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**Guidelines for the Early Management of Patients With Ischemic Stroke: 2005
Guidelines Update A Scientific Statement From the Stroke Council of the
American Heart Association/American Stroke Association**

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Guidelines for the Early Management of Patients With Ischemic Stroke

2005 Guidelines Update

A Scientific Statement From the Stroke Council of the American Heart Association/American Stroke Association

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This article serves as an update of “Guidelines for the Early Management of Patients With Ischemic Stroke,” published in *Stroke* in 2003 (<http://stroke.ahajournals.org/cgi/content/full/34/4/1056>). This update is intended to reflect advances in the field since the publication of the full guidelines. See Tables 1 and 2, reprinted in this article from the 2003 document, for explanations of grade (strength of recommendation).

Brain Imaging

CT remains the most widely used neuroimaging technique for the evaluation of patients with suspected acute ischemic stroke. Quantitative CT-based scoring systems (eg, the Alberta Stroke Program Early CT Score [ASPECTS]) are useful for identifying patients who are unlikely to recover fully despite thrombolytic therapy.¹ Substantial agreement between the ASPECTS rating performed in real time and the score obtained later by an expert can be achieved when used by an experienced reader, but correlations are not perfect (weighted κ 0.69, 95% CI 0.59 to 0.79).² This scoring system has not been assessed in general clinical practice and is limited to use in patients with infarctions suspected to be in the distribution of the middle cerebral artery. In addition, advances in CT technology, including the development of CT angiography and perfusion studies, may affect future recommendations about the use of CT in the evaluation of patients with suspected stroke.

MRI techniques also are used widely in the assessment of patients with suspected stroke or transient ischemic attack (TIA). For example, a retrospective analysis of patients having diffusion-weighted MRI studies within 3 days of TIA

demonstrated relevant abnormalities in 21% of cases.³ Changes in \approx 44% of cases are detected by T2-weighted or fluid attenuation inversion recovery MRI studies.

A scientific statement authored by a panel of the American Heart Association focused on perfusion imaging in the setting of acute ischemic stroke was published simultaneously with the original 2003 ischemic stroke guidelines.⁴ Information about the advantages and disadvantages of each imaging technique is included in the statement. The panel concluded that more comparison testing of the different techniques is needed to determine their relative abilities to differentiate tissues having normal perfusion and reversible or irreversible ischemic injury. Clinical trials must determine whether perfusion data help forecast outcomes after stroke and the ability to triage patients to specific interventions.

The 2003 ischemic stroke guidelines indicated that additional research was needed to determine the utility of MRI as a substitute for CT among patients with suspected acute stroke because detection of acute intracerebral hemorrhage via MRI had not been fully validated. A study addressing this need has been reported. In comparison with CT, MRI detected intracranial bleeding with 100% sensitivity and 100% accuracy, as identified by 3 experienced readers. Three medical students also interpreted the studies with a sensitivity of 95%. Additional studies have produced similar results.^{5,6} These results suggest that MRI may replace CT in the initial screening for hemorrhage among patients with suspected stroke. Additional experience for detection in the acute setting in real time and outside specialized academic centers in the United States is needed. Besides its utility in the diagnosis of acute brain ischemia, MRI also may help in

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TABLE 1. Levels of Evidence

Level of evidence	
Level I	Data from randomized trials with low false-positive and low false-negative errors
Level II	Data from randomized trials with high false-positive or high false-negative errors
Level III	Data from nonrandomized concurrent cohort studies
Level IV	Data from nonrandomized cohort studies using historical controls
Level V	Data from anecdotal case series
Strength of recommendation	
Grade A	Supported by level I evidence
Grade B	Supported by level II evidence
Grade C	Supported by level III, IV, or V evidence

identifying patients with previous microhemorrhages that could be associated with an increased risk of bleeding secondary to thrombolysis.^{7,8}

MRI with susceptibility-weighted imaging may be useful in detecting areas of hemorrhage after intraarterial thrombolysis in situations in which CT findings could be equivocal because of residual contrast staining.⁹ The importance of this finding needs clarification. Prospective studies are needed to determine whether the findings of susceptibility-weighted MRI affect either prognosis or treatment.

