

Acute Ischemic Stroke Review

Janice L. Hinkle, PhD RN CNRN; Mary McKenna Guanci, MS RN CNRN
J Neurosci Nurs. 2007;39(5):285-293, 310.

Abstract

More than 700,000 people have a stroke each year in the United States. A diagnosis of stroke formerly elicited a nihilistic approach, but this has substantially changed in the last decade. Currently, time is brain, and it is important for all disciplines to work together to initiate acute stroke protocols in the emergency department and identify patients within the therapeutic time window for thrombolytic and neuroprotective therapies. Evolving protocols, management, and nursing care all have important implications during the acute phase of ischemic stroke. Patient and family education on risk reduction must also be addressed by the entire healthcare team.

Introduction

Stroke is the third leading cause of mortality in the United States. Of the approximately 700,000 strokes occurring each year, about 550,000 are first strokes. About 400,000 strokes are ischemic (Thom et al., 2006). Stroke is the leading cause of adult disability with more than 4 million stroke survivors in the United States alone. Approximately 90% of stroke survivors are left with some residual deficit.

In 1994 the Stroke Council of the American Heart Association (AHA) published the first guidelines for the management of acute ischemic stroke (Adams et al., 1994). In 1996, the U.S. Food and Drug Administration (FDA) approved intravenous (IV) tissue plasminogen activator (tPA) as the first medication to treat acute ischemic stroke. The AHA subsequently published a supplement to the 1994 guidelines that addressed tPA (Adams et al., 1996). The advent of new therapies for acute ischemic stroke has brought about higher expectations for improved recovery and good outcome (Demchuk & Buchan, 2000). The AHA published guidelines for the early management of ischemic stroke in 2003 (Adams et al., 2003) and updated them in 2005 (Adams, Adams, del Zoppo, & Goldstein, 2005) and in 2007 (Adams et al., 2007).

This article reviews the pathophysiology of acute ischemic stroke and related patient care, identifies the laboratory and diagnostic tests the neuroscience nurse can expect to see in the fast-paced evaluation of a patient following an ischemic stroke, addresses the medical and nursing management approaches with which the neuroscience nurse needs to be familiar during the acute phase of the inpatient stay for ischemic stroke, and reviews patient and family education topics that must be addressed by the healthcare team.

Pathophysiology

Stroke is defined as an "acute neurologic dysfunction of vascular origin with sudden (within seconds) or at least rapid (within hours) occurrence of symptoms and signs corresponding to the involvement of focal areas in the brain" (Goldstein, Barnett, et al., 1989, p. 1412). The two main types of stroke are ischemic and hemorrhagic, accounting for approximately 85% and 15%, respectively (Hickey, 2003).

When an ischemic stroke occurs, the blood supply to the brain is interrupted, and brain cells are deprived of the glucose and oxygen they need to function. Ischemic stroke is a complex entity with multiple etiologies and variable clinical manifestations. Approximately 45% of ischemic strokes are caused by small or large artery thrombus, 20% are embolic in origin, and others have an unknown cause (Hickey, 2003).

Thrombosis can form in the extracranial and intracranial arteries when the intima is roughened and plaque forms along the injured vessel. The endothelial injury (roughing) permits platelets to adhere and aggregate, then coagulation is activated and thrombus develops at site of plaque. Blood flow through the extracranial and intracranial systems decreases, and the collateral circulation maintains function. When the compensatory mechanism of collateral circulation fails, perfusion is compromised, leading to decreased perfusion and cell death.

During an embolic stroke, a clot travels from a distant source and lodges in cerebral vessels. Microemboli can break away from a sclerosed plaque in the carotid artery or from cardiac sources such as atrial fibrillation, patent foramen ovale, or a hypokinetic left ventricle (Hickey, 2003). Emboli in the form of blood, fat, or air can occur during surgical procedures, most commonly during cardiac surgery, but also after long bone surgeries (Warlow et al., 2001).

Less common causes of ischemic stroke include carotid dissection (Bader & Littlejohns, 2004) and the presence of coagulopathies, such as those resulting from antiphospholipid antibodies (APASS Investigators, 2004). Other causes include arteritis, infection, and drug abuse, such as the use of cocaine (Blank-Reid, 1996; Hickey, 2003). While still not completely understood, the presence of periodontal disease and tooth loss is also an associated risk for ischemic stroke (Joshiyura, Hung, Rimm, Willett, & Ascherio, 2003).

As a thrombosis or emboli cause a decrease in blood supply to the brain tissue, events occur at the cellular level, referred to as the ischemic cascade. Neurons and support cells require a careful balance of variables such as temperature, pH, nutrition, and waste removal in their environment to function optimally (Hinkle & Bowman, 2003). Intensive basic scientific research during the last two decades has given healthcare professionals an increased understanding of the ischemic cascade in the format of the precise environmental alterations involved in the pathophysiology of ischemic injury at the cellular level. Understanding the ischemic cascade has led to the concept of a therapeutic time window for treatment possibilities. Often, there is a core region of dead cells surrounded by an area of hypoperfused tissue. The hypoperfused area may be rescued; this area is referred to as the penumbra region (Muir, Buchan, von Kummer, Rother, & Baron, 2006). Neuroprotection is a broad term that refers to pharmacological and nonpharmacological treatments used to halt the cellular events in the ischemic cascade, forming the theoretical basis for many of the acute stroke therapies under study (Lees et al., 2006) as well as the rationale for intervening within a therapeutic time window following ischemic stroke.

