Stroke: Acute Care and Rehabilitation

5 contact hours: $29

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Course Summary: Presents major types of stroke and risk factors. Provides a review of brain anatomy and deficits associated with various types of stroke and describes treatment regimens, prehospital, and ED treatment. Rehabilitation following acute stroke is discussed in detail. Recent advances in stroke management are tied to clinical practice.

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Course Objectives

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

- Distinguish among the main classifications of stroke.
- Explain the major risk factors for stroke and its pathophysiology.
- Identify the main structures of the brain and the common deficits caused by stroke to those structures.
- Contrast gender differences in the presentation of acute stroke.
- Summarize the efforts of public health organizations to educate the public about stroke symptoms and emergency response.
- Describe recent advances aimed at improving care of stroke victims in prehospital settings.
- Review emergency department care of persons with acute stroke.
- Discuss the role of rehabilitation therapy in the short- and long-term treatment of stroke.

Introduction

Each year in the United States there are nearly 800,000 new or recurrent strokes—an average of one stroke every 40 seconds. In 2010 the estimated direct and indirect costs associated with stroke were nearly $74 billion (AHA, 2010). About 610,000 of these strokes are first attacks and about 185,000 are recurrent attacks (AHA, 2011).

Stroke has been the third leading cause of death in the United States for more than five decades, after heart disease and cancer. In 2008 chronic lower respiratory diseases replaced stroke as the third leading cause of death, due mostly to a decrease in the numbers of deaths from stroke (NVSS, 2010).

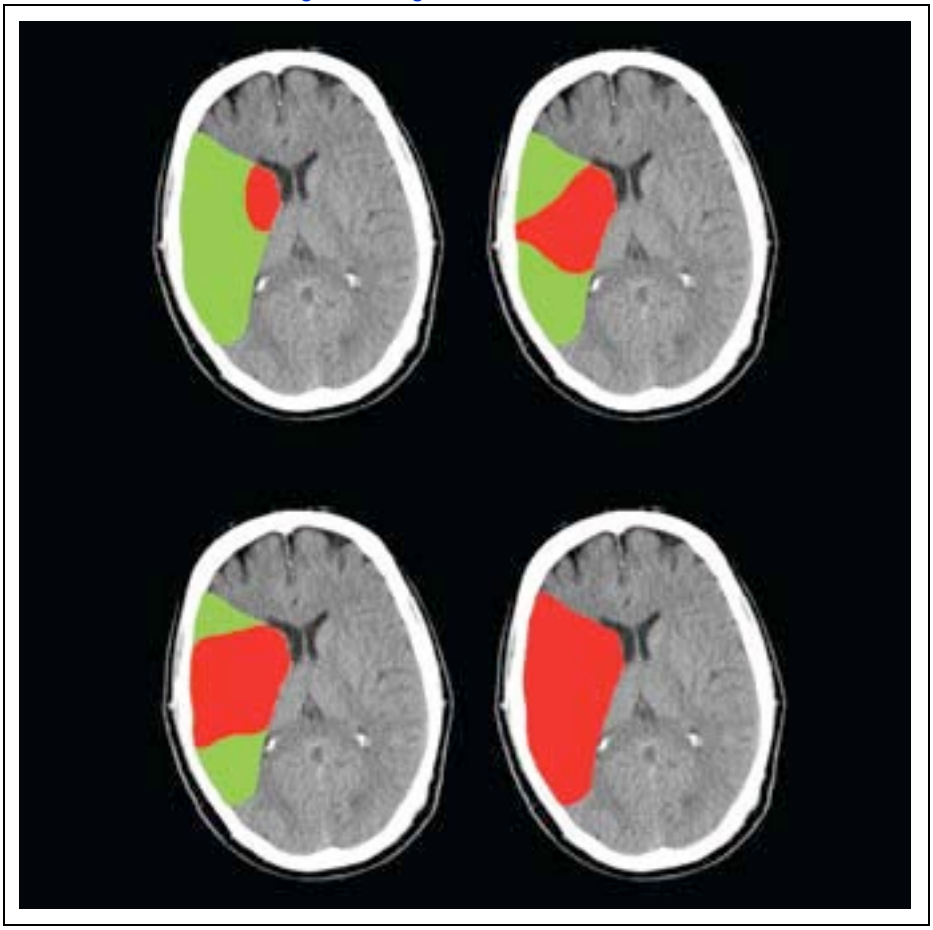
Although stroke is now the fourth leading cause of death in the United States, it remains a leading cause of long-term disability. More than 2 million people live with the neurologic after-effects of stroke. Forty percent have moderate functional impairment and 15% to 30% have severe disability (VA/DOD, 2010).

About 15% to 30% of those suffering an ischemic stroke will die within the first month. The chance of surviving a hemorrhagic stroke is much worse, with a survival rate of only about 20% (Slater, 2011). After an acute episode, the most common causes of death are pulmonary embolism (within 2–4 weeks), pneumonia (within 2–3 months), and cardiac disease (>3 months) (Slater, 2011). Twenty-one percent of men and 24% of women will die in the first year following a stroke (AHA, 2008), and only 25% recover fully.

There are two main types of stroke: ischemia and hemorrhage. An **ischemic stroke** is caused by interruption of blood flow and decrease of oxygen to the brain. If not treated rapidly, ischemia ultimately leads to infarction, in which brain cells are replaced by a fluid-filled cavity (or infarct). A **transient ischemic attack (TIA)** is also caused by blockage or interruption of blood flow to the brain. A TIA lasts only a short time but should be treated as a serious neurologic event. **Hemorrhagic stroke** occurs when a blood vessel in the brain leaks or ruptures, spilling blood into adjacent brain tissue.

The loss of oxygen and nutrients following a stroke begins a process that destroys neurons within the brain. Some cells die immediately, while others are damaged and remain at risk for death if treatment is delayed. The damaged neurons surrounding dead cells make up the ischemic **penumbra** (see illustration below) and can linger in a compromised state for several hours. With timely treatment these neurons can be saved (NINDS, 2011a).

Penumbra Surrounding Damaged Brain Tissue



Immediately after an ischemic stroke (top left), a core of irreversibly damaged brain tissue (red) is surrounded by an area of viable but at-risk tissue called the penumbra (green). Unless blood flow is restored quickly, the tissue within the penumbra will be lost (bottom right). Source: National Institutes of Health (n.d.).

Types of Stroke

Ischemic Stroke

Ischemic stroke causes a sudden onset of neurologic deficit, usually in a person with a history of hypertension, diabetes mellitus, valvular heart disease, or atherosclerosis. There are distinctive neurologic signs that indicate the region of the brain involved (McPhee and Papadakis, 2011).

Ischemic strokes occurring in the carotid circulation are the most common type of ischemic stroke, accounting for approximately 70% of all cases (Baird, 2011). They are usually caused by occlusion of one of the major intracranial arteries or one of the small single penetrating arteries.

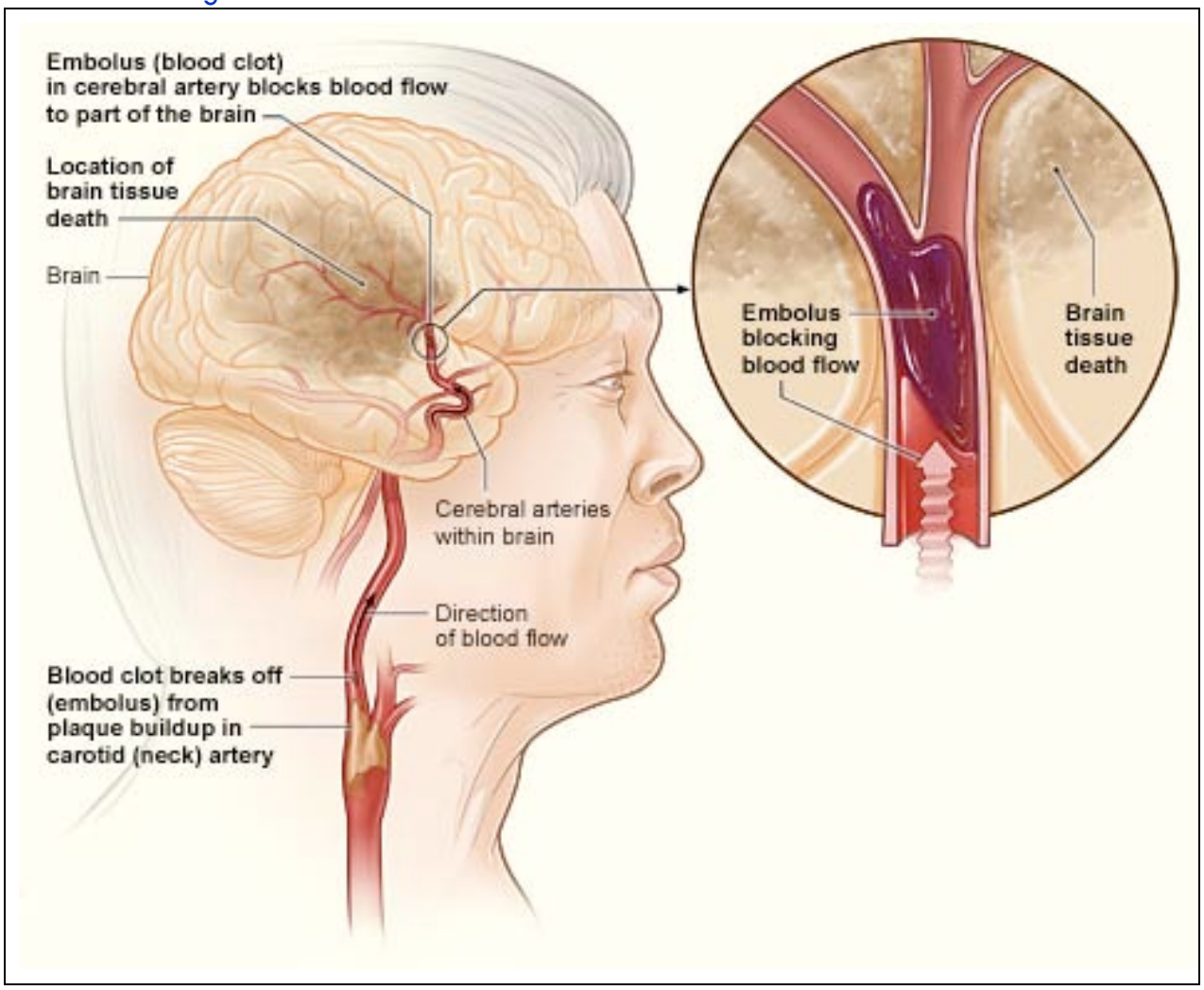
Depending on the location of the blockage, ischemic stroke can affect sensation, speech, behavior, thoughts, memory, or emotions. One side of the body may become paralyzed or weak. The five most common signs and symptoms of ischemic stroke are acute onset of:

- Numbness or weakness of the face, arm, or leg

- Confusion or trouble speaking or understanding others
- Trouble seeing in one or both eyes
- Dizziness, trouble walking, or loss of balance or coordination
- Severe headache with no known cause (CDC, 2010c)

Blood clots can cause ischemia in two ways. In the first, a clot that forms in a part of the body distant from the brain travels through the blood and becomes wedged in an artery supplying blood to the brain. This free-roaming clot is called an **embolus** and often forms in the heart. A stroke caused by an embolus is called an embolic stroke.

The Brain During an Embolic Stroke



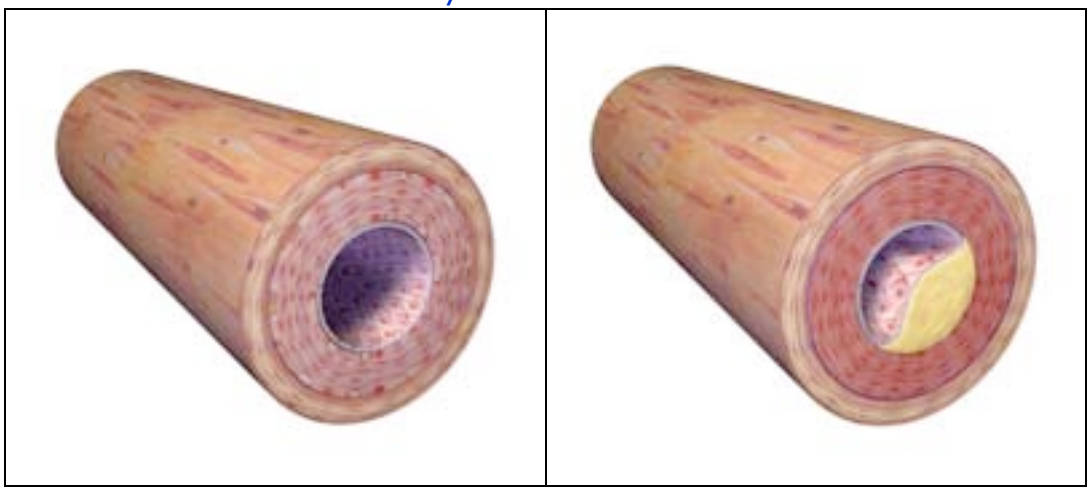
This illustration shows how an ischemic stroke can occur in the brain. If a blood clot breaks away from plaque buildup in a carotid (neck) artery, it can travel to and lodge in an artery in the brain. The clot can block blood flow to part of the brain, causing brain tissue death. Source: NIH, n.d.

The second kind of ischemic stroke, called a thrombotic stroke, is caused by **thrombosis**, the formation of a blood clot in one of the cerebral arteries that stays attached to the artery wall until it grows large enough to block blood flow (NINDS, 2011a).

Ischemic strokes can also be caused by **stenosis**, a narrowing of an artery due to the buildup of plaque and blood clots along the arterial wall. Stenosis can occur in large or small arteries and is referred to as large-vessel disease or small-vessel disease, respectively. When a stroke occurs due to small-vessel disease, a very small infarction results, sometimes called a lacunar infarction (NINDS, 2011a).

Atherosclerosis is the most common blood vessel disease that causes stenosis. In atherosclerosis, deposits of plaque build up along the inner walls of large- and medium-sized arteries, causing thickening, hardening, and loss of elasticity of artery walls along with decreased blood flow (NINDS, 2011a).

Stenosis in a Section of an Artery



Left: A sectioned elastic artery. Elastic arteries are vessels that can handle a great deal of pressure (e.g., the aorta, which takes pressure directly from the constant beating of the heart). Right: An atherosclerotic plaque, with the plaque forming on the inside wall. Source: Copyright © Zygote Media. Used with permission.

Stroke recurs in as many as 10% of stroke survivors in the first 12 months after the initial event, with an incidence of 4% per year thereafter (Baird, 2011).

Transient Ischemic Attack (TIA)

A transient ischemic attack (TIA) is an ischemic stroke that lasts only a few minutes. Its onset is acute and without warning, and recovery is usually rapid. TIAs occur when the blood supply to part of the brain is briefly interrupted—usually by an embolism. TIA symptoms are similar to those of stroke but do not last as long. Essentials features of TIA include:

- The presence of risk factors for vascular disease
- Focal neurologic deficit of acute onset
- Clinical deficit that resolves completely within 24 hours (McPhee and Papadakis, 2011)

Almost 25% of patients experiencing a TIA have resolution of neurologic symptoms within 5 minutes and 50% have resolution within 30 minutes. If the patient's symptoms persist after 1 hour, there is only a 15% chance that neurologic symptoms will disappear within 24 hours.

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, 2011). Aspirin and modification of risk factors such as high cholesterol and hypertension reduce the likelihood of heart attack and stroke (Farina, 2011).

A TIA can occur in either the anterior or the posterior circulation, and symptoms vary depending upon the location of the blockage. If ischemia affects the carotid (anterior) 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, 2011).

Other symptoms may include monocular visual loss, dysphagia, and slowness of movement. Examination during a TIA may reveal sensory changes, hyperreflexia, 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, 2011).

Because there is no way to tell whether symptoms are from a TIA or an acute stroke, people should assume that all stroke-like symptoms signal an emergency and should not wait to see if they go away. A prompt evaluation is necessary to identify the cause of the TIA and determine appropriate therapy.

Key Points About TIAs

TIA produces some or all of these symptoms:

- Numbness or weakness in the face, arm, or leg, especially on one side of the body
- Confusion or difficulty talking or understanding speech
- Trouble seeing in one or both eyes
- Difficulty walking, dizziness, or loss of balance and coordination
- Numbness/sensory changes

Source: NINDS, 2011b.

The occurrence of a TIA is a major indicator of the overall health of the cardiovascular system, and many strokes can be prevented by heeding warning signs and treating underlying risk factors. Drug therapy or surgery to reduce the risk of stroke may be indicated. The use of antiplatelet agents, particularly aspirin, is a standard treatment for patients at risk for stroke, and people with atrial fibrillation may be prescribed anticoagulants (NINDS, 2011b).

People who have suffered a TIA are at increased risk for stroke, especially in the first year after the event. In the first 3 months, stroke risk is more than 10%, with the highest risk in the 2 days following the TIA (NINDS, 2011b). 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 or speech impairment (Circulation, 2011).

Hemorrhagic Stroke

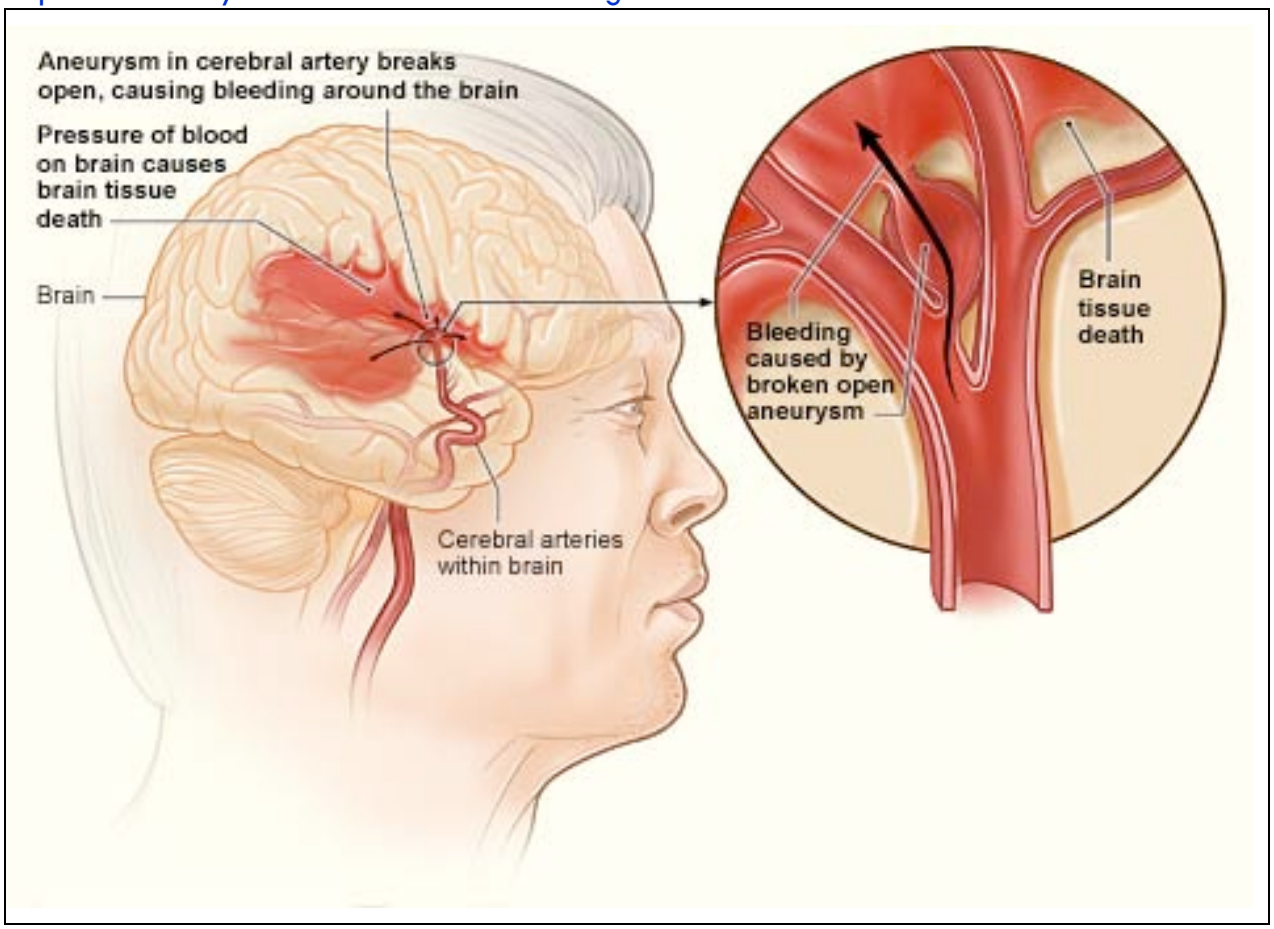
There are two types of hemorrhagic strokes—intracerebral and subarachnoid hemorrhages. Bleeding from ruptured brain arteries can either go into the substance of the brain or into the various spaces surrounding it.

