LEARNING OUTCOME AND OBJECTIVES: 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. Specific learning objectives to address potential knowledge gaps include:

- Review stroke epidemiology.
- Identify risk factors and triggers for stroke.
- Discuss the major classifications of stroke, including pathophysiology and clinical presentation.
- Describe the components of prehospital and emergency department evaluation and management.
- 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.
- Summarize hospital nursing management for stroke patients beyond 24 hours.
- Identify assessment, interventions, and goals of physical, occupational, and speech-language stroke rehabilitation in the acute setting.
- List actions to be taken in the prevention of secondary stroke.
INTRODUCTION

A stroke—also called a cerebrovascular accident (CVA) or a brain attack—is a reduction or 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

- 1 in 6 people worldwide will have a stroke at some point in their lifetime.
- 15 million people worldwide experience a stroke each year, and 5.8 million people die because of it.
- Every year, more than 795,000 people in the United States have a stroke, and about 610,000 of these are first or new strokes.
- Nearly 1 in 4 (185,000) strokes occur in people who have had a previous stroke.
- Each year about 140,000 Americans die from a stroke (1 of every 20 deaths).
- When considered separately from other cardiovascular diseases, stroke ranks fifth among all causes of death in the United States.
- In the United States, someone has a stroke every 40 seconds, and someone dies of stroke every 4 minutes.
- Stroke remains among the top 10 causes of death in children.
- Approximately 87% of all strokes are ischemic strokes, in which blood flow to the brain is blocked.
- The financial burden of stroke in the United States is estimated to be $34 billion each year, which includes the cost of healthcare services, medicines for treatment of stroke, and missed days of work.
- Stroke is a leading cause of serious long-term disability and reduces mobility in more than half of stroke survivors ages 65 and older. (CDC, 2019; AHA, 2019a; WSO, 2019)
By Age

Stroke can affect people of all ages. Nearly 75% of all strokes occur in people over the age of 65, and the risk of having a stroke more than doubles each decade after the age of 55. Although it is more common among older people, a stroke can happen to anyone at any time, including teenagers, children, newborns, and unborn babies. The risk of stroke in children is greater in the first year of life and during the periods right before and right after birth (AHA, 2019a).

By Gender

Stroke is the third leading cause of death for women and the fifth leading cause of death for men in the United States. Each year 55,000 more women have a stroke than men. One in five women have a stroke, and stroke kills twice as many women each year as breast cancer. Women in general live longer than men, and stroke has a more negative impact on their lives. More women will live alone when they have a stroke, be more apt to live in long-term care facilities, and have a worse recovery following a stroke.

Unique risk factors for women include:

- Past oral contraceptive use
- Increased blood pressure and stress on the heart during a normal pregnancy
- Preeclampsia, which doubles the risk of stroke later in life
- Hormone replacement therapy (HRT)
- Migraine headaches with aura (more common in women), which increases risk for stroke two and a half times
- Atrial fibrillation, which increases stroke risk by 20% among women over age 75 (AHA, 2019a)

By Race and Ethnicity

Blacks have nearly twice the risk of a first stroke as whites, and African Americans have the highest rate of death due to stroke, even at younger ages. Strokes among African Americans tend to occur earlier in life, and these individuals are more likely to become disabled from a stroke. Research indicates the following risk factors for African Americans:

- High prevalence of hypertension and lower likelihood to have it under control than non-Hispanic whites
- High prevalence of diabetes
- Sickle cell anemia (the most common genetic disorder among African Americans) (CDC, 2019; NSA, 2019a)
Hispanics are more likely to have a stroke at a younger age (average age of 67) compared to non-Hispanic whites (average age of 80). Stroke and heart disease cause 1 in 4 deaths among Hispanic men and 1 in 3 deaths among Hispanic women. Factors that increase the risk among this population include:

- High rates of obesity (75% of Hispanic-American men and 72% of women are overweight or obese)
- Diabetes (an estimated 30% of adult Hispanics)
- Higher risk of stroke recurrences and more severe strokes among stroke survivors with atrial fibrillation
- Delay in getting treatment related to language barriers and lack of transportation (NSA, 2019a)

Native Americans/Alaska Natives are 2.4 times more likely to have a stroke than whites, and stroke is the sixth leading cause of death. High blood pressure, diabetes, obesity, and smoking among these groups are the main factors that increase their stroke risk (NSA, 2019a).

Native Hawaiians/Pacific Islanders have been shown to have a higher prevalence of major stroke risk factors (hypertension, diabetes, and obesity) and are four times more likely to die from stroke than non-Hispanic whites (NSA, 2019a).

Asian Americans are less likely than most other minority racial/ethnic groups to die from a stroke. Overall, they have lower rates of hypertension, overweight, and obesity, and they tend to have healthier lifestyles. However, research indicates they are still 20% more likely to have a stroke than whites (NSA, 2019a).

**By Geographic Location**

An eight-state region in the southeastern United States is known as the “stroke belt.” It has been so designated because of disproportionately high stroke mortality rates, present since at least 1940, despite overall recent decreases in stroke mortality. These states include:

- Alabama
- Arkansas
- Georgia
- Louisiana
- Mississippi
- North Carolina
- South Carolina
- Tennessee
The underlying issues for higher stroke mortality in this region are not fully understood. It is thought that differences in vascular risk factors play a role. Access to primary stroke centers is also lower within the region, suggesting that differential access to care could be one important contributing factor (Karp et al., 2016).

**Effects of Stroke**

Receiving a diagnosis of stroke is frightening. A stroke can have profound effects on the body as well as the mind and emotions. The effects of a stroke depend on several factors, including the location of the obstruction or hemorrhage and how much brain tissue has been affected.

Because one side of the brain controls the opposite side of the body, a stroke affecting one side of the brain will cause neurological complications on the opposite side of the body. A stroke occurring in the **left side** of the brain will result in some or all of the following:

- Weakness, numbness, stiffness, or paralysis on the right side of the body
- Speech/language problems (aphasia, also called dysphasia)
- Slow, cautious behavioral style
- Cognitive changes
A stroke occurring in the right side of the brain will result in some or all of the following:

- Weakness, numbness, stiffness, or paralysis on the left side of the body
- Vision problems
- Quick, inquisitive behavioral style
- Cognitive changes

When a stroke occurs in the brainstem, depending on how severe the injury is, both sides of the body may be affected, and the person may be left in what is referred to as a “locked-in” state. When this occurs, the patient is unable to speak or execute any movements below the neck (AHA, 2019b).

Damage to the brain following a stroke can result in many cognitive changes. These may include problems with reasoning, planning, judgment, memory, or other thought processes and are known as vascular dementia.

It is common for emotional and behavioral changes to occur as a result of a stroke. A stroke can impact mood and outlook. Mood disorders such as depression and anxiety are common, with depression affecting between one third and two thirds of stroke survivors.

Pseudobulbar affect (PBA) is the name of a neurological condition that can be caused by a stroke. It is also known as emotional lability, reflex crying, and involuntary emotional expression disorder, among other names. PBA is more common in survivors of brainstem stroke and is characterized by a mismatch between feelings and expression, such as laughing at a funeral or crying at something that is funny (AHA, 2019b).

RISK FACTORS AND TRIGGERS FOR STROKE

Stroke risk factors have been categorized as either nonmodifiable or modifiable.

Nonmodifiable Risk Factors

- Age: The incidence of stroke increases with age, doubling for each decade after 55 years.
- Gender: The relationship of gender to stroke risk depends on age. At young ages, women have as high or higher risk of stroke as men, but at older ages the risk is slightly higher for men.
- Race/ethnicity: Disparities among racial and ethnic groups are well-documented (see above).
- Genetics: Family history of stroke increases stroke risk by 30%. Hereditary factors contribute to stroke risk, although it is challenging to determine whether risk is due to genetic mutations and/or due to shared familial exposures.
• Personal past history of stroke: About one quarter of strokes each year are recurrent events.

• Fibromuscular dysplasia: This medical disorder involves fibrous tissue growth in artery walls, which causes them to narrow.

• Brain aneurysms or arteriovenous malformations (AVMs): Aneurysms are bulges in an artery that can stretch and burst, and AVMs are tangles of faulty arteries and veins that can rupture.

• Patent foramen ovale (PFO): About 1 in 5 Americans has a PFO (hole in the heart), which puts them at an increased risk for stroke and transient ischemic attack.

• Transient ischemic attack (TIA): This brief episode of stroke-like symptoms (lasting from a few minutes to 24 hours but with no permanent damage or disability) increases the risk of stroke as much as 17% within 90 days of a TIA, with greatest risk in the first week post TIA.
  (NHLBI, 2018; NSA, 2019c)

**Modifiable Risk Factors**

• Hypertension: Elevated blood pressure is the *leading cause of stroke* and the most important controllable risk factor for stroke.

• Metabolic disorder: Diabetes mellitus doubles the risk for stroke, and stroke accounts for approximately 20% of deaths in people with diabetes.

• Cigarette smoking: Smoking remains a major risk factor, nearly doubling the risk and contributing to approximately 15% of all stroke deaths each year. Smoking cessation quickly reduces the risk, with excess risk nearly disappearing 2 to 4 years after smoking cessation. Secondhand smoke has also been identified as an independent risk factor.

• Cardiac causes: Atrial fibrillation and atrial cardiopathy are associated with cardioembolic strokes. Atrial fibrillation increases the risks fivefold. Sleep apnea can be linked to atrial fibrillation and is also associated with increased stroke risk.

• Dyslipidemia: The relationship between dyslipidemia and stroke risk is complex, with an increased risk for ischemic stroke with elevated total cholesterol and a decreased risk for ischemic stroke with elevated high-density lipoprotein (HDL) cholesterol.

• Sedentary behavior: The relationship between physical activity and stroke may be due to its association with decreased blood pressure, reduction in diabetes mellitus, and reduction in excess body weight.

• Diet/nutrition: Diet influences the risks of stroke and other stroke risk factors, including diabetes mellitus, hypertension, and dyslipidemia. Some components are well known, such as salt intake (increased hypertension) and potassium intake (associated with decreased stroke risk).
• Obesity/waist-to-hip ratio: Obesity is related to other stroke risk factors such as hypertension and diabetes mellitus. The major contributor to risk is waist-to-hip ratio rather than overall increased weight as indicated by body mass index.

• Alcohol consumption: The relationship of alcohol to stroke risk depends on the stroke type. Alcohol consumption has a more direct linear relationship with hemorrhagic stroke, and even small amounts of alcohol seem to increase risk of hemorrhages. Heavy drinking is associated with an increased risk of ischemic stroke.

• Drug abuse: Abuse of illicit substances, including cocaine, heroin, amphetamines, and ecstasy, is associated with an increased risk of ischemic and hemorrhagic subtypes of strokes.

• Inflammation: Levels of inflammatory biomarkers have been associated with increased risk of stroke, but the reasons for this association are uncertain. The inflammatory character of atherosclerotic plaque may be a contributing factor.

• Infections: Data suggest that chronic exposure to common bacterial and viral infections is a risk factor for stroke and may act as a trigger for stroke. Research has found that the risk of stroke increased after respiratory tract infection but was reduced after vaccination against influenza, pneumococcal infection, and tetanus. (NHLBI, 2018; NSA, 2019c)

Stroke Triggers

Although there is a good understanding of the major stroke risk factors listed above, what triggers a stroke to occur at a particular point in time remains to be understood. A new area of investigation in stroke epidemiology involves the determination of such stroke triggers. At this time, they may include:

• Infection: Recent infection (bacterial or viral) may be associated with new-onset atrial fibrillation.

• Air pollution: Pollutant levels, primarily particulate matter, is associated with an increase in same-day stroke hospital admissions. Similar results have been seen with carbon monoxide, nitrogen dioxide, and sulfur dioxide. Other studies have identified an increase of stroke by 6% and stroke mortality by as much as 12.5%. (Boehme et al., 2017)
PATHOLOGIES UNDERLYING STROKES

The primary pathologies underlying stroke are heart or blood vessel diseases, and the secondary manifestations in the brain are the result of one or more of these underlying diseases or risk factors.

Heart conditions may include:

- Atrial arrhythmias (fibrillation, flutter)
- Mitral or aortic valve disease
- Prosthetic and mechanical heart valves
- Sinus node dysfunction
- Recent myocardial infarction
- Congestive heart failure
- Cardiomyopathy

Blood vessel diseases may include:

- Atherosclerosis
- Hypertension
- Noninflammatory blood vessel disorders
  - Fibromuscular dysplasia
  - Vasospasm
  - Reversible cerebral vasoconstriction syndromes
  - Radiation-induced vasculopathy
  - Fabry disease
- Inflammatory blood vessel disorders
  - Isolated angiitis
  - Temporal (giant cell) arteritis
  - Cerebral vasculitis related to infection, toxins, or neoplasms
- Hematological disorders
  - Inherited and acquired blood clotting disorders
  - Prothrombotic disorders
  - Polycythemia vera
  - Genetic mutations causing disorders of the coagulation system
  - Antiphospholipid antibody syndrome
  - Sickle cell disease

(AHA, 2019c, 2016)
In order to function normally, the brain depends on receiving adequate oxygen and nutrients through a network of blood vessels. Two major sets of vessels supply blood to the brain. The anterior circulation of the brain is supplied by the right and left common carotid arteries, and the posterior portion of the brain is supplied by the right and left vertebral arteries. Every minute, about 600–700 ml of blood flow through the carotid arteries and their branches, and 100–200 ml flow through the vertebral-basilar system.

The right common carotid artery originates from the bifurcation of the brachiocephalic trunk, while the left common carotid artery originates directly from the aortic arch. Each then branches to form the external and internal carotid arteries. The external carotid arteries supply blood to the face and scalp, and the internal carotid arteries supply blood to most of the anterior portion of the cerebrum.

The vertebral arteries arise from the subclavian arteries and run alongside the medulla, giving rise to branches that supply the cervical spinal cord as well as the brainstem. They end by fusing to form the basilar artery. The vertebra-basilar arteries supply the posterior two fifths of the cerebrum, part of the cerebellum, and the brainstem.
The anterior and posterior circulations communicate through a circular anastomosis of arteries called the **circle of Willis**, which is located at the base of the brain and serves as an effective collateral circulation (UMass/ASA, 2018). The circle of Willis begins to form when the right and left internal carotid arteries (ICAs) enter the cranial cavity and each one divides into two main branches, the anterior cerebral artery (ACA) and the middle cerebral artery (MCA). The anterior cerebral arteries are then connected by the anterior communicating (ACOM) artery. Posteriorly, the basilar artery (BA) formed by the left and right vertebral arteries branches into left and right posterior cerebral arteries (PCA). The PCAs complete the circle of Willis by connection to the ICAs via the posterior communicating (PCOM) arteries (Gupta, 2017).

**TYPES OF STROKE**

The two major categories of stroke—**ischemic and hemorrhagic**—are diametrically opposite conditions, each resulting from underlying pathophysiological states. Two other subtypes of stroke are transient ischemic attack (TIA) and cryptogenic stroke.

Two primary types of stroke—hemorrhagic and ischemic. (Source: CDC, 2018.)
Ischemic Stroke

Most strokes (87%) are ischemic. They are characterized by the sudden loss of blood circulation to a specific area of the brain caused by an occlusion of a cerebral artery, resulting in a corresponding loss of neurologic function. Ischemia results in the loss of oxygen and nutrients to the brain cells, and local blood flow is limited to any residual flow in the major arterial source plus the collateral supply, if any (ASA, 2019).

Ischemic strokes may occur in three ways:

- **Thrombotic stroke:** Thrombotic strokes are responsible for almost 50% of all strokes. Cerebral thromboses are clots that form in the cerebral arterial tree. Blood clots usually form in arteries that are damaged by atherosclerotic plaque but may also be due to arterial dissection or fibromuscular dysplasia, or an inflammatory condition. 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) arising from outside the cerebral arterial tree that block arterial access to a particular brain region.

- **Systemic hypoperfusion:** This is a more general circulatory problem that manifests itself in the brain and other organs and may be the result of cardiac pump failure or reduced cardiac output related to acute myocardial ischemia, pulmonary embolism, pericardial effusion, or bleeding (Caplan, 2016; Hui et al., 2019).

**PATHOPHYSIOLOGY OF ISCHEMIC STROKE**

Interruption of blood flow through an intracranial artery leads to deprivation of oxygen and glucose in the supplied vascular territory. This initiates a cascade of events at a cellular level that, if circulation is not reestablished in time, will lead to cell death, mostly through liquefactive necrosis (Carrol & Gaillard, 2019).

Following a stroke, the affected areas of the brain that receive blood flow of less than 10 ml per each 100 grams of tissue per minute are referred to collectively as the core. The cells in the core are presumed to die within minutes of stroke onset. Zones of decreased or marginal perfusion with less than 25 ml per each 100 grams of tissue per minute are collectively called the ischemic penumbra. The tissue in the penumbra can remain viable for several hours because of this marginal tissue perfusion (Jauch, 2019).
ISCHEMIC PENUMBRA PROVIDES EARLY THERAPEUTIC WINDOW

After an ischemic stroke in which the ischemic penumbra has not yet been damaged structurally, permanent structural damage may be prevented if prompt restoration of perfusion in the penumbra can be restored. Collateral and residual blood flow can preserve neurons in the penumbra and border areas for as long as six hours after an ischemic stroke, and within this six-hour window, certain treatments can reduce the amount of damage that is irreversible (Cuccione et al., 2016).

Embolic stroke due to a thrombus formed in the atrium. (Source: NHLBI.)
Evolution of Ischemic Stroke

The temporal evolution of an ischemic stroke occurs in four stages:

1. **Hyperacute** (0 to 24 hours): Growth of the lesion begins immediately, with the largest volume of tissue infarction occurring within 10 minutes. By 30 minutes over half of the total infarct volume occurs.

2. **Acute** (24 hours to 1 week): The involved area is soft and edematous and there is a blurring of anatomic detail.

3. **Subacute** (1 week to 1 month): There is obvious tissue destruction and liquefactive necrosis of the involved brain.

4. **Chronic** (>1 month): The damaged tissue has been phagocytized and there is cavitation with surrounding gliosis, which leads to scarring. A glial scar is the body’s mechanism to protect and begin the healing process in the nervous system.

Microscopically there is also a temporal evolution of an ischemic stroke.

1. 0–48 hours: Chromatolysis (disintegration of chromophil granules in a neuron) and swollen eosinophilic neurons (anoxic neurons) are seen.

