

Acute Stroke

3.5 contact hours - \$24

Course Expires: Nov. 1, 2010

Authors: JoAnn O'Toole, RN, BSN and Lauren Robertson, BA, MPT

ATrain Education is an approved provider of continuing nursing education by the Arizona State Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation.

AzNA and ANCC Commission on Accreditation do not approve or endorse any commercial products displayed.

ATrain is an approved provider by the Florida Board of Nursing, CE Provider #50-10593.

Instructions

1. Read the course material and then complete the following forms:
 - A. Post Test
 - B. Evaluation Learning Activity
 - C. Registration Form
2. If you are not paying by credit card, prepare a check for the amount of the course made out to: *ATrain Education, Inc.*
3. Mail the completed forms and your payment to:
ATrain Education, Inc
5171 Ridgewood Rd
Willits, CA 95490

Once we receive your forms and payment, we will mail (or email, at your request) your completion certificate. If you have any questions, please call or email Info@ATrainCEU.com.

Course Objectives

When you finish this course, you will be able to:

- Describe the incidence of stroke in the United States.
- Discuss the major risk factors for stroke and its pathophysiology.
- Identify the main structures of the brain and describe common deficits caused by stroke to those structures.
- Describe the blood flow to the anterior and posterior portions of the brain.
- Distinguish between the 3 main classifications of stroke and their clinical significance.
- Describe gender differences in the presentation of acute stroke symptoms.
- Summarize the efforts of various organizations to educate the public about stroke symptoms and emergency response.
- Describe recent advances and campaigns to improve care of stroke victims in prehospital settings.
- Discuss emergency department and hospital care of persons with acute stroke.
- Describe the role of rehabilitation therapy in the short- and long-term treatment of stroke.

A glossary appears at the end of the course.

Introduction and Epidemiology

Each year in the United States, there are more than 780,000 strokes; about 600,000 are first attacks and about 180,000 are recurrent attacks (AHA, 2008). Stroke is the third leading cause of death in the U.S. and causes more serious long-term disabilities than any other disease. More than 2,000,000 people in the United States live with the neurological after-effects of stroke and stroke accounts for half of all neurologic admissions to acute hospitals.



Nearly three-quarters of all strokes occur in people over the age of 65 and the risk of having a stroke more than doubles each decade after the age of 55 (NINDS, 2003). The estimated direct and indirect cost of stroke during 2008 was \$65.5 billion (AHA, 2008).

About 15 to 30% of people will die in the first 30 days following an ischemic stroke. The survival rate after a hemorrhagic stroke is only about 20% (Slater, 2006). Approximately 21% of men and 24% of women will die in the first year following a stroke (AHA, 2008), and only 25% recover fully. After the acute episode these conditions are the most common cause of death:

- 2 to 4 weeks after a stroke, pulmonary embolism
- 2 to 3 months following the stroke, pneumonia
- More than 3 months, cardiac disease (Slater, 2006)

Hospitalizations for stroke resulted in discharge to home (50.3%), followed by discharge to a skilled nursing facility (21.0%), discharge to another facility (19.6%), and death (8.7%). More than half of persons over 85 years of age are discharged to either a skilled nursing facility or other facility, compared with 30.3% of persons aged 65 to 74 years. Higher proportions of

women than men, and blacks than whites were discharged to a facility rather than home (NIH, 1999).

Pathophysiology

The most common identifying feature of stroke is its acute onset. The vast majority of strokes are due to ischemia, caused by **thrombotic** or **embolic** occlusions of the major arteries in the cerebrum, cerebellum, or brainstem. A thrombus is an aggregation of platelets, fibrin, clotting factors, and the cellular elements of the blood attached to the interior wall of a vein or artery, sometimes occluding the lumen of the vessel. An embolism is an abnormal circulatory condition in which a foreign body travels through the bloodstream and become lodged in a blood vessel. A smaller percentage of strokes are caused by intracerebral hemorrhage, usually due to uncontrolled hypertension.

During an ischemic stroke brain tissue is damaged when a blocked or partially-blocked artery prevents oxygen from reaching the brain. During a hemorrhage brain tissue is damaged or destroyed when blood leaks into the brain, damaging sensitive nerve cells. Physical damage depends upon which blood vessel and which part of the brain is affected. Minor occlusions can occur in the intracranial or extracranial vessels without causing neurologic signs and symptoms, due to extensive collateral connections between the arterial systems of the brain.

Risk Factors for Stroke

The risk factors for stroke overlap significantly with those for ischemic heart disease. After age, **the most important risk factor is blood pressure**. The risk of stroke increases across the measured pressures for both systolic and diastolic pressure. For each 10 mm Hg increase in systolic blood pressure or 5 mm Hg increase in diastolic pressure, the relative risk of stroke increases by a factor of 2.3. Anti-hypertensive treatments significantly reduce of risk for first and subsequent stroke (Sharma, 2005).

Diabetes and smoking each increase the relative risk of stroke by a factor of 2. Hyperhomocysteinemia is an emerging risk factor. Atrial fibrillation is also a risk factor for stroke (Sharma et al, 2005).

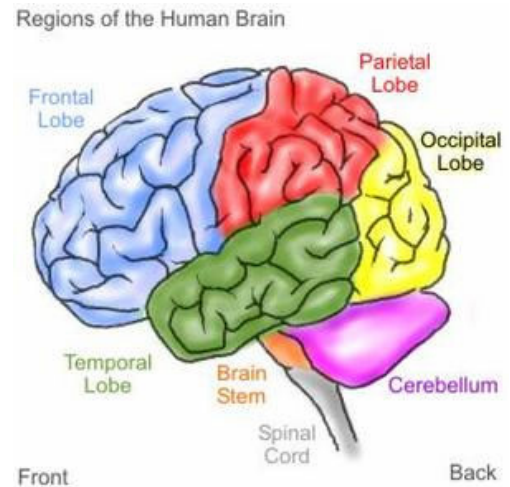
The link to serum cholesterol is somewhat more complex for stroke than for coronary artery disease. Observational studies do not show an increased risk with elevated cholesterol levels although low cholesterol may result in an increased risk of hemorrhagic stroke. In spite of this, a major therapeutic trial of lipid lowering therapy has demonstrated a reduction in ischemic stroke incidence without an increase in hemorrhagic stroke. In a sub-group within this trial, patients with prior ischemic stroke randomized to statin therapy did not show a reduced incidence of stroke; the benefit was in reducing subsequent coronary artery disease (Sharma et al, 2005).

Brain Anatomy

The brain consists of the cerebrum, cerebellum, and the brainstem. The **cerebrum** has two hemispheres, each divided into four lobes: the frontal, parietal, temporal, and occipital lobes. The lobes are named for the bones of the skull overlying them. Each lobe has extensive interaction with other lobes although specific lobes have regions that are responsible for certain cognitive functions. The nerve cells within each region are highly interconnected with other neurons in the same region, to related areas in other lobes, to areas deep in the cerebrum, and to the brainstem

and spinal cord.

The left hemisphere is referred to as the **dominant hemisphere** and the right is called the **non-dominant hemisphere**; in all the lobes, the left and right sides differ somewhat in function. Deep regions of the brain beneath the cortex contain functional groupings of cells, which are referred to as the subcortical areas of the cerebrum.



The Cerebral Cortex

Each hemisphere is covered with a thin layer of cells called the cerebral cortex. Axons arising from the estimated 100 billion cell bodies of the cerebral cortex run both horizontally and vertically and each connects with thousands of other neurons, creating a highly complex network. The cerebral cortex has historically been described by functional and structural areas such as the somatosensory, somatomotor, primary motor, visual, and auditory areas. These descriptions derived from early brain research are no longer considered to be accurate except as a broad overview. With the advent of new imaging techniques we now know that the cortex is more extensively interconnected than previously thought.

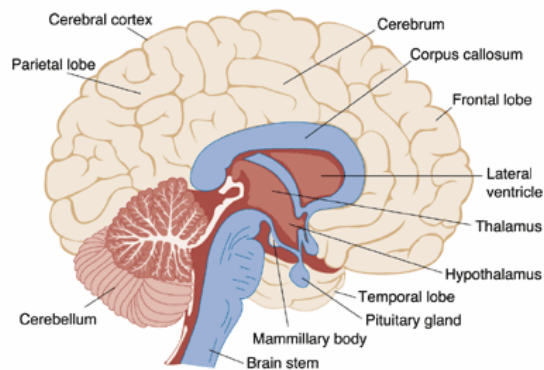
The cerebral cortex is the "thinking" and "processing" part of the brain. All cognitive functions either originate in the cerebral cortex or are processed and interpreted there. Motor commands flow out to the muscles via bundles of **efferent** nerve fibers. Sensory information from the peripheral nervous system flows into the brain for processing via bundles of **afferent** nerves.

The cerebral cortex is highly convoluted and folded, which increases the surface area of the brain—a phenomenon unique to humans. The cell bodies of the cortex have a high metabolic requirement, using six times more blood than the white matter of the brain. The interconnectedness of the nerve cells within the brain creates a flexible system, with redundancy that allows recovery of function following injury to the brain.

The cerebral cortex—especially the frontal areas—is the area of the brain most commonly affected by stroke. Damage to certain areas of the cortex is what causes the familiar stroke symptoms: speech and language dysfunction, personality changes, motor control problems, spasticity, and a host of other high level physical, cognitive, and perceptual disorders.

The Brainstem and Cerebellum

The brainstem is located above the spinal cord and beneath the thalamus and consists of the medulla oblongata, the pons, and the midbrain. The brainstem contains well-defined clusters of nerve cell bodies or nuclei that receive sensory input from the cranial nerves and send output to the thalamus for further processing. The brainstem has an ill-defined central core called the brainstem reticular formation (RF) that houses the respiratory and cardiovascular centers that influence breathing, respiration, blood pressure, circulation, and vasomotor tone.



Medial view the human brain. Oscar-Berman, M., et al. Impairments of brain and behavior: The neurological effects of alcohol. *Alcohol Health & Research World* 21(1):65–75, 1997. From: <http://www.niaaa.nih.gov/Resources/GraphicsGallery/Neuroscience/211p67.htm>.

The cerebellum, or “little brain,” is located behind and above the brainstem and makes up about 10% of the total volume of the brain. Despite its small volume, the cerebellum contains more than half of all the neurons in the brain, arranged in a highly regular and repeating pattern. It is connected to the brainstem via three pairs of peduncles (“little feet”) that are actually bundles of nerve fiber tracts entering and exiting the cerebellum. These nerve fiber tracts carry information to and from the spinal cord, cerebrum, and brainstem.

The cerebellum works with the basal ganglia and the motor portion of the cerebral cortex (motor cortex and corticospinal tract) to control and coordinate movement. Specifically, the cerebellum corrects deviations in movement by comparing one movement with another and fine-tuning subsequent movements. The cerebellum is primarily responsible for the rapid adjustments needed for normal motor activity. It also sends information to and receives information from the vestibular system and helps to control balance by controlling the axial muscles of the body.

