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Ischemic Stroke Syndromes: The Challenges of Assessment, Prevention, and Treatment

Part II: Physical Examination, Laboratory Investigations, Imaging, and Treatment

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Part I of this two-part series on stroke covered the differential diagnosis, risk factors, and prevention of stroke. This second and final part in the series will focus on the physical examination, laboratory investigations, imaging, and treatment of stroke.

—The Editor

History

A patient presenting with stroke must be evaluated rapidly and efficiently, since the goal of treatment is restoring perfusion to an area of tissue within a short time. The patient's speech may be impaired, so the interviewer must be resourceful and obtain the history from family members, paramedics, or bystanders who observed the event. The time of symptom onset must be determined; if this information is not available, the onset has to be assumed to be the time when the patient last was seen well. This assumption has to be made in patients who awaken from sleep with neurologic deficits. If the patient has a transient ischemic attack (TIA) and then has a subsequent neurologic event, the time of onset is considered to be the time of the second event. In patients with a stepwise worsening or waxing and waning of symptoms, the time of the first symptom is considered to be the time of symptom onset. Patients should be questioned about what they were doing when their symptoms began, the onset of the symptoms (gradual vs. abrupt), progression of symptoms, residual deficits, and number of attacks experienced. Cognitive changes and loss of memory or consciousness must be recorded. The history also should elicit whether the patient had seizures or visual symptoms, impairment of hearing or balance, or headache. The patient more readily may report perceived neurologic deficits such as difficulties with speech, reading, or writing; paralysis; or sensory disturbance.[1,2] To complete the history, the patient's systemic diseases, medications taken

(prescription and over the counter), and any illicit drug use must be documented.[3]

Patients presenting with pontine infarction may describe a preceding transient pain radiating from the unilateral eye to the nose, following which they developed numbness or ataxic hemiparesis on the side contralateral to the pain.[4] The "beauty parlor syndrome" has been described in elderly patrons receiving shampoo treatments. Mechanical impingement by neck rotation and hyperextension decreases vertebral artery flow and produces hypoperfusion at the atlanto-occipital-distal vertebral artery junction. Patients may present with vertigo and ataxia.[5]

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General Physical Examination

After assessment and initial stabilization of the patient's airway, breathing, and circulation (discussed below), a general physical examination must be performed. Cardiac auscultation may reveal the irregular rhythm of atrial fibrillation or the murmur of a valvular disorder. Treatment aimed at slowing ventricular rate and increasing cardiac output may be initiated.[3] A carotid bruit may be noted on the opposite side of the deficit. Pulses must be assessed bilaterally in the extremities. A deficit may indicate the presence of an aortic dissection. Skin should be inspected for needle marks (suggesting intravenous drug use) and for petechiae or ecchymosis (suggesting blood dyscrasias, the use of warfarin, or trauma). The neck should be examined for tenderness or meningismus, which may indicate trauma, subarachnoid hemorrhage, or meningitis as the cause of the deficit. Inspection of the neck may reveal a scar from carotid endarterectomy, indicating previously diagnosed and treated cerebrovascular disease. The presence of a recent operative scar may influence whether thrombolytics can be administered safely.

Ophthalmic examination is an important aspect of the physical examination in a patient presenting with cerebral ischemia.[3] On gross examination, congestion of vessels around the limbus suggests collateral circulation around the orbit. Horner's syndrome (ptosis, miosis, and facial anhydrosis) may indicate occlusion and thrombosis of the ipsilateral carotid artery. Reduction of flow to the ophthalmic artery may occur with atherosclerosis of the ipsilateral internal carotid artery prior to the branch of the ophthalmic artery, resulting in retinal ischemia. Funduscopic examination may reveal hemorrhages and exudates from retinal hypoxia. If the retina or optic nerve becomes infarcted, the retina is pale, the optic disc is white, the retinal arteries are difficult to visualize, and the veins are attenuated.

Neurologic Examination

Many scales exist for the assessment of stroke severity.[6-8] The National Institutes of Health Stroke Scale (NIHSS) (*see Table 1*) has good correlation with severity of stroke, risk of hemorrhagic transformation of ischemic stroke following tPA administration, and stroke prognosis.[9-14] This scale is used widely in the United States. Of patients with ischemic stroke and initial NIHSS score less than 10, 60-70% will have favorable outcome after one year. Only 4-16% of patients with initial NIHSS score more than 20 will have similar outcome.

The Cincinnati Prehospital Stroke Scale (CPSS),[15] a simplification of the NIHSS, has been well validated in the prehospital setting for identifying patients with stroke, particularly of the anterior circulation. A single abnormality on CPSS has a sensitivity of 100% and a specificity of 90% in identifying candidates for thrombolysis. The three questions that constitute the CPSS are listed in Table 2.

