LEARNING OUTCOME: Upon completion of this continuing education course, you will demonstrate an understanding of the anatomical alterations, pathophysiology, diagnosis, evaluation, and treatment options, emphasizing acute care and initial rehabilitation, for patients who have experienced a stroke.

LEARNING OBJECTIVES

- Review stroke epidemiology.
- Identify risk factors for stroke.
- Discuss the major classifications of stroke.
- Describe the structural anatomy of the normal brain and the alterations in function resulting from stroke.
- Outline the pathophysiology and etiology of stroke.
- Describe the components of prehospital and emergency department evaluation of stroke.
- Discuss the guidelines for early treatment and management of patients with acute stroke.
- Identify the complications and associated interventions that may occur during the ICU care of acute stroke patients.
- Identify assessment, interventions, and goals of physical, occupational, and speech therapists in the acute stages of stroke management/rehabilitation.
- List actions to be taken in the prevention of secondary stroke.

A stroke—also called a **cerebrovascular accident (CVA)** or a **brain attack**—is a reduction or an interruption of the flow of blood through an artery to one or more areas of the brain within the territory supplied by that artery. The end result is varying degrees of neurological and/or cognitive malfunction lasting longer than 24 hours. A very severe stroke can cause sudden death.
Stroke is a medical emergency, and for persons experiencing a stroke, the difference between recovery and disability or death is measured in hours. For healthcare professionals it is imperative that an understanding of stroke and the ways to take action become part of day-to-day practice. Providers are responsible for improving their skills along the continuum of care from prehospital/acute stroke to post-stroke education. In addition, educating patients about stroke prevention and recognition of stroke should be part of every provider’s practice.

**EPIDEMIOLOGY**

- An estimated 6.6 million Americans aged 20 years or older have had a stroke, and the overall prevalence is estimated to be 2.6%.
- The American Heart Association projects that by 2030, an additional 3.4 million people aged 18 years or older will have had a stroke.
- Each year over 795,000 Americans experience a new or recurrent stroke. An estimated 610,000 of these are first events and 185,000 are recurrent.
- On average, someone in the United States has a stroke every 40 seconds and someone dies of a stroke approximately every 4 minutes.
- In 2013, stroke was the cause of one of every 20 deaths in the United States. Stroke is the fifth-leading cause of death in the United States and the leading cause of serious long-term disability in America. (Mozaffarian et al., 2015; AHA/ASA, 2015b)

**By Age**

The chance of having a stroke approximately doubles for each decade of life after 55 years. Although the prevalence of stroke is higher among older adults, approximately 10% of all strokes occur in people 18 to 50 years of age. Stroke may also occur in infants and children and can even occur before birth (AHA/ASA, 2015c).

**By Gender**

Each year, women have more strokes than men, and a stroke kills more women than men. About 60% of stroke deaths occur in females and 40% in males. The use of birth control pills, pregnancy, history of preeclampsia/eclampsia or gestational diabetes, smoking, and post-menopausal hormone therapy are believed to account for this difference (AHA/ASA, 2016).
By Race

The risk of having a first stroke is almost twice as high for blacks than for whites, and blacks are more likely to die following a stroke than are whites, partially due to the higher prevalence of high blood pressure, diabetes, and obesity among this population (AHA, 2016).

The incidence of stroke among Hispanics lies between that of whites and blacks. Native Americans, Alaska Natives, and blacks are more likely to have a stroke than are other groups (CDC, 2015).

A higher incidence of intracerebral hemorrhage has been noted in Chinese, Japanese, and other Asian populations, possibly due to environmental factors (e.g., a diet rich in fish oils) and/or genetic factors (Liebeskind, 2016).

By Geographic Location

The southeastern part of the United States has been nicknamed the “stroke belt,” which includes the following eleven states:

- Alabama
- Arkansas
- Georgia
- Indiana
- Kentucky
- Mississippi
- Louisiana
- North Carolina
- South Carolina
- Tennessee
- Virginia

This part of the country has an 18% higher stroke rate than the national average, and one particular area along the eastern coastline, known as the “belt buckle,” has stroke mortality rates twice as high as the national average. This is believed to be the result of more limited access to healthcare as well as lower economic status affecting the quality and frequency of healthcare visits. In addition, ethnic-specific risks and unhealthy lifestyle traditions and patterns are believed to be contributors (Jensen, 2015).
Effects of Stroke

Worldwide, stroke is the second-leading cause of death and the leading cause of adult disability (AHA/ASA, 2015a; WHO, 2014).

Receiving the diagnosis of stroke is frightening. A stroke can have profound effects on the body as well as the mind and emotions. Effects on the body may include weakness or paralysis of the extremities, swelling of the arms or legs, stiff or painful joints, muscle tingling and spasms, or the loss of ability to perceive heat or cold. A stroke can leave a person with fatigue that makes everyday tasks difficult to accomplish.

Strokes can seriously affect how the brain processes information, depending on where the damage has occurred. Memory, ability to learn, and general awareness of surroundings can be affected, as well as the ability to communicate through speech and writing. Reading ability and concentration may also be impaired. Sleep disturbances may result in increased impairment of memory and perception.
Emotionally, a stroke can cause feelings of fear, anxiety, or depression and can result in damage to areas of the brain responsible for regulating emotions, leading to emotional lability and personality or character changes. Finally, the loss of independence that results from all of these can be the most devastating consequence of having a stroke.

Economically, in 2015 strokes cost the nation $34 billion, including the cost of healthcare services, medications, and lost productivity (CDC, 2015).

RISK FACTORS

Some risk factors are nonmodifiable:

- Low birth weight is reported to be associated with increased hemorrhagic stroke risk in adult life.
- Male gender increases the risk for ischemic stroke.
- Early menopause increases the risk for subarachnoid hemorrhage in women.
- Stroke increases markedly with age in both men and women for both ischemic and hemorrhagic stroke.
- People of African origin have a higher risk than whites.
- Genetics may play a role in the increased susceptibility of stroke (e.g., sickle cell disease).

Other risk factors are modifiable:

- Hypertension is the most important treatable risk factor for stroke.
- Atrial fibrillation can cause emboli to travel to the brain.
- Coagulation disorders have been implicated in ischemic stroke.
- Diabetes mellitus causes hyperglycemia, which in turn leads to increased fatty deposits or clots on blood vessel walls.
- Lifestyle factors impact the risk:
  - Smoking (doubles the risk for ischemic stroke)
  - Excessive alcohol consumption (however, individuals who do not use any alcohol may have a slightly increased risk as well)
  - Drug abuse
  - Obesity
  - Lack of physical activity
  - Psychological stress
Lower socioeconomic status
- Poor or inadequate nutrition

(Lindgren, 2014)

Scientists at the National Institute of Neurological Disorders and Stroke (2016a) estimate that Americans should be able to prevent 80% of all strokes. For instance, improved nutrition—including a high-vegetable, low-fat, high-fiber diet—and regular aerobic exercise have been shown to reduce risk.

CLASSIFICATIONS OF STROKE

There are two major categories of stroke, which are diametrically opposite conditions. One is characterized by an insufficient supply of blood to a part of the brain (ischemic) and the other by an excessive amount of blood within the closed cranial cavity (hemorrhagic).

Ischemic Stroke

An ischemic stroke occurs when a clot, either of local or distant origin, blocks a cerebral artery or causes oxygen deprivation with subsequent tissue damage. In the United States, ischemic stroke accounts for 87% of all stroke cases (AHA/ASA, 2014a).
Ischemic strokes may occur in two ways:

- **Thrombotic stroke**: Cerebral thromboses are clots that form in the cerebral arterial tree. Blood clots usually form in arteries that are damaged by plaque. There are two types of thrombotic stroke: large vessel thrombosis and small artery thrombosis (lacunar infarction).

- **Embolic stroke**: Cerebral emboli occur due to clots or other debris (such as pieces of plaque) that arise from outside the cerebral arterial tree—usually from the heart, pulmonary trunk, or pulmonary arteries—and travel through the arterial system until they become lodged within smaller arteries.

Ischemic strokes typically give rise to specific (focal) and often painless neurological symptoms. Onset is abrupt and may progressively evolve over 24 to 48 hours.

A **transient ischemic attack** (TIA) is a type of ischemic stroke—sometimes referred to as a “warning stroke” or a “mini stroke”—that results in no lasting damage (CDC, 2013). Although it is due to ischemia (most often the result of blood clots), TIA is different from the major types of stroke because blood flow is blocked for only a short period of time.

Neurologic dysfunction resulting from a TIA typically lasts less than an hour and results from focal cerebral, spinal cord, or retinal ischemia. It is not associated with acute tissue infarction (Nanda, 2015).

The incidence of stroke following a TIA is as high as 11% over the next seven days and 24% to 29% over the following years (Nanda, 2015). More than one third of persons who experience a TIA end up having a major stroke within one year if they do not receive treatment, and 10% to 15% will have a major stroke within three months of a TIA (CDC, 2013).

**Hemorrhagic Stroke**

Bleeding caused by a blood vessel in the brain that has leaked or ruptured is called a hemorrhagic stroke. Hemorrhagic stroke often causes tissue damage due to pressure-related changes.

Most commonly, intracerebral hemorrhages are caused by rupture of vessels due to long-term atherosclerotic damage and arterial hypertension, resulting in bleeding into the brain (intracranial hemorrhage) or the space surrounding the brain (subarachnoid hemorrhage). Such ruptures may occur due to:

- Weakened vascular walls (cerebral aneurysms)
- Congenital arteriovenous malformations (AVMs), which are dilated, often tangled, blood vessels where the arterial blood flows directly into the venous system bypassing the usual capillary bed. Over time, local damage to the tissue occurs due to compression, insufficient blood flow, irritation, or micro-hemorrhages.
Ten percent of all strokes are intracranial hemorrhages, and 3% are subarachnoid hemorrhages (Mozaffarian, et al., 2015).

A typical location of a cerebral aneurysm in the arteries that supply blood to the brain. (Source: NIH, 2011a.)

ANATOMY AND PHYSIOLOGY UNDERLYING ACUTE STROKE

Before discussing the assessment, treatment, and care of acute strokes, it is important to review the anatomy and physiology underlying the disease.

Normal Structural Anatomy of the Major Cerebral Arteries

The brain comprises 2% of the body’s mass, but it receives 17% of the heart’s output and consumes 20% of the body’s oxygen supply. The blood supply to the brain is delivered by anterior and posterior circulation systems.

The anterior circulation of the brain is formed by cerebral blood vessels that branch off the internal carotid arteries, while the posterior circulation of the brain is formed by those cerebral blood vessels that branch off the vertebral arteries. The anterior and posterior circulations communicate through a circular anastomosis of arteries called the Circle of Willis, located at the base of the brain. From this circle, other arteries arise and travel to all areas of the brain.
ANTERIOR CIRCULATION

The anterior circulation of the brain is formed by two large arteries—the right and left internal carotid arteries—that ascend from the chest in the anterior portion of the neck. The carotid arteries supply blood to about 80% of the brain, including most of the frontal, parietal, and temporal hemispheres and the basal ganglia. The two internal carotid arteries bifurcate to form two main branches:

- **Anterior cerebral arteries** supply blood to midline portions of the frontal lobes and superior medial parietal lobes.
- **Middle cerebral arteries** supply blood to most of the lateral cortex of the frontal, temporal, and parietal lobes.

POSTERIOR CIRCULATION

Also arising from the internal carotid arteries, the posterior cerebral artery contributes to the posterior circulation and supplies blood to the medial aspects of the occipital lobes, inferior portions of the temporal lobes, and cerebellum.

The rest of the posterior circulation arises from two smaller arteries—the right and left vertebral arteries—that ascend via the posterior portion of the neck. These two vertebral arteries join together to become the basilar artery.

These vertebrobasilar vessels supply blood to the remaining 20% of the brain, including the brainstem, cerebellum, and most of the posterior cerebral hemispheres.

Blood Supply of the Brain

Interior view of the brain's blood supply. (Source: Fotolia.com.)
Brain Function and Stroke Syndromes

Specific neurological functions are dependent on specialized brain regions, with each artery primarily supplying a particular region. Thus, injuries to particular branches of the major cerebral arteries produce characteristic stroke syndromes, which are symptom complexes caused by impaired blood supply to specific areas of the brain. These syndromes help clinicians to infer which brain areas have been damaged in a specific patient’s stroke.

INTERNAL CAROTID ARTERY STROKES

*Anterior Cerebral Artery (ACA) Stroke Syndrome*

Cutting off the blood supply to the entire field of one ACA will affect frontal regions on the medial surface of one half of the brain, much of the corpus callosum, part of the internal capsule, and regions of the basal ganglia. The resulting symptoms can include:

- Loss of discriminatory sensation and weakness or paralysis of the contralateral foot and leg, perhaps with some deficits in the contralateral shoulder and arm
- Occasionally, deviation of the head and eyes toward the side of the affected cerebral artery
- Occasionally, central motor problems, ranging from expressive aphasia to abulia (an absence of willpower or an inability to act decisively) to dyskinesia
- Difficulties with volition, motivation, planning, and organizing complex behavior
- Dysarthria, aphasia
• Loss of sense of smell
• Apraxia (inability to perform purposive movements or to use objects properly)
• Alien-hand syndrome (in which patients think the hand is not part of their body and that they have no control over its movement)
• Callosal disconnection syndromes (split-brain syndrome) (Godefroy, 2013; UBC, 2016)

Middle Cerebral Artery (MCA) Stroke Syndrome

The middle cerebral artery is the largest cerebral artery and the most commonly affected by stroke (Slater, 2015). Cutting off the blood supply to the entire field of one MCA will affect the primary sensory and motor cortices on the lateral surface of the cerebral hemisphere, sections of the internal capsule, and parts of the inferior parietal and lateral temporal lobes. The resulting symptoms can include:

• Full sensory loss and weakness or paralysis of the face, arm, and leg on the opposite side of the body
• Blindness in the opposite visual field (contralateral homonymous hemianopia)
• Deviation of the head and eyes toward the side of the affected MCA
• If the dominant (usually left) MCA has been occluded, global (i.e., both expressive and receptive) aphasia
• If the nondominant (usually right) MCA has been occluded, contralateral neglect (hemineglect) or the patient’s unawareness or denial of their neurological deficits (anosognosia)

Cutting off the blood supply to only the superior branches of the MCA will lead to a subset of these deficits. For example, there is often less effect on the contralateral leg and foot, and the communication difficulties are typically limited to expressive (Broca’s) aphasias (NINDS, 2016a).

Cutting off the blood supply to only the inferior branches of the MCA will lead to a subset of deficits, with little sensory or motor loss on the contralateral body side but with a full or partial contralateral homonymous hemianopia. In this case, the patient’s communication difficulties are typically limited to receptive (Wernicke’s) aphasias (NINDS, 2016a).

Posterior Cerebral Artery (PCA) Stroke Syndrome

Cutting off the blood supply to the entire field of one PCA will affect the thalamus, hippocampus, underside of the temporal lobe, medial surface of the occipital lobe, and...
motor areas of the midbrain (Helseth, 2015). PCA strokes can produce a wide variety of symptoms, including:

- Sensory loss on the entire contralateral body (all the way to the midline) (here, when sensation gradually returns, it is frequently accompanied by pain)
- Facial (VII) cranial nerve palsy, which may also be associated with hemiparesis, hemiplegia, ataxia, or decreased levels of consciousness
- Movement disorders on one side of the body, such as hemiballismus (spasms), hemichoreoathetosis (irregular involuntary twisting and writhing contractions), or hemiataxia
- Acute vision loss, specifically, homonymous hemianopia (visual field loss on the same side of both eyes)
- Dyslexia (difficulty with reading)
- Confusion
- New onset posterior cranium headache

**VERTEBRAL/BASILAR ARTERY STROKES**

**Vertebral Artery Stroke Syndrome**

Cutting off the blood supply to the entire field of one vertebral artery will affect the medulla of the brainstem. Vertebral artery strokes can produce a wide variety of symptoms, including:

- Vertigo
- Nystagmus
- Vomiting
- Ipsilateral (same-sided) ataxia
- Hypoglossal nerve dysfunction resulting in:
  - Dysarthria (unclear speech)
  - Dysphagia (difficulty swallowing)
  - Mastication (chewing)
- Ataxia from cerebellar involvement
- Reduced corneal reflex
- Hypacusis (partial hearing loss)
• Dysarthria (unclear speech)
• Paralysis of the palate, pharynx, and vocal cord
• Loss of taste in posterior third of the tongue
• Contralateral loss of pain and temperature sensation in the trunk and limbs
• Tachycardia and dyspnea
• Palatal myoclonus (involuntary jerking of the soft palate, pharyngeal muscles, and diaphragm)
  (Kaye, 2015; Tidy, 2016)

**Basilar Artery Stroke Syndrome**

Cutting off the blood supply to the entire field of the basilar artery will result in:

• Bilateral neurological problems, such as bilateral sensory and motor deficits
• Combined cerebellar and cranial nerve problems
• Hemiparesis with contralateral cranial nerve dysfunction or with ipsilateral ataxia
• Behavioral abnormalities
• Somnolence, hallucinations

Occlusion of the basilar artery is commonly catastrophic, resulting in:

• Rapid clinical deterioration in consciousness and ultimately death
• Quadriplegia
• “Locked in” syndrome (a state in which the patient can think and see but may not be able to respond in any way)
  (Hain, 2016d; Di Muzio et al., 2016)

**PATHOPHYSIOLOGY OF STROKE**

**Evolution of a Stroke**

Cerebral infarct is a process that occurs in four stages:

• Hyperacute (first 3 to 6 hours)
• Acute (1 day to 1 week)
• Subacute (1 week to 1 month)
• Chronic (>1 month)

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The hyperacute stage is a very dynamic period in the evolution of ischemic stroke. Growth of the lesion begins immediately after the onset, and the largest volume of tissue infarction occurs within 10 minutes of stroke onset. Over half of the total infarct volume occurs within 30 minutes of stroke onset (Zhang et al., 2015).

The acute stage begins six hours after the onset of the stroke. During this stage, release of inflammatory mediators from ischemic brain tissue leads to vasogenic edema with extravasation of water molecules from blood vessels to expand the interstitial spaces. This is a major deleterious type of brain edema, which persists for several days. Imaging during this period shows the involved area is soft and edematous, with a blurring of anatomic detail.

During the subacute stage there is obvious tissue destruction and liquefactive necrosis of the involved brain. Liquefactive necrosis results in a transformation of tissue into a liquid mass.

In the chronic stage of stroke, the damaged tissue has been phagocytized and there is cavitation with surrounding gliosis, a process leading to scarring (ISC, 2016).

Cell and Tissue Injuries Caused by Strokes

Stroke pathophysiology includes ischemic and mechanical damage. For decisions about acute treatment, the particular stroke syndrome is usually less important than the type of vascular injury that has occurred.

ISCHEMIC DAMAGE

The term ischemia indicates oxygen and nutrient deprivation due to an insufficient supply of blood. Both ischemic and hemorrhagic strokes cause ischemic damage. Hemorrhagic strokes cause additional ischemic damage due to the pressure that builds from the excess blood that has been released into the brain or the cerebrospinal fluid (CSF). This increase in intracranial pressure (ICP) presents additional problems for the hemorrhagic stroke patient, particularly in the acute phase and within the early recovery period.

When cerebral blood flow is reduced, the affected regions of the brain begin to stop functioning and the patient begins to lose the ability to perform the tasks that are localized in those regions.

Complete (Global) Ischemia

If the blood supply to a brain region is cut off entirely, as occurs most commonly during cardiac arrest, cell damage is widespread and neurons begin to die quickly. The brain uses energy at a high rate, but it can only store a small back-up supply of energy. Complete ischemia immediately decreases the available oxygen and glucose in the affected region of the brain, and without continual nourishment, local neurons will run low on their internal back-up stores of adenosine triphosphate (ATP) within seconds.
Once a neuron’s ATP is depleted, its membranes depolarize and extracellular ions stream in; this swells the cell with an accompanying inrush of water. The depolarization also sets off the release of unusually large amounts of extracellular excitatory neurotransmitters. These events cause the influx of calcium ions, which set off an unregulated intracellular cascade of calcium-triggered processes, including the activation of catabolic enzymes, such as proteases, phospholipases, and endonucleases. In a short time, the neuron self-destructs and dies. The entire process is termed cortical spreading depression (Ayata & Lauritzen, 2015).

**Incomplete Ischemia**

Most strokes do not produce complete ischemia. Many ischemic strokes leave arteries only partially blocked. Even when an artery is entirely occluded, the cerebral circulation has some collateral coverage with overlap and interconnections, and some blood usually gets to the affected brain regions via other routes. The perfusion that remains will vary throughout the ischemic region. A common pattern is severely reduced perfusion in the core of an ischemic region, with gradually increasing perfusion toward the edges.

Neurons become functionally silent when their arterial perfusion drops by a small amount. In a stroke, as soon as cerebral blood flow is reduced, electrical activity stops in the affected region of the brain and neurological deficits appear. For a time, the silent neurons remain alive, but they no longer have the energy to generate membrane potentials that are sufficient to respond to stimuli or to transmit signals. However, to remain alive, the silent neurons still need some arterial perfusion. If cerebral blood flow drops below 40% of normal in part of an affected region, the silent neurons begin to die (Joseph, 2015).

In most strokes, patients lose neurologic functions early, before all the neurons in the affected area are irreversibly damaged. Typically, strokes leave enough arterial perfusion that many neurons can maintain a low level of energy production sufficient to slow the onset of their deaths (Joseph, 2015).

**ISCHEMIC STROKES LEAVE AN EARLY THERAPEUTIC WINDOW**

After an ischemic stroke, the amount of irreversible damage (infarct) increases steadily as long as regions remain without sufficient blood supply. In those parts of the affected region that have no blood flow, neurons begin to die in less than 10 minutes, and for every hour untreated, the brain ages 3.6 years (Fussner, 2016).

In every infarct there is a central core of total ischemia and cell death that is irreversible. This central core is surrounded by a zone of borderline ischemic tissue called the ischemic penumbra. Ischemia in this area causes dysfunction but is not severe enough to cause structural damage. If prompt restoration of perfusion in the penumbra can be restored, permanent structural damage may be prevented.
The window of opportunity for saving the penumbra is short. And if adequate blood supply is not restored within three hours, the necrosis will extend to the penumbra (Agamanolis, 2014). It has been found that collateral and residual blood flow can preserve neurons in the penumbral and border areas for as long as six hours after an ischemic stroke. Within this six-hour window, certain treatments can reduce the amount of brain damage that is irreversible (Cuccione et al., 2016).

**MECHANICAL DAMAGE**

Besides ischemic damage, hemorrhagic strokes produce mechanical damage. The force of blood flowing extracellularly in the brain parenchyma pushes cells apart, dissects brain tissue, destroys connections, and injures brain cells.

On a larger scale, the excess pressure can also be quite physically damaging. An expanding hematoma, in combination with cerebral edema, can push portions of the brain through intracranial narrow spaces, such as the dural openings or the foramen magnum. The result is brain herniation. Herniation can irreversibly damage brain regions, and when vegetative brain centers, such as the reticular activating system or the respiratory control nuclei, are compressed, the result can be coma or death.