A **hemorrhagic stroke** occurs when a blood vessel in the brain bursts or leaks, causing blood to accumulate, compressing the surrounding brain tissue, and killing neurons. Blood also irritates delicate brain tissue and causes cerebral edema. Tissue swelling—along with the hematoma from the leaking blood—increases the mass effect, causing further damage and a general increase in intracranial pressure. Brain cells beyond the rupture are deprived of blood and are also damaged (Mayoclinic.com, 2011).

Symptoms of hemorrhagic stroke include those of ischemic stroke, but may also include nausea, vomiting, headache, and altered level of consciousness. These symptoms may indicate increased intracranial pressure and are more common with hemorrhagic strokes or large ischemic strokes. Seizures occur in up to 28% of hemorrhagic strokes (Liebeskind, 2011).

Hemorrhage can occur in several ways. One common cause of hemorrhage is a bleeding **aneurysm**—a weak or thin spot on an artery wall. Over time, these weak spots stretch or balloon out under high arterial pressure and their thin walls can rupture and spill blood into the surrounding brain cells (NINDS, 2011b). Aneurysms affect as much as 1% of the population and are sometimes hereditary. Studies have shown that the risk an aneurysm will rupture is related to its size and shape, its location, and the person's age (NIH, 2009).

Ruptured Aneurysm with Associated Bleeding in the Brain



This shows how a hemorrhagic stroke can occur in the brain. An aneurysm in a cerebral artery breaks open, which causes bleeding in the brain. The pressure of the blood causes brain tissue death. Source: NIH, n.d.

Hemorrhage also occurs when an arterial wall breaks open. Plaque-encrusted arteries eventually lose their elasticity and become brittle, thin, and prone to cracking. Hypertension increases the risk that a brittle artery wall will give way and release blood into the surrounding brain tissue (NINDS, 2011b).

An **arteriovenous malformation** can also cause a hemorrhagic stroke. A cerebral arteriovenous malformation is an abnormal connection between the arteries and veins in the brain that forms during embryonic development or soon after birth. This tangle of defective, thin-walled blood vessels and capillaries may bleed when subjected to pressure or damage. Although hemorrhage from an arteriovenous malformation can occur at any age, it is most common between the ages of 15 and 20 years. Arteriovenous malformations may develop in many different sites but those located in the brain or spinal cord can have especially widespread effects on the body (NINDS, 2011c).

Intracerebral Hemorrhage

Intracerebral hemorrhage is the most common type of hemorrhagic stroke and the second most common cause of strokes after ischemic strokes. An intracerebral hemorrhage occurs when an artery in the brain bursts, flooding the surrounding tissue with blood (CDC, 2010d). The 30-day mortality rate from intracerebral hemorrhage ranges from 35% to 52%, and half of these deaths occur within the first 2 days. Only a small number of patients who survive an intracerebral hemorrhage function independently after the event (Rordorf, 2011).

In the absence of neurovascular abnormalities such as aneurysm or angioma, nontraumatic intracerebral hemorrhage is most commonly caused by hypertensive damage to blood vessel walls (Liebeskind, 2010). A significant increase in blood pressure over time can cause hemorrhage, which often occurs after activity.

Hypertensive intracerebral 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 (McPhee and Papadakis, 2011).

Bleeding into the deep portions of the brain causes visual 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. The incidence of intracerebral hemorrhage has increased by 18% in the past ten years, possibly because of increase in the number of elders who may lack adequate blood pressure control, as well as the increasing use of anticoagulants, thrombolytics, and antiplatelet agents (Circulation, 2011).

Subarachnoid Hemorrhage

Subarachnoid hemorrhage is caused by bleeding under the meninges into the thin fluid-filled space that surrounds the brain (NINDS, 2011b). Trauma is the most common cause of subarachnoid hemorrhage (McPhee and Papadakis, 2011). About 10% of patients suffering subarachnoid hemorrhage die immediately, and up to 60% die within the first 30 days. Rebleeding is a major complication, with a mortality rate of 50% to 80% (Becske, 2010).

The first sign of subarachnoid hemorrhage is typically a severe headache with a split-second onset and no known cause. Neurologists call this a thunderclap headache and it demands immediate medical attention. About one-half of patients lose consciousness, and vomiting can present (McPhee and Papdakis, 2011). The rupture may occur in an arteriovenous malformation, but typically it is caused by an aneurysm.

Risk Factors

Risk factors for both ischemic stroke and intracerebral hemorrhage increase with age. The risk of both types of stroke doubles for each successive decade after age 55 years (JAMA, 2011). Some risks factors can be modified while others cannot. Risk factors that cannot be modified include age, gender, race/ethnicity, and family history of stroke. In contrast, other risk factors for stroke (eg, high blood pressure, cigarette smoking) can be changed or controlled by the person at risk.

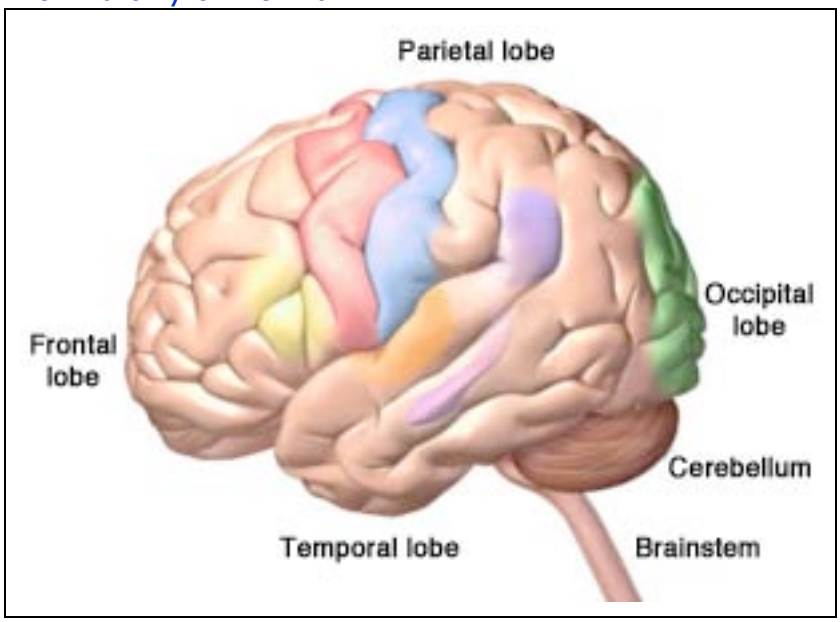
The most important risk factors for stroke are age, hypertension (HTN), diabetes, heart disease, and cigarette smoking. Others include heavy alcohol consumption, high blood cholesterol levels, and illicit drug use. When someone has more than one risk factor their overall risk of stroke is amplified. This means that the multiple risk factors compound their destructive effects and create an overall risk greater than the simple cumulative effect of the individual risk factors.

Important Risk Factors for Stroke	
Risk factor	Comments
Age	Risk of stroke doubles each decade after the age of 55 (AHA, 2011).
High blood pressure	Stroke risk is 4 to 6 times higher than for those without HTN. One-third of the adult U.S. population (including 40%–70% of those over age 65) has HTN. Forty percent to 90% of stroke patients have high blood pressure before their stroke event. Treatment of HTN can decrease stroke incidence rate by 38% and stroke fatality rate by 40% (NINDS, 2011a).
Diabetes	Stroke risk is 3 times higher than for those without diabetes. Contributing risk factors can amplify the overall risk for stroke—the prevalence of HTN is 40% higher in the diabetic population compared to the general population (NINDS 2011a).
Cigarette smoking	Doubles a person's risk for ischemic stroke and increases risk for subarachnoid hemorrhage by up to 3.5%. Promotes atherosclerosis and increases the levels of blood-clotting factors, such as fibrinogen. Weakens the endothelial lining of the cerebrovascular system, which leads to greater damage to the brain from events that occur in the secondary stage of stroke (NINDS 2011a).
Atrial fibrillation	Raises the risk for stroke because the upper chambers of the heart beat ineffectively and allow blood to pool and clot. If a clot breaks off it can lodge in the brain and cause a stroke (AHA, 2011).
High cholesterol	Contributes to stroke in the same way that it contributes to heart disease. Low-density lipoprotein (LDL or bad cholesterol) circulates in the blood, picks up excess cholesterol, and deposits it where it is needed. Excess LDL cholesterol builds up in the arteries, leading to stenosis and atherosclerosis. High density lipoprotein (HDL, good cholesterol) delivers cholesterol to the liver, where the excess is then sent to the kidneys and eliminated (NINDS, 2011a).
High alcohol consumption	Leads to an increase in blood pressure and also may deplete platelets and compromise blood clotting. Although heavy drinking is a risk for both hemorrhagic and ischemic stroke, daily consumption of smaller amounts of alcohol may provide a protective influence against ischemic stroke by decreasing the clotting ability of platelets in the blood (NINDS, 2011a).
Illicit drugs	Use of illicit drugs can cause stroke by acting on other risk factors, such as HTN, heart disease, and vascular disease. Decreases relative blood flow by up to 30%, causes vascular constriction, and inhibits vascular relaxation, leading to narrowing of the arteries. Drugs such as cocaine affect the heart, causing arrhythmias and rapid heart rate that can lead to blood clots. Marijuana decreases blood pressure and may interact with other risk factors, such as HTN and cigarette smoking, to cause rapidly fluctuating blood pressure levels, damaging blood vessels (NINDS 2011a).

Brain Anatomy

The brain is made up 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 Anatomy of the Brain



The cerebrum is made up of the parietal, frontal, occipital, and temporal lobes. Source: Copyright © Zygote Media. Used with permission.

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

The Cerebral Cortex

The cerebral cortex is a thin layer of nerve cell bodies covering the surface of each hemisphere. It is the part of the brain most often affected by stroke. Axons arising from the estimated 100 billion cell bodies of the cortex run both horizontally and vertically, and each connects with thousands of other neurons, creating a highly complex network.

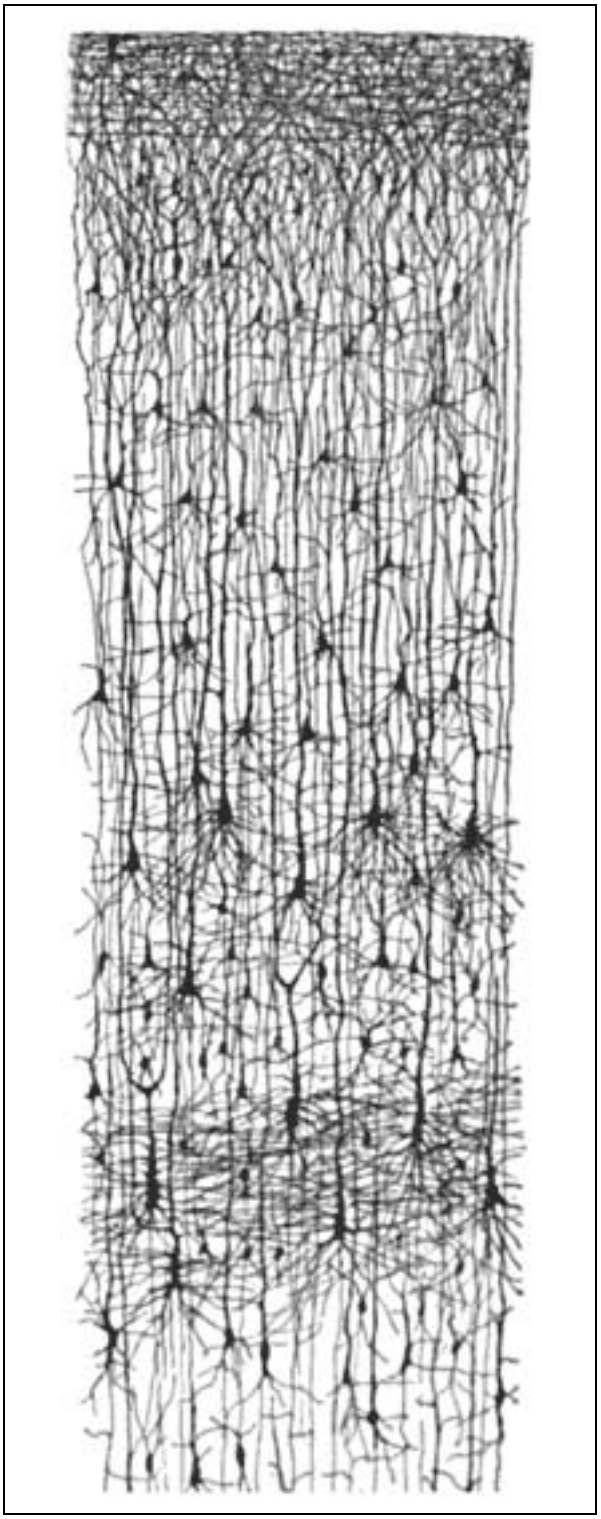
The cerebral cortex is highly convoluted and folded, which increases the surface area of the brain—a phenomenon unique to humans. The cell bodies of the cortex have a high metabolic requirement, using six times more blood than other parts of the brain. The interconnectedness of the nerve cells creates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

The cerebral cortex has historically been divided by function and structure areas into the somatosensory, somatomotor, primary motor, visual, and auditory areas. These descriptions derive from early brain research and are no longer considered to be accurate except as a broad overview. New imaging techniques show that the cortex is more extensively interconnected than previously thought.

The cerebral cortex is the thinking and processing part of the brain. The cortex both originates thoughts and commands and receives information from the periphery and other parts of the brain for processing and interpretation. **Motor commands flow from efferent nerve fibers** originating in the cortex out to the muscles.

Sensory information flows via bundles of afferent nerves residing in the cortex from the peripheral nervous system for processing. The cerebral cortex—especially the frontal areas—is the area of the brain most commonly damaged by stroke.

Nerve Cell Bodies in the Cerebral Cortex

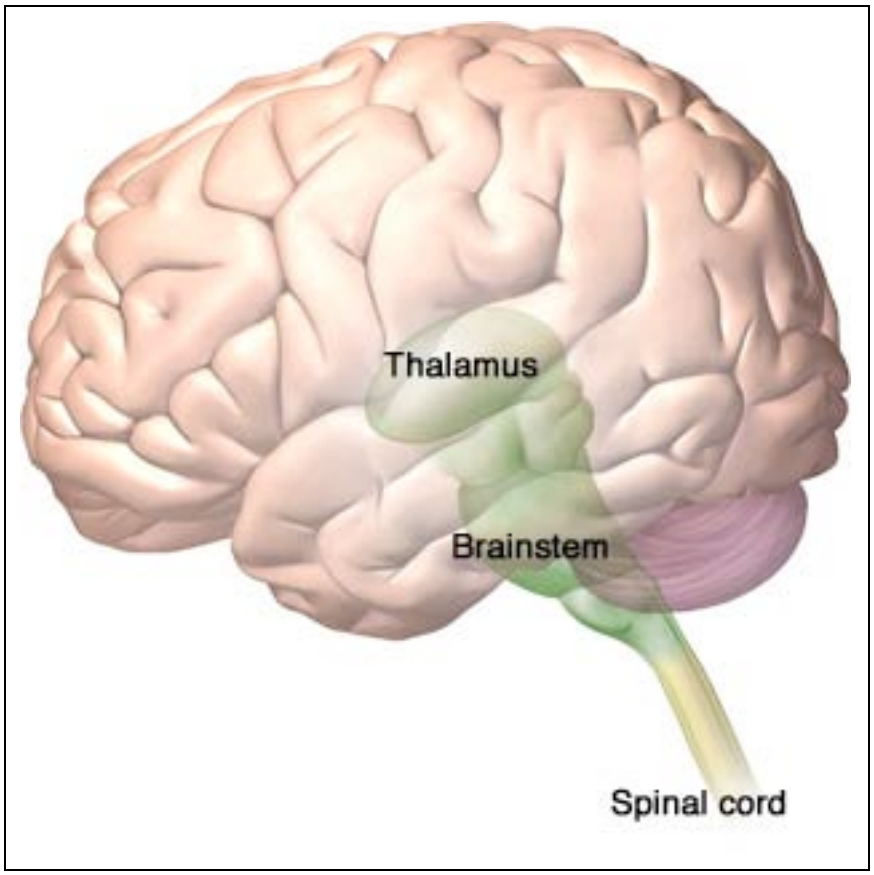


The human cortex showing a highly interconnected network of nerve cells. Source: Ramon y Cajal, 1899.

The Thalamus

The thalamus or "inner chamber" is a small ovoid mass about 3 cm long (shown below in blue) located at the base of the cerebral hemispheres. Sensation travels to the thalamus from peripheral sensory neurons. It is closely integrated with the cerebral cortex and is responsible for the initial processing of all sensory information (except olfaction).