Brain imaging is required to guide the selection of acute interventions to treat patients with stroke (grade A, no change from 2003). For most cases and at most institutions, CT remains the most important brain imaging test; however, new studies suggest that MRI also may be used to detect acute intracerebral hemorrhage and that it could be an alternative to CT. Additional studies are under way. There is general agreement that perfusion and diffusion-weighted MRI may be helpful in diagnosing and treating patients with acute stroke under some circumstances, but logistical issues, including the availability of the equipment and the presence of physicians with expertise in interpreting the tests, limit the use of MRI. At present, no data are available to show

that MRI is superior to CT for selecting patients who could be treated with intravenous recombinant tissue plasminogen activator (rtPA). The use of MRI outside the setting of clinical research studies should not delay treatment of a patient who is otherwise eligible for treatment with intravenous rtPA (grade B, no change from 2003).

Treatment of Arterial Hypertension

The treatment of arterial hypertension immediately after stroke is problematic, as stated in the 2003 guidelines. Since then, a placebo-controlled phase II safety trial tested the utility of candesartan administered from day 1 to hypertensive patients with acute ischemic stroke.¹⁰ At 12 months, patients treated with candesartan had improved survival and few subsequent vascular events. No differences in blood pressure values were noted, however, and the effects on the outcome of the stroke are not described. This preliminary observation must be confirmed by a larger clinical trial.

Pharmacological (Intravenous or Intraarterial) Thrombolysis

Symptomatic hemorrhagic transformation of the infarction remains the primary concern with the administration of intravenous rtPA in the treatment of acute ischemic stroke.¹¹ A recent pooled analysis of several trials of rtPA confirms that symptomatic hemorrhagic transformation is the primary complication of acute treatment with rtPA.¹² A meta-analysis of the postmarketing open-label studies demonstrates that the risk of hemorrhage is $\approx 5.2\%$.¹³ A subsequent report by the same group demonstrated a marked decline in major bleeding complications when the guidelines were followed.¹⁴ Schmulling et al¹⁵ found that previous use of aspirin does not increase the risk of symptomatic intracranial bleeding after the administration of rtPA. The studies show that rtPA can be given with an acceptable margin of safety in a community setting when the guidelines for selection and treatment of patients are followed.¹³

Hill et al¹⁶ reported orolingual angioedema in 9 of 176 patients treated with intravenous rtPA. In most cases, the findings were mild, transient, and contralateral to the in-

TABLE 2. Quality of Evidence Ratings for Radiological Diagnostic Tests

Level of evidence	
Class A	Evidence provided by a prospective study in a broad spectrum of persons with the suspected condition, using a "gold standard" for case definition, where test is applied in a blinded evaluation, and enabling the assessment of the appropriate tests of diagnostic accuracy.
Class B	Evidence provided by a prospective study of a narrow spectrum of persons with a suspected condition, or a well-designed retrospective study of a broad spectrum of persons with an established condition (by the "gold standard") is compared to a broad spectrum of controls, where test is applied evaluation and enabling the assessment of appropriate tests of diagnostic accuracy.
Class C	Evidence supplied by a retrospective study where either persons with an established condition or controls are of a narrow spectrum, and where test is applied in a blinded evaluation.
Class D	Any design where test is not applied in blinded evaluation OR evidence provided by expert opinion alone or in descriptive case series (without controls).
Strength of recommendation	
Grade I	Established as useful/predictive or not useful/predictive for the given condition in the specified population.
Grade II	Probably useful/predictive or not useful/predictive for the given condition in the specified population.
Grade III	Possibly useful/predictive or not useful/predictive for the given condition in the specified population.
Grade IV	Data are inadequate or conflicting. Given current knowledge, the test/predictor is unproven.

volved cerebral hemisphere. They noted that the likelihood was increased among patients who were taking angiotensin-converting enzyme inhibitors and among those who had evidence of ischemia in the frontal cortex and insula on CT. Other cases of more severe edema of the throat and mouth also have been described.^{17–19} Although the previous use of angiotensin-converting enzyme inhibitors is not a contraindication for the administration of rtPA, physicians should be aware of this potential complication. Presumably, medications used to treat angioedema would be indicated to treat a severely affected patient.