2. 24–72 hours: Neuronal cell necrosis and acute inflammatory response are seen.

3. 3–5 days: An influx of mononuclear cells begins to phagocytize necrotic debris.

4. 1–2 weeks: Vascular proliferation and reactive astrogliosis occurs whose function is to maintain a neuron’s working environment.

5. Over 1 month: Necrotic tissue will be completely removed and a cystic cavity surrounded by a glial scar will be formed.

(ISC, 2019a)

**CLINICAL PRESENTATION OF ISCHEMIC STROKE**

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. Most patients are involved in normal daily activities and notice these common symptoms:

- Sudden numbness or weakness of the face, arm, or leg, especially involving one side of the body
- Sudden confusion or trouble speaking or understanding
- A change in the vision of one or both eyes that occurs suddenly with no known cause
- A quick onset of dizziness, loss of coordination/balance, or other problems walking
- Sudden, severe headache with no known cause
Some patients may also experience a sudden loss of consciousness, fainting or a seizure without a known cause, and vomiting or fever that occurs within minutes or hours that cannot be explained by another cause. In large vessel ischemic stroke, headache may occur prior to, during, or following stroke onset.

The effects of an acute ischemic stroke may cause additional symptoms in women, including:

- Face, arm, or leg pain
- Hiccups or nausea
- Chest pain or palpitations
- Shortness of breath
  (Beaumont, 2018; Ramzan & Fisher, 2019)

**ISCHEMIC STROKE SYNDROMES**

Specific neurological functions are dependent on specialized brain regions, with each artery primarily supplying a particular region. Thus, occlusion in 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.

![Brain regions and associated neurological functions. (Source: Activase Image Library.)](image-url)
**Anterior Cerebral Artery (ACA) Stroke Syndrome**

The anterior cerebral artery provides blood to the medial portion of the brain: frontal, prefrontal, primary motor, primary sensory, and supplemental motor cortices. The sensory and motor cortices receive sensory information and control movement of the contralateral lower extremity. The supplemental motor area contains Broca’s area, which is involved in initiation of speech. The prefrontal cortex is involved in organization and planning of complex behavior and may influence the personality.

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
- Bilateral leg weakness if both ACAs are involved
- 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 (power or faculty of using one’s will), motivation, planning, and organizing complex behavior
- Disinhibition
- 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—inaibility to directly share information between the two hemispheres) (Jin, 2017; UBC, 2016)

**Middle Cerebral Artery (MCA) Stroke Syndrome**

Two thirds of ischemic strokes occur in the MCA because of the size of the territory and the direct flow from internal carotid artery into the middle cerebral artery. This provides the easiest path for thromboembolism. Clinical presentation will depend on the extent of the infarct and hemispheric dominance. MCA covers a large portion of the hemisphere and can involve the frontal, temporal, and parietal lobes and can also involve the basal ganglia. 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
  - Alexia (inability to read)
  - Acalculia (loss of ability to do simple arithmetic calculations)
  - Agraphia (loss of ability to write)
- If the nondominant (usually right) MCA has been occluded:
  - Contralateral neglect (hemineglect)
  - Unawareness or denial of their neurological deficits (anosognosia)
  - Flat affect
  - Loss of prosody of speech (intonation, stress pattern, loudness variations, pausing and rhythm)

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 (Jin, 2019; 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**

The PCA supplies blood to multiple brain regions (occipital lobe, inferomedial temporal lobe, a large portion of the thalamus, and the upper brainstem and midbrain.)
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)
- Achromatopsia (difficulty perceiving colors)
- Hallucinations (uncommon)
- Palinopsia (seeing images persist even after image is removed)
- Prosopagnosia (difficulty recognizing familiar faces)
- Confusion
- Alteration in consciousness
- New onset posterior cranium headache
  
  (El-Feky & Bronson, 2019; Kuybu et al., 2019)

**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 (repetitive, uncontrolled eye movement)
- Vomiting
- Ipsilateral (same-sided) ataxia
- Hypoglossal nerve dysfunction resulting in:
  - Dysarthria (unclear speech)
  - Dysphagia (difficulty swallowing)
  - Difficulty chewing
• Ataxia (lack of voluntary coordination of muscle movements) 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**

Occlusion of the basilar artery may cause brainstem or thalamic ischemia, and is rare (<1% of all strokes). If not treated early, brainstem infarction results in rapid deterioration in the level of consciousness and, ultimately, death. Basilar artery stroke presents with sudden and dramatic neurological impairment, the exact characteristics dependent upon the site of occlusion:

• Sudden death/loss of consciousness
• Bilateral sensory deficits
• Motor dysfunction often absent
• Visual and oculomotor deficits
• Combined cerebellar and cranial nerve problems
• Hemiparesis with contralateral cranial nerve dysfunction or with ipsilateral ataxia
• Behavioral abnormalities
• Somnolence, hallucinations, and dream-like behavior

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)
  (Sharma & D’Souza, 2019)
CRYPTOGENIC STROKE

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 (Prabhakaran & Elkind, 2019).

Transient Ischemic Attack (TIA)

Different from the major types of stroke, a transient ischemic attack (sometimes referred to as a mini-stroke) is a brief interruption of blood flow, most often caused by thrombosis, to part of the brain, spinal cord, or retinas. TIA may cause temporary stroke-like symptoms but does not damage brain cells or cause permanent disability. There is brief neurological dysfunction, with clinical symptoms typically lasting less than one hour and without evidence of acute infarction.

Patients with TIA or mild stroke are at risk for developing stroke in the near future: 10% to 15% of patients will have a stroke within three months, with half occurring within 48 hours. A TIA precedes approximately 15% of all strokes, and up to 25% of people who suffer a TIA die within one year (ASA, 2018a; CDC, 2018).

CLINICAL PRESENTATION OF TIA

A person experiencing a transient ischemic attack may have one or more of the following signs or symptoms:

- Weakness or numbness in the arms and/or legs, usually on one side of the body
- Dysphasia
- Dizziness
- Vision changes
- Paresthesias (tingling)
- Abnormal taste and/or smells
- Confusion
- Loss of balance
- Altered consciousness and loss of consciousness
  (CDC, 2018)

Hemorrhagic Stroke

Intracranial bleeding caused by a blood vessel within the cranium that has leaked or ruptured is called a hemorrhagic stroke. Hemorrhagic strokes are less common than ischemic strokes,
making up about 15% of all strokes. They are, however, responsible for about 40% of all stroke deaths (NSA, 2019c). There are two types of hemorrhagic strokes:

- **Intracerebral hemorrhage (ICH)**, the most common type, occurs when a blood vessel within the brain ruptures.
- **Subarachnoid hemorrhage (SAH)** refers to bleeding in the subarachnoid space, the area between the brain and the meninges that cover it.

**PATHOPHYSIOLOGY OF HEMORRHAGIC STROKE**

An intracranial hemorrhage is most commonly the result of hypertension. Bleeding occurs suddenly and rapidly. There are usually no warning signs, and bleeding can be severe enough to cause coma or death. Subarachnoid hemorrhage is often due to an aneurysm or an arteriovenous malformation (AVM) but can also be caused by trauma.

An **aneurysm** is a focal dilatation of arteries, which can be congenital or develop later in life due to factors such as hypertension and atherosclerosis. The most frequently encountered type is the berry (or saccular) aneurysm. Berry aneurysms are most often isolated lesions whose formation results from a combination of hemodynamic stresses and acquired or congenital weakness in the vessel wall. These aneurysms typically occur at vascular bifurcations, with more than 90% occurring in the anterior circulation of the brain. Because of the weakness in the vessel wall, an abnormal widening, ballooning, or bleb (blister, often hemispherical) develops and there is a risk for rupture. When the vessel bursts, blood is released into the brain tissue (Liebeskind, 2019).

Aneurysmal subarachnoid hemorrhage is a devastating event. Approximately 10% of patients die prior to reaching the hospital, 25% die within 24 hours of SAH onset, and about 45% die within 30 days; only one third of patients have a good outcome after treatment (Rordorf & McDonald, 2019).
Arteriovenous malformations (AVMs) are dilated tangled blood vessels in which the arterial blood flows directly into the venous system, bypassing the capillary bed within the brain tissue or on its surface. Numerous genetic causes may predispose to AVM in the brain, and more than 50% of patients with an AVM have an intracranial hemorrhage. The abnormal and weak blood vessels dilate over time and may eventually burst from the high-pressure flow from the arteries (Liebeskind, 2019; NSA, 2019b).

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 intracranial pressure (ICP) can 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).

CLINICAL PRESENTATION OF HEMORRHAGIC STROKE

Symptoms of intracerebral hemorrhage often begin with a sudden headache occurring during activity. However, headache may be mild or absent in the older adult. Loss of consciousness is common, often within seconds or a few minutes. Nausea, vomiting, delirium, and focal or generalized seizures are common.
Neurologic deficits usually are sudden and progressive. Large hemorrhages, when occurring in the hemispheres, cause hemiparesis; when occurring in the posterior fossa, they cause cerebellar or brainstem deficits, such as stertorous (low-pitched, nonmusical) breathing, pinpoint pupils, coma, or conjugate eye deviation.

A large intracerebral hemorrhage is fatal within a few days in approximately one half of patients. In those who survive, consciousness returns and neurologic deficits gradually diminish to different degrees as the blood is resorbed. Some patients may have only a few neurologic deficits due to the fact that hemorrhage is less destructive to the brain tissue than an infarct.

Small hemorrhages may result in focal deficits with no impairment of consciousness and with no headache or nausea, and they may mimic ischemic stroke (Giraldo, 2019a).

Symptoms of subarachnoid hemorrhage begin abruptly with a severe headache (often referred to as a thunderclap headache), which peaks within seconds. Pain may radiate into the neck and even down the back into the legs. Vomiting occurs soon after onset. Loss of consciousness may follow, usually immediately but sometimes not for several hours. Severe neurologic deficits may develop and become irreversible within minutes or a few hours. Sensorium may be impaired, and the patient may become restless. There is a possibility of seizures, and the heart or respiratory rate is often abnormal (Giraldo, 2019b).

PREHOSPITAL MANAGEMENT OF ACUTE STROKE

Because fast recognition and treatment of a stroke can reduce the possibility of death and long-term disabilities, the American Heart Association developed the “Stroke Chain of Survival.” This chain involves eight links or steps that should be taken by patients, family members, and prehospital and emergency room personnel in caring for stroke patients. This approach can be an effective way to make certain that appropriate care is delivered as rapidly as possible, increasing the odds for a full recovery. The eight links include:

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

Prehospital management of acute stroke involves the first three links of the chain: detection, dispatch, and delivery (ACLS, 2019).
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 recognition of potential strokes. This information should include the following symptoms:

- Sudden numbness or weakness of face, arm, or leg, especially on one side of the body
- Sudden confusion or trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden severe headache with no known cause
  (NINDS, 2019a)

Classic signs of a stroke. (Source: NIH/NINDS.)

If they are experiencing any of these symptoms, patients should call 911 or get someone else to call 911 (see below).

BARRIERS TO RECOGNIZING A STROKE IN ONESELF

Even people who know the warning signs may not realize 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 (NINDS, 2019b).

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 bringing an emergency medical service (EMS) team to the patient is the most important action to take for a stroke victim.

In an emergency, people often believe that time is being lost by waiting for an EMS team to arrive, and so family members or bystanders often hurriedly drive patients to the hospital. In fact, patients usually get to the appropriate hospital more quickly if they use the EMS system by calling 911. EMS teams are trained to choose the most appropriate hospital in the region, which may not be the closest hospital. In addition, the care and assessment that an EMS team provides a stroke victim shortens the time lag between the onset of stroke symptoms and the evaluation and treatment of the stroke.

When calling 911, it is important to:

• Provide the emergency dispatch operator with the location of the emergency.
• If calling from a cell phone, provide the operator with the wireless phone number so the emergency operator can call back in case the call gets disconnected.
• Remember that many emergency operators currently lack the technical capability to receive texts, photos, and videos.
• Learn and use the state’s designated number for highway accidents or other non-life-threatening incidents.

The FCC’s basic 911 rules require wireless service providers to transmit all 911 calls to a Public Safety Answering Point (PSAP) regardless of whether the caller subscribes to the provider’s service or not. Phase II E911 rules require wireless service providers to provide the latitude and longitude of callers to PSAPs. This information must be accurate to within 50 to 300 meters depending on the type of location technology used (FCC, 2018).
The Role of Emergency Response

EMS DISPATCHERS (PUBLIC SAFETY TELECOMMUNICATORS)

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 with speed to bring EMS to the patient

Dispatchers play a key role in the diagnosis of stroke. EMS dispatchers are the first medical contact the patient has. Their job is to interrogate the caller about the presence or absence of priority symptoms. 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 patient
- Getting critical background information about the patient
  (ADH, 2018)

**Identifying the Problem**

With a few key questions, EMS dispatcher can respond by alerting an EMS team and shorten time-critical response. Using a stroke diagnostic tool, such as FAST (see below), the dispatcher will ask the patient (or ask the caller to ask the patient) to:

- Smile to check for facial drooping
- Raise both arms to check for weakness or paralysis on either side
- Repeat a simple phrase such as “the early bird catches the worm” to hear if speech is unusual

Patients are scored based on their response. If the score is high, it is more likely the person is having a stroke.
FAST STROKE ASSESSMENT TOOL

The mnemonic **FAST** (also known as the *Cincinnati Prehospital Stroke Scale + Time*) is an easy way for EMS dispatchers to remember the sudden signs of stroke.

**F** – **Face drooping.** Ask the person to smile. Is the person’s smile uneven?

**A** – **Arm weakness.** Ask the person to raise both arms. Is one arm weaker or numb? Does one drift downward?

**S** – **Speech difficulty.** Ask the person to repeat a simple sentence such as, “The sky is blue.” Is speech slurred? Is the person unable to speak or hard to understand? Is the sentence repeated correctly?

**T** – **Time.** Take note of the time in order to report when the first symptoms appeared.

(See also the box “Cincinnati Prehospital Stroke Scale” later in this course.)

If the caller is someone other than the patient, the dispatcher will ask:

- Is the person conscious and breathing?
- What does the person look like? Does the face look uneven?
- Does the person have a sudden loss of balance?
- Can the person respond to you and follow simple commands?
- Can the person answer your questions?
- Is the person able to speak in full sentences?
- Is the person’s speech slurred?
- Is the person complaining of pain?
- Is the person diabetic?
- Has the person had a seizure recently?
- Has the person had a severe headache recently?

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 (ADH, 2018).
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 are given a priority dispatch requiring the same level of emergency treatment as heart attacks and trauma. American Heart Association/American Stroke Association 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. 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 (Powers et al., 2018).

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 (Powers et al., 2018).

Advising on Possible First Aid

Following determination of priority and dispatch of EMS, the dispatcher offers prearrival instructions, which can include:

- If the caller is the patient, instruct him or her to lie down.
- If the person is unconscious, provide instructions on airway control.
- Keep the person calm and reassure the person that help is on the way.
- Do not allow the person to move around.
- If the person is having difficulty breathing, keep the neck straight and remove pillows.
- Cover the person to prevent heat loss.
- Do not give the person anything to eat or drink.
- Gather the person’s medications (if any).
- Do not give the person any medications, including aspirin.
- Unlock the doors to allow EMS quick entry.
- If anything changes or the person’s condition worsens, call back immediately. (ADH, 2018)
Collecting Critical Information

When an EMS operator suspects that a call concerns an individual experiencing a stroke, the operator also 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.

- Past medical or surgical history
- Past history of a stroke
- Recent trauma or injury
- Time the person was last known well (LKW) without any symptoms of stroke
- Medications the person is currently taking

(ADH, 2018)

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

EMS best practice states that responders take 10 minutes or less on the scene to complete an assessment and begin transport. Upon arrival at the scene, EMS responders initially manage CABs (chest compressions, airway, breathing) and give oxygen if needed to bring O₂ saturation to 94% or greater, and then complete an assessment. Point-of-care testing for glucose can also help rule out hypoglycemia as a common stroke mimic (Colmer et al., 2018).

Confirming a Possible Stroke

A prehospital stroke assessment is completed using an assessment tool. The tool most commonly used is the Cincinnati Prehospital Stroke Scale (CPSS) (see below), a simple three-item scale based on the National Institutes of Health Stroke Scale and designed specifically for use by EMS. Another tool, the Los Angeles Prehospital Stroke Screen (LAPSS), comprises multiple elements, including the history, blood glucose, and specific physical findings.

Most of the prehospital stroke assessment tools can be performed in less than one minute, and use of these tools has been shown to increase paramedic sensitivity to stroke identification to ≥90% (NSA, 2019c).
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 does not 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
   (Kothara et al., 1999)

Determining Stroke Severity

When a potential stroke has been confirmed, a stroke severity tool is utilized to differentiate a patient with large vessel occlusion from one without. This distinction is critical for EMS when determining the best destination hospital. Such assessment tools include:

- RACE (Rapid Arterial Occlusion Evaluation Scale)
- FAST-ED (Field Assessment Stroke Triage for Emergency Destination)

First responders and emergency personnel can also access a mobile application (app) to assess the severity of the stroke using one of several stroke scales. These scales measure certain physical indicators, which may include the ability to squeeze and release a hand, control eye movement, make facial expressions, feel a pin prick, and more. Based on results from the stroke scale, the app recommends the type of facility where a stroke patient can receive appropriate treatment (SNIS, 2019).

Collecting Critical Background Information

Regardless of the information provided to the responders that has been collected by the 911 dispatcher, EMS responders attempt to collect other essential information about the patient. The history is direct and focused to prevent delaying transport. A medication list with a focus on anti-coagulation is obtained.
It is particularly important to determine when the patient was **last known well (LKW)**, since time is important in determining treatment. A patient who woke up with new symptoms should be considered LKW at the last time he or she was seen awake, even if that was the evening prior.

Because time is of the essence, responders also 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 is helpful if next of kin are immediately available for consent (NSA, 2019c).

**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 fully 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 the assessment indicates the patient is experiencing a stroke. While the patient is being prepared for transport, critical background information is obtained and a stroke severity assessment is completed by one of the team using the tool **FAST-ED**. The team is able to quickly transport the patient, whose vital signs remain stable, in under 10 minutes to the nearby 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.