Laboratory Investigations

As part of the initial evaluation of patients with stroke, the following diagnostic studies should be obtained to rule out mimics or to find the cause of the stroke:[16]

• Fingerstick glucose—to rapidly eliminate the possibility of

Table 1. The National Institutes of Health Stroke Scale

TEST ITEM	TITLE	SCORES AND RESPONSES
1a	Level of consciousness	0 - alert 1 - drowsy 2 - obtunded 3 - coma/unresponsive
1b	Orientation questions (2)	0 - answers both correctly 1 - answers one correctly 2 - answers neither correctly
1c	Response to commands (2)	0 - performs both tasks correctly 1 - performs one task correctly 2 - performs neither correctly
2	Gaze	0 - normal horizontal movements 1 - partial gaze palsy 2 - complete gaze palsy
3	Visual fields	0 - no visual field defect 1 - partial hemianopia 2 - complete hemianopia 3 - bilateral hemianopia
4	Facial movement	0 - normal 1 - minor facial weakness 2 - partial facial weakness 3 - complete unilateral palsy
5	Motor function (arm) a. Left arm b. Right arm	0 - no drift 1 - drift before 5 seconds 2 - falls before 10 seconds 3 - no effort against gravity 4 - no movement
6	Motor function (leg) a. Left leg b. Right leg	0 - no drift 1 - drift before 5 seconds 2 - falls before 5 seconds 3 - no effort against gravity 4 - no movement
7	Limb ataxia	0 - no ataxia 1 - ataxia in one limb 2 - ataxia in two limbs
8	Sensory	0 - no sensory loss 1 - mild sensory loss 2 - severe sensory loss
9	Language	0 - normal 1 - mild aphasia 2 - severe aphasia 3 - mute or global aphasia
10	Articulation	0 - normal 1 - mild dysarthria 2 - severe dysarthria
11	Extinction or inattention	0 - absent 1 - mild (loss of 1 sensory ability) 2 - severe (loss of 2 modalities)

Adapted from: www.strokecenter.org/trials/scales/nihss.html. Accessed 2/11/2004.

hypoglycemia or hyperglycemia as the cause of the neurologic deficit.

• Serum electrolytes—may identify hypernatremia or hyponatremia, acute renal failure, or acidosis.

• Complete blood count—may rule out hyperviscosity syndrome, thrombocytosis, or thrombocytopenia. Leukocytosis may be a result of acute stress, the presence of necrotic brain tissue, or a preceding infection. A recent (within one week) infection, particularly of bacterial origin, has been noted as a factor associated with stroke in patients of all ages.

• Coagulation studies—a coagulopathy must be identified, since it may have been the precipitating factor for a stroke or may disqualify a patient from receiving thrombolytic therapy.

• Electrocardiogram—may reveal atrial fibrillation or other rhythm disturbance as the cause of a stroke. Deep symmetric T-wave inversions, prominent U waves, and QT prolongation may suggest subarachnoid hemorrhage.

In selected patients, the following additional tests may be obtained:

• Liver function tests;
• Toxicology screens;
• Blood alcohol level;
• Pregnancy test;
• Arterial blood gas;
• Chest x-ray film;
• Electroencephalogram;
• Lumbar puncture.

Imaging

Computed Tomography. CT scan identifies hemorrhage and helps find other nonvascular causes of stroke-like symptoms.[17] CT scan can be used quickly and reliably to determine whether an acute stroke is hemorrhagic, the major branch point in therapy for stroke. Despite multiple advancing technologies, unenhanced CT scan remains the only radiologic test necessary for evaluation of the brain in an acute stroke before initiation of thrombolytic therapy (if indicated). Other tests may be beneficial in better defining the area of the ischemic penumbra and quantifying perfusion to the brain but currently are not necessary to determine acute treatment pathways. Their availability may be limited to specialized stroke centers.

Recently, attention has been given to signs that may be seen on early CT, which might predict outcome or risk of hemorrhage if thrombolytics are administered to patients with acute ischemic stroke. These signs are listed in Table 3.[16,18] One study reported that the hyperdense middle cerebral artery sign (HMCAS) indicates there is thrombus or embolus in the first portion of the middle cerebral artery (MCA) and is associated with neurologic deterioration.[19] The same investigators also found early CT evidence of more than 50% MCA involvement to be predictive of neurologic deterioration.