Indicators of Cerebellum Damage

Damage to the cerebellum affects the timing and force of movement, disrupts spatial awareness, and impairs motor learning. As might be expected, a cerebellar stroke can cause vertigo, nausea, vomiting, and nystagmus. Other features of cerebellar damage include ipsilateral limb ataxia, contralateral sensory loss in the limbs, and disruption in balance, timing, and force of movement.

The hallmarks of cerebellar stroke are **ataxia**, intention tremor, and hypotonia. Ataxia is motor incoordination due to irregularities in the timing, rate and force of a muscular contraction. Ataxia manifests in unsteady, grossly uncoordinated or “drunken” gait, loss of balance, and a tendency to fall. Ataxia causes delays in initiating responses, errors in the range of a movement (dysmetria), and errors in the rate and rhythm of movements.

Intention or **action tremor** is another common type of abnormal movement associated with cerebellar damage. The tremor is not present at rest (as with Parkinson's) but occurs as soon as a movement is attempted. For example, a person may reach for a glass of water but be unable to control the force and range of the movement, especially at the end of the movement. While reaching for the glass the tremor increases, and the individual may overshoot the glass entirely, touch the glass with too much force, or lift it too rapidly. Intention tremor typically decreases at rest and increases with movement.

Finally, hypotonia occurs with cerebellar strokes. **Hypotonia** is a decreased resistance to the passive stretch of a joint. Muscles feel soft to the touch and lack normal tone. Hypotonia can be tested by tapping the patellar tendon reflex with a reflex hammer. A tap on the patellar tendon will normally produce a quick extension of the lower leg, which will come to rest after one or two swings. If cerebellar damage is present, a tap on the patellar tendon will cause the lower leg to oscillate 6 or 7 times before coming to rest. This is called a **pendular swing** and is typical of cerebellar damage.

Deficits Associated with Stroke

Motor Deficits

Paralysis and **paresis** are the two of the most common and debilitating disabilities resulting from stroke. The muscle weakness and dysfunction is usually seen in the part of the body opposite the side of the brain damaged by stroke. If the stroke occurs on the left side of the brain, the effects will be felt on the right side of the body. One-sided paralysis is called **hemiplegia** (one-sided weakness is called **hemiparesis**).

Following the acute stroke, motor recovery progresses from flaccidity and areflexia (low tone) to spasticity (too much tone). Spasticity is often followed by the development of "synergy patterns" in which the failure of muscles to relax when complimentary muscles contract leads to co-contraction of the muscles (Gould and Barnes, 2007).

Sensory Deficits

Stroke patients often experience sensory deficits in which the ability to feel touch, pain, temperature, or position is lost. Sensory deficits can hinder the ability to recognize objects by touch and can be severe enough to cause loss of recognition of one's own limbs. Some stroke patients experience pain, numbness, or odd sensations such as tingling or prickling, a condition known as **paresthesia**.

Stroke survivors can experience a variety of chronic pain syndromes resulting from stroke-induced damage to the nervous system (**neuropathic pain**). Patients who have a seriously weakened or paralyzed arm commonly experience moderate to severe pain that radiates outward from the shoulder. The most common factors associated with shoulder pain following a stroke are:

- Subluxation and capsular stretch
- Contractures
- Complex regional pain syndrome (CRPS)
- Rotator cuff injury
- Spastic muscle imbalance of the glenohumeral joint (Gould and Barnes, 2007)

In some stroke patients, pathways for sensation in the brain are damaged, causing transmission of false signals that result in the sensation of pain in a limb or side of the body that has the sensory deficit. The most common of these pain syndromes is called **thalamic pain syndrome**, which can be difficult to treat even with medications.

The loss of urinary continence is fairly common immediately after a stroke and often results from a combination of sensory and motor deficits. Stroke survivors may lose the ability to sense the need to urinate or the ability to control muscles of the bladder. Some may lack enough mobility to reach a toilet in time. Loss of bowel control or constipation may also occur. Permanent incontinence after a stroke is uncommon, but even a temporary loss of bowel or bladder control can be emotionally difficult for stroke patients.

Language Deficits

At least one-fourth of all stroke survivors experience **aphasia**, deficits in the ability to speak and understand spoken and written language. A stroke-induced injury to any of the brain's language-control centers can severely impair verbal communication. Damage to the language center known as Broca's area causes **expressive aphasia**, difficulty conveying thoughts through words or writing. Patients lose the ability to speak the words they are thinking or to put words together in coherent, grammatically correct sentences.

In contrast, damage to the language center known as Wernicke's area results in **receptive aphasia**. People with this condition have difficulty understanding spoken or written language and often have incoherent speech. Although they can form grammatically correct sentences, their utterances are often devoid of meaning.

The most severe form of aphasia, **global aphasia**, is caused by extensive damage to several areas involved in language function. People with global aphasia lose nearly all their linguistic abilities; they can neither understand language nor use it to convey thought. A less severe form of aphasia, called **anomic** or **amnesic aphasia**, occurs when there is only a minimal amount of brain damage; its effects can be subtle. People with anomic aphasia may simply selectively forget interrelated groups of words, such as the names of people or particular kinds of objects.

Cognitive Deficits

Stroke can cause damage to parts of the brain responsible for memory, learning, and awareness. Stroke survivors may have dramatically shortened attention spans or may experience deficits in short-term memory. Individuals also may lose their ability to make plans, comprehend meaning, learn new tasks, or engage in other complex mental activities.

Two fairly common deficits resulting from stroke are **anosognosia**, an inability to acknowledge the reality of the physical impairments resulting from stroke, and **neglect**, the loss of the ability to respond to objects or sensory stimuli located on one side of the body, usually the stroke-impaired side. Stroke survivors who develop **apraxia** lose their ability to plan the steps involved in a complex task and to carry out the steps in the proper sequence. Stroke survivors with apraxia may also have problems following a set of instructions. Apraxia appears to be caused by a disruption of the subtle connections that exist between thought and action.

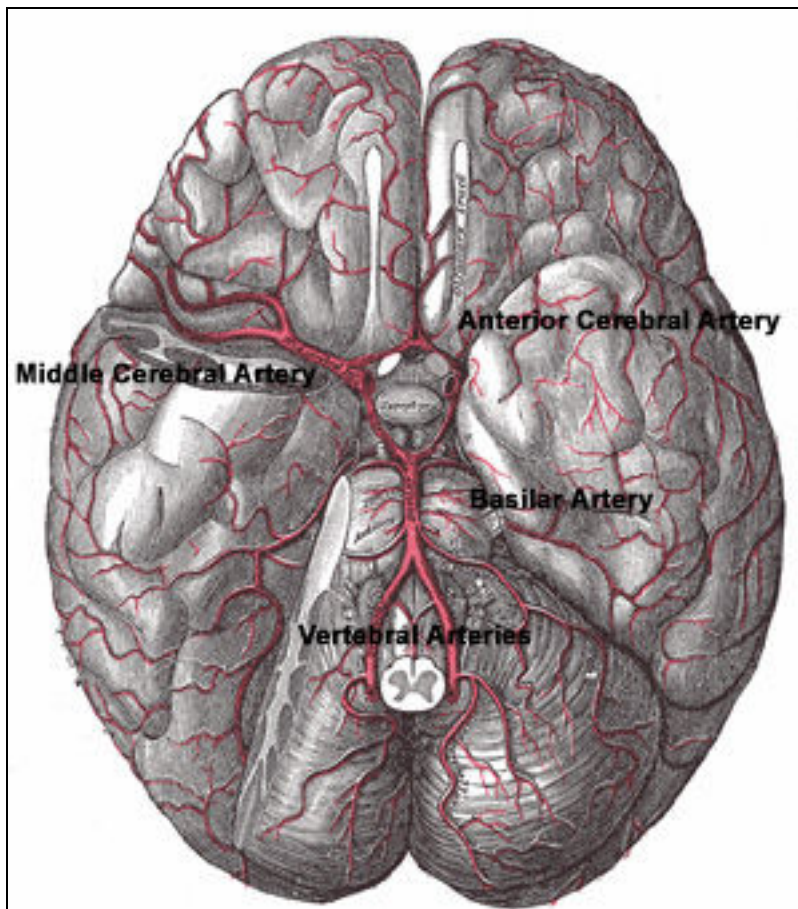
Emotional Deficits

Many people who survive a stroke feel fear, anxiety, frustration, anger, sadness, and a sense of grief for their physical and mental losses. These feelings are a natural response to the psychological and physical trauma of stroke. Alternately, some emotional disturbances and personality changes are caused by the physical effects of brain damage.

Clinical depression, which is a sense of hopelessness that disrupts an individual's ability to function, appears to be the emotional disorder most commonly experienced by stroke survivors. Signs of clinical depression include sleep disturbances, a radical change in eating patterns that may lead to sudden weight loss or gain, lethargy, social withdrawal, irritability, fatigue, self-loathing, and suicidal thoughts. Poststroke depression can be treated with antidepressant medications and psychological counseling.

Anatomy: Blood Flow to the Brain

Blood flows from the heart to the brain via two large arterial systems: the **carotid** and the **vertebrobasilar** arterial systems.



The arteries of the base of the brain.

From: http://en.wikipedia.org/wiki/Anterior_communicating_artery.

Carotid (Anterior) Circulation

The vast majority of strokes, both ischemic and hemorrhagic occur in the part of the brain supplied by the carotid circulation, which channels blood to most of the cerebral hemispheres. The three clinically-important branches of the carotid circulation are the middle cerebral artery, anterior cerebral artery, and the ophthalmic artery. The middle cerebral artery (MCA) supplies blood to the **lateral or outer surface** of the brain as well as most of the basal ganglia and parts of the internal capsule.

The anterior cerebral artery (ACA) supplies the **medial surface**, and the ophthalmic artery supplies blood to the eye and adjacent structures of the face. Deep branches from the carotid system also supply blood to the regions of the brain below the cerebral cortex—the basal ganglia and the thalamus, together formerly (and still sometimes) referred to as the extrapyramidal system.

The **middle cerebral artery** is the largest of the cerebral arteries and the most common artery involved with stroke; embolism is the most common cause of blockage (Slater, 2006). Men are affected by MCA stroke more often than women at a male-to-female ratio of 3:1 (Slater, 2006). Damage to the middle cerebral artery causes contralateral weakness and sensory loss in the face, neck, and arm (and to a lesser degree in the leg) and homonymous hemianopsia (loss of half of the visual fields of both eyes).

If a stroke occurs on the left (dominant) side of the brain, **aphasia** (Broca's and Wernicke's) can occur. Aphasia is an acquired communication disorder in people who previously had normal ability. **Dysphagia** or difficulty swallowing due to paralysis of tongue and larynx and dysarthria or the inability to articulate clearly due to facial paralysis is also common result of damage in the area of the brain served by the MCA.

If damage occurs on the right (nondominant) side of the brain, speech and comprehension are usually unaffected but other high-level deficits can occur such as behavioral changes, general confusion and disinhibition, unintentional fabrication of information, memory deficits, attentional deficits, apraxia, and neglect. **Apraxia** is the loss of the ability to organize a movement or perform a purposeful act.