A recent report offered a pooled analysis of data from several clinical trials of rtPA.¹² The data from each of these trials have been reported independently. Although the trials used different definitions of outcomes, the combined analysis applied definitions used in the National Institute of Neurological Disorders and Stroke trials (eg, no or minimal disability at 3 months as measured by modified Rankin Scale, the Barthel Index, and the National Institutes of Health [NIH] Stroke Scale) plus a global statistical test. The lower 95% confidence limit for the adjusted odds ratio for a favorable outcome crossed unity at 4.5 hours from symptom onset. This finding suggests that some patients may benefit from treatment beyond the current 3-hour window; however, additional information is necessary to move the maximal time window to 4.5 hours in the guidelines. Ongoing studies are evaluating the potential utility of rtPA given >3 hours after the onset of stroke.

Hsia et al²⁰ found that the subtypes of ischemic stroke do not influence responses to treatment with rtPA. This finding implies that the determination of the subtype of stroke (eg, cardioembolism, large artery atherosclerosis, or small artery occlusion) is not a prerequisite for the administration of rtPA.

Intraarterial administration of thrombolytic agents has considerable appeal.²¹ A review of the available data shows that intraarterial thrombolysis is associated with a reduction in mortality and an improvement in favorable outcomes after a stroke, but it is also associated with an increased risk of hemorrhagic complications.²² Additional studies have been published since the development of the 2003 guidelines. In general, the results are similar to those published previously.^{23–26} Studies testing the utility of intraarterial thrombolysis are ongoing. Recommendations for the design and organization of such trials were published recently.²⁷ At present, no evidence is available to show that intraarterial thrombolysis is superior to intravenous treatment. Therapy should not be withheld from patients who are eligible for treatment with intravenous thrombolysis so that medications can be administered intraarterially, except in the setting of a comparative research clinical trial.

The combination of administering intravenous therapy and then intraarterial therapy is being tested. This strategy could allow for early treatment of stroke with intravenous medication while the resources to deliver intraarterial therapy are organized.^{21,28,29} Additional reports that have become available since the 2003 guidelines reflect mixed results.^{30,31} Clinical trials testing the utility of the combination of intravenous and intraarterial therapy are in progress, and addi-

tional data are needed to support a recommendation for combination treatment.

Using transcranial Doppler ultrasonography, Alexandrov and Grotta³² found that approximately one third of patients develop reocclusion of the artery after intravenous thrombolysis. Patients with partial recanalization were the most likely to experience reocclusion and poorer neurological outcomes. These results are stimulating research on adjunctive antithrombotic therapies that help maintain arterial patency. Among the interventions are anticoagulants and rapidly acting parenterally administered antiplatelet agents.^{15,33–36} Although preliminary results are promising, experience is limited. Additional data are needed before changing the current recommendations to withhold adjunctive antithrombotic therapy for the first 24 hours after administration of rtPA.

Because of the current time requirements for the administration of rtPA, all aspects of the healthcare system must respond with a sense of urgency. Community-wide stroke programs are increasing the number of patients that can be treated.^{37–39} Delays within the hospital emergency department also need to be addressed.³⁹ Telemedicine and emergency air transportation are among the ways to speed the treatment of patients with acute stroke.^{40,41}

Novel thrombolytic agents such as desmoteplase, reteplase, and tenecteplase are being evaluated, but prospective data comparing these drugs with intravenous rtPA are few. Although experience is limited, thrombolytic agents have been given successfully to children with acute ischemic stroke.⁴²

Recommendations

The recommendation for the intravenous administration of rtPA within 3 hours of onset of stroke in carefully selected patients should not be changed (grade A, no change from 2003). The evidence is strong that all delays in treating patients should be avoided (grade A, new recommendation). Although intraarterial thrombolysis alone or in combination with intravenous thrombolysis holds great promise, the use of these approaches is preferable in the setting of randomized clinical trials. A correction is needed in Table 7 of the 2003 Guidelines. Patients with an INR level of 1.7 or below can be treated with rtPA.