The European Cooperative Acute Stroke Study (ECASS) showed the presence of early ischemic changes on CT scan as well as severity of initial clinical deficit was associated with increased risk of hemorrhagic infarction or hemorrhagic transfor-

Table 2. The Three Questions of the Cincinnati Prehospital Stroke Scale: Evaluation of Facial Palsy, Arm Weakness, and Speech Abnormalities[15]

FACIAL DROOP
Ask the patient to show his/her teeth or to smile. • Normal: Both sides of the face move equally. • Abnormal: One side of the face does not move as well as the other.
ARM DRIFT
Ask the patient to close his/her eyes and extend both arms straight out for 10 seconds. • Normal: Both arms move the same or both arms do not move at all. • Abnormal: One arm does not move or one arm drifts down compared with the other.
SPEECH
Ask the patient to repeat the statement, "The sky is blue in Cincinnati." • Normal: The patient says the words correctly with no slurring. • Abnormal: The patient slurs words, says the wrong words, or is unable to speak.

mation.[20] In another study, patients had increased risk of fatal brain hemorrhage if the initial CT scan was abnormal or showed hypoattenuation in more than one-third of the MCA territory but had increased benefit if they had less than one-third hypoattenuation of the MCA territory.[21] CT evidence of mass effect or early edema also has been associated with an eight-fold increase in risk of intracranial hemorrhage.[16,22]

Interpretation of CT scans by physicians can vary.[23] Scoring systems for CT interpretation may improve diagnosis and provide prognostic information but currently are not validated. More study is needed.

CT Angiography (CTA). CTA is emerging as a study complementary to standard unenhanced CT. It may be performed in fewer than five minutes following initial CT without moving the patient. CTA has good correlation with confirming studies such as digital subtraction angiography (DSA) and ultrasound (US). It is less invasive than DSA and less time-consuming and more readily available than either DSA or US. CTA evidence of occlusion at presentation correlates strongly and independently with clinical outcome.[24]

CTA can provide important diagnostic and prognostic information. In a study of 40 patients, CTA identified subgroups that may not benefit from intravenous thrombolytic therapy (those with autolyzed thrombi, occlusion of internal carotid artery bifurcation, and poor leptomeningeal collaterals).[25] In another small study involving 15 patients, CTA identified an aneurysm.[26]

Xenon-enhanced CT cerebral blood flow (XeCT CBF) also has been studied in conjunction with CTA. One study of 51 patients suggested that if patients had normal CBF (> 30 mL/100 g/min), they were more likely to have a good functional outcome and not benefit from thrombolytic therapy. In contrast, patients with reversible ischemia (7-29 mL/100 g/min) were theoretically most likely to benefit from thrombolytics. Patients with irreversible ischemia (< 7 mL/100 g/min) were deemed not likely to benefit from thrombolytic treatment. The authors concluded that XeCT CBF and/or CTA may be used to identify subgroups that may benefit most from thrombolytics and those that may be extended beyond the three-hour window.[27]

In some centers, CTA already is a standard part of the initial evaluation of patients with acute stroke. It is likely that more information will emerge from studies with CTA to better define subgroups that will benefit most from thrombolytic therapy and those that should be excluded from it.

Magnetic Resonance Imaging (MRI). Standard MRI images are insensitive to changes in acute ischemia within the first hours; changes are found in fewer than 50% of patients. In addition, MRI is less sensitive than CT in detecting intracranial hemorrhage. Coupled with the other limitations associated with MRI (i.e., metallic implants of any type, time, cost, access issues, and claustrophobia), it is unlikely that MRI will replace CT in the imaging of acute stroke patients anytime in the near future other than in specialized stroke centers.

Diffusion weighted imaging (DWI) can visualize areas of ischemia within minutes of symptom onset because of early changes in ischemic brain tissue (decreased water diffusion). Perfusion weighted imaging (PWI) with paramagnetic contrast agent can provide measures of cerebral hemodynamic status. DWI can detect lesion size, site, and age and can give information about the involved vascular territory. It has reasonably high sensitivity (88-100%) and specificity (95-100%) in detecting acute ischemia.[16,28] With the advances in MRI technology, many authors advocate the use of MRI to develop better protocols for patients who will benefit from thrombolytics, potentially even beyond the three-hour window.[29,30]

Treatment

Emergency Department Management. Basic principles of care apply to all stroke patients. Attention should be paid to airway, breathing, and circulation. Patients who require emergent airway intervention to avoid obstruction, hypoventilation, and aspiration should be intubated using rapid sequence intubation. Oxygen therapy is indicated if the patient requires support to maintain an appropriate level of oxygenation; however, routine supplemental oxygen is not indicated in all patients with acute ischemic stroke.[31] Patients should have intravenous access and initial cardiac monitoring. Blood glucose should be measured and corrected.

