke*. 1998;29:1544–1549.

80. Tanne D, Verro P, Mansbach H, Levine SR. Overview and summary of phase IV data on use of t-PA for acute ischemic stroke. *Stroke Interventionalist*. 1998;1:3–5.
81. Tanne D, Bates VE, Verro P, Kasner SE, Binder JR, Patel SC, Mansbach HH, Daley S, Schultz LR, Karanjia PN, Scott P, Dayno JM, Vereczkey-Porter K, Benesch C, Book D, Coplin WM, Dulli D, Levine SR; t-PA Stroke Survey Group. Initial clinical experience with IV tissue plasminogen activator for acute ischemic stroke: a multicenter survey. *Neurology*. 1999;53:424–427.
82. Trouillas P, Nighoghossian N, Getenet JC, Riche G, Neuschwander P, Froment JC, Turjman F, Jin JX, Malicier D, Fournier G, Gabry AL, Ledoux X, Derex L, Berthezène Y, Adeleine P, Xie J, Ffrench P, Dechavanne M. Open trial of intravenous tissue plasminogen activator in acute carotid territory stroke: correlations of outcome with clinical and radiological data. *Stroke*. 1996;27:882–890.
83. Wattigney WA, Croft JB, Mensah GA, Alberts MJ, Shephard TJ, Gorelick PB, Nilasena DS, Hess DC, Walker MD, Hanley DF Jr, Shwayder P, Girusus M, Neff LJ, Williams JE, LaBarthe DR, Collins JL; Paul Coverdell National Acute Stroke Registry. Establishing data elements for the Paul Coverdell National Acute Stroke Registry: part 1: proceedings of an expert panel. *Stroke*. 2003;34:151–156.
84. Molyneux A, Kerr R, Stratton I, Sandercock P, Clarke M, Shrimpton J, Holman R; International Subarachnoid Aneurysm Trial (ISAT) Collaborative Group. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. *Lancet*. 2002;360:1267–1274.
85. Bruno A, Saha C, Williams LS. Percent change on the National Institutes of Health Stroke Scale: a useful acute stroke outcome measure. *J Stroke Cerebrovasc Dis*. 2009;18:56–59.
86. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med*. 1991;325:445–453.
87. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA*. 1995;273:1421–1428.
88. SAMMPRIS: Stenting vs. Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis-ongoing trial. Available at: <http://www.strokecenter.org/trials/TrialDetail.aspx?tid=819>. Accessed November 5, 2009.
89. Hobson RW 2nd. CREST (Carotid Revascularization Endarterectomy versus Stent Trial): background, design, and current status. *Semin Vasc Surg*. 2000;13:139–143.
90. Furie KL, Kasner SE, Adams RJ, Albers GW, Bush RL, Fagan SC, Halperin JL, Johnston SC, Katzan I, Kernan WN, Mitchell PH, Ovbiagele B, Palesch YY, Sacco RL, Schwamm LH, Wassertheil-Smoller S, Turan TN, Wentworth D. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. October 21, 2010. DOI: 10.1161/STR.0b13e3181f7d043. Available at: <http://stroke.ahajournals.org/cgi/reprint/STR.0b13e3181f7d043>. Accessed December 19, 2010.
91. Goldstein L, Bushnell CD, Adams RJ, Appel LJ, Braun LT, Chaturvedi S, Creager MA, Culebras A, Eckel RH, Hart RG, Hinchey JA, Howard VJ, Jauch EC, Levine SR, Meschia JF, Moore WS, Nixon JV, Pearson TA. Guidelines for the primary prevention of stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association on behalf of the American Heart Association Stroke Council, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, Council on High Blood Pressure Research, Council on Peripheral Vascular Disease, and Council on Quality and Outcomes Research. *Stroke*. December 2, 2010. DOI: 10.1161/STR.0b13e3181f7d238. Available at: <http://stroke.ahajournals.org/cgi/reprint/STR.0b13e3181f7d238>. Accessed December 19, 2010.
92. Qureshi AI, Hutson AD, Harbaugh RE, Stieg PE, Hopkins LN; North American Trial of Unruptured and Ruptured Aneurysms Planning Committee. Methods and design considerations for randomized clinical trials evaluating surgical or endovascular treatments for cerebrovascular diseases. *Neurosurgery*. 2004;54:248–264.