The **anterior cerebral artery**, which supplies much of the medial surface of the brain is the second most common artery involved in stroke. A stroke in the ACA circulation can cause contralateral weakness and sensory loss, primarily in the leg. There may be some weakness in the contralateral arm, especially proximally.

A clot lodging in the **ophthalmic artery** leads to sudden and brief ipsilateral monocular loss of vision and **homonymous hemianopsia**. A temporary lack of blood flow to the retina may cause **amaurosis fugax** or "fleeting blindness"—often described as a "curtain" passing vertically across the visual field with complete loss of vision in that eye for a few minutes. There is a similar curtain effect as the fleeting blindness passes. Amaurosis fugax does not cause permanent damage but may be a sign of impending stroke.

Vertebrobasilar or Posterior Circulation

Blood traveling through the two vertebral arteries join together at the level of the brainstem to form the **basilar artery**. The vertebrobasilar artery supplies blood to the posterior part of the cerebral hemispheres, including the occipital lobes and the posterior portions of the temporal lobes, the cerebellum, and the brainstem.

Posterior circulation ischemia causes a variety of symptoms that are distinctly different from those found with carotid artery strokes. If the damage is in the area of the brainstem there may be loss of brainstem function, cranial nerve abnormalities (with or without hemiparesis), hemi-sensory deficits, dysphagia, and dysarthria.

Damage to the cerebellum affects motor control causing ataxia, hypotonia, dysmetria, loss of equilibrium and vertigo. Ataxia is gross motor incoordination due to cerebellar damage and is a hallmark of cerebellar stroke. Symptoms from cerebellar strokes are usually on the same side as the stroke (ipsilateral). Loss of blood flow in the posterior cerebral artery causes diplopia (double vision) and bilateral hemianopsia.

Types of Stroke

Stroke is caused by either ischemia or hemorrhage. In **ischemic stroke** there is an interruption in blood flow and decrease of oxygen vital brain tissue. A **transient ischemic attack (TIA)** lasts only a short time but should be treated as a serious neurologic event. Ischemic strokes and TIAs are closely related; both are caused by blockage or interruption of blood flow to the brain.

Hemorrhagic stroke is less common than ischemic stroke and is the result of blood seeping out of compromised blood vessels into adjacent brain tissue. Because blood is caustic, it easily damages delicate brain tissue.

Transient Ischemic Attack (TIA)

Essentials features of TIA include 1) the presence of risk factors for vascular disease, 2) focal neurologic deficit of acute onset, and 3) clinical deficit resolves completely within 24 hours (McPhee and Papadakis, 2007).

A transient ischemic attack (TIA) lasts only a few minutes. It occurs when the blood supply to part of the brain is briefly interrupted by a blockage. Transient ischemic attack symptoms, which usually occur suddenly, are similar to those of stroke but do not last as long. Most symptoms of a TIA disappear within an hour, although they may persist for up to 24 hours.

Almost 25% of patients experiencing a TIA have resolution of neurologic symptoms within five minutes and 50% have resolution within thirty minutes. If the patient's symptoms persist after one hour, there is only a 15% chance that neurologic symptoms will disappear within 24 hours. Symptoms of a TIA can include: numbness or weakness in the face, arm, or leg, especially on one side of the body; confusion or difficulty in talking or understanding speech; trouble seeing in one or both eyes; and difficulty with walking, dizziness, or loss of balance and coordination.

The majority of TIAs are caused by cardioembolism, which most commonly originate from the heart or one of the major extracranial arteries of the neck. A number of disorders increase the risk of TIA, including rheumatic heart disease, mitral valve disease, cardiac arrhythmias, infective endocarditis, atrial myxoma, and complications following myocardial infarction. Emboli that break loose from ulcerated atherosclerotic plaques in a major artery are also a cause. Patients with AIDS are at increased risk for developing TIAs and stroke (McPhee and Papadakis, 2007).

A TIA can involve either the anterior or the posterior circulation, and symptoms vary depending upon the location of the blockage. Onset is acute and without warning, and recovery is usually rapid. If ischemia affects the carotid circulation, the symptoms include weakness and heaviness on the contralateral arm, face, or leg. Numbness and sensory changes may also occur, either alone or in combination with motor deficit (McPhee and Papadakis, 2007).

Other symptoms may include monocular visual loss, dysphagia, and slowness of movement. Examination during a TIA may reveal sensory changes, hyperreflexia, an extensor plantar response, and flaccid weakness. Once the symptoms pass, examination will reveal no neurologic deficits, but carotid bruit or cardiac abnormalities may be present (McPhee and Papadakis, 2007).

A small clot (microembolus) in the ophthalmic artery, the first branch of the internal carotid artery, can cause temporary loss of vision in one eye (amaurosis fugax) and without treatment 20% of people with this condition will go on to suffer a stroke. Antiplatelet therapy decreases the rates of transient ischemic attacks by 5% (Way & Dougherty, 2003).

About 30% of people with stroke have a history of TIAs, and proper treatment is important for prevention of stroke (McPhee and Papadakis, 2007). The occurrence of a TIA is a major indicator of the overall health of the cardiovascular system. People who have suffered a TIA are at increased risk for stroke, especially in the first year after the attack. After the first year, the risk of a subsequent stroke decreases to about 8% per year. Patients with TIA who are considered at high risk for a stroke include those over the age of 60, diabetics, or those with TIAs lasting longer than 10 minutes and with weakness, speech impairment, or gait disturbance (McPhee and Papadakis, 2007).

Ischemic Stroke

Essential features of ischemic stroke include 1) sudden onset of neurologic deficits, 2) history of hypertension, diabetes mellitus, valvular heart disease, atherosclerosis, 3) distinctive neurological signs, which indicate the region of the brain involved (McPhee and Papadakis, 2007). Ischemia is caused by a reduction in the flow of oxygenated blood to a portion of the brain due to vascular occlusion. Occlusion can be caused by:

- **Embolism**, in which a foreign body travels through the bloodstream and becomes lodged in a blood vessel
- **Stenosis**, in which a blood vessel becomes narrowed
- **Thrombosis**, in which a clot, consisting of platelets, fibrin, clotting factors, and blood cells attach to the interior wall of an artery, occluding the vessel

Ischemic strokes account for 80% to 85% of all strokes; thrombotic occlusion causes approximately 55% and embolic occlusions cause approximately 25%. About 20% of ischemic strokes occur in the internal carotid arteries (anterior circulation), 20% in the vertebrobasilar arterial system (posterior circulation) and 20% in the deep penetrating arterioles in the basal parts of the brain (Westmoreland, 1994).

Although the majority of people recover from an initial stroke, the hospital mortality rate for initial stroke can be as high as 20% to 30%. More than half of those experiencing a first stroke with neurologic deficits will experience a second stroke with further neurologic damage. Even in the absence of neurologic deficit, the risk of a recurrent stroke is high, ranging from 10% to 50%, with a 33% fatality rate.

Hemorrhagic Stroke

Signs and symptoms of hemorrhagic stroke include 1) lost or impaired consciousness in 50% of patients, 2) vomiting and headache at onset of bleeding, 3) rapidly evolving neurological deficit with hemiplegia or hemiparesis (McPhee and Papadakis, 2007).

Hemorrhagic stroke represents 15 to 20% of all strokes. In the absence of neurovascular abnormalities such as aneurysm or angioma, the most common cause of spontaneous intracerebral hemorrhage is hypertension (Way et al., 2003). Bleeding into the brain can be caused by a significant increase in systolic/diastolic blood pressures over time and often occurs after activity. Hypertensive hemorrhage occurs most often in the basal ganglia and less frequently in the pons, cerebellum, thalamus, and white matter. Nontraumatic cerebral hemorrhage is also associated with bleeding disorders, anticoagulant therapy, liver disease, and brain tumors.

Bleeding into the deep portions of the brain causes loss of conjugate lateral gaze, loss of upward gaze, downward deviation of the eyes, lateral gaze palsies, and unequal pupils. If hemorrhage is in the cerebellum, there may be sudden onset of nausea and vomiting, headache, disequilibrium, and loss of consciousness. Treatment for hemorrhage is generally conservative and supportive. Surgical intervention may be indicated in the presence of a hematoma, especially in the cerebellum.

Gender Differences in Acute Stroke

Three out of five strokes in the United States occur in women, largely because women live longer than men. Several studies have indicated a potential difference in the way men and women present with stroke. As with myocardial infarction, women have a higher occurrence of nontraditional symptoms and may be less likely to be triaged and diagnosed with acute stroke (Labiche et al, 2002).

In one study women reported nontraditional symptoms 62% more often than men. This included pain, change in level of consciousness, disorientation, and non-neurologic symptoms such as nausea, non-focal weakness, and hiccups. Other non-specific symptoms included chest pain, shortness of breath and palpitations (Labiche et al, 2002).

Failure to recognize the presence of an acute stroke has severe consequences and can—and often does—delay evaluation and treatment. Pre-hospital EMS personnel and triage nurses play critical roles in identifying stroke symptoms because they are the first healthcare professionals to assess the patient and are responsible for assigning priority for evaluation by the ED physician.

Studies have shown that slower triage is associated with a worse outcome in patients with acute stroke. Other studies have shown that male patients achieve a better outcome than female patients in motor, cognitive, and functional outcomes (Labiche et al., 2002).

Public Awareness



From: <http://www.stroke.ninds.nih.gov/materials/>

Many gaps have been identified in the public knowledge of stroke symptoms. Although rapid identification and treatment clearly improve outcomes, studies have shown that the general public is largely ignorant about stroke symptoms. A telephone survey conducted in the Cincinnati area revealed that 70% of the 2,173 respondents could correctly identify at least one symptom of stroke but high-risk groups—including people over 75 years of age, males, and blacks—were the least knowledgeable. In a national U.S. phone survey of 750 adults over age 50, 42% could not identify limb numbness or weakness as stroke symptoms. Forty percent of respondents were unaware that stroke occurs in the brain. Of patients with stroke, 39% were unable to identify any symptoms of stroke. This proportion was worst for those over the age of 65 (Sharma et al, 2005). It has long been the goal of the Centers for Disease Control, in conjunction with the American Heart Association, and the American Stroke Association to increase public awareness of stroke signs and symptoms. Messages about prevention have focused on several key areas of public education including reducing high blood pressure, reducing cholesterol, improving emergency response, decreasing tobacco use, improving nutrition, increasing physical activity, decreasing obesity, and decreasing and controlling diabetes.

Blood Pressure

Sixty-five million Americans have high blood pressure, and another 59 million are pre-hypertensive. A 12 to 13 point reduction in systolic blood pressure can reduce heart attacks by 21%, strokes by 37%, and all deaths from cardiovascular disease by 25%. Nearly 70% of people with high blood pressure do not have it under control. The Dietary Approaches to Stop Hypertension (DASH) study has shown that following a healthy eating plan can both reduce a person's risk of developing high blood pressure and lower an already elevated blood pressure. Medications can also help reduce high blood pressure (CDC, 2006).