Antipyretics should be administered if the patient is febrile, since pyrexia has been associated with poor neurologic outcomes in patients presenting with acute stroke.[32] One study found that a difference in body temperature of 1° C was equivalent to a four-point difference in stroke severity on admission score, a 15-mm difference in infarct size, an 80% difference in mortality, and a four-point difference in stroke severity score

on discharge.[33] Fever after stroke onset also has been associated with increased morbidity and mortality.[34] The source of fever should be investigated and treated, and efforts should be made to lower the temperature pharmacologically or with a cooling blanket. Hypothermia has been shown to be neuroprotective after experimental global and focal hypoxic brain injury. Modest hypothermia may be an avenue for further investigation.[35]

Blood Pressure. Transient elevations in blood pressure may be seen in acute stroke, and elevated pressures may fall without pharmacologic intervention.[36,37] There are no good data to support the lowering of blood pressure in patients with acute ischemic stroke and no data to define which levels of arterial hypertension necessitate emergent treatment.[37] Management of blood pressure should balance the theoretical increased risk of cerebral edema and hemorrhagic transformation against the detriment of aggressive blood pressure treatment leading to secondary reduction of perfusion and expansion of the size of infarct. Generally, blood pressure should be left untreated unless urgent antihypertensive medication is needed for organ failure (hemorrhagic infarct, acute renal failure, hypertensive encephalopathy, aortic dissection, acute pulmonary edema, or acute myocardial infarction [MI]). The American Stroke Association (ASA) guidelines for the early management of patients with ischemic stroke state antihypertensives should be withheld unless the systolic blood pressure is greater than 220 mmHg or the diastolic blood pressure is greater than 120 mmHg.[16] Agents that easily are titrated and that minimally affect cerebral perfusion (e.g., labetalol) are preferred. Nitroprusside may be used if necessary. Oral agents such as captopril and nicardipine also can be used. Nifedipine and other agents that precipitously lower blood pressure should be avoided.

Persistent hypotension is rare in association with stroke, but if it occurs, it must be addressed. The source of the hypotension must be investigated. Possible etiologies include MI, arrhythmias, hypovolemia, and aortic dissection. An electrocardiogram should be obtained as part of the patient's initial management. Volume replacement or the use of pressor agents may be required.

Thrombolytics should not be administered in patients with systolic blood pressure greater than 185 mmHg or diastolic blood pressure greater than 110 mmHg, as this is associated with parenchymal hemorrhage.[20,21] If blood pressure can be controlled with one or two doses of labetalol, then the patient remains eligible for thrombolytic therapy. More aggressive management to maintain desired blood pressure levels contraindicates thrombolytic therapy.

CT scanning of the brain should be performed as rapidly as possible. CT scanning to rule out intracranial hemorrhage will delineate the treatment pathway and help determine eligibility for thrombolytic therapy. CT examination should be completed and interpreted, ideally within 45 minutes of the patient's arrival. Repeat CT scan should be obtained if the patient worsens; the scan can be used to determine if hemorrhagic transformation has occurred.

Table 3. Computed Tomography Indications of Poor Outcome after Administration of Thrombolytics

- Hyperdense middle cerebral artery sign
- Loss of gray-white matter differentiation in the lentiform nucleus or cortical ribbon
- Sulcal effacement is an early (within 6 hours) sign of ischemia in the territory of the middle cerebral artery
- Early ischemic changes
- Hypoattenuation in more than one-third of the middle cerebral artery territory
- Mass effect or early edema

Thrombolytics. Three early trials with streptokinase were terminated due to increases in the incidence of adverse outcomes (intracranial hemorrhage and death) in the treatment group.[38-40] Recombinant tissue plasminogen activator (tPA) is the only thrombolytic approved by the U.S. Food and Drug Administration (FDA) for the treatment of acute ischemic stroke. The FDA based its approval on one prospective multicenter trial, conducted by the National Institute of Neurological Disorders and Stroke (NINDS), the results of which were published in 1995.[22]

The NINDS trial was divided into two parts; in both parts, patients were randomized to receive tPA, 0.9 mg/kg, or placebo within three hours after onset of stroke symptoms. Patients in the treatment arm were given 0.9 mg/kg tPA (maximum dose, 90 mg) in a 10% bolus followed by a constant infusion of the remaining 90% over 60 minutes. The first part enrolled 291 patients and measured whether they had improvement in their NIHSS score of four points or more over baseline or resolution of deficits within 24 hours of onset of stroke. In this portion of the trial, there was no statistical difference between the tPA group and the placebo recipients ($P = 0.21$). However, in post hoc analysis, the authors point out that the median NIHSS scores were two points lower in the 0- to 90-minute range and four points lower in the 90- to 180-minute range when the tPA group was compared with placebo (P value not provided). The second part enrolled 333 patients and assessed clinical outcomes at three months. Patients who received tPA were at least 30% more likely to have minimal or no disability based on four measures of neurologic disability (NIHSS, modified Rankin, Glasgow outcome scale, and Barthel Index). The primary hypothesis in part two was tested with a global statistic to simultaneously test for effect in all four outcome measures.

Symptomatic intracerebral hemorrhage occurred in 6.4% of tPA recipients but only 0.6% of placebo recipients ($P < 0.001$). Mortality at three months was 17% in the tPA group and 21% in the placebo group ($P = 0.30$).