Anticoagulants

Current data do not provide evidence in support of the efficacy of early anticoagulation in improving outcomes after acute ischemic stroke.⁴³ The recommendations of the 2003 guidelines are in agreement with other statements indicating that most stroke patients do not need emergency administration of anticoagulants.^{44–46} Despite the lack of supporting data, anticoagulants are still given frequently.⁴⁷

A preliminary clinical study of argatroban has been completed and the agent was deemed to be safe.⁴⁸ Burak et al⁴⁹ administered enoxaparin to 8 children with stroke and concluded that the low-molecular-weight heparin was a safe and effective alternative to heparin for children. Anticoagulants also are being explored as an adjunct to thrombolytic therapy.¹⁵ Although the preponderance of past acute anticoagula-

TABLE 7. Characteristics of Patients With Ischemic Stroke Who Could Be Treated With rtPA

Diagnosis of ischemic stroke causing measurable neurological deficit
The neurological signs should not be clearing spontaneously
The neurological signs should not be minor and isolated
Caution should be exercised in treating a patient with major deficits
The symptoms of stroke should not be suggestive of subarachnoid hemorrhage
Onset of symptoms <3 hours before beginning treatment
No head trauma or prior stroke in previous 3 months
No myocardial infarction in the previous 3 months
No gastrointestinal or urinary tract hemorrhage in previous 21 days
No major surgery in the previous 14 days
No arterial puncture at a noncompressible site in the previous 7 days
No history of previous intracranial hemorrhage
Blood pressure not elevated (systolic <185 mm Hg and diastolic <110 mm Hg)
No evidence of active bleeding or acute trauma (fracture) on examination
Not taking an oral anticoagulant or if anticoagulant being taken, INR ≤1.7
If receiving heparin in previous 48 hours, aPTT must be in normal range
Platelet count ≥100 000 mm ³
Blood glucose concentration ≥50 mg/dL (2.7 mmol/L)
No seizure with postictal residual neurological impairments
CT does not show a multilobar infarction (hypodensity >1/3 cerebral hemisphere)
The patient or family understand the potential risks and benefits from treatment

tion trials has failed to show a benefit, newer clinical trials testing heparin and other anticoagulants continue.

Recommendations

No data are available to support changing the recommendations about the use of anticoagulants in the urgent treatment of patients with acute ischemic stroke.

Antiplatelet Aggregating Agents

Since the publication of the 2003 guidelines, Roden-Jullig et al⁵⁰ have reported the results of a placebo-controlled trial of aspirin (325 mg/day) for the treatment of patients with stroke. The trial enrolled 441 patients (220 took aspirin) within 48 hours of the onset of stroke. Patients were treated for 5 days; no significant reduction in the rate of neurological worsening was noted. No differences in outcomes were noted at 3 months. This study was underpowered to detect the mild beneficial effects of aspirin identified in earlier megatrials. A small study found that the combination of aspirin and a low-molecular-weight heparin did not improve outcomes after stroke.⁵¹

Other rapidly acting antiplatelet agents are being evaluated for their usefulness in treating patients with stroke. These agents are being administered as a monotherapy or in combination with thrombolysis.^{33–36} In a placebo-controlled study, abciximab was administered within 6 hours of the onset of stroke. The results have been presented in abstract

form and are apparently promising but have not been published.

Recommendations

Although the new data do not change the recommendation that most patients should receive aspirin within 48 hours of stroke, the data also support the conclusion that the effects of aspirin are modest (grade A, no change from 2003). Aspirin should not be considered as an alternative to intravenous thrombolysis or acute therapies aimed at improving outcomes after stroke. Additional research on abciximab or other rapidly acting antiplatelet agents is needed before any recommendation about their use can be made.