**Transport and Delivery**

One of the most important components of stroke care EMS provides is a prenotification report to the receiving hospital. Prenotification allows the receiving institution to activate local protocols, ready necessary medications, prepare and hold the CT scanner, and prepare to assess the patient as soon as they arrive (Colmer et al., 2018).
**Additional Care En Route**

Instructions for care en route can include:

- Assess and reassess ABCs. Do not treat hypertension unless directed by medical command.
- Perform cardiac monitoring. Do not delay transport to obtain a 12-lead ECG.
- Provide oxygen to maintain oxygen saturation 94%. Routine oxygen administration is not indicated.
- Perform blood glucose assessment. Treat if less than 60 mg/dL. Do not treat with oral medication. Maintain strict NPO.
- Establish IV access. Do not administer excess fluid or glucose.

(ASA, 2019c; Colmer et al., 2018)

**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.

**EMERGENCY DEPARTMENT STROKE EVALUATION AND MANAGEMENT**

EMS delivery ideally involves transporting the patient to the nearest facility with appropriate stroke resources. Acute stroke treatment protocols involve specialized knowledge and practical experience. However, the facilities, equipment, and personnel for acute stroke management are expensive and are not available at most hospitals.

Emergency department care addresses these links in the stroke chain of survival:

- **Data:** Obtaining laboratory results, performing physical and neurological exams,
and brain imaging

- **Decision:** Determining appropriate treatment
- **Drug:** Administering drug therapy if appropriate

### Types of Stroke Care Facilities

Facilities capable of providing stroke care all 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 to 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. A specific individual, often an advanced practice nurse, leads the initiative and is responsible for organizing the process (Jacobson, 2017).

The support team includes members from many disciplines, including neurology, emergency medicine, neurosurgery, nursing, radiology, pharmacy, laboratory, physical medicine, and rehabilitation. It consists of physicians, nurses, physical therapists, occupational therapists, speech and language therapists, and social workers. Best practice indicates that a vascular neurologist serve as leader in the stroke unit (Jacobson, 2017).

The American Stroke Association and The Joint Commission (TJC) classify hospitals into four categories based upon the level of care they are able to provide for stroke patients:

1. Acute stroke-ready hospital
2. Primary stroke center
3. Thrombectomy-capable stroke center
4. Comprehensive stroke center

### ACUTE STROKE-READY HOSPITAL (ASRH)

Acute stroke-ready hospitals tend to be smaller hospitals located in rural and suburban areas. An acute stroke-ready hospital differs from a non-stroke center in that they have around-the-clock access to stroke expertise (either by telephone or in person) and the ability to administer IV thrombolytics prior to transferring a patient for more advanced care.

TJC certification for these facilities requires that they 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, providing educational opportunities toprehospital personnel

© 2019 WILD IRIS MEDICAL EDUCATION, INC.
• Initial assessment performed by an emergency department physician, nurse practitioner, or physician assistant
• CT, MRI (if used), and laboratory capability 24/7
• Access to a neurologist 24/7 in person or by telemedicine
• Neurosurgical services available within three hours (through transfer)
• IV thrombolytic administration capability
• Transfer protocols with one primary stroke center (PSC) or comprehensive stroke center (CSC) (see below)
• Telemedicine available within 20 minutes
• Required ED staff education minimum of twice a year, core team at least 4 hours annually
• Educational opportunities to prehospital personnel
• Use of three inpatient and two outpatient standardized measures to evaluate clinical performance

Following initial certification, recertification is required every two years following an onsite review (TJC, 2018).

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, and 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, laboratory, CTA, MRA availability 24/7; at least one modality for cardiac imaging when needed
• Access to neurosurgical services within two hours or availability 24/7 in a PSC that provides neurosurgical services
• Telemedicine available
• Transfer protocols for neurosurgical emergencies
• Required ED staff education a minimum of twice a year, core team at least 8 hours annually
• 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
Following initial certification, recertification is required very two years following an onsite review (TJC, 2018).

**TELESTROKE CONSULTATION**

Ideally, the treatment of all acute strokes is provided in PSCs or higher-level facilities, 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 emergency medicine doctors to recommend diagnosis and treatment that can be given in the local community. This avoids the need for transfer to another medical center, thereby reducing the delay between recognition of stroke and appropriate treatment.

Like a direct onsite consultation, physicians and patients communicate using digital video cameras, internet telecommunications, robotic telepresence, smartphones, tablets, and other technology.

In telestroke, many people work together as a team, including a program manager, clinical coordinator, vascular neurologists, neurosurgeons and radiologists at the distance site, and emergency medicine physicians and other staff at the originating site. Radiology technicians, informational technology staff, researchers, nurses, nurse practitioners, and other staff also are important members of the team (Mayo Clinic, 2018a).

**THROMBECTOMY-CAPABLE STROKE CENTER**

A thrombectomy-capable stroke center is a facility that has performed mechanical thrombectomy and postprocedure care for at least 15 patients with ischemic stroke in the past 12 months or at least 30 patients over the past 24 months. The requirements for this certification are the same as those of a PSC, with the following exceptions and additions:

- Program medical director with a neurology background and ability to provide clinical and administrative guidance to program
- Dedicated neuro-intensive care beds for complex stroke patients available and onsite critical care coverage 24/7
- Neurologist accessible 24/7 via in person or telemedicine and written call schedule for attending physicians available 24/7
- Ability to provide mechanical thrombectomy
- Ability to administer intra-arterial thrombolytics
- Education for nurses and other ED staff 2 hours annually, stroke nurses and core stroke teams 8 hours annually
• Use of eight PSC stroke measures as well as give ischemic hemorrhagic CSTK (comprehensive stroke) measures for a total of 13 in evaluation of clinical performance (TJC, 2018)

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 together with these additional requirements:

• Program medical director with extensive expertise available 24/7
• Dedicated neuro-intensive care unit beds for complex stroke patients and neuro-intensivist coverage 24/7
• Advanced imaging capabilities
• Neurologist who meets emergent needs of multiple complex stroke patients and written call schedule for attending physicians providing availability 24/7
• Neurosurgical service available 24/7
• Capabilities for microsurgical neurovascular clipping of aneurysm, neuroendovascular coiling of aneurysm, stenting of extracranial carotid arteries, carotid endarterectomy, and 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
• Use of eight core stroke measures and ten comprehensive stroke measures for a total of 18 to evaluate clinical performance (TJC, 2018)

MOBILE STROKE UNIT (MSU)

Mobile stroke units have been slowly spreading across the United States, with an estimated 20 units in 2019.

An MSU is an ambulance equipped with all the traditional medications, tools, and resources used to treat stroke patients in a hospital setting. It may include an EMS driver, a critical care paramedic, a critical care nurse, and a CT technologist. It is also fitted with an onboard CT scanner, since a CT scan is the fastest way to determine what type of stroke the patient is experiencing and what treatment is required.
As the CT scan and other onboard tests are being performed, results are sent to a stroke neurologist and a radiologist at the hospital via a wireless VPN router. The physician can be live-streamed into the back of the ambulance, where an assessment of the patient can be done. In some cities, a single stroke code team is mobile and travels between multiple hospitals that by themselves may not have such resources. These stroke code teams avoid time delays and costs incurred through transfer of a patient to a single site (Nyberg et al., 2018; Jacobson, 2017).

### STROKE PROTOCOLS

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** to accomplish the goal of rapid treatment includes:

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, 2017)

**Triage**

**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.

About one half of all stroke patients will not enter the emergency room by ambulance. The ED registration staff must be trained to recognize signs of possible stroke. 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.

**IDENTIFICATION OF A POTENTIAL STROKE VICTIM**

The **Emergency Severity Index (ESI)** is a five-level emergency department triage algorithm that provides clinically relevant stratification of patients into five groups from 1 (most urgent) to 5 (least urgent) on the basis of acuity and resource needs, including labs, ECG, X-rays, CT, MRI,
ultrasound-angiography, IV hydration, IV or IM medications, specialty consultation, and simple and complex procedures (AHRQ, 2018).

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 their 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.

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.

3. Levels 3, 4, and 5 are least urgent and would not apply to a stroke patient. They are dependent upon the number of resources required to provide appropriate care. (AHRQ, 2018)
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 or less
- From the door to admission to a monitored bed: 3 hours

(Jauch, 2017)

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 LKW
   - 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 60 minutes of the patient’s arrival)
     - 3-hour time window after onset of symptoms
     - 4.5-hour time window in well screened patients who are at low risk for bleeding
     - 4.5- to 6-hour time window, evaluate candidate for mechanical thrombectomy
   - Door to admission time of 3 hours after arrival for all patients

   (ACLS, 2019; Filho & Samuels, 2018)

The time sheet then follows the patient to keep providers, nurses, and technicians on schedule.
ROLE OF THE EMERGENCY DEPARTMENT RN

The emergency department nurse who initially evaluates the patient:

1. Notifies CT to anticipate an emergent CT scan
2. Obtains a serum glucose, unless EMS glucose value is already known
3. Anticipates orders for:
   - CT without contrast or MRI to rule out ICH or nonstroke lesions
   - CT with contrast head/neck
   - Laboratory tests for cardiac-specific troponin and chemistry panel
   - Coagulation studies
   - Arterial blood gas analysis to assess for hypoxemia
   - 12-lead EKG to rule out myocardial infarction (MI) and atrial fibrillation
   - Electroencephalogram (EEG) to rule out ongoing seizures
   - Chest X-ray if clinically indicated
(LNC, 2019a; OHSU Healthcare, 2017)

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 (see below). 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 (ACLS, 2019).

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, by mouth twice daily; lisinopril (Zestril), 5 mg, by mouth daily; and hydrochlorothiazide (Esidrix), 25 mg, by mouth 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 AND TREAT MEDICAL PROBLEMS**

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 <94%, give O₂ via nasal cannula at 2 to 3 L/min. Supplemental oxygen is not recommended in nonhypoxic patients with acute ischemic stroke.
- Hypoperfusion and hypovolemia should be corrected.
- Patients with elevated blood pressure who are eligible for fibrinolytic therapy should have BP carefully lowered to <185 mmHg systolic and <110 mmHg diastolic before IV fibrinolytic therapy is begun.
- Sources of elevated temperature >100.4 °F should be identified and treated. Antipyretic medications should be administered if indicated.
- Treat hyperglycemia to achieve blood glucose levels in the range of 140 to 180 mg/dL.
- Treat hypoglycemia (<60 mg/dL) in all patients with acute ischemic stroke.
- 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.
If the patient is alcoholic or malnourished, thiamine should be given.  
(ACLS, 2019; Powers et al., 2018)

**Stroke Diagnostic Studies**

A **blood glucose** laboratory test must precede initiation of fibrinolytic therapy. **Coagulation studies** (international normalized ratio [INR], activated partial thromboplastin time [aPTT], and platelet count) may also be required if there is suspicion of coagulopathy. Because of the very low risk of unsuspected abnormal findings, fibrinolytic treatment should not be delayed while waiting for testing if there is no reason to suspect abnormal results (Powers et al., 2018).

Additional laboratory tests are tailored to the individual patient and may include the following:

- Cardiac biomarkers
- Toxicology screen
- Fasting lipid profile
- Erythrocyte sedimentation rate (ESR)
- Pregnancy test
- Antinuclear antibody (ANA)
- Rheumatoid factor
- Homocysteine level
- Rapid plasma reagent (RPR)

A urine pregnancy test should be obtained for all women of childbearing age with stroke symptoms. The safety of the fibrinolytic agent recombinant tissue-type plasminogen activator (rtPA) in pregnancy has not been studied in humans (Jauch, 2019).

**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
• Coagulopathies
• Illicit drug use (especially cocaine)
• Migraines
• Oral contraceptive use
(Jauch, 2019)

Patient Examination

Patient examination includes a focused physical examination, a neurological examination, and a formal stroke assessment.

FOCUSED PHYSICAL EXAMINATION (WITH ECG)

The purpose of the focused general physical is to:

• Detect extracranial causes of stroke symptoms
• Distinguish stroke from stroke mimics
• Determine and document for future comparison degree of neurological deficit using the NIH Stroke Scale to determine severity and possible location of the stroke
• Localize the lesion
• Identify comorbidities
• Identify conditions that can influence treatment decisions, such as recent surgery or trauma, active infection, or active bleeding

The physical examination must include all major organ systems, including a careful head and neck exam for signs of trauma, infection, and meningeal irritation. Vital signs can point to impending clinical deterioration and may assist in narrowing the differential diagnosis.

A search for the cardiovascular causes of stroke requires examination of the following:

• Ocular fundi for retinopathy, embolic, or hemorrhage
• Heart for arrhythmias, such as atrial fibrillation, gallop, or murmur
• Peripheral vascular including palpation of carotid, radial and femoral pulses, and auscultation for carotid bruit
• Unequal pulses or blood pressures in the extremities, which may indicate the presence of an aortic dissection
(Jauch, 2019)
NEUROLOGICAL EXAMINATION

A brief but accurate neurological exam should be done with the goals of:

- Confirming the presence of stroke syndrome
- Distinguishing stroke from stroke mimics
- Establishing a neurological baseline to assess improvement or deterioration of condition
- Establishing stroke severity to assist in prognosis and therapeutic selection

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

The skull and spine should be examined for signs of meningismus (neck stiffness, headache, and other symptoms suggestive of meningeal irritation (Jauch, 2019).

FORMAL STROKE ASSESSMENT

AHA/ASA guidelines 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  
  (Powers et al., 2018)

### 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>Drowsy</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Obtunded (dulled or less sharp)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Comatose-unresponsive</td>
<td>3</td>
</tr>
<tr>
<td><strong>Orientation questions:</strong> What month is it?</td>
<td>Answers both correctly</td>
<td>0</td>
</tr>
<tr>
<td>What is your age?</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>Response to commands:</strong> Open and close eyes. Grip and release nonparetic hand.</td>
<td>Performs both tasks correctly</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Performs one task correctly</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Performs neither</td>
<td>2</td>
</tr>
<tr>
<td><strong>Best gaze:</strong> Follow my finger with your eyes.</td>
<td>Normal horizontal movements</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Partial gaze palsy</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Complete gaze palsy</td>
<td>2</td>
</tr>
<tr>
<td><strong>Visual fields</strong></td>
<td>No visual field deficit</td>
<td>0</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>
<tr>
<td><strong>Facial palsy:</strong> Show teeth. Raise eyebrows. Squeeze eyes shut.</td>
<td>Normal, symmetrical</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Minor facial weakness</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Partial facial weakness</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Complete unilateral palsy</td>
<td>3</td>
</tr>
<tr>
<td><strong>Motor arms:</strong> Extend arms (each arm tested and scored separately).</td>
<td>No drift</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Drift before 10 seconds</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Falls before 10 seconds</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>
**Motor legs:** In supine position, raise leg 30 degrees (each leg tested and scored separately).

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No drift</td>
<td>0</td>
</tr>
<tr>
<td>Drift before 5 seconds</td>
<td>1</td>
</tr>
<tr>
<td>Falls before 5 seconds</td>
<td>2</td>
</tr>
<tr>
<td>Falls, no effort against gravity</td>
<td>3</td>
</tr>
<tr>
<td>No movement</td>
<td>4</td>
</tr>
</tbody>
</table>

**Cerebellar testing:** Limb ataxia (finger-nose-finger and heel-shin tests on both sides).

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>0</td>
</tr>
<tr>
<td>Ataxia in one limb</td>
<td>1</td>
</tr>
<tr>
<td>Ataxia in two limbs</td>
<td>2</td>
</tr>
</tbody>
</table>

**Sensory:** Pinprick to face, arm, leg.

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No sensory loss</td>
<td>0</td>
</tr>
<tr>
<td>Mild sensory loss</td>
<td>1</td>
</tr>
<tr>
<td>Severe sensory loss</td>
<td>2</td>
</tr>
</tbody>
</table>

**Sensory:** Extinction. Double simultaneous test.

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No neglect</td>
<td>0</td>
</tr>
<tr>
<td>Partial neglect (1 sensory modality lost)</td>
<td>1</td>
</tr>
<tr>
<td>Complete neglect (2 modalities lost)</td>
<td>2</td>
</tr>
</tbody>
</table>

**Best language:** Ask the patient to name items and describe pictures.

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aphasia</td>
<td>0</td>
</tr>
<tr>
<td>Mild to moderate aphasia</td>
<td>1</td>
</tr>
<tr>
<td>Severe aphasia</td>
<td>2</td>
</tr>
<tr>
<td>Mute, global aphasia</td>
<td>3</td>
</tr>
</tbody>
</table>

**Dysarthria:** Assess speech clarity to “mama, baseball, huckleberry, tip-top, fifty-fifty.”

<table>
<thead>
<tr>
<th>Test</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal articulation</td>
<td>0</td>
</tr>
<tr>
<td>Mild to moderate dysarthria</td>
<td>1</td>
</tr>
<tr>
<td>Severe dysarthria with near to unintelligible or worse</td>
<td>2</td>
</tr>
</tbody>
</table>

**Total score:** ____

**Score interpretation**

<table>
<thead>
<tr>
<th>Score Interpretation</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No stroke</td>
<td>0</td>
</tr>
<tr>
<td>Minor stroke</td>
<td>1–4</td>
</tr>
<tr>
<td>Moderate stroke</td>
<td>5–15</td>
</tr>
<tr>
<td>Moderate/severe stroke</td>
<td>15–20</td>
</tr>
<tr>
<td>Severe (major) stroke</td>
<td>21–42</td>
</tr>
</tbody>
</table>

(Powers et al., 2018)

For hemorrhagic strokes, another neurological assessment tool, the **Glasgow Coma Scale (GCS)**, 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 GCS, 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.)

### GLASGOW COMA SCALE (GCS)

<table>
<thead>
<tr>
<th>Neurological Aspect</th>
<th>Scale</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td>Spontaneously</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>To sound</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No eye opening</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor response</strong></td>
<td>Obey command</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion withdrawal</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion (decorticate)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Abnormal extension (decerebrate)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No motor response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal response</strong></td>
<td>Oriented and converses</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Disoriented and confused</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No verbal response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total score:</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Score interpretation</th>
<th>Minor brain injury</th>
<th>13–15</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Moderate brain injury</td>
<td>9–12</td>
</tr>
<tr>
<td></td>
<td>Severe brain injury (coma)</td>
<td>3–8</td>
</tr>
</tbody>
</table>

(Jain et al., 2019)

**Hypothesis and Diagnosis 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.
FORMING THE HYPOTHESIS

Hypothesis generation begins as soon as the first information about the patient becomes available, and as information is gathered, the clinician proceeds systematically from the more general to the more specific.