A number-needed-to-treat analysis of the NINDS trial indicates that for every eight acute stroke patients treated, one will benefit. One patient in 17 will suffer an intracranial hemorrhage, and one in 40 will die. These numbers may be helpful in explain-

ing risks and benefits to patients who are eligible for thrombolytic treatment.

Opponents of the use of thrombolytics and critics of the NINDS study point to the low numbers of patients enrolled per center. The largest controversy centers on the baseline NIHSS score in the treatment group vs. the placebo group. A later report from the NINDS study group divulged an imbalance in baseline NIHSS scores in the 91- to 180-minute subgroup: The treatment group's score was significantly lower than the placebo group's score.[41] This difference is significant because it has been reported that the NIHSS score strongly is related to outcome.[42]

An independent panel of three biostatisticians, one emergency physician, one neurologist, and one internist recently confirmed the results of the NINDS data, announcing their findings at conferences in Valencia and Boston. They concluded that, despite subgroup imbalances, there is statistically significant benefit in treating acute ischemic stroke with tPA within three hours of symptom onset, with an apparent increase in the benefit odds ratio from 1.7 to 2.1 in the original analysis. Publication of their data and methodology is pending.

The European Cooperative Acute Stroke Study (ECASS)[43] is the competing study with negative conclusions. In the ECASS trial, 620 patients were randomized to receive placebo or tPA, 1.1 mg/kg, within six hours. ECASS measured disability using the Barthel Index and modified Rankin scale at 90 days as well as combined Barthel Index and Rankin scale at 90 days, the Scandinavian Stroke Scale at 90 days, and 30-day mortality. Results showed some statistically significant improvement in functional measures and neurologic outcome in a defined subgroup of patients with moderate to severe deficit without extended infarct signs on initial CT scan. The authors concluded, however, that since this subgroup is hard to define and there was an increase in the mortality rate at 30 days and a significant increase in parenchymal hemorrhage in the tPA-treated group, they could not recommend the use of thrombolytics in an unselected population.[43]

Critics of the ECASS and supporters of the NINDS study point to the difference in dose and timing of tPA administration and the different outcome measures between the two studies. Post ad hoc analysis of ECASS using NINDS endpoints was more favorable.[44] The authors of the more recent ECASS II concluded that thrombolysis in selected patients may improve clinical outcome, although their original primary endpoint improvement in modified Rankin Score was not statistically significant.[45]

A Cochrane database meta-analysis[46] encompassed 17 trials and 5216 patients. Trials were heterogeneous, with different agents, doses, routes, and measured endpoints, although about 50% of the data came from trials using tPA. Thrombolytic therapy significantly increased the odds of death within the first 10 days and at the end of follow-up and increased the risk of symptomatic intracranial hemorrhage. Thrombolytics administered within six hours also significantly reduced the number of patients who were dead or dependent at the end of follow-up. Patients treated within three hours received the most benefit, with less effect on the incidence of death. The reviewers concluded that tPA may be associated with less hazard and more benefit and, thus, its use may be justified in experienced clinical centers for selected patients. They further stated that widespread use of tPA in routine clinical practice cannot be supported at this time and that further study is needed.

As the controversy continues, both the American College of Emergency Physicians (ACEP) and the American Academy of Emergency Medicine (AAEM) have published policy statements addressing the use of thrombolytics for acute ischemic stroke. ACEP endorses cooperation between emergency medical services personnel and emergency department personnel to identify hospital capabilities and states that tPA may be efficacious for patients meeting NINDS criteria but that more study is necessary to better define the group of patients who will benefit the most.[47] AAEM concluded that there is insufficient evidence to consider tPA as the standard of care and that, given the lack of definitive evidence, it is inappropriate to conclude that use or non-use represents "standard of care."[48]

The ACEP web site offers a policy and resource education paper, listing indications and contraindications (both absolute and relative) for the use of tPA in acute ischemic stroke (*see Table 4*) as well as a summary of the major trials involving thrombolytics for stroke.[49]

More recently, the Society for Academic Emergency Medicine (SAEM) published its own position on optimizing the care of patients with stroke. The authors of the paper state that, although there is evidence for therapeutic benefit for an important minority of patients, there are few available data on which subgroups of patients most likely will benefit and those that most likely will be harmed. SAEM supports ongoing scientific investigation; the creation of national research initiatives, including a data registry to gather outcomes for stroke patients whether or not thrombolytic therapy was administered; and improved education of health care providers and the lay public.[50]

Antiplatelet Agents. Aspirin has been proven to be of benefit in reducing the risk of recurrence of stroke. The International Stroke Trial (IST) and the Chinese Acute Stroke Trial (CAST), with approximately 20,000 patients in each, showed small but significant benefits. IST found a reduced incidence of recurrent strokes at 14 days and decreased rates of death or dependency at six months.[51] CAST also found some early benefit in mortality to aspirin but did not reach significance with its endpoint of death or dependence at hospital discharge.[52] When the two trials are combined, there is a small but significant long-term benefit from aspirin—with nine fewer deaths or nonfatal strokes per 1000 (absolute risk reduction of 0.9%; number needed to treat = 111).[53,54] Aspirin should not be given within 24 hours of tPA administration.