Cholesterol

A 10% decrease in total blood cholesterol levels may reduce the incidence of coronary heart disease by as much as 30%. Only 18% of adults with high blood cholesterol have it under control. Lowering saturated fat and increasing fiber in the diet, maintaining a healthy weight, and getting regular physical activity can reduce a person's risk for cardiovascular disease by lowering LDL (bad) cholesterol and raising HDL (good) cholesterol. Statins can reduce deaths from heart disease by reducing cholesterol levels (CDC, 2006).

Emergency Response

Forty–seven percent of heart attack deaths occur before an ambulance arrives and 48% of stroke deaths occur before hospitalization. Only 3% to 10% of eligible stroke victims get the emergency therapy (rt-PA) that can lead to recovery (CDC, 2006).

Tobacco

Cigarette smokers are 2 to 4 times more likely than nonsmokers to develop coronary heart disease. Cigarette smoking approximately doubles a person's risk for stroke. People who quit smoking reduce their risk of death from cardiovascular disease by half within a few years. Each year, secondhand smoke results in an estimated 35,000 deaths due to heart disease among nonsmokers (CDC, 2006).

Nutrition

Fruits and vegetables are high in nutrients and fiber and relatively low in calories. A diet that is rich in fruits and vegetables can lower a person's risk of developing heart disease, stroke, and hypertension. Grain products provide complex carbohydrates, vitamins, minerals, and fiber. A diet high in grain products and fiber can help reduce a person's cholesterol level and risk of cardiovascular disease. Foods that are high in saturated fats such as full–fat dairy products, fatty meats, and tropical oils raise cholesterol levels. People can lower their blood pressure by reducing the salt in their diets, losing weight, increasing physical activity, increasing potassium, and eating a diet rich in vegetables, fruit, and low–fat dairy products (CDC, 2006).

Physical Activity

Regular physical activity can decrease a person's risk of cardiovascular disease and prevent or delay the development of high blood pressure. People of all ages should get a minimum of 30 minutes of moderate–intensity physical activity (such as brisk walking) on most, if not all, days of the week (CDC, 2006).

Obesity

Because people who are overweight or obese have an increased risk for cardiovascular disease, diabetes, and hypertension, weight management can reduce a person's risk for these conditions (CDC, 2006).

Diabetes

Adults with diabetes have heart disease death rates about 2 to 4 times higher than adults without diabetes, and the risk for stroke is 2 to 4 times higher among people with diabetes. About 65% of deaths among people with diabetes are due to heart disease and stroke (CDC, 2006).

In addition to emphasizing public education in the areas listed above, CDC, AHA, and NSA among others have developed public health programs that emphasize quick recognition of stroke signs and symptoms. In June 1998, the Brain Attack Coalition, a group of professional, volunteer, and government entities dedicated to reducing stroke-related death and disability, reached consensus on the symptoms of stroke. Previously, standardized definitions for stroke signs and symptoms did not exist (Wall, 2008).

The consensus symptoms are: 1) sudden numbness or weakness of face, arm, or leg, especially on one side of the body; 2) sudden confusion or trouble speaking or understanding speech; 3) sudden trouble seeing in one or both eyes; 4) sudden trouble walking, dizziness, or loss of balance or coordination; 5) and sudden severe headache with no known cause. These “suddens” are commonly used to convey stroke symptoms in clinical and public health settings and among advocacy organizations concerned with stroke (Wall, 2008).

The “suddens” were adopted by several national and state-based educational campaigns. Since consensus was reached, advocacy organizations in Massachusetts have annually conducted at least one campaign on the signs and symptoms of stroke. Yet in 2003, only 18% of Massachusetts adults were aware of all signs and symptoms of stroke, but 80% would call 9-1-1 if they thought someone was having a stroke or heart attack. Because early recognition leads to early treatment and improved clinical outcomes, increasing symptom recognition could vastly improve stroke survival and quality of life (Wall, 2008).

To address the lack of recognition of stroke symptoms in their state, the Massachusetts Department of Public Health Heart Disease and Stroke Prevention and Control Program (HSPC) hired a social marketing and communications company, Geovision, Inc, to conduct research on existing messages about stroke signs and symptoms. Geovision conducted a comprehensive literature search and informal inventory of other unpublished sources. The results were used to develop an evidence-based approach to educating the lay public to recognize the signs of stroke and respond by calling 9-1-1 (Wall, 2008).

The search revealed that a comprehensive public awareness campaign that includes mass media can increase stroke recognition but should target family, coworkers, and caregivers of those at highest risk for stroke. Moreover, educational efforts should focus on behaviors that promote early seeking of hospital (Wall, 2008).

In their search for stroke education messages with evidence-based effectiveness they modified the Cincinnati Prehospital Stroke Scale (CPSS), a three-item scale based on a simplification of the National Institutes of Health Stroke Scale. The CPSS has high sensitivity and specificity for identifying stroke patients who are candidates for thrombolytic treatment when performed by a physician and has similar results when used by prehospital care providers. The CPSS has been accurately administered by untrained laypeople to identify stroke signs in mock patients and in stroke survivors when prompted by a 9-1-1 telecommunicator (Wall, 2006).

The researchers learned that the CPSS had been successfully modified by adding a fourth item, so that it could be used by laypeople before they called 9-1-1. This item provided instructions for laypeople to call 9-1-1 if they observed stroke signs in someone. What came out of this research was the development of the Stroke Heroes Act FAST campaign. In a retrospective chart review of 3500 stroke patients, the FAST method identified 88.9% of patients with stroke or transient ischemic attack (Wall, 2008).

Testing of an animation using the Stroke Heroes Act FAST message yielded positive results for message recall and aesthetics. Despite concerns from stroke educators during concept testing that FAST does not convey all the signs and symptoms of stroke it was demonstrated that the FAST acronym successfully identified 88.9% of stroke patients (Wall, 2008).






<p>Face</p>  <p>Does the face look uneven? Ask them to smile.</p>		<p>F: Does the face look uneven?</p>
<p>Arm</p>  <p>Does one arm drift down? Ask them to raise both arms.</p>	<p>Is it a stroke?</p>	<p>A: Does the arm drift down?</p>
<p>Speech</p>  <p>Does their speech sound strange? Ask them to repeat a phrase.</p>	<p>Check these signs FAST!</p>	<p>S: Does their speech sound strange.</p>
<p>Time</p>  <p>Every second brain cells die. Call 9-1-1 at any sign of stroke!</p>	<p>Call 9-1-1 at any sign of stroke.</p>	<p>T: Every second brain cells die. Call 9-1-1 at any sign of stroke.</p>

Illustration courtesy CDC.

Pre-Hospital Care

The American Heart Association recently recommended the establishment of processes to provide rapid access to EMS for people with acute stroke and promoting the use of transport and treatment protocols by EMS personnel. In addition, hospital care for acute stroke should include strategies to ensure appropriate and efficient triage and treatment of stroke patients and use of clinical pathways (i.e., procedures and protocols outlined through a decision tree, usually provided on a paper document attached to a medical chart) (Tsai, 2008).

First responders, EMTs, and paramedics are often the first source of medical information and play a role in the initial triage of the potential stroke victim. Failure to recognize and report both traditional and nontraditional symptoms of stroke can mislead hospital triage teams, causing them to defer the patient's immediate evaluation and treatment. One study showed that between 29% and 65% of people with symptoms of stroke access the medical care via their local EMS and the majority of these people present within the 3-hour time period (Saver, 2007).

The ability to reach the emergency department within 3 hours of onset is an important goal. The use of time-dependent medications and treatments for the acute treatment of stroke has decreased morbidity and mortality following acute stroke. Because of these new drugs, training of emergency medical personal (EMS) in the rapid identification of stroke has become critical issue. EMS personnel are being trained to approach stroke as an urgent medical emergency similar to acute myocardial infarction.

In 2006, the Minnesota Department of Health (MDH) and the Minnesota Stroke Partnership (MSA) conducted two surveys. The survey for emergency medical services organizations, mailed to every licensed ambulance service in Minnesota, asked about transportation policies and training needs. The survey for hospitals, mailed to every hospital in the state, asked about capacity to treat acute stroke (Tsai, 2008).

In the portion of the survey assessing stroke patient transportation and advance notification, less than two-fifths (37%) of respondents reported the existence of a written transportation protocol for stroke patients. Ninety-one percent would not bypass a hospital to send a patient to a destination with greater capacity for stroke care; according to written comments, ambulances take patients to a hospital chosen by the patient or patient's family.

Most ambulances in the MDH survey (78%) provide advance notice to hospitals about stroke patients needing urgent care, with notification on scene (10%), immediately en route (21%), or as soon as possible en route (54%); the questionnaire did not define the difference between "immediately" and "as soon as possible." Ten percent of ambulance services alert the destination hospital within 5 minutes before reaching it. Air transport is used infrequently; 70% never use a helicopter (Tsai, 2008).

During prehospital care and management, seventy-six percent of services surveyed indicated they have a written protocol for prehospital stroke management. The Cincinnati Stroke Scale (a modified version of the National Institutes of Health Stroke Scale) is the most commonly used stroke scale assessment tool. The stroke scale score is reported to hospital staff "sometimes" in 21% of cases, "always" in 34% of cases, and "rarely" or "never" in 36% of cases; 9% of respondents did not know whether the stroke scale score is reported to hospital staff (Tsai, 2008).

A majority (60%) of respondents surveyed reported their personnel are trained on stroke at least once a year. Nearly 30% reported their personnel receive stroke training once every 2 or 3 years. Classroom training was the most commonly cited format, followed by DVDs or videos. Other types of training reported include run reviews and Advanced Cardiac Life Support refresher courses. Most respondents reported offering training opportunities annually (56%) or semiannually (24%) is most effective (Tsai, 2008).

Forms of training reported most often as most effective by those surveyed include agency-sponsored conferences and seminars, regional conferences, Internet-based training, and statewide conferences. Agency-sponsored conferences or seminars was the kind of training most commonly reported effective for Emergency Medical Technician (EMT)-Basic (79%) and, to a lesser degree for paramedics (32%). This form of training also was reported as effective for paramedics (32%). Internet-based training was the second most common method for paramedics (22%), with regional (18%) and statewide (16%) conferences cited about equally. Teleconferences, videoconferences, and national conferences were least frequently cited as effective training vehicles (Tsai, 2008).

The Minnesota Stroke Partnership made several recommendations as a result of the survey:

- Prehospital EMS personnel should continue to treat stroke as an emergent event.
- First responders should be taught to recognize the signs of stroke and to communicate such information to ambulance personnel.
- All ambulance service organizations should be encouraged to use the Cincinnati Stroke Scale as the standard for assessing patients suspected of having stroke.
- Hospital ED staff should be informed that the Cincinnati Stroke Scale standard is being encouraged, and prehospital providers should include instructions in their standard operating procedures to always verbally provide stroke scale information to hospital ED staff.
- Prehospital EMS providers should be informed about the locations of primary stroke centers and educated about recent recommendations for stroke systems of care.
- Training should be provided for prehospital EMS providers on stroke issues annually or semiannually. This training should be offered, but not mandated, for ambulance personnel.
- Classroom education or regional conferences and seminars should be provided. Internet-based training also should be provided as an option for continuing education (Tsai, 2008).