The Thalamus



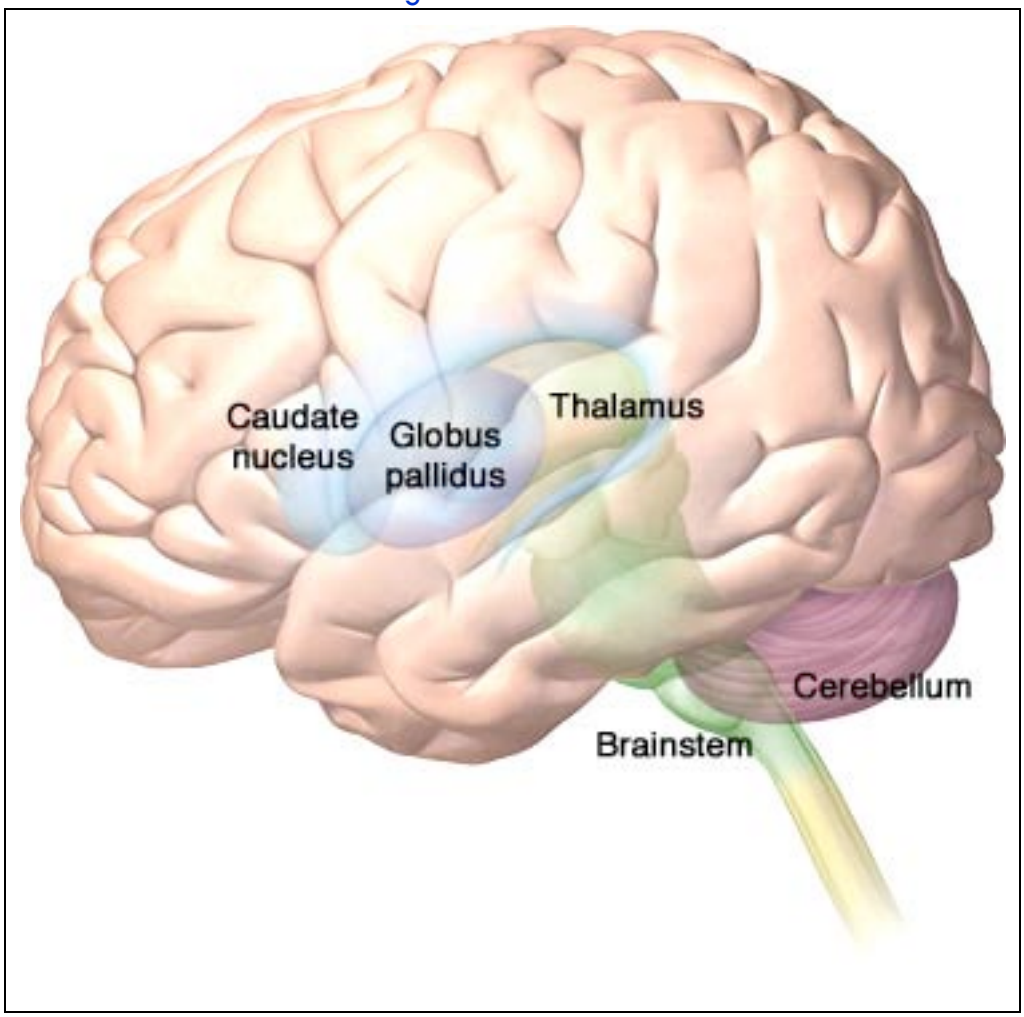
The thalamus (in blue) is the destination of spinothalamic tract—the sensory pathway responsible for processing pain, temperature, and crude touch. Source: © Zygote Media. Used with permission.

The thalamus accepts and sifts sensory information and is the part of the brain where sensation is first consciously experienced or felt.

Subcortical Structures (Basal Ganglia, or Extrapyramidal System)

The basal ganglia are three large masses of cells (ganglia) that lie at the base of the cerebral cortex and surround the thalamus. The three masses that compose the basal ganglia are called caudate nucleus, putamen, and globus pallidus. The names of these three structures are combined in various ways: the caudate nucleus and the putamen together are referred to as the **striatum** and the globus pallidus and the putamen together are known as the **lentiform nucleus**.

Lateral Brain with Basal Ganglia



This image illustrates the left lateral view of the brain and spinal cord, as well as the caudate nucleus and basal ganglia deep in the brain, and a contour of the rest of the thalamus. The cerebral hemisphere (in pink) surrounds the caudate nucleus thalamus (in the center). Source: Copyright © Zygote Media. Used with permission.

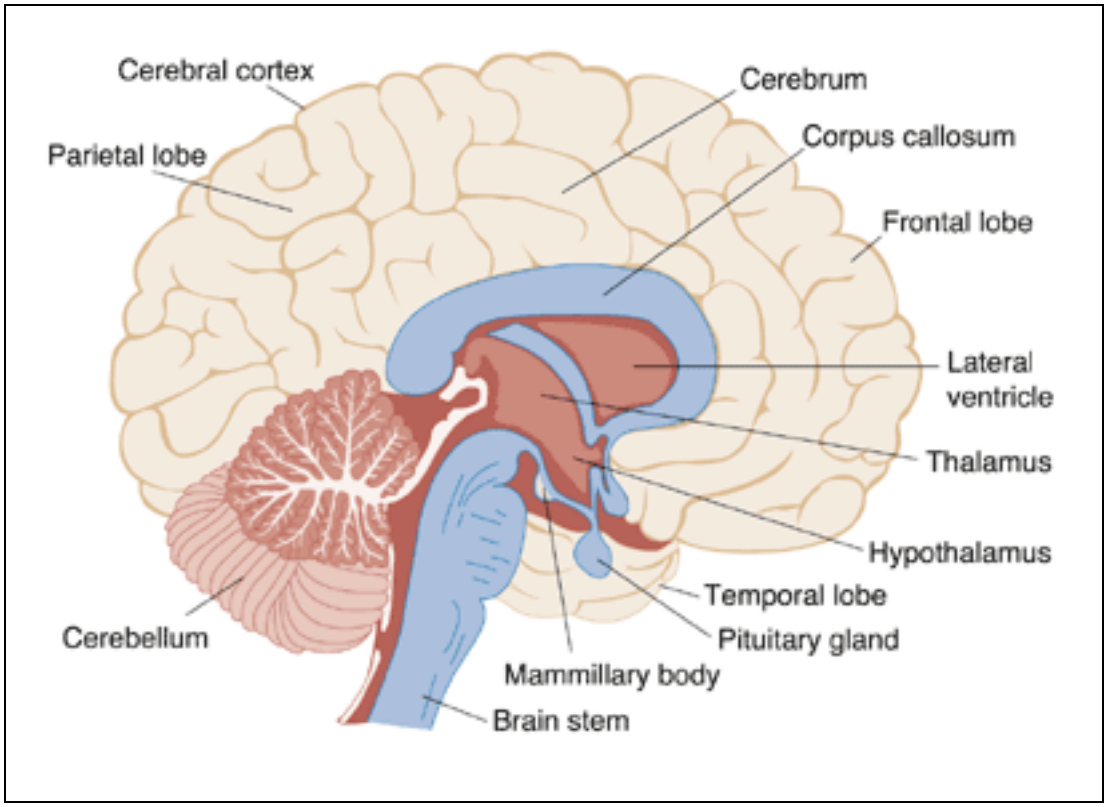
The basal ganglia, together with the cerebellum and the motor cortex, are involved with motor control. A motor command initiated by the cortex is modified and processed within the basal ganglia. This part of the brain helps the cerebral cortex to execute subconscious, learned movements. It scales movement and determines how large, small, fast, or slow a movement needs to be for optimum performance. The basal ganglia also work in conjunction with the substantia nigra as part of the dopamine circuit, which is damaged in Parkinson's disease.

The basal ganglia are sometimes referred to as the "extrapyramidal system" to differentiate them from the pyramidal system (now referred to as the corticospinal tract). It originates in the frontal lobe and is responsible for voluntary movement. Disorders affecting the basal ganglia are still sometimes referred to as extrapyramidal 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 this information upstream to the thalamus for further processing. The brainstem has an ill-defined central core called the brainstem **reticular formation** that houses the respiratory and cardiovascular centers that influence breathing, respiration, blood pressure, circulation, and vasomotor tone.

Medial View of the Brain



This illustration identifies the various areas of the human brain. Source: Oscar-Berman et al., 1997.

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 size, 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 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 other motor control areas of the brain 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.

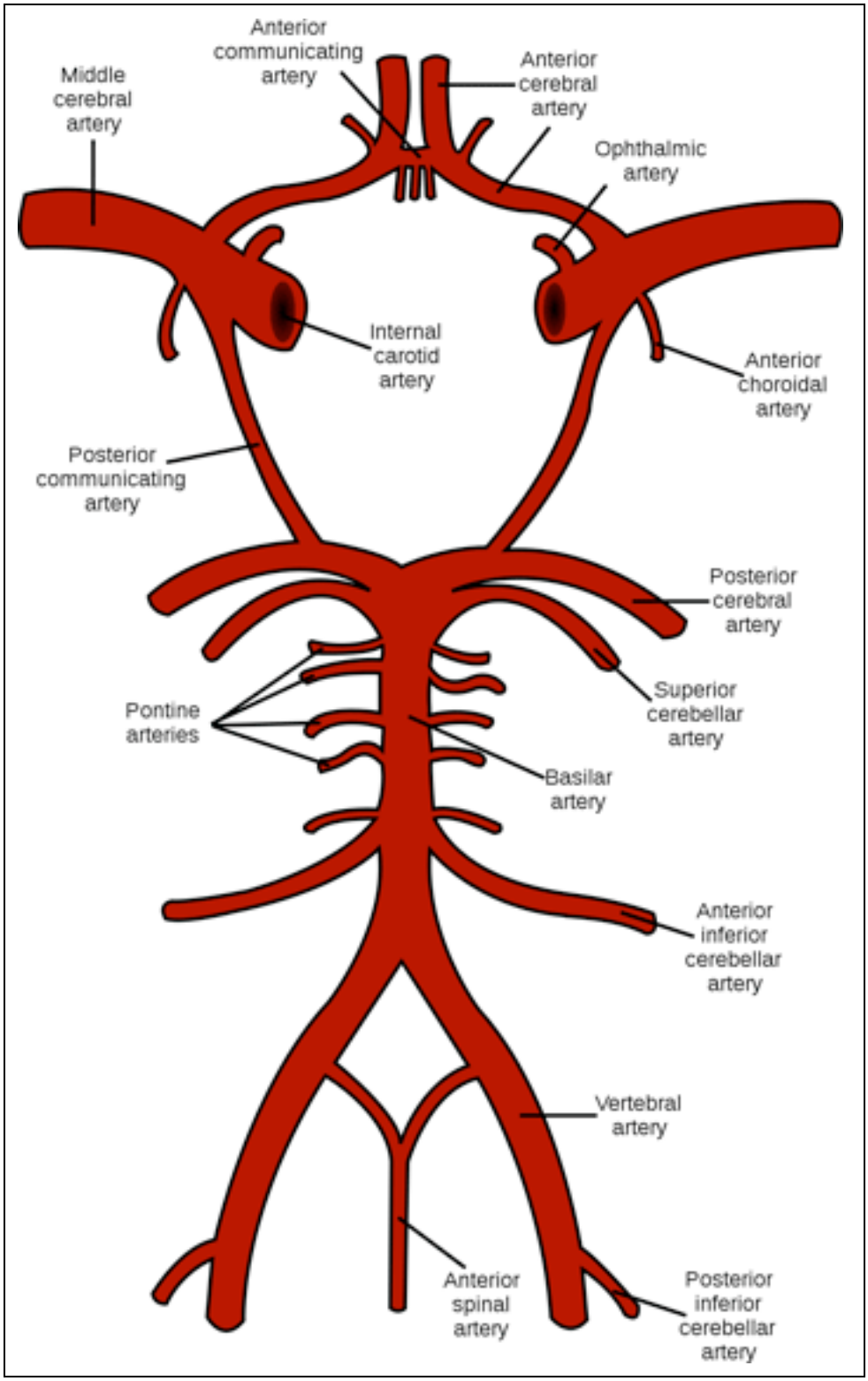
Blood Flow to the Brain

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

Carotid Circulation

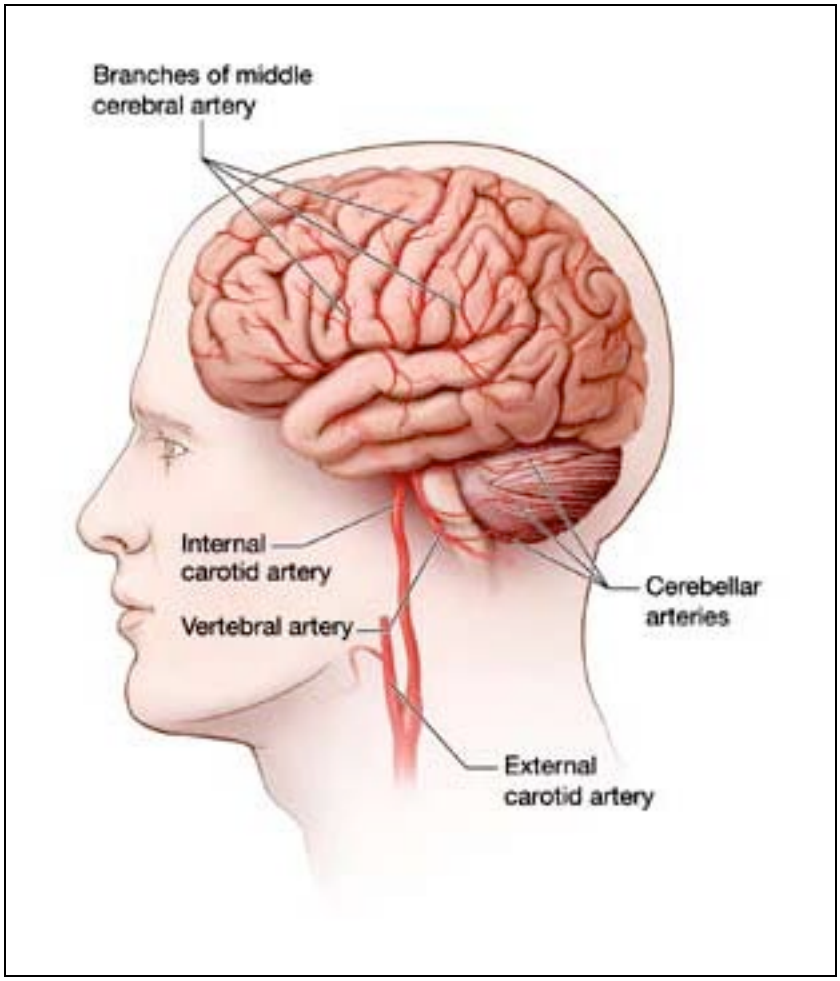
The three clinically important branches of the carotid circulation are the middle cerebral artery, the anterior cerebral artery, and the ophthalmic artery. The middle cerebral artery supplies blood to the lateral of the brain as well as most of the basal ganglia and parts of the internal capsule. At the base of the brain, the carotid and vertebrobasilar arteries form a circle of communicating arteries known as the **circle of Willis**.

The Circle of Willis



Schematic representation of the circle of Willis showing the arteries of the brain and brain stem. Source: Wikimedia Commons. Used with permission.

Carotid and Vertebral Arteries



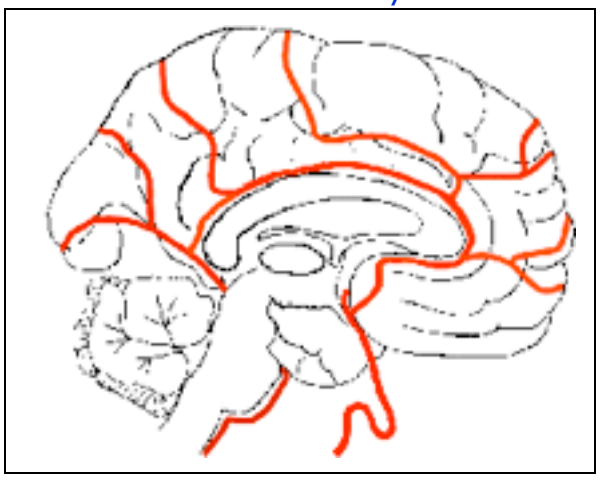
The carotid and vertebral arteries ascend through the neck and divide into branches that supply blood to different parts of the brain. Source: NINDS, Stroke Challenge Brochure, p. 18.

The **middle cerebral artery**, which supplies blood to the lateral surface of each hemisphere, is the largest of the cerebral arteries and the most common artery involved with stroke; embolism is the most common cause of blockage (Slater, 2011). Men are affected by middle cerebral artery stroke more often than women at a male-to-female ratio of 3 to 1 (Slater, 2011).

Because the middle cerebral artery is the area most commonly affected by ischemic stroke, its symptoms are the most familiar to medical workers: 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), as well as cognitive deficits that affect speech, language, and comprehension.

The **anterior cerebral artery** supplies the medial surface of the brain, 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 sometimes referred to as the extrapyramidal system, as noted earlier.

The Anterior Cerebral Artery



Medial surface of the brain showing the areas perfused by the anterior cerebral artery. Source: Lauren Robertson. Used by permission.

Vertebrobasilar or Posterior Circulation

Blood traveling through the two vertebral arteries joins 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.

Disorders Associated with Stroke

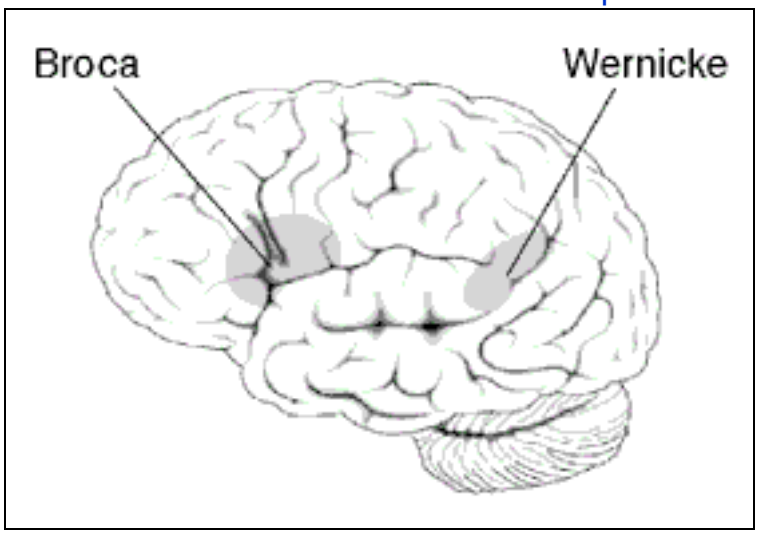
> Neurology's favorite word is "deficit," denoting an impairment or incapacity of neurological function: loss of speech, loss of language, loss of memory, loss of vision, loss of dexterity, loss of identity, and myriad other lacks and losses of specific function (or faculties). For all of these dysfunctions (another favorite term), we have privative words of every sort—aphonia, aphemia, aphasia, alexia, apraxia, agnosia, ataxia—a word for every specific neural or mental function of which patients, through disease or injury, or failure to develop, may find themselves partly or wholly deprived.
>
> Oliver Sachs
>
> *The Man Who Mistook His Wife for a Hat*

Carotid Circulation Disorders

A stroke in any of the major arteries within the carotid circulation (middle cerebral, anterior cerebral, and ophthalmic artery) disrupts the higher cognitive motor and sensory processing. The most common problems—aphasia, apraxia, agnosia, and hemi-neglect, and other cognitive disorders—occur in the areas of the brain supplied by the middle cerebral artery. Similar problems occur with occlusions of the anterior cerebral artery, but the lower extremities and proximal upper extremities are more affected.

If a stroke occurs on the left side of the brain in the middle cerebral artery, **aphasia** (language impairment) is a common occurrence. Aphasia is an acquired communication disorder in people who previously had normal ability. Aphasia is typically defined by the specific region of the brain that has sustained damage. **Wernicke's aphasia** is caused by damage to the lateral surface of the left temporal lobe. It is sometimes referred to as receptive or fluent aphasia because a patient's is fluent but the words carry no meaning. Sentences can be long and meandering—usually longer than seven words.

Areas Related to Broca's and Wernicke's Aphasia



Source: Wikimedia Commons. Used with permission.

Broca's aphasia is caused by damage to the lateral surface of the left frontal lobe. It is sometimes referred to as expressive or non-fluent aphasia because a patient is unable to communicate and sentences are short and choppy—usually less than seven words. **Global aphasia** is a combination of Wernicke's and Broca's aphasia in which a person is unable to understand the spoken word or communicate with speech. A severe stroke may begin with global deficits then slowly resolve to a lesser deficit.