Initial Assessment

Assessment of the patient with a stroke begins with recognition of the event as a stroke in the prehospital phase of care and continues throughout care. Emergency medical technicians and ambulance staff members need training in the recognition of signs and

symptoms of stroke. Tools such as the Face Arm Speech Test (Harbison et al., 2003) and the shortened National Institutes of Health Stroke Scale (sNIHSS; Tirschwell et al., 2002) have been tested and found to be effective in increasing the diagnostic accuracy of ambulance staff. The National Association of EMS Physicians has published standards for acute stroke prehospital care (Sahni, 2000) which the AHA did not seek to duplicate but continued to emphasize the need for immediate diagnosis and evaluation (Adams et al., 2003).

In the emergency department (ED), as the patient arrives, preferably by ambulance, a suspected stroke is treated as an acute event until diagnostic evidence suggests otherwise. Neurological assessment is based on both subjective and objective data, and a careful medical history is crucial to establish the exact time of onset of stroke signs and symptoms. Essential data to include are a quick history of timing of the event, pertinent past medical history, and risk factors. The full NIHSS (Fig 1) can be used to guide the neurologic assessment (Goldstein, Bertels, & Davis, 1989; Goldstein & Samsa, 1997; Richardson, Murray, House, & Lewenkopf, 2006).

Medscape®		www.medscape.com
Category	Results	Scoring
1a. Level of consciousness (LOC; patients who score 2 or 3 on this item should be assessed using the Glasgow Coma Scale)	Alert, keenly responsive	0
	Not alert (arousable—minor stimulation)	1
	Not alert (arousable—painful stimulation)	2
	Unresponsive	3
1b. LOC questions (month, age)	Answers both correctly	0
	Answers one correctly	1
	Answers neither correctly	2
1c. LOC commands (open and close eyes, make fist, release fist)	Performs both tasks correctly	0
	Performs one correctly	1
	Performs neither correctly	2
2. Best gaze	Normal	0
	Partial gaze palsy	1
	Forced deviation	2
3. Visual	No visual loss	0
	Partial hemianopia	1
	Complete hemianopia	2
	Bilateral hemianopia	3
4. Facial palsy	Normal	0
	Minor paralysis	1
	Partial paralysis	2
	Complete	3
5. Motor function (arm)	No drift	0
	a. left Drift before 5 seconds	1
	b. right Drift before 10 seconds	2
	No effort against gravity	3
	No movement	4
6. Motor function (leg)	No drift	0
	a. left Drift before 5 seconds	1
	b. right Drift before 10 seconds	2
	No effort against gravity	3
	No movement	4
7. Limb ataxia	No ataxia	0
	Ataxia—one limb	1
	Ataxia—two limbs	2
8. Sensory	No sensory loss	0
	Mild sensory loss	1
	Severe sensory loss	2
9. Language	Normal	0
	Mild aphasia	1
	Severe aphasia	2
	Mute or global aphasia	3
10. Articulation	Normal	0
	Mild to moderate dysarthria	1
	Severe dysarthria	2
11. Extinction and inattention	Absent	0
	Mild (1 sensory modality)	1
	Severe (2 modalities)	2

Note. The full NIH Stroke Scale is available at www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

Source: J Neurosci Nurs © 2007 American Association of Neuroscience Nurses

Figure 1.

National Institutes of Health Stroke Scale (NIHSS)

Symptoms of ischemic stroke according to the areas of cerebral circulation are shown in Figure 2. In addition to these symptoms, determining dominance is important as the dominant hemisphere is primarily responsible for language function. Handedness determines dominance for most people. Right-handed people are left-hemisphere dominant; left-handed people are also left-hemisphere dominant about 60% of time. The clinical features that are more common with a dominant left cerebral hemisphere lesion include aphasia, agraphia, acalculia, apraxias, a left gaze preference, a right visual field deficit along with right-sided hemiparesis, and a right-sided hemisensory loss. Common features of a nondominant right cerebral hemisphere include neglect (left-sided hemiattention), right gaze preference, left visual field deficit, dysarthria, flat affect, left-sided hemiparesis, and left-sided hemisensory loss (Bader & Littlejohns, 2004; Hickey, 2003).

Brainstem

Hemiparesis or quadriparesis

Motor or sensory loss in all four limbs

Eye movement abnormalities, such as diplopia and dysconjugate gaze

Oropharyngeal weakness

Vertigo, tinnitus

Nausea, vomiting

Dysmetria

Cerebellum

Ipsilateral limb ataxia

Gait ataxia

Vetebrobasilar Circulation

Symptoms correlate with brainstem and cerebellar functions as above

Cranial nerve deficits in cranial nerves III–XII

Anterior Circulation Symptoms

Carotid artery

Contralateral motor and sensory loss

Amaurosis fugax or transmonocular blindness (caused by emboli to retinal artery)

Anterior Cerebral Artery

Confusion

Personality change

Incontinence

Contralateral motor or sensory loss in leg greater than arm

Middle Cerebral Artery

Contralateral motor or sensory loss (arm greater than leg)

Contralateral motor loss in lower face

Contralateral visual field loss

Language deficit (dominant hemisphere)

Spatial-perceptual deficit (nondominant hemisphere)

Posterior Cerebral Artery

Contralateral sensory loss

Ipsilateral visual field deficit

Cortical blindness

Source: J Neurosci Nurs © 2007 American Association of Neuroscience Nurses

Figure 2.