Moreover, the global compression caused by increased intracranial pressure from a hemorrhagic stroke can cause the cardiovascular system to malfunction, and significant increases in ICP lead to reduced consciousness, global brain ischemia, and death (Grotta et al., 2015).

**ETIOLOGY OF STROKE**

The reduction of the blood flow to a region of the brain leads to an ischemic stroke. Causes of such reductions include:

- Most commonly, an arterial blockage, initiated by local processes (thrombi) or as a result of clots from afar (emboli)
- Occasionally, a systemic cardiovascular problem, such as shock or cardiac arrest (atherosclerosis can also predispose to thrombi and/or emboli)
- Other causes such as genetic factors and inflammatory processes

**Arterial Blockage**

Many ischemic strokes result from clots (thrombi) that form within the cerebral arteries. It is helpful to divide the conditions leading to such locally generated obstructions into large vessel pathologies and small vessel pathologies.
• **Large vessel pathologies.** Atherosclerosis is the most common cause of large vessel occlusive disease. Atherosclerotic thrombi are usually formed along plaque that has become ulcerated or disrupted, and such plaque disruptions tend to occur at places where the blood flow is turbulent (e.g., at arterial branch points). Atherosclerotic thrombi can enlarge in situ and reduce distal blood flow, or they can break off and occlude smaller arteries upstream. Besides atherosclerosis, other occlusive conditions of large cerebral vessels include vasoconstriction (as in migraine disease) and arterial dissections (Jauch, 2015).

• **Small vessel pathologies.** Small vessel damage usually results from lipohyalinosis, which thickens the media in the walls of small arteries and eventually leads to small artery occlusions and stroke. Lipohyalinosis, which is produced by hypertension combined with atherosclerosis, is especially destructive to those branches of the middle cerebral, vertebral, basilar, and Circle of Willis arteries that come off at right angles to the parent artery and that dive into the brain parenchyma. Obstruction of these penetrating arteries produces small, deep, noncortical infarcts called lacunae, and the clinical results are called lacunar strokes (Jauch, 2015).

Other ischemic strokes are caused by **emboli** (debris and clots that arise elsewhere and are subsequently swept into the cerebral circulation). Extracranial stroke emboli are formed by large vessel pathologies and by other conditions that foster the formation of blood clots that can crumble or be dislodged. One common source of stroke emboli is the left atrium of the heart, where thrombi can form during atrial fibrillation. Another source of stroke emboli is the aorta and the carotid artery, from which atherosclerotic plaque and clots detach and are then carried deeper into the cerebral vasculature. Acute myocardial infarction is associated with a 2% to 3% incidence of embolic stroke, and of these, 85% occur in the first month after infarct (Jauch, 2015).
Atrial fibrillation can lead to stasis of blood in the left atrium. This allows blood clots to form. If a piece of clot breaks off and travels to an artery in the brain, it can block the blood flow through the artery, causing a stroke. (Source: NHLBI, 2014.)

Systemic Cardiovascular Problems

Widespread cerebral hypoperfusion can produce global brain ischemia. The causes of cerebral hypoperfusion range from arrhythmias to cardiac arrest and from respiratory failure to bleeding or shock. The symptoms of a global reduction of blood flow to the brain are diffuse, bilateral, and nonfocal, and they include the signs of circulatory compromise—pallor, sweating, tachycardia, and hypotension (CUMC, 2016).

Symptoms of stroke can occur due to inadequate cerebral blood flow because of decreased blood pressure (specifically, decreased cerebral perfusion pressure), hematologic hyperviscosity from sickle cell disease, or other hematologic diseases (i.e., multiple myeloma and polycythemia vera).
Genetic and Inflammatory Mechanisms

Evidence has shown that inflammation and genetic factors play important roles in developing atherosclerosis and, specifically, in stroke. Atherosclerosis has been found to be a dynamic, chronic inflammatory condition caused by a response to endothelial injury. LDL cholesterol and smoking contribute to this injury, and infections have also been suggested as a contributor to endothelial injury and atherosclerosis (Sultan, 2015).

A number of genes are also known to increase a person’s susceptibility to ischemic stroke, and research is currently underway in this area to identify them (Sultan, 2015).

Ischemic strokes can be caused by sickle cell disease, which is seen in about 6% of children with stroke. The primary site of strokes in these patients is in the large arteries. Because sickle cells are rigid, they clump up along the walls of these arteries, damage vessel walls, and cause more sickle cells to accumulate, further narrowing the vessels (Sultan, 2015; Talama et al., 2014).

Intracranial Hemorrhage

Intracranial hemorrhages occur within the cranium. They include epidural, subdural, subarachnoid, intracerebral, and intraventricular bleeds.

Intracranial bleeds and subsequent hemorrhagic strokes can be produced by either external or internal causes (e.g., head trauma as a result of a fall, bleeding tumors, hypertension, etc.). Intracranial hemorrhages can rapidly cause brain damage and can be life threatening. They can happen in persons of any age (Liebeskind, 2016; Sykora et al., 2014).

INTRACEREBRAL HEMORRHAGE (ICH)

ICH accounts for 8% to 13% of all strokes. ICH results from a wide range of disorders and is more likely to result in death or major disability than ischemic or subarachnoid hemorrhage. ICH is usually caused by bleeding from smaller arteries or arterioles. The most common causes are:

- Hypertension
- Trauma
- Amyloid angiopathy
- Bleeding diatheses (either genetic or acquired)
- Cocaine or amphetamine use
- Ruptured vascular malformations or aneurysms
  (Liebeskind, 2016)
SUBARACHNOID HEMORRHAGE (SAH)

Many subarachnoid hemorrhages are due to trauma, but spontaneous ruptures of a cerebral aneurysm are also common. Most of these aneurysms are on or near the anterior portions of the Circle of Willis (Sunderrajan et al., 2013).

The causes of aneurysm formation and rupture are still debated. Some relevant observations include:

- Two percent of the population have unruptured cerebral aneurysms >3 mm in diameter.
- Cerebral aneurysms develop gradually and most are not fully formed at birth. Rupture of a cerebral aneurysm is most often a condition of middle age, peaking in people aged 35 to 65 years.
- Most ruptured cerebral aneurysms occur in people with normal blood pressure.
- The larger aneurysms are the ones most likely to rupture. The average cerebral aneurysm is 7.5 mm in diameter, but ruptured aneurysms tend to have been larger than 10 mm in diameter.

Cryptogenic Strokes

One third of ischemic strokes are classified as cryptogenic. Cryptogenic strokes are ischemic strokes in which a comprehensive evaluation cannot define the cause. Most cryptogenic strokes produce symptoms similar to those of strokes known to be caused by emboli; nonetheless, the strokes are labeled cryptogenic if available tests cannot document the specific cause (AHA/ASA, 2015d).

PREHOSPITAL AND EMERGENCY DEPARTMENT EVALUATION FOR STROKE

Strokes produce the sudden loss of neurocognitive function, including motor and sensory dysfunction. Many things can be done to reverse or to temper the effects of a stroke, but successful medical therapy depends on immediate medical attention. Therefore, patients having a stroke need to be taken immediately to an emergency department (ED) that has the personnel and equipment to provide comprehensive acute stroke treatment, preferably a certified stroke center.

Certified Stroke Centers

To carry out acute treatment protocols, medical care providers need both specialized knowledge and practical experience. However, the facilities, equipment, and personnel for acute stroke management are expensive and are not available at most hospitals.
Stroke centers have a permanent stroke team with two divisions. The code team—a neurologist (or ED stroke specialist) and a neurology nurse—is always available to respond to a page and institute emergency care. The larger support team is a task force that keeps the stroke program organized, efficient, and up-to-date with a unit staffed by a multidisciplinary team specializing in treating acute stroke and stroke-related complications.

The support team includes members from many disciplines, including neurology, emergency medicine, neurosurgery, nursing, radiology, pharmacy, laboratory, physical medicine, and rehabilitation (Ellmers, 2015). It consists of physicians, nurses, physical therapists, occupational therapists, speech and language therapists, and social workers. Best practice indicates that a vascular neurologist should play the role of leader in the stroke unit.

In North America, the Joint Commission (TJC) offers three advanced levels of certification for stroke programs:

- Acute Stroke-Ready Hospital
- Primary Stroke Center
- Comprehensive Stroke Center

**ACUTE STROKE-READY HOSPITAL (ASRH)**

In 2015, the Joint Commission developed a new certification program for Acute Stroke-Ready Hospitals. Facilities that receive this certification provide the following:

- A medical program director with sufficient knowledge of cerebrovascular disease
- Stroke protocols and an acute stroke team available 24 hours/7 days per week and at bedside within 15 minutes
- Collaborative relationship with local EMS, encouraging training in field assessment tools and communication prior to bringing a stroke patient to the emergency department
- Initial assessment performed by an emergency department physician, nurse practitioner, or physician assistant
- CT, MRI, and lab capability 24/7
- Access to a neurologist 24/7 in person or by telemedicine (within 20 minutes)
- Neurosurgical services available within three hours (through transfer)
- IV thrombolytic administration capability
- Transfer protocols with one PSC or CSC

(TJC, 2016)
PRIMARY STROKE CENTER (PSC)

To be certified as a Primary Stroke Center, an emergency department (or its hospital) must meet all the criteria for Acute Stroke-Ready Hospitals, plus additional criteria, including:

- A stroke unit or designated beds for the acute care of stroke patients
- Initial assessment completed by an emergency department physician
- CT, MRI, labs, CTA, MRA availability 24/7; cardiac imaging when needed
- Access to neurosurgical services within two hours or availability 24/7 in a PSC that provides neurosurgical services
- Provision of education to prehospital personnel and a minimum of two stroke education activities per year to the public
- Use of eight standardized measures to evaluate clinical performance (TJC, 2016)

COMPREHENSIVE STROKE CENTER (CSC)

Comprehensive Stroke Center certification is available only in Joint Commission–accredited acute care hospitals. Organizations seeking CSC certification must meet all of the general eligibility requirements for PSC certification. In addition, CSCs are required to:

- Have a program medical director with extensive expertise available 24/7
- Have dedicated neuro-intensive care unit beds for complex stroke patients that provide neuro-critical care 24/7
- Use advanced imaging capabilities
- Have capabilities for microsurgical neurovascular clipping of aneurysm, neuroendovascular coiling of aneurysm, stenting of extracranial carotid arteries, carotid endarterectomy, endovascular therapy
- Protocols for receiving transfers and circumstances for not accepting transfers
- Sponsor a minimum of two public education opportunities annually; present a minimum of two educational courses annually for internal staff or individuals external to the CSC (e.g., hospitals)
- Participate in stroke research approved by the institutional review board (TJC, 2016)
STROKE CENTER TIME TARGETS

Stroke centers are dedicated to quick, efficient care. The recommended time targets for key steps in the management of acute stroke are as follows:

- From the door to a physician, 10 minutes
- From physician to neurological expertise, 15 minutes
- From the door to a completed CT, 25 minutes
- From the door to the reading of the CT scan by a specialist, 45 minutes
- From the door to treatment, 60 minutes
- From the door to admission to a monitored bed, 3 hours

(Jauch, 2015a)

The time of 45 minutes is set as the first milestone in the ED stroke protocol. Within the first 45 minutes of a patient’s evaluation, they should be channeled into one of two treatment pathways:

1. Stroke patients with any sign of intracranial bleeding are managed using a protocol that is based on close monitoring and individualized treatment of medical complications.

2. Stroke patients with no bleeding and with evidence of an obstructed cerebral artery (i.e., an ischemic stroke) are managed using a protocol that can lead to rapid intervention, typically with the IV administration of a “clot-busting” drug.

By 45 minutes, the stroke team should have distinguished ischemic from hemorrhagic strokes. The rapid diagnosis of an acute stroke and the determination of its type allow a stroke team the widest range of direct treatment options. Thrombolytic treatment (“clot-busting”) of ischemic strokes is recommended within a limited time window (currently, 4.5 hours after the initial stroke symptoms). Time is such a critical element that a written time sheet is maintained for each stroke patient. Timekeeping is one of the important tasks for the ED and stroke team nurses; nurses are the team members who keep stroke care on schedule (Jauch et al., 2013).

TELESTROKE CONSULTATION

The time-dependent stroke treatments, such as intravenous rtPA, are only recommended for use in hospitals with experienced staff and well-equipped facilities. Ideally, the treatment of all acute strokes would be done in Primary Stroke Centers, but many areas of the country are far from such centers. One way to extend the range of acute stroke treatment, especially the administration of thrombolytic agents, into areas far from stroke specialists is by using video teleconsultation, or telestroke.

Telestroke (telemedicine) is a two-way videoconference between distant stroke-care specialists (neurologists) and local bedside-care providers. Telestroke works exactly like a direct onsite consultation, and as with onsite consultations, patients or their families are kept involved and
Barriers to telestroke programs include issues of medical liability and malpractice across state lines. Additionally, telemedicine is increasingly subject to extensive regulation, and medical licensing laws may limit the use of out-of-state telestroke consultations.

Source: Greve, 2015; Gold, 2014.

FOLLOWING THE STROKE CHAIN OF SURVIVAL

To best streamline the response to potential stroke patients, the American Heart Association developed the “Stroke Chain of Survival.” This chain involves steps that should be followed by patients, families, prehospital, and emergency personnel in response to a patient who is experiencing a stroke. This chain involves:

- Detection
- Dispatch
- Delivery
- Door
- Data
- Decision
- Drug
- Disposition

Source: ACLS, 2016.

The Role of Patients and Bystanders

The role of patients and bystanders involves the first two links in the stroke chain of survival:

- **Detection:** Recognizing a stroke
- **Dispatch:** Responding by calling 911

RECOGNIZING A POTENTIAL STROKE

Recognizing that a stroke may be taking place is the first step in caring for the patient, so public education and information is required in order to increase the recognition of potential strokes.
Persons experiencing a stroke from ischemia or hemorrhage can have the following symptoms:

- Weakness: hemiparesis, monoparesis, or (rarely) quadriplegia
- Hemisensory deficits
- Monocular or binocular visual loss
- Visual field deficits
- Diplopia (double vision)
- Dysarthria (difficulty in speech articulation)
- Facial droop
- Ataxia (lack of muscle control)
- Vertigo (rarely in isolation)
- Aphasia (lack of language abilities)
- Sudden decrease in level of consciousness
- Sudden severe headache in approximately 50% of those with ICH
- Seizure with no prior history
  (Jauch, 2015; Liebeskind, 2016)

Persons experiencing a transient ischemic attack may also present with one or more of these symptoms:

- Sudden weakness, numbness, or paralysis in the face, arm, or leg (most often on one side of the body)
- Confused, muttered, slurred, or garbled speech
- Difficulty in understanding when someone else is speaking
- Sudden blindness in one or both eyes or double vision
- Dizziness, loss of balance, or loss of coordination
  (Nanda, 2015)
Patients should be told that if they are having any of these symptoms, they should call 911 or get someone else to call 911 (see below).

However, even people who have been taught the warning signs may not realize that they are having a stroke. Some factors contributing to this problem are:

- Stroke can change a person’s level of consciousness.
- Stroke can make a person confused.
- Stroke victims can misunderstand the seriousness of their bodies’ signals; for instance, pain is a major symptom of illness, but most strokes are painless.
- Stroke victims with damage to their nondominant parietal lobe can lose the ability to recognize that they are ill.
- The person may be in denial.

For these reasons, it is often a family member or bystander who first realizes that a medical problem is occurring. The public should understand that if there is the possibility that someone is having a stroke, they should not hesitate—they should call 911 immediately.

**STROKE ASSESSMENT TOOL**

The mnemonic *FAST* (also known as the Cincinnati Prehospital Stroke Scale + Time) is the easy way to remember the sudden signs of stroke. When these signs are spotted, call 911 immediately:

- **F** – Face drooping. Ask the person to smile. Is the person’s smile uneven?
- **A** – Arm weakness. Is one arm weaker or numb? Ask the person to raise both arms. Does one drift downward?
- **S** – Speech difficulty. Is speech slurred? Is the person unable to speak or hard to understand? Ask the person to repeat a simple sentence such as, “The sky is blue.” Is the sentence repeated correctly?
- **T** – Time. Call 911 if someone shows any of these symptoms. Even if the symptoms go away, call 911 and get the patient to the hospital immediately. Take note of the time in order to report when the first symptoms appeared.

Source: NINDS, 2016c.

**RESPONDING BY CALLING 911**

People often wonder what first aid to give to a stroke victim. The best first aid is professional transport to a hospital, and getting an ambulance is the most important thing a bystander can do for a stroke victim.
CALL 911 OR GO DIRECTLY TO THE HOSPITAL?

In an emergency, people often feel that time is being lost by waiting for an EMS (emergency medical service) team to arrive, and family members or bystanders often hurriedly drive patients to the hospital. In fact, however, patients usually get to the appropriate hospital faster if they use the EMS system by calling 911. EMS teams are trained to choose the most appropriate hospital in the region, and this is not necessarily the closest hospital. In addition, the care and assessment that an EMS team gives a stroke victim shortens the time lag between the onset of stroke symptoms and the evaluation and treatment of the stroke.

EMS teams should advocate for widely available 911 capabilities in their region. All landlines and wireless phones should be able to reach local 911 operators. It is also important that the caller’s number and location be displayed automatically for dispatchers. At the moment, two telephone systems do not always give 911 operators the detailed locations of callers: Multiline Telephone Systems (MLTS), which are used by many large organizations, and Voice over Internet Protocol (VoIP) services.

Since wireless phones are mobile, they are not associated with one fixed location or address. Wireless phones may provide a general idea of the caller’s location, but it is not always possible to provide assistance quickly. Some providers may offer new location-capable phones (E911). The FCC has addressed this issue, and new standards for location now apply to outdoor measurements. Indoor cell phone location continues to pose obstacles (FCC, 2015).

The Role of Emergency Response

EMS DISPATCHERS

The role of EMS dispatchers (911 operators) also involves the first two links in the stroke chain of survival:

- **Detection:** Identifying a possible stroke
- **Dispatch:** Responding by dispatching EMS

Dispatchers play a key role in the diagnosis of stroke. EMS dispatchers are the first medical contact the patient has. In general, EMS dispatchers have these responsibilities:

- Identifying the presenting problem
- Choosing, notifying, and sending the team of responders that is appropriate for each emergency
- Advising the callers on possible first aid for the victim
- Getting critical background information about the victim
Identifying the Problem

Dispatchers should recognize that a person may have had a stroke if any of the signs and symptoms of stroke (as noted above) are reported by the patient or bystander.

When the caller’s description includes any of the preceding signs, the 911 operator asks these questions about the person:

- Is she/her completely awake (alert)?
- Is she/he breathing normally?
- Is there facial drooping? Is the smile uneven?
- Is there arm weakness? Does one arm drift downward?
- Is she/he having speech problems? Can he/she understand what is being said?
- What time did this start (happen)?

When a dispatcher is able to flag a possible stroke victim, the EMS team can be given time to review and plan during their outbound trip and to notify the nearest stroke center.

Assigning Potential Strokes High Priority

EMS dispatchers decide what type of response is appropriate for each emergency. They choose:

- The skill level and equipment of the EMS response team: basic life support (BLS) or advanced life support (ALS)
- The type of vehicle to send
- The initial speed requirement (e.g., sirens, flashing lights, etc.)

Acute strokes require the same level of emergency treatment as heart attacks and trauma. American Heart Association/American Stroke Association (AHA/ASA) guidelines recommend that potential strokes be given the highest level of priority and that EMS dispatchers send the highest level of emergency care available. When available, an ALS team should be sent (Jauch, 2015a). If a choice has to be made, however, speed of transport to a stroke center is the first consideration. Therefore, if an ALS team is not immediately available, a BLS team should be dispatched.

Stroke should be a priority dispatch with a prompt EMS response. Designated stroke centers will be the preferred destination for patients with acute stroke symptoms. Telemedicine is also a new technology that has the ability to provide timely expert advice to rural and underserved clinics and hospitals (Jauch, 2015a).
When patients having a stroke are more than one hour’s travel time by ambulance from a hospital that is equipped to treat acute strokes, then air transport should be considered. Helicopters or other aircraft can be used to take the EMS team to the patient and then to transport the patient and the EMS team to a stroke center. Helicopters can also be used for secondary transport of patients from a remote receiving ED to a stroke center (Jauch, 2015a).

**Advising on Possible First Aid**

While waiting for an ambulance to arrive, the dispatcher can offer assistance to the caller to provide first aid for the patient.

- If the caller is the patient, instruct him/her to lie down.
- Reassure the person that help is on the way.
- Assist to a position of comfort, supporting the head.
- Check airway to make certain it is clear and open.
- Wipe secretions from the mouth to prevent aspiration.
- Cover the person to prevent heat loss.
- Loosen any tight clothing.
- Do not give the person anything to eat or drink.
- Do not give the person any medications, including aspirin.
- If the person is unconscious, place in recovery (side-lying) position with head supported.
- Unlock the doors for EMS to gain quick entry.

(O’Neill Hill, 2015)

**Collecting Critical Information**

When an EMS operator suspects that a call concerns an individual experiencing a stroke, the operator begins collecting critical background information. Dispatchers should make a special effort to get an estimate of the elapsed time since any potential stroke symptoms first appeared and to collect as much relevant data as possible.
CRITICAL BACKGROUND INFORMATION ABOUT POTENTIAL STROKE VICTIMS

1. Obtain the patient’s medical history, inquiring specifically about:
   - Past strokes
   - TIAs
   - Hypertension
   - Diabetes
   - Myocardial infarction and other heart problems
   - Atherosclerosis and peripheral artery disease
   - Bleeding disorders
   - Recent surgeries
   - Liver disease

2. Record the patient’s current medications, inquiring specifically about:
   - Aspirin, anticoagulants, and antiplatelet agents
   - Beta blockers
   - Calcium channel blockers
   - Insulin
   - Antihypertensives
   - Cocaine, amphetamine, and other “street” drugs
   - Alcohol intake

3. Note the time when the symptoms first appeared and the last time that the patient did not have the symptoms.

4. Ask about any recent injury, inquiring specifically about head trauma.

EMS RESPONDERS

The links in the stroke chain of survival that EMS responders are concerned with include:

- **Detection**: Rapid EMS confirmation of a possible stroke
- **Delivery**: Rapid management and transport
- **Door**: Appropriate triage to a stroke center or high-acuity area facility

Upon arrival at the scene, EMS responders initially manage CABs (chest compressions, airway, breathing) and give oxygen if needed.
Confirming a Possible Stroke

A prehospital stroke assessment is completed using an assessment tool such as the Cincinnati Prehospital Stroke Scale (CPSS) or Los Angeles Prehospital Stroke Screen (LAPSS). The tool most commonly used is the CPSS (see below), a simple three-item scale based on the National Institutes of Health Stroke Scale and designed specifically for use by EMS. The LAPSS comprises multiple elements, including the history, blood glucose, and specific physical findings. Use of these tools has been shown to increase paramedic sensitivity to stroke identification to ≥90% (NSA, 2016).