Heparin. Neither unfractionated heparin (UFH) nor low-molecular-weight heparin (LMWH) has been shown to decrease mortality or stroke-related morbidity if given within

Table 4. Indications and Contraindications for the Use of Tissue Plasminogen Activator (tPA) in Patients with Ischemic Stroke

INDICATIONS

- Ischemic stroke onset within 3 hours of drug administration.
- Measurable deficit on the NIH stroke scale examination.
- Computed tomography (CT) scan does not show hemorrhage or nonstroke cause of deficit.
- Age >18 years.

CONTRAINDICATIONS

- Symptoms are minor or improving rapidly.
- Patient had seizure at onset of stroke.
- Patient had another stroke or serious head trauma within the past 3 months.
- Patient had major surgery within the past 14 days.
- Patient has a known history of intracranial hemorrhage.
- Patient has sustained systolic blood pressure >185 mmHg.
- Patient has sustained diastolic blood pressure >110 mmHg.
- Aggressive treatment is necessary to lower the patient's blood pressure.
- Patient has symptoms suggestive of subarachnoid hemorrhage.
- Patient had gastrointestinal or urinary tract hemorrhage within the past 21 days.
- Patient had arterial puncture at a noncompressible site within the past 7 days.
- Patient received heparin within the past 48 hours and has elevated partial thromboplastin time (PTT).
- Prothrombin time (PT) is >15 seconds.
- Platelet count is < 100,000 mL.
- Patient's serum glucose is < 50 mg/dL or > 400 mg/dL.

RELATIVE CONTRAINDICATIONS

- Patient has a large stroke with NIH Stroke Scale score > 22.
- CT scan shows evidence of large middle cerebral artery territory infarction (sulcal effacement or blurring of gray-white junction in more than one-third of MCA territory).

Reprinted with permission from: American College of Emergency Physicians. Policy Resource and Education Paper: Use of intravenous tPA for the management of acute stroke in the emergency department. ACEP web site www.acep.org/1,5005,0.html, February 2002. Accessed 2/12/2004.

48 hours of the onset of acute ischemic stroke.[54] UFH or LMWH likewise has not been shown to reduce the rate of stroke recurrence if given within 48 hours. Nor is there any benefit from heparin of either type if specific subgroups are chosen (atrial fibrillation or advancing stroke).[51] Heparin does reduce the risk of deep vein thrombosis (DVT) in acute stroke, with evidence for both UFH and LMWH types, but there is not enough evidence to suggest a decrease in the incidence of pulmonary embolism.[54]

Neuroprotectants. There was initial excitement about potential neuroprotectants, but no trials have shown any significant improvement with neuroprotectants of any class. Their benefit has yet to be proven. Future studies will assess magnesium and combination therapies of neuroprotectants and thrombolytics.

Disposition

All patients with acute stroke should be admitted to the hospital, preferably to a stroke unit. Level of care can be determined by the severity of symptoms and the patient's stability. Frequent neurologic checks are important to monitor symptom progression. Neurologic deterioration may indicate hemorrhagic transformation; repeat head CT should be performed in this situation.

Patients with TIA likely would benefit from hospital admission. Approximately 15% of ischemic strokes are preceded by TIA.[55] One study found that patients presenting to the emergency department with TIA have subsequent stroke rates of 5.3% at 2 days.[56] Another study documented a stroke risk of 8.6% at 7 days and 12% at 30 days after first-ever TIA.[57] If ocular TIAs were excluded, the risk was 5.1% at 2 days, 10.3% at 7 days, and 14.3% at 30 days.

Compared with their counterparts in the United States, European stroke units typically offer more comprehensive rehabilitative care. Patients managed in stroke units in Europe have decreased mortality and disability rates and improved quality of life, with favorable effects lasting years.[58,59] Benefits were found in the community setting and regardless of age, sex, comorbidity, or initial stroke severity.[60] Reviews of multiple stroke unit trials confirm these benefits.[61-63] The American Stroke Association recommends the use of stroke units incorporating comprehensive rehabilitation.[16]

Conclusion

Acute ischemic stroke is a complex disease spectrum affecting millions of people. This emergency has a time-limited acute treatment window: Patients must be stabilized and evaluated quickly but thoroughly to rule out hemorrhage and mimics of stroke. New modalities available in brain imaging can aid the emergency physician in guiding therapy, but evaluation and assessment for thrombolytic therapy remain principal goals. Informed consent should be obtained from/for patients who are candidates for thrombolytic therapy because of the risk of intracranial hemorrhage. Stroke units have significant beneficial effect and should be established more widely.