Symptoms of Ischemic Stroke According to Cerebral Circulation

The presence of a transient ischemic attack or other conditions need to be ruled out to ensure that patients receive the appropriate treatment for their condition (Tietgen, 2005;

Townend, Hanson, Sturm, & Whyte, 2005). A wide range of abnormalities can mimic a stroke, including hypoglycemia, migraine, seizure, and trauma (Fig 3).

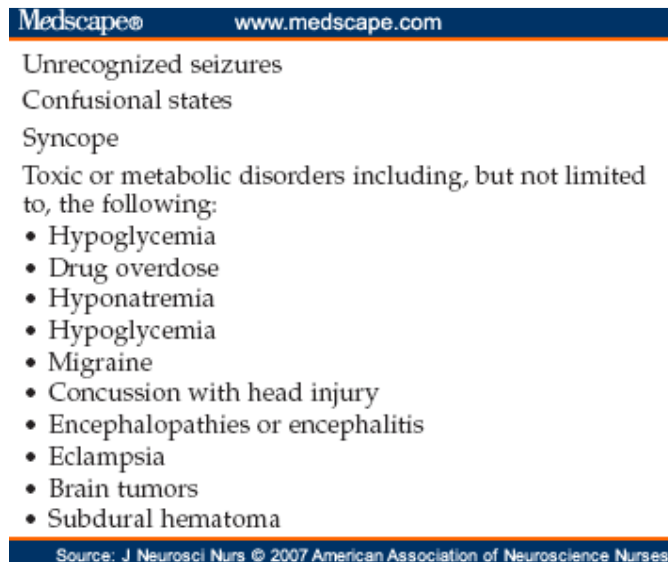


Figure 3.

Conditions that Mimic Ischemic Stroke

Diagnostic studies help to confirm stroke, detect early potentially life-threatening complications, and direct specific care given; those recommended in the AHA guidelines are shown in Figure 4 (Adams et al., 1994; Adams et al., 2003; Adams et al., 2007). These diagnostic tests are available in most EDs 24 hours a day. Blood glucose can be checked in the ambulance with a finger stick or upon ED arrival and is helpful in ruling out hypoglycemia as a cause for the event or hyperglycemia as a compounding factor. A computed tomography (CT) scan without contrast is recommended to rule out the presence of a hemorrhagic stroke that would preclude the use of thrombolysis (Muir et al., 2006). Adjunct studies may include a CT angiogram, magnetic resonance imaging (MRI), and cerebral angiography. A CT angiogram can be used to identify large vessel stenoses or occlusion. MRI allows for better visualization of possible infarcted areas, and angiography is used when intraarterial (IA) thrombolysis is indicated or when surgical interventions are being considered.

All Patients

CT of the brain without contrast
MRI can be considered at qualified centers
Electrocardiogram
Markers of cardiac ischemia
Complete blood count with platelet count
Serum electrolytes
Blood glucose
Prothrombin time, activated partial thromboplastin time, and international normalized ratio
Renal function tests
Oxygen saturation

Selected Patients

Chest X ray
Hepatic function tests
Arterial blood gas levels (if hypoxia suspected)
Lumbar puncture (if subarachnoid hemorrhage is suspected and CT is negative)
Erythrocyte sedimentation rate (ESR), syphilis serology
Lipid profile
Toxicology screen
Blood alcohol level
Pregnancy test
Electroencephalogram (when seizures suspected)

Note. CT = computed tomography; MRI = magnetic resonance imaging. Compiled from "Guidelines for the management of patients with acute ischemic stroke," by H. P. Adams, T. G. Brott, R. M. Crowell, A. J. Furlan, C. R. Gomez, J. Grotta, et al., 1994, *Stroke*, 25(9), 1901-1914, "Guidelines for the early management of patients with ischemic stroke," by H. P. Adams, R. Adams, T. Brott, G. J. Zoppo, A. J. Furlan, L. B. Goldstein, et al., 2003, *Stroke*, 34(4), 1056-1083, and "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," by H. P. Adams, G. del Zoppo, M. J. Alberts, D. L. Bhatt, L. Brass, A. Furlan, et al., 2007, *Stroke*, 38(5), 1655-1711. Copyright 1994, 2003, and 2007 by Lippincott Williams & Wilkins.

Source: J Neurosci Nurs © 2007 American Association of Neuroscience Nurses

Figure 4.

Recommended Tests in Evaluation of Acute Ischemic Stroke

Acute Stroke Teams

An element critical to the success of stroke treatment is the formation of an acute stroke team that will guide all patients through the hyperacute and acute phases of stroke care (Bonnono et al., 2000; Rapp et al., 1997). Team members include emergency medical services (EMS) personnel, ED staff members, intensive care unit (ICU) staff members, stroke physicians, radiologists or neuroradiologists, interventionalists, staff nurses from additional areas as needed, and others. Most stroke teams follow treatment protocols, guidelines, standing orders, or pathways and prospectively collect data for review to provide guidance and refine future stroke care (Holloway, Vickrey, Benesch, Hinchey, & Bieber, 2001).