CINCINNATI PREHOSPITAL STROKE SCALE

In the CPSS, the patient is asked to perform three actions. The presence of all three components has been found to identify 100% of patients with stroke. An abnormal response to any of the three indicates that it is likely that the patient is having or has recently had a stroke. The actions and the range of stroke and nonstroke responses are:

1. “Can you show me your teeth?”
   - Stroke likely = the sides of the face look different
   - Stroke less likely = the sides of the face look the same
2. “Please hold both arms out in front of you.”
   - Stroke likely = one arm drifts more or one arm doesn’t move
   - Stroke less likely = both arms move the same or both arms do not move at all
3. “Please repeat this sentence: ‘The sky is blue in Cincinnati.’”
   - Stroke likely = no speech, incorrect words, or slurring
   - Stroke less likely = correct words are repeated without slurring

Collecting Critical Background Information

Regardless of the information already collected by the 911 dispatcher, EMS responders should attempt to collect essential information about the patient (see the “Critical Background Information about Potential Stroke Victims” box above).

Because time is of the essence, responders should gather telephone numbers of relatives and witnesses. If knowledgeable acquaintances are available, they are asked to meet responders at the receiving hospital, or if necessary, to travel with responders. For emergency treatments, it will be helpful if next-of-kin are immediately available for consent.
Records are completed and then passed on to the medical team at the receiving hospital. Ideally, EMS teams will use developed checklists with the essential questions to capture all the critical information.

CASE

Marcella has just finished her training to become an EMS first responder. She performed well in all the training classes, but she is still quite nervous about her first call as a full-fledged EMS professional. Within the first half hour of her first shift, Marcella hears the call from the dispatcher about a likely stroke victim. Rushing to the scene, Marcella and her team are greeted at the door by the patient’s daughter, who is frantic with worry.

The patient is an 86-year-old African American woman sitting on the sofa. Marcella does an initial visual assessment and notices that the woman’s face appears to be sagging on the right side. While another team member is getting the woman’s vital signs, Marcella asks the woman to “Smile and show me your teeth.” The woman’s face clearly shows asymmetry. Then Marcella asks the woman to stretch out her arms as far apart as she can. The woman tries, but Marcella notices that her left arm is drifting down. More certain that the team is dealing with a stroke victim, Marcella asks the woman to repeat the sentence “The sky is blue in Cincinnati.” When the woman slurs her words, Marcella tells the other team members that they need to take the woman to the nearby stroke center. The team is able to quickly transport the woman, whose vital signs remain stable, in under 10 minutes to the stroke center.

Later that evening, while reflecting on her first day as an EMS professional, Marcella realizes the importance of her stroke training. Within 30 minutes of the onset of symptoms, the woman was examined by stroke specialists and now has a good prognosis for eventual recovery.

Transporting the Patient

Maintaining circulation, airway, and breathing are the first priorities. For strokes, keeping the head flat (i.e., supine or 0-degrees elevation) usually offers better brain circulation than keeping the head elevated when the flat position does not impair the CABs.

After stabilizing the patient, time is paramount. As soon as possible, begin transporting the patient to the appropriate ED and continue the rest of the prehospital care en route. Each EMS unit should be provided with maps showing the nearest appropriate ED for stroke management in any area (NSA, 2016).

Information is exchanged between the EMS team and the ED stroke team. The hospital stroke team can tell the paramedics about the size and placement of the IV access that will be needed, and hospital specialists can advise the paramedics about managing complications, such as severe hypertension, hyperglycemia, or cardiac dysfunction.
Additional Care En Route

**Oxygen.** Strokes are crises of insufficient oxygen delivery to the brain, so it is important to keep the patient’s blood oxygen saturation at normal levels. Attach a pulse oximeter and treat hypoxemia (in this case, oxygen saturation <94%) with supplemental O₂. Currently, there is no indication that supplemental oxygen will benefit a patient who already has normal levels of blood oxygen saturation.

**IV access.** When acute resuscitation is needed, insert an IV line immediately. Otherwise, consider starting an IV en route after consulting the destination ED. Some key brain imaging studies require large bore IV lines that must be inserted proximally (i.e., no more distal than the antecubital fossa). If the receiving hospital will need a specialized IV line, placing the appropriate line in advance can save time.

**IV fluids.** Treat shock or significant dehydration with balanced salt solutions (isotonic crystalloids, such as normal saline). Otherwise, saline lock the IV or set the IV to drip the minimum amount of balanced salt solution to keep the line open. In general, the goal is to add only a minimal amount of extra fluid, because overhydration can cause cerebral edema. Another concern is hyperglycemia, which can worsen the injury in a stroke. Therefore, do not use dextrose solutions except to correct hypoglycemia.

**Blood glucose level.** Hypoglycemia produces symptoms that look like stroke, and persistent hypoglycemia will cause brain injury. Therefore, as soon as possible, check the patient’s capillary blood glucose level and treat hypoglycemia with 50% dextrose.

**ECG.** Attach a 3-lead ECG and monitor the patient’s heart continuously with two specific objectives:

1. Watch for serious cardiac consequences. The brain’s reaction to stroke includes an increase in the body’s sympathetic tone, and this predisposes a person to arrhythmias and myocardial infarction.

2. Screen for cardiac causes. Strokes can be caused by preexisting atrial fibrillation or by atherosclerosis, which can already have caused heart damage that can be seen in ECG recordings.

**Hypertension management.** Hypertension is a common finding in acute stroke. Monitor blood pressure but do not treat arterial hypertension.
NURSE EDUCATORS FOR EMS TEAMS

Nurse educators are often responsible for teaching first response techniques for strokes to local emergency medical technicians. The basic information to be covered is found in the American Heart Association’s ACLS provider manual and online. Nurse educators place special emphasis on:

- Stroke victims needing immediate care in specialized emergency departments
- Stroke being given the same high priority as myocardial infarction
- EMS teams having written stroke-specific protocols and/or checklists prepared in advance
- Simple stroke assessments, such as the Cincinnati Prehospital Stroke Scale, being quickly completed in the field
- Obtaining needed information from the patient or bystanders, including the time of onset of neurological symptoms or the last time the patient was without neurological symptoms
- EMS teams needing to know the closest acute care stroke hospitals
- After alerting the destination ED, EMS responders then staying in touch with the ED staff for advice

As an EMS instructor, a nurse tailors the emergency response protocols to the local region. First, the nurse must know which medical techniques can be performed by paramedics and emergency medical technicians under local regulations. Second, the nurse must learn which area hospitals are equipped and staffed for treating acute strokes.

CASE

Recently trained as an EMS provider, John takes a call from the dispatcher about an 83-year-old female patient with a possible stroke. On arrival, after taking the patient’s vital signs, John notes that the patient has a blood pressure of 200/90 mm Hg, a respiration rate of 28 breaths/minute, and a blue tinge around her mouth. John’s supervisor instructs him to place an oxygen mask on the patient, start an IV line, and continue monitoring the patient’s blood pressure.

When John asks about the potential dangers of the patient’s high blood pressure, the supervisor tells him that during an acute stroke, the current recommendations are to avoid attempting to control blood pressure until the patient can be fully evaluated by medical personnel. John continues to monitor the patient’s blood pressure, which remains the same, and her other vital signs. After five minutes on oxygen, John notices the patient’s color and her respiration rate normalizing. Another five minutes later, the EMS team and the patient arrive at the hospital, where the stroke team takes over the patient’s care.
In the Emergency Department

Once the patient has been admitted to an emergency room, the links in the stroke chain of survival include:

- **Data:** Obtaining laboratory results, performing physical and neurological exams, brain imaging
- **Decision:** Determining appropriate treatment
- **Drug:** Administering drug therapy if appropriate

When a potential stroke patient enters any ED, staff must begin a protocol that can lead directly to the administration of a thrombolytic drug at the present hospital or at a stroke center. The main goals are rapid access to thrombolysis for ischemic stroke patients and stabilization and rapid admission to a stroke unit for all stroke patients.

A typical protocol that is used to accomplish the goal of rapid treatment should include:

1. Triage
2. Stabilization of comorbid medical problems
3. Stroke diagnostic studies
4. Medical history
5. Patient examination
6. Diagnosis and hypothesis for stroke type and etiology
7. Cranial imaging to confirm diagnosis

(Jauch, 2016)

**TRIAGE**

Emergency department care begins with triage. The EMS team ideally will have identified any potential stroke victims that it is bringing, but approximately one half of all stroke patients will not use an EMS service or transportation to the ED. Therefore, the ED registration staff must be trained to look for signs of possible stroke (Jauch et al., 2013; Cavallini et al., 2015).

The front desk nurse should have a written stroke-recognition checklist. This will ensure that any triage nurse can quickly channel potential stroke victims into the ED’s stroke protocol.

**Time-to-treatment is critical.** Therefore, patients with suspected acute stroke are assigned the same high priority as patients with acute myocardial infarction or serious trauma, regardless of the severity of the neurological deficits.
Identification of Potential Stroke Victims

The Emergency Severity Index (ESI) is a triage tool for use in the emergency department. It is a five-level triage algorithm that provides clinically relevant stratification of patients into five groups (“1” being the most severe and “5” being the least) by determining acuity and resource needs. The system puts all stroke patients in the level 2 or “needs immediate assessment” category, the same as for an unstable trauma patient or a critical-care cardiac patient (Jauch et al., 2013).

A 5-level ESI triage algorithm. (Source: Adapted from Gilboy et al., 2011.)

This algorithm triages patients based on the severity of symptoms.

1. Level 1 represents a patient who has no pulse, may be intubated or unable to breathe on his or her own, and may be unresponsive to noxious stimuli (P/U on APVU scale) or to verbal commands. Immediate life-saving intervention such as resuscitation is required for Level 1.
2. Level 2 (where stroke patients should be placed) is a high-risk, emergency situation. The recommendation here is to imagine the hospital had “one last bed.” The stroke patient should get priority. This is true whether or not the patient is confused, lethargic, or disoriented and whether or not the patient is in pain.

**Time Sheet Documentation during Triage**

For patients with an acute onset of neurological signs, triage nurses complete the following:

1. A stroke recognition checklist
2. A time sheet documenting:
   - Time of onset of symptoms, or last time when the patient was symptom free
   - Time of the patient’s arrival in the ED
   - Time goal for the initial provider’s assessment (i.e., 10 minutes after the patient’s arrival at the ED)
   - Time goal for a completed CT scan (i.e., 25 minutes after the patient’s arrival)
   - Time windows for the rtPA treatment of eligible patients
     - ED goal (e.g., within 1 hour of the patient’s arrival)
     - 3-hour time window after onset of symptoms
     - 4.5-hour time window after onset of symptoms
     - 6-hour time window after onset of symptoms
   - Time goal for admission to a monitored bed (i.e., 3 hours after arrival) (Jauch et al., 2013; Cavallini et al., 2015)

The time sheet then follows the patient to keep providers, nurses, and technicians on schedule.

**Mobilization of the Hospital’s Stroke Team**

When a potential stroke patient has been identified, a stroke page is initiated from the incoming EMS vehicle or from the ED triage nurse. The stroke code team then reports to the ED, joins the ED receiving team, and begins the acute stroke protocol once the patient is medically stable.

The first parts of the stroke protocol include drawing blood and taking a medical history; these can be done immediately by the nurses, who should have standing orders. Next, the patient undergoes a selected physical examination and a complete neurological examination with a formal stroke assessment—the NIH Stroke Scale and, for patients
with a reduced level of consciousness, a Glasgow Coma Scale Score. In this time-limited evaluation stage, a chest X-ray is warranted only when needed for immediate decisions about heart or lung problems. Finally, all suspected stroke patients need cranial imaging.

**CASE**

Eleanor, a 62-year-old African American female patient, arrives to the emergency department accompanied by her daughter. Eleanor presents with sudden onset of left-eye blindness beginning 30 to 45 minutes ago while she was at home reading a magazine. Her daughter called 911 for immediate transport. Eleanor says it was as if “someone had dropped a gray curtain over my left eye” but that her vision is improving.

The nurse in the ED, Joan, asks the patient if she has had a headache, weakness, dizziness, tingling, fatigue, or slurred speech in the past. Beyond occasional headaches, Eleanor denies any of these symptoms and adds that this blindness has never happened to her before. Eleanor’s health history reveals that she has well-controlled type 2 diabetes and hypertension, with untreated hyperlipidemia that was recently diagnosed. Eleanor’s medications include metformin (Glucophage), 1000 mg, twice daily; lisinopril (Zestril), 5 mg, daily; and hydrochlorothiazide (Esidrix), 25 mg, daily. Eleanor was also on estrogen replacement therapy for eight years post-hysterectomy. Her pertinent family history includes a mother who had a cerebrovascular event at age 82 years.

Based on Eleanor’s symptoms, medical history, and family history, the nurse immediately consults with the ED physician and alerts the stroke team. The nurse also reassures Eleanor and her daughter that they were right to call 911.

**STABILIZATION OF COMORBID MEDICAL PROBLEMS**

Within 10 minutes of arrival, a general examination is done to identify other potential causes of the patient’s symptoms and coexisting comorbidities or issues that may impact the management of a stroke.

*Identify Medical Problems and Begin Treatment*

A quick but thorough examination is done to assess for circulation, airway, breathing, and vital signs and to medically stabilize any problems the patient may have in addition to the stroke.

- For oxygen saturation <92%, give O₂ via nasal cannula at 2 to 3 L/min.
- Establish IV access if not yet done. Patients eligible for rtPA therapy will need a minimum of two IV sites—one for IV fluids and/or IV medications and one dedicated to rtPA administration.
- Establish continuing cardiac monitoring.
- Hypoglycemia can mimic a stroke by causing focal neurological deficits.
Hyperglycemia is toxic to oxygen-starved but still-surviving brain cells and will worsen stroke damage. Either condition requires treatment.

- If the patient is hypoglycemic, dextrose should be administered.
- If blood glucose is >300 mg/dL, insulin should be administered.

- If the patient is alcoholic or malnourished, thiamine should be given.

- Hyperthermia (>37.6 °C or 99.6 °F) increases neurological deterioration. Acetaminophen may be administered or a cooling blanket may be utilized.

- Increased intracranial pressure can suppress the respiratory reflex and control mechanisms in a stroke victim, and intubation may be needed to ensure sufficient ventilation. Vomiting can be another consequence of increased intracranial pressure, and intubation can protect the lungs from aspiration.

- The patient should be kept on bed rest with the neck straight and the airway patent. Head position is decided on an individual basis. In general, keeping the head flat will maximize blood flow to the brain. Elevating the head 25 to 30 degrees is suggested for:
  - Suspected increased intracranial pressure
  - Aspiration risk
  - Airway obstruction risk
  - Chronic respiratory problems
  - Heart failure

(Jauch et al., 2013; Cavallini, 2015; Carl, 2016)

**STROKE DIAGNOSTIC STUDIES**

The two most essential laboratory tests for acute stroke patients are **blood sugar level** and **coagulation studies**, which can be done quickly using point-of-care-testing. Before using a thrombolytic agent (e.g., rtPA) to treat an ischemic stroke, the likelihood of inducing hemorrhaging must be assessed with, minimally, a platelet count and a prothrombin time or INR. A complete blood count, electrolytes, and basic chemistry panel are useful as baseline studies.

Additional laboratory testing is tailored to the individual patient. For certain patients, hepatic and renal function tests, lipid profile, toxicology screening, blood alcohol level, or pregnancy test will also be appropriate. In addition, in the case of an intracranial hemorrhage, blood typing and cross matching should be done if fresh frozen plasma may be needed to reverse a coagulopathy. The ED’s stroke protocol should explain how to determine whether any of these extra tests are necessary (Jauch, 2015a).

**MEDICAL HISTORY**

The medical history should include the patient’s chief complaint and the history of the present illness. **The most important piece of historical data, however, is the time of symptom onset.**
The history should include all symptoms the patient has experienced, as well as the time and sequence of each of them. Obtaining this history may require interviewing a family member and/or a witness.

A medical history for stroke patients includes a review of systems eliciting the following information:

- Hypertension
- Diabetes mellitus
- Tobacco use
- High cholesterol
- History of coronary artery disease, coronary artery bypass, or atrial fibrillation
- Recent trauma
- Recent illnesses
- Coagulopathies
- Illicit drug use (especially cocaine)
- Migraines
- Oral contraceptive use
- Current medications, asking specifically about insulin, oral hypoglycemia, and anticoagulants (e.g., Coumadin/warfarin)  
  (Jauch, 2015a)

The time and sequence (clinical course) of the patient’s symptoms are elicited in an effort to help determine the type of stroke that has occurred.

- An abrupt onset with maximal neurologic defect occurring immediately may indicate an embolic stroke.
- Rapid recovery also indicates possible embolic stroke.
- Fluctuating symptoms and a course that “stutters” along indicates a thrombotic stroke.
- Symptoms that evolve over hours or days can indicate a lacunar stroke.
- Large vessel ischemia evolves over a longer period of time.
- Symptoms that are progressive over minutes to hours and do not improve in the early phase point to intracerebral hemorrhage.
- Subarachnoid hemorrhage due to ruptured aneurysm has a very abrupt onset and focal neurologic deficits are not common.  
  (Cameron, 2013)
PATIENT EXAMINATION

Focused Physical Examination (with ECG)

The purpose of the focused general physical is to look for possible underlying causes of stroke in the patient.

- **Vital signs** are crucial, as blood pressure is often elevated in stroke.
  - Systolic pressure of >220 mm Hg may indicate intracerebral hemorrhage.
    Blood pressure fluctuations may indicate cardiac problems.
  - Stroke can decrease the respiratory drive or cause airway objections.
    Hypoventilation can cause cerebral vasodilation and increase intracranial pressure.
  - Elevated temperature may be caused by bleeding into the ventricles.

- Examination of the **pulses** may reveal absent radial, carotid, or lower extremity pulses, which could indicate a possible diagnosis of atherosclerosis with thrombosis or emboli.

- A **cardiac examination** including an ECG is vital, as patients with stroke, especially ischemic stroke, often have cardiac problems. The following may be the cause of stroke:
  - Atrial fibrillation
  - Myocardial infarction
  - Murmurs

- Examination of the **eyes:**
  - Fundal cholesterol crystals, white intravascular occlusions, or red clot emboli may indicate vessel occlusions due to emboli from atherosclerosis or blood clots.
  - Subhyaloid hemorrhage (beneath the vitreous membrane) may indicate subarachnoid hemorrhage.
  - Papilledema (swollen optic disc) indicates increased intracranial pressure.

- Examining the **head for trauma** for a cause for neurological symptoms:
  - Contusions or tongue lacerations could indicate a seizure.
  - Retro-orbital bruits may indicate atherosclerosis or intracranial vascular lesions.
• Examination of the **neck**:
  o Carotid bruits may signal atherosclerotic disease.
  o Jugular venous distension (JVD) may indicate heart failure.

• Examination of the **skin** may show signs of coagulation problems:
  o Jaundice
  o Ecchymosis
  o Purpura
  o Petechiae

**Neurological Exam**

The neurological examination should try to confirm the findings from the history and provide a quantifiable measurement of any neurologic deficits to be found over time. The goals of the neurological exam are to:

• Confirm the presence of a stroke syndrome
• Distinguish stroke from stroke mimics
• Establish a neurologic baseline should the patient’s condition improve or deteriorate
• Establish a stroke severity to assist in prognosis and therapeutic selection (Jauch, 2015a)

The essential components of the neurological examination include:

• Cranial nerves
• Motor function
• Sensory function
• Cerebellar function
• Gait
• Deep tendon reflexes
• Expressive and receptive language capabilities
• Mental status and level of consciousness
**Formal Stroke Assessment**

The AHA/ASA guidelines (Go et al., 2013; Sidney et al., 2013) recommend all potential stroke victims be assessed using the **NIH Stroke Scale (NIHSS)**. This is a measure of the severity of neurologic deficits and can be used to objectively monitor the improvement or deterioration of the stroke. The NIHSS scale is designed to be simple, valid, and reliable and can be administered consistently by physicians, nurses, or therapists.

Standardized stroke assessment tools do not replace a neurologic exam. Instead, the stroke scale is an efficient way to objectively determine the severity and possible location of the stroke. NIHSS scores are helpful in identifying patients who would likely benefit from fibrinolytic therapy and those at greater risk of hemorrhagic complications of fibrinolytic use.

The NIHSS focuses on six major areas of the neurologic examination: These include:

- Level of consciousness
- Visual function
- Motor function
- Cerebellar function
- Sensation and extinction (formerly known as neglect)
- Language

*(Jauch, 2015a)*

### NIH STROKE SCALE (NIHSS)

<table>
<thead>
<tr>
<th>Instructions</th>
<th>Scale</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level of consciousness (LOC) observed</strong></td>
<td>Alert</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Somnolent but arousable</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Stuporous but responds to deep stimulus</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Comatose-unresponsive</td>
<td>3</td>
</tr>
<tr>
<td><strong>LOC questions:</strong> What month is it? What is your age?</td>
<td>Answers both correctly</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Answers one correctly</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Answers both incorrectly</td>
<td>2</td>
</tr>
<tr>
<td><strong>LOC commands:</strong> Open and close eyes. Grip and release nonparetic hand.</td>
<td>Obey both correctly</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Obey one correctly</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Obey both incorrectly</td>
<td>2</td>
</tr>
<tr>
<td><strong>Best gaze:</strong> Follow my finger with your eyes.</td>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Partial gaze palsy</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Forced deviation</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Total gaze paresis not overcome by oculocephalic reflex (doll’s head maneuver)</td>
<td>3</td>
</tr>
<tr>
<td><strong>Visual fields</strong></td>
<td>No visual loss</td>
<td>0</td>
</tr>
<tr>
<td>--------------------</td>
<td>----------------</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td>Partial hemianopia</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Complete hemianopia</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Bilateral hemianopia</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Facial palsy:</strong> Show teeth. <strong>Raise eyebrows.</strong> Squeeze eyes shut.</th>
<th>Normal, symmetrical</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Minor, asymmetry on smiling</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Partial, total, or near total of lower face</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Complete in upper and lower face on one or both sides</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Motor arms:</strong> Extend arms (each arm tested and scored separately).</th>
<th>No drift downward</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Drift</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Drift with some effort against gravity</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Falls, no effort against gravity</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>No movement</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Motor legs:</strong> In supine position, raise leg 30 degrees (each leg tested and scored separately).</th>
<th>No drift downward</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Drift</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Drift with some effort against gravity</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Falls, no effort against gravity</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>No movement</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Cerebellar testing:</strong> Limb ataxia (finger-nose-finger and heel-shin tests on both sides).</th>
<th>Absent</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present in one limb</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>President in two limbs</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Sensory:</strong> Pinprick to face, arm, leg.</th>
<th>Normal</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Partial loss</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Severe loss</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Sensory:</strong> Extinction. Double simultaneous test.</th>
<th>No neglect</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Partial neglect</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Complete neglect</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Best language:</strong> Ask the patient to name items and describe pictures.</th>
<th>No aphasia</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild to moderate aphasia</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Severe aphasia</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Mute, global aphasia</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Dysarthria:</strong> Assess speech clarity to “mama, baseball, huckleberry, tip-top, fifty-fifty.”</th>
<th>Normal articulation</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild to moderate dysarthria</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Near to unintelligible or worse</td>
<td>2</td>
</tr>
</tbody>
</table>
For hemorrhagic strokes, another neurological assessment tool, the **Glasgow Coma Scale**, is used to describe the general level of consciousness in patients with traumatic brain injury or suspected hemorrhage stroke. Like the NIHSS, the GCS is not a diagnostic tool, and it does not replace the neurological exam.