Volume Expansion and Drug-Induced Hypertension

Medical measures to improve cerebral blood flow are being evaluated. In addition to its ability to improve flow to the ischemic region, albumin may have neuroprotective effects and is being tested. In a pilot study, Hillis et al⁵² found that drug-induced hypertension can improve blood flow and lessen the neurological consequences of stroke. This regimen has been used to treat patients with vasospasm after subarachnoid hemorrhage. Although drug-induced hypertension holds promise, this therapy may be associated with an increased risk of brain edema, hypertensive encephalopathy, or hemorrhagic transformation of the infarction. Additional vasopressor-related complications may include cardiac ischemia or arrhythmias. The intervention also may require admission to an intensive care unit and close monitoring. Further testing of drug-induced hypertension is in progress.

Recommendations

At present, drug-induced hypertension cannot be recommended for the treatment of most patients with ischemic stroke (grade A, new recommendation).

Surgical and Endovascular Procedures

Gay et al⁵³ successfully performed carotid endarterectomy in 21 patients with acute ischemic symptoms. In another study of 67 patients, emergency carotid endarterectomy achieved recanalization in all but 5 cases.⁵⁴ The patients who were selected for surgery had normal preoperative flow in the middle cerebral artery. The aim was to avoid performing surgery on the internal carotid artery if an ipsilateral embolic occlusion of the middle cerebral artery had already occurred. Another study found that the presence of a diffusion/perfusion mismatch could be used to help select patients for surgery.⁵⁵

Endovascular and adjunctive mechanical thrombolytic methods include lasers, intraarterial suction devices, snares, angioplasty, and clot-retrieval devices.^{56,57} In some cases, these devices have been used in conjunction with pharmacological thrombolysis.⁵⁸ In addition, therapeutic ultrasonography has been used to help break fibrin monomers, dissolve thrombi, and improve recanalization.^{59,60} Although these preliminary reports suggest that mechani-

cal thrombolysis has great potential for the treatment of patients with acute ischemic stroke, these procedures have not been tested sufficiently to make any recommendation about their use.

Recommendations

At present, none of the methods of mechanical thrombolysis has been adequately tested to draw conclusions about efficacy. These interventions cannot be recommended outside the setting of clinical trials (grade A, no change from 2003).

Neuroprotective Agents

The last full guideline statement reviewed the results of several clinical trials that tested putative neuroprotective agents. No agent had demonstrated clinical benefit. Since the publication of the guidelines, the results of the IMAGES (Intravenous Magnesium Efficacy in Stroke) study have been reported.⁶¹ No overall difference in outcomes was noted between patients given mag-

nesium and patients given placebo when the medication was administered within 12 hours of the onset of stroke; however, only 3% of the patients were enrolled within 3 hours of the onset of symptoms. Another trial of magnesium is under way⁶²; in this trial, the medication is initiated while the patient is being transported to the hospital.

Citicoline is another putative neuroprotective agent that has been studied extensively. Although no significant benefit was associated with use of citicoline based on the primary, predetermined end points of any of the stroke trials, Davalos et al performed a meta-analysis of individual patient data.⁶³ The analysis tested the hypothesis of whether 6 weeks of treatment with oral citicoline would improve outcomes at 3 months. Data from patients receiving various doses of citicoline or placebo who were enrolled in 4 clinical trials were analyzed. Only patients with compatible neuroimaging results, a moderate-to-severe neurological deficit (NIH Stroke Scale score ≥ 8), and a prestroke modified Rankin Scale score of 0 or 1 were