If damage occurs on the right side of the brain, speech and comprehension are usually unaffected but other high-level cognitive deficits occur, including behavioral changes, general confusion and disinhibition, unintentional fabrication of information, memory deficits, attentional deficits, apraxia, and neglect.

Apraxia is another common cognitive problem with strokes in the carotid circulation. **Apraxia** is the loss of the ability to organize a movement or perform a purposeful act. It is a disorder of the execution of movement that cannot be attributed to weakness, incoordination, sensory loss, poor language comprehension, or attention deficit. Apraxia is a weakening of the top-down formulation of an action—the inability to sustain the intent to complete a movement. As a result, the nervous system is easily influenced by irrelevant input—a sort of pathologic absent-mindedness.

Apraxia affects all modalities including speech, writing, gesturing, dressing, and all activities of daily living (ADLs). It is difficult to for caregivers to understand and identify. Examples of apraxia are: picking up a telephone and beginning to talk without dialing, lighting a candle and trying to smoke it as if it were a cigarette, using a knife to brush one's hair, using a pencil to butter bread. In all these examples the brain commands the body to perform a movement but the command fades before the movement is completed. The patient tries to complete the movement but has already forgotten what the task was. Nevertheless, an attempt is made to complete the task—perhaps by guessing.

Case

Barbara is a 73-year-old woman who recently had a stroke and is the rehabilitation unit of a large nursing home. She has been diagnosed with severe apraxia but has no weakness or trouble with her mobility. She is sitting at the side of the bed and, with the help of a nursing assistant, is trying to get dressed. She picks up a sock and moves to put the sock on her right foot. Instead, she places the sock next to the phone. The nursing assistant, in a hurry, hands the sock back to Barbara and tells her to finish getting dressed. Barbara again moves to put the sock on her right foot but slips it over her right hand. The nursing assistant grabs the sock and puts it on Barbara's foot, thinking Barbara is being intentionally uncooperative. After she gets Barbara dressed, the nursing assistant reports to the charge nurse that Barbara is uncooperative and refused to get dressed.

In fact, Barbara is not being uncooperative or refusing to follow instructions. She wants to do what she is asked to do but can't seem to remember how to do anything. Unfortunately, her apraxia will show up in every activity she attempts—from eating to bathing to dressing. If the nursing assistant understood the nature of Barbara's difficulties she could ask for help in dealing with apraxia. The most obvious tactic is to break tasks down for Barbara and understand that she very quickly forgets what she is trying to do. Lots of verbal reinforcement and patience is needed to help Barbara complete her daily activities.

Agnosia is a sensory disorder in which a person is unable to recognize an object by sight, touch, or hearing in the absence of defects in the sensory apparatus of these systems. The person can touch, hear, and see but cannot recognize or identify the object. Agnosia is usually tested by asking a person to identify a series of objects that are placed out of sight in a bag or behind a partition. The person with agnosia will be unable to name an object by touch alone but will be able to identify the object using vision.

Anosognosia (hemi-neglect) is a sensory disorder caused by damage to the parietal lobe in which a person is unaware of the contralateral (opposite) side of the body including half of the visual field. It causes a disruption of a person's body schema and spatial orientation and affects balance and safety awareness. The person is often unaware that the second half of the body exists and will deny that anything is wrong. Those with hemi-neglect may ignore food on the left side of a plate, walk into objects in the left half of the visual field, and completely ignore the left extremities. They may even claim that the affected arm or leg belongs to another person.

A stroke in the ACA circulation affects the medial surface of the brain. It can cause contralateral weakness and sensory loss, primarily in the leg. There may be some weakness in the contralateral arm, especially proximally. It affects the lower extremities more than the upper extremities, leading to difficulties with balance, gait, and mobility. Behavioral disturbances and confusion may be present, and urinary incontinence is not uncommon.

A small clot (microembolus) in the ophthalmic artery, the first branch of the internal carotid artery, can cause partial or complete loss of vision in one eye lasting seconds to minutes; this is called temporary monocular blindness or *amaurosis fugax* (fleeting blindness). It is caused by temporary loss of blood flow to the retina and can be a sign of an impending stroke. It is often described as a gray or black shade that comes down over the eye or as blurring, fogging, or dimming of vision. A clot lodged in the ophthalmic artery can also lead to a sudden and brief bilateral symmetric loss of vision in half of the visual fields that is called **homonymous hemianopsia**.

Loss of Visual Fields in Homonymous Hemianopsia

Paris as seen with right homonymous hemianopsia. The right visual field is missing in both eyes. Source: Wikimedia Commons. Used by permission.

Thalamic Disorders

After a stroke affecting the thalamus, a person may become hypersensitive to pain. This syndrome, called thalamic pain or "central pain syndrome," is due to damage to the spinal tracts that carry pain and temperature sensation from the periphery to the thalamus. Damage to these tracts, called the **spinothalamic** or **trigeminothalamic** tracts result in severe, spontaneous pain in the parts of the body connected to the damaged tracts. Thalamic pain starts several weeks after the stroke and presents as an intense burning pain on the side of the body affected by the stroke; it is often worsened by cutaneous stimulation.

Pain is typically constant, may be moderate to severe in intensity, and is often made worse by touch, movement, emotions, and temperature changes, usually cold temperatures. One or more types of pain sensations may be present—the most prominent being burning. Mingled with the burning may be sensations of pins and needles; pressing, lacerating, or aching pain; and brief, intolerable bursts of sharp pain similar to the pain caused by a dental probe on an exposed nerve. Individuals may have numbness in the areas affected by the pain. The burning and loss of touch sensations are usually most severe on the distant parts of the body, such as the feet or hands.

Basal Ganglia Disorders

In addition to the lateral surface of the cerebral cortex, the middle cerebral artery also supplies blood to the basal ganglia. A stroke affecting the basal ganglia usually causes motor control problems rather than hemiparesis. Damage typically causes too much movement (hyperkinesia) or too little movement (hypokinesia).

Hyperkinesia

> What then is the opposite of deficit—an excess or superabundance of function? Neurology has no word for this—because it has no concept. A function, or functional system, works—or it does not; these are the only possibilities it allows. Thus a disease which is "ebullient" or "productive" in character challenges the basic mechanistic concepts of neurology, and this is doubtless one reason why such disorders—common, important and intriguing as they are—never have received the attention they deserve. And this alone suggests that our basic concept or vision of the nervous system—as a sort of machine or computer—is radically inadequate, and needs to be supplemented by concepts more dynamic, more alive.
>
> Oliver Sachs
>
> *The Man Who Mistook His Wife for a Hat*

Hyperkinesis is too much movement, and we have many words to describe such disorders. **Chorea** is a hyperkinetic movement disorder characterized by arrhythmic, rapid, involuntary movement that flows from one part of the body to another. The most common type of non-drug–related chorea is Huntington's chorea. **Dystonia** is a hyperkinetic movement disorder characterized by involuntary movement that is twisting, sustained, and repetitive. Over time, the affected body part may assume a fixed posture involving one joint (focal dystonia), two joints (segmental dystonia), or several joints (generalized dystonia).

Athetosis is a hyperkinetic movement disorder characterized by spontaneous writhing movements of the hand, arm, neck, or face. **Tardive dyskinesia** is a slow-onset, drug-induced hyperkinetic movement disorder characterized by rhythmic, unwanted movements of the face and extremities such as facial grimacing, tongue movements, and pill-rolling motions with the fingers. **Tourette syndrome** is characterized by excessive energy, tics, jerks, verbal noises, compulsive behavior, and grimaces. It is also associated with other behavioral disorders such as attention deficit disorder.

Hypokinesia

Hypokinesis is too little movement. Parkinson's disease (*paralysis agitans*) is one of the most common hypokinetic movement disorders and is characterized by resting tremor, rigidity, masked faces, bradykinesia, and festinating gait. Parkinson's disease is caused by widespread destruction of a portion of the brainstem (the substantia nigra), which is responsible for sending dopamine to the basal ganglia. Although Parkinson's disease is not caused by stroke it is mentioned here as an example of a hypokinetic movement disorder.

Vertebrobasilar Circulation Disorders

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), or hemi-sensory deficits.

If damage is in the area of the cerebellum, you can expect to see ataxia, intention tremor, and hypotonia. **Ataxia** is motor incoordination due to irregularities in the timing, rate, and force of a muscular contraction. Ataxia causes unsteady, grossly uncoordinated, or "drunken" gait, loss of balance, and a tendency to fall. It also affects the ability to judge the distance or scale of a movement, typified by overshooting or undershooting an object (dysmetria). As a result, vertigo, nausea, vomiting, and nystagmus are common occurrences following a cerebellar stroke.

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 initiated. 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.

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.

Gender, Age, and Racial Differences

Gender Differences

Although men have a higher risk for stroke (1.25 times that for women), more women die from stroke. Because men do not live as long as women and are usually younger when they have a first stroke, men have a higher rate of survival than women. Even though women have fewer strokes than men, women are generally older when they have strokes and are more likely to die from them (NINDS, 2011a).

Nontraditional symptoms are more frequently reported by women than men, including pain and reduced level of consciousness. Women may also have other symptoms, such as nausea, face, arm or leg pain, hiccups, feeling very tired, chest pain, shortness of breath, or a racing heartbeat (Womenshealth.gov, 2010). Recognizing nontraditional symptoms is critical in order to prevent a delay in diagnosis and treatment of stroke.

Some risk factors for stroke apply only to women (eg, pregnancy, childbirth, menopause) and are tied to hormonal fluctuations and changes that affect a woman in various stages of life. Research in the past few decades has shown that high-dose oral contraceptives can increase the risk of stroke in women by as much as 20%; fortunately, they have been replaced with safer and more effective ones containing lower doses of estrogen. Some studies have shown the newer low-dose oral contraceptives may not increase the risk of stroke in women significantly (NINDS, 2011a).

Pregnancy and childbirth can also put a woman at an increased risk for stroke. Pregnancy increases the risk of stroke as much as 3 to 13 times, although it still remains a relatively small risk—approximately 8 in 100,000 women. Up to 25% of strokes during pregnancy end in death, and hemorrhagic strokes are the leading cause of maternal death in the United States. Subarachnoid hemorrhage, in particular, causes 1 to 5 maternal deaths per 10,000 pregnancies (NINDS, 2011a).

The risk of stroke during pregnancy is greatest in the six weeks following childbirth. The risk of ischemic stroke after pregnancy is about 9 times higher and the risk of hemorrhagic stroke is more than 28 times higher for postpartum women than for women who are not pregnant or postpartum. Both preeclampsia and eclampsia cause a rise in blood pressure and an increased tendency to form blood clots that can contribute to this increased stroke incidence (NINDS, 2011a).

Hormonal changes at the end of the childbearing years can increase the risk of stroke. Several studies have shown that menopause can increase a woman's risk of stroke and that hormone replacement may reduce that risk. The mechanism by which estrogen can prove beneficial to postmenopausal women could include its role in cholesterol control. Studies have shown that estrogen acts to increase levels of HDL while decreasing LDL levels (NINDS, 2011a).

Strokes in Young People

People 18 to 45 years of age are considered young adults and have risk factors for stroke such as drug use, alcohol abuse, pregnancy, head and neck injuries, heart disease or heart malformations, and infections. Some other causes of stroke in the young are linked to genetic diseases (NINDS, 2011a).

Hemorrhagic stroke is the most common type of stroke in young adults. Hemorrhagic strokes represent 20% of all strokes in the United States and young people account for many of these. Intracranial hemorrhage accounts for 41% and subarachnoid hemorrhage accounts for 17% of these strokes. The remaining 42% of strokes due to ischemia in the young adult usually require a more exhaustive workup to determine the cause. Despite advances in diagnostic procedures, 20% of strokes in young people continue to be of unknown etiology (Slater, 2011).

Strokes in Children

Medical conditions that can lead to stroke in children include intracranial infection, brain injury, vascular malformations, occlusive vascular disease, and genetic disorders such as sickle cell anemia, tuberous sclerosis, and Marfan's syndrome (NINDS, 2011a).

The symptoms of stroke in children are different from those in mature adults and young adults. A child experiencing a stroke may have seizures, a sudden loss of speech, a loss of expressive language (including body language and gestures), hemiparesis (weakness on one side of the body), hemiplegia (paralysis on one side of the body), dysarthria (impairment of speech), convulsions, headache, or fever. It is a medical emergency when a child shows any of these symptoms (NINDS, 2011a).

In children with stroke, the underlying conditions that led to the stroke should be determined and managed to prevent future strokes. A recent study found that giving blood transfusions to young children with sickle cell anemia greatly reduces the risk of stroke (NINDS, 2011a).

Most children who experience a stroke will do better than most adults after treatment and rehabilitation. This is due in part to the young brain's greater plasticity and the ability to adapt to deficits and injury. Children who experience seizures along with stroke do not recover as well as children who do not have seizures. Some children may experience residual hemiplegia, though most will eventually learn how to walk (NINDS, 2011a).

Strokes in African Americans

African Americans have a higher prevalence of hypertension, diabetes, obesity, smoking, and sickle cell anemia, leading to their having almost twice as many strokes as Caucasians. The incidence of stroke in African American males is 93 per 100,000 and in African American females it is 79 per 100,000. The incidence of stroke in Caucasian males is 63 per 100,000 and in Caucasian females is 59 per 100,000 (MD Guidelines, n.d.).

Prevention

Despite the advent of new treatments for acute ischemic stroke and the promise of other acute therapies, prevention remains the best approach for reducing the incidence of stroke. Age, gender, race, family history, and one's medical history (such as a previous stroke) are non-modifiable risk factors for stroke. But, those who practice a healthy lifestyle have an 80% lower risk of a first stroke compared with those who do not (JAMA, 2011). The public needs education about lifestyle changes that can reduce their risk of stroke.

Once a stroke occurs, it is essential that it is rapidly recognized in order for the stroke victim to receive clot-busting drugs or other stroke treatment as rapidly as possible, because "time is brain." However, many gaps have been identified in the public knowledge of stroke symptoms. It has long been the goal of the Centers for Disease Control (CDC), in conjunction with the American Heart Association (AHA) and the American Stroke Association (ASA) to increase public awareness of stroke signs and symptoms.

Messages about prevention have focused on several key areas of public education including modifiable risk factors such as 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

> Hypertension remains the most important, well-documented, modifiable risk factor for stroke, and treatment of hypertension is among the most effective strategies for preventing both ischemic and hemorrhagic stroke.
>
> *Journal of the American Heart Association*, 2011

About 1 out of 3 American adults has high blood pressure and another 25% have pre-hypertension—blood pressure numbers that are higher than normal, but not yet in the high blood pressure range. In 2010 high blood pressure cost the United States $76.6 billion in healthcare services, medications, and missed days of work. About 70% of those with high blood pressure who took medication had their high blood pressure controlled (CDC, 2011b).

Cholesterol

Approximately 1 in every 6 adults—more than 16% of the U.S. adult population—has high total cholesterol (240 mg/dL and above). People with high total cholesterol have approximately twice the risk of heart disease as people with optimal levels (below 200 mg/dL). 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 and stroke by lowering cholesterol levels. In addition to lifestyle changes, statins (eg, lovastatin, simvastatin) may be needed to reduce cholesterol levels (CDC, 2010a).

Emergency Response

Heart attacks and strokes are life-and-death emergencies in which every second counts. Nearly half of all stroke and heart attack deaths occur before patients are transported to hospitals. For this reason, prehospital emergency medical service (EMS) organizations and providers are vital partners with public health to reduce death and disability from heart attacks and strokes. Additionally, it is important for the public to recognize the major warning signs and symptoms and the need to immediately call 911(CDC, 2011a).

Tobacco

Cigarette smokers have twice the risk of stroke compared to nonsmokers. Smoking decreases the amount of oxygen in the blood, causing the heart to work harder. Smoking promotes atherosclerosis and increases levels of blood clotting factors (NINDS 2011a).

Nutrition

A healthy diet can reduce the risk for acquiring medical conditions such as hypertension, diabetes, high lipid levels, coronary artery disease, and obesity. All of these conditions increase the chance of having a stroke. Recent studies indicate that a diet rich in fruits and vegetables can lower the risk of heart disease and stroke. Those people who ate more than five servings of fruits and vegetables per day had roughly a 20% lower risk of coronary heart disease and stroke compared with individuals who ate less than three servings per day (Harvard School of Public Health, 2011a).

Another study found that a diet rich in fruits, vegetables, and low-fat dairy products lowered systolic blood pressure by 11mm Hg and diastolic blood pressure by almost 6mm Hg—as much as achieved by medications (Harvard, 2011a).

The average American consumes 3400 mg of sodium each day, most of which comes from processed, store-bought, and restaurant foods. Only about 5% comes from salt added during cooking and about 6% comes from adding salt at the table. Current dietary guidelines recommend that adults should consume no more than 2,300 mg of sodium per day. However, the following population groups should consume no more than 1,500 mg per day:

- People 40 years of age or older
- African Americans
- Those with hypertension (CDC, 2009)

Two out of three (69%) adults in the United States fall into one or more of these three groups that are at especially high risk for health problems from too much sodium (CDC, 2009).

Blood pressure rises with increasing amounts of sodium in the diet, and sodium reduction lowers cardiovascular disease and death rates over the long term. Higher salt intake is associated with a 23% increase in stroke and a 14% increase in heart disease (Harvard, 2011b).

Physical Activity

Physical activity can help maintain a healthy weight and lower cholesterol and blood pressure. The Surgeon General recommends that adults should engage in moderate-intensity exercise for at least 30 minutes on most days of the week (CDC, 2010b).

Obesity

Because people who are overweight or obese have an increased risk for cardiovascular disease, diabetes, hypertension, and stroke, weight management can reduce a person's risk from these factors (CDC, 2010b).

Diabetes

People who have diabetes are at least twice as likely as someone who does not have diabetes to have heart disease or a stroke. People with diabetes also tend to develop heart disease or have strokes at an earlier age than other people. Women who have not gone through menopause usually have less risk of heart disease than men of the same age. But women of all ages with diabetes have an increased risk of heart disease because diabetes cancels out the protective effects of being a woman in her childbearing years (National Diabetes Information Clearinghouse, 2005).

People with diabetes who have already had one heart attack run an even greater risk of having a second one. In addition, heart attacks in people with diabetes are more serious and more likely to result in death. High blood-glucose levels over time can lead to atherosclerosis (NDIC, 2005). If blood-glucose levels are high at the time of a stroke, then brain damage is usually more severe and extensive than when blood glucose is well-controlled. Treating diabetes can delay the onset of complications that increase the risk of stroke (NINDS, 2009b).

Carotid Endarterectomy

Carotid endarterectomy is a surgical procedure in which fatty deposits are removed from one of the two carotid arteries located in the neck. Carotid endarterectomy is done to prevent stroke for those who have a certain level of blockage and to prevent recurrent stroke; this is not an acute stroke treatment.

The carotid arteries are the main suppliers of blood to the brain. Two recent NINDS trials showed that carotid endarterectomy is a safe and effective stroke prevention therapy for most people with greater than 50% stenosis of the carotid arteries when performed by a qualified and experienced neurosurgeon or vascular surgeon (NINDS, 2011a).