Administering the scale takes 3 to 5 minutes and requires no special equipment. External stimuli are given to a patient, and the tester rates three neurological aspects of the patient’s response: eye opening, limb movement, and vocalization.

On the Glasgow Coma Scale, points are given for higher levels of response and consciousness. Final scores can range from 3 to 15, with lower scores indicating more severe neurological deficiency. (Note that this is the **reverse** of the NIHSS, in which higher scores indicate more severe deficits.)

<table>
<thead>
<tr>
<th>GLASGOW COMA SCALE (GCS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neurological Aspect</strong></td>
</tr>
<tr>
<td>Eye opening</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Motor response</td>
</tr>
<tr>
<td></td>
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<td></td>
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<tr>
<td></td>
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<tr>
<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Verbal response</td>
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<tr>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
</tbody>
</table>
DIAGNOSIS AND HYPOTHESIS FOR STROKE TYPE AND ETIOLOGY

As information accumulates, the stroke team builds evidence for the diagnosis of “stroke” or “nonstroke.” For likely strokes, the team will also be weighing the evidence for and against intracranial bleeding.

**Rule Out Stroke Mimics**

Approximately 74% of patients presenting with apparent acute stroke symptoms are ultimately diagnosed with stroke. This means that 26% had symptoms produced by stroke mimics. Making a prompt diagnosis requires ruling out a great number of mimics that include structural intracranial abnormalities, infection, syncope, vertigo, seizure, and migraine patterns as well as underlying psychiatric causes and demyelinating diseases.

The most common mimics are:

- Seizure and postictal paresis
- Syncope
- Sepsis (most commonly urinary tract infection)
- Functional (psychiatric disorders such as conversion disorder)
- Primary headache disorder (migraine)
- Space-occupying lesions (tumor, abscess)
- Metabolic (hypoglycemia, hyperglycemia, hyponatremia)
- Peripheral neuropathy (nerve compression, radiculopathy)
- Unilateral facial paralysis (Bell’s palsy)
- Drugs and alcohol
- Neurodegenerative disorders (e.g., multiple sclerosis, Guillain-Barré syndrome, optic neuritis)

It is also important to remember that acute infections can unmask or reactivate focal neurological deficits from previous strokes or TIAs. For patients having sustained a prior stroke, all potential focal neurological deficits should be considered within the context of
their medical history, past presenting symptoms, and subsequent residual deficit (Martel et al., 2015).

**Forming the Hypothesis**

As the stroke team learns details of the medical history and physical state of a patient, they formulate a hypothesis as to the type of stroke and its etiology based upon the following information obtained:

**History**

- Clinical course
- Patient demographics
- Hypertension
- Previous TIA
- Smoking
- Birth control pills
- Physical activity/sexual intercourse
- Recent head/neck trauma
- Associated symptoms
  - Fever
  - Infectious disease
  - Headache/seizures and/or nausea and vomiting
  - Decreased level of consciousness

**Physical examination**

-Absent pulses
- Cardiac findings
- Fundoscopic exam

**CRANIAL IMAGING TO CONFIRM DIAGNOSIS**

Because time is of primary importance, there should be a standing order for a cranial scan for all potential stroke patients. There should also be a plan for getting the scan read quickly. Cranial imaging should be completed within 25 minutes of the patient’s arrival at the ED, and the interpretation by the radiologist on call should be available within 20 minutes of the scan’s completion.
Imaging Studies

**Noncontrast computed tomography (NCCT)** of the head is the recommended initial diagnostic imaging tool in the hyperacute stage of stroke. NCCT has excellent sensitivity for detection of ischemic information; it is rapid, inexpensive, and widely available. The total scan time for a CT is approximately 5 minutes.

**Multimodal CT**, including CT perfusion and CT angiography, is capable of addressing all acute imaging needs:

- Ruling out thrombectomy candidates
- Ruling out hemorrhage
- Identifying large vessel occlusion
- Detecting infarct core and penumbra
- Assessing collateral flow

**CT perfusion** has become a critical tool in the selection of patients for thrombolytic treatment as well as increasing the accurate diagnosis of ischemic stroke by nonexpert readers four-fold compared to routine noncontrast CT. It allows both the core of the infarct to be identified as well as the surrounding penumbra that can potentially be salvaged.

**Magnetic resonance imaging (MRI)** is a major advance in neuroimaging of stroke, providing greater structural detail and recognition of early cerebral edema. Rapid MRI can take 10 to 15 minutes. Although the scan time difference between CT and MRI is small, there are a number of hindrances that can add delays of up to 18 to 30 minutes to MRI times. These may include availability of a scanner, screening for MRI safety, transporting the patient to a scanner that is often outside the ED, transferring the patient in and out of the scanner with MRI-compatible equipment, and positioning the patient, among others.

MRI is contraindicated in up to 20% of patients due for reasons including any electronic or metal implants. Other contraindications for MRIs are respiratory or hemodynamic instability, vomiting, agitation, impaired consciousness, or patient claustrophobia (Vo et al., 2015; Jauch, 2015a).

At this time, **diffusion weighted imaging (DWI)** is the best clinically available technique to identify hyperacute infarction. DWI can detect a new infarct within minutes of its occurrence. It provides information on the viability of brain tissue, showing image contrast that is dependent on the molecular motion of water, which may be substantially altered by disease. DW imaging time ranges from a few seconds to two minutes (Yo et al., 2015).
LUMBAR PUNCTURE (LP)

If an acute subarachnoid hemorrhage (SAH) is a possibility but it cannot be identified in the imaging results, lumbar puncture is indicated. SAH leads to blood in the cerebrospinal fluid (CSF) in less than 30 minutes. LP is most sensitive 12 hours after the onset of symptoms. LP findings can be negative in approximately 10% to 15% of patients with SAH. With a small hemorrhage, there may only be several hundred red blood cells per cc of fluid; nonetheless, even a few hundred blood cells per cc will make the normally crystal-clear CSF appear cloudy.

A common confounding factor is blood that has leaked into the CSF sample from vessels injured by the lumbar puncture needle (a “traumatic tap”). One indication that the blood probably came directly from the CSF is the finding that the blood count does not decrease in consecutive collecting tubes (Becske, 2015).

Other Diagnostic Procedures

Once a determination of the cause of a stroke is made, other diagnostic procedures may be required to aid in decision making for treatment.
**Carotid ultrasound** is a two-step procedure that uses sound waves to create detailed images of the buildup of plaque and spectral analysis to measure blood flow velocity in the carotid arteries. Doppler ultrasonography and pulse Doppler ultrasonography have been used to detect and determine the degree of stenosis (Silver, 2015a). In patients with subarachnoid hemorrhage, transcranial Doppler (TCD) ultrasonography is useful in detecting vasospasm of the intracranial arteries.

**Cerebral angiogram** gives detailed views of the arteries in the brain and neck. It is the definitive preoperative diagnostic tool for patients with intracranial aneurysm. In the hyperacute stroke setting, it is usually performed to document the site of occlusion and to evaluate collateralization to the area (Vinas, 2015).

**Echocardiogram** is used to evaluate cardioembolic stroke. Approximately 20% of ischemic strokes are considered cardioembolic. Transesophageal echocardiography has been shown to be a superior method of identification of most cardiac sources of emboli (Schneck, 2015).

**EARLY TREATMENT AND ACUTE STROKE MANAGEMENT**

**Ischemic Stroke Treatment**

For ischemic strokes, the goal is to preserve tissue in the penumbra, where perfusion, although decreased, remains sufficient to prevent further infarction. Attempts to establish revascularization includes fibrinolysis by the administration of intravenous recombinant tissue-type plasminogen activator (rtPA) and intra-arterial approaches.
It is recommended to treat patients with ischemic stroke with the fibrinolytic drug rtPA (alteplase). This is a time-limited treatment (see also box below):

- As of 2013, it is **recommended** that eligible patients with an acute ischemic stroke be given IV rtPA treatment if the drug can be administered **within 3 hours** of the onset of clearly defined stroke symptoms.
- IV rtPA is **suggested** if treatment can be started **between 3 and 4.5 hours** after the onset of symptoms.
- Generally, IV rtPA is **not suggested after 4.5 hours**, though this is often a decision based on the clinical situation (Jauch, 2016).

<table>
<thead>
<tr>
<th>TIME-LIMITED rtPA RECOMMENDATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time Since Symptom Onset</strong></td>
</tr>
<tr>
<td>Within 3 hours</td>
</tr>
<tr>
<td>3 to 4.5 hours</td>
</tr>
<tr>
<td>After 4.5 hours</td>
</tr>
</tbody>
</table>

Source: Jauch, 2016.

Certain patients to whom IV rtPA cannot be given may be eligible instead for catheter-administered intra-arterial rtPA. Other treatments for ischemic stroke are being tested, but none is yet recommended for widespread use later than 4.5 hours after the stroke symptoms appeared.

**FIBRINOLYSIS WITH rtPA**

A variety of interventional stroke treatments are actively being pursued, but the only FDA-approved interventional treatment recommended for general use for acute ischemic stroke is the thrombolytic drug rtPA.

For eligible stroke patients, IV rtPA can significantly improve outcomes. The odds of a good outcome following stroke are 75% higher for patients who receive alteplase within 3 hours of the onset of their symptoms compared with those who did not. Those who received alteplase within 3 to 4.5 hours had an increase of 26% for a good outcome. In those patients treated following a 4.5-hour delay, there was a nonsignificant increase of 15%.

Alteplase is most effective if given within 90 minutes of the onset of stroke symptoms, and those with NIHSS scores in the 7 to 12 range have better outcomes. The value of using rtPA is reduced but beneficial within 180 minutes, and the benefits still outweigh the risks at 270 minutes.
Limitations of intravenous rtPA are:

- Reperfusion dependent on available blood clotting substrate and time to lyse clot
- Stimulates platelet activation
- May not be as effective with larger and older clots
- Risk of hemorrhage
  (Broderick, 2016)

Trials on the use of ultrasound enhancement of fibrinolysis are currently underway, as are the use of combinations of fibrinolytic agents with anticoagulants and/or antiplatelet agents (Jauch, 2015a; Broderick, 2016).

**WHAT IS rtPA?**

*tPA* is the abbreviation for *tissue plasminogen activator*, a naturally occurring human enzyme. rtPA is tPA that has been made in the lab using recombinant DNA technology.

Tissue plasminogen activator is a protease that turns plasminogen into plasmin, which is a molecule that cuts apart the fibrin strands holding blood clots together.

The generic name for rtPA is alteplase and the brand names are Activase and Cathflo Activase. The drug is a white powder that is reconstituted in sterile water. Besides being used to treat acute ischemic stroke, rtPA is used to treat acute myocardial infarction.

**Eligibility for rtPA**

rtPA is used to treat an acute ischemic stroke, not a hemorrhagic stroke. Eligible patients should not have any risk factors for significant bleeding events: they cannot have had recent major surgery, myocardial infarction, stroke, or other internal injuries, and they must have normal clotting functions and a sufficient number of platelets. Additionally, the patient should not have significant hypertension (Jauch, 2015).

When the time of symptom onset is known, the stroke team has up to 4.5 hours to give a patient rtPA. However, when the stroke occurs while someone is asleep (known as a “wake-up” stroke) and the patient has no idea or is unable to relate a time, rtPA is not an option under current treatment guidelines (Fesmire et al., 2014).
CONDITIONS UNDER WHICH A PATIENT IS ELIGIBLE FOR rtPA

Stroke Status
- The patient has a diagnosis of ischemic stroke causing measurable neurological deficits
- The neurological signs are not clearing spontaneously
- The neurological signs are not minor and isolated
- The symptoms of stroke are not suggestive of subarachnoid hemorrhage
- There has not been a seizure with postictal residual neurological impairments
- CT does not show a multilobar infarction (hypodensity >1/3 cerebral hemisphere) or intracerebral hemorrhage
- The blood glucose concentration is >50 mg/dL (2.7 mmol/L)

Blood Vessel Status
- No head trauma or prior stroke in the previous 12 weeks
- No myocardial infarction in the previous 12 weeks
- No gastrointestinal or urinary tract hemorrhage in the previous 3 weeks
- No major surgery in the previous 2 weeks
- No arterial puncture at a noncompressible site in the previous 1 week
- No history of previous intracranial hemorrhage
- Blood pressure is not too high (specifically, systolic pressure is <185 mm Hg and diastolic pressure is <110 mm Hg)
- No evidence of active bleeding or acute trauma (e.g., a fracture) on examination

Thrombotic Status
- Not taking an oral anticoagulant, or
  - If anticoagulant is being taken, INR <1.7
  - If patient received heparin in previous 48 hours, their aPTT (activated partial thromboplastin time) must be in a normal range
- Platelet count >100,000 mm$^3$

Understanding of Risks/Benefits
- Patient or family understands the potential risks and benefits of treatment
- In the absence of the ability of the patient or a surrogate to provide information and consent, patient can be treated under the principle of presumption of consent

Source: Jauch, 2015; Fugate, 2015; Filho, 2016.
Administration of rtPA

The protocol for administering rtPA should be written, and the involved members of the stroke team should review it in advance. Treating an ischemic stroke with rtPA must be done promptly. Therefore, stroke EDs need electronic standing orders for the drug and an established procedure for quickly dispensing the drug from the pharmacy at any hour.

The 2013 American Stroke Association guidelines for early management of ischemic stroke state that informed patient consent for IV rtPA is indicated. Regulatory precedents in the United States and internationally support the use of IV rtPA in patients lacking capacity if alternative form of consent cannot be obtained within the treatment window. Data suggest that issues with consent are the most common reasons for delay in IV rtPA (Cohn, 2015).

INFORMED CONSENT OR PRESUMED CONSENT?

In life-threatening emergencies involving incapacitated patients without surrogates, clinicians may intervene without obtaining informed consent. This is based on the assumption that a reasonable person would consent to treatment under the circumstances. Controversy exists as to whether or not this rationale applies to the treatment of acute stroke with intravenous thrombolysis because this treatment is not life preserving but rather improves functional outcomes.

A recent survey of older adults showed that 76% of older adults would want thrombolysis for acute ischemic stroke, but nearly a quarter would not. Despite this controversy, professional organizations have recently endorsed the presumption of consent. It is recommended that policies must be informed by normative considerations for appropriate treatment such as the role of clinical judgment and the values of life and independence (Chiong et al., 2014).

A registered nurse administers rtPA and ensures:

- Vital signs and neurologic assessment have been completed
- CT scan has been completed and interpreted
- Inclusion/exclusion criteria have been met and stroke scale is completed
- Continuous ECG and SpO₂ monitoring is in place
- Appropriate lab studies are completed
- Patient’s identity has been verified according to institutional protocol
- rtPA dose is verified to the order with a second RN or physician
- All procedures that might cause bleeding (Foley catheters, NG tubes) are completed
- At least two large-bore IVs are in place
Initiating the infusion: The nurse makes certain that the rtPA is run by itself with no other drugs.

During administration:

- The dose is administered as ordered. The total dose is 0.9 mg/kg up to a maximum of 90 mg (i.e., all patients weighing >100 kg [220 lbs.] receive a total of 90 mg of drug).
- The first 10% of the dose is given as a bolus, and the remainder is delivered as a 60-minute infusion.
- Complete neurologic assessment and vital signs are taken every 15 minutes during the 60-minute infusion.
- The patient is monitored and the physician notified for adverse allergic reaction.
- Infusion is discontinued and an emergency CT scan and appropriate lab work obtained if the following signs of intracranial bleeding occur:
  - Acute hypertension
  - Severe headache
  - Nausea and/or vomiting

Post administration:

The rtPA infusion is completed in one hour. Vital signs should be obtained:

- Every 15 minutes for the next hour, then
- Every 30 minutes for 6 hours, then
- Every 6 hours for 16 hours

For the next 24 hours, all invasive procedures except for necessary venous blood draws or fasting blood sugars are avoided. The patient is monitored for blood in the urine, stool, emesis, or from puncture sites (Murnen & Kornack, 2014).

During the first 24 hours, blood pressure is maintained at <180/105 mm Hg if judged clinically necessary, no antiplatelet or anticoagulant drugs are given, and no arterial punctures are done. Likewise, intra-arterial catheters, nasogastric tubes, and indwelling bladder catheters are not inserted during the first 24 hours.

Risks and Complications

About 6% of patients have intracerebral hemorrhage associated with early worsening following rtPA. Half of these patients experience an altered final outcome as a result. About 10% have major early hemorrhage, but many occur in already infarcted areas and
do not alter final outcomes. Complications that occur less frequently include systemic hemorrhage, angioedema, and allergic reactions (Saver, 2015).

Clinical factors associated with an increased risk of ICH after alteplase infusion are:

- Increasing age
- Higher NIHSS score on admission
- Elevated blood pressure
- Elevated serum glucose concentration
- Pretreatment use of aspirin and clopidogrel combined or alone
- History of statin prescription
- History of hypertension
- Congestive heart failure
- Ischemic heart disease
- Atrial fibrillation
- Renal impairment
  (Karaszewski et al., 2015)

**Intra-Arterial Fibrinolysis with rtPA**

Normally, rtPA is given intravenously. Theoretically, a higher concentration can be delivered to the clot by injecting rtPA through an intra-arterial catheter placed near the clot, but initiating of intra-arterial administration requires a longer time and may mitigate some of this advantage. Intra-arterial thrombolysis has been the traditional approach for patients with stroke from basilar artery occlusion. Ongoing studies do not, however, support unequivocal superiority of intra-arterial fibrinolysis over intravenous fibrinolysis (Saver, 2015).

**TREATMENT WITH OTHER ANTITHROMBOTIC DRUGS**

A patient who has a stroke is at risk for another. Treatments or procedures to reduce the risk of a second stroke include:

- Antiplatelet therapy: aspirin
- Platelet aggregation inhibitors: clopidogel (Plavix), ticlopidine HCL (Ticlid)
- Anticoagulants: heparin, warfarin (Coumadin)

New oral agents demonstrate several advantages over warfarin, including a rapid onset of action, a broad therapeutic window, low interpatient variability, and minimal drug or dietary interactions.
These new agents are:

- Dabigitran (Pradaxa)
- Rivaroxaban (Xarelto)
- Apixaban (Eliquis)
- Endoxaban (Savaysa)

(Perez et al., 2013)

For acute ischemic stroke patients who are not receiving rtPA, IV heparin, or oral anticoagulants, daily aspirin (325 mg) on the first day followed by 150 mg to 325 mg/day thereafter is recommended, beginning within the first 48 hours. Antiplatelet therapy helps prevent new clots from developing but does not dissolve clots that are already present (Caplan, 2015).

Studies of anticoagulation with heparin or low molecular-weight heparin have not demonstrated significant benefits for most acute ischemic stroke patients. Anticoagulation cannot be used in patients with hemorrhagic stroke; and stroke patients with large infarctions, uncontrolled hypertension, or bleeding conditions should not be given full-dose anticoagulation treatment (Cruz-Flores, 2015).

(See also “Preventing Secondary Stroke” later in this course.)

**ENDOVASCULAR TREATMENT**

*Mechanical Thrombectomy*

In 2015 AHA/ASA (Powers et al., 2015) updated its guidelines on endovascular treatment using stent retrievers for acute ischemic strokes and strongly recommends its use in certain patients. The procedure is a mechanical thrombectomy in which a large blood clot is removed by a wire-caged device called a stent retriever threaded from the groin to the artery in the brain.

Guidelines state that patients should receive endovascular therapy with a stent retriever if they meet **all** the following criteria:

- Prestroke modified Rankin Scale (mRS) score of 0 to 1
- Acute ischemic stroke with receipt of IV rtPA within 4.5 hours of onset
- Causative occlusion of the internal carotid artery or proximately middle cerebral artery
- Age 18 years or older
- NIHSS score of >6
- ASPECT score of >6
- Treatment can be initiated (groin puncture) within 6 hours of symptom onset (Hughes, 2015; Badhiwala et al., 2015)
ASSESSMENT TOOLS FOR MEETING ENDOVASCULAR TREATMENT CRITERIA

**Modified Rankin Scale (mRS).** The mRS assesses disability in patients who have had a stroke and is compared over time to check for recovery and degree of continued disability. The mRS correlates with physiological indicators such as stroke type, lesion size, and neurologic impairment. A score of 0 to 4 indicates no disability; a score of 5 indicates disability requiring constant care for all needs; a score of 6 indicates death.

**Alberta Stroke Program Early CT Score (ASPECTS).** ASPECTS is a 10-point quantitative CT scan score that uses a reproducible grading system to assess early ischemic changes on pretreatment CT studies in patients with middle cerebral artery stroke. An ASPECTS score of less than or equal to 7 predicts worse functional outcome at three months as well as symptomatic hemorrhage.

Source: Hacking, 2016; Alberta Health Services, n.d..

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**Carotid Endarterectomy and Carotid Angioplasty and Stenting**

A **carotid endarterectomy (CEA)** is a surgical procedure done to open and clean a carotid artery with the goal of secondary stroke prevention. The highest risk period for recurrent stroke is within the first two weeks, so whenever possible, patients should be treated within this window. This procedure may be recommended for:

- Patients with a moderate (50%–79%) blockage of a carotid artery if experiencing symptoms of stroke or TIA
- Patient with severe (80% or more) blockage even if the patient has no symptoms (Chong, 2013)

**Carotid angioplasty and stenting (CAS)** involves the passage of a balloon into a narrowed artery to expand it, followed by implantation of screens called stents to open up and support the blood vessel. Currently, the Centers for Medicare and Medicaid Services have approved reimbursement for CAS only in the following patients:

- Those with high-grade stenosis greater than 70% who are considered to be at high risk for a carotid endarterectomy
- Those who are at high risk for a carotid endarterectomy and have asymptomatic carotid stenosis greater than 80% (Rodriguez, 2016)

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**BLOOD PRESSURE CONTROL IN ISCHEMIC STROKE**

Acute ischemic strokes usually present with elevated blood pressure, but this is not always an indication for aggressive treatment of the hypertension. After a stroke, some degree of hypertension may be needed to maintain adequate perfusion of the brain, although very high
blood pressure (systolic pressure >200 mm Hg) has been linked to higher mortality rates after stroke (Jauch, 2016).

There are no good data available to guide blood pressure management in the hyperacute phase of ischemic stroke (first 12 hours) when the penumbra is at risk for irreversible damage if the blood flow to the brain is reduced by lowering blood pressure (Filho, 2015).