The nursing focus is on patient assessment, blood pressure management, injury prevention, prevention of complications such as aspiration, and other aspects of care. Early blood pressure management is critical to prevent further ischemic injury during acute stroke. The American Stroke Association (ASA) has recommendations for blood pressure (BP) management, which are shown in [Table 1](#). The recommendations differ according to whether the patient is eligible for thrombolysis and according to their BP measurement.

The advent of thrombolytic therapy has been responsible for driving the current acute stroke protocols. Eligibility criteria for IV thrombolysis therapy must be met before administration of a thrombolytic drug. Criteria for tPA are shown in Figure 5 (Bader & Littlejohns, 2004; Kongable, 1997). IV tPA must be initiated within 3 hours of symptom onset. If the BP or blood glucose values are not within the criteria but can be brought within the recommended ranges within the 3-hour window, tPA can be administered. The tPA dose is 0.9 mg/kg, not to exceed 90 mg. It is administered by a 10% bolus followed by the remaining 90% of the dose infused during the next hour.

Inclusion Criteria

Acute ischemic stroke with a definable 3-hour time frame from onset of symptoms

Exclusion Criteria

Current use of anticoagulants or PT >15 seconds
INR >1.7

Use of heparin in the previous 48 hours and a prolonged PTT

A platelet count <100,000 K/ μ l

Another stroke or serious head injury in the last 3 months

Major surgery within the last 14 days

Systolic BP >185 mm Hg or diastolic BP >110 mm Hg

Neurologic signs that are improving rapidly

Prior ICH

Blood glucose <50 mg/dl or >400 mg/dl

Seizure at the onset of stroke

GI or urinary bleeding within the preceding 21 days

Recent MI

Severe stroke (NIHSS score >22)

Note. BP = blood pressure; GI = gastrointestinal; ICH = intracerebral hemorrhage; INR = international normalized ratio; MI = myocardial infarction; NIHSS = National Institutes of Health Stroke Scale; PT = prothrombin time; PTT = partial thromboplastin time; tPA = tissue plasminogen activator.

Figure 5.

Inclusion and Exclusion Criteria for Intravenous tPA

Nursing care for a patient during and after IV tPA infusion includes neurologic assessment every 15 minutes for 2 hours and then every 30 minutes for 6 hours to include monitoring for signs and symptoms of intracerebral hemorrhage (ICH). If a decrease in the level of consciousness or worsening neurologic deficit occurs during infusion, notify the physician and stop the infusion. There is a 50% mortality rate associated with ICH. The patient should be monitored for gastrointestinal or genitourinary hemorrhage or bleeding at puncture sites. Bleeding precautions should be instituted that include monitoring the patient in an ICU or stroke unit and avoiding placement of a nasogastric tube, indwelling bladder catheter, or IA pressure catheters in the first 24 hours following tPA administration (Adams et al., 2003).

The ability to deliver IA tPA extends the possible acute-therapy window from 3 hours to 6–12 hours for selected patients (Adams et al., 2003). The IA route for tPA is a treatment consideration when rapid recanalization is desired. IA tPA must be given within 6 hours of onset of anterior stroke symptomatology and within 12 hours of onset of posterior circulation stroke symptoms. The immediate nursing concerns are similar to those of IV tPA with the additional concern of the possibility of a retroperitoneal bleed or groin hematoma when a femoral access is used for catheterization and medication delivery (Adams et al., 2003). The need to provide adjunct therapies to maintain vessel patency after an arterial procedure may complicate this therapy. These therapies may include heparin administration to prevent acute reocclusion and use of IIb/IIIa receptor antagonists (Integrilin) to prevent acute reocclusion and endothelial injury, especially if a stent has been used. *Bridging therapy* is a term used to describe the use of both IV tPA and IA tPA. This approach is used when telemedicine is used to treat stroke in areas without neurointerventional teams available (LaMonte et al., 2003). A patient may receive IV tPA at the transferring hospital followed by IA tPA when they reach a hospital with a neurointerventional team qualified to administer IA therapy.

Anticoagulation is often used to prevent recurrent stroke. Traditional anticoagulants, such as IV heparin followed by oral warfarin sodium (Coumadin), may be used to prevent an early recurrent embolus, assist in maintaining collateral blood flow, and halt progression of additional thrombus by interfering with the clotting cascade. Antiplatelet agents such as aspirin, ticlopidine hydrochloride (Ticlid), dipyridamole (Persantine), and IIb/IIIa receptor antagonists are used to inhibit platelet aggregation for secondary stroke prevention.

The goals of acute stroke care are to support recovery, prevent complications, prevent recurrent stroke, and ensure that the patient reaches the highest possible functional outcome. This requires an interdisciplinary approach with nurses playing a major role in all aspects of care. Admission of the patient to a stroke unit is a highly rated and an agreed-upon performance measure that helps all disciplines meet these goals (Adams et al., 2003; Holloway et al., 2001).