After a history is obtained, the clinician plans the physical examination to look for additional findings to help confirm or refute the preliminary diagnoses. Overall, the process of diagnosis should be logical, systematic, and sequential.

When physical examination is being done, it is important to consider the differential diagnoses that can mimic stroke. It has been reported that 19% of patients diagnosed with acute ischemic stroke by neurologists before cranial imaging actually had noncerebrovascular causes for their symptoms.

COMMON STROKE MIMICS

The most frequent stroke mimics include:

- Migraine
- Epilepsy and seizures
- Postictal state
- Sepsis
- Drug overdose
- Mass lesions (abscesses, primary and metastatic tumors, arteriovenous malformations, subdural hematomas)
- Toxic-metabolic disorders (e.g., hyponatremia, hyperglycemia, hypoglycemia)
- Vestibular neuritis
- Psychiatric disorder (e.g., conversion disorder) (Jauch, 2019)

In neurology, two diagnostic questions always require an answer: 1) **What** is the disease mechanism? and 2) **Where** is the lesion? In determining disease mechanism, the clinician considers historical data:

- Past and present patient and family illnesses
- Presence nature of past strokes or TIA
- Activity at the onset of stroke
- Temporal course and progression of the findings
- Fluctuations between normal and abnormal
Accompanying symptoms such as headache, vomiting, seizures, and decreased level of consciousness

The general physical examination uncovers disorders not known from the history and adds to the information used for diagnosing the stroke mechanism.

Following review of the basic elements on which diagnosis is based and the preliminary diagnostic impression from the clinical encounter, imaging and laboratory testing should be planned to test, confirm, and elaborate on the hypothesis of stroke mechanism and anatomical location. When deciding on which tests to conduct, the following questions are asked:

1. Is the brain lesion caused by ischemia or hemorrhage, or is it related to a nonvascular stroke mimic?
2. Where is the brain lesion? What is its size, shape, and extent?
3. What is the nature, site, and severity of the vascular lesion, and how do brain perfusion abnormalities relate to the brain lesion?
4. Are abnormalities of blood constituents causing or contributing to brain ischemia or hemorrhage?
5. Is the patient having seizures?
6. Are there genetic abnormalities that may clarify etiology and potentially guide treatment? (Caplan, 2016)

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

Imaging is done to exclude hemorrhage, assess degree of brain injury, and identify the lesion responsible for the ischemic deficit.

Noncontrast computed tomography (NCCT) of the head remains the mainstay in the setting of acute stroke. It is the most rapid and cost-effective strategy available. The goals of CT in the acute setting are to exclude intracranial hemorrhage, which would preclude thrombolysis.

Multimodal CT techniques, including CT perfusion imaging and CT angiography, make CT 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 fourfold 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.

CT angiography may identify thrombus within an intracranial vessel and may guide intra-arterial thrombolysis or clot retrieval. It may also establish stroke etiology and evaluate carotid and vertebral arteries in the neck.

Multiphase or delayed CT angiography is showing benefit, either replacing CT perfusion or as an additional fourth step in the stroke CT protocol, as it guides patient selection for endovascular therapy by assessing collateral blood flow in the ischemic and infarct tissue.

MRI with magnetic resonance angiography (MRA) has been a major advance in the neuroimaging of stroke. MRI not only provides great structural detail but also can demonstrate early cerebral edema. In addition, MRI has proved to be sensitive for detection of acute intracranial hemorrhage. However, MRI is not as available as CT scanning is in emergencies, many patients have contraindications to MRI imaging (e.g., pacemakers, implants), and interpretation of MRI scans may be more difficult.

At this time, diffusion weighted imaging (DWI) in combination with perfusion MRI outlines salvageable areas of ischemia, providing a useful guide for stroke management. 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.

Transcranial doppler ultrasound (TCD) has been utilized for the diagnosis of intracranial vessel occlusion, as well as the differentiation between ischemic and hemorrhagic stroke, in the context of a negative CT and a clinically suspicious patient presentation (Jauch, 2019; Carroll & Gaillard, 2019).
Image of a transverse (axial) noncontrast CT scan of a patient with a hemorrhagic stroke. Blood is seen as light areas along the left cerebral ventricle (arrows). (The front of the head is at the top of the image.) (Source: Internet Stroke Center.)

LUMBAR PUNCTURE (LP)

A lumbar puncture is done to rule out meningitis or subarachnoid hemorrhage when the CT scan is negative but the clinical suspicion remains high. Persons with suspected subarachnoid hemorrhage and a normal CT scan should undergo a lumbar puncture to detect bilirubin. Red blood cells can be found in both subarachnoid hemorrhage and a traumatic spinal tap. Distinguishing between them means being aware that only red blood cells within the human body break down into bilirubin. Red blood cells in cerebrospinal fluid collected from a traumatic tap will break down into oxyhemoglobin, but not into bilirubin.

Because the breakdown of red blood cells can take up to 12 hours, guidelines recommend that the lumbar puncture should wait until 12 hours after the initial onset of symptoms. Bilirubin will turn fluid yellow (xanthochromia), but visual inspection alone is not considered sufficiently reliable. Therefore, all specimens should undergo spectrophotometry analysis to detect bilirubin, which can be detected as long as two weeks after the initial onset of symptoms (Becske, 2018).
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. It is done if the patient has a TIA or a medical condition that increases the risk of stroke (Mayo Clinic, 2019).

**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.

**Echocardiogram** is most commonly used to evaluate cardioembolic stroke, but transesophageal (TEE) provides images of the internal structures of the heart and is a superior method of identification of most cardiac sources of emboli (ISC, 2019b).

EARLY TREATMENT AND ACUTE STROKE MANAGEMENT

Early treatment with drug administration, if appropriate, is the next link in the chain of survival. If the patient is not a candidate for drug therapy, endovascular therapy may be the treatment of choice.

**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.

**FIBRINOLYSIS WITH rtPA (ALTEPLASE)**

It is recommended to treat carefully selected patients with ischemic stroke with the fibrinolytic drug rtPA (alteplase). The FDA has approved its use in patients who meet specific criteria. Initially, the drug was to be given within 3 hours of the onset of clearly defined stroke symptoms, and only after CT scanning had ruled out hemorrhagic stroke. Subsequently, the AHA/ASA revised the guidelines and expanded the window of treatment from **3 hours to 4.5 hours** after the onset of symptoms.

Studies have found that administration of alteplase within 4.5 hours of stroke onset significantly improves outcomes, irrespective of age or stroke severity, with earlier treatment providing the greatest benefit. The odds of a good stroke outcome were 75% higher for patients who received alteplase within 3 hours of symptom onset compared with those who did not. Patients given
alteplase 3 to 4.5 hours after symptom onset had a 26% increased chance of a good outcome, and patients with a delay of more than 4.5 hours had a nonsignificant 15% increase in the chance of a good recovery (Jauch, 2019).

**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)

---

**WHAT IS rtPA?**

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

The generic name for rtPA is *alteplase* and 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**

The following table indicates inclusion criteria guidelines for administration of rtPA in patients whose onset of symptoms is known to be under 3 hours.

<table>
<thead>
<tr>
<th>INCLUSION CRITERIA GUIDELINES FOR rtPA ADMINISTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>By stroke status:</strong></td>
</tr>
<tr>
<td>• Patient has a diagnosis of ischemic stroke causing measurable neurological deficits.</td>
</tr>
<tr>
<td>• Neurological signs are not clearing spontaneously.</td>
</tr>
<tr>
<td>• Neurological signs are not minor and isolated.</td>
</tr>
<tr>
<td>• Symptoms of stroke are not suggestive of subarachnoid hemorrhage.</td>
</tr>
<tr>
<td>• There has not been a seizure with postictal residual neurological impairments.</td>
</tr>
<tr>
<td>• CT does not show a multilobar infarction (hypodensity &gt;1/3 cerebral hemisphere).</td>
</tr>
<tr>
<td>• Blood glucose concentration is &gt;50 mg/dL (2.7 mmol/L).</td>
</tr>
</tbody>
</table>
By blood vessel status:
- No head trauma or prior stroke in the previous 3 months
- No myocardial infarction in the previous 3 months
- No gastrointestinal or urinary tract hemorrhage in the previous 21 days
- No major surgery in the previous 14 days
- No arterial puncture at a noncompressible site in the previous 7 days
- No history of previous intracranial hemorrhage
- Systolic blood pressure <185 mmHg and diastolic pressure <110 mmHg
- No evidence of active bleeding or acute trauma (e.g., a fracture) on examination

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

Other criteria:
- Blood glucose >50 mg/dL

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

(Saver, 2018)

Eligibility criteria for rtPA at 3 hours to 4.5 hours from onset are similar but more stringent, with any one of the following being an additional exclusion criterion.

- Over 80 years of age
- Use of oral anticoagulants, regardless of the INR
- Baseline score on the National Institutes of Health Stroke Scale (NIHSS) greater than 25
- History of diabetes
- History of stroke
(Jauch, 2019)
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.

INFORMED CONSENT OR PRESUMED CONSENT?

The 2018 AHA/ASA guidelines state: “The benefit of IV alteplase is well established for adult patients with disabling stroke symptoms regardless of age and stroke severity. Because of this proven benefit and the need to expedite treatment, when a patient cannot provide consent (e.g., aphasia, confusion) and a legally authorized representative is not immediately available to provide proxy consent, it is justified to proceed with IV thrombolysis in an otherwise eligible adult patient with a disabling AIS” (Powers et al., 2018).

The dosage is calculated based on the patient’s weight, with a maximum total of 90 mg over 60 minutes. Ten percent of the dose is given as an IV bolus over 1 minute, followed by an IV infusion of the remainder of the dose over 1 hour. It may be administered intravenously or intra-arterially (an off-label route).

Prior to administration, a registered nurse 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 are 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 (indwelling urinary catheters, nasogastric tubes) are completed
- At least two large-bore IVs are in place

Initiating the infusion: The nurse makes certain that the rtPA is infused with no other drugs.

During administration, the nurse:

- Maintains the patient on strict bedrest during treatment
Completes a neurologic assessment and take vital signs every 15 minutes during the 60-minute infusion

Monitors the patient and notifies the physician for adverse allergic reactions

Discontinues infusion and obtains an emergency CT scan and appropriate laboratory work if the following signs of intracranial bleeding occur:
  - Acute hypertension
  - Severe headache
  - Nausea and/or vomiting
  - Worsening neurological exam

Post administration:

- Complete rtPA infusion within one hour.
- Check vital signs and neurologic status:
  - Every 15 minutes for 2 hours, then
  - Every 30 minutes for 6 hours, then
  - Every 60 minutes until 24 hours after rtPA treatment
- Withhold antiplatelet or anticoagulant therapy and invasive procedures for 24 hours following treatment.
- Monitor for serious adverse events, such as bleeding and angioedema.
  - Concomitant use of angiotensin-converting enzyme (ACE) inhibitors may increase the risk of orolingual angioedema.
  - Concomitant use of anticoagulants and drugs that inhibit platelet function increase the risk of bleeding.
- Delay insertion of nasogastric tubes, indwelling catheters, or intra-arterial pressure catheters if patient can be managed without them.
- Obtain follow-up CT or MRI scan 24 hours after treatment before starting anticoagulants or antiplatelet agents.

ADVERSE REACTIONS TO rtPA TREATMENT

- Bleeding (most common)
- Orolingual angioedema
- Arrhythmias
- Hypotension
- Edema
• Cholesterol embolization
• Venous thrombosis
• Re-embolization of deep venous thrombi (DVT) in patients with pulmonary embolism
• Nausea
• Vomiting
• Hypersensitivity reactions
  (LNC, 2019a)

Management of bleeding within 24 hours after administration of alteplase:

• Stop alteplase infusion.
• Obtain CBC, PT (INR), aPTT, fibrinogen level, and type and cross-match.
• Obtain emergent nonenhanced head CT.
• Per order, administer cryoprecipitate (includes factor VIII): 10 U infused over 10–30 minutes (onset in 1 hour, peaks in 12 hour); administer additional dose for fibrinogen level <200 mg/dL.
• Per order, administer tranexamic acid 1,000 mg IV infused over 10 min OR ε-aminocaproic acid 4–5 g over 1 hour, followed by 1 g IV until bleeding is controlled.
• Obtain hematology and neurosurgery consult.
• Manage BP, intracranial pressure (ICP), cerebral perfusion pressure (CPP), mean arterial pressure (MAP), temperature, and glucose.

Management of orolingual angioedema:

• Maintain airway.
  ○ Intubation may not be needed if edema is limited to anterior tongue and lips.
  ○ Edema involving larynx, palate, floor of mouth, oropharynx with rapid progression (within 30 minutes) poses higher risk of respiratory compromise requiring intubation.
  ○ Awake fiberoptic intubation is preferred.
• As ordered, perform the following:
  ○ Discontinue IV alteplase infusion and hold ACE-inhibitors.
  ○ Administer IV methylprednisolone 125 mg.
  ○ Administer IV diphenhydramine 50 mg.
  ○ Administer ranitidine 50 mg IV or famotidine 20 mg IV.
○ If there is an increase in angioedema, administer epinephrine (0.1%) 0.3 mL subcutaneously or by nebulizer 0.5 mL.

○ Administer icatibant (selective bradykinin B2 receptor antagonist), 3 mL (30 mg) subcutaneously in abdomen.

(Powers et al., 2018)

**Complications**

Symptomatic intracranial hemorrhage (ICH) after IV rtPA for ischemic stroke occurs in 2% to 7% of patients. Approximately one half of symptomatic intracranial hemorrhages occur by 10 hours after treatment, with the rest occurring by 36 hours. Intracranial hemorrhage occurring after 36 hours is not likely due to rtPA.

Certain patients are more susceptible to ICH following rtPA administration. These may include patients with:

- Larger strokes
- Older age
- Higher baseline glucose
- Greater stroke severity
- Hypertension
- Congestive heart failure
- Renal impairment
- Diabetes mellitus
- Ischemic heart disease
- Atrial fibrillation
- Baseline antiplatelet use
- Microhemorrhages on MRI
- Leukoaraiosis (damage in the white matter regions of the brain)

If symptomatic intracranial hemorrhage occurs, reversal of the alteplase-induced coagulopathy may be necessary. Cryoprecipitate is probably the first choice, but clear superiority has not been shown for any of the multiple available agents. In some instances, if clinically necessary, neurosurgical intervention may also be considered (Wilner, 2018).

**Other Fibrinolysis Administration Options**

Normally, rtPA is given intravenously, but intra-arterial fibrinolysis has been found to offer moderate net benefits when applied to all patients with potentially disabling deficits
and large artery cerebral thrombotic occlusions. Compared with intravenous therapy, intra-arterial therapy offers several advantages, including a higher concentration of lytic agent delivered to the clot target, a lower systemic exposure to drug, and higher recanalization rates.

Disadvantages include additional time required to initiate therapy, availability only at specialized centers, and mechanical manipulation within potentially injured vessels. Because of these disadvantages, intra-arterial fibrinolytic therapy is commonly administered as an off-label therapy at tertiary centers within 6 hours of onset in the anterior circulation and up to 12–24 hours after onset in the posterior circulation.

There are additional trials ongoing that combine both IV and IA thrombolysis, bringing together the speed of initiating IV and the higher rates of recannulization of the intra-arterial route (Saver, 2018).

Trials on the use of transcranial ultrasound enhancement of fibrinolysis are currently underway. By delivering mechanical pressure to the thrombus, ultrasound can theoretically expose more of the thrombus’s surface to the circulating fibrinolytic agent. Further research is needed to determine the exact role it plays (Jauch, 2019).

TREATMENT WITH OTHER ANTITHROMBOTIC DRUGS

Results of trials using other intravenous fibrinolytic agents in selected patients are consistent with rtPA results but as yet have not identified another proven agent. Three trials of streptokinase found no net benefit of high dose, late IV lytic therapy. A pilot trial of tenecteplase suggested a potential safety and benefit ratio greater than or equal to that of rtPA, but the study concluded that tenecteplase was not superior to alteplase (Saver, 2018).

Aspirin administration is recommended in patients with acute ischemic stroke within 24 to 48 hours after onset. For those treated with IV alteplase, aspirin administration is commonly delayed until 24 hours later. Aspirin, however, is not recommended as a substitute for acute stroke treatment in patients who are otherwise eligible for IV alteplase or mechanical thrombectomy.

In patients presenting with minor stroke, treatment for 21 days with dual antiplatelet therapy (aspirin and clopidogrel) begun within 24 hours can be beneficial for early secondary stroke prevention for a period of up to 90 days from symptom onset.

Urgent anticoagulation—with the goal of preventing early recurrent stroke, halting neurological worsening, or improving outcomes after ischemic stroke—is not recommended for treatment of patients with AIS (Powers et al., 2018).
ENDOVASCULAR TREATMENT

Mechanical Thrombectomy

Mechanical thrombectomy with a stent retriever is a minimally invasive surgical procedure done under general anesthesia or conscious sedation using a small device (called a stent retriever) to remove blood clots in the brain and restore blood flow.

Only second-generation stent retriever devices should be used for mechanical thrombectomy. Catheterization is performed via femoral artery puncture. The catheter is guided to the internal carotid artery and from there to the site of the occlusion. The tiny net-like stent retriever is then inserted into the catheter and guided to the occlusion. The stent is then pushed directly through the clot, after which it expands to the size of the artery wall. At this point, the retriever has captured the clot, and it is removed as the device is pulled back.

Mechanical thrombectomy devices can remove a clot in a matter of minutes, whereas pharmaceutical thrombolytics, even those delivered intra-arterially, may take as long as two hours to dissolve a thrombus.

Following 20 years of studies, endovascular treatment in selected patients with large vessel strokes compared to medical therapy alone has been shown to be of benefit. Intra-arterial treatment with second-generation mechanical thrombectomy devices is safe and effective for reducing disability and is superior to standard treatment with IV thrombolysis alone for ischemic stroke due to large artery occlusion in the proximal anterior circulation.

Prior to the studies, endovascular therapy provided a treatment opportunity for those patients unable to receive IV alteplase. As a result of the latest clinical trials, it is now the standard of care to consider mechanical thrombectomy for those patients with acute ischemic stroke due to a large vessel occlusion in the anterior circulation who can be treated within 24 hours of time LKW (last known well), regardless of whether they receive IV alteplase for the same ischemic stroke event.

