Neurovascular Emergencies

Introduction

Neurovascular emergencies require timely and accurate assessments and treatments to ensure the best clinical outcomes. This course will give a brief overview of the anatomy of the neurovascular system, describe some of the common neurovascular emergencies, and explore the immediate assessment and treatment recommendations for each type of emergency as well as some of the potential complications care providers for these patients may encounter.

Neurovascular Anatomy

The neurovascular system consists of the brain, spinal cord and associated vasculature. This course will focus mainly on issues of the brain. The anatomy of the brain is complex due its intricate structure and function. This amazing organ acts as a control center by receiving, interpreting, and directing sensory information throughout the body. The brain is made up of many specialized areas that work together. The cortex is the outermost layer of brain cells. Thinking and voluntary movements begin in the cortex.

The basal ganglia are a cluster of structures in the center of the brain. The basal ganglia coordinate messages between multiple other brain areas. The limbic system is a group of structures that controls emotions, emotional responses, hormonal secretions, mood, motivation, as well as pain and pleasure sensations. The limbic system consists of the amygdala, cingulate gyrus, fornix, hippocampus, hypothalamus, olfactory cortex, and the thalamus.

The brain is composed of the frontal, occipital, temporal, and parietal lobes as well as the cerebellum and the brain stem. The brain is surrounded by several layers of tissue including the meninges and the dura. The skull (cranium) helps protect the brain from injury.

Frontal Lobe

The frontal lobe is located in the anterior part of the brain. It is involved in planning, organizing, problem solving, selective attention, personality and a variety of higher cognitive functions including behavior and emotions. The most anterior portion of the frontal lobe is called the prefrontal cortex. It is very important for the higher cognitive functions and the determination of the personality. The posterior portion of the frontal lobe consists of the premotor and motor areas. Nerve cells that produce movement are located in the motor areas. The premotor areas serve to modify movements.

Occipital Lobe

The occipital lobe is in the back of the brain and processes visual information. Not only is the occipital lobe mainly responsible for visual reception, it also contains association areas that help in the visual recognition of shapes and colors. Damage to this lobe can cause visual deficits.
Temporal Lobe
The temporal lobes are located on each side of the brain at about the level of the ears. These lobes allow one to differentiate sounds and smells. They also help in sorting new information and are believed to be responsible for short-term memory. The right temporal lobe is mainly involved in visual memory (i.e., memory for pictures and faces) while the left temporal lobe is mainly involved in verbal memory (i.e., memory for words and names).

Parietal Lobe
The parietal lobes are located on each side of the brain behind the frontal lobe at the top of the brain. The parietal lobes contain the primary sensory cortex which controls sensation (touch, pressure). Behind the primary sensory cortex is a large association area that controls fine sensation (judgment of texture, weight, size, shape.) Damage to the right parietal lobe can cause visuo-spatial deficits (e.g., the patient may have difficulty finding their way around new, or even familiar, places). While damage to the left parietal lobe may disrupt a patient's ability to understand spoken and/or written language.

Cerebellum
The cerebellum is the portion of the brain (located at the back) which helps coordinate movement (balance and muscle coordination). Damage to this area may result in ataxia which is a problem of muscle coordination. This can interfere with a person's ability to walk, talk, eat, and to perform other self-care tasks.

Brainstem
The brainstem is the lower extension of the brain where it connects to the spinal cord. Neurological functions located in the brainstem include those necessary for survival (breathing, digestion, heart rate, blood pressure) and for arousal (being awake and alert).
Most of the cranial nerves come from the brainstem. The brainstem is the pathway for all fiber tracts passing up and down from peripheral nerves and spinal cord to the highest parts of the brain. The brainstem is further divided into the midbrain, medulla oblongata, and the pons.

**Cerebral Vasculature**

The cerebral vascular system is also complex and intricate. The brain receives blood from two sources: the internal carotid arteries (ICAs), which arise at the point in the neck where the common carotid arteries bifurcate; and the vertebral arteries. The internal carotid arteries branch to form two major cerebral arteries, the anterior and middle cerebral arteries (ACA and MCA respectively). The right and left vertebral arteries come together at the level of the pons on the ventral surface of the brainstem to form the midline basilar artery. The basilar artery joins the blood supply from the internal carotids in an arterial ring at the base of the brain called the circle of Willis. The posterior cerebral arteries (PCAs) arise at this confluence, as do two small bridging arteries, the anterior and posterior communicating arteries. Conjoining the two major sources of cerebral vascular supply via the circle of Willis presumably improves the chances of any region of the brain continuing to receive blood if one of the major arteries becomes occluded.