Preventing Acute Complications

Supportive care includes continuous assessment of airway, ventilatory support, and supplemental oxygen to maintain a pulse oximetry level >92% (Adams et al., 2007). Normothermia is promoted as fever has been associated with poor outcome after stroke. The patient is monitored for cardiac arrhythmia (Adams et al., 2007).

Although the optimal management of acute arterial hypertension has not been established, there is little scientific basis and no clinically proven benefit for lowering BP (Adams et al., 2007). The consensus is that antihypertensive agents should be withheld unless the diastolic BP is >120 mm Hg or the systolic BP is >220 mm Hg ([Table 1](#)).

Medical interventions are used to induce hypertension in some centers to maintain perfusion to an ischemic area. A combination of volume expansion with albumen and intravenous medications such as phenylephrine (Neo-Synephrine) may be used to increase blood pressure and augment cerebral blood flow in an attempt to improve the patient's neurologic status (Adams et al., 2005; Rordorf, Koroshetz, Ezzeddine, Segal, &

Buonanno, 2001). The patient also needs to be monitored in an ICU for hypotension, and the BP must be supported with appropriate medications if needed.

Surgical interventions for stroke depend on the exact pathology. Hemicraniectomy may be used to control increased intracranial pressure that occurs from cerebral edema following a large hemispheric stroke. Other surgical interventions, such as carotid endarterectomy or extracranial/intracranial bypass, can help increase brain perfusion; however, such procedures are rarely performed during the acute phase of care as they are measures to prevent secondary stroke. Neurointerventional procedures in the treatment of stroke include angioplasty and carotid stenting. Angioplasty, with or without carotid stenting, may be used to improve perfusion to areas of the brain at risk. The FDA has approved a clot retrieval device that has been used in a small number of cases to restore perfusion (Felton et al., 2005). Research on clinical outcomes continues.

Neurologic examination of the patient is of vital importance particularly in the acute phase of care. The exam includes observation for changes in the patient before, during, and after therapies. Frequent nursing assessment is necessary because of the risk of increasing intracranial pressure from ICH, reperfusion injury, and cerebral edema. The NIHSS is a useful tool for assessment as it is a standardized neurologic examination intended to describe the neurologic deficits in patients with stroke, and it is reliable and valid when used by physicians and nurses (Goldstein, Bertels, et al., 1989; Goldstein & Samsa, 1997). It does not, however, contain all the components of a neurologic exam, so it cannot be used exclusively. Additional neurologic assessment needs to include assessment of size and reactivity of pupils and cranial nerves (American Association of Neuroscience Nurses [AANN], 2004).

BP management within the prescribed goals is critical to prevent secondary complications ([Table 1](#)). BP goals are dependent upon stroke etiology and individual risks. Observations for changes in the neurologic exam with BP manipulation are vital in achieving therapeutic goals.

Glycemic control is needed to prevent increase in infarction size and edema and to lower recanalization rates that can occur with acute and chronic hyperglycemia (Ribo et al., 2005). Hypoglycemia can cause further neurologic signs and lead to further brain injury. Blood glucose target range is 80–120 mg/dL (Adams et al., 2003).

Decreases in level of consciousness, tongue or throat weakness, or a decreased gag reflex may compromise the patient's airway. Nursing interventions that promote an adequate airway include assessment of the patient's ability to clear secretions. Oxygen saturation monitoring is used to evaluate oxygenation levels. The patient is positioned with his or her head midline and the head of bed elevated 30° to decrease risk of aspiration and enhance venous return (AANN, 2004). Chest physical therapy, suctioning, and incentive spirometry help to clear secretions and maintain a patent airway. Ventilator care, including weaning and evaluating for tracheostomy, is important in moving the patient through the continuum of care. An oral care guideline will aid in prevention of ventilator-associated pneumonia.

Aspiration is a major concern when caring for a patient with stroke because of limited ability to protect the airway and risk of dysphagia. Dysphagia may be caused by decreased level of consciousness or cranial nerve impairment. A swallow screen should

be completed before a patient receives anything by mouth, especially water or medications. A swallow screen should include evaluation of level of consciousness, ability to cough, phonation, pharyngeal sensation, and the ability to swallow water. A positive gag reflex should not be interpreted as a valid swallow screen, because patients with intact gag reflexes may not be effectively able to swallow. Insertion of a nasogastric or orogastric tube may be necessary to provide nutrition and medications if a patient fails a preliminary swallow screen. A gastric tube is needed if long-term impairment is anticipated. Guidelines suggest that a feeding tube should not be inserted for 24 hours after tPA administration because of increased risk of bleeding; however, a patient may need protection if vomiting and aspiration is a concern. Patients should be fed within 48 hours of admission if possible. Keeping the head of the bed elevated at least 30° may aid in preventing aspiration, but this is always done with caution and monitoring to avoid hypotension. A consult with a speech pathologist and a dietitian aids in acute care and rehabilitation planning.

The patient's ability to communicate must also be assessed, particularly after a dominant hemisphere stroke. Communication boards, an enhanced call system, or computers may help the patient communicate needs. Reassurance, reorientation, and education about the present condition and rehabilitation needs are necessary to prepare the patient for discharge. Speech therapists will also assist in the evaluation of communication skills and cognitive status.