For patients who have failed IV rtPA or are outside the window for IV rtPA administration, mechanical thrombectomy is often the only viable treatment option. Recent trial results support use of mechanical thrombectomy within a 24-hour time window for patients who meet eligibility criteria (Powers et al., 2018; Deshmukh, 2019).

In 2018 AHA/ASA updated its guidelines on endovascular treatment using stent retrievers for acute ischemic strokes and strongly recommends its use in patients who meet all the following criteria:

- Age 18 years or older
- Have minimal prestroke disability
• Have prestroke mRS score of 0 to 1
• Have causative occlusion of the internal carotid or proximal middle cerebral artery
• Have a NIHSS >6
• Have a reassuring noncontrast head CT per ASPECTS score
• Can be treated within 6 hours of time LKW (Powers et al., 2018)

**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 (Medscape Education, 2017).

**Emergency CEA/Carotid Angioplasty**

A carotid endarterectomy (CEA) is a surgical procedure done to open and clean a carotid artery. AHA/ASA 2018 guideline recommendations for carotid endarterectomy/carotid angioplasty indicate it is useful when brain imaging suggests a small infarct core with a large penumbra that is compromised by inadequate flow from a critical carotid stenosis or occlusion. It is also useful in cases of neurological deficit following a carotid endarterectomy in which acute thrombosis of the surgical site is suspected but not well established.

In patients with unstable neurological status (e.g., stroke-in-evolution), the efficacy of emergency or urgent CEA is not well established (Powers et al., 2018).

**BLOOD PRESSURE CONTROL IN ISCHEMIC STROKE**

The question of whether or not to treat high blood pressure during treatment of stroke has been debated since 1985 without reaching a definitive answer. There is epidemiological evidence that hypertension is associated with a poor outcome after ischemic stroke and is related to hematoma expansion and functional outcome after intracerebral hemorrhage. On the other hand, pathophysiological concerns include the presence of dysfunctional cerebral autoregulation during stroke and that lowering blood pressure will reduce tissue perfusion, increase lesion size, and ultimately worsen outcome (Bayh et al., 2018).
Current guidelines indicate elevated blood pressure should **not** be treated acutely in patients who are not treated with thrombolytic therapy **unless**:

- Hypertension is extreme (systolic blood pressure >220 mmHg or diastolic blood pressure >120 mmHg)
- The patient has:
  - Active ischemic coronary artery disease
  - Heart failure
  - Aortic dissection
  - Hypertensive encephalopathy
  - Preeclampsia/eclampsia

When treatment is indicated, a cautious lowering of blood pressure by approximately 15% during the first 24 hours after stroke is suggested.

Patients who have elevated BP and are eligible for treatment with fibrinolysis should have their blood pressure carefully lowered so that systolic pressure is <185 mmHg and diastolic <110 mmHg before fibrinolytic therapy is initiated (Powers et al., 2018).

Systemic hypotension and hypovolemia should be corrected so as to improve cerebral blood flow and systemic organ function. However, drug-induced hypotension has not been proven for ischemic stroke (Fihlo & Mullen, 2019).

**SUPPORTIVE CARE**

Supportive care for patients during treatment for ischemic stroke includes:

- Bed rest for the first 24 hours
- Routine placement on aspiration, deep venous thrombosis, fall, and seizure precautions
- Avoidance of prophylactic antibiotics
- Head of bed elevated at a minimum of 30 degrees unless contraindicated
- Airway and ventilation maintenance
- Continuous hemodynamic monitoring and neurologic assessment
- Nothing by mouth (NPO) for the first 24 hours (Powers et al., 2018; LNC, 2019b)
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 at this time.** A promising very early-stage treatment may be evacuation of hematoma, either via open craniotomy or endoscopy, which may improve long-term prognosis.

Currently, hemorrhagic stroke is treated with **medical management** that includes:

- Basic life support measures
- Control of bleeding
- Seizure control
- Blood pressure control
- Intracranial pressure control

(Liebeskind, 2019; Rordorf & McDonald, 2019)

**BASIC LIFE SUPPORT MEASURES**

Management begins with rapid stabilization of vital signs and the simultaneous acquisition of an emergent CT scan. Endotracheal intubation for patients with a decreased level of consciousness and poor airway protection may be necessary. Patients with elevated intracranial pressure are intubated and hyperventilated. Mannitol is administered for further control, and glucose levels are monitored (Powers et al., 2018).

**CONTROL OF BLEEDING**

Anticoagulation-associated intracranial hemorrhage has a high morbidity and mortality rate. Over half of these patients die within 30 days. Patients on warfarin have an increased incidence of hemorrhagic stroke. The necessity of **reversing warfarin anticoagulation is a medical emergency**, and 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)

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.

There is controversy as to whether or not patients who are taking antiplatelet medications, such as aspirin, should be given desmopressin (DDAVP) and/or platelet transfusions. Guidelines recommend platelet transfusions only when such hemorrhaging complicates severe thrombocytopenia (Liebeskind, 2019).

Newer direct oral anticoagulants (DOACs), such as Pradaxa, are increasingly used as alternatives to warfarin therapy. A number of suggestions have been put forth to guide treatment in the setting of hemorrhagic complications; however, there is less clinical experience with their reversal in the setting of ICH (Rordorf & McDonald, 2019).

SEIZURE CONTROL

Up to 28% of patients with intracranial hemorrhage have early nonconvulsive seizure activity. Guidelines recommend that patients with clinical seizures or with EEG seizure activity accompanied by changes in mental status receive antiepileptic medication. For rapid control of seizures, a benzodiazepine (lorazepam, diazepam) are given, followed by phenytoin or fosphenytoin (Cerebyx) loading for longer-term control (Liebeskind, 2019).

BLOOD PRESSURE CONTROL

There are no defined optimum BP levels for patients with acute hemorrhagic stroke, but highly elevated BP is thought to lead to rebleeding and expansion of the hematoma. 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.

For patients who present with SBP >220 mmHg, it is recommended to aggressively reduce blood pressure with a continuous intravenous infusion of antihypertensive medication and frequent (every 5 minutes) blood pressure monitoring. The target blood pressure is uncertain, but a SBP of 140–160 mmHg is considered reasonable.

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 SBP is >200 mmHg or MAP is >150 mmHg, aggressive intravenous medication should be administered, checking blood pressure every 5 minutes (Liebeskind, 2019; Rordorf & McDonald, 2019).

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 degrees 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.

More aggressive therapies include osmotic therapy (mannitol or hypertonic saline), barbiturate anesthesia, and neuromuscular blockage, which require monitoring of ICP and BP to maintain adequate cerebral perfusion pressure of 70 mmHg.

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

For patients with extension of hemorrhage (IVH) or hydrocephalus, placement of an external ventricular drain may be necessary in order to provide CSF diversion. This also adds the capability to monitor ICP (Liebeskind, 2019).

SURGICAL INTERVENTIONS

Surgical interventions, such as early hematoma evacuation, is a potential treatment for hemorrhagic stroke but 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 8 hours of stroke
- Volume of the hematoma is 20–50 ml
- Glasgow Coma Score is 9–12
- Patient age is 50–59 years
  (Liebeskind, 2019)

The role of surgical treatment in ICH remains controversial, and outcomes are conflicting. Patients with hemorrhages greater than 3 cm in diameter or those with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression or hydrocephalus are recommended for surgical treatment (Rordorf & McDonald, 2019).

Decompression surgery relieves pressure on the brain, allowing pooled blood to be removed and repair to damaged blood vessels.

Craniotomy with open surgery is performed when hematoma is very large or when it is compressing the brainstem. It involves removal of a portion of the skull to drain the hematoma and repair blood vessels.

Simple aspiration involves drilling a small hole in the skull and draining the hematoma using a needle (Knopman, 2017).

TREATMENT OF ANEURYSMS

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.

Endovascular coiling is a less-invasive procedure than clipping and has increasingly been used in recent years with excellent success. 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. Endovascular treatment may be preferred over surgical clipping when:

- An aneurysm is in a difficult location to access surgically
- The aneurysm is small-necked and located in the posterior fossa
- The patient is elderly
- The patient has a poor clinical grade
  (Liebeskind, 2019)

**ARTERIOVENOUS MALFORMATION (AVM) TREATMENT**

Treatment options for AVM depend on its type, location, and size. Surgery is the most common treatment for AVM and is most effective with more easily accessible lesions of smaller size. There are three potential surgical options: surgical resection, endovascular embolization, and stereotactic radiosurgery.

**Surgical resection.** If an AVM has bled and/or is in an area that can be easily accessed, then surgery is recommended 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.

**Endovascular embolization.** This procedure involves 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).** SRS is done for an AVM that is not too large but is in an area difficult to reach by regular surgery. This treatment 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 (ASA, 2018b; NINDS, 2019c).
SUBARACHNOID HEMORRHAGE (SAH) TREATMENT

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 and Ischemic Infarction**

Symptomatic vasospasm is associated with a clinical decline and signals a poor prognosis. 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. Increases in mean arterial pressure alone have been shown to be of benefit for patients with vasospasm, and a number of medications are used to achieve this goal. These include phenylephrine, norepinephrine, and dopamine.

More invasive means of treatment of vasospasm depend on the utilization of cerebral angiography and include intra-arterial vasodilator administration and balloon angioplasty.

Nimodipine is currently recommended as first-line medical treatment for preventing post-SAH cerebral vasospasm (Ashley, 2018).

**Hydrocephalus**

Hydrocephalus can be an acute or delayed complication in 20% of SAH cases. Acute obstructive hydrocephalus usually occurs within the first 24 hours and can precipitate life-threatening brainstem compression and blood vessel occlusion.

Treatment for acute hydrocephalus includes ventricular drainage (ventriculostomy), depending on the severity of clinical neurologic dysfunction or CT scan findings. Rapid lowering of intracranial pressure during intraventricular catheter placement is associated with a higher risk of rebleeding and should be avoided.

Ventriculostomy, when done correctly, is a relatively low-risk procedure that can result in dramatic and immediate clinical improvement in about two thirds of patients (Becske, 2018).

**SUPPORTIVE CARE**

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 30 degrees; 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
• Maintaining NPO status until swallowing function is evaluated
• Avoiding hypoglycemia
• Correcting hyperglycemia to 140–180 mg/dL
• Correcting metabolic acidosis
• Continuous ICP using direct measurement
• Hypertension treatment with IV medication
  (Rordorf & McDonald, 2019)

PREVENTING AND MANAGING COMPLICATIONS

After the patient’s stroke has stabilized for 12 to 24 hours, collaborative care shifts from preserving life to preventing complications, reducing disability, and attaining optimal functioning. This most often requires intensive care in a unit staffed by ICU nurses who are trained to recognize and manage intracranial complications.

Postprocedural Care

For patients who have had surgery, complications may develop within 24 to 48 hours of catheter sheath removal. The site should be monitored for:

• Bruising
• Groin hematoma/excessive bleeding
• Signs of infection, such as localized edema and erythema
• Retroperitoneal bleed
• Pseudo-aneurysm or arteriovenous fistula
• Arterial occlusion
• Peripheral pulses for the first 24 to 48 hours  
  (Hill et al., 2018)

Patients who have received CT contrast during a procedure should be:

• Given fluids IV to flush contrast from kidneys
• Monitored for fluid overload, especially in patients with cardiac comorbidities
• Monitored for cardiac arrhythmias  
  (Hill et al., 2018)

**Deterioration of 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.

During the first 24 hours, acute stroke patients require 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).

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 GCS 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 et al., 2016).

**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:

• Cranial nerve problems, especially asymmetry, loss or reduction of pupillary responses
• Peripheral motor deficits
• Irregular respirations
• Neck pain or stiffness
• Nausea and vomiting
• Loss of consciousness
Brain herniation is a life-threatening emergency, and ventriculostomy or decompression craniectomy may be a necessary treatment option for many patients (LNC, 2019a; Ishida, 2018).

**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 and **Cushing triad** (bradycardia, respiratory depression, and hypertension). While the mechanism of Cushing triad is controversial, many believe it is related to brainstem compression and is an especially ominous sign requiring urgent intervention.

Patients with signs of increased ICP or herniation should be intubated and hyperventilated. Excessive hyperventilation is to be avoided, as it may potentiate vasospasm and ischemia. Other interventions for increased ICP include:

- Osmotic agents (e.g., mannitol), which can decrease ICP dramatically (50% 30 minutes post administration)
- Loop diuretics (e.g., furosemide)
- Although controversial, intravenous steroids (e.g., dexamethasone)

Patients must remain on strict bedrest with head of the bed elevated at 30 degrees to ensure optimal venous drainage. To decrease stimuli, the patient may be placed in a darkened, quiet room and given mild sedation if agitated. Sedatives and analgesics, however, should be used cautiously to avoid masking neurological findings (Becske, 2018).

**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 represents the conversion of an ischemic infarction into an area of hemorrhage. Hemorrhagic transformation of an ischemic infarct occurs within 2–14 days post ictus, is more commonly seen following cardioembolic strokes, and is more likely with larger infarct size. Hemorrhagic transformation is also more likely following administration of fibrinolytic treatment. Hemorrhagic transformation can range from the development of small petechial hemorrhages to the formation of hematomas that produce neurologic decline,
necessitating surgical evacuation or decompressive hemiepicondylectomy (Jauch, 2019; Danziger, 2018).

- **Intracerebral hemorrhages.** Enlargement of blood clot occurs in the first 24 hours after onset in about a third of patients and is a major concern. One major factor in the rebleed is the use of anticoagulant medication. Fifty percent of patients admitted with brain hemorrhage while on anticoagulation deteriorate in the first 24 to 48 hours due to additional bleeding, with a high mortality rate of 64% by 6 months (MCN, 2017).

- **Subarachnoid hemorrhages.** The most dreaded early complication of SAH is rebleeding, the greatest risk being within the first 24 hours of rupture, particularly within six hours of initial hemorrhage. The cumulative risk of rebleeding is 19% at 14 days. Overall mortality rate from rebleeding is reported to be a high as 78%. Measures to prevent rebleeding include bed rest in a quiet, darkened room; analgesia; and sedation. Stool softeners are given to prevent the Valsalva maneuver with resultant peaks in SBP and ICP. The only effective treatment is prevention by obliterating the ruptured aneurysm by clipping surgically or occluding endovascularly with a coil (Becske, 2018).

**Pulmonary Complications**

**PNEUMONIA**

Pneumonia, one of the most common respiratory complications of acute stroke, occurs in approximately 5%–7% of stroke patients. Stroke-related pneumonia is associated with a higher mortality and poorer long-term outcome. Aspiration is the cause of close to 60% of post-stroke pneumonia, usually due to dysphagia.

Measures to prevent aspiration include 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 by a trained healthcare professional. Patients who fail the swallowing screening should be referred to a speech pathologist for a comprehensive assessment (Chalela, 2018; Powers et al., 2018; LNC, 2019a).

**COMPLICATIONS RELATED TO INTUBATION AND MECHANICAL VENTILATION**

Intubation and mechanical ventilation of patients with ischemic stroke is usually done in cases of pulmonary edema or for inability to protect the airway because of a decreased level of consciousness from effects of the stroke or seizure, partial airway obstruction, hypoventilation, or aspiration pneumonia. The morbidity and mortality in patients intubated after acute stroke is very high (71%).

Complications related to endotracheal intubation include laryngeal injury (e.g., laryngeal edema, vocal cord dysfunction), swallowing impairment, tracheal stenosis, tracheoesophageal fistula, cuff leaks, sinusitis, and biofilm formation (Chalela, 2018).
ABNORMAL BREATHING PATTERNS

Abnormal breathing patterns occur in approximately 60% of patients with neurologic disorders, including strokes. The underlying causes are cardiac and respiratory abnormalities. Abnormal breathing patterns are more common among patients with impaired consciousness, severe deficits, during sleep, and in patients with medullary infarcts. Most respiratory patterns do not have prognostic significance or imply involvement of specific areas of the central nervous system. Only sustained tachypnea with low PaCO₂ is associated with poor outcome.

Abnormal respiratory patterns are described below:

- **Cheyne-Stokes** is characterized by recurrent central apnea alternating with a crescendo-decrescendo pattern of tidal volume. It is the most commonly recognized abnormal respiratory pattern following stroke, and in approximately 90% of patients it reflects underlying cardiopulmonary disease.

- **Periodic breathing**, a variant of Cheyne-Stokes respiration, is characterized by regular, recurrent cycles of changing tidal volumes in which the lowest tidal volume is less than half the maximal tidal volume in that cycle. It is the most frequent abnormal respiratory pattern directly related to stroke rather than underlying systemic disease, occurring in approximately 25% of patients. Periodic breathing may be more common among patients with subarachnoid hemorrhage.

- **Gasping** is an abnormal breathing pattern characterized by an attenuated inspiratory period followed by a disproportionately long period of expiration. It is most commonly seen in medullary strokes, and respiratory failure almost invariably ensues.

- **Complete apnea** following stroke is extremely rare in the absence of brain death, and the prognosis for recovery is generally poor.

Close observation of the stroke patient for these potential disturbances and implementation of prophylactic measures can prevent significant morbidity and mortality (Chalela, 2018).

OXYGEN DESATURATION

In patients with acute ischemic and hemorrhagic stroke, oxygen desaturation detected by continuous pulse oximetry is associated with increased age, higher NIHSS score, and presence of dysphagia. Patients with a history of cardiac and pulmonary diseases are also at higher risk for desaturation. In studies, most patients were found to be asymptomatic during periods of desaturation, suggesting that silent desaturation may be more common than previously recognized.

Patients with very low oxygen levels must be treated with oxygen, and so it is important to monitor oxygen levels to maintain oxygen saturation >94%. However, there is clear and unambiguous evidence that stroke patients do not need routine oxygen and that it does not improve recovery from stroke (Chalela, 2018; Roffe et al., 2017).
NEUROGENIC PULMONARY EDEMA (NPE)

Neurogenic pulmonary edema is a rare form of pulmonary edema that most often develops abruptly and progresses quickly after a neurological insult. NPE is an increase in interstitial and alveolar fluid that can occur following stroke, particularly 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 (Chalela, 2018).