TABLE 6. Approach to Elevated Blood Pressure in Acute Ischemic Stroke

Blood Pressure Level, mm Hg	Treatment
A. Not eligible for thrombolytic therapy	
Systolic ≤ 220 OR diastolic ≤ 120	Observe unless other end-organ involvement (eg, aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy) Treat other symptoms of stroke (eg, headache, pain, agitation, nausea, vomiting) Treat other acute complications of stroke, including hypoxia, increased intracranial pressure, seizures, or hypoglycemia
Systolic ≤ 220 OR diastolic 121–140	Labetalol 10–20 mg IV for 1–2 min May repeat or double every 10 min (max dose 300 mg) OR Nicardipine 5 mg/h IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/h every 5 min to max of 15 mg/h Aim for a 10%–15% reduction in blood pressure
Diastolic >140	Nitroprusside 0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ IV infusion as initial dose with continuous blood pressure monitoring Aim for a 10%–15% reduction in blood pressure
B. Eligible for thrombolytic therapy	
Pretreatment	
Systolic >185 OR diastolic >110	Labetalol 10–20 mg IV for 1–2 min May repeat 1 time or nitropaste 1–2 in
During/after treatment	
1. Monitor blood pressure	Check blood pressure every 15 min for 2 h, then every 30 min for 6 h, and finally every hour for 16 h
2. Diastolic >140	Sodium nitroprusside 0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ IV infusion as initial dose and titrate to desired blood pressure
3. Systolic >230 OR diastolic 121–140	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10 min to maximum dose of 300 mg, or give initial labetalol dose, then start labetalol drip at 2–8 mg/min OR Nicardipine 5 mg/h IV infusion as initial dose and titrate to desired effect by increasing 2.5 mg/h every 5 min to maximum of 15 mg/h; if blood pressure is not controlled by labetalol, consider sodium nitroprusside
4. Systolic 180–230 OR diastolic 105–120	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10–20 min to maximum dose of 300 mg or give initial labetalol dose, then start labetalol drip at 2–8 mg/min

included. Recovery was assessed on the basis of a global estimate of effect on the modified Rankin Scale, NIH Stroke Scale, and the Barthel Index. Recovery at 3 months was found in 25.2% of citicoline-treated patients versus 20.2% of placebo-treated patients ($P=0.0034$). The data for this exploratory, post hoc analysis were obtained from a highly selected group of patients. Of particular concern is that none of the individual clinical trials, which were the source of the data, was able to find a benefit from treatment with citicoline. Thus, additional research is needed to substantiate these results.

Recommendations

At present, no agent with putative neuroprotective effects can be recommended for the treatment of patients with acute ischemic stroke (grade A, no change from 2003).

Nutrition and Hydration

In a randomized trial, the FOOD (Feed Or Ordinary Diet) Trial Collaboration is testing the utility of several feeding strategies including oral supplementation, early versus delayed nasogastric tube feeding, and nasogastric versus percutaneous endoscopic gastrostomy feeding. A preliminary report based on 3012 patients indicates that poor baseline nutritional status is associated with worse outcomes at 6 months.⁶⁴ Although weakened, this relationship persists after adjustment for other factors including the patient’s age,

increased risk of infections including pneumonia, gastrointestinal bleeding, and pressure sores. Data about the effectiveness of specific therapies aimed at improving nutrition are not yet available. Still, these data provide a strong rationale for assessment of the patient’s nutritional status at the time of admission. In addition, measures should be implemented to maintain or improve the nutritional status of all patients with recent stroke.

Recommendations

Assessment of the patient’s baseline nutritional status and institution of measures to correct any major nutritional problems are recommended (grade C, new recommendation).

Hypothermia

Small preliminary clinical studies suggest that hypothermia may be feasible and beneficial for treatment of acute stroke.^{65–68} Two important articles in the *New England Journal of Medicine* showed significant benefits for hypothermia in cardiac arrest survivors.^{69,70} Hypothermia for acute stroke is a promising area for development, but data are insufficient to recommend it.

Table 6

Table 6 of the 2003 Guidelines has been updated with the table on page 920. The 2003 Guidelines online now show this update, and the table is being printed here for reference.