93. Qureshi AI, Alexandrov AV, Tegeler CH, Hobson RW 2nd, Dennis Baker J, Hopkins LN. Guidelines for screening of extracranial carotid artery disease: a statement for healthcare professionals from the multi-disciplinary practice guidelines committee of the American Society of Neuroimaging: cosponsored by the Society of Vascular and Interventional Neurology. *J Neuroimaging*. 2007;17:19–47.
94. Brott TG, Hobson RW 2nd, Howard G, Roubin GS, Clark WM, Brooks W, Mackey A, Hill MD, Leimgruber PP, Sheffet AJ, Howard VJ, Moore WS, Voeks JH, Hopkins LN, Cutlip DE, Cohen DJ, Popma JJ, Ferguson RD, Cohen SN, Blackshear JL, Silver FL, Mohr JP, Lal BK, Meschia JF; CREST Investigators. Stenting versus endarterectomy for treatment of carotid-artery stenosis [published corrections appear in *N Engl J Med*. 2010;363:198 and *N Engl J Med*. 2010;363:498]. *N Engl J Med*. 2010;363:11–23.
95. Citron SJ, Wallace RC, Lewis CA, Dawson RC, Dion JE, Fox AJ, Manzione JV, Payne CS, Rivera FJ, Russell EJ, Sacks D, Yakes WF, Bakal CW; Society of Interventional Radiology; American Society of Interventional and Therapeutic Neuroradiology; American Society of Neuroradiology. Quality improvement guidelines for adult diagnostic neuroangiography: cooperative study between ASITN, ASNR, and SIR. *J Vasc Interv Radiol*. 2003;14(pt 2):S257–S262.
96. Bertrand ME, Legrand V, Boland J, Fleck E, Bonnier J, Emmanuelson H, Vrolix M, Missault L, Chierchia S, Casaccia M, Niccoli L, Oto A, White C, Webb-Peploe M, Van Belle E, McFadden EP. Randomized multicenter comparison of conventional anticoagulation versus antiplatelet therapy in unplanned and elective coronary stenting: the Full Anticoagulation Versus Aspirin and Ticlopidine (FANTASTIC) Study. *Circulation*. 1998;98:1597–1603.
97. Goods CM, al-Shaibi KF, Liu MW, Yadav JS, Mathur A, Jain SP, Dean LS, Iyer SS, Parks JM, Roubin GS. Comparison of aspirin alone versus aspirin plus ticlopidine after coronary artery stenting. *Am J Cardiol*. 1996;78:1042–1044.
98. Bhatt DL, Kapadia SR, Bajzer CT, Chew DP, Ziada KM, Mukherjee D, Roffi M, Topol EJ, Yadav JS. Dual antiplatelet therapy with clopidogrel and aspirin after carotid artery stenting. *J Invasive Cardiol*. 2001;13:767–771.
99. McKeivitt FM, Randall MS, Cleveland TJ, Gaines PA, Tan KT, Venables GS. The benefits of combined anti-platelet treatment in carotid artery stenting. *Eur J Vasc Endovasc Surg*. 2005;29:522–527.
100. SSVLIVA Study Investigators. Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSVLIVA): study results. *Stroke*. 2004;35:1388–1392.
101. Bose A, Hartmann M, Henkes H, Liu HM, Teng MM, Szikora I, Berlis A, Reul J, Yu SC, Forsting M, Lui M, Lim W, Sit SP. A novel, self-expanding, nitinol stent in medically refractory intracranial atherosclerotic stenoses: the Wingspan study. *Stroke*. 2007;38:1531–1537.
102. Fiorella D, Levy EI, Turk AS, Albuquerque FC, Niemann DB, Aagaard-Kienitz B, Hanel RA, Woo H, Rasmussen PA, Hopkins LN, Masaryk TJ, McDougall CG. US multicenter experience with the Wingspan stent system for the treatment of intracranial atheromatous disease: periprocedural results. *Stroke*. 2007;38:881–887.
103. Siddiq F, Vazquez G, Memon MZ, Suri MF, Taylor RA, Wojak JC, Chaloupka JC, Qureshi AI. Comparison of primary angioplasty with stent placement for treating symptomatic intracranial atherosclerotic diseases: a multicenter study. *Stroke*. 2008;39:2505–2510.
104. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg*. 1968;28:14–20.
105. Hemphill JC 3rd, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. *Stroke*. 2001;32:891–897.
106. Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. *J Neurosurg*. 1986;65:476–483.
107. Bederson JB, Connolly ES Jr, Batjer HH, Dacey RG, Dion JE, Diringer MN, Duldner JE Jr, Harbaugh RE, Patel AB, Rosenwasser RH. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association [published correction appears in *Stroke*. 2009;40:e518]. *Stroke*. 2009;40:994–1025.
108. Rosen DS, Macdonald RL. Subarachnoid hemorrhage grading scales: a systematic review. *Neurocrit Care*. 2005;2:110–118.
109. Aulmann C, Steudl WI, Feldmann U. Validation of the prognostic accuracy of neurosurgical admission scales after rupture of cerebral aneurysms [in German]. *Zentralbl Neurochir*. 1998;59:171–180.
110. Braun V, Rath S, Antoniadis G, Richter HP, Börm W. Treatment and outcome of aneurysmal subarachnoid haemorrhage in the elderly patient. *Neuroradiology*. 2005;47:215–221.
111. Cedzich C, Roth A. Neurological and psychosocial outcome after subarachnoid haemorrhage, and the Hunt and Hess scale as a predictor of clinical outcome. *Zentralbl Neurochir*. 2005;66:112–118.
112. Oshiro EM, Walter KA, Piantadosi S, Witham TF, Tamargo RJ. A new subarachnoid hemorrhage grading system based on the Glasgow Coma Scale: a comparison with the Hunt and Hess and World Federation of

- Neurological Surgeons Scales in a clinical series. *Neurosurgery*. 1997;41:140–147.
113. Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, Khoury J. The ABCs of measuring intracerebral hemorrhage volumes. *Stroke*. 1996;27:1304–1305.
  114. Morgenstern LB, Hemphill JC 3rd, Anderson C, Becker K, Broderick JP, Connolly ES Jr, Greenberg SM, Huang JN, MacDonald RL, Messe SR, Mitchell PH, Selim M, Tamargo RJ; on behalf of the American Heart Association Stroke Council and Council on Cardiovascular Nursing. Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2010;41:2108–2129.
  115. Clarke JL, Johnston SC, Farrant M, Bernstein R, Tong D, Hemphill JC 3rd. External validation of the ICH score. *Neurocrit Care*. 2004;1:53–60.
  116. Del Brutto OH, Campos X. Validation of intracerebral hemorrhage scores for patients with pontine hemorrhage. *Neurology*. 2004;62:515–516.
  117. Hemphill JC 3rd, Farrant M, Neill TA Jr. Prospective validation of the ICH Score for 12-month functional outcome. *Neurology*. 2009;73:1088–1094.
  118. Andrade-Souza YM, Zadeh G, Ramani M, Scora D, Tsao MN, Schwartz ML. Testing the radiosurgery-based arteriovenous malformation score and the modified Spetzler-Martin grading system to predict radiosurgical outcome. *J Neurosurg*. 2005;103:642–648.
  119. Zhao J, Wang S, Li J, Qi W, Sui D, Zhao Y. Clinical characteristics and surgical results of patients with cerebral arteriovenous malformations. *Surg Neurol*. 2005;63:156–161.
  120. Fisher CM, Roberson GH, Ojemann RG. Cerebral vasospasm with ruptured saccular aneurysm: the clinical manifestations. *Neurosurgery*. 1977;1:245–248.
  121. Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale. *J Neurosurg*. 1988;68:985–986.
  122. Chyatte D, Fode NC, Sundt TM Jr. Early versus late intracranial aneurysm surgery in subarachnoid hemorrhage. *J Neurosurg*. 1988;69:326–331.
  123. Kassell NF, Drake CG. Timing of aneurysm surgery. *Neurosurgery*. 1982;10:514–519.
  124. Kassell NF, Torner JC, Jane JA, Haley EC Jr, Adams HP. The International Cooperative Study on the Timing of Aneurysm Surgery: part 2: surgical results. *J Neurosurg*. 1990;73:37–47.
  125. Mayberg MR, Batjer HH, Dacey R, Diringer M, Haley EC, Heros RC, Sternau LL, Torner J, Adams HP Jr, Feinberg W, Thies W. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke*. 1994;25:2315–2328.