Cardiovascular Complications

CARDIAC PROBLEMS

Cardiac complications include arrhythmias, myocardial infarction, and neurogenic cardiac injury. Angina, myocardial infarction, and cardiac ischemia complicate 6% of acute strokes and are serious or life-threatening medical events in 1% of stroke patients. Cardiac arrhythmia in the first 72 hours following an admission for acute stroke occurs in approximately 25% of patients, with atrial fibrillation being the most common. Cardiac events and cardiac death after acute stroke may be caused by acute myocardial infarction, heart failure, ventricular arrhythmias such as ventricular tachycardia or fibrillation, and cardiac arrest.

Initial ECG monitoring should be undertaken for all patients with stroke. The duration and mode of monitoring is generally recommended for at least the first 24 hours, but for patients with embolic stroke of uncertain source, longer-term ECG monitoring (external or implantable) should be used.

ECG changes are likely mediated by the autonomic nervous system, and concurrent stroke and myocardial infarction are not uncommon. 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.

It is helpful to note that elevated cardiac enzymes following acute ischemic stroke may not be an indication of myocardial injury. However, elevated troponin T after stroke may be a poor prognostic sign, and troponin levels may be useful when attempting to differentiate between neurogenic left ventricular dysfunction associated with subarachnoid hemorrhage and that of dysfunction associated with myocardial infarction (Ishida, 2018; Chalela & Jacobs, 2019).

Options for treatment of acute MI are limited in the setting of acute ischemic stroke. MI is a contraindication to thrombolysis, and thrombolysis is contraindicated in MI (Chalela & Jacobs, 2019).

HYPERTENSION

Hypertension is common in acute stroke and is associated with poor outcomes. Studies of antihypertensive treatment in this setting, however, have produced conflicting results. One
drawback of blood pressure reduction theoretically is that elevated blood pressure may counteract dysfunctional cerebral autoregulation as a result of stroke, but evidence suggests that antihypertensive treatment in acute stroke does not change cerebral perfusion.

All acute stroke patients should have their blood pressure closely monitored in the first 48 hours after stroke onset. Patients with acute ischemic stroke with blood pressure >220/120 mmHg should have their blood pressure cautiously reduced. For these patients, a reasonable goal is to lower blood pressure by 15% during the first 24 hours after the onset of stroke. Care must be taken to avoid lowering blood pressure too quickly or aggressively, since this could worsen perfusion in the penumbra.

During the acute phase of stroke, there is no evidence supporting the use of any particular antihypertensive agent to achieve the recommended blood pressure goals. Reversible and titratable intravenous agents are considered the best option for obtaining precise blood pressure lowering. Guidelines suggest the use of intravenous labetalol, nicardipine, and clevidipine as first-line antihypertensive agents that allow for rapid and safe titration to reach blood pressure goals. Medications that can cause a prolonged or rapid decline in blood pressure (such as nifedipine) should be avoided (Filho & Mullen, 2019).

It is recommended to start or restart antihypertensive therapy (orally or via nasogastric tube) during hospitalization in neurologically stable patients with BP >140/90 mmHg to improve long-term BP control unless contraindicated (Jauch, 2019; Powers et al., 2018).

**VENOUS THROMBOEMBOLISM (VTE)**

Venous thromboembolism includes deep vein thrombosis (DVT) and pulmonary embolism (PE). DVT may develop as early as 24–48 hours after stroke onset and has a peak incidence between two to seven days. The risk of death associated with untreated DVT in stroke patients is about 15%. The rate of DVT development is higher in patients of advanced age, those with a high stroke severity, and those with hemiparesis or immobility.

DVT is a serious issue, as it may lead to pulmonary embolism. Pulmonary embolism accounts for 13%–25% of early deaths following stroke and is the most common cause of death two to four weeks after stroke onset. The risk of VTE remains elevated in the first one to three months following stroke, due in part to stroke-related immobility.

Without treatment, pulmonary embolism has a mortality rate of about 30%. Anticoagulation is the recommended treatment, but it carries the risk of hemorrhage transformation in patients with ischemic stroke and risk of hematoma expansion and rebleeding in patients with cerebral hemorrhage. Embolectomy can also be used for patients with embolism who have contraindications to anticoagulation and thrombolysis if necessary.

The best option for prevention of DVT for stroke patients whose mobility is restricted is an intermittent pneumatic compression (IPC) device, preferably thigh-length. IPC should not be started for patients who have already been at bed rest or immobilized without any form of VTE.
prophylaxis for >72 hours after stroke onset because of the theoretical risk of dislodging a venous clot (Ishida, 2018).

**Other Common Complications**

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

**HYPERTHERMIA**

Fever is independently associated with poor outcomes. All stroke patients should have their temperature monitored at least four times a day for 72 hours. Sources of hyperthermia (temperature >100.4 °F) are identified and treated with antipyretic medications. Elevated temperature in the first 24 hours of being admitted to ICU has been associated with an increased risk of in-hospital death.

The benefit of hypothermia is not well established and should only be offered in the context of ongoing clinical trials. Studies suggest that induction of hypothermia is associated with an increased risk of infection, including pneumonia (Powers et al., 2018).

**HYPERGLYCEMIA AND HYPOGLYCEMIA**

Numerous studies have shown that hyperglycemia exacerbates neuronal damage after stroke. However, despite the marked occurrence of neurologic or medical complications and overall clinical deterioration associated with both high or low post-stroke blood glucose, there are still no precise guidelines regarding a gold standard euglycemic management after stroke.

Current guidelines utilize the American Diabetic Association recommendations of maintaining glucose in the range of 140–180 mg/dL, with close monitoring to prevent hypoglycemia (<60 mg/dL) (Powers et al., 2018).

Typically, hyperglycemia is treated with subcutaneous insulin on a sliding scale. Refractory hyperglycemia may require the use of intravenous insulin; however, IV insulin increases the risk of hypoglycemia.

When hypoglycemia is discovered, the glucose level must be brought expeditiously to a normal level. IV fluids, such as dextrose 25% in water (D25W) or dextrose 50% in water (D50W), may be necessary. Treatment of hypoglycemia beyond the initial therapy depends on the condition’s underlying cause.

Neurologists typically do not treat patients with glucose-containing fluids without coadministration of thiamine in order to avoid the possibility of precipitating acute Wernicke encephalopathy or chronic Korsakoff psychosis (Chawla, 2018).
DYSPHAGIA

Post-stroke dysphagia is a very common complication (37%–78%) and is the major risk factor for pneumonia. Patients with acute stroke should have their swallowing screened within four hours of arrival at the hospital and before being given any oral food, fluid, or medication. Personnel specifically trained in swallowing screening using a validated tool should undertake screening to observe how well swallowing muscles move and to help determine which nerves, muscles, and reflexes are impaired.

The water swallowing test (WST) is a bedside screening tool used to assess for aspiration in clinical practice, but it is limited in its accuracy. Different food texture and viscosity might be used according to the levels of dysphagia, which cannot be demonstrated by WST. A more ideal bedside screening tool is the volume-viscosity swallow test (V-VST), which uses boluses of different volumes and viscosities of food. It has an advantage by indicating the appropriate diet for stroke patients in order to minimize the risk of complications.

Patients who fail the swallowing screening are referred to a speech pathologist for a comprehensive assessment, which may include instrumental examination. Video-fluoroscopy (VFS) and fiberoptic endoscopic evaluation of swallowing (FEES) are considered the gold standard for evaluating swallowing function. However, both are invasive and require trained staff.

There is insufficient data as to whether screening protocols decrease death or dependency, but that does not mean screening is ineffective. Those who fail screening are older, have more comorbidities, present with weakness and speech difficulties, have lower levels of consciousness, and have higher stroke severity (Chalela, 2018; Ye et al., 2018).

URINARY TRACT INFECTION

Urinary tract infection is a common problem following stroke and occurs in approximately 11%–15% of patients who are followed for up to three months. It is a serious complication that impairs stroke recovery, is associated with poorer neurological outcomes and longer hospital stays, and can be life-threatening in about 1% of cases. UTI has also found to be a common complication in patients who have been followed for up for 30 months.

Factors that are considered to increase the risk of UTI include:

- Stroke severity
- Stroke-induced immunosuppression
- Depressed level of consciousness
- Bladder dysfunction
- Urinary catheterization
It is a common practice to place an indwelling catheter in patients with stroke due to incontinence, immobility, or convenience. This is an important risk factor for infection, and the duration of catheterization is directly related to the risk. Therefore, an indwelling catheter should be avoided whenever possible. The use of external catheter systems or intermittent catheterization are alternatives associated with a lower risk of urinary tract infections (Ishida, 2018).

GASTROINTESTINAL COMPLICATIONS

Gastrointestinal hemorrhage is one of the more common complications and has both serious and nonserious manifestations in about 2%–3% of stroke patients.

Stress ulcer prophylaxis with proton pump inhibitors or H2 antagonist is effective for reducing overt GI bleeding but may increase the risk of nosocomial pneumonia. Therefore, prophylaxis is not routinely used for patients with acute stroke but is reserved for patients who have one or more of the following risk factors:

- Mechanical ventilation for >48 hours
- Coagulopathy
- History of GI ulceration or bleeding within the past year
- Two or more of the following risk factors:
  - Sepsis
  - Intensive care stay lasting >1 week
  - Occult GI bleeding lasting ≥6 days

Evidence suggests that enteral nutrition alone may reduce the risk of GI bleeding due to ulceration and that stress ulcer prophylaxis may be ineffective or harmful among patients who are receiving enteral nutrition (Ishida, 2018).

NURSING CARE BEYOND 24 HOURS

After the first 24 hours, the nursing staff can begin to focus on other patient management issues that may arise. During this same period, acute rehabilitation is started—usually within 24 to 48 hours—and continues from stroke onset to four days following ischemic stroke and from onset to seven days following hemorrhagic stroke. Following this acute stage, the patient is transferred to a setting that will provide comprehensive long-term stroke rehabilitation.

The highest priorities 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
  (Physiopedia, 2018)

The interdisciplinary team approach is utilized during this stage. There is strong evidence that organized, interprofessional stroke care reduces mortality rates and the likelihood of institutional care and long-term disability, enhances recovery, and increases independence in ADLs (Winstein et al., 2016).

Once the patient is stabilized medically, nursing, physical therapy, occupational therapy, and speech-language therapy professionals assess the following secondary stroke and stroke-related impairments and potential complications in order to provide appropriate interventions:

• Impaired physical mobility
• Acute pain
• Self-care deficit
• Disturbed sensory pattern
• Impaired swallowing (dysphagia) and nutrition
• Alteration in bowel and bladder elimination
• Risk for falls and fractures
• Risk for impaired skin integrity
• Impaired communication (due to aphasia/dysphasia)
• Emotional changes
  (ASA, 2018c; NINDS, 2019d)

(See also “Rehabilitation in the Acute Stroke Setting” later in this course for occupational therapy, physical therapy, and speech-language therapy interventions.)

**Impaired Physical Mobility**

Impaired physical mobility may be related to hemiparesis, loss of balance and coordination, spasticity, or brain injury. Nursing interventions for improving mobility and preventing deformities include:

• Positioning to prevent contractures, including using measures to relieve pressure, assist in maintaining good body alignment, and prevent compressive neuropathies
• Applying a splint at night to prevent flexion of an affected extremity
• Preventing adduction of an affected shoulder using a pillow placed in the axilla
• Elevating an affected arm to prevent edema and fibrosis
• Positioning fingers so that they are barely flexed by placing hand in slight supination; (when upper extremity spasticity is noted, avoiding use of a hand roll and using a dorsal wrist splint)

• Changing position every two hours; placing patient in a prone position for 15 to 30 minutes several times a day to maintain hip extension

• Collaborating with physical and occupational therapy to establish an exercise program and to receive instructions for correctly performing active and passive range of motion exercises

• Providing full range of motion four or five times daily to maintain joint mobility, regain motor control, prevent contractures in the paralyzed extremity, prevent further deterioration of the neuromuscular system, and enhance circulation; if tightness occurs in any area, performing range of motion exercises more frequently

• During exercise, observing for signs of pulmonary embolus or excessive cardiac workload (e.g., shortness of breath, chest pain, cyanosis, and increasing pulse rate)

• Supervising and supporting the patient during exercises; planning frequent short periods of exercise; encouraging patient to exercise unaffected side at intervals during the day (RNpedia, 2019)

**Acute Pain**

Patients may experience pain related to hemiplegia and disuse. Interventions include:

• Using proper patient movements and positioning; placing flaccid arm on a table or pillows when the patient is seated

• Never lifting the patient by the flaccid shoulder or pulling the affected arm or shoulder

• Elevating arm and hand to prevent edema

• Administering analgesic agents as indicated (RNpedia, 2019)

**Self-Care Deficits**

Following a stroke with functional deficits, the patient requires assistance in managing self-care, which includes bathing, hygiene, toileting, dressing, grooming, and feeding. The nursing care plan to enhance self-care may include:

• Collaborating with the interdisciplinary team, including occupational therapy

• Encouraging personal hygiene activities as soon as the patient can sit up; selecting activities that can be done with one hand
• Working with the patient to set realistic goals and add a new task daily
• Encouraging the patient to carry out all self-care activities on the unaffected side
• Ensuring that the patient does not neglect the affected side, providing assistive devices as necessary
• To help improve morale, making sure the patient is fully dressed during ambulatory activities
• Assisting with dressing activities using clothing with Velcro closures; putting the garment on the affected side first
• Keeping the environment uncluttered and organized
• Providing emotional support, encouragement, and positive feedback for accomplishments and efforts
  (RNpedia, 2019)

**Disturbed Sensory Perception**

Sensory perception disturbance includes kinesthetic, tactile, or visual problems related to altered sensory perception, transmission, and/or integration. To help manage sensory perceptual difficulties, nursing care includes:

• Situating items (e.g., food) toward the patient’s unaffected side
• Teaching the patient to turn and look in the direction of the defective visual field to compensate for the loss
• Making eye contact with the patient and drawing attention to the affected side
• Increasing natural or artificial lighting in the room
• Reminding the patient with hemianopsia of the other side of the body and placing extremities so the patient can see them
  (RNpedia, 2019)

**Impaired Swallowing (Dysphagia) and Nutrition**

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

• Starting enteral diet within seven days of admission following acute stroke
• Consulting with speech-language therapist to evaluate gag reflexes and to assist in teaching alternate swallowing techniques:
  o Advising the patient to take small boluses of food
  o Informing the patient of foods that are easier to swallow
Providing thickened liquids or pureed diet as indicated

- Having suction equipment available at the bedside, especially during early feeding attempts

- For patients with dysphagia, using nasogastric tubes for feeding in the early phase of stroke (first 7 days) and percutaneous gastrostomy tube if unable to swallow safely for longer than 2–3 weeks

- Preparing for tube feedings by elevating the head of the bed, checking tube placement before feeding; administering the feeding slowly and ensuring the cuff of tracheostomy tube is inflated (if applicable), monitor and reporting excessive retained or residual feeding

- Having the patient sit upright, preferably in a chair, while eating and drinking, maintaining upright position for 45 to 60 minutes after eating

- Advancing the diet as tolerated

- Considering nutritional supplements for those who are or are at risk for malnourishment

- Implementing oral hygiene protocols to reduce risk of pneumonia (RNpedia, 2019; Powers et al., 2018; LNC, 2019b)

**Alteration in Bowel and Bladder Elimination**

It is common for patients to have problems controlling their bladder and/or bowels following a stroke. Urinary incontinence is more common than fecal incontinence, occurring in nearly 79% of patients on admission. Twenty-eight percent are incontinent on discharge. Up to 40% of patients develop urinary incontinence at 7–10 days following acute stroke related to flaccid bladder, detrusor instability, confusion, or difficulty communicating. The prevalence of incontinence increases with time, and risk factors include hemiparesis, depression, age >75 years, and large infarcts (NSA, 2019e).

Urinary problems may include:

- Urgency
- Frequency
- Nocturnal incontinence
- Functional incontinence
- Reflex incontinence
- Overflow incontinence
Bowel problems can include:

- Fecal incontinence
- Constipation
- Constipation with overflow
- Fecal impaction

The nursing care plan for prevention or improving bowel and bladder elimination includes:

- Collaborating with physical and occupational therapy
- Performing intermittent sterile catheterization during periods of loss of sphincter control
- Analyzing the patient’s voiding pattern and offering urinal, bedpan, or bedside commode on patient’s voiding schedule
- Assisting male patients to an upright posture for voiding
- Initiating a bladder and bowel training program
- Performing a bedside bladder ultrasound after voiding to check for residual early in the program
- Maintaining fluid intake of 2,000–3,000 ml per day unless contraindicated
- Requesting and administering stool softeners or laxatives
- Administering enemas if needed
  (RNpedia, 2019: NSA, 2019e)

**Risk for Falls**

A very common complication of acute stroke is falling. Hip fractures represent 45% of post-stroke fractures and are two to four times more common in stroke patients due to decreased mobility and disuse of paretic limbs. Falls are commonly seen in people who have had a stroke and occur in 7% of people in the first week after their stroke (Verheyden et al., 2018).

Nursing interventions for prevention of falls include:

- Placing the patient on a fall prevention protocol
- Collaborating with physical and occupational therapy to determine ambulation and mobility needs
- Orientating the patient to the environment and assessing their ability to use call light, side rails, and bed controls; considering alternatives for patients unable to use the call light
• Routinely assisting with toileting on the patient’s toileting schedule
• Assessing, documenting, and addressing visual or hearing impairments
• Checking footwear for secure fit, nonskid soles, and no trailing laces
• Ensuring that the patient’s ambulatory assistive devices are within reach at all times, with appropriate precautions taken if patient exhibits confusion and/or diminished safety awareness
• Ensuring patient/family are informed of the increased risk of falls
(RBFT, 2016)

Risk for Impaired Skin Integrity

Stroke patients are at risk for skin breakdown as a result of the inability to feel or move extremities, incontinence, inability to communicate needs, pain, discomfort, and decreased nutritional status. Nursing interventions include:

• Performing regular skin assessment during hospitalization and inpatient rehabilitation using an objective scale such as the Braden Scale
• Providing skin hygiene measures, such as emollients for dry skin
• Maintaining nutrition and hydration
• Turning and repositioning every two hours; positioning patient on affected side for only 30 minutes
• Minimizing skin friction and providing pressure relief early mobility, using special mattresses, wheelchair cushions, and seating until mobility returns
(LNC, 2019b)

Impaired Communication (Aphasia/Dysphasia)

Speech problems following stroke sometimes resolve within hours or days. Some problems, however, are more permanent and require speech and language therapy to improve communication. There are four broad categories of communication problems:

• **Expressive (Broca’s) aphasia**: Patients know what they want to say but cannot find the words.