Stroke patients often have an alteration in physical mobility related to weakness, immobility caused by disruption of movement pathways, and neglect. This increases the risk of deep vein thrombosis, pulmonary complications such as pneumonia, and falls. Elastic stockings, compression boots, and drugs such as low-molecular-weight heparin may be used to prevent deep vein thrombosis. If a patient is on bed rest, monitoring of skin integrity and a turning schedule will help prevent skin breakdown. An evaluation of motor strength, balance, range of motion, and neglect is needed. Early mobilization is recommended, if possible, within the first 48 hours (Holloway et al., 2001). Physical and occupational therapy consults are necessary to evaluate for splinting, further evaluation, and rehabilitation planning. Stroke is a life-changing event. Optimizing independence is important in the plan of care.

Secondary Prevention and Education

The patient's stroke risk factors must be assessed, and the patient and family learning needs should be addressed. A baseline assessment of the degree of knowledge deficit related to modifiable risk factors and the need for secondary prevention needs to be conducted. The degree of readiness to change must also be assessed (Miller & Spilker, 2003).

An overview of the recommendations for the secondary prevention of stroke according to the most common risk factors can be found in Figure 6. Hypertension needs to be addressed with the goal of eventually getting the systolic BP lower than 120 mm Hg and diastolic BP lower than 80 mm Hg (National Institutes of Health, 2003; Sacco et al., 2006). Patients with diabetes mellitus must have glucose levels controlled. Cholesterol levels must be evaluated, diet changes made, lipid-lowering drugs started, and cigarette smoking eliminated. Smoking-cessation interventions are most effective when started while the patient is in the hospital (Rice, 2006). Excessive alcohol use should be

eliminated. Physical inactivity must be addressed, and the benefits of an exercise program and a healthy diet leading to weight loss in obese individuals should be explained (Sacco et al.).

Medscape® www.medscape.com	
Risk Factor	Intervention
Hypertension	BP needs to be below 120/80 mm Hg
Diabetes	Glucose needs to be controlled
Cholesterol	Elevated cholesterol levels should be treated with lifestyle modifications, dietary guidelines, and medication
Cigarette smoking	Patients who have smoked in the last year should be strongly advised to quit
Alcohol	Consumption of no more than two drinks per day for men and one drink per day for nonpregnant women is advised
Obesity	Patients should be advised to maintain a BMI between 18.5 and 24.9 kg/m ² and a waist circumference of <35 inches for women and <40 inches for men
Physical activity	Patients who are capable of engaging in physical activity should engage in at least 30 minutes of moderate-intensity physical exercise most days

Note. BMI = body mass index; BP = blood pressure. From "Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack," by R. L. Sacco, R. Adams, G. Albers, M. J. Alberts, O. Benavente, K. Furie, et al., 2006, *Stroke*, 37(2), 577-617. Copyright 2006 by Lippincott Williams & Wilkins. Adapted with permission.

Source: J Neurosci Nurs © 2007 American Association of Neuroscience Nurses

Figure 6.

Stroke Risk Factors and Stroke Prevention Interventions

When new medications are prescribed for secondary prevention, the patient needs to receive education regarding their indication, duration, monitoring, and potential side effects. Warfarin (Coumadin) is the most common oral anticoagulant used in the treatment of cardiothrombotic stroke in patients with atrial fibrillation. The International Normalized Ratio is used to monitor and maintain anticoagulation in the therapeutic range of 2-3 (Sacco et al., 2006). Unless contraindicated, aspirin therapy is often recommended if anticoagulation is not used as a treatment strategy, and patients must understand the associated risks for this therapy if prescribed. Further recommendations for the prevention of recurrent stroke in a variety of other circumstances can be found elsewhere (Sacco et al.).

Educational interventions need to be tailored to the individual's education level and learning style (Hickey, 2003; Moore, 2001). Materials tailored to the needs of the patient and family include a wide variety of media. Printed materials on specific medications are available from most hospital pharmacies, many in alternate languages as needed. Videotapes or DVDs on medications and procedures are available for visual

learners. Patients who are computer savvy may prefer recommended Web sites, such as the ASA Web site (www.strokeassociation.org) and the National Stroke Association Web site (www.stroke.org). Each patient and family should receive printed materials outlining the warning signs of stroke. These warning signs are available from the ASA in both English and Spanish (ASA, 2000). Printed patient teaching materials about stroke risk factors and modifiable risk factors are also available through the ASA's Get With The Guidelines program, available at www.americanheart.org.

Education concerning rehabilitation needs and support services available may assist the patient and family during recovery. Social work and chaplaincy consults are needed in the support of patients and families. Psychiatry and neuropsychiatry can further support the patient's recovery.

Summary

All healthcare professionals want the best outcomes for people who have had an ischemic stroke. A nihilistic approach toward a patient with a diagnosis of acute ischemic stroke is no longer appropriate. Today's healthcare professionals are aware that it is important for all disciplines to work together to achieve better outcomes for patients by being knowledgeable about early and aggressive evaluation and treatment recommendations. Healthcare professionals also work hard to assure that patients and family members are knowledgeable about stroke prevention. Many important areas of patient teaching begin in the acute setting and continue in the outpatient setting, home or rehabilitation.