  126. Allen GS, Ahn HS, Preziosi TJ, Battye R, Boone SC, Chou SN, Kelly DL, Weir BK, Crabbe RA, Lavik PJ, Rosenbloom SB, Dorsey FC, Ingram CR, Mellits DE, Bertsch LA, Boisvert DP, Hundley MB, Johnson RK, Strom JA, Transou CR. Cerebral arterial spasm: a controlled trial of nimodipine in patients with subarachnoid hemorrhage. *N Engl J Med*. 1983;308:619–624.
  127. Ohman J, Heiskanen O. Timing of operation for ruptured supratentorial aneurysms: a prospective randomized study. *J Neurosurg*. 1989;70:55–60.
  128. US Food and Drug Administration. NDA 18-869/S-014. FDA approved Labeling text. Nimotop® (nimodipine) Capsules for oral use. Available at: [http://www.accessdata.fda.gov/drugsatfda\\_docs/label/2006/018869s014lbl.pdf](http://www.accessdata.fda.gov/drugsatfda_docs/label/2006/018869s014lbl.pdf). Accessed December 20, 2010.
  129. Schievink WI, Wijidicks EF, Piepgras DG, Chu CP, O'Fallon WM, Whisnant JP. The poor prognosis of ruptured intracranial aneurysms of the posterior circulation. *J Neurosurg*. 1995;82:791–795.
  130. Rajshekhkar V, Harbaugh RE. Results of routine ventriculostomy with external ventricular drainage for acute hydrocephalus following subarachnoid haemorrhage. *Acta Neurochir (Wien)*. 1992;115:8–14.
  131. Hasan D, Vermeulen M, Wijidicks EF, Hijdra A, van Gijn J. Management problems in acute hydrocephalus after subarachnoid hemorrhage. *Stroke*. 1989;20:747–753.
  132. Ransom ER, Mocco J, Komotar RJ, Sahni D, Chang J, Hahn DK, Kim GH, Schmidt JM, Sciacca RR, Mayer SA, Connolly ES. External ventricular drainage response in poor grade aneurysmal subarachnoid hemorrhage: effect on preoperative grading and prognosis. *Neurocrit Care*. 2007;6:174–180.
  133. Haley EC Jr, Kassell NF, Torner JC. The International Cooperative Study on the Timing of Aneurysm Surgery: the North American experience. *Stroke*. 1992;23:205–214.
  134. Lysakowski C, Walder B, Costanza MC, Tramèr MR. Transcranial Doppler versus angiography in patients with vasospasm due to a ruptured cerebral aneurysm: a systematic review. *Stroke*. 2001;32:2292–2298.
  135. Murayama Y, Malisch T, Guglielmi G, Mawad ME, Viñuela F, Duckwiler GR, Gobin YP, Klucznick RP, Martin NA, Frazee J. Incidence of cerebral vasospasm after endovascular treatment of acutely ruptured aneurysms: report on 69 cases. *J Neurosurg*. 1997;87:830–835.
  136. Sloan MA, Alexandrov AV, Tegeler CH, Spencer MP, Caplan LR, Feldmann E, Wechsler LR, Newell DW, Gomez CR, Babikian VL, Lefkowitz D, Goldman RS, Armon C, Hsu CY, Goodin DS. Assessment: transcranial Doppler ultrasonography: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*. 2004;62:1468–1481.
  137. Wardlaw JM, Offin R, Teasdale GM, Teasdale EM. Is routine transcranial Doppler ultrasound monitoring useful in the management of subarachnoid hemorrhage? *J Neurosurg*. 1998;88:272–276.
  138. The Joint Commission. Performance measurement and improvement for disease-specific care certification programs. Available at: [http://www.jointcommission.org/performance\\_measurement\\_and\\_improvement\\_for\\_disease-specific\\_care\\_certification\\_programs/](http://www.jointcommission.org/performance_measurement_and_improvement_for_disease-specific_care_certification_programs/). Accessed December 20, 2010.
  139. Qureshi AI, Janardhan V, Hanel RA, Lanzino G. Comparison of endovascular and surgical treatments for intracranial aneurysms: an evidence-based review. *Lancet Neurol*. 2007;6:816–825.