• **Receptive (Wernicke’s) aphasia**: Patients hear what is being said but cannot make sense of the words.

• **Anomic or amnesia aphasia**: Patients have difficulty using the right names for objects, people, places, or events.
Global aphasia: Patients cannot speak or understand speech, nor can they read or write.
(ASA, 2018c)

Nursing interventions for patients experiencing impaired communication may include:

- Collaborating with speech-language therapy, along with active patient participation, to establish goals
- Reinforcing the individually tailored program
- Making the milieu conducive to communication; remaining sensitive to the patient’s reactions and needs
- Responding to the patient in an appropriate manner and treating the patient as an adult
- Providing emotional support and understanding to allay anxiety
- Avoiding completing the patient’s sentences
- Being consistent in daily routines; providing a written schedule, checklists, or other means to help with memory and concentration (e.g., communication board)
- When speaking to the patient, speaking slowly, giving one instruction at a time and allowing the patient time to process
- Talking to aphasic patients while performing care activities to provide social contact (LNC, 2019b)

Emotional Changes

Emotional changes are common after a stroke and can impact rehabilitation outcome. Nurses are among the members of the rehab team who are expected to address the psychosocial needs of patients by providing support and guidance to improve coping. Occupational therapists as well are responsible for promoting coping and adjustment to the consequences of stroke.

Changes in emotions and emotional behaviors are often the result of changes in abilities and difficulties in understanding what is happening. As a result, patients may experience feelings of helplessness and hopelessness. They may have emotional lability; exaggerated or inappropriate responses to anger, sadness, or happiness; and difficulty expressing themselves. Additionally, a stroke can cause changes in personality so that to others the patient seems like a different person.

Stroke patients often experience anger that is directed toward hospital staff as well as family members. This anger may be the result of damage to the brain, the loss of ability to communicate, or inability to make choices about their daily activities. The only way for a patient to exert control may be to refuse to do tasks or to be involved in treatment.

Stroke patients can easily become irritated, frustrated, and angry and may use language that they did not use prior to the stroke. This may occur when the person is attempting to accomplish
something that formerly was easy and has become difficult post stroke. Interventions can include:

- Talking in a calm and gentle tone
- Drawing the person’s attention away from what is causing anger or frustration
- Giving the person something easier to do

It is important to explore the patient’s previous methods of dealing and coping with life’s problems and the presence and quality of their support systems in order to build on past successes and to mobilize resources (Memorial Health System, 2019).

**DEPRESSION AND ANXIETY**

Psychological reactions to stroke can appear in the days, months, and years after a stroke, and a psychological assessment is made soon after a stroke to determine what, if any, psychological or emotional problems are present. As patients recover, they may begin to understand their situation and what has happened to them. At this point they are at risk for depression or other adjustment difficulties and must be closely monitored.

Two of the most common emotional/psychological problems that can result from a stroke are depression and anxiety. Many patients may experience bouts of crying, feel hopeless, and withdraw from social activities. Others may experience general feelings of fear and anxiety, which may result in acute anxiety attacks.

In the acute care setting, the patient is monitored for signs and symptoms of depression, including sleep disturbance, increased difficulty concentrating, negative coping statements, lethargy, withdrawal, lack of interest in usually enjoyable undertakings, and loss of appetite (WakeMed, 2019; NHS, 2019).

**FAMILY EDUCATION**

It is important to assess the patient’s spouse, partner, family members, and other home caregivers to review expectations and coping skills. The family is also given education and assistance in dealing with the psychological impact of stroke, including the impact on relationships with and among family members as well as sexual relationship issues. Anxiety and worry may begin to increase if information on the impact of the stroke is not provided (WakeMed, 2019; Tsai et al., 2015).

**REHABILITATION IN THE ACUTE STROKE SETTING**

Beyond nursing care, other members of the interdisciplinary team play a prominent role in preparing the patient to be discharged or transferred to an appropriate setting for long-term rehabilitation.
Stroke rehabilitation is based on the awareness that the brain has the capability to compensate for damage through reorganization and creation of new connections among neurons. There are two main types of neuroplasticity:

- **Structural neuroplasticity**, in which the strength of the connections between neurons (synapses) changes
- **Functional neuroplasticity**, which describes the permanent changes in synapses due to learning and development

The best way to encourage neuroplasticity in stroke recovery is to use two key methods:

- Task repetition
- Task-specific practice
  (Ackerman, 2018)

The primary goals of rehabilitation in the acute setting involve:

- Prevention of medical complications
- Prevention of deconditioning and contractures
- Training of new skills
- Optimizing post-stroke rehabilitation:
  - Early assessment with standardized evaluations and validated assessment tools
  - Early employment of evidence-based interventions relevant to individual patient needs
  - Patient access to an experienced multidisciplinary rehabilitation team
  - Ongoing medical management of risk factors and comorbidities
  (Physiopedia, 2018)

**Physical and Occupational 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:

**MOBILITY**

- **Activities of Daily Living (ADL) Index**: Determines abilities with basic ADL and mobility tasks
- **Activity Measure for Post-Acute Care (AM-PAC)**: Measures function in three domains—basic mobility, daily activities, and applied cognition
• **AM-PAC 6 Clicks**: Electronically administered short form of AM-PAC that assesses mobility and self-care abilities through six questions

• **Stroke Impact Scale**: Measures stroke recovery in eight domains including strength, hand function, mobility, ADLs, emotion, memory, communication, and social participation

• **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

**CHANGING AND MAINTAINING BODY POSITION**

• **Assessment of Motor and Process Skills (AMPS)**: Provides an objective assessment of various motor and process skills; requires training to administer

• **Berg Balance Scale (BBS)**: Measures static and dynamic balance abilities using functional tasks commonly performed in everyday life

• **Performance-Oriented Mobility Assessment (POMA)**: Measures both static and dynamic balance using tasks testing balance and gait

• **Timed Get Up and Go Test**: Measures dynamic balance and mobility

**CARRYING, MOVING, AND HANDLING OBJECTS**

• **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)

• **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

• **Jebsen Hand Function Test (JHFT)**: Assesses upper extremity function

• **Arm Motor Ability Test**: Includes unilateral and bilateral tasks completion using basic ADL tasks; best for higher-functioning patients with active movement of wrist and hand

• **Rivermead Motor Assessment (RMA)**: Assesses functional mobility including gait, balance, and transfer

• **Wolf Motor Function Test**: Assesses motor ability of individuals with moderate to severe upper-extremity motor deficits; includes reaching and functional activities requiring fine motor coordination
PERFORMING SELF-CARE

- **Cleveland Scale of Activities of Daily Living**: Evaluates basic ADL abilities in persons with dementia
- **Barthel Index (BI)/Modified Barthel Index**: Widely used measure of functional disability involving evaluation of mobility and independence
- **Patient-Specific Functional Scale**: Patient determines functional ability with five activities (determined by therapist or patient), with ability rated on a 10-point scale

SWALLOWING

- **Mann Assessment of Swallowing Ability (MASA)**: Designed for use in bedside evaluation of patients referred for swallowing function assessment
- **Acute Stroke Dysphagia Screen**: Easily administered and reliable tool with sufficient sensitivity to detect both dysphagia and aspiration risk in acute stroke patients
- **Victorian Dysphagia Screening Model ASSIST Tool**: Recommended in the presence of persisting acute stroke symptoms by personnel who have successfully completed approved training in dysphagia screening
- **Acute Stroke Dysphagia Screen**: Designed for use by practitioners who are not trained speech therapists to determine quickly and accurately that dysphagia exists and to refer for further swallowing assessment

PHYSICAL AND FUNCTIONAL IMPAIRMENTS

- **Function Independence Measure (FIM)**: Widely utilized tool that assesses physical and cognitive disabilities in terms of the burden required for taking care of the patient
- **Orpington Prognostic Scale (OPS)**: Simple, objective bedside evaluation to obtain a baseline assessment of stroke severity
- **Chedoke-McMaster Stroke Assessment**: Used in conjunction with the Functional Independence Measure, employing the same rating methods for disability inventory
- **Motor Assessment Scale (MAS)**: Assesses performance of functional tasks rather than isolated patterns of movement
- **Manual Muscle Testing**: Evaluates function and strength of specific muscles and muscle groups based on the effective performance of a particular movement/movements in relation to the forces of gravity and manual resistance
- **Fugl-Meyer Assessment (FMA):** Stroke-specific, performance-based impairment index that assesses motor functioning, balance, sensation, and joint functioning to determine disease severity, describe motor recovery, and plan and assess treatment

  (AOTA, 2013; Dutton, 2016; Rehab Measures, 2019; Physiopedia, 2019a)

### CMS INPATIENT REHABILITATION FACILITY—PATIENT ASSESSMENT INSTRUMENT (IRF-PAI)

The Center for Medicare and Medicaid Services (CMS) requires the IRF-PAI to assess a patient’s memory status and ability to perform personal care for all patients who receive services in an inpatient rehabilitation setting. Self-care tasks are assessed in Section GG of the IRF-PAI. These tasks include:

1. Eating
2. Oral hygiene
3. Toileting hygiene
4. Showering/bathing self
5. Upper-body dressing
6. Lower-body dressing
7. Putting on/taking off footwear

Tasks are rated on a 6-point scale ranging from dependent to independent and assessed both upon admission and at discharge from the inpatient rehabilitation setting (CMS, 2019).

### 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:

- Preventing complications
- Mobilizing the patient
- Encouraging resumption of self-care activities
- Screening for rehabilitation and choice of settings

Physical therapy goals in **rehabilitation care** involve:

- Developing the rehabilitation plan and monitoring progress
- Managing sensory-motor deficits
• Improving functional mobility and independence
• Preventing and treating complications
• Discharge planning
• Community reintegration
  (Das, 2019)

**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
• Prior activity level
• History of any recent alterations in function prior to stroke

Physical examination includes objective testing of the following:

• Passive range of motion
• Sensation
• Motor control
• Strength
• Coordination
• Postural control/balance
• Functional status
• Gait and locomotion
• Upper limb control
• Endurance
  (Physiopedia, 2019b)

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**: Assessment of passive hypo- or hypermobility of joints, structure and integrity of the joint surfaces, and periarticular soft tissue qualities

• **Range of motion**: Assessment of accessory movement at joint surfaces; tissue extensibility, such as muscle-tension length and movement; and muscle tone, including the presence of spasticity

• **Motor function**: Assessment 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**: Assessment for the excitability of the nervous system and the integrity of the neuromuscular system

• **Circulation**: Evaluation of cardiovascular and lymphatic drainage systems and their ability to adequately meet demands at rest and with activity

• **Ventilation and respiration**: Assessment for 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**: Assessment to determine the presence and underlying cause of gait deviations in the areas of rhythm, cadence, step, stride, and speed

• **Balance**: Assessment of 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**: Assessment 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**: Assessment of the patient’s ability to perform activities of daily living and instrumental activities of daily living

• **Bowel and bladder**: Assessment for flaccidity during the acute stage

• **Assistive technologies**: Assessment to determine the need for assistive and adaptive devices
  
  (Surbala, 2013; Georgiev, 2015; APTA, 2016)

**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. Physical (and occupational) therapists may elect to use one approach or any combination of them.
Neurodevelopmental treatment approach/Bobath is a multidisciplinary approach based on the neuroplasticity of the brain and involving physical therapists, occupational therapists, and speech therapists. It recognizes that all patients with neurodisability have the potential for enhanced function. It 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 (Physiopedia, 2019c).

Proprioceptive neuromuscular facilitation (PNF) stimulates nerve-muscle/sensory receptors to evoke response through manual stimuli in diagonal and reciprocal patterns along with necessary visual component added to increase the ease of movement and to promote function (Bruno-Petrina, 2018).

Rood approach is a therapy in which the modification of muscle tone and voluntary motor activity is attempted using cutaneous sensorimotor stimulation. Movement is directed toward functional goals. 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 stresses the importance of cognitive and perceptual function as well as motor function and discourages compensatory movement. Treatment is functional and task oriented (e.g., stair mobility, getting in and out of the bath). It teaches general strategies for solving motor problems (Physio.co.uk., 2019).

Brunnstrom approach movement therapy. Stroke can affect collaboration between muscles, resulting in abnormal movement patterns. This approach teaches patients how to use the abnormal synergy patterns to their advantage 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/proprioceptive stimulation enhances the synergies. Brunnstrom movement therapy is based on seven stages of recovery from stroke (see box below) (Bruno-Petrina, 2016; Saebo, 2018).

<table>
<thead>
<tr>
<th>BRUNNSTROM 7 STAGES OF RECOVERY FROM STROKE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stage 1:</strong> This is a period of flaccidity when neither reflex nor voluntary movements are present.</td>
</tr>
<tr>
<td><strong>Stage 2:</strong> Basic limb synergies may appear, spasticity appears, and minimal voluntary movement responses may be present.</td>
</tr>
<tr>
<td><strong>Stage 3:</strong> 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.</td>
</tr>
<tr>
<td><strong>Stage 4:</strong> Spasticity begins to decline, and some movement combinations are mastered.</td>
</tr>
</tbody>
</table>

© 2019 WILD IRIS MEDICAL EDUCATION, INC.
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.
(Saebo, 2018)

**ACUTE CARE PHYSICAL THERAPY INTERVENTIONS**

Early initiation of mobility-focused physical rehabilitation as soon as the patient is medically stabilized has been associated with decreased deconditioning, improved long-term functional outcomes, and decreased risk of hospital readmission. Studies of rehab intensity following CVA in the acute setting have found higher-intensity levels to correlate more strongly with decreased readmission than lower intensity levels (Joshi, 2018; Andrews et al., 2015).

However, substantial variation has been observed with regards to the consistency with which post-CVA patients receive physical therapy consultations in the acute care phase, despite considerable association of early rehab initiation with improved long-term outcomes. One longitudinal study found that nearly 25% of CVA patients did not receive a rehabilitation consult during the acute care phase, with factors such as increased severity of stroke, lack of premorbid ambulation ability, and hemorrhagic stroke associated with lower likelihood of receiving an acute care rehab consult (Capo-Lugo et al., 2019).

In the acute care setting, evaluation and interventions provided by physical therapists may include any of the following, depending on a patient’s functional deficits and ongoing needs:

- **Positioning/bed mobility**: Physical therapists advise on safe and correct positioning of the patient in multiple positions, including supine, side-lying (on both affected and unaffected sides), and sitting, in order to avoid injury and promote the patient’s ability to self-mobilize. Early bed mobility training may include teaching patients how to roll side-to-side, transition from supine to/from sitting, sit supported in bed, and sit supported out of bed (with or without back support). Proper positioning can help reduce muscle pain, spasms, slowness, or stiffness that can occur following a stroke.

- **Range of motion (ROM) exercises**: Both passive and active exercises may be initiated early and performed daily in order to promote and maintain joint mobility, protect compromised joints (such as a subluxed shoulder), prevent contractures, increase circulation to extremities, and decrease vascular complications of immobility. ROM exercises may be performed more frequently if patients have increased risk of joint contractures. Effective positioning strategies are important in maintaining soft tissue length and to encourage proper joint alignment. Patients may also be taught ROM self-activities.
• **Managing spasticity:** If spasticity is present, early mobilization and daily stretching may be employed to maintain length of spastic muscles and soft tissues and promote optimal positioning. Modalities may include application of cold or heat, massage, and electrical stimulation.

• **Facilitating upright sitting:** Sitting upright is an important way to build endurance, provide maximum stimulation, and give the patient a sense of normalcy during the acute care phase. Early training in sitting focuses on achieving a symmetrical posture with optimal spine and pelvic alignment.

• **Exercises to improve respiratory and circulatory functions:** If not medically contraindicated, exercises to optimize respiratory and circulatory function may be initiated during this phase. Exercises may include deep breathing and coughing; chest expansion exercises; ankle pumps; and active, active-assisted, or passive upper and/or lower extremity exercises.

• **Decubiti prevention measures:** In order to prevent the complication of pressure injury/ulcers, physical therapists work with and make recommendations to the interdisciplinary team to ensure that patients are properly positioned and that pressure points are protected by appropriate padding, cushioning, and/or unweighting. PT may advise on the use of pressure-reducing devices such as specialized beds/mattresses, foot/ankle positioners, or pressure-relieving wheelchair cushions (such as those with a gel or air-cell core). Improving a patient’s ability to independently mobilize is one of the more important physical therapy interventions with regards to preventing pressure injuries.

• **Transfer techniques:** Physical therapy interventions include teaching the patient how to safely transfer between multiple types of surfaces, including from bed to/from chair and sitting to/from standing. Physical therapists also advise clinical staff on how to appropriately support/assist the patient during transfers (including demonstrating and performing correct transfer technique using assistive devices such as gait belts, assistive devices, or mechanical lifts) as well as recommending the appropriate level of assistance to be used.

• **Balance improvement measures:** As allowed by a patient’s functional mobility status, physical therapists may assist with early-stage balance training activities, including specific bed exercises (such as pelvic bridging), sitting on the edge of bed (with or without external support), standing (with or without support), and progressing to ambulation as appropriate. Improving static and dynamic balance, along with improved ability to ambulate (or self-propel a wheelchair) can lead to greater independence and overall well-being as a patient prepares for discharge.

• **Deconditioning prevention measures:** To prevent deconditioning, physical therapists make recommendations for and encourage early bed mobility and as much out-of-bed time as medically appropriate and tolerated by the patient. Such activities may include side-to-side rolling, transitioning from supine to/from sitting, sitting upright in an
appropriate chair, transferring sit to/from stand, and (when appropriate) ambulation with appropriate assistive devices or propelling a wheelchair.

- **Assistive device training**: Prior to discharge, physical therapists may recommend and/or train the patient in the use of appropriate assistive devices, such as:
  - Wheelchairs
  - Walkers (rolling, standard, hemi-, etc.)
  - Canes (straight, quadripod, etc.)
  - Orthoses (i.e. when foot drop is present)
  - Any devices previously used by the patient (such as orthotics, prosthetics, etc.)

- **Patient/family education**: Physical therapists work with family members/caregivers in order to provide training in how to help with appropriate exercises for the patient as well as how to safely help the patient with functional mobility at home. They also provide education about the physical effects of the stroke and what continued rehabilitation may be able to accomplish for the patient.