Patients may need a carotid endarterectomy if they have:

- Had a TIA or stroke with at least 70% narrowing of the carotid artery.
- Had a TIA or mild stroke in the past 6 months that did not leave them completely disabled, and the carotid arteries are at least 50% narrowed.
- Not had a TIA or stroke, but the carotid arteries are narrowed 60% or more and they have a low risk of complications from the surgery. (WebMD, 2009)

Those most likely to benefit from surgery are people who have had symptoms and have 70% or greater narrowing (stenosis) of their carotid artery. People with less than 50% narrowing do not seem to benefit from surgery (WebMD, 2009).

Carotid Endarterectomy

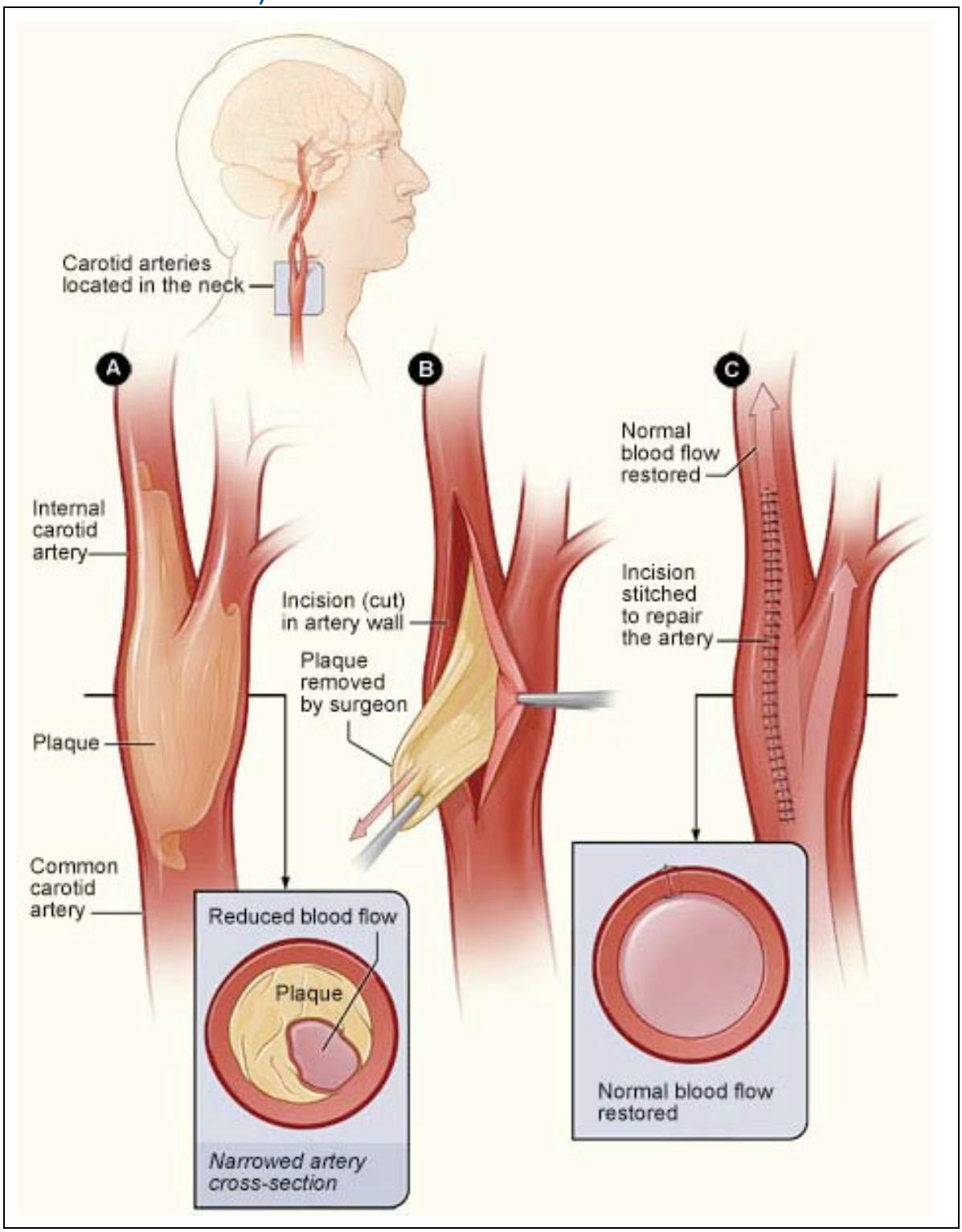


Figure A shows a carotid artery with plaque buildup. The inset image shows a cross section of the narrowed carotid artery. Figure B shows how the carotid artery is cut and how the plaque is removed. Figure C shows the artery stitched up and normal blood flow restored. The inset image shows a cross section of the artery with plaque removed and normal blood flow restored. Source: NIH, n.d.

A large clinical trial was done to test the effectiveness of carotid endarterectomy versus carotid stenting. Stenting involves inserting a long, thin catheter into an artery in the leg and threading the catheter through the vascular system into the stenosis of the carotid artery. Once the catheter is in place, the radiologist expands the stent with a balloon on the tip of the catheter to open the stenosis (NINDS, 2011a).

Following up after an average of 2.5 years, there was no difference in the estimated 4-year rates of early stroke and later stroke, heart attack, or death—between carotid artery stenting and carotid endarterectomy. The study also found that the age of the patient made a difference. At age 69 and younger, stenting results were slightly better. Conversely, for patients older than 70, surgical results were slightly superior to stenting (NINDS, 2010a).

Carotid endarterectomy has been shown to reduce the risk of TIA and stroke in people with moderate to severe narrowing (70%–99%) of the carotid arteries. Carotid endarterectomy is three times more effective than treatment with medication alone in these patients (WebMD, 2009).

Symptomatic and asymptomatic patients undergoing carotid endarterectomy should be given aspirin (81to 325 mg/day) prior to surgery and for at least 3 months following surgery to reduce the risk of myocardial infarction or stroke. It is recommended that aspirin be continued indefinitely provided that contraindications are absent. Aspirin at 650 or 1,300 mg/day is less effective in the perioperative period. The data are insufficient to recommend the use of other antiplatelet agents in the perioperative setting (AHRQ, 2010c).

Recognizing Stroke Symptoms

The most common identifying feature of stroke is its acute onset. Every second a clot blocks blood flow to the brain, 32,000 brain cells die. Administration of clot-busting thrombolytic drugs must happen as soon as possible after onset of symptoms to prevent further brain damage. A New York study determined that only 20% of patients arrived at a designated stroke center within 3 hours of stroke symptom onset (the recommended time frame for use of thrombolytics). This study showed that more than 70% of respondents would call 911 if they noticed someone having difficulty speaking, but only 33% would call 911 for double vision or trouble seeing (Jurkowski et al., 2008).

Stroke Symptoms

Source: National Institutes of Health.

The delay between symptom onset and arrival at a hospital is influenced by:

- Identification of stroke symptoms
- Determination that the symptoms require immediate emergency care
- Calling 911
- The time it takes until hospital arrival

Evidence suggests that most of the delay between symptom onset and hospital arrival occurs before the call to 911 is made (Jurkowski et al., 2008).

The CDC, AHA, and ASA, 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, 2010).

The consensus symptoms are:

- Sudden numbness or weakness of face, arm, or leg, especially on one side of the body
- Sudden confusion or trouble speaking or understanding speech
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, or loss of balance or coordination
- Sudden severe headache with no known cause

The "suddens" were adopted by several national and state-based educational campaigns and are used to convey stroke symptoms in clinical and public health settings and among advocacy organizations concerned with stroke.

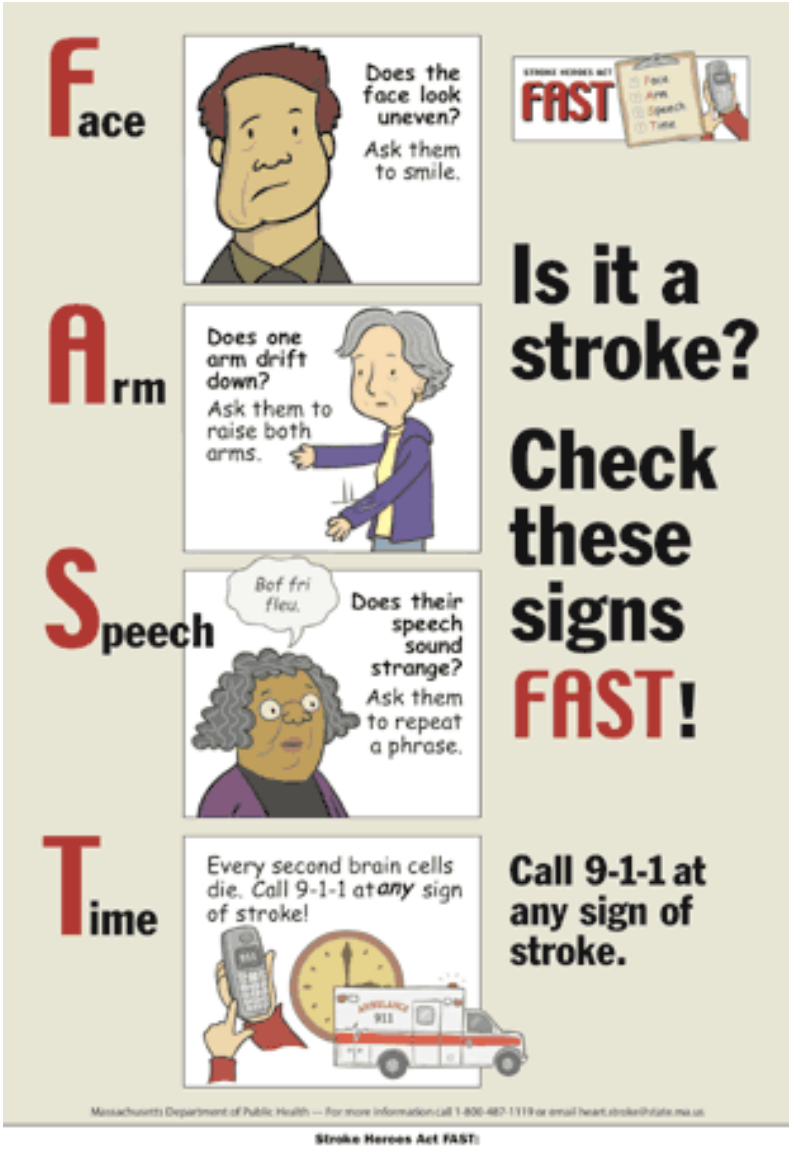
Although consensus on stroke symptoms has been achieved, public awareness has still lagged behind. For example, advocacy organizations in Massachusetts have annually conducted at least one campaign on the signs and symptoms of stroke. Yet in 2003 only 18% of Massachusetts adults were aware of all signs and symptoms of stroke, but 80% said they would call 911 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, 2010).

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 to develop an evidence-based approach to educate the public to recognize the signs of stroke and respond by calling 911. The campaign showed that a 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 care (Wall, 2010).

In another instance, the Cincinnati Prehospital Stroke Scale (CPSS), a three-item scale based on a simplification of the National Institutes of Health Stroke Scale was accurately used by untrained laypeople to identify stroke signs in mock patients and in stroke survivors when prompted by a 911 telecommunicator. The CPSS can identify stroke patients who are candidates for thrombolytic treatment when performed by a physician and has similar results when used by prehospital care providers (Wall, 2010).

The CPSS was modified with the addition of a fourth item, so that it could be used by lay people before they called 911. These and other studies lead to the development of the **Stroke Heroes Act FAST** campaign which, in a retrospective chart review of 3500 stroke patients, successfully identified almost 90% of patients with stroke or transient ischemic attack (Wall, 2010).

Checking for Signs of Stroke



F: Does the face look uneven?

A: Does the arm drift down?

S: Does the speech sound strange.

T: Every second brain cells die. Call 911 at any sign of stroke.

Source: CDC.

Acute Stroke Treatment

Pre-Hospital Care

Most strokes occur at home and the EMS is the first medical contact for more than half of all patients who have a stroke. Because EMS transport significantly shortens arrival time to the emergency department and because of the narrow window for delivery of time-dependent medications, EMS plays a pivotal role in the rapid treatment of stroke. A recent 3-year data analysis found that stroke patients brought in by EMS were twice as likely to receive a timely CT scan compared to those who did not use EMS (National Stroke Association, 2011).

Nine-one-one dispatchers, first responders, EMTs, and paramedics are often the first source of medical information and play a vital role in the initial triage of potential stroke victims. It is imperative that the stroke system of care provide training and education for these people in order to minimize delays in pre-hospital dispatch, assessment, and transport (Circulation, 2010).

Studies have found that the greatest portion of the delay between onset of symptoms and emergency treatment is the time it takes for a patient to recognize the signs of stroke and decide to seek medical care. Between one-half and three-quarters of ischemic stroke patients do not arrive at the hospital within the 3-hour window of treatment that is needed to make an assessment and begin therapy. Some of the factors in the delays include lack of knowledge regarding stroke symptoms, treatment options, and the need for quick therapy (Williams et al., 2009).

Delays in treating stroke also occur because of poor recognition of stroke by 911 dispatchers and misdiagnosis of stroke by EMS personnel. Dispatch is a crucial link in the chain of care, yet dispatchers miss as many as 70% of stroke cases because they do not have the understanding or tools to properly assess the symptoms reported by callers. A similarly high misdiagnosis rate (61%) was documented for the responding EMS personnel when diagnosing stroke in the field (Williams et al., 2009).

These findings underscore the challenges facing the emergency systems of care. The lack of close coordination of stroke care among healthcare providers has resulted in a fragmented system for stroke prevention, emergency care, treatment, and rehabilitation (Williams et al., 2009).

To improve stroke outcome, a better integration of the facilities, agencies, and professionals that provide stroke care is needed. There needs to be rapid access to EMS, use of diagnostic algorithms, and EMS protocols that reflect the most current stroke treatment, as well as recommendations to dispatch EMS as rapidly as possible to improve outcomes. Emergency physicians and stroke experts should be involved in the design of protocols and training programs, stroke assessment and thrombolytic screening tools, and should encourage rapid transport to a stroke center (Williams et al., 2009).

The CDC, in collaboration with the North Carolina office of EMS, created the NEMSIS-based North Carolina Prehospital Medical Information System (PreMIS), to develop and implement EMS stroke performance improvement toolkits. The EMS toolkits focus on:

- Prompt recognition of stroke through the use of stroke screening
- Documentation of stroke symptom onset—the last time the person was seen well
- Screening the blood glucose of the patient for hypoglycemia

- Maintaining EMS scene times of 10 minutes or less
- Rapid transport (with early notification) to a stroke center (Williams et al., 2009)

EMS providers should provide pre-arrival notification to a medical facility capable of providing acute stroke care. Pre-arrival notification has been found to increase the number of stroke patients who receive fibrinolytic therapy. Recent studies indicate a favorable benefit from triage of stroke patients directly to designated stroke centers (Circulation, 2010).

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).

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.
- Ambulance service organizations should be encouraged to use the Cincinnati Stroke Scale as the standard for assessing patients suspected of having stroke.
- Hospital emergency 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 emergency 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 semi-annually. 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)

Emergency Department Care

As in the prehospital phase, initial patient assessments made in the emergency department (ED) are based on evaluation of airway, breathing, and circulation, vital signs, and neurologic status. Most acute ischemic stroke patients arrive to the ED hemodynamically stable; however, patients with decreased level of consciousness may require airway management (Stroke, 2009).

Acute stroke produces an increase in blood pressure in approximately 80% of patients and pressure must be monitored frequently. Control of blood pressure is important because significant elevation in blood pressure is an exclusion criterion for administration of thrombolytic therapy (recombinant tissue plasminogen activator, or rtPA). Elevated blood pressure after administration of rtPA is associated with increased risk of intracerebral hemorrhage.

When a suspected stroke patient arrives to the ED, triage is usually the function of the nursing staff. 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. The triage nurse, using the 5-level Emergency Severity Index, will assign most stroke patients an acuity level 2, meaning the patient needs immediate assessment. The nurse must be able to recognize symptoms suggestive of stroke and determine the last time the patient was seen normal (Stroke, 2009).

Because the benefit of thrombolytics in acute ischemic stroke is strongly time-dependent, ED 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. Each minute that goes by, more nerve cells die. Treatment is most beneficial in the first minutes after stroke and declines steadily during the first 3 hours (Jeffrey, 2009).

Because there is no way to tell whether symptoms are from a TIA or an acute stroke, assume that all stroke-like symptoms signal an emergency—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 treat or reduce the risk of stroke. 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. Evaluation and treatment of acute stroke within one hour of arrival to the emergency department must include:

- An initial patient evaluation within 10 minutes of arrival in the ED. This should include patient history, insertion of 2 to 3 peripheral IVs, bedside blood glucose, initiation of lab work, and NIHSS (National Institutes of Health Stroke Scale) assessment by the physician. An electrocardiogram does not take priority over the CT scan, but should be done as soon as possible (Circulation, 2010).
- Notify the stroke team within 15 minutes of arrival.
- Initiate a CT scan within 25 minutes of arrival.
- Interpret the CT scan and labs within 45 minutes of arrival.
- Ensure a door-to-drug (needle) time of 60 minutes from arrival for eligible patients.

The patient should be transferred to an inpatient setting, preferably a stroke unit, within 3 hours of arrival to the emergency department (Circulation, 2010).

Because of the narrow therapeutic window for the use of thrombolytic medications, acute stroke teams are recommended to ensure the fastest onset-to-treatment time possible. The teams should comprise:

- **Code team**—responds to a code pager and delivers urgent treatment. May consist of a neurologist, ED physician, and nurse.
- **Task force**—works daily to facilitate patient access to treatment. May include members from neurology, emergency medicine, neurosurgery, nursing, pharmacy, laboratory, physical medicine, and rehabilitation (Ellmers, 2011)

The NIH Stroke Scale

The American Heart Association recommends that all emergency departments assess the severity of stroke using the National Institutes of Health Stroke Scale (NIHSS). The NIHSS is a graded neurologic examination assessing consciousness, eye movements, visual fields, motor and sensory impairments, ataxia, speech, cognition, and inattention. The scale was developed as a communication tool; it is simple and quick, and has shown significant reliability across diverse groups, settings, and languages. However the NIHSS also contains items with poor reliability and redundancy. The modified NIHSS (mNIHSS) minimizes redundancy and eliminates items with poor reliability (Meyer et al., 2010).

Note: Since the mNIHSS is more reliable, it allows for improved communication, better medical care, and refinement of trial enrollments. The mNIHSS should serve as the primary stroke clinical deficit scale for clinical and research aims (Meyer et al., 2010)

The NIHSS is used to evaluate the level of impairment sustained by a stroke patient, immediately and at intervals post onset. Scores increase as neurologic deficits increase. Although a level of neurologic deficit on the NIHSS has not been established for treatment with thrombolytics, the Food and Drug Administration (FDA) has included a package insert in rtPA instructions listing a score greater than 22 as a warning. Additionally, patients with a score higher than 22 are at greater risk for hemorrhage transformation if they are given rtPA. The mNIHSS is summarized in the table below.