  140. Qureshi AI, Janardhan V, Memon MZ, Suri MF, Shah QA, Miley JT, Puchta AE, Taylor RA. Initial experience in establishing an academic neuroendovascular service: program building, procedural types, and outcomes. *J Neuroimaging*. 2009;19:72–79.
  141. Molyneux AJ, Kerr RS, Yu LM, Clarke M, Sneade M, Yarnold JA, Sandercock P; International Subarachnoid Aneurysm Trial (ISAT) Collaborative Group. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. *Lancet*. 2005;366(9488):809–817.
  142. Vanninen R, Koivisto T, Saari T, Hernesniemi J, Vapalahti M. Ruptured intracranial aneurysms: acute endovascular treatment with electrolytically detachable coils: a prospective randomized study. *Radiology*. 1999;211:325–336.
  143. Pelz DM, Lownie SP, Fox AJ. Thromboembolic events associated with the treatment of cerebral aneurysms with Guglielmi detachable coils. *AJNR Am J Neuroradiol*. 1998;19:1541–1547.
  144. Sakai H, Sakai N, Higashi T, Iihara K, Takahashi J, Kogure S, Anei R, Yamada N, Imakita S, Nagata I. Embolic complications associated with neurovascular intervention: prospective evaluation by use of diffusion-weighted MR imaging [in Japanese]. *No Shinkei Geka*. 2002;30:43–49.
  145. Soeda A, Sakai N, Sakai H, Iihara K, Yamada N, Imakita S, Nagata I. Thromboembolic events associated with Guglielmi detachable coil embolization of asymptomatic cerebral aneurysms: evaluation of 66 consecutive cases with use of diffusion-weighted MR imaging. *AJNR Am J Neuroradiol*. 2003;24:127–132.
  146. Fraser JF, Riina H, Mitra N, Gobin YP, Simon AS, Stieg PE. Treatment of ruptured intracranial aneurysms: looking to the past to register the future. *Neurosurgery*. 2006;59:1157–1166.
  147. Flibotte JJ, Hagan N, O'Donnell J, Greenberg SM, Rosand J. Warfarin, hematoma expansion, and outcome of intracerebral hemorrhage. *Neurology*. 2004;63:1059–1064.
  148. Aguilar MI, Hart RG, Kase CS, Freeman WD, Hoeben BJ, Garcia RC, Ansell JE, Mayer SA, Norrving B, Rosand J, Steiner T, Wijidicks EF, Yamaguchi T, Yasaka M. Treatment of warfarin-associated intracerebral hemorrhage: literature review and expert opinion [published correction appears in *Mayo Clin Proc*. 2007;82:387]. *Mayo Clin Proc*. 2007;82:82–92.
  149. Freeman WD, Aguilar MI. Management of warfarin-related intracerebral hemorrhage. *Expert Rev Neurother*. 2008;8:271–290.
  150. Goldstein JN, Rosand J, Schwamm LH. Warfarin reversal in anticoagulant-associated intracerebral hemorrhage. *Neurocrit Care*. 2008;9:277–283.
  151. Guidelines on oral anticoagulation: third edition. *Br J Haematol*. 1998;101:374–387.
  152. Baker RI, Coughlin PB, Gallus AS, Harper PL, Salem HH, Wood EM; Warfarin Reversal Consensus Group. Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis [published correction appears in *Med J Aust*. 2005;182:48]. *Med J Aust*. 2004;181:492–497.

153. Lankiewicz MW, Hays J, Friedman KD, Tinkoff G, Blatt PM. Urgent reversal of warfarin with prothrombin complex concentrate. *J Thromb Haemost.* 2006;4:967–970.
154. Lindley CM, Sawyer WT, Macik BG, Lusher J, Harrison JF, Baird-Cox K, Birch K, Glazer S, Roberts HR. Pharmacokinetics and pharmacodynamics of recombinant factor VIIa. *Clin Pharmacol Ther.* 1994;55:638–648.
155. Makris M, Greaves M, Phillips WS, Kitchen S, Rosendaal FR, Preston EF. Emergency oral anticoagulant reversal: the relative efficacy of infusions of fresh frozen plasma and clotting factor concentrate on correction of the coagulopathy. *Thromb Haemost.* 1997;77:477–480.