Typically, blood pressure drops in the first 24 hours after acute stroke, whether or not the patient receives antihypertensive treatment. Currently it is the consensus recommendation that blood pressure be lowered only if the systolic blood pressure is greater than 220 mm Hg or the diastolic pressure is greater than 120 mm Hg. Treatment of hypertension in acute ischemic stroke may vary, depending on whether or not the patient is a candidate for fibrinolysis.

In those who are rtPA candidates, the drug of choice is IV labetalol (Trandate). One to two inches of transdermal nitropaste may also be used. Another drug option is IV nicardipine (Cardene). The goal of therapy for these patients is to reduce blood pressure by 15% to 25% in the first day, with continued blood pressure control throughout hospitalization.

For patients who are not candidates for rtPA and who have a systolic blood pressure greater than 220 mm Hg or a diastolic blood pressure greater than 120 mmHg, labetalol infusion is recommended. Alternatively, nicardipine may be used. The goal for these patients is to reduce blood pressure by 10% to 15%.

Control of hypotension should be managed in standard fashion using aggressive fluid resuscitation, a thorough search for the etiology, and, if needed, vasopressor support (Jauch, 2016).

**SUPPORTIVE TREATMENT**

Patients being treated for ischemic strokes require:

- Bed rest for the first 24 hours
- Head of bed below 30° (Avoid hypoperfusion in evolving infarcts; focially ischemic brain has impaired autoregulator capacity and may not compensate for changes in blood pressure.)
- Airway and ventilation maintenance
- Continuous hemodynamic monitoring and neurologic assessment
- NPO for the first 24 hours except for very mild or rapidly resolving deficits
- Bedside swallowing screening and assessment for dysphagia
- IV fluids with normal saline over the first 24 hours
- Symptomatic treatment of confusion, agitation, headache pain, nausea, and vomiting
- Referral for chest physiotherapy, if required
  (Jichici, 2015)

**Intracranial Hemorrhagic Stroke Treatment**

Management and treatment of intracranial hemorrhagic stroke depends on the type of intracranial hemorrhage that has occurred (intracerebral or subarachnoid) and the cause of the bleeding (e.g., hypertension, use of anticoagulant medications, head trauma, blood vessel malformation). No effective targeted therapy for hemorrhagic stroke exists yet, and hemorrhagic stroke is treated with medical management that includes:

- Basic life support measures
- Reversal of anticoagulants
- Seizure control
- Blood pressure control
- Intracranial pressure (ICP) control
  (Becske, 2015; Caplan, 2015; Hemphill et al., 2015)

**VENTILATION AND OXYGENATION**

Endotracheal intubation may be required for patients with a decreased level of consciousness and poor airway protection, using neuromuscular blockade or sedative-hypnotics to allow for rapid return of motor control and neurologic assessment. Etomidate (Amidate) is favored for induction when elevated blood pressure is a concern.

**REVERSAL OF ANTICOAGULANTS**

Anticoagulation-associated intracranial hemorrhage has a high morbidity and mortality rate. Over half of these patients die within 30 days. The necessity of reversing warfarin anticoagulation is a medical emergency. Reversals must be accomplished as quickly as possible to prevent further expansion of the hematoma. Options for reversal therapy include:

- Intravenous vitamin K
- Fresh-frozen plasma (FFP)
- Prothrombin complex concentrate (PCC) to treat cerebral hemorrhages related to warfarin therapy
- Recombinant factor 7 for hemorrhages related to the new oral anticoagulants
- Hemodialysis for patients who are taking dabigatran
- Protamine to treat patients with hemorrhage associated with heparin therapy
  (Liebeskind, 2016; Alberts, 2015; Hemphill et al., 2015)
Vitamin K requires more than six hours to normalize the INRA and should be given along with either FFP or PCC. FFP is the standard of care in the United States. It does, however, require a large-volume infusion. PCC involves a smaller-volume infusion but is associated with high rates of thrombotic complications.

Patients receiving heparin who develop a hemorrhagic stroke should have anticoagulation reversed immediately with protamine. Those patients with severe deficiency of a specific coagulation factor should receive factor replacement therapy.

Management after a hemorrhagic stroke is most often decided on a case-by-case basis, though antithrombotics, including antiplatelet and anticoagulant agents, should be discontinued after an acute intracranial hemorrhage (Liebeskind, 2016).

**SEIZURE CONTROL**

In up to 28% of patients with intracranial hemorrhage, seizures are often nonconvulsive. In those patients whose mental status is depressed out of proportion to the degree of brain injury, continuous EEG monitoring should be considered.

Guidelines recommend that patients with clinical seizures or with EEG seizure activity accompanied by changes in mental status should receive antiepileptic medication. For rapid control of seizures, a benzodiazepine (lorazepam, diazepam) should be given, followed by phenytoin or fosphenytoin (Cerebyx) loading for longer-term control (Liebeskind, 2016).

**BLOOD PRESSURE CONTROL IN HEMORRHAGIC STROKE**

Blood pressure control management is more controversial. Excessively high BP increases the risk of bleeding, and low BP increases the risk of cerebral hypoperfusion (Liebeskind, 2016).

Intensive reduction of blood pressure early in the treatment of hemorrhagic stroke appears to lessen the absolute growth of hematomas. This is particularly true in patients who have received antithrombotic therapy.

Medications used in the treatment of elevated blood pressure are beta blockers (e.g., labetalol [Trandate]) and angiotensin-converting enzymes inhibitors (e.g., enalapril [Vasotec]). In refractory cases, nicardipine (Cardene) and hydralazine (Apresoline) are used. The AHA/ASA recommends that if systolic blood pressure is over 200 mm Hg or mean arterial pressure (MAP) is over 150 mm Hg, the aggressive intravenous medication should be administered, checking blood pressure every five minutes (Liebeskind, 2016).

**INTRACRANIAL PRESSURE CONTROL**

Elevated intracranial pressure can be the result of the hematoma, the surrounding edema, or both. To improve jugular venous outflow and lower intracranial pressure, the head of the bed should be elevated to 30° and the head should be maintained at midline and not turned to either side. The patient may require analgesia (morphine or alfentanil [Afentna]) and sedation (propofol) to assist
with this recommendation. Dehydrating agents such as mannitol are sometimes administered, with the goal of making the blood plasma hyperosmolar (300 to 310 mOsm/L).

Hyperventilation is not recommended, as its effect is transient. It also decreases cerebral blood flow and may result in rebound elevated intracranial pressure (Liebeskind, 2016).

SURGICAL INTERVENTIONS

Surgical interventions such as early hematoma evacuation is a potential treatment for hemorrhagic stroke, but this remains controversial and is not recommended for the majority of patients.

There is evidence, however, of improved outcome with this intervention if any of the following apply:

- Surgery is undertaken within eight hours of stroke
- Volume of the hematoma is 20 ml to 50 ml
- Glasgow Coma Score is 9 to 12
- Patient age is 50 to 59 years
  (Liebeskind, 2015)

Surgical treatment, however, may be recommended to prevent or stop bleeding or reduce the pressure inside the skull. The procedure has shown improved outcome for those with a large or expanding cerebellar hemorrhage that is causing brainstem compression or for relieving hydrocephalus. For patients with a large intracerebral hemorrhage that is causing coma, shift, and increased IP, the recommendation is decompressive hemicraniotomy and hemicraniectomy (Alberts, 2015).

**Aneurysm Treatment**

There are two types of treatment for aneurysm: surgical clipping and endovascular coiling.

**Surgical clipping** is a procedure done to close off an aneurysm to prevent rebleeding. A portion of the skull is removed to access the aneurysm and a metal clip is placed on the neck of the aneurysm to stop blood flow to it. Following, the skull portion is replaced.

**Coil embolization** is a less-invasive procedure than clipping. During this procedure a tiny coil is advanced through an artery in the groin to the aneurysm, filling the area with a coil. A blood clot forms within the coil, blocking blood flow to the aneurysm and preventing it from rupturing again (Mayo Clinic, 2015; Caplan, 2015).
**Arteriovenous Malformation (AVM) Treatment**

Surgery is the most common treatment for AVM and is most effective with more easily accessible lesions that are of smaller size. There are three potential surgical treatment options for brain AVM whose main goal is to prevent hemorrhage: surgical resection, endovascular embolization, and stereotactic radiosurgery.

**Surgical resection** is the removal of the AVM via conventional brain surgery. Using a high-powered microscope, the AVM is sealed off with special clips and carefully removed from the surrounding brain tissue. Resection is usually done if the AVM can be removed with little risk of hemorrhage or seizures.

**Endovascular embolization** is a procedure involving the insertion of a long, thin catheter through a groin artery and threaded through blood vessels to the brain using X-ray imaging. The catheter is positioned into one of the arteries feeding the AVM and an immobilizing agent is injected, such as quick-acting acrylate glue, thrombus-inducing coils (e.g., microcoils), or other materials, to block the artery and reduce blood flow to the AVM. This procedure is less invasive than surgical resection and is frequently used prior to it to make the procedure safer by reducing the size of the AVM or the likelihood of bleeding.

**Stereotactic radiosurgery (SRS)** is a treatment that uses proton beam, linear accelerator, or gamma knife methods to precisely deliver a high dose of radiation to destroy the AVM. SRS directs many highly targeted radiation beams at the AVM to damage the blood vessels and cause scarring. These scarred AVM blood vessels then slowly clot off in one to three years following treatment (Mayo Clinic, 2014; Sen, 2014).

**SUPPORTIVE TREATMENT**

Patients being treated for hemorrhagic stroke require:

- Constant hemodynamic monitoring in an ICU
- Frequent neurologic assessment
- Bed rest with sedation and head of bed elevated to 20° to 30° (for subarachnoid hemorrhage), strict bed rest until etiology of hemorrhage is determined
- Cautious use of sedatives and analgesics due to potential to mask neurologic findings
- Pain control for headache
- Temperature control (elevated temperature can increase the degree of ischemic damage; maintain normal temperature using acetaminophen PO/PR; consider cooling devices)
- Maintaining hydration with intravenous IV normal saline
- Correcting electrolyte deficits

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Correcting acid-base disorders
Correcting hyperglycemia to less than 140 dL by instituting insulin therapy
Continuous ICP using direct measurement
Hypertension treatment with IV medication
Antacids to prevent associated gastric ulcers
(Domocmat 2014; Liebeskind, 2016)

SUBARACHNOID HEMORRHAGES

For patients who have had a subarachnoid hemorrhage, the medical management includes supportive care and is directed toward preventing and treating the common complications of SAH.

Vasospasm

In the majority of patients, intravascular volume becomes depleted in the days following a subarachnoid hemorrhage, and this greatly increases the chances of an ischemic infarction from vasospasm, a major complication of SAH. Fluids are given to maintain an above-normal circulating blood volume and central venous pressure. Nimodipine, the best-known and most widely used calcium channel blocker, may be used to dilate vessels (Becske, 2015; Hackin et al., 2016).

Hydrocephalus

Subarachoid hemorrhage causes one third of hydrocephalus cases. Normal pressure hydrocephalus (NPH) is a form of communicating hydrocephalus resulting from an aneurysm rupture. It is an abnormal buildup of CSF in the brain’s ventricle or cavities and occurs if the normal flow of CSF throughout the brain and spinal cord is blocked in some way.

Hydrocephalus is treated by lumbar puncture or external ventricular drainage, depending on the severity of the neurologic dysfunction or CT scan findings. Rapid lowering of ICP during intraventricular catheter placement is associated with a high risk of rebleeding and should be avoided. Periodically, resolution of hydrocephalus may be assessed by blocking CSF drainage while monitoring ICP (Lowth, 2015; Becske, 2015).
PREVENTING AND MANAGING COMPLICATIONS

In addition to acute treatment for ischemic strokes and intracerebral and subarachnoid hemorrhages, efforts are necessary to prevent and manage specific complications that can arise in the ICU after the acute treatment phase. **Within the first 24 hours of the onset of symptoms, many acute stroke patients will need intensive care** in a unit staffed by ICU nurses who are also trained to recognize and manage intracranial complications.

Besides neurological deterioration, non-neurological problems are frequent, and the stroke patient may face:

- Myocardial infarction
- Heart failure
- Aspiration pneumonia
- Pulmonary embolism

Watchful monitoring and quick reaction to developing complications are the bases of effective acute care for stroke patients (Liebeskind, 2015; Domocmat, 2014; Becski, 2015).

The Stroke ICU

Careful monitoring is the key to optimal stroke management. A significant number of stroke patients will deteriorate within the first 24 hours post-stroke. Because strokes also often leave victims in a medically unstable condition, acute stroke patients are monitored during their first 24 hours in an ICU with a nurse-to-patient ratio of 1:2. Even stroke patients with minor symptoms but with no radiological evidence of a stroke are typically monitored in a stroke ICU (or an ED observation unit) for 6, 12, or 23 hours, depending on the patient’s condition.

Stroke patients who may be eligible for fibrinolytic treatment are first channeled into the rtPA treatment protocol, while stroke patients with symptoms suggesting the need for neurosurgical intervention are first channeled into the neurosurgical evaluation protocol. However, the end station for both protocols is also typically the stroke ICU.

Besides monitoring the patient’s neurological functioning, ICU care aims to keep the patient’s physiology stable and to prevent or treat additional medical complications. Meanwhile, during the ICU monitoring, the stroke team physicians work to establish the specific cause of the stroke and begin to plan a strategy to avoid reoccurrences.

Nursing in the Stroke ICU

Nurses take the lead in ICU care by implementing a **clinical pathway** or care map. Clinical pathways are standardized, evidence-based, multidisciplinary management plans that identify an appropriate sequence of clinical interventions, timeframes, milestones, and expected outcomes for a homogenous patient group. Representatives from each discipline should take part in
determining the content of the written pathway or protocol (Ellmers, 2015). Nurses also develop an individualized nursing care plan that is used to outline the patient’s nursing care on a daily basis.

**NURSES IN A COMPREHENSIVE STROKE CENTER (CSC)**

In their “Recommendations for Comprehensive Stroke Centers,” the Brain Attack Coalition offers these guidelines for staffing a stroke unit:

High-quality nursing care is a key factor in determining patient outcomes after a stroke. The majority of nurses caring for stroke patients in an ICU, stroke unit, or ward should be registered nurses. The nurses in a CSC should be familiar with standard neurologic assessments and scales, stroke protocols, care maps, ongoing research projects, and new patient-care techniques related to stroke.

Nurses who care primarily for stroke patients should attend training sessions sponsored by the CSC (i.e., in-services, seminars, specialized lectures) three times per year. Such nurses should participate in 10 hours of continuing education units or other educational programs annually that are related to or focused on cerebrovascular disease. Each nurse should have a file that documents his/her participation in the above activities. It is suggested that each CSC nurse (stroke unit or ICU) attend one national or regional meeting every other year that focuses on some aspect of cerebrovascular disease.

An advanced practice nurse (APN) is a vital team member involved in several important aspects of a CSC, such as patient care, care maps, research activities, stroke registries, educational programs, and quality assurance. The designation of APN could include a nurse practitioner, master’s-prepared clinical nurse specialist, or American Board of Neuroscience Nurses–certified nurse. It is recommended that a CSC have one APN (or similar personnel) to implement and coordinate [care under the various stroke protocols].

Source: Alberts et al., 2011.

**Monitoring Neurological Functioning**

The key to managing complications in the stroke ICU is recognizing them quickly. The deterioration in a patient’s neurological status is always a signal to search quickly for a complication.

Stroke ICU nurses are characterized by their experience in performing neurological function assessments. During the first 24 hours, acute stroke patients need a neurological assessment at least every four hours. Stroke assessments are usually made along with the check of vital signs (pulse, blood pressure, temperature, oxygen saturation, blood glucose, and respiratory pattern).
(A sample flowchart for a nursing neurological assessment can be found online; see “Resources” at the end of this course).

When first admitting an acute-care stroke patient to the ICU, the nurse conducts a full neurological assessment and determines the patient’s baseline NIHSS and Glasgow Coma Scale scores (see “Neurological Assessments” above). Thereafter, stroke protocols often recommend only a brief neurological examination unless some neurological deterioration is detected. Deterioration of neurological functioning has been defined as an increase of two or more points on the NIHSS score within 24 hours (Siegler, 2016).

**Acute Complications**

A stroke ICU has plans in place for the most common medical complications. Members of the ICU staff will always have different levels of expertise, but written protocols and standardized stroke orders ensure that the best care can always be given without delay and with few mistakes.

**ISCHEMIC BRAIN SWELLING**

Injured brain tissue swells from edema, and sufficient swelling will push the brain against the skull or nondispensable edges of the dura. In these situations, the brainstem is often squeezed, and the patient will show signs of **cerebral herniation**. Cerebral herniation should be suspected when new neurological signs include both cranial nerve problems (especially loss or reduction of pupillary responses) and peripheral motor deficits. Brain herniation is a life-threatening emergency.

Edema is an occasional consequence of any ischemic stroke, but brain edema is most likely when ischemic strokes involve occlusions of the MCA or multilobar and cerebellar infarcts. Decompression craniectomy is a necessary treatment option in many patients. Excluding patients with cerebellar infarction, serious episodes of brain edema typically occur three to five days after an acute ischemic stroke rather than in the first 24 hours (Wijdicks et al., 2014; Lutsep, 2013).

**WATCH LIST FOR BRAIN SWELLING**

Besides the particular ischemic stroke symptoms, other factors that put a patient on the watch list for serious brain swelling include:

- A history of hypertension or heart failure
- An elevated white blood cell count
- >50% MCA hypodensity in head images
- Ischemic changes beyond the territory of the MCA
- Currently on mechanical ventilation

Source: Lutsep, 2013.
If brain edema is suspected, steps to reduce the swelling include:

- Raise the head of the bed to 20 to 30 degrees
- Restrict hypo-osmolar fluids
- Correct hypoxemia and hypercarbia
- Reduce any increases in body temperature
- Avoid vasodilators

### INCREASED INTRACRANIAL PRESSURE

Both ischemic and hemorrhagic strokes sometimes increase intracranial pressure indirectly as a result of brain edema. Hemorrhagic strokes can also increase intracranial pressure directly by adding extravascular blood to the restricted intracranial space.

During a stroke, an increase in intracranial pressure further reduces cerebral perfusion, which can cause global neurological dysfunction and additional ischemia. Increased intracranial pressure can also cause lethal brainstem compression.

Clinically, elevated ICP presents as headache, vomiting, and a decreased level of consciousness. Papilledema can be seen in a funduscopic exam, and sometimes there is periorbital bruising. The appearance of **Cushing’s triad**—bradycardia, respiratory depression, and hypertension—is an especially ominous sign.

Treatments for increased ICP include positioning the head of the bed at a 20- to 30-degree angle, controlling pain (e.g., with morphine or alfentanil), aggressively treating fever with acetaminophen and mechanical cooling, and avoiding techniques and situations that increase intrathoracic pressure. (Moving some stroke patients to an upright posture will worsen their neurological status, so position changes must be done cautiously.)

Other measures to lower ICP include reducing the extravascular fluid volume with intravenous mannitol, inducing respiratory alkalosis with forced hyperventilation, sedation (e.g., barbiturates or propofol), avoiding hypotension, directly draining some CSF, or performing a craniotomy to mechanically decompress the intracranial space (Becske, 2015).

### INTRACRANIAL REBLEEDING

Another cause of deteriorating neurological functioning in the stroke ICU is additional intracranial bleeding. This problem can be recognized using brain imaging, usually by CT scan.

- **Ischemic strokes.** Hemorrhage transformation (HT) is estimated to occur in over half of all cerebral infarcts at some state. Cardioembolic strokes, especially large ones, are more likely to undergo hemorrhagic transformation than atherothrombotic strokes. HT can occur spontaneously but is especially common after thrombolytic therapy. The risk for HT development increases dramatically when massive cerebral infarction is present. At
the present time, there are no evidence-based guidelines for the treatment of hemorrhage transformation post rtPA (Chong, 2014; Zhang et al., 2014; Yuranga & Gillard, 2016).

- **Intracerebral hemorrhages.** Rebleeding within the first 24 to 48 hours is the leading cause of death in persons surviving the initial bleed. Approximately 70% of patients who rebleed will die. Onset of a rebleed is usually accompanied by a sudden severe headache, which is often associated with severe nausea and vomiting, a decrease in or loss of consciousness, and new neurological deficits. Surgical or endovascular interventions are most effective. Prevention of recurrent hemorrhage, especially in a patient with hypertensive hemorrhage, is to aggressively control blood pressure to normotension. Blood pressure should be monitored and treated, with a goal SBP of under 160 when ICP is not elevated (BAF, 2016; Chong, 2013).

- **Subarachnoid hemorrhages.** The most dreaded early complication of SAH is rebleeding, the greatest risk being within the first 24 hours of rupture. Overall mortality rate from rebleeding is reported to be a high as 78%. Risk factors include high systolic pressure and presence of an intracerebral or intraventricular hematoma. Measures to prevent rebleeding include bed rest in a quiet, darkened room; analgesia; and sedation. Stool softeners are given to prevent Valsavla maneuvers with resultant peaks in SBP and ICP. The only effective treatment is prevention by obliterating the ruptured aneurysm, which can be clipped surgically or occluded endovascularly with a coil (Becske, 2015).

**SEIZURES**

After a stroke, seizure activity has been seen in between 2% to 23% of all patients within the first days after stroke. They are usually focal but may be generalized. Although primary prophylaxis for post-stroke seizures is not indicated, secondary prevention of subsequent seizures with standardized antiepileptic therapy is recommended (Jauch, 2015).

**PULMONARY COMPLICATIONS**

**Pneumonia**

Pneumonia, one of the most common respiratory complications of acute stroke, occurs in approximately 5% to 9% of stroke patients. Pneumonia incidence is higher in patients with acute ischemic stroke and in those who require nasogastric tube feeding. Aspiration is the cause of close to 60% of post-stroke pneumonia, usually due to dysphagia.

Measures to prevent aspiration keeping the patient NPO initially and subsequently modifying diet for those who have a persistent dysphagia.

The patient should be screened on admission for swallowing problems using a formal screening protocol. Other interventions may include positioning, medications, oral hygiene, tube feeding, and influenza and pneumococcal vaccines (File, 2015; Chalela, 2016).
Abnormal Breathing Patterns

An acute stroke patient commonly has periods of abnormal breathing, especially when the patient has decreased consciousness or a large or serious stroke. Tachypneic (fast breathing) patterns can be a problem if they lower blood levels of carbon dioxide (hypocapnia), thereby reducing cerebral perfusion. Other more common abnormal respiratory patterns, however, do not signal impending neurological deterioration.

Abnormal breathing patterns occur in approximately 60% of patients with neurologic disorders, including strokes. The underlying causes are cardiac and respiratory abnormalities.

- **Periodic breathing.** Approximately 25% of stroke patients have episodes of patterned breathing. The recurrent pattern usually alternates a set of shallow breaths with a set of deep breaths. Patients with subarachnoid hemorrhage are especially prone to developing periodic breathing.