Disclosure

Writing Group Member Name	Research Grant	Speakers Bureau/Honoraria	Stock Ownership	Consultant/Advisory Board	Other
Harold Adams	National Institute of Neurological Disorders and Stroke; Boehringer Ingelheim; Centocor; Eli Lilly; Sanofi-Synthelabo; NMT Medical; Astra-Zeneca; Merck; GlaxoSmithKline	Boehringer Ingelheim; Bristol-Myers Squibb	None	None	None
Robert Adams	National Heart, Lung, and Blood Institute-National Institutes of Health	Boehringer Ingelheim; Bristol-Myers Squibb; Wyeth Laboratories; Sanofi-Synthelabo; Novartis	None	Boehringer Ingelheim; Bristol-Myers Squibb; Sanofi-Synthelabo; Wyeth Laboratories; Department of Veterans Affairs	Siemens ACUSON; Advanced Testing Laboratory; Boehringer Ingelheim; Bristol-Myers Squibb
Gregory Del Zoppo	National Institutes of Health	None	None	Boehringer Ingelheim	None
Larry B. Goldstein	Grants: National Institutes of Health, Department of Veterans Affairs, Centers for Disease Control and Prevention/University of North Carolina-Chapel Hill; clinical trials site: Boehringer Ingelheim, AGA Medical Corp	Bayer; Pfizer/Parke-Davis	None	AstraZeneca; Bristol-Myers Squibb/Sanofi-Synthelabo EVEREST; CuraGen Corp; D-Pharm; GlaxoSmithKline; Johnson & Johnson; Merck Research Laboratories; Pfizer/Parke-Davis; AGA Medical Corp	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit.

prestroke functional level, living conditions, and severity of stroke. A poor nutritional status was associated with an

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KEY WORDS: AHA/ASA Scientific Statements ■ stroke ■ thrombolytic therapy ■ anticoagulation ■ evaluation

Correction

In the April 2005 issue of *Stroke*, the AHA/ASA Scientific Statement, “Guidelines for the Early Management of Patients With Ischemic Stroke: 2005 Guidelines Update: A Scientific Statement From the Stroke Council of the American Heart Association/American Stroke Association” by Adams et al (*Stroke*. 2005;36:916–923), contained typographical errors in Table 6 on page 920. In the left column, row 3, the row heading “Systolic \leq 220 OR diastolic 121–140” should have read “Systolic $>$ 220 OR diastolic 121–140.” In addition, the original table contained two errors that were corrected in a previous erratum (*Stroke*. 2005;36:1352). The publisher regrets these typographical errors. The corrected Table 6 appears below, and the online version of the statement has been updated.

TABLE 6. Approach to Elevated Blood Pressure in Acute Ischemic Stroke

Blood Pressure Level, mm Hg	Treatment
A. Not eligible for thrombolytic therapy	
Systolic \leq 220 OR diastolic \leq 120	Observe unless other end-organ involvement (eg, aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy) Treat other symptoms of stroke (eg, headache, pain, agitation, nausea, vomiting) Treat other acute complications of stroke, including hypoxia, increased intracranial pressure, seizures, or hypoglycemia
Systolic $>$ 220 OR diastolic 121–140	Labetalol 10–20 mg IV for 1–2 min May repeat or double every 10 min (max dose 300 mg) OR Nicardipine 5 mg/h IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/h every 5 min to max of 15 mg/h Aim for a 10%–15% reduction in blood pressure
Diastolic $>$ 140	Nitroprusside 0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ IV infusion as initial dose with continuous blood pressure monitoring Aim for a 10%–15% reduction in blood pressure
B. Eligible for thrombolytic therapy	
Pretreatment	
Systolic $>$ 185 OR diastolic $>$ 110	Labetalol 10–20 mg IV for 1–2 min May repeat 1 time or nitropaste 1–2 in
During/after treatment	
1. Monitor blood pressure	Check blood pressure every 15 min for 2 h, then every 30 min for 6 h, and finally every hour for 16 h
2. Diastolic $>$ 140	Sodium nitroprusside 0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ IV infusion as initial dose and titrate to desired blood pressure
3. Systolic $>$ 230 OR diastolic 121–140	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10 min to maximum dose of 300 mg, or give initial labetalol dose, then start labetalol drip at 2–8 mg/min OR Nicardipine 5 mg/h IV infusion as initial dose and titrate to desired effect by increasing 2.5 mg/h every 5 min to maximum of 15 mg/h; if blood pressure is not controlled by labetalol, consider sodium nitroprusside
4. Systolic 180–230 OR diastolic 105–120	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10–20 min to maximum dose of 300 mg or give initial labetalol dose, then start labetalol drip at 2–8 mg/min

[Correction for Vol 36, Number 4, April 2005. Pages 916–923.]