These recommendations were in agreement with those put forth by the Expert Panel on Emergency Medical Services Systems and the Stroke Council (Acker et al, 2007), which examined the challenges associated with integrating emergency medical services activation and response within stroke systems of care.

The panel made the following recommendations, which were approved by the American Heart Association Science Advisory and Coordinating Committee in 2007:

- For activating and dispatching the emergency medical services response for stroke patients, stroke systems should require appropriate processes that ensure rapid access to EMS for acute stroke patients.
- For EMS responders, EMS services should use protocols, tools, and training that meet current ASA/AHA guidelines for stroke care.
- Prehospital providers, emergency physicians, and stroke experts should collaborate in the development of EMS training, assessment, treatment, and transportation protocols for stroke.
- Patients should be transported to the nearest stroke center for evaluation and care if a stroke center is located within a reasonable transport distance and transport time (Acker et al, 2007).

Emergency Department and Hospital Care

Because there is no way to tell whether symptoms are from a transient attack or an acute stroke, assume that all stroke-like symptoms signal an emergency and do not wait to see if they go away. A prompt evaluation (within 60 minutes) is necessary to identify the cause of the event and determine appropriate therapy. Depending on the patient's medical history and the results of a medical examination, the physician may recommend drug therapy or surgery to reduce the risk of stroke.

In the emergency department, acute stroke patients should be identified as quickly as possible to determine those eligible for thrombolytic therapy, which must be administered within 3 hours of known onset of stroke symptoms. ER personnel are trained to "think fast" meaning they look for (F) facial droop, (A) arm drift, (S) slurred speech, and (T) time to act quickly.

Patients seen as ineligible for thrombolytic therapy immediately undergo a rapid secondary categorization to establish their treatment plan. Response systems—including optimal time frames—must be established, maintained, and monitored in all emergency departments. The goal should be to:

- 1) Perform an initial patient evaluation within 10 minutes of arrival in the emergency department.
- 2) Notify the stroke team within 15 minutes of arrival.
- 3) Initiate a CT scan within 25 minutes of arrival.
- 4) Interpret the CT scan within 45 minutes of arrival.
- 5) Ensure a door-to-drug (needle) time of 60 minutes from arrival.
- 6) Transfer the patient to an inpatient setting within 3 hours of arrival. (AHA, 2006)

The American Heart Association recommends that all emergency departments assess the severity of stroke using the National Institutes of Health (NIH) Stroke Scale. The NIHSS is used to evaluate the level of impairment sustained by a stroke patient, immediately and at intervals post onset. The Stroke Scale is summarized in Table 2.

The NIHSS scores range from 0 to 42 with the higher score indicating more severe deficits. Although a level of neurologic deficit on the NIHSS has not been established for treatment with thrombolytics, the Federal Drug Administration has included a package insert in rt-PA listing a score greater than 22 a warning. Additionally, patients with a score higher than 22 are at greater risk for hemorrhage transformation if they are given rt-PA. Table 3 shows the prognostic value of the NIH Stroke Scale for patients who are **not** treated with a thrombolytic drug such as rt-PA.

Table 2: Modified NIH* Stroke Scale Summary (NINDS, 2003)

Item	Name	Response
1A	Level of consciousness	0=Alert 1=Not alert 2=Unresponsive
1B	Level of questions	0=Answers both questions correctly 1=Answers one question correctly 2=Answers neither correctly
1C	Level of commands	0=Performs both tasks correctly 1=Performs one task correctly 2=Performs neither task
2	Best gaze	0=Normal 1=Partial gaze palsy 2=Total gaze palsy
3	Visual fields	0=No visual loss 1=Partial hemianopsia 2=Complete hemianopsia 3=Bilateral hemianopsia
4	Facial palsy	0=Normal 1=Minor paralysis 2=Partial paralysis 3=Complete paralysis
5	Motor arm	a. Left or b. Right <ul style="list-style-type: none"> • 0=No drift • 1=Drift before 10 sec • 2=Fall before 10 sec • 3=No effort against gravity • 4=No movement
6	Motor leg	a. Left or b. Right <ul style="list-style-type: none"> • 0=No drift • 1=Drifts before 5 sec • 2=Fall before 5 sec • 3=No effort against gravity • 4=No movement
7	Ataxia	0=Absent 1=One limb 2=Two limbs
8	Sensory	0=Normal 1=Mild loss 2=Severe loss
9	Language	0=Normal 1=Mild aphasia 2=Severe aphasia 3=Mute or global aphasia
10	Dysarthria	0=Normal 1=Mild 2=Severe
11	Extinction/inattention	0=Normal 1=Mild 2=Severe
12	Distal motor	0= Normal 1=Some extension after 5 seconds 2=No extension after 5 seconds

Table 3 Prognostic Value of NIHSS* In Patients Not Treated With t-PA

NIHSS score	Prognosis
>20 + age >75	Mortality rate 45% (48% with t-PA)
>17 + atrial fibrillation	Positive predictive value for poor outcome 96%
=16	High probability of death or severe disability
>7	Only 2.5% of patients functionally normal at 48 hours
=7	45% of patients functionally normal at 48 hours
=6	Good spontaneous recovery

*National Institutes of Health Stroke Scale

For hemorrhagic strokes the Glasgow Coma scale is used to determine level of consciousness and severity of the underlying change of consciousness. A maximum score of 14 to 15 indicates mild dysfunction; 11 to 13 indicates moderate to severe dysfunction, and a score of 10 or below indicates severe dysfunction (AHA, 2006).

Imaging Studies

The most important function of an imaging study for acute stroke is to differentiate between ischemic and hemorrhagic stroke. It can also be used to rule out other conditions such as tumors. Further, imaging studies can be used to determine the extent of the ischemic damage and to guide the use of therapies.

A noncontrast cranial CT scan is the standard with which other brain imaging techniques are compared although it is relatively insensitive in detecting acute and small cortical or subcortical infarctions, especially in the posterior fossa (Adams, 2007). A CT scan demonstrates whether stroke is ischemic or hemorrhagic in 95% of cases; it can also define almost all intracranial hematomas >1 cm, as well as more than 95% of subarachnoid hemorrhages.

Although standard MRI sequences (T1 weighted, T2 weighted, and proton density) are relatively insensitive to the changes of acute ischemia (Adams, 2007), multi-modal MRI can be used for emergency diagnosis and has some advantages when compared to CT. Despite the drawbacks of MRI, its use is increasing in acute stroke centers.

In general MRI has some advantages over CT:

- Better gray- and white-matter resolution of all parenchymal structures
- Better identification of normal and abnormal tissue
- Ability to perform multiplanar images with ease
- Does not involve radiation
- Shows evidence of ischemic stroke earlier than CT

Disadvantages of MRI are:

- Not generally available.
- More expensive than CT.
- Difficult to monitor the seriously ill patient while in scanner.
- Requires greater time than CT.
- Motion artifacts are a serious problem.
- Acute intracranial hemorrhage can be easily missed.

Monitoring Blood Pressure

Acute stroke produces an increase in blood pressure in approximately 80% of patients. Blood pressure must be monitored frequently. Control of blood pressure is important for the following reasons:

- Significant elevation in blood pressure is an exclusion criterion for administration of rt-PA.
- Elevated blood pressure after administration of rt-PA is associated with increased risk of intracerebral hemorrhage.

Thrombolytics

Thrombolytic agents are used to dissolve clots when the presence of a blood clot seriously diminishes the flow of blood to some part of the body. **Recombinant tissue plasminogen activator**, or **rt-PA** (generic name, alteplase; drug name, Activase) is a thrombolytic drug used successfully for treatment of acute ischemic stroke in those people who meet the inclusion/exclusion criteria for its administration (McPhee and Papadakis, 2007).

Table 4a Inclusion Criteria for Thrombolytic Therapy (t-PA) (AHA, 2006)

Inclusion Criteria

- Ischemic stroke by clinical evaluation
- Initiation of treatment within 3 hr of onset of symptoms (when patient was last seen to be normal)
- Neurologic deficit measured by NIH stroke scale (NIHSS)
- Cranial CT negative for hemorrhage

Table 4b Exclusion Criteria for Thrombolytic Therapy (t-PA) (AHA, 2006)

Exclusion Criteria

- Evidence of acute intracerebral or intracranial hemorrhage on noncontrast CT scan of head
- Symptoms suggesting subarachnoid hemorrhage
- Multilobar infarction on CT scan
- History of intracranial hemorrhage
- Pretreatment systolic blood pressure >185 or diastolic >110, or, if aggressive treatment is required not reduced to 185/110 or lower at time treatment should begin
- Known intracranial neoplasm, arteriovenous malformation, or aneurysm
- Witnessed seizure at the onset of stroke
- Active internal bleeding or acute trauma
- Platelet count < 100,000 per mm³
- Heparin within past 48 hr and prolonged activated partial thromboplastin time (aPTT)
- Current use of anticoagulant with INR >1.7 or PT time >15 sec
- Other stroke, serious head trauma, or intracranial surgery within past 3 months
- Arterial puncture at a noncompressible site in past 7 days

Relative Contraindications / Precautions

- Rapid improvement of neurologic deficits based on clinical evaluation
- Major surgery in past 14 days
- GI or GU hemorrhage in past 21 days
- Recent acute MI (within past 3 months)
- Postmyocardial infarction pericarditis
- Glucose concentration <50 mg/dL or >400 mg/dL

In an important study conducted by the National Institute of Neurological Disorders and Stroke and published in *New England Journal of Medicine* in 1995, alteplase was shown to improve neurologic outcome significantly in approximately 10% of patients with ischemic stroke—especially those with ischemic stroke in the carotid or vertebrobasilar circulation (NINDS, 1995). As a result of the NINDS study, alteplase was approved by the Food and Drug Administration (FDA) in 1996 for treatment of acute stroke and was designated by the American Heart Association (AHA) as an optional treatment (class IIb).

The NINDS alteplase study was conducted at nine centers across the United States and involved 624 patients who received either intravenous alteplase or a placebo within three hours of the onset of stroke symptoms. The results of the study indicated that the number of patients with complete or almost complete recovery was increased by 30% over patients receiving the placebo, as measured by four medical outcome measures (NINDS, 1995).

The NINDS study was a major breakthrough in the treatment of ischemic stroke. The Federal Drug Administration (FDA), American Heart Association (AHA), the National Stroke Association (NSA), and the media touted the effectiveness of alteplase. The AHA launched a nationwide campaign that encouraged use of the term **brain attack** instead of stroke, largely based on the purported effectiveness of alteplase.

The complication of greatest concern in the use of alteplase for acute stroke is intracranial hemorrhage occurring after administration of the drug. According to the original NINDS study, hemorrhage is most likely to occur in the first three hours after infusion but may occur up to 36 hours later. In the original study intracranial hemorrhage occurred within 36 hours of treatment in 6.4% of the NINDS patients who received t-PA as compared to 0.6% who received the placebo (NINDS, 1995). Nearly half of those hemorrhages were fatal.