- **Cheyne-Stokes respiration** is known to occur with unilateral hemispheric and brainstem infarcts. Hypocapnia is almost always present and may require treatment. During Cheyne-Stokes respiration, a patient stops breathing temporarily. When starting to breathe again, the patient takes increasingly deeper breaths. After a peak, the breaths become shallower and shallower until breathing again stops. The cycle then repeats. Cheyne-Stokes respiration is usually a reflection of underlying heart or lung problems; when it occurs, it is a warning that the patient may be hypocapnic or hypoxemic (Chalela, 2016).

Need for Mechanical Ventilation

Self-regulated breathing can be a problem for stroke victims, especially patients with hemorrhagic stroke or damage to the brainstem. Patients with breathing dysfunction usually have impaired consciousness or impaired airway reflexes, and an endotracheal tube is inserted if the patient’s protective airway mechanisms have been compromised.

Patients who develop aspiration pneumonia, pulmonary edema, stupor with reduced respiratory reflexes, or seizures are likely to require mechanical ventilation. The need for endotracheal intubation is a poor sign; morbidity and mortality in patients requiring intubation following acute stroke is very high (71%–76%) (Chalela, 2016).

Decreased Oxygenation

Ischemia from poor oxygenation of brain tissue is a major cause of the neurological deficits of a stroke, and longer periods of oxygen deprivation produce more extensive and irreversible damage. Therefore, to save brain tissue, it is critical to maintain normal blood oxygen saturation. Currently, there is no evidence that either supplemental or hyperbaric oxygen is helpful for stroke patients who already have normal blood oxygen saturation levels.
In the ICU, oxygen saturation is monitored continuously. Hypoxemia ≤92% is treated with supplemental oxygen at 2 L/min to 4 L/min. **Continuous pulse oximetry** is required because patients can be hypoxemic without showing clinical symptoms (Chalela, 2016; Mecham, 2016).

The appearance of hypoxemia should also alert the nurse to check:

- Airway patency
- Bed positioning
- Level of consciousness
- Lung sounds

If hypoxemia persists on supplemental oxygen, then blood gases and a chest film should be obtained.

**Neurogenic Pulmonary Edema**

Neurogenic pulmonary edema (NPE) most often develops abruptly and progresses quickly after a stroke occurs. NPE is an increase in interstitial and alveolar fluid that can occur following subarachnoid hemorrhage. The patient presents with dyspnea, tachycardia, hypertension, and bilateral rales. The condition most often resolves spontaneously but can be fatal in severe cases.

Supportive management and supplemental oxygen are required, along with treatment of the underlying condition. Episodes of NPE are well tolerated and usually resolve within 48 to 72 hours (Wemple et al., 2015; Chalela, 2016).

**CARDIOVASCULAR COMPLICATIONS**

**Cardiac Problems**

Strokes and heart problems are frequent companions. Stroke patients often present with existing cardiac problems. Strokes can also be the cause of such heart problems as arrhythmias and myocardial infarctions (MI). In a stroke patient, one must actively search for these problems, because myocardial infarctions concurrent with a stroke are often silent. Therefore, the initial evaluation of acute stroke patients includes a cardiac exam, an ECG, and blood tests for cardiac markers (in particular, an elevated troponin T, which is a specific marker of myocardial necrosis).

Options for treatment of acute MI are limited in the setting of acute ischemic stroke. MI is a contraindication to thrombolysis, and thrombolysis in contraindicated in MI.
Following a stroke, particularly a subarachnoid hemorrhage, neurogenic cardiac damage may become evident. Neurogenic cardiac damage may be due to underlying coronary disease, but this may not be the only mechanism, as it can occur in patients with subarachnoid hemorrhage who are often young and do not have underlying heart disease. Neurogenic cardiac damage can be detected by ECG abnormalities, elevated troponin levels, and evidence of left ventricular dysfunction (Chalela & Jacobs, 2016).

**Hypertension**

Ideally, ischemic stroke patients will have their blood pressures maintained at a systolic pressure of about 180 mm Hg and a diastolic pressure of 105 to 110 mm Hg during the first 24 hours. Higher blood pressures are treated cautiously. The temptation to immediately reduce high blood pressure should be tempered by two observations:

- Some degree of hypertension is often needed to maintain adequate cerebral perfusion after a stroke.
- In many patients, the acutely elevated blood pressure of a stroke will spontaneously decline during the first 24 hours.

Before using antihypertensive drugs to treat a patient’s high blood pressure, nurses should consider remedying other factors that may be elevating the blood pressure. Pain, nausea, a full bladder, or a loud environment can all raise a patient’s blood pressure. Intracranial problems, such as increased bleeding, can also raise blood pressure (Jauch, 2016).

**Venous Thromboembolism**

Venous thromboembolism (VTE) includes deep vein thrombosis (DVT) and pulmonary embolism (PE) and can be life threatening. Pulmonary embolism accounts for 13% to 25% of early deaths following stroke and is the most common cause of death two to four weeks post-stroke onset.

DVT may develop as early as 24 to 48 hours after stroke onset, and peak of incident ranges from two to seven days. Deep vein thrombosis is a serious issue, as it may lead to pulmonary embolism. The rate of DVT development is higher in patients of advanced age, in those with a high stroke severity, and those with hemiparesis or immobility.

The best options for prevention of VTE for patients with ischemic stroke include anticoagulants, aspirin, and intermittent pneumatic compression devices, preferably thigh-length for patients within 72 hours of onset whose mobility is restricted (Filho, 2015).

The safety of using antithrombotic therapy in patients with acute intracranial hemorrhage has not yet been adequately established. Intermittent pneumatic compression is the
recommended option for these patients, and subcutaneous heparin may be considered (Wijdicks, 2014).

OTHER COMMON PROBLEMS

Other complications that occur frequently in ICU stroke patients include fevers, hyperglycemia, dysphagia, and infections.

Fever

Hyperthermia accelerates ischemic neuronal injury. A high body temperature in the first 12 to 24 hours after the onset of stroke has been associated with poor functional outcome. Treatment of fever should be directed at its underlying cause, which in most cases is infection:

- Patients who have been receiving mechanical ventilation for more than 48 hours may develop a fever accompanied by new or progressive pulmonary infiltrate leukocytosis and purulent tracheobronchial secretions.
- Intravascular catheter-related infection may present with fever but no localized signs or symptoms. Other presentations may include purulent drainage or cellulitis at the insertion site.
- Urinary catheter-related infections may present with signs and symptoms of cystitis, pyelonephritis, or urosepsis.
- Intracranial pressure monitor catheter may be another source of infection.

All infections have the potential to progress to sepsis and septic shock. It is recommended that all patients who develop a new fever be treated with empiric antibiotics if they are deteriorating. There is conflicting data, however, about the use of antipyretics and cooling devices. Oral antipyretics are only marginally effective in lowering elevated body temperature and may have unintended adverse consequences; it is recommended that when fever is impairing management and may be detrimental to the outcome, they be considered (Jauch, 2015; MacLaren & Spelman, 2016).

Hyperglycemia

Both hyperglycemia and hypoglycemia are associated with increased brain injury after an acute stroke. Hyperglycemia worsens outcome and increases the rate of mortality from stroke, which is thought to be due to a pro-constrictive, pro-thrombotic and pro-inflammatory state resulting in harm to endothelial cells and the vascular tree. Post-stroke hyperglycemia is prevalent (almost 2 in every 3) in acute ischemic stroke patients. The exact mechanism for this is unknown, but it may be due to impaired recanalization, decreased perfusion, increased reperfusion injury, or cerebral lactic acidosis.
Despite the poor outcomes, there is no agreed-upon specific management. Due to complicating effects of feeding, patients with acute stroke and hyperglycemia are often kept NPO. U.S. stroke guidelines recommend treatment with subcutaneous sliding-scale insulin, requiring monitoring every 6 hours. If refractory, IV insulin is recommended, which requires rigorous hourly monitoring since IV insulin increases the risk of hypoglycemia. There are no guidelines for the duration of insulin therapy, but 48 hours has been found to be sufficient and longer than 48 hours unnecessary (Mandava, 2015; Sulaiman et al., 2014).

**Dysphagia**

A common complication and major risk factor for aspiration pneumonia in stroke patients is oropharyngeal dysphagia. An estimated 40% to 50% of stroke patients with dysphagia aspirate, and up to 70% experience silent aspiration. Prevention of aspiration includes keeping the patient NPO and assessing the patient’s swallowing function. Such tests are associated with a significant decrease in the risk for aspiration pneumonia.

Video-fluoroscopy and fiberoptic endoscopic evaluation of swallowing (FEES) are considered the gold standard for evaluating swallowing function. However, both are invasive and require trained staff.

Bedside tests to screen are useful but not completely accurate. They can, however, determine whether an evaluation by a speech-language pathologist is needed, whether the patient should be taken off NPO status, and whether a nutritional consult or IV hydration is needed.

Two such screening tools recommended by AHA/ASA are:

- **Toronto Bedside Swallowing Screening Test**, which assesses voice quality, tongue movement, and ability to manage water by teaspoon or cup. The test takes 10 minutes to perform and requires four hours of training to be certified in its use (McCullough & Martino, 2013).

- The **3-ounce (90 cc) Water Swallow Challenge**, in which 3 ounces of water are given to the patient to drink uninterrupted from a cup. The patient is observed for one minute after the swallow for coughing or wet/hoarse vocal quality (Ishida, 2016).

**Beyond 24 Hours**

In the United States, the average inpatient length of stay for stroke patients is 6.1 days (CDC, 2016). For ICU nurses, the first 24 hours of this stay are focused on stabilizing the patient’s physiology while closely monitoring for complications. Meanwhile, besides providing acute care, physicians use the first 24 to 48 hours to identify the cause of the stroke and to formulate a plan to correct the cause or to otherwise reduce the risk of additional strokes.
After the first 24 hours, the nursing staff can begin to shift away from acute care and begin to monitor for other patient management issues that may arise. The following concerns are addressed in the patient’s nursing care plan:

**IMPAIRED PHYSICAL MOBILITY**

Nursing actions for stroke patients with impaired physical mobility include:

- Collaborate with the interdisciplinary team, including physical and occupational therapy.
- Assess the extent of impairment initially and on a regular basis.
- Reposition the patient at least every 2 hours, and more if placed on affected side, which has poorer circulation.
- If tolerated, position prone once or twice daily to maintain hip extension.
- Prop extremities in functional position, use footboard, keep head in neutral position to prevent contractures and foot drop. If indicated, use arm sling when the patient is upright to reduce shoulder subluxation or shoulder-hand syndrome.
- Evaluate the need for positional aids/splint to prevent flexion contractures.
- Abduct affected arm by placing a pillow under the axilla, and elevate to promote venous return and prevent edema.
- Use trochanter rolls to prevent hip rotation, and place knee and hip in extended position to maintain function.
- Observe affected side for signs of compromised circulation.
- Monitor skin integrity. Use pressure-relieving devices as necessary.
- Beginning from admission, active and passive range of motion should be done to all extremities to minimize atrophy, promote circulation, and help prevent contractures. (Vera, 2013; Malones, 2015)

Currently, there is a lack of evidence-based guidelines for patients with stroke regarding the timing, frequency, and amount of out-of-bed activity recommended to improve outcome or prevent harm. Recent studies have suggested that early post-stroke mobilization may be harmful, in that the brain may need a “cooling off” period. In the largest study to date, it was found that the higher the dose of mobilization, a very early mobilization protocol was associated with a reduction in the odds of favorable outcome at three months. The data showed that an early, lower-dose out-of-bed activity regimen is preferable to very early, frequent, high-dose intervention (AVERT Trial Collaboration Group, 2015).
SELF-CARE DEFICIT

Following a stroke with functional deficits, the patient will need assistance in managing self-care. The nursing care plan may include:

- Collaborate with the interdisciplinary team, including occupational therapy.
- Assess the patient’s abilities and level of deficit for performing activities of daily living.
- Allow the patient to complete care for him/herself and provide assistance as needed.
- Observe for impaired judgment to promote safety.
- Allow sufficient time for the patient to accomplish tasks.
- Provide positive feedback for accomplishments and for efforts.
- Assist the patient with visual deficits by situating things (e.g., food) on a tray toward the patient’s unaffected side.
- Assist the patient to utilize self-help devices.
- Assess communication abilities to express toileting needs. (Vera, 2013; Malones, 2015)

RISK FOR ASPIRATION RELATED TO DYSPHAGIA

In order to maintain nutrition and hydration and avoid aspiration for patients with dysphagia, the nursing care plan should include:

- Collaborate with the interdisciplinary team, including speech and language therapy.
- Have suction equipment available at the bedside, especially during early feeding attempts.
- Place the patient in an upright position (45 degrees or higher) during and after feeding to facilitate the use of gravity in swallowing; maintain upright position for 45 to 60 minutes after eating.
- Place food of appropriate consistency, as determined by speech and language therapy, on the unaffected side of the mouth.
- Allow 30 to 45 minutes for meals.
- Limit or avoid the use of drinking straws.
- Administer IV fluid and/or tube feedings as directed. (Vera, 2013; Malones 2015)
ALTERATION IN BOWEL AND BLADDER ELIMINATION

Following a stroke, bowel and bladder problems are common. The nursing care plan for prevention or improving bowel and bladder elimination should include:

- Collaborate with physical and occupational therapy.
- Care for an indwelling urinary catheter and remove catheter as soon as possible.
- Initiate bladder and bowel training programs.
- Bedside bladder ultrasound should be done after voiding to check for residual early in the program.
- Schedule toileting at regular intervals.
- Maintain fluid intake of 2,000 ml per day unless contraindicated.
- Request and administer stool softeners or laxatives.
- Administer enemas if needed.
  (Ignatavicius & Workman, 2015)

RISK FOR FALLS

Stroke patients are at risk for falls related to mobility impairment, and universal fall precautions should be followed. Universal precautions apply to all patients at all times regardless of fall risk and revolve around keeping the environment safe and comfortable. In addition, stroke patients require collaboration between nursing and rehabilitation personnel to determine progressive ambulation needs and customization of all fall prevention interventions, such as utilization of bed or chair alarms, and sitters if necessary. Patient, family, and healthcare personnel should be alerted about the precautions instituted to prevent falls (AHRQ, 2013).

ALTERED NUTRITION AND HYDRATION

Stroke patients should have their nutritional and hydration status assessed on admission and regularly thereafter. The nursing care plan for nutrition and hydration includes:

- Collaborate with speech-language therapy.
- Institute nasogastric tube feeding within 24 hours of admission if unable to take adequate nutrition and fluids orally.
- Refer to a dietary specialist for detailed nutritional assessment, individualized advice, and monitoring.
- Give food, fluids, and medication in a modified form for patients with dysphagia.
- Provide recommended oral nutritional supplements as ordered.
- Administer long-term (greater than four weeks) nutritional support via percutaneous enteric gastrostomy tube.
- Assess for special problems that interfere with eating, such as neglect of one side; hemiplegia; aphasia; and cognitive, visual and communication challenges. (Poulia, 2015)

ACUTE STROKE REHABILITATION

One of the most important phases of stroke recovery is rehabilitation. Stroke rehabilitation begins during the acute hospital stay as soon as the patient has been medically stabilized and is able to tolerate and benefit from it—usually within 24 to 48 hours following the stroke. The highest priorities during this stage are to prevent a recurrent stroke and complications, ensure the proper management of general health functions, mobilize the patient, encourage resumption of self-care activities, and provide emotional support to the patient and family.

The goals of stroke rehabilitation in the acute care setting are to:

- Prevent, recognize, and manage comorbid medical conditions (e.g., pressure sores, DVT, aspiration pneumonia)
- Minimize impairments
- Maximize functional independence in activities of daily living

An interdisciplinary team approach is utilized in the rehabilitation of stroke patients. Once stabilized medically, the rehabilitation services of physical therapy, occupational therapy, and speech-language therapy should be consulted to assess rehabilitation needs, to begin early rehabilitation efforts, and to recommend the most appropriate post-stroke setting (NINDS, 2016d).

NEUROPLASTICITY AND THE REHABILITATION PROCESS

Stroke rehabilitation is based on the capability of the brain to reorganize by forming new neural connections, referred to as neuroplasticity. The underlying principle is that individual connections within the brain are constantly being removed or recreated, dependent on how they are used. The brain is malleable and constantly changing in response to experience.

Neuroplasticity involves both the function and the structure of the brain. Functional neuroplasticity is the brain’s ability to move functions from a damaged area to undamaged areas. Structural plasticity is the ability to actually change physical structure as a result of learning. The brain can reorganize pathways, create new connections, and even create new neurons throughout the lifespan.

Addressing Stroke-Related Impairments

Following a stroke, patients are often left with one or more of the following impairments that are addressed by members of the acute stroke rehabilitation team:

- Altered sensation
  - Pain (central pain or thalamic pain syndrome characterized by constant, severe burning pain with intermittent sharp pains)
  - Hyperalgesia
- Pain that is triggered by loud sounds, bright lights, etc.
- Altered vision
  - Homonymous hemianopia (a visual field loss on left or right side of the vertical midline in one or both eyes)
  - Visual neglect (an unconscious decreased awareness of part of the field of view or other stimuli to one side of the body)
  - Problems with depth perception, and spatial relationships
- Weakness (distal muscles more affected than proximal muscles)
- Alteration of muscle tone
  - Flaccidity present immediately after a stroke
  - Spasticity in a great percentage of cases
- Abnormal muscle synergy (coactivation of multiple muscles recruited by a single neural command)
- Abnormal reflexes
- Altered coordination
- Altered motor programming (apraxia)
- Impaired postural control and balance
- Emotional changes (e.g., depression is extremely common)
- Altered bladder and bowel function
- Speech, language, and swallowing deficits
Indirect impairments related to the above may eventually result in musculoskeletal changes such as:

- Loss of voluntary movement and immobility, resulting in loss of range of motion and contractures
- Disuse atrophy and muscle weakness
- Osteoporosis from decreased physical activity
  (Ignatavicius & Workman 2015)

**Physical and Occupation Therapy Assessment Tools**

Standardized evaluations and valid assessment tools are essential to evaluate patients following a stroke in order to develop a comprehensive treatment plan. Many of the following assessment tools are employed by both physical and occupational therapists:

- **Function Independence Measure (FIM):** A widely utilized tool that assesses physical and cognitive disabilities in terms of the burden required for taking care of the patient
- **Activities of Daily Living (ADL) Index:** Assesses mobility, walking, and moving around
- **Orpington Prognostic Scale (OPS):** A simple, objective bedside evaluation to obtain a baseline assessment of stroke severity
- **Stroke Rehabilitation Assessment of Movement (STREAM):** A quick and simple means designed for use by physical therapists to provide a qualitative evaluation of motor functioning for stroke patients
- **Barthel Index (BI):** Another widely used measure of functional disability involving a simple evaluation of independence to determine the ability of a patient to perform self-care
- **Berg Balance Scale (BBS):** Provides a quantitative assessment of static balance and fall risk in older adults
- **Chedoke-McMaster Stroke Assessment:** Designed to be used in conjunction with the Functional Independence Measure, employing the same rating methods for disability inventory
- **Motor Assessment Scale (MAS):** A task-oriented approach to evaluation assessing performance of functional tasks rather than isolated patterns of movement
- **Action Research Arm Test (ARAT):** An observer-rated, performance-based upper limb assessment of a patient’s ability to handle objects differing in size, weight, and shape; assesses grasp, grip, pinch, and gross movement
- **Assessment of Motor and Process Skills (AMPS):** Used to evaluate a person’s “quality” of performance of activities of daily living
• Functional Reach Test/Modified Functional Reach: Assesses a patient’s stability by measuring the greatest distance the patient can reach forward while standing in a fixed position (modified version requires the person to sit in a fixed position)

• Manual Muscle Testing: A procedure to evaluate function and strength of muscles and muscle groups based on the effective performance of a movement in relation to the forces of gravity and manual resistance

• Mann Assessment of Swallowing Ability (MASA): Designed for bedside evaluation and allows for determination of those who require more involved instrumental evaluation

• Acute Stroke Dysphagia Screen: Designed for use by practitioners who are not trained speech therapists to detect swallowing difficulty in stroke patients quickly and accurately (AOTA, 2013; Salter et al., 2013; APTA, 2016a; Dutton, 2016; Rehab Measures, 2015, 2013a, 2013b, 2013c)

FUNCTIONAL INDEPENDENCE MEASURE (FIM)

Tasks Assessed: This assessment tool contains 18 items composed of 13 motor tasks and 5 cognitive tasks that are considered basic activities of daily living. Areas assessed include:

- Eating
- Grooming
- Bathing
- Upper-body dressing
- Lower-body dressing
- Toileting
- Bladder management
- Bowel management
- Toilet transfer
- Locomotion (ambulatory or wheelchair level)
- Stairs
- Cognitive comprehension
- Social interaction
- Problem solving
- Memory

Scoring: Tasks are rated on a 7-point scale ranging from complete dependence to complete independence. Scores range from a low of 18 to a high of 126 and indicate level of function; they are generally obtained at admission and again at discharge.

Physical Therapy for Acute Stroke Rehab

Physical therapy is one of the core professional disciplines involved in stroke rehabilitation. Principles of physical therapy during the **acute phase** of treatment involve:

- Prevention of medical complications
- Prevention of deconditioning and contractures
- Training of new skills
  (Chatterjee, 2015; Ndjamen, 2015)

**Long-term** physical therapy goals are to:

- Improve sensory function
- Improve flexibility and joint integrity
- Improve strength
- Manage spasticity
- Improve movement control
- Improve postural control and functional mobility
- Manage shoulder pain
- Improve LE function
- Improve balance
- Improve locomotion
- Improve aerobic function
- Improve motor learning
- Educate patient and family
  (Chatterjee, 2015)

**PHYSICAL THERAPY ASSESSMENT**

Following an acute stroke, physical therapists begin assessment by obtaining:

- History of the present illness
- Past medical history
- Standardized review of systems
- List of medications being taken
- Family history
- History of any recent alterations in function prior to stroke
Physical examination involves observation of the patient for:

- Abnormal limb posturing
- Facial asymmetry
- Primitive movements (synergy patterns) of the upper and lower extremities, which dominate reflex and voluntary efforts that result from the loss of control in higher centers of the brain
- Abnormal gait pattern and use of any walking aids

The physical therapist continues assessment using tests and measures (see above) to identify impairments, many of which are done at rest, during, and following activity. Evaluations may include:

Joint integrity and mobility, for passive hypo- or hypermobility of joints, structure and integrity of the joint surfaces, and periarticular soft tissue qualities

- Range of motion, for accessory movement at joint surfaces; tissue extensibility, such as muscle-tension length and movement; and muscle tone, including the presence of spasticity
- Motor function, for weakness, paralysis, dysfunctional movement patterns and postures, abnormal timing, poor coordination, clumsiness, and the patient’s ability to control voluntary postures and movement patterns
- Reflex integrity, to determine the excitability of the nervous system and the integrity of the neuromuscular system
- Circulation, to determine the status of the cardiovascular and lymphatic drainage systems and their ability to adequately meet demands at rest and with activity
- Ventilation and respiration, to determine the presence of dyspnea during exercise, respiratory muscle strength, decreased tidal volume and vital capacity, and the respiratory system’s ability to meet oxygen demands
- Gait and locomotion, to determine the presence and underlying cause of gait deviations by assessing rhythm, cadence, step, stride, and speed
- Balance, to assess the patient’s ability to maintain equilibrium with gravity while stationary and while moving, as well as when standing, ambulating, and sitting, either support or unsupported
- Posture, for alignment and position of the body in relation to gravity, the center of mass, or the base of support in efforts to prevent injury or progressive deformity
- Functional assessment, to determine the patient’s ability to perform activities of daily living and instrumental activities of daily living
• Bowel and bladder, for flaccidity during the acute stage
• Need for assistive and adaptive devices
  (Surbala, 2013; Georgiev, 2015; APTA, 2016b)

PHYSICAL REHABILITATION APPROACHES

There are various approaches to physical rehabilitation that can be used following stroke, and debate is ongoing about the effectiveness of these approaches. Therapists may elect to use one approach or any combination of them.