Modified NIH Stroke Scale Summary (mNIHSS)		
Item	Name	Response
1A	Level of consciousness	0=Alert 1=Not alert 2=Unresponsive
1B	Level of consciousness questions. Patient is asked the month and his/her age.	0=Answers both questions correctly 1=Answers one question correctly 2=Answers neither correctly
1C	Level of consciousness commands. Patient is asked to open and close the eyes and then to grip and release the non-paretic hand.	0=Performs both tasks correctly 1=Performs one task correctly 2=Performs neither task
2	Best gaze. Only horizontal eye movement will be tested.	0=Normal 1=Partial gaze palsy 2=Total gaze palsy
3	Visual fields: Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate.	0=No visual loss 1=Partial hemianopsia 2=Complete hemianopsia 3=Bilateral hemianopsia
4	Facial palsy. Ask the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace.	0=Normal 1=Minor paralysis 2=Partial paralysis 3=Complete paralysis
5	Motor arm: The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds.	a. Left or b. Right • 0=No drift • 1=Drift before 10 sec • 2=Fall before 10 sec • 3=No effort against gravity • 4=No movement
6	Motor leg: The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds.	a. Left or b. Right • 0=No drift • 1=Drifts before 5 sec • 2=Fall before 5 sec • 3=No effort against gravity • 4=No movement

Modified NIH Stroke Scale Summary (mNIHSS)		
Item	Name	Response
7	Ataxia: Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness.	0=Absent 1=One limb 2=Two limbs
8	Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient.	0=Normal 1=Mild loss 2=Severe loss
9	Language: The patient is asked to describe pictures and read a list of sentences.	0=Normal 1=Mild aphasia 2=Severe aphasia 3=Mute or global aphasia
10	Dysarthria: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from an attached list.	0=Normal 1=Mild 2=Severe
11	Extinction/inattention: Sufficient information to identify neglect may be obtained during the prior testing.	0=Normal 1=Mild 2=Severe

Source: NIH, n.d.

For hemorrhagic strokes, the Glasgow Coma Scale is used in addition to the NIH Stroke Scale to determine level of consciousness and severity of the underlying change of consciousness (AHRQ, 2010b). The Glasgow Coma Scale ranges from 3 to 15, in which progressively higher scores indicate higher levels of consciousness.

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.

There are a variety of imaging devices used to evaluate stroke patients. The most widely used imaging procedure is the computed tomography (CT) scan. Also known as a CAT (computed axial tomography) scan, CT creates a series of cross sectional images of the head and brain. Because it is readily available at most major hospitals and produces images quickly, CT is the most commonly used diagnostic technique for acute stroke.

CT also has unique diagnostic benefits. It will quickly rule out a hemorrhage, can occasionally show a tumor that might mimic a stroke, and may even show evidence of early infarction. Infarctions generally show up on a CT scan about 6 to 8 hours after the start of stroke symptoms (NINDS, 2011a).

If a stroke is caused by hemorrhage, a CT can show evidence of bleeding into the brain almost immediately after stroke symptoms appear. Hemorrhage is the primary reason for avoiding thrombolytic therapy. Thrombolytic therapy cannot be used until the doctor can confidently diagnose the patient as suffering from an ischemic stroke because this treatment could make a hemorrhagic stroke worse (NINDS, 2011a).

Magnetic resonance imaging (MRI) is also used with stroke patients. MRI uses magnetic fields to detect subtle changes in brain-tissue water content. One effect of stroke is the slowing of water movement, called diffusion, through the damaged brain tissue. MRI can show this type of damage within the first hour after stroke symptoms start. The benefit of MRI over a CT scan is more accuracy and earlier diagnosis of infarction, especially for smaller strokes, while showing equivalent accuracy in determining when hemorrhage is present. MRI is more sensitive than CT for other types of brain disease, such as brain tumor, that might mimic a stroke. MRI cannot be performed in patients with certain types of metallic or electronic implants, such as pacemakers (NINDS, 2011a).

In 2010 the American Academy of Neurology concluded that diffusion-weighted imaging MRI is superior to noncontrast CT scans for diagnosing ischemic stroke within 12 hours of onset of symptoms. However, due to time constraints for administration of thrombolytics, the longer time required to complete an MRI, and because MRI is often not immediately available, a noncontrast CT is still recommended to rule out hemorrhagic stroke in patients being considered for thrombolysis (Anderson, 2010).

Other types of MRI scans—often used for the diagnosis of cerebrovascular disease and to predict the risk of stroke—are magnetic resonance angiography and functional magnetic resonance imaging (fMRI). Neurosurgeons use magnetic resonance angiography to detect stenosis (blockage) of the brain arteries inside the skull by mapping flowing blood. Functional MRI uses a magnet to pick up signals from oxygenated blood and can show brain activity through increases in local blood flow (NINDS, 2011a).

Duplex Doppler ultrasound and arteriography are two diagnostic imaging techniques used to decide if an individual would benefit from carotid endarterectomy—used to remove fatty deposits from the carotid arteries to help prevent stroke. Doppler ultrasound is a painless, noninvasive test in which sound waves above the range of human hearing are sent into the neck. Echoes bounce off the moving blood and the tissue in the artery and can be formed into an image. Ultrasound is fast, painless, risk-free, and relatively inexpensive compared to magnetic resonance angiography and arteriography, but it is not considered to be as accurate as arteriography (NINDS, 2011a).

Arteriography involves use of an x-ray of the carotid artery that is taken as dye is injected into the artery. The procedure carries its own small risk of causing a stroke and is costly to perform. The benefits of arteriography over magnetic resonancing techniques and ultrasound are that it is extremely reliable and still the best way to measure stenosis of the carotid arteries (NINDS, 2011a).

Ischemic Stroke Therapies

Recombinant tissue plasminogen activator (rtPA) is the only drug approved by the FDA for acute ischemic stroke and must be given intravenously within 3 hours of time of onset (the last time the patient was seen normal) of stroke symptoms. Other procedures such as interventional radiology and mechanical removal of the clot have increased the time frame for ischemic stroke treatment (Gesensway, 2010).

Thrombolytics

The FDA approved the use of rtPA in 1996, partly on the basis of a study done by the National Institutes of Neurological Disorders and Stroke (NINDS). The study, published in *New England Journal of Medicine* in 1995, showed neurologic improvement was achieved in 31% to 50% of patients treated with rtPA compared with 20% to 38% of patients given a placebo. The major risk of treatment was symptomatic intracranial hemorrhage (NIH, 2009).

The 1995 NINDS study was a major breakthrough in the treatment of ischemic stroke. The FDA, the AHA, the National Stroke Association (NSA), and the media touted the effectiveness of rtPA (also known as "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.

A 1998 follow-up analysis of the NINDS trial found that after their initial hospitalization, people who received rtPA were less likely to require inpatient rehabilitation or nursing home care. The authors estimated that this lower dependency on long-term care would translate into a savings to the healthcare system of more than $4 million for every one thousand individuals treated with rtPA (NIH, 2009).

Because rtPA interferes with blood clotting and has also been shown to increase leaking along the blood-brain barrier, it carries a risk of intracerebral hemorrhage. According to the original NINDS study, hemorrhage is most likely to occur in the first 3 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 tPA as compared to 0.6% who received the placebo (NINDS, 1995). Nearly half of those hemorrhages were fatal.

Subsequent studies have demonstrated that using rtPA more liberally than is recommended in the NINDS protocol resulted in a higher rate of intracranial hemorrhage. Complications are more likely when rtPA is used in patients over 70 years old, those with more severe stroke (NIHSS over 15), or those with glucose over 300 mg/dl. Therefore, rtPA is not recommended for patients who do not meet the inclusion and exclusion criteria. Because brain cells rapidly die as they are deprived of oxygen and because the risk of rtPA-induced hemorrhage increases over time from stroke onset, its use is limited to the first 3 hours (in most cases) after the start of stroke symptoms (NIH, 2009).

Thrombolytic agents are used to treat an ongoing, acute, ischemic stroke; they halt the stroke by dissolving the blood clot that is blocking blood flow to the brain. rtPA is a genetically engineered form of tPA, a thrombolytic or clot-busting substance made naturally by the body. It should be used only after it has been confirmed that the patient has suffered an ischemic stroke (NINDS, 2011b).

In an ischemic stroke, there is an area of irreversibly damaged brain tissue surrounded by an area of at-risk but viable tissue called the penumbra. During the first 3 hours after the onset of symptoms, reperfusion therapy can save this viable tissue by using clot-busting drugs (thrombolytics) to dissolve the clot. The longer therapy is delayed, the more brain cells will die (NIH, 2009).

rtPA is currently the only drug approved by the FDA for use in acute ischemic stroke (Rivera-Bou, 2010). It has a 30% recanalization rate when given within 3 hours of symptom onset in the acute setting, and has been shown to improve overall recovery rates at 1 year post stroke (Cronin, 2011).

For patients with ischemic stroke who meet the inclusion and exclusion criteria, the American College of Chest Physicians' evidence-based clinical practice guidelines recommend administration of IV rtPA in a dose of 0.9 mg/kg (maximum of 90 mg), with 10% of the total dose given as an initial bolus and the remainder infused over 60 minutes (AHRQ, 2010a).

Contraindications for treatment with rtPA include recent hemorrhage, increased risk of hemorrhage, arterial puncture at a non-compressible site, and systolic pressure >185 mm Hg or diastolic pressure >110 mm Hg (McPhee and Papdakis, 2011).

Treatment must be initiated within 3 hours of clearly defined symptom onset and all unnecessary delays must be avoided because the benefits of rtPA therapy diminish rapidly over time. For patients with extensive and clearly identifiable hypodensity on CT, the guideline developers suggest not using rtPA (AHRQ, 2010a).

When considering thrombolytics for a stroke patient with symptom onset greater than 3 hours and less than 4.5 hours, the following exclusion criteria should be added (Rivera-Bou, 2010):

- Age greater than 80 years
- Those taking anticoagulants regardless of their INR
- NIH stroke scale score >25
- A history of stroke and diabetes

Note: In 2009 the American Heart Association (AHA) and the American Stroke Association (ASA) published a science advisory recommending that the time window for rtPA administration be increased to 4.5 hours after onset of stroke symptoms, although this change has not been approved by the FDA (Slater, 2011). Despite the potential benefit of rtPA extending out to 4.5 hours, it should be administered as early as possible for the best outcome (Saver, 2011).

Interventional Radiology

Interventional radiology offers a longer treatment window for some patients with acute ischemic stroke. Intra-arterial rtPA can be given up to 6 hours after onset of stroke symptoms and has the same efficacy as intravenous rtPA. Using x-ray guidance, a catheter is threaded through the femoral artery into the tiny arteries of the brain where rtPA is delivered directly into the clot (Cronin, 2011). Because less rtPA is used, there is less chance of intracranial bleeding.

Mechanical clot removal can be a treatment option for stroke patients who may arrive at the hospital too late, or have contraindications to the use of rtPA. The mechanical embolus removal in cerebral ischemia (MERCI) retriever can be used up to 8 hours after symptom onset. The device is threaded through the femoral artery to the site of the stroke to retrieve the clot. The retriever has received approval from the FDA for use in patients with persistent vessel occlusion after IV rtPA (Lutsep, 2011).

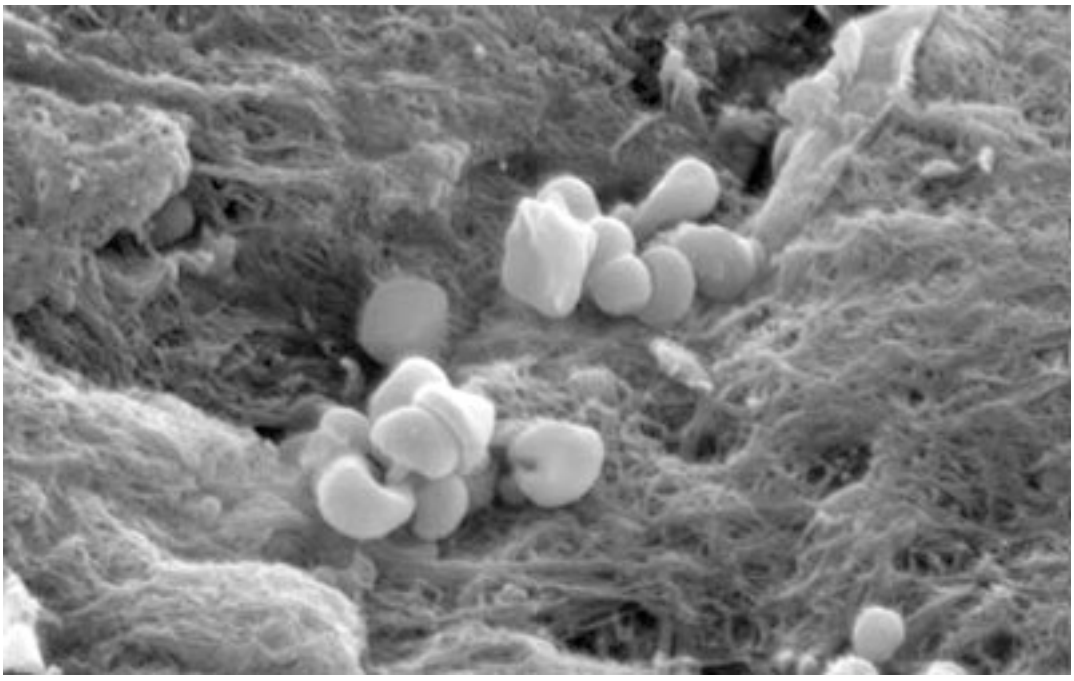
One study showed recanalization occurred in 55% of patients who were treated with a MERCI device alone and in 68% of patients who were treated with a MERCI device plus adjuvant treatment (Lutsep, 2011).

Approved in 2007 by the FDA, the penumbra system uses aspiration to remove a clot. The system is threaded through the femoral artery via catheter to the thrombus site where the radiologist breaks up the clot with a separator and then aspirates it. This system can be used up to 8 hours from symptom onset and has shown up to an 82% recanalization success rate (Cronin, 2011).

Antiplatelets

Antiplatelet therapy is used for both the prevention and management of acute ischemic stroke. Antiplatelet drugs inhibit the activity of cells called platelets, which stick to damaged areas inside blood vessels and lay the foundation for blood clots. Antiplatelets do not break up clots. Patients with ischemic stroke or TIA who are not on anticoagulation should be taking an antiplatelet agent such as aspirin. When patients are aspirin-intolerant, clopidogrel, or a combination of low-dose aspirin and dipyridamole, modified release, should be used. Dipyridamole may also be taken alone (AHRQ, 2010d).

Platelets Attached to Blood Vessels



Platelets (magnified here thousands of times) cling to damaged areas of blood vessels and contribute to the formation of clots. Antiplatelet drugs can help reduce the risk of ischemic stroke. Source: NIH, n.d.

Aspirin is the oldest and most common antiplatelet medicine. In a meta-analysis of trials of aspirin in the secondary prevention of cardiovascular and cerebrovascular events, aspirin reduced the number of strokes by more than 20%. For patients with acute ischemic stroke who are not receiving thrombolysis, early aspirin therapy is recommended to stop the growth of the clot (initial dose of 150 to 325 mg) (AHRQ, 2010d). Aspirin has also been proven to be the most effective immediate treatment after an ischemic stroke to reduce the likelihood of another stroke (Mayo Clinic, 2010). Aspirin should not be given within 24 hours of administration of rtPA.

The maximal antiplatelet effect of aspirin may take several days when given in doses as low as 75 mg/day, so low doses are used for the long-term prevention of heart attacks and strokes. When immediate anti-clotting effects are needed, moderate doses of aspirin (160–325 mg) are used (Medicinenet.com, 2011).

Anticoagulants

Anticoagulants reduce stroke risk by preventing the formation of clots and the extension of existing clots, but do not break them up. The most commonly used anticoagulants include warfarin (Coumadin), heparin, and enoxaparin (NINDS, 2011a).

There have been several trials to test the efficacy of anticoagulants versus antiplatelet drugs. It has been found that, although aspirin is an effective therapy for the prevention of a second stroke in most patients with atrial fibrillation, some patients with additional risk factors do better on warfarin therapy. A recent study tested the effectiveness of low-molecular-weight heparin (enoxaparin) in stroke prevention. This study showed that heparin anticoagulants are not generally effective in preventing recurrent stroke or improving outcomes (NINDS, 2011a).

Warfarin is often prescribed to prevent the possibility of clotting and stroke in patients with atrial fibrillation. Atrial fibrillation raises the risk of stroke 4 to 6 times. Some of these patients have a lower risk of stroke and are treated with aspirin, which reduces clotting but is not as strong as warfarin. Other treatments include medications such as beta blockers or calcium channel blockers to slow the heartbeat, and anti-arrhythmic drugs or electrical cardioversion to normalize the heartbeat (NINDS, 2009c).

Patients with acute ischemic stroke treated with rtPA should not be treated with anticoagulation for at least 24 hours post thrombolysis (Cruz-Flores, 2011). If heparin or any other anticoagulant is indicated after 24 hours, a non-contrast CT scan or other sensitive diagnostic imaging method should be done to rule out intracranial hemorrhage before starting an anticoagulant (The Brain Attack Coalition, n.d.).

Hemorrhagic Stroke Therapies

[This section taken largely from National Heart Lung and Blood Institute, 2011.]

Hemorrhagic stroke occurs if an artery in the brain leaks blood or ruptures. Patients with hemorrhagic stroke usually present with neurologic symptoms similar to ischemic stroke patients, but they tend to be sicker. The first step in treating a hemorrhagic stroke is to find the cause of bleeding in the brain and then control it.

Surgery may be needed to treat a hemorrhagic stroke. If an aneurysm is the cause of a stroke, aneurysm clipping or coil embolization may be done. An **aneurysm clipping** is done to block off the aneurysm from the blood vessels in the brain. During the procedure, a surgeon places a tiny clamp at the base of the aneurysm.

Coil embolization is a less complex procedure for treating an aneurysm. The surgeon inserts a catheter into an artery in the groin that is threaded to the site of the aneurysm. A tiny coil is then pushed through the tube and into the aneurysm, causing a blood clot to form, blocking blood flow through the aneurysm, and preventing it from bursting again.

If an arteriovenous malformation (a tangle of faulty arteries and veins that can rupture within the brain) is the cause of hemorrhagic stroke, a repair may be done to prevent further bleeding. Types of repair are:

- Surgery to remove the arteriovenous malformation
- Injection of a substance into the blood vessels of the arteriovenous malformation to block blood flow
- Radiation to shrink the blood vessels of the arteriovenous malformation

Did you know. . .

All strokes are treated as ischemic until a CT scan confirms otherwise. The reason is that, with an ischemic stroke, the staff must follow the strict timeline and protocol for possible rtPA administration. If a hemorrhage is present, different treatment is required.

Hospital Care

In some cases stroke patients are transferred to intensive care until they are stabilized. Those who have received rtPA are always sent to intensive care, as are many of those with hemorrhagic stroke.

Delivery systems for acute stroke hospital care are relatively primitive compared to systems for state-of-the-art emergency cardiac care. This is partly a reflection of being able to provide only supportive care to ischemic stroke patients until the approval of rtPA. The approval of intravenous rtPA for selected patients with ischemic stroke exposed these deficiencies and mandated changes in the hospital care system. These long-needed changes will also benefit patients with hemorrhagic strokes (NINDS, 2011c).

Once a patient has been evaluated and treated in the ED, the patient should be transferred to a specialized stroke unit (usually intensive care). Primary and comprehensive stroke centers have stroke units with specially trained staff and a multi-disciplinary approach to treatment and care of stroke patients. These units have been shown to be superior to general medical units and have been shown to result in positive effects that last for years (Circulation, 2010). Stroke unit care reduces the likelihood of death and disability by as much as 30% in men and women of any age with mild, moderate, or severe stroke (AHRQ, 2009).