Further research with advances in imaging technology and NIHSS correlations likely will better define the role of thrombolytics in specific patient categories. Many avenues of further research are available, as the optimal treatment of stroke patients continues to be defined. Prevention and education remain key strategies in the management of stroke, with an emphasis on smoking cessation and blood pressure control.

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Physician CME Questions

41. Which of the following should be included in the general physical examination of a stroke patient?
    A. Pulses should be assessed bilaterally in the extremities.
    B. The neck should be examined for tenderness or meningismus.
    C. The skin should be inspected for needle marks, petechiae, or ecchymosis.
    D. Funduscopic examination may reveal hemorrhages and exudates from retinal hypoxia.
    E. All of the above.

42. Pyrexia has been associated with poor neurologic outcomes in patients presenting with acute stroke.
    A. True
    B. False

43. Regarding blood pressure and stroke, which of the following statements is true?
    A. Generally, blood pressure should be left untreated unless urgent antihypertensive medication is needed for organ failure, acute pulmonary edema, or acute myocardial infarction.
    B. The ASA guidelines state that, in the early management of patients with ischemic stroke, antihypertensives should be withheld unless the systolic blood pressure is greater than 220 mmHg or the diastolic blood pressure is greater than 120 mmHg.
    C. Agents that easily are titrated and that minimally affect cerebral perfusion, such as labetalol, are preferred.
    D. Nifedipine and other agents that precipitously lower blood pressure should be avoided.
    E. All of the above.

44. Standard MRI can detect changes in acute ischemia within the first few hours.
    A. True
    B. False

45. CT studies demonstrate poor outcomes from ischemic stroke after administration of thrombolytics with which of the following findings?
    A. Early ischemic changes
    B. Hyperdense middle cerebral artery sign
    C. Loss of gray-white matter differentiation in the lentiform nucleus or cortical ribbon
    D. Mass effect or early edema
    E. All of the above

46. All of the following are contraindications for the use of tPA in patients with ischemic stroke *except*:
    A. patient who had a seizure at onset of stroke.
    B. gastrointestinal bleed six months ago.
    C. symptoms that are rapidly improving.
    D. measurable deficit on the NIHSS.
    E. systolic blood pressure greater than 185.

47. All of the following are the indications for the use of tPA in patients with ischemic stroke *except*:
    A. ischemic stroke onset within 3 hours of drug administration.
    B. patient with a large stroke with NIH stroke scale score greater than 22.
    C. age greater than 18 years.
    D. CT scan that does not show hemorrhage or nonstroke cause of deficit.
    E. measurable deficit on the NIH stroke scale examination.

48. Which of the following statements is true regarding treatment of ischemic stroke?
    A. Cochrane database review of thrombolytic therapy supports widespread use of thrombolytics for acute ischemic stroke.
    B. Streptokinase has been proven to decrease mortality in acute ischemic stroke and has been approved by the FDA for treatment of acute ischemic stroke.
    C. MRI is the preferred method of intracranial imaging for the evaluation of ischemic stroke.
    D. Patients managed in stroke units in Europe have decreased mortality and disability rates and improved quality of life.
    E. Aspirin should be given at the same time as tPA if the patient is eligible for treatment with thrombolytics.

49. Which of the following statements is true regarding antiplatelet agents?
    A. Aspirin has been proven to be of benefit in reducing the risk of recurrence of stroke.
    B. Aspirin should not be given within 24 hours of tPA

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

administration.

C. The IST and CAST trials found small but significant benefits from aspirin.

D. All of the above

50. No trials of neuroprotectants of any class have shown any significant improvement.

A. True

B. False

In Future Issues:

ST Elevation MI

CME Answer Key

41. E	46. D
42. A	47. B
43. E	48. D
44. B	49. D
45. E	50. A

Emergency Medicine Reports

CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

Emergency Medicine Reports
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Stroke Part II