**Traditional therapy** includes range of motion exercises, strengthening exercises, and mobilization strategies, as well as compensatory techniques.

**Bobath concept** is the most commonly used neurodevelopmental training approach, in which muscle patterns and not isolated movements are utilized for motion. The stroke patient with motor deficits is not able to direct nervous impulses to muscles in the different combinations used normally. The therapy suppresses abnormal muscle patterns before normal patterns are introduced. Abnormal patterns are modified at key points of control—the neck, spine, shoulder, and pelvis.

**Proprioceptive neuromuscular facilitation** (PNF) stimulates nerve-muscle/sensory receptors to evoke response through manual stimuli to increase the ease of movement and to promote function.

**Sensorimotor therapy/Rood Approach** is a therapy in which the modification of muscle tone and voluntary motor activity is attempted using cutaneous sensorimotor stimulation. It utilizes quick stretch, icing, fast brushing, slow stroking, tendon tapping, vibration, and joint compression to promote contraction of proximal muscles.

**Motor relearning program/Carr and Shepherd Approach** uses cognitive motor relearning theory to allow the patient to relearn how to move functionally and how to problem solve during attempts at new tasks. It teaches general strategies for solving motor problems.

**Brunnstrom Approach movement therapy** uses primitive synergistic patterns in training to improve motor control through central facilitation using what is referred to as Twitchell’s recovery. Patients are taught to use and voluntarily control motor patterns available to them at a particular point in the recovery process. Cutaneous/proprrioceptive stimulation enhances the synergies. Brunnstrom movement therapy is based on seven stages of recovery from stroke (see below) (Cuccurullo, 2014; Bruno-Petrina, 2016; NINDS, 2014; Nudo 2013; Lighthill & Atkins, 2013).
BRUNNSTROM 7 STAGES OF RECOVERY FROM STROKE

Stage 1 — This is a period of flaccidity when neither reflex nor voluntary movements are present.

Stage 2 — Basic limb synergies may appear, spasticity appears, and minimal voluntary movement responses may be present.

Stage 3 — The patient starts to regain control over movement synergies. Spasticity reaches its peak, and the patient is able to initiate movement but is unable to control it.

Stage 4 — Spasticity begins to decline, and some movement combinations are mastered.

Stage 5 — More difficult movement combinations are mastered. Spasticity continues to decline.

Stage 6 — Individual joint movement becomes possible, coordination approaches normalcy, spasticity disappears, and the individual is more capable of full movement patterns.

Stage 7 — Normal motor functions are restored.

Source: Chatterjee, 2015.

ACUTE-CARE PHYSICAL THERAPY INTERVENTIONS

As soon as the patient is medically stabilized, physical therapy employs the following interventions in the acute-care setting:

• **Positioning:** The therapist advises on positioning the patient in supine, side lying on both affected and normal side, and sitting. Physical therapy teaches patients to roll to sit up, to sit supported in bed, and to sit supported out of bed. Proper positioning helps reduce muscle pain, spasms, slowness, or stiffness that can occur following a stroke.

• **Range of motion exercises:** Both passive and active exercises are begun and done daily to promote and maintain joint mobility, prevent contractures, increase circulation to the extremities, and decrease vascular complications of immobility.

• **Sitting practice:** Sitting is important to build up tolerance, provide maximum stimulation and give the patient a sense of normalcy.

• **Breathing exercises:** Deep breathing and coughing, chest expansion exercises, and passive and active ankle and toe exercises are begun to improve respiratory and circulatory function if there are no contraindications.
• **Decubiti prevention measures:** In order to prevent the complication of pressure ulcers, the therapist ensures that the patient is properly positioned and that pressure points are protected by padding and/or cushioning. PT may advise on the use of special beds such as air mattresses, water beds, or foam mattresses.

• **Deconditioning prevention measures:** To prevent deconditioning, the therapist encourages early bed mobilization such as active turning, moving from supine to sitting, sitting to supine, sitting, sit to stand, and finally, walking.

• **Transfer techniques:** Physical therapy teaches the patient safe transfer from bed to chair and from a sitting to a standing position. PT also advises staff on how to support the patient during transfers, demonstrating and performing transfers using assistive devices such as a gait belt or a hydraulic lift.

• **Balance improvement measures:** With increased patient mobility, the therapist assists with balance improvement using pelvic bridging exercises. Improving balance along with walking leads to greater independence and improves well-being. Current tools do not allow for balance training under load conditions sufficiently low for acute stroke patients. A new and novel tool, Balance Bed, has the ability to provide whole-body functional balance exercises, including balance perturbations, for patients in the acute phase of stroke (Oddsson et al., 2015).

• **Assistive device training:** Prior to discharge, the physical therapist fits a patient to a wheelchair and/or trains patients in the use of mobility assistive devices such as:
  - Orthoses
  - Prostheses
  - Canes
  - Walkers
  - Wheelchairs

• **Patient/family education:** PT teaches family members how to help with exercises for the patient and how they can help the patient move or walk. They also teach them about the disease process and what rehabilitation may be able to accomplish for the patient.

• **Discharge planning** Throughout the hospitalization, assessment has been continuous in an effort to assist in determining the most appropriate setting for the next level of care. The physical therapist may make a home visit prior to discharge to determine the need for architectural modification. (NINDS, 2016d; Surbala, 2013)
Occupational Therapy for Acute Stroke Rehab

Occupational therapy plays a very significant role in facilitating early mobilization, restoring function, preventing further decline, and coordinating care, including transition and discharge planning.

Treatment approaches are aimed to meet the ultimate goal of maximizing function and independence. They include:

- Rehabilitate and restore function using physical, cognitive, perceptual, and functional activities
- Teach compensatory techniques (adaptive equipment)
- Provide preventative education to help patients gain an understanding of their condition, maintain improvements, and avoid problems in the future

OCCUPATIONAL THERAPY ASSESSMENT

An initial assessment by occupational therapy is completed to understand the impact of changes in motor function, sensation, visual perception, and cognition on the stroke patient’s abilities to manage the tasks of daily living. Elements of an occupational therapy assessment include:

- Interviews with the patient and/or family to establish the patient’s prior life roles and the tasks and activities involved in those roles
- Observation of the acute stroke patient’s abilities to perform personal self-care (e.g., showering, dressing, toileting, grooming, eating)
- Visual-perceptual screening for impairments that can interfere with the ability to organize, interpret, and give meaning to information that is seen, impacting the ability to learn
- Memory, cognition, and executive functioning screening to determine the impact of changes on abilities to resume daily functioning
- Sensory, motor, and upper limb assessment, with particular emphasis on the upper limb and hand function
  (Boyt Schell & Scaffa, 2015)

ACUTE-CARE OCCUPATIONAL THERAPY INTERVENTIONS

Occupational therapy interventions in the acute stage include:

- **Positioning and seating**: Correct positioning in good body alignment to reduce the risk of:
  - Aspiration
• **Upper limb positioning:** Addressing upper limb positioning to prevent shoulder trauma, lessen pain, reduce swelling, and encourage independence in feeding and other self-care activities.

• **Mobilization techniques:** Utilizing positioning, turning, and transferring techniques to assist with mobility.

• **Prevention of decubiti:** Utilizing methods to prevent decubiti, such as:
  - Cushions and padding
  - Barrier sprays
  - Lubricants
  - Special mattresses
  - Protective dressings
  - Splinting
  - Use of positioning devices

• **Splinting:** Utilizing splints to correct biomechanical malalignment and protect joint integrity, as well as to prevent shortening of soft tissues and development of contractures.

• **Personal ADLs:** Providing training in self-care activities (e.g., bathing, dressing) with adaptive or durable medical equipment and/or compensatory techniques if needed, such as linking behaviors that naturally go together, providing cueing, focusing on one task or step at a time, and completing it before moving on to the next.

• **Assistive and adaptive devices:** Providing training in the use of upper extremity adaptive devices and wheelchair management (adaptive devices may be recommended for eating, bathing, grooming, and transferring).

• **Management of shoulder pain:** Beginning prophylactic management early; educating all members of the team, patient, and family on the correct way to handle a vulnerable shoulder and to never use the affected arm to aid transfers; supporting the affected arm to maintain alignment; strapping the hemiplegic shoulder to reduce pain.

• **Discharge planning:** Making recommendations for ongoing rehab in settings appropriate to the level of the patient’s rehabilitation needs, including outpatient settings and the home; completing a home assessment to identify potential difficulties such as safety and accessibility.

(AOTA, 2016; Teasell et al., 2012; Gillen, 2016)
Speech and Language Therapy

Speech-language therapy is another core element of stroke rehabilitation and has a key role in the identification, assessment, and management of potentially life-threatening eating, drinking, and swallowing problems (dysphagia).

All stroke patients should be screened at bedside for dysphagia before being given food or fluids. Following a failed bedside screening, the speech-language therapist completes a risk assessment of current eating and drinking, determines the safety of food trials, and chooses further swallowing assessment using videofluoroscopy or fiberoptic endoscopic evaluation (FESS) (NINDS, 2016d).

A clinical judgment is then made regarding how to proceed with food and fluids, and if necessary, modification are made of food and liquid consistency, temperature, taste, and texture in order to improve swallowing function and efficiency.

SPEECH-LANGUAGE INTERVENTIONS

The treatment plan for stroke patients with dysphagia may consist of strengthening exercises for the oral, laryngeal, and pharyngeal musculature as well as compensatory strategy training.

Typical strategies may include:

- Altering bolus size, consistency, or method of presentation
- Altering patient posture/position
- Utilizing sensory stimulation for heightened sensory input
- Utilizing therapeutic maneuvers (e.g., Mendelsohn Maneuver, Supraglottic Swallow)
- Utilizing exercise programs (tongue resistance, ROM, tongue base, chewing) (Knutsen, 2014; Gragga, 2015)

Strokes can also result in difficulties with a patient’s ability to communicate ideas, needs, and feelings. These problems may include:

- **Apraxia**: Patients have difficulty or cannot move their mouth and tongue to speak.
- **Aphasia**: Impairment of language, affecting production or comprehension of speech and ability to read or write. Three common types of aphasia seen in stroke patients are:
  - Broca’s aphasia (nonfluent or expressive). Speech output is severely reduced and is limited to short utterances of four words or less. The person may understand speech and be able to read but be limited in writing.
  - Wernicke’s aphasia (fluent or receptive): The patient may speak easily and fluently in long, complex sentences that don’t make sense or that include
unrecognizable, incorrect, or unnecessary words. They commonly don’t understand spoken language and often don’t recognize that others can’t understand them.

- **Global aphasia:** Results from extensive damage to the language areas of the brain. These patients have impaired ability in both expression and comprehension.

- **Dysarthria:** Patients have impaired intelligibility of speech as a result of weakness, paralysis, or incoordination of speech musculature.

- **Cognitive deficits:** Patients with cognitive deficits have problems with attention, memory, perception, insight and judgment, organization, processing speed, problem solving, reasoning, and executive functioning. (Mayo Clinic, 2015; Tactus therapy, 2016)

In the acute phase of stroke recovery, the speech-language therapist works with the patient to develop verbal and nonverbal strategies in order to provide a communication lifeline for the patient to make choices, get needs met, and better cope with the stroke experience. These strategies are taught to staff and family members as well, and may include:

- Use of gestures or symbols
- Writing
- Communication charts
- Letter boards
- Drawing
  (Stroke Association, 2012)

**Discharge from the Hospital**

Nurses are the key players in organizing a patient’s discharge from the hospital. Nurses are with the patient throughout the day, and they have seen the full range of the patient’s limitations and dependencies. While a patient is still in the hospital, nurses on the stroke team initiate the patient’s transition into the appropriate supervised rehabilitation programs.

As the time of discharge approaches, nurses arrange to have a patient’s limitations assessed formally by specialists—including physical therapists, occupational therapists, speech-language pathologists, psychologists, and nutritionists. These professionals then make recommendations that can be taken into account before physicians have begun discharging the patient.

**MEDICAL REHABILITATION**

After a stroke, the patient’s health is unstable, and they are at risk for cardiovascular problems and for additional strokes. Medically, the post-hospital goals for a stroke patient are to avoid or to quickly deal with medical complications and to prevent the recurrence of strokes and TIAs. Plans to safeguard a patient’s health can be called medical rehabilitation.
The physician’s role in medical rehabilitation is the coordination of longer-term care, preventing second strokes, blood pressure control, diabetes management, and the elimination of risk factors such as smoking, excess weight, dyslipidemia, and excessive alcohol consumption.

PATIENT AND FAMILY EDUCATION

As the stroke team hands a stroke patient over to a medical rehabilitation program, the patient, family, and caregivers need to be informed, educated, and kept informed. After the hospitalization, these individuals will be making the day-to-day healthcare decisions for the patient, and these decisions need to be based on accurate, realistic information.

The interdisciplinary team is involved in educating the patient and family on the recovery process, explaining the pathology of the patient’s particular stroke, practical problems that the patient will face, and methods for preventing a recurrence of the stroke. Advice will be given to caregivers and family about communicating with a patient who is aphasic or who has significant motor or sensory deficits. Follow-up visits with a physician are scheduled and family and patient are made aware of these appointments.

In discussions with the patient and family, they should be counseled that almost three quarters of stroke survivors will eventually need their family’s assistance at home and that the practicalities and costs of that home help should be thought through in advance. Finally, a social worker or community liaison provides referrals to government and nonprofit help agencies, support groups, and other helpful community resources (Kaltenbach et al., 2013; Kerr, 2012).

Patient education will already have begun during the patient’s stay by medical, nursing, and rehabilitation personnel, and the influx of medical information at discharge can be overwhelming. Therefore, patients and families should be given instructions and guidelines in the form of printed materials that can be reviewed at home as long as patient can cognitively comprehend the instructions. If not, a SLP may be able to assist. Nurses should also include a list of medically accurate stroke websites; the “Resources” section below offers some suggestions.

PREVENTING SECONDARY STROKE

While a major component in the prevention of secondary stroke is early, aggressive rehabilitation, additional interventions are necessary to optimize the patient’s chances of a satisfying and productive life after stroke. The mnemonic \textit{ABCDE} describes important elements in preventing secondary stroke:

\begin{itemize}
  \item A – Antiaggregants and anticoagulants
  \item B – Blood pressure-lowering medications
  \item C – Cholesterol-lowering medications, cessation of smoking
  \item D – Diet
  \item E – Exercise
\end{itemize}

(Silver, 2015b)
Medications Used for Prevention of Secondary Stroke

For patients who have had an ischemic stroke related to atherosclerosis or embolus, antiaggregant and anticoagulant medications are recommended for prevention of secondary stroke.

ANTIAGGREGANTS

- Aspirin: Adverse effects include gastritis, tinnitus, and hearing loss.
- Clopidogrel (Plavix): Requires no blood monitoring; adverse effects are similar to aspirin.
- Extended-release dipyridamole (Persantine): More effective in combination with aspirin than aspirin alone; combination adverse effects are similar to aspirin with the exception of an increased incidence of headache and GI disturbance.
- Ticlopidine (Ticlid): Blood monitoring is required every 2 weeks for 3 months; adverse effects include diarrhea, skin rash, and reversible agranulocytosis.
- Vorapaxar (Zontivity): A 2014 FDA-approved antiplatelet medication that is a protease-activated receptor 1 (PAR-1) inhibitor, is given in addition to aspirin and/or clopidogrel.

ANTICOAGULANTS

- Warfarin (Coumadin): Patients with atrial fibrillation should receive warfarin if they are high-risk or are older than 75 years regardless of risk.
- Vitamin K antagonists (VKS): In stroke in patients with atrial fibrillation, VKS significantly reduces the risk of recurrent stroke and is recommended by guidelines for three to six months.
- New option anticoagulants (NOACs). NOACs have been approved by the FDA and are as effective or more so in preventing stroke as warfarin or VKS; do not require routine INR monitoring; and are safer from a hemorrhagic-stroke standpoint. They include:
  - Dabigatran (Pradaxa)
  - Rivaroxaban (Xeralto)
  - Apixaban (Eliquis)

(Baeusler et al, 2015; Jauch, 2013; Silver, 2015b; Chong, 2013)

BLOOD PRESSURE-LOWERING MEDICATIONS

High blood pressure puts a stroke victim at risk for additional strokes; therefore, reducing hypertension is a generally accepted post-stroke goal. One common guideline suggests gradually reducing the blood pressure of a post-stroke patient over several months, with an end goal of <130/80 mm Hg.
First-line agents for control of hypertension in the prevention of secondary stroke include:

- Diazide diuretics
- Calcium-channel blockers, angiotensin-converting enzyme (ACE) inhibitors
- Angiotensin receptor blockers (ARBs)

ACE inhibitor (ramipril) added to other medical therapy, including antiplatelet agents, reduces the risk of stroke; it is believed this is due to endothelial protection. The combination of peridopril (an ACE inhibitor) and indapamide (a thiazide diuretic) substantially reduces the recurrence of stroke, mainly as a result of lowering blood pressure.

Beta blockers are second-line agents, as they are considered inferior to the others in preventing secondary strokes.

**CHOLESTEROL-LOWERING MEDICATIONS (STATINS)**

Studies have shown a 10-year reduction of recurrent stroke when statin therapy is added after a first stroke. HMG-CoA reductase inhibitors include:

- Pravastatin (Provachol): In patients with a history of coronary artery disease, pravastatin decreases the risk of future stroke, even in those with normal serum cholesterol levels.
- Simvastatin (Zocor): In patients with a history of coronary artery disease, other vascular disease, or diabetes, simvastatin has also shown a reduction in secondary stroke risk. (Jauch, 2013; Silver, 2015b)

**Cessation of Smoking**

Smoking increases the risk for developing atherosclerosis, and reversing atherosclerosis is an important part of preventing future heart attack or stroke. Inhaling cigarette smoke produces several affects that damage the cerebrovascular system. Women who are taking oral contraceptives and smoke increase their risk of stroke many times. Cigars and pipes are not a safer alternative to cigarettes, and people who smoke them actually have a higher risk for cardiovascular disease.

It is also important to avoid secondhand smoke. The risk of stroke for nonsmokers exposed to secondhand smoke is increased by an estimated 20% to 30% (AHA, 2016).

Written prescriptions for exercise and medications for smoking cessation (nicotine patch, bupropion, varenicline) increase the likelihood of smoking cessation (Silver, 2015b).
Diet Modifications

Studies have been done showing several diets that are beneficial in the reduction of risk for secondary stroke. These include:

- Mediterranean diet: Primarily fruits and vegetables, whole grains, legumes and nuts. Butter is replaced with healthy fats, and herbs and spices instead of salt are used to flavor foods. Red meat is eaten no more than a few times a month. Fish and poultry are eaten at least twice weekly. Moderate intake of alcohol is included.

- The Dietary Approaches to Stop Hypertension (DASH) diet is similar to the Mediterranean diet, emphasizing fruits, vegetables, and a low intake of red meat and sweets.

- The Prudent diet also includes a higher intake of fruits and vegetables, whole grains, legumes, and fish.

(Dearborn et al, 2015; White, 2015)

Exercise

Following ischemic stroke or TIA, patients who are capable of engaging in activity should consider at least 30 minutes of moderate-intensity physical exercise such as walking briskly or using an exercise bicycle each day. This may reduce risk factors and comorbid conditions that increase the risk for recurrent stroke (Billinger et al., 2014).

SUMMARY

Strokes Are Sudden Disruptions of Blood Flow to the Brain

Strokes, also called cerebrovascular accidents (CVAs), result from limitations in cerebral perfusion, usually due to clots. Occasionally, the reductions in perfusion are accompanied by intracranial bleeding.

Approximately 6 million Americans have had a stroke, and about 800,000 people suffer a stroke each year. Strokes do not strike equally throughout the population. Strokes are usually a condition of the elderly; the most susceptible age group is in the 80- to 84-year-old range. More women than men die of stroke each year.

Most strokes result from blockages of an artery by a local blood clot or by an embolus from the heart or carotid artery. These strokes are called ischemic, and they are typically the product of years of atherosclerosis and hypertension. About 10% of all strokes are quite different, having been caused by intracranial bleeds. These are called hemorrhagic strokes, and they result from a ruptured cerebral artery or aneurysm. Hypertension is typically involved in generating a hemorrhagic stroke.
Symptomatically, all strokes appear as acute impairments in brain functioning. A person may suddenly have difficulty walking, seeing, speaking, or understanding. With severe hemorrhagic strokes, the person may lose consciousness. A common presentation of a stroke is the sudden loss of sensation or movement on one side of the body or face. Most ischemic strokes are painless, although hemorrhagic strokes can produce severe headache.

**Acute Strokes Are High Priority Emergencies**

An acute ischemic stroke is a medical emergency, much like a myocardial infarction; a “brain attack” needs fast, organized care just as does a heart attack. The acute treatments are also similar. Both strokes and myocardial infarctions can be caused by clots obstructing arteries, both can leave some tissue underperfused, and in both, underperfused tissue can sometimes be revived if local circulation can be reestablished within a critical time window.

Like the treatment for an acute myocardial infarction, treatment for an acute stroke is given high priority by EMS teams and emergency room personnel. For a stroke, there is a 4.5-hour interval after the onset of symptoms in which thrombolytic therapy (i.e., intravenous administration of rtPA) has a chance to reopen clogged cerebral arteries and save some of the underperfused brain tissue. Given this time constraint, EMS teams have the goal of getting potential stroke victims stabilized, evaluated, and to a stroke center in less than an hour.

The early recognition and diagnosis of a stroke is facilitated by using standardized tests, such as the Cincinnati Prehospital Stroke Scale, which can be administered in three to five minutes using no special equipment. Such standardized diagnostic tools give accurate and reproducible predictions of the likelihood that a person has had an acute stroke. It has been shown that 911 operators can even administer the Cincinnati Prehospital Stroke Scale over the phone with the help of cooperative bystanders.

**ED Evaluation of a Stroke Requires Head Imaging**

While making a detailed diagnosis, the first step in treating a stroke is to establish an airway, possibly by intubation. Next, one must check for evidence of head trauma and consider immobilizing the spine. If the patient’s neurological condition is deteriorating (e.g., if there is a decreasing level of consciousness, pupillary dysfunction suggesting brainstem damage, or decorticate or decerebrate posturing), there may be cerebral edema or continued hemorrhaging, so neurosurgery must be consulted.