Nursing care focuses on continued stabilization of the stroke patient. Vital signs and neuro checks must be done every 15 minutes for 2 hours after administration of rtPA, then every 30 minutes for 6 hours, and every hour for the next 16 hours. The NIH stroke scale is done every hour for the first 24 hours after rtPA administration. The NINDS rtPA Stroke Study and the AHA recommendations for rtPA include strict monitoring and regulation of blood pressure below defined upper limits with antihypertensive agents. There are protocols for emergency management of hemorrhage in rtPA-treated patients, specific guidelines for the control of hypertension, and recommendations for management of bleeding complications. Any neurologic changes must be reported to the physician immediately (NINDS, 2011a).

In addition to careful monitoring and treatment of blood pressure and neurologic status, it is important to prevent hypoxia, maintain euthermia (normal body temperature), and control blood glucose. Other complications from stroke (eg, deep vein thrombosis, prevention of urinary tract infection) must be addressed. All patients who have had a stroke must have a swallow evaluation prior to being given anything by mouth to prevent aspiration pneumonia. During this time the focus will be to discover the cause of the initial stroke, prevent complications from treatment, and initiate therapies to prevent another stroke (Circulation, 2010).

Stroke Centers

In 2000 the Brain Attack Coalition recommended the creation of two types of stroke centers: primary and comprehensive. This is similar to the system of different levels used by trauma centers. In 2003 the Joint Commission developed stroke center certification requirements (in collaboration with the AHA and ASA) in order to foster better outcomes for stroke patients (Cronin, 2011).

Primary Stroke Centers

A primary stroke center has the resources and staff to treat most uncomplicated acute strokes and to administer thrombolytics. As of October 2009, there were more than six hundred certified primary stroke centers in the United States (Cronin, 2011).

Primary stroke centers must meet Brain Attack Coalition guidelines. Their emergency departments should be able to offer approved therapies to appropriately selected patients whether the stroke is ischemic or hemorrhagic. Requirements include the following (NINDS, 2008a):

- Improving the NINDS chain of recovery
- Agreements with EMS systems for pre-notification
- 24/7 physician-staffed emergency department
- Written care protocols
- A defined acute stroke team (should include ED staff)
- A named director of acute stroke treatment for the institution
- Necessary support:
    - Commitment and support of the medical organization
    - Neuro-imaging services (24/7)
    - Laboratory services (24/7)

  - Inpatient services appropriate to the patient's level of illness with close neurologic and cardiorespiratory monitoring (inpatient services are required only for those primary stroke centers that will provide ongoing inpatient care for patients with stroke)
- On-site neurosurgical services or pre-specified transfer agreements
- Outcome and quality improvement activities
- Continuing medical education

In addition to providing emergency department evaluation and treatment of the acute stroke patient, specialized stroke centers offer an organized approach to inpatient care aimed at preserving and restoring neurologic function and preventing future neurologic damage (NINDS, 2008a).

Comprehensive Stroke Centers

A comprehensive stroke center offers the full spectrum of state-of-the-art stroke care for patients with ischemic as well as hemorrhagic stroke. The comprehensive stroke system is made up of primary stroke centers and their referral hospitals, with more advanced services to which individual stroke patients are appropriately transferred. Pathways for patient care, patient transfer, and stroke prevention; inter-hospital communication; ongoing assessment and improvement of the quality of stroke care; and public and professional stroke education are also essential components of a comprehensive stroke system (NINDS, 2008a).

In January 2011, the AHA/ASA published new guidelines for comprehensive stroke centers. Primary stroke centers must have standard neurosurgical capabilities or the ability to transfer patients to a facility that has these capabilities. In addition to the requirements for primary stroke centers, comprehensive stroke centers must have the ability to perform neurosurgic and endovascular procedures on site and to have intensive care services for these patients (Anderson, 2011).

Rehabilitation Following Stroke

> A considerable body of evidence, mainly from countries in Western Europe, indicates that better clinical outcomes are achieved when patients with acute stroke are treated in a setting that provides coordinated, multidisciplinary stroke-related evaluation and services. Skilled staff, better organization of services, and earlier implementation of rehabilitation interventions appear to be important components.
>
> Agency for Healthcare Policy and Research (AHCPR)
>
> *Guideline for Post Stroke Rehabilitation*, 1995

Each year in the United States about two-thirds of those who suffer a stroke receive rehabilitation services of some type. The organization and delivery of these services range from minimal outpatient services to intensive inpatient care on a specialized rehabilitation unit.

In the United States, the most common location for post-stroke rehabilitation is an inpatient rehabilitation facility. This can include a hospital-based rehabilitation unit or freestanding rehabilitation hospital. Most of these facilities offer physical, occupational, and speech/language therapy as well as comprehensive nursing and medical care, family planning services, caregiver training, and social services.

Under Medicare guidelines, patients are generally eligible for rehabilitation services if there is a loss of functional or cognitive abilities following a stroke and if there is potential for improvement. If pre- and post-stroke functional status is equivalent, or if the prognosis is judged to be poor, rehabilitation services are usually not appropriate (VA/DOD, 2010).

The amount and type of rehabilitation services are (for the most part) dictated by Medicare regulations and the amount and intensity of services as well as discharge decisions can be strongly influenced by these regulations. Under these guidelines, the average stay for a post-stroke patient in an inpatient rehabilitation facility is 15 days, with about 70% of patients discharged to home. The remaining 30% either return to the acute care setting or transfer to a skilled nursing facility for continued rehabilitation services (Conroy, DeJong, and Horn, 2009).

The Rehabilitation Program

The goal of post-stroke rehabilitation is to improve functional outcomes and, if possible, allow a return to independent living, which requires not only the ability to perform basic activities of daily living, but also the more complex instrumental activities of daily living such as shopping, meal preparation, telephone use, driving, and money management.

Initially, the main focus is to prevent recurrence of stroke, manage comorbidities, and prevent complications. Mobilization and resumption of self-care tasks can begin as soon as the patient is stabilized and able to participate in these activities. If physical or cognitive deficits are severe it may not be possible for patients to return to their former home and plans must be made for continued inpatient rehabilitation.

For less severely disabled stroke patients, early supported discharge (providing interdisciplinary rehabilitation in the home instead of in a hospital) may be a viable option. Early supported discharge is a model of care that links inpatient care with community services and allows the patients to be discharged home sooner with support of the rehabilitation team. Early supported discharge should only be considered when adequate community services for rehabilitation and for caregiver support are available, and when services can be provided at the level of intensity needed (VA/DOD, 2010).

When the decision has been made that a patient will benefit from rehabilitation services, the medical team must determine the best setting for ongoing rehabilitation. Regardless of the setting, the patient should be cared for by a coordinated, multi-disciplinary team and assessments should be completed using standardized tools. There is evidence that higher intensity of physical and occupational therapy results in improved functional outcomes, particularly in patients with moderate impairment (VA/DOD, 2010).

When considering the most appropriate setting, the following points should be carefully considered:

- Severity of the patient's impairment and rehabilitation needs
- Availability of family and social support and resources
- Patient and family goals and preferences
- Availability of early supported discharge services for people with mild to moderate disability

Patients will likely remain in an inpatient setting for their rehabilitation care if they need nursing services, intensive physician care, or multiple therapeutic interventions.

A large portion of time (19%–43%) in inpatient stroke rehabilitation is spent on training for activities of daily living (ADLs) because the ability to perform these activities is often necessary to move to levels of less structured care, particularly the ability to return to community living.

Typically, ADLs are addressed with a mixture of **restorative** interventions such as motor training for the paretic upper extremity and **compensatory** interventions such as one-handed techniques, or adaptive equipment. In the United States, much less time is spent on independent (IADL) training during inpatient rehabilitation because of the short lengths of stays (VA/DOD, 2010).

Assessment of Progress

Because medical insurance companies require that a patient show steady progress toward stated goals, regular assessment is needed. During re-assessment, progress towards attainment of agreed-upon goals should be documented and discussed with the rehab team, patient, and caregivers. Goals that are met should be modified and overall progress should be discussed and documented.

If a patient's functional status deteriorates or a patient falls short of the anticipated goals, re-evaluation provides an opportunity for education, psychosocial evaluation, and re-assessment of community resources (VA/DOD, 2010).

Assessing Progress

- Patients should be regularly re-evaluated during rehabilitation.
- Attention should focus on progress toward stated goals.
- Patients who show a decline in functional status may not be eligible for continued rehabilitation.
- Considerations about the cause of a decline and its prognosis can help guide decisions about when or if further rehabilitation evaluation should occur.
- Psychosocial status and community integration needs should be re-assessed, particularly for a patient who has experienced a functional decline or reached a plateau.

Source: VA/DOD, 2010.

Discharge

Discharge planning begins immediately upon admission and should involve the patient, family, primary care provider, social services, and rehabilitation team. Prior to discharge, all necessary equipment and support services must be in place. Depending upon the discharge location, full independence may not be required or expected (eg, a patient returning to an assisted-living situation). Patients are ready for discharge from an inpatient setting when:

- There are no skilled nursing needs, or if needs are present they can be met by a caregiver or community support services.
- They no longer require regular physician care.
- There is an environment available that supports specific functional deficits.

- They are functionally independent or can be assisted by family or caregiver.
- If needed, rehabilitation services are available and accessible in the community (VA/DOD, 2010)

For most people, discharge from a formal rehabilitation program is only the beginning. The first few weeks after discharge can be difficult as the patient attempts to use newly learned skills without the support of the rehabilitation team. The full impact of the stroke may not become apparent until the patient has been home a few weeks.

Although rehabilitation services are effective in improving functional ability, gains may be lost when a patient returns home. To ensure long-term benefits, patients may benefit from continued therapy services in the home or sub-acute care setting (VA/DOD, 2010).

Caregiver Issues

A **caregiver** is someone who provides assistance to a person in need. It may be physical, financial, or emotional assistance. Caregivers can help with basic ADLs such as bathing, dressing, walking, and cooking, or more complex tasks such as medication management and home management, among other things. They may give direct care or manage care from a distance or for others. They can be a family member, a neighbor, or a medical professional.

Caregiving can be extremely taxing, both physically and emotionally, and clinicians should be aware of the potential adverse effects of caregiving on family function and the health of the caregiver. Caregivers are at increased risk for depression, increased use of health services, and self-administration of medications prescribed originally for the stroke patient (VA/DOD, 2010). Depression has been associated with physical abuse of the patient and a greater likelihood of nursing home placement. Opportunities for respites may be extremely important.

Clinicians should work with the patient and caregivers to ensure they are knowledgeable about the patient's needs. Pre-existing family habits and characteristics play an important role in a patient's successful transition to community living. Caregivers are more likely to give adequate support if they are knowledgeable about stroke and its disabilities, are not depressed, and live in an otherwise well-functioning family unit (VA/DOD, 2010).

Community support can help buffer the effects of disabilities on the patient, family, and caregivers. Educational support can be provided through printed materials, videos, computer programs, information on support group, and so on. The availability of emotional and physical support services such as homemaker home health, Meals on Wheels, devices, and equipment are also crucial to a successful outcome. A home assessment for all patients who will be discharged home with functional impairments should be encouraged (VA/DOD, 2010).

Family Education

The patient and family or caregiver should be actively engaged in the rehabilitation program. They should participate in decision making and the setting of relevant goals and realistic strategies for reaching them.

In order for patients to sustain the gains made during inpatient care and make further progress in the community, the rehabilitation team should view the patient and family as the unit of care. The rehabilitation team must have a comprehensive understanding of the needs and preferences of the family and be familiar with the post-discharge environment. The team must understand the patient and family history, expectations, coping styles, resources, emotional support system, and family dynamics (VA/DOD, 2010).

Stroke patients and family caregivers should receive a psychosocial assessment with psychosocial intervention and referrals as needed. The psychosocial assessment of both the patient with stroke and the primary family caregiver should address the following areas:

- Pre-stroke functioning of the patient and the primary caregiver
- Capabilities and abilities of the primary caregiver
- Caregiver's understanding of the patient's needs
- Family dynamics and relationships
- Availability and anticipated involvement of other family members
- Spiritual and cultural preferences
- Leisure time and preferred activities
- Understanding of the condition and expectations for recovery
- Stroke-related outcomes and resources for follow-up care (VA/DOD, 2010)

Motor Impairment and Recovery

Rehabilitation can help individuals cope with disability and relearn skills that have been lost. This relearning is made possible by the brain's **plasticity**—its ability to reorganize itself by forming new connections between neurons. Plasticity soars in the developing brain and wanes as the brain matures, but even the aged brain appears to retain some plasticity and thus some capacity to repair itself after a stroke (NINDS, 2009c).

Following a stroke, changes in movement patterns can affect functional independence, safety, and hygiene. Impaired strength, endurance, or coordination and neuromuscular recruitment abnormalities alter gait speed, increase energy expenditure, and decrease mobility. Impaired mobility can significantly reduce functional independence and increase the burden of care (VA/DOD, 2010).

Several theoretical models of motor control serve as the foundation for treatment approaches following a stroke. Traditional approaches such as neurodevelopmental treatment (also known as NDT or Bobath), Brunnstrom, proprioceptive neuromuscular facilitation (PNF), are based on reflex or hierarchical models of motor control.

Contemporary models focus on the interaction of higher and lower centers of control and view the nervous system as one system among many that influence motor behavior. **Task-oriented** approaches focus on the interaction of multiple systems and assume that motor control and behavior are organized around goal-directed and functional activities, rather than on muscles or movement patterns (VA/DOD, 2010).

All of these approaches have been shown to be useful; however, no single approach has proven more useful than the others. Rehabilitation therapists should incorporate a combination of these techniques throughout the recovery process (VA/DOD, 2010).

Strengthening and Exercise

Weakness of the upper and lower extremities is a common impairment after a stroke because of damage to upper motor neurons in the damaged part of the brain. This leads to weakness and atrophy of muscles. Weakness in the lower extremities affects strength and balance and causes difficulty with mobility and walking. Weakness in the upper extremities affects the use of the hand and arm and affects the ability to do activities of daily living. Weakness is usually not confined to the extremities but also affects the muscles of the trunk, neck, face, and eyes.

Strengthening exercises do not repair upper motor neuron damage but work at the level of skeletal muscle using remaining innervation. Strengthening exercises may include weight lifting, weight bearing, and isokinetic exercise. Exercise may also include tasks that incorporate sequencing and motor control (VA/DOD, 2010).

Range of Motion

Weakness and paresis following stroke commonly contributes to decreased active and passive range of motion (ROM) of the involved joints. Profound hemiparesis can lead to joint contractures, which severely impair mobility and may lead to pressure ulcers. Passive (PROM) and active (AROM) exercise are used to reduce the risk of secondary musculoskeletal impairment from decreased joint ROM.

Stroke can also worsen pre-existing conditions such as osteoarthritis, or can lead to osteoarthritis by producing muscle imbalances that result in inappropriate forces across joints. Maladaptive activity patterns or postures can develop in upper or lower extremities after stroke as an individual attempts to regain function. For example, hyperextension of the knee is common maladaptive pattern that allows for weight-bearing on a weakened or paretic lower extremity, but it can cause osteoarthritis and joint pain if this pattern continues over time.

Balance and Mobility

Balance is the ability to maintain your center of gravity over your base of support and it is often severely affected following a stroke. Sensory changes alter the ability to respond normally to losses of balance and affect the ability to adapt quickly to environmental changes. The visual and vestibular systems may be affected, causing visual disorientation and vertigo. The goal of a balance assessment is to find the cause of the balance impairment and design a program to improve balance, decrease the risk of falls, and to determine the need for equipment, home modification, and assistive devices.

To assess balance and mobility effectively, an easy-to-use assessment tool is necessary. One such tool, the Berg Balance Scale, is a reliable clinical tool for the assessment of functional mobility and gait. The Berg Balance Scale consists of 14 tasks scored from 0 to 56, which assess a variety of functional, balance, and gait activities. Each task is scored on a 1–4 scale; a score of 0 indicates an inability to perform the task while a score of 4 means the patient is independent with that task. The Berg Balance Scale has excellent internal consistency and good test/retest reliability and requires little specialized training. It can be performed with minimal equipment in a small space and can be used in any clinical setting.

Those with impaired mobility and balance should be referred to a mobility-training program where specific and individualized goals can be established. Improving balance and mobility requires adequate practice, a progression in the difficulty of the tasks, sufficient intensity, frequency, and duration of practice sessions, and functional task-specific activities. Combining specific interventions, repetition, and practice as well as engaging the patients will lead to better outcome (VA/DOD, 2010).

Although the existing evidence is limited by poor trial designs, some studies have shown that improvement of mobility and gait improves ADLs, participation, and quality of life. A meta-analysis of randomized controlled trials conducted by Langhorne in 2009 demonstrated that "cardiovascular fitness training, incorporating a mixture of cardiorespiratory and strength training, high-intensity physiotherapy, and repetitive task training showed a consistent pattern of improvement in walking speed" (VA/DOD, 2010).

Balance and Sensory Organization

Of the many systems and organs that provide sensory input to the central nervous system, the somatosensory, visual, and vestibular systems are the ones most directly involved with balance. Understanding how these sensory systems malfunction helps explain the profound difficulties patients have with balance and mobility following a stroke.

The **somatosensory system** has perhaps the strongest influence on balance. Somatosensory input from touch, heat, cold, pressure, joint position, muscle stretch, and pain, among others, is continuously fed to the brain. There the sensory information is automatically processed and used to make the many quick adjustments that keep us balanced. Somatosensory input provides critical feedback about our position in space, body sway, and changes in terrain. This allows our muscles to make constant, automatic adjustments to maintain balance and avoid falls.

The **visual system** is another key contributor to balance. It allows us to determine the movement of objects in our environment; it tells us where we are in relation to parts of our own body and to other objects. Our visual system does this using both central and peripheral vision. Central vision is processed mostly through the macula—the part of the retina that allows us to see clearly. Peripheral vision provides information to the brain about general spatial orientation and is more important for postural control and balance than central vision. Vision works in conjunction with the vestibular system, comparing information about velocity and rotation with actual visual information (Shumway-Cook and Woollacott, 2007).

Our eyes don't always provide accurate information to our brains. We've all had the experience of being stopped at a stoplight and having the car next to us start to move—we think we are moving and slam our foot on the brake. As soon as our foot touches the brake we instantly know that we weren't moving and even feel a little foolish. In this example, the brain gives preference to visual input for a split second—momentarily overriding somtosensory input—and introduces a sensory conflict. The sensory conflict is quickly resolved, thanks to the somatosensory and vestibular systems. The touch of our foot on the brake and position receptors in our back and legs quickly tell us that we were, in fact, sitting still. At the same time the hair cells in the vestibular system let us know that there is no forward motion and we relax.

The **vestibular system** is responsible for processing information about movement in relation to gravity—specifically, rotation, acceleration/deceleration, and head stabilization during gait. The vestibular system works with the visual and somatosensory systems to help us maintain our orientation in space; it works with the visual system to stabilize the eyes and maintain posture during walking (vestibulo-ocular reflex). Vestibular disorders cause a feeling of dizziness and unsteadiness and affect the ability of the nervous system to mediate inter-sensory conflicts such as in the example given above.

The vestibular system declines with age, and there may be as much as a 40% loss of vestibular nerve and hair cells by age 70. Vestibular decline has a profound effect on balance and postural control. This is because it is used as a reference system by the visual and somatosensory systems when those systems are in conflict. Vestibular impairment can lead to problems with gaze stabilization, blurred vision, and vertigo (Shumway-Cook and Woollacott, 2007).