The National Institutes of Health Stroke Scale

TEST ITEM	TITLE	SCORES AND RESPONSES
1a	Level of consciousness	0 - alert 1 - drowsy 2 - obtunded 3 - coma/unresponsive
1b	Orientation questions (2)	0 - answers both correctly 1 - answers one correctly 2 - answers neither correctly
1c	Response to commands (2)	0 - performs both tasks correctly 1 - performs one task correctly 2 - performs neither correctly
2	Gaze	0 - normal horizontal movements 1 - partial gaze palsy 2 - complete gaze palsy
3	Visual fields	0 - no visual field defect 1 - partial hemianopia 2 - complete hemianopia 3 - bilateral hemianopia
4	Facial movement	0 - normal 1 - minor facial weakness 2 - partial facial weakness 3 - complete unilateral palsy
5	Motor function (arm) a. Left arm b. Right arm	0 - no drift 1 - drift before 5 seconds 2 - falls before 10 seconds 3 - no effort against gravity 4 - no movement
6	Motor function (leg) a. Left leg b. Right leg	0 - no drift 1 - drift before 5 seconds 2 - falls before 5 seconds 3 - no effort against gravity 4 - no movement
7	Limb ataxia	0 - no ataxia 1 - ataxia in one limb 2 - ataxia in two limbs
8	Sensory	0 - no sensory loss 1 - mild sensory loss 2 - severe sensory loss
9	Language	0 - normal 1 - mild aphasia 2 - severe aphasia 3 - mute or global aphasia
10	Articulation	0 - normal 1 - mild dysarthria 2 - severe dysarthria
11	Extinction or inattention	0 - absent 1 - mild (loss of 1 sensory ability) 2 - severe (loss of 2 modalities)

Adapted from: www.strokecenter.org/trials/scales/nihss.html. Accessed 2/11/2004.

Computed Tomography Indications of Poor Outcome after Administration of Thrombolytics

- Hyperdense middle cerebral artery sign
- Loss of gray-white matter differentiation in the lentiform nucleus or cortical ribbon
- Sulcal effacement is an early (within 6 hours) sign of ischemia in the territory of the middle cerebral artery
- Early ischemic changes
- Hypoattenuation in more than one-third of the middle cerebral artery territory
- Mass effect or early edema

Indications and Contraindications for the Use of Tissue Plasminogen Activator (tPA) in Patients with Ischemic Stroke

INDICATIONS

- Ischemic stroke onset within 3 hours of drug administration.
- Measurable deficit on the NIH stroke scale examination.
- Computed tomography (CT) scan does not show hemorrhage or nonstroke cause of deficit.
- Age >18 years.

CONTRAINDICATIONS

- Symptoms are minor or improving rapidly.
- Patient had seizure at onset of stroke.
- Patient had another stroke or serious head trauma within the past 3 months.
- Patient had major surgery within the past 14 days.
- Patient has a known history of intracranial hemorrhage.
- Patient has sustained systolic blood pressure >185 mmHg.
- Patient has sustained diastolic blood pressure >110 mmHg.
- Aggressive treatment is necessary to lower the patient's blood pressure.
- Patient has symptoms suggestive of subarachnoid hemorrhage.
- Patient had gastrointestinal or urinary tract hemorrhage within the past 21 days.
- Patient had arterial puncture at a noncompressible site within the past 7 days.
- Patient received heparin within the past 48 hours and has elevated partial thromboplastin time (PTT).
- Prothrombin time (PT) is >15 seconds.
- Platelet count is < 100,000 mL.
- Patient's serum glucose is < 50 mg/dL or > 400 mg/dL.

RELATIVE CONTRAINDICATIONS

- Patient has a large stroke with NIH Stroke Scale score > 22.
- CT scan shows evidence of large middle cerebral artery territory infarction (sulcal effacement or blurring of gray-white junction in more than one-third of MCA territory).

Reprinted with permission from: American College of Emergency Physicians. Policy Resource and Education Paper: Use of intravenous tPA for the management of acute stroke in the emergency department. ACEP web site www.acep.org/1,5005,0.html, February 2002. Accessed 2/12/2004.

The Three Questions of the Cincinnati Prehospital Stroke Scale: Evaluation of Facial Palsy, Arm Weakness, and Speech Abnormalities

FACIAL DROOP

Ask the patient to show his/her teeth or to smile.

- Normal: Both sides of the face move equally.
- Abnormal: One side of the face does not move as well as the other.

ARM DRIFT

Ask the patient to close his/her eyes and extend both arms straight out for 10 seconds.

- Normal: Both arms move the same or both arms do not move at all.
- Abnormal: One arm does not move or one arm drifts down compared with the other.

SPEECH

Ask the patient to repeat the statement, "The sky is blue in Cincinnati."

- Normal: The patient says the words correctly with no slurring.
- Abnormal: The patient slurs words, says the wrong words, or is unable to speak.

Supplement to *Emergency Medicine Reports*, March 8, 2004: Ischemic Stroke Syndromes: The Challenges of Assessment, Prevention, and Treatment. Part II: Physical Examination, Laboratory Investigations, Imaging, and Treatment." *Authors:* **Marcia Cort, MD,** Assistant Professor, Division of Emergency Medicine, University of Maryland Medical Systems, Baltimore; **Dick Kuo, MD,** Assistant Professor, Division of Emergency Medicine, University of Maryland School of Medicine, Baltimore.