156. Raj G, Kumar R, McKinney WP. Time course of reversal of anticoagulant effect of warfarin by intravenous and subcutaneous phytonadione [published correction appears in *Arch Intern Med.* 2000;160:986]. *Arch Intern Med.* 1999;159:2721–2724.
157. Schulman S. Clinical practice: care of patients receiving long-term anticoagulant therapy. *N Engl J Med.* 2003;349:675–683.
158. Steiner T, Rosand J, Diringer M. Intracerebral hemorrhage associated with oral anticoagulant therapy: current practices and unresolved questions. *Stroke.* 2006;37:256–262.
159. The ARUBA Trial. Available at: <http://www.arubastudy.org/frameSetDescripTrial.html>. Accessed June 3, 2010.
160. Ogilvy CS, Stieg PE, Awad I, Brown RD Jr, Kondziolka D, Rosenwasser R, Young WL, Hademenos G. AHA scientific statement: recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. *Stroke.* 2001;32:1458–1471.
161. n-BCA Trial Investigators. N-butyl cyanoacrylate embolization of cerebral arteriovenous malformations: results of a prospective, randomized, multi-center trial. *AJNR Am J Neuroradiol.* 2002;23:748–755.
162. Diringer MN, Edwards DF. Admission to a neurologic/neurosurgical intensive care unit is associated with reduced mortality rate after intracerebral hemorrhage. *Crit Care Med.* 2001;29:635–640.
163. Mirski MA, Chang CW, Cowan R. Impact of a neuroscience intensive care unit on neurosurgical patient outcomes and cost of care: evidence-based support for an intensivist-directed specialty ICU model of care. *J Neurosurg Anesthesiol.* 2001;13:83–92.
164. Suarez JI, Zaidat OO, Suri MF, Feen ES, Lynch G, Hickman J, Georgiadis A, Selman WR. Length of stay and mortality in neurocritically ill patients: impact of a specialized neurocritical care team. *Crit Care Med.* 2004;32:2311–2317.
165. Varelas PN, Conti MM, Spanaki MV, Potts E, Bradford D, Sunstrom C, Fedder W, Haccin Bey L, Jaradeh S, Gennarelli TA. The impact of a neurointensivist-led team on a semiclosed neurosciences intensive care unit. *Crit Care Med.* 2004;32:2191–2198.
166. Bershad EM, Feen ES, Hernandez OH, Suri MF, Suarez JI. Impact of a specialized neurointensive care team on outcomes of critically ill acute ischemic stroke patients. *Neurocrit Care.* 2008;9:287–292.
167. Varelas PN, Schultz L, Conti M, Spanaki M, Genarrelli T, Haccin-Bey L. The impact of a neuro-intensivist on patients with stroke admitted to a neurosciences intensive care unit. *Neurocrit Care.* 2008;9:293–299.
168. Beer R, Pfausler B, Schmutzhard E. Management of nosocomial external ventricular drain-related ventriculomegnitis. *Neurocrit Care.* 2009;10:363–367.
169. Lozier AP, Sciacca RR, Romagnoli MF, Connolly ES Jr. Ventriculostomy-related infections: a critical review of the literature. *Neurosurgery.* 2002;51:170–181.
170. Management of Stroke Rehabilitation. Washington, DC: Department of Veterans Affairs and Health Affairs Department of Defense; 2003. Office of Quality and Performance publication No. 10Q CPG/STR-03.
171. Cifu DX, Stewart DG. Factors affecting functional outcome after stroke: a critical review of rehabilitation interventions. *Arch Phys Med Rehabil.* 1999;80(suppl 1):S35–S39.
172. Teasell RW, Foley NC, Bhogal SK, Speechley MR. An evidence-based review of stroke rehabilitation. *Top Stroke Rehabil.* 2003;10:29–58.
173. Arias M, Smith LN. Early mobilization of acute stroke patients. *J Clin Nurs.* 2007;16:282–288.
174. Langhorne P, Tong BL, Stott DJ. Association between physiological homeostasis and early recovery after stroke. *Stroke.* 2000;31:2518–2519.
175. Asplund K, Stegmayr, B, Peltonen M. From the twentieth to the twenty-first century: a public health perspective on stroke. In: Ginsberg MD, Bogouslavsky J, eds. *Cerebrovascular Disease: Pathophysiology, Diagnosis, and Management.* Vol 2. Malden, MA: Blackwell Science; 1998.