In subsequent clinical studies in the United States, Canada, and Germany, strict adherence to the NINDS protocol has produced promising results, similar to the original NINDS study. Adverse effects, such as increased intracranial hemorrhage, dependence, and death, and poor long-term outcomes have been reported in a number of studies, although significant deviation from the NINDS protocol occurred in many of these studies (Gladstone & Black, 2001). Deviations from protocol included failure to control blood pressure, failure to recognize symptoms of hemorrhage on a CT scan, administration of t-PA later than three hours after onset of stroke, and inclusion of patients who did not meet the inclusion/exclusion criteria (Gladstone & Black, 2001).

Thrombolysis for acute stroke has received widespread, though not universal, support. It is a complex intervention making intensive use of resources and personnel, with a narrow therapeutic window. The Brain Attack Coalition (BAC) in the U.S. and the Heart and Stroke Foundation of Canada (HSFC) advocate for multilevel system changes to increase the number of patients eligible for, and receiving, thrombolytic therapy. Such advocacy has influenced public policy (Sharma et al, 2005).

The use of rt-PA for acute stroke is steadily evolving. Sattin and others have proposed an **expedited code stroke protocol** that recommends the use of rt-PA earlier than 3 hours and have recommended an onset-to-treatment time of 2 hours (Sattin et al, 2006). Portions of this expedited protocol are already in use in many stroke centers in the United States. The key features of the expedited code stroke protocol are:

- 2-hr onset-to-treatment benchmark
- In-person triage of all code stroke calls
- Unmixed rt-PA at bedside
- Proceed without coagulation test results unless specifically indicated
- Proceed without chest x-ray unless specifically indicated
- No delay for formal CT interpretation
- No delay for written consent (Sattin et al, 2006)

In a recent study published in the New England Journal of Medicine researchers administered alteplase or a placebo to 821 patients 3 to 4.5 hours following onset of symptoms of acute stroke. The results indicate that compared with a placebo, intravenous alteplase administered 3 to 4.5 hours after the onset of symptoms significantly improved clinical outcomes without an increase in the rate of symptomatic intracranial hemorrhage (Hacke, et al, 2008).

Antiplatelets

The use of antiplatelet agents, particularly aspirin, is a standard treatment for patients at risk for stroke. Antiplatelets act to prevent the blood from clotting. Aspirin is the oldest and most common antiplatelet agent. Other antiplatelets include ticlopidine, clopidogrel, and an extended-release antiplatelet containing dipyridamole plus aspirin.

The AHA and ASA recommend 325 mg of aspirin within 24 to 48 of an acute stroke. Other recommendations include:

- Aspirin should not be considered a substitute for other acute interventions for treatment of stroke, including the intravenous administration of rtPA.
- The administration of aspirin as an adjunctive therapy within 24 hours of thrombolytic therapy is not recommended.
- The administration of clopidogrel alone or in combination with aspirin is not recommended for the treatment of acute ischemic stroke.
- Outside the setting of clinical trials, the intravenous administration of antiplatelet agents that inhibit the glycoprotein IIb/IIIa receptor is not recommended (Adams, 2007).

Anticoagulants

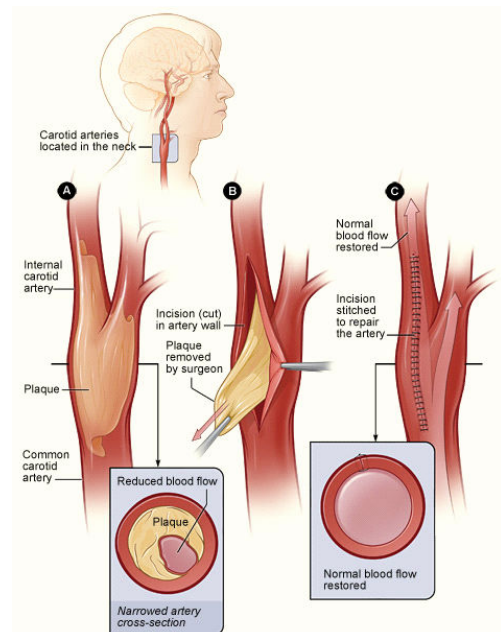
Anticoagulants—sometimes referred to as “blood thinners”—make it harder for the blood to clot. Warfarin, coumadin, and heparin are anticoagulants that may be prescribed for people with atrial fibrillation. They have been in use for the treatment of acute ischemic stroke for more than 50 years (NINDS, 2005).

Recent studies have demonstrated that early administration of heparin or low-molecular weight (LMW) heparins/danaparoid does **not** lower the risk of early recurrent stroke or lessen the risk of early neurological worsening. In addition, the administration of these drugs is associated with an increased risk of bleeding complications (Adams, 2007). The AHA has made the following recommendations:

- Urgent anticoagulation with the goal of preventing early recurrent stroke, halting neurological worsening, or improving outcomes after acute ischemic stroke is **not** recommended for treatment of patients with acute ischemic stroke.
- Urgent anticoagulation is **not** recommended for patients with moderate to severe strokes because of an increased risk of serious intracranial hemorrhagic complications.
- Initiation of anticoagulant therapy within 24 hours of treatment with intravenously administered rtPA is **not** recommended (Adams, 2007).

Carotid Endarterectomy

Carotid endarterectomy is a surgical procedure in which fatty deposits are removed from one of the two carotid arteries. It is performed to prevent stroke. About 180,000 carotid endarterectomies are performed in the United States each year (Kracjer, 2005). The surgery has been found highly beneficial for persons who have already had a stroke or experienced the symptoms of a stroke and have a severe stenosis of 70 to 99%. In this group, surgery reduces the estimated 2-year risk of stroke or death by more than 80%, from greater than 1 in 4 to less than 1 in 10 (NINDS, 2004).



http://www.nhlbi.nih.gov/health/dci/images/cad_endarterectomy.jpg

The carotid endarterectomy procedure was first described in the mid-1950s. It began to be used increasingly as a stroke prevention measure in the 1960s and 1970s. Its use peaked in the mid-1980s at which time several authorities began to question the trend and the risk-benefit ratio for some groups, and the use of the procedure dropped precipitously. Two clinical trials were performed in the early 1990s. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) evaluated the benefit of the procedure in symptomatic patients and the Asymptomatic Carotid Atherosclerosis Study (ACAS) evaluated the benefits in asymptomatic patients (Kracjer, 2004).

For patients who have already had transient or mild stroke symptoms due to moderate carotid stenosis (50 to 69%), surgery reduces the 5-year risk of stroke or death by 6.5%. The failure rate for ipsilateral stroke or death for the medical group is 22.2%, and for the surgery group is 15.7% from greater than 1 in 4 to less than 1 in 7. The procedure has also been found highly beneficial for persons who are symptom-free but have a carotid stenosis of 60 to 99%. In this group, the surgery reduces the estimated 5-year risk of stroke by more than one-half, from about 1 in 10 to less than 1 in 20 (NINDS, 2004).

In one study, carotid endarterectomy and aspirin was shown to be more beneficial than aspirin alone. Patients with stenoses of less than 50% did not benefit from surgery (Way et al., 2003). In

another study, patients with carotid stenoses greater than 60% were given either a carotid endarterectomy or medical therapy consisting of antiplatelet drugs and reduction of risk factors (eg, smoking cessation, treatment of hypertension, reduction of cholesterol). After five years, the risk for stroke or death was less in the surgical group (5.1%) than in the medical therapy group (11%) (Way et al., 2003).

For stroke prevention, women undergo fewer angiograms and carotid endarterectomies compared with men (Labiche et al, 2002).

Stroke Centers

In 2000, the Brain Attack Coalition (BAC) recommended the creation of 2 types of stroke centers: primary and comprehensive. A primary stroke center has the resources and staff to treat most uncomplicated acute strokes and to administer thrombolytics. There are currently 648 accredited primary stroke centers in the United States.

A comprehensive stroke center (CSC) is defined as a facility or system with the necessary personnel, infrastructure, expertise, and programs to diagnose and treat stroke patients who require a high intensity of medical and surgical care, specialized tests, or interventional therapies (Albert et al, 2005). There are currently about 200 comprehensive stroke centers in the United States.

Rehabilitation Following Stroke

After the acute phase, physical, occupational and speech therapy play an important role in recovery for those with motor, sensory, or cognitive deficits. Early mobilization helps prevent contractures and active movement improves strength and reinforces normal movement patterns. The types and degrees of disability following a stroke are determined by which area of the brain is damaged.

In the United States approximately two-thirds of the more than 780,000 people who suffer a stroke each year survive and require rehabilitation. The goals of rehabilitation are to help survivors become as independent as possible and to attain the best possible quality of life. Even though rehabilitation does not cure the damage caused by the stroke, rehabilitation can help people achieve a good long-term outcome (NINDS, 2002).

Rehabilitation helps stroke survivors relearn skills that are lost when part of the brain is damaged. These skills can include coordinating leg movements in order to walk, or carrying out the steps involved in a complex activity. Rehabilitation also teaches survivors new ways of performing tasks that circumvent or compensate for any residual disabilities (NINDS, 2002).

Patients may need to learn how to bathe and dress using only one hand, or how to communicate effectively when their ability to use language has been compromised. The most important element in any rehabilitation program is carefully directed, well-focused, repetitive practice—the same kind of practice used by all people when they learn any new motor or cognitive skill (NINDS, 2002).

Rehabilitative therapy begins in the acute-care hospital after the patient's medical condition has been stabilized, usually 24 to 48 hours after the stroke. The first steps involve promoting independent movement, because many patients are paralyzed or seriously weakened. Patients are taught to change positions frequently while lying in bed and to engage in range-of-motion (ROM) exercises to strengthen their stroke-impaired limbs (NINDS, 2002).

Passive ROM exercises are those in which the therapist actively moves a limb repeatedly; active or active-assisted ROM exercises are performed by the patient with physical assistance from the therapist. Patients progress from sitting up and transferring between the bed and a chair to standing, bearing their own weight, and walking, with or without assistance (NINDS, 2002).

Rehabilitation therapists help patients perform progressively more complex and demanding tasks, such as bathing, dressing, and using a toilet, and they encourage patients to begin using their stroke-impaired limbs while engaging in those tasks. Beginning to reacquire the ability to carry out these basic activities of daily living represents the first stage in a stroke survivor's return to functional independence (NINDS, 2002).

Successful rehabilitation requires a coordinated interdisciplinary approach. The goal of rehabilitation is to reduce morbidity and mortality, reduce the length of the hospital stay, and reduce or eliminate the need for long-term care. Many factors affect the outcome of care, including the presence of an organized plan of care, the skill of the rehabilitation team, and the cooperation of the patient, family, and friends (NINDS, 2002).

The earlier rehabilitation begins, the more likely survivors are to regain lost abilities and skills.

Rehabilitation helps an individual who has experienced a stroke reach the highest possible level of independence. Because stroke survivors often have complex rehabilitation needs, progress and recovery are unique for each person. Although a majority of functional abilities may be restored soon after a stroke, recovery is an ongoing process and improvement may be possible for years after the stroke has occurred (NINDS, 2002).