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Correction

In the article by Adams et al¹, “Guidelines for the Early Management of Patients With Ischemic Stroke: 2005 Guidelines Update: A Scientific Statement From the Stroke Council of the American Heart Association/American Stroke Association,” which appeared in the April 2005 issue of the *Stroke*, an error appears on page 921. In the paragraph that begins, “In a randomized trial, the FOOD. . .,” the sentence should read, “In a randomized trial, the FOOD (Feed Or Ordinary Diet) Trial Collaboration is testing the utility of several feeding strategies including oral supplementation, early versus delayed nasogastric tube feeding, and nasogastric versus percutaneous endoscopic gastrostomy feeding.” The corrected version of this article is available online at <http://stroke.ahajournals.org/cgi/content/full/36/4/916>. (The previous version, if needed, can be accessed by selecting the “Previous Version of This Article” link.)

¹[Correction for Vol 36, Number 6, April 2005. Pages 916–923.]
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Correction

In the April 2005 issue of *Stroke*, the AHA/ASA Scientific Statement, “Guidelines for the Early Management of Patients With Ischemic Stroke” by Adams et al,¹ contained two dosing errors in Table 6 on page 920. In the table, there are 2 rows labeled “Diastolic >140.” Because of an error in production, in each of these rows the dose for nitroprusside was given as 0.5 mg·kg⁻¹·min⁻¹.

Please note that the correct dose in both cases should be 0.5 μg·kg⁻¹·min⁻¹. The corrected Table 6 appears below.

TABLE 6. Approach to Elevated Blood Pressure in Acute Ischemic Stroke

Blood Pressure Level, mm Hg	Treatment
A. Not eligible for thrombolytic therapy	
Systolic ≤220 OR diastolic ≤120	Observe unless other end-organ involvement (eg, aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy) Treat other symptoms of stroke (eg, headache, pain, agitation, nausea, vomiting) Treat other acute complications of stroke, including hypoxia, increased intracranial pressure, seizures, or hypoglycemia
Systolic ≤220 OR diastolic 121–140	Labetalol 10–20 mg IV for 1–2 min May repeat or double every 10 min (max dose 300 mg) OR Nicardipine 5 mg/h IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/h every 5 min to max of 15 mg/h Aim for a 10%–15% reduction in blood pressure
Diastolic >140	Nitroprusside 0.5 μg·kg ⁻¹ ·min ⁻¹ IV infusion as initial dose with continuous blood pressure monitoring Aim for a 10%–15% reduction in blood pressure
B. Eligible for thrombolytic therapy	
Pretreatment	
Systolic >185 OR diastolic >110	Labetalol 10–20 mg IV for 1–2 min May repeat 1 time or nitropaste 1–2 in
During/after treatment	
1. Monitor blood pressure	Check blood pressure every 15 min for 2 h, then every 30 min for 6 h, and finally every hour for 16 h
2. Diastolic >140	Sodium nitroprusside 0.5 μg·kg ⁻¹ ·min ⁻¹ IV infusion as initial dose and titrate to desired blood pressure
3. Systolic >230 OR diastolic 121–140	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10 min to maximum dose of 300 mg, or give initial labetalol dose, then start labetalol drip at 2–8 mg/min OR Nicardipine 5 mg/h IV infusion as initial dose and titrate to desired effect by increasing 2.5 mg/h every 5 min to maximum of 15 mg/h; if blood pressure is not controlled by labetalol, consider sodium nitroprusside
4. Systolic 180–230 OR diastolic 105–120	Labetalol 10 mg IV for 1–2 min May repeat or double labetalol every 10–20 min to maximum dose of 300 mg or give initial labetalol dose, then start labetalol drip at 2–8 mg/min

¹[Correction for Vol 36, Number 4. Pages 916–923.]

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