- **Discharge planning**: Throughout the hospitalization, physical therapists continually reassess the patient’s functional mobility status 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 and/or other safety modifications.
  (NINDS, 2016d; Surbala, 2013; O’Sullivan et al., 2019; APTA, 2019)

**Occupational Therapy for Acute Stroke Rehab**

Occupational therapy plays a significant role in acute care settings by facilitating early mobilization, improving function, preventing further decline, and coordinating care, including transition and discharge planning. Being an occupational therapist working in acute care is vastly different than other settings since time to work with the patient is limited, typically to only a few days. Rather than working with patients in a full hour-long session, OT must focus on quick, functional interventions.

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

- Rehabilitating and restoring function using physical, cognitive, perceptual, and functional activities
- Teaching restorative or compensatory techniques with or without the use of adaptive equipment, as appropriate
- Providing education on energy conservation techniques that address self-care, functional ability, or therapeutic exercise
- Recommending adaptive equipment and home modifications, if needed

**OCCUPATIONAL THERAPY ASSESSMENT**

An initial assessment by the occupational therapist is performed 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
- Analysis of prehospitalization roles and the patient’s likelihood of resuming them
- Observation of the patient’s abilities to perform personal self-care (e.g., showering, dressing, toileting, grooming, eating)
- Identifying what the patient needs and wants to do, including the supports and barriers
- 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 and motor assessments, with particular emphasis on upper limb and hand function, functional mobility, and transfers to assess ability to complete ADL skills (AOTA, 2017; Stromsdorfer, 2019)

**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
  - Shoulder pain
  - Pressure areas
  - Deep vein thrombosis and pulmonary embolism
  - Contractures
  - Chronic pain in affected joints
- Muscle spasticity
- Extremity swelling

- **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 and using neuromuscular re-education, trunk stabilization, and balance activities to improve the patient’s ability to move in and out of bed and maintain an upright posture necessary to perform self-care

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

- **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, substituting hook and loop fasteners (e.g., Velcro) for buttons on clothing, etc.

- **Cognition and perception:** Addressing cognitive and perceptual deficits, including compensatory techniques

- **Assistive and adaptive devices and techniques:** 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:** Strategies for protecting the joint and reducing pain, including:
  - Positioning and supporting the arm during rest, functional mobility, and wheelchair use by using a hemi-tray or arm trough
  - During the flaccid state, using slings to prevent injury (however, beyond the flaccid stage, slings remain controversial)
Strapping the shoulder
Avoiding the use of overhead pulleys

- **Discharge planning**: Making recommendations for ongoing rehab in settings appropriate to the level of the patient’s rehabilitation needs, including inpatient, outpatient, skilled nursing facilities, home health, or other post-acute settings, can also include community and nontraditional (nonmedical model) settings. Interventions in each of these settings vary depending on the patient’s ability level and stroke severity. (AOTA, 2017; Harrison, 2015; Gillen, 2016)

### Speech-Language Therapy for Acute Stroke Rehab

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) and the development of other means of communication.

All stroke patients are 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). A clinical judgment is then made regarding how to proceed with food and fluids, and if necessary, modifications are made in food and liquid consistency, temperature, taste, and texture in order to improve swallowing function and efficiency (NINDS, 2019d).

### 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 interventions 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) (ASHA, 2019)

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

- Apraxia: Difficulty or inability to move the mouth and tongue to speak
- Aphasia: Impaired language, affecting production or comprehension of speech and ability to read or write
• Dysarthria: Impaired intelligibility of speech as a result of weakness, paralysis, or incoordination of speech musculature

• Cognitive deficits: Problems with attention, memory, perception, insight and judgment, organization, processing speed, problem solving, reasoning, and executive functioning (Mayo Clinic, 2018b; Tactus Therapy, 2018)

Speech-language therapists are the mainstay of dysarthria treatment. The primary aim is to maximize the patient’s ability to communicate with others using the following types of interventions:

• Impairment-level interventions to target impairment of function
  o Nonspeech and oro-motor exercises to improve speed, range, strength, and accuracy of speech/respiratory musculature
  o External stimulation of the muscles

• Activity-level interventions to increase intelligibility by modifying existing speech
  o Modifying the rate of speech
  o Using augmentative or alternative communicator devices such as a communication chart, gestures, writing, drawing

• Participation-level interventions, including support or education for the individual and significant others (Mitchel et al., 2017)

DISCHARGE FROM THE HOSPITAL

As the time of discharge approaches, a patient’s limitations are 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 begin discharging the patient. Nurses on the stroke team also initiate the patient’s transition into the appropriate supervised rehabilitation programs.

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 **ABCDE** describes important elements in preventing secondary stroke:

  A – **Antiaggregants**: Aspirin, clopidogrel, extended-release dipyridamole; and **anticoagulants** (warfarin, apixaban, dabigatran, revaraxaban, edoxaban)
**B – Blood pressure–lowering medications**: thiazide diuretics, calcium-channel blockers, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blockers (ARBs)

**C – Cholesterol-lowering medications** (pravastatin, simvastatin), **cessation** of smoking (prescription nicotine patch, bupropion, varenicline), **carotid** revascularization

**D – Diet**: Such as the Dietary Approaches to Stop Hypertension (DASH), Mediterranean diet, Prudent diet, etc.

**E – Exercise**: Such as 30 minutes of moderate-intensity physical exercise such as walking briskly or using an exercise bicycle each day (Silver, 2019)

**Patient and Family Education and Support**

Following stroke, patients and families are typically faced with multiple life changes and challenges as the patient transitions through the stages of recovery. Both the patient and family should be assessed, educated, and prepared for transitions between care stages and settings and screened for level of coping, risk for depression, and other physical and psychological issues.

Education and support for patient, families, and caregivers may include:

- Written discharge instructions and recommendations that identify collaborative action plans, follow-up care, and goals
- Access to a designated contact person in the hospital or community for continuity of care and queries
- Accurate and up-to-date information about the next care setting, what the patient and family can expect, and how to prepare
- Ongoing access to and advice from health and social service organizations appropriate to needs and stages of transition and recovery
- Links to and information about local community agencies, such as stroke survivor groups, peer survivor visiting programs, meal provider agencies, and other services and agencies
- Shared decision-making/participation regarding transitions between stages of care
- Counseling, preparation, and ongoing assessment for adjustment to:
  - Change of living setting
  - Change in physical needs and increased dependency
  - Change in social roles and leisure activities
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. They 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 most commonly 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 or surrounding 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 primary care provider 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.

Both heart disease and strokes can 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 a stroke. If you have atrial fibrillation, ask your primary care provider how you can reduce your chance of getting a stroke. Also ask your PCP 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. The drug alteplase has been approved by the U.S. Food and Drug Administration for dissolving blood clots in the brain.

Alteplase is usually injected in a vein, where it is carried in the blood stream to the clot in order to break 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 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 PCP’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 PCP to suggest a stop-smoking plan.

3. Control your **diabetes**. People with diabetes have a higher risk of having a stroke. Follow your PCP’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 PCP’s recommendations for your diet and take any medications that are prescribed.

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 PCP for help in making a realistic weight loss plan.
6. Stay active. Regular **exercise** lowers your risk of developing a stroke.

**CONCLUSION**

Strokes, often called *cerebrovascular accidents (CVAs)*, result from limitations or interruptions in cerebral perfusion. Stroke ranks fifth among all causes of death in the United States when considered apart from other cardiovascular diseases.

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. Strokes caused by intracranial bleeds 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. Most ischemic strokes are painless, although hemorrhagic strokes can produce severe headache.

An acute ischemic stroke is a medical emergency, requiring fast, organized care. 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 critical step in evaluating an acute stroke is making the distinction between ischemic and hemorrhagic strokes, and at this point, treatment paths for ischemic and hemorrhagic stroke patients diverge. For ischemic strokes, IV recombinant tissue plasminogen activator (rtPA) is administered. For hemorrhagic strokes due to a ruptured subarachnoid aneurysm, treatment by surgically clipping the aneurysm remnant or by endovascularly inserting a coil may be done.

Following initial evaluation and treatment, stroke patients are monitored in the ICU, and 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.
RESOURCES

American Stroke Association  
http://www.strokeassociation.org

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

Internet Stroke Center  
http://www.strokecenter.org

Neurological Flowsheet (Sutter Medical Center)  

Stroke information (National Institute of Neurologic Disorders and Stroke)  

Stroke assessment scales overview (Internet Stroke Center)  

REFERENCES


© 2019 WILD IRIS MEDICAL EDUCATION, INC.
DISCLOSURE

Wild Iris Medical Education, Inc., provides educational activities that are free from bias. The information provided in this course is to be used for educational purposes only. It is not intended as a substitute for professional healthcare. Neither the planners of this course nor the author have conflicts of interest to disclose. (A conflict of interest exists when the planners and/or authors have financial relationship with providers of goods or services which could influence their objectivity in presenting educational content.) This course is not co-provided. Wild Iris Medical Education, Inc., has not received commercial support for this course. There is no “off-label” use of medications in this course. All doses and dose ranges are for adults, unless otherwise indicated. Trade names, when used, are intended as an example of a class of medication, not an endorsement of a specific medication or manufacturer by Wild Iris Medical Education, Inc., or ANCC. Product trade names or images, when used, are intended as an example of a class of product, not an endorsement of a specific product or manufacturer by Wild Iris Medical Education, Inc., or ANCC. Accreditation does not imply endorsement by Wild Iris Medical Education, Inc., or ANCC of any commercial products or services mentioned in conjunction with this activity.

ABOUT THIS COURSE

You must score 70% or better on the test and complete the course evaluation to earn a certificate of completion for this CE activity.

ABOUT WILD IRIS MEDICAL EDUCATION

Wild Iris Medical Education offers a simple CE process, relevant, evidence-based information, superior customer service, personal accounts, and group account services. We’ve been providing online accredited continuing education since 1998.

ACCREDITATION INFORMATION FOR WILD IRIS MEDICAL EDUCATION
TEST

[ Take the test online at wildirismedicaleducation.com ]

1. Which is a correct statement about the incidence and prevalence of stroke in the United States?
   a. Native Americans are more likely to have a stroke than whites.
   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 a unique stroke risk factor for women?
   a. Alcohol intake
   b. Smoking history
   c. Age over 50 years
   d. Use of hormone replacement therapy

3. Speech and language problems are typically associated with a stroke that occurs in the:
   a. Right side of the brain.
   b. Left side of the brain.
   c. Frontal lobe of the brain.
   d. Brainstem.

4. Which is the leading cause and most important controllable risk factor for stroke?
   a. Diabetes mellitus
   b. Atrial fibrillation
   c. Hypertension
   d. Obesity

5. Exposure to which pollutant is hypothesized to trigger a stroke?
   a. Helium
   b. Nitrogen dioxide
   c. Hydrogen
   d. Carbon dioxide
6. Which structure is not a part of the circle of Willis?
   a. The basilar artery
   b. The right and left anterior cerebral arteries
   c. The posterior communicating artery
   d. The posterior cerebral arteries

7. 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).

8. Which type of stroke constitutes the majority (87%) of strokes?
   a. Transient ischemic attack
   b. Subarachnoid hemorrhage
   c. Intracranial hemorrhage
   d. Ischemic

9. The majority of thrombotic strokes are caused by:
   a. Pieces of plaque.
   b. Cardiac pump failure.
   c. Reduced cardiac output.
   d. Blood clots.

10. Following an ischemic stroke, permanent structural damage within the ischemic penumbra can be prevented if blood supply is restored within:
    a. 36 hours.
    b. 24 hours.
    c. 8 hours.
    d. 6 hours.

11. The stroke syndrome referred to as “locked-in” syndrome is the result of occlusion of the:
    a. Anterior cerebral artery.
    b. Basilar artery.
    c. Posterior cerebral artery.
    d. Vertebral artery.
12. An ischemic stroke for which a comprehensive evaluation cannot define the cause is called a:
   a. Middle cerebral artery stroke.
   b. Cryptogenic stroke.
   c. Anterior cerebral artery stroke.
   d. Transient ischemic attack.

13. A hemorrhagic stroke that results in bleeding into the space surrounding the brain is called:
   a. An intracerebral hemorrhage.
   b. A subarachnoid hemorrhage.
   c. A cerebral infarction.
   d. An arteriovenous malformation.

14. During a hemorrhagic stroke, mechanical damage can increase intracranial pressure, which may result in:
   a. A decrease of blood released into the cerebrospinal fluid.
   b. Brain herniation.
   c. Dissection of brain tissue.
   d. ATP depletion.

15. The most important step for a family member or bystander to take when someone appears to be experiencing a stroke is to:
   a. Quickly 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. Which is not the role of EMS dispatchers in regard to a potential stroke emergency?
   a. Identifying the problem
   b. Assigning high priority
   c. Confirming a possible stroke
   d. Advising on possible first aid
17. EMS best practice states that responders should spend how much time on the scene to complete an assessment and begin transport?
   a. 20 minutes or less
   b. 15 minutes or less
   c. 10 minutes or less
   d. 5 minutes or less

18. In the Cincinnati Prehospital Stroke Scale, the patient is asked to:
   a. “Lie down in a comfortable position.”
   b. “Loosen any tight clothing.”
   c. “Tell me when your symptoms began.”
   d. “Show me your teeth.”

19. While en route to the hospital, emergency responders are instructed to:
   a. Immediately treat hypertension.
   b. Perform a 12-lead ECG.
   c. Routinely administer oxygen.
   d. Treat glucose if less than 60 mg/dL.

20. A facility certified by the Joint Commission (TJC) as an acute stroke-ready hospital must provide:
   a. CT, MRI, and laboratory 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.

21. The stroke center time target for the management of acute stroke from door to treatment is not more than:
   a. 15 minutes.
   b. 25 minutes.
   c. 60 minutes.
   d. 3 hours.
22. The two most essential laboratory tests before treatment of acute stroke are blood glucose level and:
   a. Coagulation studies.
   b. Lipid profile.
   c. Blood typing and crossmatch.
   d. Hematocrit level.

23. 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.

24. 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.

25. The main imaging recommendation for stroke diagnosis is:
   a. Multimodal computed tomography scan.
   b. Noncontrast computed tomography (NCCT) scan.
   c. Magnetic resonance imaging (MRI) scan.
   d. Diffusion weighted imaging (DWI) technique.

26. 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
27. For which reason would a patient be excluded from receiving rtPA within 3 to 4.5 hours from the onset of a stroke?
   a. Patient’s symptoms are not suggestive of subarachnoid hemorrhage
   b. Patient has not had a myocardial infarction in the previous 3 months
   c. Patient is not taking an oral anticoagulant
   d. Patient is over 80 years of age

28. 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. Severe headache, nausea, and/or vomiting.

29. Which is an advantage of intra-arterial rtPA over intravenous rtPA administration?
   a. It delivers a higher concentration of the drug to the clot.
   b. It results in a higher systemic exposure to the drug.
   c. It can be started within 36 hours of stroke onset.
   d. It does not require additional time to initiate.

30. Criteria for receiving endovascular treatment using mechanical thrombectomy include:
   a. Having an asymptomatic carotid artery blockage.
   b. Evidence of a reassuring noncontrast head CT.
   c. Diagnosis with a moderate blockage of the carotid artery.
   d. Being at high risk for carotid endarterectomy.

31. Which is not a true statement regarding managing blood pressure during treatment of stroke?
   a. High blood pressure should be carefully lowered for patients who will receive fibrinolysis.
   b. Drug-induced hypotension has been proven effective for ischemic stroke.
   c. Systemic hypovolemia should be corrected.
   d. Hypertension should be treated if patient has heart failure.
32. Which is a **correct** statement regarding early management of hemorrhagic stroke patients?
   a. Lowering blood pressure does not lessen the absolute growth of hematomas.
   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.

33. A major complication of subarachnoid hemorrhage with poor prognosis is:
   a. Ischemic infarction from vasospasm.
   b. Elevated blood pressure.
   c. Renal impairment due to hypovolemia.
   d. Uncontrolled convulsive seizures.

34. Which is a sign of possible cerebral herniation in a patient with an acute stroke?
   a. Appearance of Cushing’s triad
   b. Sudden severe headache
   c. Intracranial rebleeding
   d. Loss of consciousness

35. 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. Stroke-related pneumonia is associated with a higher mortality.
   d. Common complications of mechanical ventilation are abnormal breathing patterns.

36. 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.

37. Which is a **correct** statement regarding hyperglycemia and hypoglycemia after stroke?
   a. Hypoglycemia exacerbates neuronal damage.
   b. IV dextrose should not be administered.
   c. IV insulin is the treatment of choice.
   d. There is no gold standard for euglycemic management.
38. Which is considered a better bedside screening tool for dysphagia in patients with acute stroke?
   a. Water swallowing test (WST)
   b. Video-fluoroscopy (VFS)
   c. Volume-viscosity swallow test (V-VST)
   d. Fiberoptic endoscopic evaluation of swallowing (FEES)

39. Which intervention should be included in the nursing plan of care for a stroke patient with dysphagia?
   a. Having suction equipment available during early feeding attempts
   b. Starting enteral diet within 24 hours of admission
   c. Maintaining upright position for 15 minutes after eating
   d. Maintaining fluid intake of 1,000 ml per day

40. The patient recovering from a stoke who refers to a fork as a door has which type of aphasia?
   a. Global
   b. Anomic
   c. Receptive
   d. Expressive

41. Which tool is used to assess a patient’s ability to handle objects differing in size, weight, and shape?
   a. Functional Independence Measure (FIM)
   b. Action Research Arm Test (ARAT)
   c. Fugl-Meyer Assessment (FMA)
   d. Assessment of Motor and Process Skills (AMPS)

42. By engaging a stroke patient in early mobilization, a physical therapist is primarily targeting:
   a. Gait abnormalities.
   b. Inability to transfer independently.
   c. Muscle spasms.
   d. Deconditioning.
43. The occupational therapist uses strapping in acute stroke rehabilitation in order to:
   a. Assist with early mobilization.
   b. Prevent contractures.
   c. Manage shoulder pain.
   d. Prevent pressure injuries.

44. Which intervention is used by a speech-language therapist to manage a patient with dysphagia following a stroke?
   a. External stimulation of muscles
   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

45. Which medication is recommended to prevent secondary stroke?
   a. Vitamin K
   b. Ibuprofen
   c. Lorazepam
   d. Clopidogrel

46. When responding to a patient’s question about the relationship between heart disease and stroke, the clinician explains that:
   a. Blood clots caused by an irregular heartbeat can travel to the brain and cause stroke.
   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.