CE Disclaimer

The print version of this article was originally certified for CE (continuing education) credit. For accreditation details, please contact the publisher, American Association of Neuroscience Nurses (AANN), 4700 W. Lake Avenue, Glenview, IL 60025-1485

Table 1. 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 (e.g., aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy) Treat other symptoms of stroke (e.g., 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 minutes May repeat or double dose every 10 minutes (maximum 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 minutes to maximum of 15 mg/h Aim for a 10%–15% reduction in blood pressure
Diastolic >140	Nitroprusside 0.5 µg/kg/min 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 minutes May repeat 1 time or nitropaste 1–2 inches
During and after treatment	
Monitor blood pressure	Check blood pressure every 15 minutes for 2 hours, then every 30 minutes for 6 hours, and finally every hour for 16 hours
Diastolic >140	Sodium nitroprusside 0.5 µg/kg/min IV infusion as initial dose and titrate to desired blood pressure
Systolic >230 OR diastolic 121–140	Labetalol 10 mg IV for 1–2 minutes May repeat or double labetalol every 10 minutes 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 minutes to maximum of 15 mg/h; if blood pressure is not controlled by labetalol, consider sodium nitroprusside
Systolic 180–230 OR diastolic 105–120	Labetalol 10 mg IV for 1–2 minutes May repeat or double labetalol every 10–20 minutes to maximum dose of 300 mg or give initial labetalol dose, then start labetalol drip at 2–8 mg/min

Note. From "Guidelines for the early management of patients with ischemic stroke: 2005 guidelines update," by H. Adams, R. Adams, G. del Zoppo, & L. B. Goldstein, 2005, *Stroke*, 36(4), 916–921. Copyright 2005 by Lippincott Williams & Wilkins. Reprinted with permission.

References

1. Adams, H. P., Adams, R., Brott, T., Zoppo, G. J., Furlan, A. J., Goldstein, L. B., et al. (2003). Guidelines for the early management of patients with ischemic stroke. *Stroke*, *34*(4), 1056–1083.
2. Adams, H. P., Adams, R., del Zoppo, G., & Goldstein, L. B. (2005). Guidelines for the early management of patients with ischemic stroke: 2005 guidelines update. *Stroke*, *36*(4), 916–921.
3. Adams, H. P., Brott, T. G., Crowell, R. M., Furlan, A. J., Gomez, C. R., Grotta, J., et al. (1994). Guidelines for the management of patients with acute ischemic stroke. *Stroke*, *25*(9), 1901–1914.
4. Adams, H. P., Brott, T. G., Furlan, A. J., Gomez, C. R., Grotta, J., Helgason, C. M., et al. (1996). Guidelines for thrombolytic therapy for acute stroke: A supplement to the guidelines for the management of patients with acute ischemic stroke. *Stroke*, *27*(9), 1711–1718.
5. Adams, H. P., del Zoppo, G., Alberts, M. J., Bhatt, D. L., Brass, L., Furlan, A., et al. (2007). 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*, *38*(5), 1655–1711.
6. American Association of Neuroscience Nurses. (2004). *Guide to the care of the patient with ischemic stroke*. Glenview, IL: Author.
7. American Stroke Association. (2000). *Every second counts*. Dallas: Author.
8. APASS Investigators. (2004). Antiphospholipid antibodies and subsequent thrombo-occlusive events in patients with ischemic stroke. *Journal of the American Medical Association*, *291*(5), 576–584.
9. Bader, M. K., & Littlejohns, L. R. (Eds.). (2004). *AANN core curriculum for neuroscience nursing* (4th ed.). St. Louis: Elsevier.
10. Blank-Reid, C. (1996). How to have a stroke at an early age: The effects of crack, cocaine and other illicit drugs. *Journal of Neuroscience Nursing*, *28*(1), 19–27.
11. Bonnono, C., Criddle, L. M., Lutsep, H. L., Stevens, P., Kearns, K., & Norton, R. (2000). Emergi-paths and stroke teams: An emergency department approach to acute ischemic stroke. *Journal of Neuroscience Nursing*, *32*(6), 298–305.
12. Demchuk, A. M., & Buchan, A. M. (2000). Predictors of stroke outcome. *Neurologic Clinics*, *19*(2), 455–473.
13. Felton, R. P., Ogden, N. R. P., Pena, C., Provost, C., Schlosser, M. J., & Witten, C. M. (2005). The Food and Drug Administration medical device review process: Clearance of a clot retriever for use in ischemic stroke. *Stroke*, *36*(2), 404–406.
14. Goldstein, L. B., Bertels, C., & Davis, J. N. (1989). Interrater reliability of the NIH Stroke Scale. *Archives of Neurology*, *46*, 660–662.
15. Goldstein, L. B., & Samsa, G. P. (1997). Reliability of the National Institutes of Health Stroke Scale. *Stroke*, *28*(2), 307–310.
16. Goldstein, M., Barnett, H. J. M., Orgogozo, J. M., Sartorius, N., Symon, L., & Vereshchagin, N. V. (1989). Stroke—1989: Recommendations on stroke prevention, diagnosis, and therapy. Report of the WHO Task Force on Stroke