Facilities for Stroke Rehabilitation

Rehabilitation services have been shown to play a critical role in recovery from acute stroke, although the quality of service varies widely depending on location. Specialized rehabilitation facilities are unevenly distributed and almost totally absent in non-urban and rural communities. A 1991 analysis of Medicare claims showed that patients receiving rehabilitation services were distributed as follows:

- 17% were admitted to hospital-based inpatient rehab programs
- 23% were treated in skilled nursing facilities
- 40% received outpatient or home care services (NINDS, 2002)

It is not known what percentage of the stroke population receive which services but evidence suggest that only 16% receive full services and as many as 25% of stroke patients receive no rehabilitation services whatsoever (NINDS, 2002).

Inpatient Rehabilitation Units

Inpatient facilities may be freestanding or part of larger hospital complexes. Patients stay in the facility, usually for 2 to 3 weeks, and engage in a coordinated, intensive program of rehabilitation. Such programs often involve at least 3 hours of active therapy a day, 5 or 6 days a week. Inpatient facilities offer a comprehensive range of medical services, including full-time physician supervision and access to the full range of therapists specializing in poststroke rehabilitation (NINDS, 2002).

Outpatient Units

Outpatient facilities are often part of a larger hospital complex and provide access to physicians and the full range of therapists specializing in stroke rehabilitation. Patients typically spend several hours, often three days each week, at the facility taking part in coordinated therapy sessions and then return home at night. Comprehensive outpatient facilities frequently offer treatment programs as intense as those of inpatient facilities, but they may also offer less demanding regimens, depending on the patient's physical capacity (NINDS, 2002).

Nursing Facilities

Rehabilitative services available at nursing facilities are more variable than are those at inpatient and outpatient units. Skilled nursing facilities usually place an emphasis on rehabilitation, whereas traditional nursing homes emphasize residential care. In addition, fewer hours of therapy are offered compared to outpatient and inpatient rehabilitation units (NINDS, 2002).

Home-Based Rehabilitation Programs

Home rehabilitation allows for great flexibility so that patients can tailor their program of rehabilitation and follow individual schedules. Stroke survivors may participate in an intensive level of therapy several hours per week or follow a less-demanding regimen. These arrangements are often best suited for people who lack transportation or who require treatment by only one type of rehabilitation therapist (NINDS, 2002).

Patients dependent on Medicare coverage for their rehabilitation must meet Medicare's "homebound" requirements to qualify for such services; at this time, lack of transportation is not a valid reason for home therapy. The major disadvantage of home-based rehabilitation programs is the lack of specialized equipment. However, undergoing treatment at home gives people the advantage of practicing skills and developing compensatory strategies in the context of their own living environment (NINDS, 2002).

Conclusion

The onset of stroke is a dramatic and discouraging event both for the person with the stroke and for the family. A person may go from complete independence to complete dependence in a matter of minutes. Upon entering the medical system, the person with a stroke is confronted with a bewildering array of tests, people, and places, as well as confusion about treatment and services.

Medical care for stroke survivors is often fragmented and inconsistent. In some cases the patient is moved quickly from the hospital to a skilled nursing facility for rehabilitation. In urban areas, care may be more comprehensive and organized, but in many other areas follow-up care may be hit and miss. Nursing and rehabilitation professionals can play a critical role in the care of the person with a stroke by being educated about the effects of stroke and familiar with available services. Many physicians may be unfamiliar with common treatment options, and services may go unordered simply out of ignorance.

There are astounding advances taking place in the emergency, acute, and long-term treatment of people with cerebral vascular accidents. It is hoped that this course has familiarized you with some of these advances and that you will be able to apply what you have learned to the clinical setting. The desire of all healthcare professionals is to provide the highest possible level of care for their clients.

References

- Acker JE, Pancioli AM, Crocco TJ, et al. (2007). *Implementation strategies for emergency medical services within stroke systems of care: a policy statement from the American Heart Association/American Stroke Association Expert Panel on Emergency Medical Services Systems and the Stroke Council*. *Stroke* 2007 Nov;38(11):3097–115. Accessed 6-11-08 from: <http://stroke.ahajournals.org/cgi/content/full/38/11/3097>.
- Adams HP. (2007). AHA/ASA Guideline: Guidelines for the Early Management of Adults with Ischemic Stroke. *Circulation*. 2007;115:e478-e534. Accessed 6-13-08 from: <http://www.circ.ahajournals.org/cgi/content/full/115/20/e478#TBL7>.
- Alberts MJ et al. (2005). Recommendations for Comprehensive Stroke Centers: A Consensus Statement from the Brain Attack Coalition. *Stroke* 36 (7): 1597. Accessed 6-13-08 from: <http://stroke.ahajournals.org/cgi/reprint/36/7/1597?ijkey=fe6ee3bcc6143d3d6fe668e3a5304ade6daf24a1>.
- American Heart Association (2006). Handbook of Emergency Cardiovascular Care for Healthcare Providers. American Heart Association. AmericanHeart.org/cpr.
- American Heart Association (2008). *Heart Disease and Stroke 2008 Update At-a-Glance Statistics*. Accessed 6-10-08 from: http://www.americanheart.org/downloadable/heart/1200082005246HS_Stats%202008.final.pdf.
- Centers for Disease Control Division of Heart Disease and Stroke Prevention (2006). *What Science Tells Us*. Accessed 6-12-08 from: http://www.cdc.gov/DHDSP/library/moving_into_action/science.htm.
- Gladstone DJ, Black SE. (2001). *Update on intravenous tissue plasminogen activator for acute stroke: from clinical trials to clinical practice*. *CMAJ* August 7, 2001; 165 (3). Accessed 6-14-08 from <http://www.cmaj.ca/cgi/content/full/165/3/311#T323>.
- Gould R and Barnes SS. (2007). Shoulder and Hemiplegia. eMedicine. Accessed 6-14-08 from: <http://www.emedicine.com/pmr/TOPIC132.HTM>.
- Hacke W et al. for the ECASS Investigators. (2008). Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. *N Engl J Med* 2008 Sep 25; 359:1317.
- Krajcer Z. *Carotid Stenting*. *Tex Heart Inst J*. 2005; 32(3): 369–371. PMID: PMC1336711. Accessed 6-10-08 from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1336711>.
- Labiche LA, Chan W, Saldin KR, Morgenstern LB. (2002). *Sex and Acute Stroke Presentation*. *Ann Emerg Med* 40:453–60. Accessed 6-12-08 from: <http://www.annemergmed.com/article/PIIS0196064402000768/fulltext>.
- National Institutes of Neurological Disorders and Stroke (NINDS) (2004). *Questions and Answers about Carotid Endarterectomy*. Accessed 6-10-08 from: http://www.ninds.nih.gov/disorders/stroke/carotid_endarterectomy_background.htm.

National Institutes of Neurological Disorders and Stroke (NINDS) (2003). *Know Stroke: Know the Signs, Act in Time*. Accessed 6-10-08 from: <http://www.stroke.ninds.nih.gov/>.

National Institutes of Neurological Disorders and Stroke (NINDS). (2002). Post-Stroke Rehabilitation Fact Sheet, NINDS. 2002. NIH Publication No. 02-4846. Accessed 6-13-08 from: <http://www.ninds.nih.gov/disorders/stroke/poststroke rehab.htm#organizations>.

National Institutes of Neurological Disorders and Stroke (NINDS), rt-PA Stroke Study Group. (1995). *Tissue plasminogen activator for ischemic stroke*. *N Eng J Med* 333:1581-87.

Sacco RL, Sung GY. (2005). *Secondary Stroke Prevention: Antiplatelets*. Conversations with the Experts. Presentation at University of Colorado, Neurocritical Care and Stroke Program. Retrieved September 15, 2005 from http://strokensa.healthology.com/focus_article.asp?f=stroke_experts&b=strokensa&c=stroke_sacco.

Sattin JA, Olson SE, Liu L, Raman R, Lyden PD (2006). *An Expedited Code Stroke Protocol Is Feasible and Safe*. *Stroke* 37 (12): 2935. (2006) Accessed 6-11-08 from: <http://stroke.ahajournals.org/cgi/reprint/37/12/2935>.

Saver JL, Kalafut M. (2007). *Thrombolytic therapy in stroke*. eMedicine. June 13, 2008 from <http://www.emedicine.com/neuro/topic370.htm>.

Sharma M, Clark H, Armour T, et al. (2005). *Acute Stroke: Evaluation and Treatment. Evidence Report/Technology Assessment No. 127*. AHRQ Publication No. 05-E023-2. Rockville, MD: Agency for Healthcare Research and Quality. July 2005. Accessed 6-12-08 from: <http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=hstat1b.chapter.8162>.

Slater D (2006). *Middle Cerebral Artery Stroke*. eMedicine. Accessed 6-11-08 from: <http://www.emedicine.com/PMR/topic77.htm>.

McPhee SJ and Papadakis MA. Tierney LM ed. (2007). *Current Medical Diagnosis and Treatment 2007*. Forty-Sixth Edition. New York: McGraw-Hill Medical.

Tsai AW. (2008). *Prehospital and emergency department capacity for acute stroke care in Minnesota*. *Prev Chronic Dis* 2008;5(2). Accessed 6-10-08 from: http://www.cdc.gov/pcd/issues/2008/apr/07_0212.htm.

Wall HK, Beagan BM, O'Neill HJ, Foell KM, Boddie-Willis CL. (2008). *Addressing stroke signs and symptoms through public education: the Stroke Heroes Act FAST campaign*. *Prev Chronic Dis* 2008;5(2). Accessed 6-12-08 from: http://www.cdc.gov/Pcd/issues/2008/apr/07_0214.htm.

Way LW, Doherty GM (eds.). (2003). *Current Surgical Diagnosis and Treatment*, 11th ed. New York: Lange Medical Books/McGraw-Hill.

Westmoreland BF, Benarroch EE, Daube JR, Reagan TJ, Sandok BA. (2004). *Medical Neurosciences: An Approach to the Anatomy, Pathology, and Physiology by Systems and Levels*. Little, Brown, and Company 1994 by the Mayo Foundation.

Post Test

1. Stroke in the United States:
 - a) Is the leading cause of death among those 55 years of age and older.
 - b) Causes more serious long-term disabilities than any other disease.
 - c) Is mainly hemorrhagic.
 - d) Results in death in about 50% of people in the first 30 days following an ischemic event.
2. Ischemic strokes:
 - a) Are caused by blood that leaks into the brain, damaging sensitive nerve cells.
 - b) Make up a small percentage of the total number of strokes.
 - c) Are caused by thrombotic or embolic occlusions of the major arteries of the brain.
 - d) Occur when a major vessel of the heart is occluded by a blood clot.
3. Risk factors for stroke:
 - a) Have little in common with heart disease risk factors.
 - b) Include diabetes and smoking which each increase stroke risk by a factor of 2.
 - c) Include hypertension which is the most important risk factor.
 - d) Include atrial fibrillation which is the most important risk factor.
4. The cerebrum:
 - a) Is divided into 4 hemispheres called frontal, parietal, temporal and occipital.
 - b) Is another name for the cerebellum.
 - c) Contains the left hemisphere which is referred to as the non-dominant hemisphere and the right hemisphere which is the dominant hemisphere.
 - d) Has 2 hemispheres, each divided into 4 lobes: the frontal, parietal, temporal and occipital lobes.
5. Each hemisphere of the brain is covered with a thin layer of cells:
 - a) Called the cerebrum, where most strokes occur.
 - b) That consists of the medulla oblongata, pons and midbrain.
 - c) Called the cerebral cortex which is the "thinking" and "processing" part of the brain.
 - d) Called the cerebral cortex which has a low metabolic requirement allowing for recovery of function after brain injury.
6. The area of the brain responsible for control of blood pressure and breathing is the:
 - a) Brainstem reticular formation
 - b) Medulla oblongata
 - c) Cerebellum
 - d) Thalamus
7. The cerebellum or "little brain":
 - a) Makes up more than half of the total volume of the brain.
 - b) Houses the respiratory and cardiovascular centers.
 - c) Helps to control and coordinate movement.
 - d) Is highly convoluted and folded, which increases the surface area of the brain.