After a brief medical history (that includes defining the time course of the onset of symptoms) and a physical exam (with special attention to the neurological and cardiac exams), stat blood work (blood glucose, serum electrolytes, renal function tests, cardiac markers, CBC, prothrombin time/INR, a PTT, and a toxicology screen if drug use is suspected) is drawn.

As in pre-hospital (i.e., EMS) stroke management, emergency department stroke care is facilitated by using standardized tests. In the ED, the recommended assessment tool is the NIH Stroke Scale, which can be administered in five to eight minutes using no special equipment. The
NIH Stroke Scale quantifies the severity of a stroke, and it has been widely used to measure both deterioration and improvement of stroke patients.

The critical step in evaluating an acute stroke is making the distinction between ischemic and hemorrhagic strokes. For this, there is no clinical test; the determination must be made by CT (or MRI) imaging, as interpreted by an experienced radiologist. Emergency head imaging (usually, a noncontrast CT scan) is needed within 25 minutes of the patient’s delivery to the ED, and a completed radiologic evaluation is needed less than 20 minutes later.

**Eligible Victims of Acute Ischemic Strokes Can Be Treated with a Fibrinolytic Drug**

At this point, treatment paths for ischemic and hemorrhagic stroke patients diverge. For ischemic strokes, IV recombinant tissue plasminogen activator (rtPA) should be administered to eligible patients within 4.5 hours of the onset of symptoms. To be eligible, patients must not be pregnant, must have a sufficiently high platelet count, and can have no indication of intracranial hemorrhage, no recent major surgery, no evidence of internal bleeding, no known bleeding diatheses, and no current anticoagulant therapy. After receiving IV rtPA, patients must be carefully monitored for at least 24 hours in an ICU.

For hemorrhagic strokes due to a ruptured subarachnoid aneurysm, neurosurgery is consulted for possible treatment by surgically clipping the aneurysm remnant or by endovascularly inserting a coil. For other subarachnoid hemorrhages, intracerebral hemorrhages, and ischemic strokes ineligible for rtPA treatment, patients are admitted directly to an ICU and monitored carefully.

**ICU Care of Acute Stroke Victims Focuses on Preventing or Quickly Treating Complications**

In the ICU, stroke patients have their vital signs and neurological functioning checked regularly. The acute management of a stroke patient’s hypertension cannot be an automatic process; treatment must balance the threat of additional hypertensive tissue damage against the need to maintain adequate cerebral perfusion. Extreme hypertension is reduced gradually, but most patients are allowed to remain mildly hypertensive early in their ICU course.

Overall, 30% of strokes will deteriorate within the first 24 hours. Deteriorating vital or neurological signs can be due to cerebral edema, increased intracranial pressure, or rebleeding, as well as to cardiopulmonary problems. Neurosurgery should be involved in assessing a deteriorating patient.

**Acute Stroke Rehabilitation**

As soon as the patient is stabilized medically, usually within 24 to 48 hours, the rehabilitation team is consulted to assess rehabilitation needs, begin early rehabilitation efforts, and recommend the most appropriate post-stroke setting. The goals of rehabilitation in the acute
setting are to prevent, recognize, and manage comorbid medical conditions; to minimize impairments; and to maximize functional independence.

**Most Stroke Patients Need Long-Term Physical and Medical Rehabilitation**

The recovery from a stroke usually requires long-term coordinated and continuing medical and physical rehabilitation. Patients who have been left with severe disabilities from their stroke may still be gradually improving more than five months afterward.

Patients frequently recover from strokes, even serious strokes, because of the ability of the brain to learn new ways to accomplish old tasks. This learning takes time, and one medical rehabilitation goal is to maintain a patient’s health sufficiently for their brain to relearn what it can; the second medical goal is to prevent additional strokes.

At the same time, the goals of physical rehabilitation are to maximize the speed at which the brain retrains itself to complete functional tasks and to substitute tasks that are more manageable for those functions that cannot be relearned.

**QUESTIONS PATIENTS MAY ASK**

**First Response to a Stroke**

**Q.** What should I do if I think I may be having a stroke?

**A.** A stroke is an emergency like a heart attack. Call 911 immediately, or get someone to call for you. Don’t wait for the symptoms to go away, and don’t worry that you may be mistaken. Paramedics would much rather come and reassure you than see you suffer the consequences of an untreated stroke.

**Q.** I’m close to a hospital; shouldn’t I drive myself rather than waste time calling 911?

**A.** Strokes can disrupt your ability to drive, so do not drive anywhere if you think you are having a stroke. It’s also better medically for you to wait for an EMS team, so don’t let someone else drive you to a hospital if it is possible to get trained professionals to take you.

Strokes need immediate treatment, but they must be treated properly. The EMS team that comes when you call 911 knows the best first aid to administer. They know which treatments to start on the way to the hospital, they know which hospital can give you the best stroke treatments, and they will call ahead so that the hospital will be prepared to speed you past the front desk and into a treatment room.
Q. How can I tell if someone is having a stroke?

A. Strokes come on suddenly. Sometimes there is a severe headache, but many times there is no pain at all. When you have a stroke, you are suddenly not able to do something that you could do before. Classic stroke symptoms are:

- A sudden weakness of your face, arm, or leg, often to just one side of your body
- A sudden numbness of your face, arm, or leg, often to just one side of your body
- Sudden confusion, trouble speaking, or difficulty understanding things
- Sudden trouble seeing with one eye or with both eyes
- Sudden trouble walking, sudden dizziness, or a sudden loss of balance or coordination
- A sudden severe headache that you can’t explain

A person having a stroke may show one or more of these signs. Any of the above symptoms signals an emergency, so call 911 just as you would if you saw a car accident or if a person was choking, had sudden chest pain, or became unconscious or unresponsive. You don’t have to be certain that the person is actually having a stroke.

Q. What first aid should I give someone with a stroke?

A. Make sure the person is in a safe place, then call 911. Calling for assistance is the most critical first aid. The 911 operator will give you further advice about first aid.

Q. What happens when someone has a stroke?

A. A person has a stroke when a part of their brain stops getting enough blood. Usually, strokes happen all of a sudden, so the stroke patient finds that they have suddenly lost some ability. The patient may suddenly not be able to move an arm, or they may lose the ability to feel things, to speak clearly, or to walk.

Infrequently, a stroke will show up with a sudden severe headache, but most often strokes are painless, and a person may not realize they have had a stroke until they try to use one of the affected muscles. For example, they may suddenly realize that they can’t hold something in their hand; they may fall when they stand up because one of their legs isn’t working; or they may be confused or unable to talk clearly.

The best treatments for strokes need to begin quickly. If you think that you or someone around you may be having a stroke, call 911 immediately.
Informational Questions

Q. What is a stroke? What are the different types?

A. There are two main types of stroke: ischemic and hemorrhagic.

The most common type of stroke is **ischemic**. In an ischemic stroke, a brain artery becomes blocked by a **blood clot**. The region of the brain normally supplied by that artery no longer gets enough blood, and that part of the brain becomes starved for oxygen and sugar. Without oxygen and sugar, nerve cells stop working, so the affected region of the brain can no longer perform its particular functions, such as moving an arm or a leg.

Brain cells will stop working when they get less than the normal amount of blood—even when the blood supply hasn’t stopped completely but has only been reduced. If the blood flow can be restored quickly enough, many of the brain cells will start working again and the difficulties that the person was having will go away partly or completely. On the other hand, if it takes too long to restore the blood flow, brain cells will die. In this case, the difficulties caused by the stroke will remain.

A less common type of stroke is **hemorrhagic**, which means “bleeding.” In a hemorrhagic stroke, an artery is torn and blood begins to leak out and form a pool in the brain. When the blood is leaking out of the artery, it is not carrying sufficient oxygen and sugar to the region that it normally supplies, and the person has the same problems as in an ischemic stroke. In addition, in a hemorrhagic stroke, the pool of blood expands and pushes on the neighboring blood vessels and brain cells. The pressure of the expanding pool of blood causes additional brain damage.

Q. What is the difference between a stroke, a brain attack, and a cerebrovascular accident (CVA)?

A. These are three different names for the same thing.

Q. I have heart disease, and my doctor said I might have a stroke. How can heart disease affect the brain?

A. Most strokes are caused by clots that become stuck inside arteries in the head and then cut off the supply of blood to the brain.

One relation of heart disease to strokes is that they can both be caused by **atherosclerosis**. Just as in a stroke, heart attacks and attacks of chest pain (called **angina**) are often caused by blood clots. Blood clots in the heart usually come from atherosclerosis. Atherosclerosis is a disease that can affect all the large arteries in the body, and some clots formed by atherosclerosis can be swept into the brain. Therefore, if a person has blood clots in their heart, then they also have a chance of getting blood clots elsewhere, such as in their brain.
Another relation between heart disease and strokes has to do with problems in the rhythm of the heartbeat. Irregular heart rhythms can cause blood clots. One particular heart rhythm irregularity, called **atrial fibrillation**, is notorious for putting a person at risk for getting a stroke. If you have atrial fibrillation, ask your doctor how you can reduce your chance of getting a stroke. And be sure to also ask your doctor to teach you the warning signs of a stroke.

**Q. Can a stroke be stopped?**

**A.** A stroke is the set of symptoms that follow when a brain artery is blocked or bleeding. The brain can often recover if the cause of the stroke can be reversed and fresh blood can be gotten to the blood-starved areas soon enough.

When the underlying problem is a blocked artery, the stroke symptoms will sometimes lessen or even disappear if the obstructing clot is removed or dissolved quickly enough. In the case of bleeding arteries, these will sometimes stop bleeding on their own, and sometimes they can be coaxed to slow down or stop. If the bleeding can be stopped, the stroke symptoms will sometimes lessen.

All treatments depend on speed, so call 911 immediately if someone might be having a stroke.

**Q. What are clot-dissolving or clot-busting drugs?**

**A.** Clot-dissolving drugs are enzymes that break the bonds holding clots together. Clot-dissolving drugs have been used for a long time to treat blood clots elsewhere in the body. One drug has been approved by the U.S. Food and Drug Administration (FDA) for dissolving blood clots in the brain. This drug is called alteplase.

Alteplase is usually injected in a vein, and it is carried in the blood stream to the clot, where it breaks up the threads of protein that hold the clot together. Not all strokes can be treated with alteplase, and alteplase can sometimes cause bleeding in the brain. Nonetheless, when an experienced physician recommends using alteplase for a person who has just had a stroke, the benefits outweigh the risks.

**Q. My mother died of a stroke. Am I likely to have a stroke, too? What about my children?**

**A.** People whose parents, grandparents, brothers, or sisters had a stroke have a higher risk of having a stroke themselves. You can reduce your chances of having a stroke and protect yourself and your children by paying special attention to six things in your lifestyle.

1. **Keep your blood pressure** in a healthy range. High blood pressure can cause a stroke. People can have high blood pressure without knowing it, so get your blood pressure checked. If you have high blood pressure, follow your primary care provider’s recommendations.

2. **Stop smoking.** Smokers have a greater risk of having a stroke, and smokers who are also taking birth control pills have an even higher risk. Ask your primary care provider to
suggest a stop-smoking plan.

3. Control your diabetes. People with diabetes have a higher risk of having a stroke. Follow your primary care provider’s recommendations for controlling your blood sugar levels.

4. Keep your cholesterol level low. High blood cholesterol makes a person more likely to develop atherosclerosis, and atherosclerosis is a major cause of strokes. Get your cholesterol level checked. If your cholesterol levels are unhealthy, follow your primary care provider’s recommendations for your diet and take any medications that he or she prescribes.

5. Keep your weight low. Obesity is another condition that will increase your risk of developing a stroke. Losing weight is difficult, so ask your primary care provider for help in making a realistic weight loss plan.


RESOURCES

American Stroke Association (A division of the American Heart Association)
http://www.strokeassociation.org

Brain Attack Coalition
https://www.brainattackcoalition.org/

National Stroke Association
http://www.stroke.org

Neurological Flowsheet (Sutter Medical Center, Sacramento)

NIH Stroke Scale training
http://www.nihstrokescale.org/

Stroke (National Institute of Neurologic Disorders and Stroke)

Stroke assessment scales (Internet Stroke Center)
http://www.strokecenter.org/professionals/stroke-diagnosis/stroke-assessment-scales-overview/
REFERENCES


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1. Which is a correct statement about the incidence and prevalence of stroke in the United States?
   a. Alaska Natives and blacks are more likely to have a stroke than are other groups.
   b. Men have more strokes than women.
   c. The northwestern part of the United States is referred to as the “stroke belt.”
   d. Stroke does not occur in infants and children.

2. Which is the most important treatable risk factor stroke?
   a. Diabetes mellitus
   b. Atrial fibrillation
   c. Hypertension
   d. Obesity

3. The two major categories of stroke are:
   a. Thrombotic and embolic.
   b. Ischemic and hemorrhagic.
   c. Warning stroke and mini stroke.
   d. Lacunar infarction and transient ischemic attack (TIA).

4. Which type of stroke constitutes the majority of strokes?
   a. Transient ischemic attack (TIA)
   b. Subarachnoid hemorrhage
   c. Intracranial hemorrhage
   d. Ischemic

5. Cerebral emboli that cause strokes usually originate in the:
   a. Middle cerebral arteries.
   b. Deep leg veins.
   c. Left vertebral artery.
   d. Heart, pulmonary trunk, or pulmonary arteries.
6. A hemorrhagic stroke that results in bleeding into the space surrounding the brain is called:
   a. An intracranial hemorrhage.
   b. A subarachnoid hemorrhage.
   c. A cerebral infarction.
   d. An arteriovenous malformation (AVM).

7. Which arteries supply blood to 80% of the brain?
   a. The right and left internal carotids
   b. The right and left vertebral arteries
   c. The Circle of Willis
   d. The vertebrobasilar arteries

8. The stroke syndrome caused by occlusion of the basilar artery can result in:
   a. Contralateral facial (VII) cranial nerve palsy.
   c. Blindness in the opposite visual field.
   d. Both expressive and receptive aphasia.

9. During the acute phase, a patient with hemorrhagic stroke is at risk for additional ischemic damage due to:
   a. A decrease of blood released into the cerebrospinal fluid.
   b. A rise in intracranial pressure.
   c. Traumatic injury.
   d. ATP depletion.

10. Following an ischemic stroke, the penumbra can be saved from necrosis if blood supply is restored within:
    a. 6 hours.
    b. 4.5 hours.
    c. 3 hours.
    d. 45 minutes.

11. Ischemic stroke due to reduction of blood flow to a region of the brain is most commonly caused by:
    a. Genetic factors such as sickle cell disease.
    b. Widespread cerebral hypoperfusion.
    c. A ruptured aneurysm near the Circle of Willis.
    d. An arterial blockage associated with thrombi or emboli.
12. Cocaine or amphetamine use is among the most common causes of which type of stroke?
   a. Basilar artery stroke
   b. Incomplete ischemic stroke
   c. Intracerebral hemorrhage
   d. Subarachnoid hemorrhage

13. A facility that is certified as an Acute Stroke-Ready Hospital must provide:
   a. CT, MRI, and lab capability available at all times.
   b. Dedicated neuro-intensive care unit beds.
   c. Capabilities for angioplasty and stenting.
   d. A stroke unit for the acute care of stroke patients.

14. The stroke center time target for the management of acute stroke from door to treatment is:
   a. 15 minutes.
   b. 25 minutes.
   c. 60 minutes.
   d. 3 hours.

15. The most important step for a family member or bystander to take when someone appears to be experiencing a stroke is to:
   a. Drive the person to the nearest hospital.
   b. Immediately call 911.
   c. Wait to see if it is a transient ischemic attack.
   d. Give the person an aspirin.

16. In the Cincinnati Prehospital Stroke Scale, the patient is asked to: “Hold both arms out in front of you,” “Repeat the sentence ‘The sky is blue in Cincinnati,’” and:
   a. “Lie down in a comfortable position.”
   b. “Loosen any tight clothing.”
   c. “Tell me when your symptoms began.”
   d. “Show me your teeth.”

17. En route to the emergency department, emergency medical services (EMS) responders should:
   a. Treat shock or significant dehydration with a 5% dextrose in 0.45 NaCl solution.
   b. Treat arterial hypertension.
   c. Cardiovert atrial fibrillation.
   d. Check glucose level and treat hypoglycemia with 50% dextrose.
18. The two most essential laboratory tests before treatment of acute stroke are blood glucose levels and:
   a. Coagulation studies.
   b. Lipid profile.
   c. Blood typing and crossmatch.
   d. Hematocrit level.

19. The most important piece of historical data to obtain in the medical history of a patient with acute stroke is:
   a. History of hypertension.
   b. Recent trauma.
   c. Time of symptom onset.
   d. History of atrial fibrillation.

20. Which is a correct statement regarding the Glasgow Coma Scale?
   a. It is a tool used to rule out stroke mimics.
   b. It is helpful in identifying candidates for fibrinolytic therapy.
   c. It is a tool used to describe a patient’s level of consciousness.
   d. It focuses on six major areas of the neurological exam.

21. The recommended initial diagnostic imaging in the hyperacute stage of stroke is a:
   a. Multimodal computed tomography scan.
   b. Noncontrast computed tomography (NCCT) scan.
   c. Magnetic resonance imaging (MRI) scan.
   d. Diffusion weighted imaging (DWI) technique.

22. Under the recommendations for treatment of ischemic stroke, recombinant tissue plasminogen activator (rtPA) is:
   a. “Suggested” to be given more than 4.5 hours post stroke.
   b. “Not suggested” to be given during the first 90 minutes post stroke.
   c. “Recommended” to be given between 3 to 4.5 hours after onset of stroke symptoms.
   d. “Recommended” to be given within 3 hours of onset of stroke symptoms.
23. Which condition **excludes** a patient from receiving recombinant tissue plasminogen activator (rtPA)?
   a. Neurological signs not clearing spontaneously
   b. Normal clotting functions
   c. A history of previous intracranial hemorrhage
   d. A history of stroke six years ago

24. The nurse correctly **discontinues** an infusion of recombinant tissue plasminogen activator (rtPA) for a patient with acute stroke who develops:
   a. Acute hypotension and nausea.
   b. Spontaneous clearing of symptoms.
   c. Elevated serum glucose and hypotension.
   d. Acute hypertension or nausea and vomiting.

25. Which is a correct statement concerning treatment with antithrombotics to reduce the risk of a second stroke?
   a. New oral agents are more advantageous than warfarin.
   b. Daily aspirin is no longer recommended for ischemic stroke patients.
   c. Heparin demonstrates significant benefits.
   d. Anticoagulant treatment is recommended for patients with large infarctions.

26. Criteria for receiving endovascular treatment using mechanical thrombectomy include:
   a. Asymptomatic carotid artery blockage.
   b. Acute ischemic stroke with receipt of IV rtPA within 4.5 hours of onset.
   c. Moderate blockage of the carotid artery.
   d. Being at high risk for carotid endarterectomy.

27. Which is a **correct** statement regarding early management of ischemic and hemorrhagic stroke patients?
   a. Elevated blood pressure in ischemic stroke is always an indication for aggressive treatment.
   b. Effective targeted treatment for hemorrhagic stroke is now available.
   c. The necessity for reversing warfarin anticoagulation is a medical emergency.
   d. The recommended treatment for elevated intracranial pressure is hyperventilation.
28. A common complication of subarachnoid hemorrhage is:
   a. Ischemic infarction from vasospasm.
   b. Elevated blood pressure.
   c. Renal impairment due to hypovolemia.
   d. Uncontrolled convulsive seizures.

29. Which is a sign of possible brain herniation in a patient with an acute ischemic stroke?
   a. Appearance of Cushing’s triad
   b. Sudden severe headache
   c. Intracranial rebleeding
   d. Loss of pupillary response

30. Which is a correct statement regarding pulmonary complications following stroke?
   a. The primary prevention of aspiration pneumonia is early initiation of nasogastric feeding.
   b. Intubation decreases morbidity and mortality in stroke patients.
   c. Patients can be hypoxemic without showing clinical symptoms.
   d. Neurogenic pulmonary edema is common following ischemic stroke.

31. The best option for preventing deep vein thrombosis (DVT) in patients with acute intracranial hemorrhage whose mobility is restricted is:
   a. Intermittent pneumatic compression devices.
   b. Low-dose oral anticoagulants.
   c. Aspirin, 81 mg, once a day.
   d. Low-dose intravenous heparin.

32. Which is a correct statement concerning early mobilization of stroke patients?
   a. Studies indicate that very early post-stroke mobilization improves outcomes.
   b. Accepted guidelines call for frequent out-of-bed activity.
   c. Data indicate that it may be harmful to allow the brain to “cool off” following a stroke.
   d. An early, lower-dose activity regimen is preferable to a very early, higher-dose regimen.
33. Which intervention is part of a recommended nursing care plan for a stroke patient with dysphagia?
   a. Have suction equipment available at the bedside.
   b. Allow 10 to 20 minutes for meals.
   c. Maintain head-of-bed elevation at 30 degrees for 60 to 90 minutes after eating.
   d. Use drinking straws instead of cups or glasses.

34. The Functional Independence Measure (FIM) tool is used for patients with stroke to assess:
   a. Motor and cognitive abilities related to activities of daily living.
   b. Grasp, grip, pinch, and gross movement.
   c. Static balance and fall risk in older adults.
   d. Upper limb strength in handling objects differing in size, weight, and shape.

35. By engaging a stroke patient in early out-of-bed mobilization, a physical therapist is primarily targeting the prevention of:
   a. Gait abnormalities.
   b. Poor balance.
   c. Muscle spasms.
   d. Deconditioning.

36. The occupational therapist uses splinting in acute stroke rehabilitation in order to:
   a. Assist with early mobilization.
   b. Prevent contractures.
   c. Manage shoulder pain.
   d. Prevent decubiti.

37. Which is a strategy used by a speech-language therapist to manage a patient with dysphagia following a stroke?
   a. Assessing function and motor abilities
   b. Implementing the use of a letter board
   c. Teaching the patient’s family to feed him/her
   d. Altering the bolus size and consistency

38. Which measure is recommended to prevent secondary stroke in a patient?
   a. Assisting the family with day-to-day management of the patient’s care
   b. Decreasing physical exercise intensity
   c. Ceasing smoking
   d. Avoiding antiaggregant medications
39. Which medication is recommended to prevent secondary stroke in patients who have had an ischemic stroke related to atherosclerosis or embolus?
   a. Vitamin K (Mephyton)
   b. Ibuprofen (Advil)
   c. Lorazepam (Ativan)
   d. Clopidogrel (Plavix)

40. A patient asks how heart disease can affect the brain. The best response would be:
   a. Blood clots caused by an irregular heartbeat can travel to the brain.
   b. Cholesterol plaque that builds up in the heart contributes to the formation of brain aneurysms.
   c. Ventricular fibrillation increases the risk for blood clots to form in the brain.
   d. Heart disease causes a decrease in blood flow to the parts of the brain involved in movement.