- and other Cerebrovascular Disorders. *Stroke*, 20(10), 1407–1431.
17. Harbison, J., Hossain, O., Jenkinson, D., Davis, J., Louw, S., & Ford, G. A. (2003). Diagnostic accuracy of stroke referral from primary care, emergency room physicians, and ambulance staff using the face arm speech test. *Stroke*, 34(1), 71–76.
 18. Hickey, J. V. (2003). *The clinical practice of neurological and neurosurgical nursing* (5th ed.). Philadelphia: Lippincott, Williams & Wilkins.
 19. Hinkle, J. L., & Bowman, L. (2003). Neuroprotection for ischemic stroke. *Journal of Neuroscience Nursing*, 35(2), 114–118.
 20. Holloway, R. G., Vickrey, B. G., Benesch, C., Hinchey, J., & Bieber, J. (2001). Development of performance measures for acute ischemic stroke. *Stroke*, 32(9), 2058–2074.
 21. Josphipura, K. J., Hung, H., Rimm, E., Willett, W., & Ascherio, A. (2003). Periodontal disease, tooth loss, and incidence of ischemic stroke. *Stroke*, 34(1), 47–52.
 22. Kongable, G. (1997). Code Stroke: Using t-pa to prevent ischemic brain injury. *American Journal of Nursing*, 97(11), 16bb–16hh.
 23. LaMonte, M., Bahouth, M. N., Hu, P., Pathan, M. Y., Yarbrough, K. L., Gunawardane, R., et al. (2003). Telemedicine for acute stroke triumphs and pitfalls. *Stroke*, 34(3), 725–728.
 24. Lees, K. R., Zivin, J. A., Ashwood, T., Davalos, A., Davis, S. M., Diener, H. C., et al. (2006). NXY-059 for acute ischemic stroke. *New England Journal of Medicine*, 354(6), 588–600.
 25. Miller, E. T., & Spilker, J. (2003). Readiness to change and brief educational interventions: Successful strategies to reduce stroke risk. *Journal of Neuroscience Nursing*, 35(4), 215–222.
 26. Moore, K. (2001). Managing TIA: A nursing perspective. *Transient ischemic attack (TIA) disease management guide*. Montvale, NJ: Medical Economics Company.
 27. Muir, K. W., Buchan, A. M., von Kummer, R., Rother, J., & Baron, J. C. (2006). Imaging of acute stroke. *Lancet Neurology*, 5, 744–768.
 28. National Institutes of Health. (2003). *The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7)*. Department of Health and Human Services. Retrieved November 21, 2003, from www.nhlbi.nih.gov/guidelines/hypertension/.
 29. Rapp, K., Bratina, P., Barch, C., Braimah, J., Daley, S., Donnarumma, R., et al. (1997) Code Stroke: Rapid transport, triage and treatment using rt-PA therapy. The NINDS rt-PA Stroke Study Group. *Journal of Neuroscience Nursing*, 29(6), 361–366.
 30. Ribo, M., Molina, C., Montaner, J., Rubiera, M., Delgado-Mederos, R., Arenillas, J. F., et al. (2005). Acute hyperglycemia state is associated with lower tPA-induced recanalization rates in stroke patients. *Stroke*, 36(8), 1705–1709.
 31. Rice, V. H. (2006). Nursing intervention and smoking cessation: Meta-analysis update. *Heart and Lung*, 35(3) 147–163.
 32. Richardson, J., Murray, D., House, K., & Lewenkopf, T. (2006). Successful implementation of the National Institutes of Health Stroke Scale on a stroke/neurovascular unit. *Journal of Neuroscience Nursing*, 38(Suppl. 4), 309–314.
 33. Rordorf, G., Koroshetz, W. J., Ezzeddine, M. A., Segal, A. Z., & Buonanno, F. S. (2001). A pilot study of drug-induced hypertension for treatment of acute

- stroke. *Neurology*, 56(9), 1210–1213.
34. Sacco, R. L., Adams, R., Albers, G., Alberts, M. J., Benavente, O., Furie, K., et al. (2006). Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack. *Stroke*, 37(2), 577–617.
 35. Sahni, R. (2000). Acute stroke: Implications for prehospital care. *Prehospital Emergency Care*, 4(3), 270–272.
 36. Thom, T., Haase, N., Rosamond, W., Howard, V. J., Rumsfeld, J., Manolio, T., et al. (2006). Heart disease and stroke statistics—2006 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, 113(6), e85–151.
 37. Tietgen, G. E. (2005). The risk of stroke in patients with migraine and implications for migraine management. *CNS Drugs*, 19(8), 683–692.
 38. Tirschwell, D. L., Longstreth, W. T., Becker, K. J., Gammans, R. E., Saboujian, L. A., Hamilton, S., et al. (2002). Shortening the NIH stroke scale for use in the prehospital setting. *Stroke*, 33(12), 2801–2806.
 39. Townend, B. S., Hanson, J. A., Sturm, J. W., & Whyte, S. (2005). Stroke or encephalitis? *Emergency Medicine Australasia*, 17(4), 401–404.
 40. Warlow, C., Dennis, M., van Gign, J., Hankey, G. J., Sandercock, P., Bamford, J., et al. (2001). *Stroke: A practical guide to management* (2nd ed.). London: Blackwell Science.

--

Luis H. Tello, DVM, MS
Quality Assurance Medical Advisor
Banfield The Pet Hospital
P: 503-922-5851
Fax: 503-922-6851
luis.tello@banfield.net