The nervous system is very good at compensating for disabilities and declines. Once an injury has occurred, the nervous system immediately goes to work attempting to compensate for neurologic changes, weakness, and loss of function. But the brain doesn't always choose the best (or even a good) compensation; it chooses the fastest and most efficient in an attempt to continue functioning. One of the immediate goals of therapy is to help the nervous system develop strategies and compensations that minimize musculoskeletal damage and maximize function.

Sensory loss may lead to inflexible or improper sensory weighting. A person may depend on one particular sense for postural control even if that sense leads to further instability (Shumway-Cook and Woollacott, 2007). You may notice a person walking with head down, carefully watching every step. In this case, vision has become the dominant sense being used for balance. Retraining would involve improving the use of somatosensory and vestibular input to reduce dependence on visual input.

Locomotor Training

[The following section is from NINDS, 2011d. "Large NIH Funded Rehabilitation Study Looks at Getting Stroke Patients Back on Their Feet."]

> More than 4 million stroke survivors experience difficulty walking. Rigorously comparing available physical therapy treatments is essential to determine which is best. The Locomotor Experience Applied Post-Stroke (LEAPS) study shows that the more expensive, high-tech therapy is not superior to intensive home strength and balance training, but both were better than lower intensity physical therapy.
>
> Walter Koroshetz, MD, Deputy Director
>
> National Institute of Neurological Disorders and Stroke

The Locomotor Experience Applied Post-Stroke (LEAPS) trial set out to compare the effectiveness of the body-weight supported treadmill training with walking practice started at two different stages—two months post stroke (early locomotor training) and six months post stroke (late locomotor training). The locomotor training was also compared against a home exercise program managed by a physical therapist, which was aimed at enhancing flexibility, range of motion, strength, and balance as a way to improve their walking. The primary measure was improvement in walking at 1 year after the stroke.

In the LEAPS trial—the largest stroke rehabilitation study ever conducted in the United States—stroke patients who had physical therapy at home improved their ability to walk just as well as those who were treated in a training program that requires the use of a body-weight supported treadmill device followed by walking practice. The study, funded by the NIH, also found that patients continued to improve up to 1 year after stroke—defying conventional wisdom that recovery occurs early and tops out at 6 months. In fact, even patients who started rehabilitation as late as 6 months after stroke were able to improve their walking.

More than four hundred patients with either severe or moderate walking impairments were randomly assigned into the three study groups and participated in thirty-six 90-minute sessions over 12 to 16 weeks. The average age of the patients was 62 years. Fifty-four percent were men, 46% were women, and 22% were black. The trial took place at six inpatient rehabilitation centers in Florida and California.

The walking program involved having a patient walk on a treadmill in a harness that provides partial body weight support. This form of rehabilitation, which is known as **locomotor training**, has become increasingly popular. After the patients complete their training on the treadmill, they practice walking.

Locomotor Training

An example of a body-weight supported treadmill.
Source: NIH.

Previous studies suggested that these devices, also called **commercial lifts** or **robot-assisted treadmill steppers**, are an effective intervention in helping stroke patients walk. But this walking program had not been tested on a large scale or examined in terms of the most appropriate timing for therapy.

The investigators had hypothesized that the body-weight supported treadmill and walking program, especially early locomotor training, would be superior to a home exercise program. However, they found that all groups did equally well, achieving similar gains in walking speed, motor recovery, balance, social participation, and quality of life.

The patients' measure of improvement was based on how well they were able to walk independently by the end of the study period. For example, severely impaired stroke patients were considered improved when they were able to walk around inside the house, whereas the patients who were already mobile at home were considered improved when they could progress to walking independently in the community.

At the end of 1 year, 52% of study participants had made significant improvements in their ability to walk. The timing of the locomotor training program did not seem to matter. At 1 year no differences were found in the proportion of patients who improved walking with the early or late treadmill training program, nor did the severity of their stroke affect their ability to make progress by the end of the year. All groups achieved similar gains in the speed and distance of their walking, physical mobility, motor recovery, and social participation, resulting in an improved quality of life.

All study participants started out with **usual care**, which involved a variable number of physical therapy sessions of about 1 hour each. Following this they were assigned to one of the study groups. The study found that earlier was better when it comes to rehabilitation therapy. The patients who were not assigned to a study group until 6 months after their stroke recovered only about half as much as the participants who received one of the two therapy programs at 2 months. This finding suggests that either the treadmill training program or the at-home sessions are effective forms of physical therapy, and both are superior to usual care.

The patients in the body-weight supported treadmill and walking program group that started at 6 months made significant improvements in walking speed, despite the widely held assumptions and reports that most functional improvements after stroke are complete by 6 months. The researchers said this suggests that recovery beyond 6 months can be influenced by further therapy.

Individuals in the locomotor training groups were likely to feel faint and dizzy during the exercise, and those who received early locomotor training experienced more falls. Fifty-seven percent of participants experienced one fall, 34% had multiple falls, and 6% had a fall resulting in injury.

The at-home group was the most likely to stick with the program; only 3% dropped out of this part of the study, compared to 13% of the locomotor training groups. The physical therapy training programs in this study were progressive, intensive, and repetitive, and were highly effective in improving functional status and levels of walking ability, and quality of life at one year post stroke. The home exercise programs require less expensive equipment, less training for the therapists, and fewer clinical staff members.

"We were pleased to see that stroke patients who had a home physical therapy exercise program improved just as well as those who did the locomotor training," said Pamela W. Duncan, principal investigator of LEAPS and professor at Duke University School of Medicine. "The home physical therapy program is more convenient and pragmatic. Usual care should incorporate more intensive exercise programs that are easily accessible to patients to improve walking, function, and quality of life."

Adaptive Equipment

Following a stroke, many patients require assistive devices, adaptive equipment, mobility aids, wheelchairs, and orthoses to maximize independent functioning. Many types of adaptive devices and durable medical equipment are available. Type and level of functional deficit, the amount of learned adaptation, and the structural characteristics of the living environment help determine the need for a particular item. Walking devices are helpful for patients with mild gait impairments. These devices increase the base of support around a patient's center of gravity and reduce the effort needed to walk. Walking aids include (but are not limited to):

- Single-point canes
- Tripod or quad canes
- Four-point walkers
- Wheeled walkers

Wheelchairs should be provided for patients with severe motor weakness or for those who easily fatigue. Wheelchair designs vary greatly and a wheelchair prescription should be specific to the patient's needs and environment and to patient and caregiver preferences.

A vast array of adaptive devices are available, including devices to make eating, bathing, grooming, and dressing easier for patients with functional limitations. These devices should serve as a supplement and should not be expected to take the place of the patient mastering the task in question. Additionally, many patients may need to use adaptive devices early during the rehabilitation following a stroke, but will not require long-term use. This should be taken into account when considering a device. Adaptive devices include:

- Eating utensils with built-up handles
- Rocker knives
- Plate guards
- Non-skid place mats
- Long-handled sponges for bathing
- Hand-held showers
- Tub and shower chairs
- Grab bars for bathrooms
- Elevated toilet seats

Lower extremity orthoses, such as ankle-foot orthoses and knee-ankle-foot orthoses, may be required if the patient has persistent weakness and instability at the ankle or knee joint following a stroke. Proper timing for using an orthosis can facilitate gait training and should be considered early on in the rehabilitation process to permit gait training as early as possible.

An orthosis should not be used as a substitute for functional exercise directed at regaining muscle strength and control, particularly if the prognosis for motor recovery is good. Prefabricated orthoses can be used in the early stages of gait training, but a custom-fit device should be provided if it is determined that the patient may require long-term use of the orthosis (VA/DOD, 2010).

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, patients are 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 acute hospital to a skilled nursing facility or center 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 educating themselves about the effects of stroke and becoming familiar with available services as well as the astounding advances taking place in the emergency, acute, and long-term treatment of people who have had a stroke.

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(Post test begins on next page)

Post Test

Use the Answer Sheet following the test to record your answers. There are 38 questions.

1. Stroke in the United States:
   a. Is the first-leading cause of death.
   b. Causes more serious long-term disabilities than any other disease.
   c. Is not a significant cause of long-term disability.
   d. Results in death within a month for about 50% of people.

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 involving the major brain arteries.
   d. Occur when a major vessel of the heart is occluded by a blood clot.

3. Transient ischemic attacks (TIAs):
   a. Are small strokes in which the symptoms resolve within 2 to 3 days.
   b. Are usually caused by blood leaking into the brain.
   c. Usually have a gradual onset of symptoms similar to stroke followed by a gradual decrease in symptoms.
   d. Are often caused by embolism, which most commonly originate from the heart or neck arteries.

4. Hemorrhagic stroke:
   a. Has the same symptoms as an ischemic stroke.
   b. May include nausea, vomiting, headache, and altered level of consciousness.
   c. Is the result of blockage of one or more of the major arteries in the brain.
   d. Rarely causes severe neurologic after-effects.

5. Risk factors for stroke:
   a. Have little in common with heart disease risk factors.
   b. Includes moderate alcohol consumption.
   c. Includes bradycardia, which is the most important risk factor.
   d. Includes hypertension, which is one of the most important risk factors.

6. The cerebrum:
   a. Is divided into four hemispheres.
   b. Is another name for the cerebellum.
   c. Is not very interconnected with other parts of the nervous system.
   d. Has two hemispheres, each divided into four lobes.

7. The cerebral cortex:
   a. Controls the right hemisphere of the brain exclusively.
   b. Is a thin layer of nerve cells that smoothes out the convolutions of the brain.
   c. Is a thin layer of nerve cells that covers the surface of both hemispheres.
   d. Controls the left hemisphere of the brain exclusively.

8. The respiratory and cardiovascular centers of the brain comprise the:
   a. Brainstem reticular formation.
   b. Medulla oblongata.
   c. Cerebellum.
   d. Thalamus.

9. The cerebellum, or "little brain":
   a. Makes up nearly 40% 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.

10. Blood flows from the heart to the brain via two large arterial systems:
    a. The middle cerebral and the anterior cerebral arteries.
    b. The ophthalmic artery and the middle cerebral artery.
    c. The carotid and the vertebrobasilar systems.
    d. Broca's and Wernicke's systems.

11. The middle cerebral artery (MCA):
    a. Is not generally vulnerable to emboli.
    b. Is the smallest of the arteries supplying blood to the brain.
    c. Supplies blood to the right hemisphere of the brain.
    d. Is the most common artery involved in stroke.

12. Broca's aphasia is signaled by short, choppy sentences, and Wernike's aphasia by long, meandering sentences in patients who have stroke-induced injury to the brain's language control centers.
    a. True
    b. False

13. A stroke affecting the basal ganglia:
    a. Usually causes motor control problems rather than hemiparesis.
    b. Can cause Parkinson's disease.
    c. Usually causes contralateral hemiparesis.
    d. Will cause apraxia and aphasia.

14. Damage to the cerebellum may result in all of the following **except**:
    a. Ataxia, intention tremor, and hypotonia.
    b. Apraxia and resting tremor.
    c. Tardive dyskinesia.
    d. Speech and language dysfunction.

15. Strokes in women:
    a. Occur more often than in men largely because women have smaller carotid arteries.
    b. May include nontraditional symptoms such as altered level of consciousness, nausea, and hiccups.
    c. May have a better outcome than male patients in motor, cognitive, and functional recovery.
    d. Are easier to recognize because of a higher occurrence of traditional symptoms.

16. Those who practice a healthy lifestyle have an 80% lower risk of a first stroke compared with those who do not.
    a. True
    b. False

17. Carotid endarterectomy:
    a. Is of great benefit in patients with acute hemorrhagic stroke.
    b. Should not be used in conjunction with aspirin therapy.
    c. Is most often performed in women with severe carotid stenosis.
    d. Reduces the risk of TIA and stroke in people with moderate to severe narrowing of the carotid arteries.

18. The "suddens" refers to:
    a. A sudden increase in blood pressure, weight, and diabetes leading to increases in heart attack and stroke.
    b. The need for quick assessment of symptoms by EMTs to ensure prompt treatment of stroke victims.
    c. Sudden weakness or numbness, confusion, trouble seeing, trouble walking, and severe headache.
    d. The lack of awareness of stroke symptoms by the general public.

19. Stroke Heroes Act FAST:
    a. Identifies almost 98% of stroke victims when used by untrained lay people.
    b. Stands for facial weakness, altered level of consciousness, slurred speech, and time to call 911.
    c. Stands for facial weakness, arm drift, slurred speech and time to call 911.
    d. Is too difficult for lay people to remember and is not effective in identifying stroke victims.

20. Recommendations made by the Minnesota Stroke Partnership advise that:
    a. Prehospital personnel should transport any suspected stroke victim to the nearest ED.
    b. EMS personnel should not attempt to identify stroke victims.
    c. The Glasgow Coma Scale should be used as the standard for assessing suspected stroke patients.
    d. EMS personnel should treat stroke as an emergent event and transport suspected patients to the nearest stroke center.

21. The goal of reaching an emergency department within 3 hours of the onset of stroke symptoms is:
    a. To differentiate a true stroke from a TIA.
    b. Important because x-rays do not show the difference between hemorrhagic and ischemic stroke after 3 hours.
    c. Only important if the patient has symptoms of a hemorrhagic stroke so they can receive thrombolytic drugs.
    d. Critical because of the use of time-dependent medications and treatments for acute stroke.

22. When a suspected stroke patient arrives in an emergency department, the goal should be:
    a. An initial evaluation within 1 hour.
    b. A CT scan initiated within 10 minutes.
    c. Interpretation of the CT scan and labs within 15 minutes.
    d. Door-to-drug (needle) time within 60 minutes of arrival.

23. The most important function of an imaging study for acute stroke is to: A
    a. Differentiate between ischemic and hemorrhagic stroke.
    b. Determine the area of the brain damaged by stroke.
    c. Determine the amount of arterial blockage.
    d. Determine if an individual would benefit from carotid endarterectomy.

24. Thrombolytic agents such as rtPA:
    a. Are effective for the treatment of hemorrhagic strokes.
    b. Can halt an ischemic stroke by dissolving the blood clot that is blocking blood flow to the brain.
    c. Can be used for up to 24 hours following acute onset.
    d. Has been shown to be dangerous and ineffective with ischemic strokes.

25. Interventional radiology:
    a. Must be used within 1 hour of acute symptom onset.
    b. Increases the chance of intracranial bleeding from rtPA.
    a. Uses an x-ray guided catheter to deliver rtPA directly into the clot.
    d. Is an effective treatment for intracranial hemorrhage.

26. The use of aspirin:
    a. Should be considered as a substitute for rtPA.
    b. Should be given at the same time as rtPA.
    c. Is a standard treatment for patients at risk for stroke.
    d. Should be combined with clopidogrel for the treatment of acute ischemic stroke.

27. All strokes are treated as ischemic until a CAT scan confirms otherwise.
    a. True
    b. False

28. A comprehensive stroke center differs from other types of stroke centers in that:
    a. It has the staff to treat most uncomplicated acute strokes and to administer thrombolytics.
    b. It focuses on stabilization of the stroke patient.
    c. It has a type 2 trauma center on site.
    d. It is able to perform neurosurgical and endovascular procedures on site.

29. The goal of post-stroke rehabilitation is to:
    a. Improve functional outcomes.
    b. Help patients regain 100% of their pre-stroke capabilities.
    c. Provide early supported discharge for all stroke patients.
    d. Keep patients in a rehabilitation setting for as long as possible.

30. A patient should be discharged from inpatient therapy when:
    a. They no longer have any deficits.
    b. They are able to drive short distances.
    c. Their family can take over nursing care duties.
    d. They are functionally independent.

31. Caregivers:
    a. Are usually well-trained and able to help a family member who has had a stroke.
    b. Are at increased risk for depression.
    c. Should be kept out of the rehabilitation process.
    d. Adapt easily to changes in a loved one following a stroke.

32. Balance and mobility:
    a. Are usually not affected following a stroke.
    b. Improve quickly with the use of an assistive device.
    c. Are often severely affected following a stroke.
    d. Usually do not improve after someone has had a stroke.

33. The sensory organs that contribute most to balance are:
    a. The somatosensory, visual, and vestibular systems.
    b. The cochlea and retina.
    c. Sensory organs for heat, cold, and light touch.
    d. The primary visual cortex.

34. The LEAPS trial found all of the following to be true **except**:
    a. Those with home physical therapy improved just as much as those who did locomotor training with a body-weight supported treadmill.
    b. Earlier is better when it comes to rehabilitation therapy.
    c. Patients continued to improve up to 1 year after stroke.
    d. Usual care (a variable number of physical therapy sessions of about 1 hour each) was just as good as an intensive home care program.

(Answer sheet follows on next page)

Answer Sheet

Stroke: Acute Care and Rehabilitation

Name (Please print your name): _____
Date: _____

Passing score is 80%

1. _____
2. _____
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34. _____

(Course evaluation follows on next page)

Course Evaluation

Please use this scale for your course evaluation. Items with asterisks (*) are required.

5 = Strongly agree

4 = Agree

3 = Neutral

2 = Disagree

1 = Strongly disagree

*1. Upon completion of the course, I was able to:

a. Distinguish among the main classifications of stroke.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

b. Explain the major risk factors for stroke and its pathophysiology.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

c. Identify the main structures of the brain and the common deficits caused by stroke to those structures.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

d. Contrast gender differences in the presentation of acute stroke.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

e. Summarize the efforts of public health organizations to educate the public about stroke symptoms and emergency response.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

f. Describe recent advances aimed at improving care of stroke victims in prehospital settings.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

g. Review emergency department care of persons with acute stroke.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

h. Discuss the role of rehabilitation therapy in the short- and long-term treatment of stroke.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*2. The course was written in a way that facilitated my learning.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*3. This course was free from commercial bias.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*4. The course met my continuing education needs.
❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*5. The material presented was supported by evidence.

❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*6. The author avoided the use of anecdotal information as the main source of material.

❑ 5 ❑ 4 ❑ 3 ❑ 2 ❑ 1

*7. The course was free of product promotion.

❑ Yes ❑ No**

** If you answered no, please answer #8.

8. Was product promotion the sole purpose of the presentation?

❑ Yes ❑ No**

*9. It took me 60 minutes per contact hour to complete the course, test, and evaluation.

❑ Yes ❑ No**

** If your answer was no, how long did it take?

_____

10. 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): _____

11. I heard about ATrain Education from:

❑ Search engine ❑ Advertisement

❑ Government or Board website ❑ Returning customer

❑ Friend ❑ Publication (Magazine, etc.)

❑ Other _____

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

❑ Yes ❑ No

13. Comments or suggestions (optional): _____

_____

_____

(Registration on next page)

Registration Information

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

* Name: _____

* Address: _____

* City: _____ State: _____ Zip: _____

* Phone: _____

* Professional Designation: _____

* License Number and State: _____

Please email my certificate: ❑ Yes ❑ No
Email (required if you want your certificate sent by email): _____
(If you request an email certificate we will **not** send a copy of the 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**.
5 contact hours: $29

Credit card information:
Name _____

Address (if different from above): _____

City: _____State: _____ Zip: _____

Card type: ❑ Visa ❑ MC ❑ American Express ❑ Discover

Card number _____ CVS # _____

Expiration date _____

Test Completion and Mailing Instructions

1. Complete all forms:
   - ❑ Answer Sheet
   - ❑ Evaluation Learning Activity
   - ❑ Registration Form (this page)
2. If you are **paying by check**, prepare a check for $29 made out to ATrain Education, Inc.
3. Mail the completed forms and your payment to:

   ATrain Education, Inc
   5171 Ridgewood Rd
   Willits, CA 95490

When 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 Sharon@ATrainCEU.com. And thanks for taking the ATrain!