8. Damage to the cerebellum may result in all of the following except:
- Ataxia
 - Intention or action tremor
 - Hypotonia
 - Speech and language dysfunction
9. Stroke associated deficits may include:
- Paralysis of one side of the body which is called hemiparesis.
 - Pain, numbness or odd sensations such as tingling or prickling, called thalamic pain syndrome.
 - Chronic pain syndrome from damage to the nervous system, called neuropathic pain.
 - Thalamic pain syndrome which is easily treated with medication.
10. Stroke-induced injury to the brain's language-control centers:
- May result in receptive aphasia, which is the inability to plan the steps involved in a complex task and carry them out in the proper sequence.
 - May result in expressive aphasia caused by damage to Broca's area, which is difficulty conveying thoughts through words or writing.
 - Causes global aphasia which is minimal brain damage with subtle effects.
 - Results in aphasia in more than 1/2 of all stroke patients.
11. Blood flows from the heart to the brain via 2 large arterial systems:
- The middle cerebral and the anterior cerebral arteries.
 - The ophthalmic artery and the middle cerebral artery.
 - The carotid and the vertebrobasilar systems.
 - Broca's and Wernicke's.
12. The middle cerebral artery:
- Is a branch of the vertebrobasilar system.
 - Supplies blood to much of the medial surface of the brain.
 - Is the second most common artery involved in stroke which may cause contralateral weakness and sensory loss, primarily in the leg.
 - Is the most common artery involved in stroke causing contralateral weakness and sensory loss in the face, neck and arm.
13. Posterior circulation ischemia:
- Causes contralateral symptoms.
 - May cause diplopia which is difficulty swallowing.
 - May cause ataxia which is gross motor incoordination due to cerebellar damage.
 - May cause amaurosis fugax or "fleeting blindness".
14. TIAs or transient ischemic attacks:
- Are small strokes in which the symptoms resolve within 5 days.
 - Are usually caused by a narrowing of the vessels by plaque that eventually occludes the blood supply.
 - Usually have a gradual onset of symptoms similar to stroke followed by a gradual decrease in symptoms.
 - Are often caused by cardioembolism, which most commonly originate from the heart or neck arteries.

15. TIAs are a major indicator of the overall health of the cardiovascular system and about 30% of people with stroke have a history of TIAs.
- True
 - False
16. Ischemic stroke:
- Represent 15 to 20% of all strokes.
 - Can be caused by a significant increase in blood pressures over time and often occurs after activity.
 - Is caused by a reduction in blood flow to a portion of the brain due to vascular occlusion.
 - May be caused by thrombosis in which a foreign body becomes lodged in a blood vessel.
17. Hemorrhagic strokes:
- Occur most often in the cerebellum.
 - Occur when a vessel of the brain is occluded by embolism, stenosis or thrombosis.
 - Account for 80 to 85% of all strokes.
 - Result in lost or impaired consciousness in 50% of patients.
18. Strokes in women:
- Occur more often than in men largely because women have smaller carotid arteries.
 - May include nontraditional symptoms such as altered level of consciousness, nausea and hiccups.
 - May have a better outcome than male patients in motor, cognitive and functional recovery.
 - Are easier to recognize because of a higher occurrence of traditional symptoms.
19. Risk factors for stroke that can be changed by education and intervention include all of the following except:
- A family history of stroke.
 - Hypertension
 - A sedentary life style.
 - Smoking
20. The "suddens" refers to:
- A sudden increase in blood pressure, weight and diabetes seen in Americans over the last 10 years that has contributed to a sudden increase in heart attack and stroke.
 - The need for sudden awareness of symptoms by EMTs to assure prompt treatment of stroke victims.
 - Sudden weakness or numbness, confusion, trouble seeing, trouble walking and severe headache and are used to convey stroke symptoms.
 - The lack of awareness of stroke symptoms by the general public.
21. Stroke Heroes Act FAST:
- Has been shown to identify almost 98% of stroke victims when used by untrained laypeople.
 - Uses the acronym FAST which stands for facial weakness, altered level of consciousness, slurred speech and time to call 9-1-1.
 - Uses the acronym FAST which stands for facial weakness, arm drift, slurred speech and time to call 9-1-1.
 - Has been shown to be too difficult for the layperson to remember and therefore is not effective in identifying stroke victims.

22. The goal of reaching an emergency department within 3 hours of the onset of stroke symptoms is:
- To differentiate a true stroke from a TIA.
 - Important because x-rays do not show the difference between hemorrhagic stroke and ischemic stroke after 3 hours.
 - Only important if the patient has symptoms of a hemorrhagic stroke so they can receive thrombolytic drugs.
 - Critical because of the use of time-dependent medications and treatments for acute stroke.
23. Recommendations made by the Minnesota Stroke Partnership advise that:
- Prehospital personnel should transport any suspected stroke victim to the nearest emergency department.
 - EMS personnel should not attempt to identify stroke victims because they lack the proper education.
 - EMS personnel should treat stroke as an emergent event and transport suspected patients to the nearest stroke center.
 - The Glasgow Coma Scale should be used as the standard for assessing suspected stroke patients.
24. When a suspected stroke patient arrives in an emergency department the goal should be:
- An initial evaluation within 1/2 hour.
 - A CT scan initiated within 10 minutes.
 - Interpretation of the CT scan within 25 minutes.
 - Door-to-drug time within 60 minutes.
25. Thrombolytic agents are used to dissolve clots that seriously diminish blood flow to some part of the body.
- True
 - False
26. Inclusion criteria for the administration of thrombolytic agents include:
- An ischemic stroke diagnosed by clinical evaluation.
 - Initiation of treatment within 5 hours of the time when the patient was last seen normal.
 - Neurological deficits as measured by the Glasgow Coma Scale.
 - Evidence of a subarachnoid hemorrhage on the CT scan.
27. Exclusion criteria for thrombolytic therapy include all of the following except:
- Evidence of acute intracerebral bleed on a non-contrast CT scan.
 - Symptoms suggesting subarachnoid hemorrhage.
 - A witnessed seizure at the onset of the stroke.
 - The sudden onset of a left facial droop within the last 3 hours.
28. Relative contraindications for administration of thrombolytics include:
- Major surgery within the last 10 years.
 - A myocardial infarction within the last year.
 - Rapid improvement of neurologic deficits based on clinical evaluation.
 - GI or GU hemorrhage within the last 3 months.

29. Recent proposals for an expedited code stroke protocol recommend:
- A 2 hour onset-to-treatment benchmark.
 - Written consent by family members if the patient is not able to give consent.
 - Chest x-ray within 5 minutes.
 - CT scan only if an intracranial bleed is suspected.
30. The use of aspirin:
- Should be considered as a substitute for rt-PA.
 - Is recommended as an adjunctive therapy that should be given at the same time as rtPA.
 - Is a standard treatment for patients at risk for stroke.
 - Combined with clopidogrel is recommended for the treatment of acute ischemic stroke.
31. Urgent anticoagulation with heparin is not recommended for patients with moderate to severe strokes because of an increased risk of serious intracranial hemorrhagic complications.
- True
 - False
32. Carotid endarterectomy:
- Has been shown to be of great benefit in patients with acute hemorrhagic stroke.
 - Should not be used in conjunction with aspirin therapy.
 - Is most often performed in women with severe carotid stenosis.
 - Reduces 5 year stroke risk by 1/2 in patients who are symptom-free, but have a carotid stenosis of 60 to 90%.
33. Rehabilitation following a stroke:
- Should not begin until at least 5 days after an acute stroke.
 - Should help a stroke victim reach the highest possible level of independence.
 - Begins with frequent turning of stroke patients by the physical therapists.
 - Includes passive range of motion exercises performed by the patient with physical assistance from the therapist.
34. Stroke rehabilitation:
- Has been shown to be unnecessary because brain damage from a stroke cannot be reversed.
 - Is not done in as many as 50% of stroke patients.
 - Can be done as an outpatient only if the patient is able to walk.
 - Is offered in hospital-based, skilled nursing and outpatient facilities in addition to home care services.

Course Evaluation

Please answer each of the following questions. Questions with asterisks (*) are required.

* 1. This course met the goals and learning objectives.

Yes No

* 2. The author was well prepared to write about the content in a way that facilitated my learning.

Yes No

* 3. This course was free from commercial bias.

Yes No

* 4. The learning activity met my continuing education needs.

Yes No

* 5. The learning activity took me 60 minutes per contact hour. (If you answer “No”, please enter the total time it took to finish the course, test, and evaluation.)

Yes

No**

** If your answer was “No”, how long did it take to finish the course, test, and evaluation?

6. My professional educational level is (check one):

Nursing

Nurse Aide LVN/LPN RN (diploma) RN (AD)

BSN MSN Nurse Practitioner / Advanced Practice Nurse

PhD / DNSc

Therapy

OT Aide COTA OT MOT OTD

PT Aide PTA PT MPT MSPT DPT PhD

Other (please specify): _____

7. I heard about ATrain Education from:

Search engine

Advertisement

Government or Board website

Returning customer

Friend

Publication (Magazine, etc.)

Other _____

(continued on next page)

8. I found the ATrainCEU.com website easy to use:

Yes No_____

9. Comments or suggestions (optional): _____

Registration Information

Please answer all of the following questions (*required).

*Name: _____

*Address: _____

*Phone: _____

*Professional Designation: _____

*License Number and State: _____

Please e-mail my certificate: Yes No

Email (required if you want your certificate sent by email): _____

(Note: If you request an email certificate we will not send a copy of your certificate by US Mail)

Payment Options

You may pay by credit card or by check.

Fill out this section only if you are **paying by credit card**.

3.5 contact hours - \$24

Fill out this section if paying by credit card

Name _____

Address (if different than above) _____

Card type: Visa MC American Express Discover

Card number _____

Expiration date _____

Test Completion and Mailing Instructions

1. Complete all forms:

- Post Test
- Evaluation Learning Activity
- Registration Form (this page)

2. If you are **paying by check**, prepare a check for \$24 made out to ATrain Education, Inc.

3. Mail the completed forms and your payment to:

ATrain Education, Inc
5171 Ridgewood Rd
Willits, CA 95490

Once we receive your forms and payment, we will mail (or email, if you request it) your certificate of completion. If you have any questions or concerns, please call or contact us at info@ATrainCEU.com. And thanks for taking the ATrain!