176. Gresham GE, Alexander D, Bishop DS, Giuliani C, Goldberg G, Holland A, Kelly-Hayes M, Linn RT, Roth EJ, Stason WB, Trombly CA. American Heart Association Prevention Conference: IV: prevention and rehabilitation of stroke: rehabilitation. *Stroke.* 1997;28:1522–1526.
177. Indredavik B, Bakke F, Slordahl SA, Rokseth R, Håheim LL. Stroke unit treatment: 10-year follow-up. *Stroke.* 1999;30:1524–1527.
178. Indredavik B, Bakke F, Solberg R, Rokseth R, Haaheim LL, Holme I. Benefit of a stroke unit: a randomized controlled trial. *Stroke.* 1991;22:1026–1031.
179. Indredavik B, Slørdahl SA, Bakke F, Rokseth R, Håheim LL. Stroke unit treatment: long-term effects. *Stroke.* 1997;28:1861–1866.
180. Jørgensen HS, Nakayama H, Raaschou HO, Larsen K, Hübbe P, Olsen TS. The effect of a stroke unit: reductions in mortality, discharge rate to nursing home, length of hospital stay, and cost: a community-based study. *Stroke.* 1995;26:1178–1182.
181. Kaste M, Skyhøj Olsen T, Orgogozo J, Bogousslavsky J, Hacke W. Organization of stroke care: education, stroke units and rehabilitation: European Stroke Initiative (EUSI). *Cerebrovasc Dis.* 2000;10(suppl 3):1–11.
182. Stroke Unit Trialists' Collaboration. Collaborative systematic review of the randomised trials of organised inpatient (stroke unit) care after stroke. *BMJ.* 1997;314:1151–1159.
183. Lorish TR. Stroke rehabilitation. *Clin Geriatr Med.* 1993;9:705–716.
184. Sacco RL, Benjamin EJ, Broderick JP, Dyken M, Easton JD, Feinberg WM, Goldstein LB, Gorelick PB, Howard G, Kittner SJ, Manolio TA, Whisnant JP, Wolf PA. American Heart Association Prevention Conference: IV: prevention and rehabilitation of stroke: risk factors. *Stroke.* 1997;28:1507–1517.
185. Helgason CM, Wolf PA. American Heart Association Prevention Conference IV: prevention and rehabilitation of stroke: executive summary. *Circulation.* 1997;96:701–707.
186. Indredavik B, Bakke F, Slordahl SA, Rokseth R, Håheim LL. Stroke unit treatment improves long-term quality of life: a randomized controlled trial. *Stroke.* 1998;29:895–899.
187. Strand T, Asplund K, Eriksson S, Hägg E, Lithner F, Wester PO. A non-intensive stroke unit reduces functional disability and the need for long-term hospitalization. *Stroke.* 1985;16:29–34.
188. Legg L, Langhorne P; Outpatient Service Trialists. Rehabilitation therapy services for stroke patients living at home: systematic review of randomised trials. *Lancet.* 2004;363:352–356.
189. SPOTRIAS (Specialized Program of Translational Research in Acute Stroke). Available at: <http://www.spotrias.com/Default.aspx>. Accessed October 14, 2009.
190. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, Amelink GJ, Schmiedeck P, Schwab S, Rothwell PM, Boussier MG, van der Worp HB, Hacke W; DECIMAL, DESTINY, and HAMLET Investigators. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. *Lancet Neurol.* 2007;6:215–222.
191. Adams HP Jr, Davis PH, Leira EC, Chang KC, Bendixen BH, Clarke WR, Woolson RF, Hansen MD. Baseline NIH Stroke Scale score strongly predicts outcome after stroke: a report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology.* 1999;53:126–131.
192. Smith EE, Shobha N, Dai D, Olson DM, Reeves MJ, Saver JL, Hernandez AF, Peterson ED, Fonarow GC, Schwamm LH. Risk score for in-hospital ischemic stroke mortality derived and validated within the Get With the Guidelines-Stroke Program. *Circulation.* 2010;122:1496–1504.

## Metrics for Measuring Quality of Care in Comprehensive Stroke Centers: Detailed Follow-Up to Brain Attack Coalition Comprehensive Stroke Center Recommendations: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

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