The major branches that arise from the internal carotid artery - the anterior and middle cerebral arteries - form the anterior circulation that supplies the front part of the brain. These arteries also originate from the circle of Willis. Each gives rise to branches that supply the cortex and branches that penetrate the basal surface of the brain, supplying deep structures such as the basal ganglia and thalamus. Especially prominent are the lenticulostriate arteries that branch from the middle cerebral artery. These arteries also supply the basal ganglia and thalamus. The posterior circulation of the brain supplies the posterior cortex, the middle part of the brain and the brainstem. It comprises arterial branches arising from the posterior cerebral, basilar and vertebral arteries. The pattern of arterial distribution is similar for all the subdivisions of the brainstem. Midline arteries supply medial structures, lateral arteries supply the lateral brainstem and dorsal-lateral arteries supply dorsal-lateral brainstem structures and the cerebellum. Among the most important dorsal-lateral arteries are the posterior inferior cerebella artery (PICA) and the anterior inferior cerebella artery (AICA) which supply specific areas of the medulla and pons. These arteries, as well as branches of the basilar artery that penetrate the brainstem from its ventral and lateral surfaces are especially common sites of occlusion and result in specific functional deficits of cranial nerve, somatic sensory and motor function.
Common Neurovascular Emergencies

Transient Ischemic Attack (TIA)
The term TIA was first introduced in the early 1950s based upon the recognition that transient focal loss of neurologic function often preceded strokes. Since then, transient ischemic attack has been defined as a temporary episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction. The end point of such an episode is biologic (tissue injury) rather than an arbitrary time limit.

TIA was originally defined as a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours and caused by a reversible cerebral ischemia. However, this classic definition of TIA was inadequate for several reasons. Most importantly, there is risk of permanent tissue injury (i.e. infarction) even when focal transient neurologic symptoms last less than one hour. Additionally, about one-half of patients with classically defined TIA syndromes have corresponding appropriate ischemic lesions by brain MRI.

So, the benign connotation of the term TIA was replaced by an understanding that even relatively brief ischemia can cause permanent brain injury. Perhaps more importantly, patients with TIA are at increased risk of recurrent stroke, and therefore require urgent evaluation and treatment since immediate intervention may substantially reduce the risk of recurrent stroke.
**Initial Evaluation**

Patients who have a suspected TIA require urgent evaluation due to the high stroke risk associated with TIA. Additionally, immediate intervention after patients experience a TIA may prevent a significant number of strokes.

The initial evaluation of suspected TIA includes basic laboratory studies that are suggested by the history and physical examination, an electrocardiogram, brain imaging, and neurovascular imaging. Laboratory testing is helpful to rule out metabolic and hematologic causes of neurologic symptoms, including hypoglycemia, hyponatremia, and thrombocytosis.

Several neurologic disorders give rise to transient focal neurologic symptoms, and these should be considered before determining a diagnosis of TIA. In addition to TIAs, the most frequent and important causes of discrete self-limiting attacks include seizures, migraine auras and syncope. Less frequent causes include pressure or position related peripheral nerve or nerve root compression that causes transient numbness and tingling and metabolic disturbances such as hypoglycemia and hepatic, renal, and pulmonary encephalopathies that can produce temporary changes in behavior and movement.

Whether patients presenting with TIA should be hospitalized or not is not clear, but urgent assessment and management is essential in either case. In 2009 the American Heart Association (AHA) and American Stroke Association (ASA) set guidelines for the definition and evaluation of TIA. These guidelines state that it is reasonable to hospitalize patients with TIA who present within 72 hours of symptom onset and meet any of the following criteria:

- ABCD² score of 3 or greater
- ABCD² score of 0 to 2 and uncertainty that the diagnostic workup can be completed within two days as an outpatient
- ABCD² score of 0 to 2 and other evidence that the event was caused by focal ischemia

The ABCD² score (i.e. ABCD squared, for Age, Blood pressure, Clinical features, Duration of symptoms, and Diabetes) is a simple prognostic assessment tool with moderate predictive accuracy that was designed to identify patients at high risk of ischemic stroke in the first two days after TIA. This tool will be discussed further later in this course.

The National Stroke Association recommends that hospitalization be considered for patients with a first TIA within the past 24 to 48 hours, and is generally recommended for patients with the following conditions:

- Crescendo TIAs
- Duration of symptoms greater than one hour
- Symptomatic internal carotid artery stenosis greater than 50%
- Known cardiac source of embolus such as atrial fibrillation
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- Known hypercoagulable state
- High risk of early stroke after TIA

Patients who need urgent evaluation and are not hospitalized should have rapid access to brain imaging with head CT and/or MRI, neurovascular studies such as CT angiography (CTA), MR angiography (MRA), and/or ultrasound; and electrocardiogram.

All patients with a TIA within the past two weeks who are not hospitalized should undergo investigations within 24 to 48 hours to determine the mechanism of ischemia and subsequent preventative therapy. In addition, patients who are not admitted should be informed that they need to go to an Emergency Department immediately if symptoms recur.

**Treatment**
Thorough evaluation and assessment should occur as soon as possible for all patients with suspected TIA. The preferred approach to treatment of TIA is to determine the pathophysiology of the event so that specific stroke preventative therapy can be prescribed. This includes brain imaging with CT or MRI; cardiac evaluation including a 12 lead electrocardiogram and possible echocardiogram; cardiac monitoring to exclude atrial fibrillation; and other blood tests such as complete blood count (CBC), proteomic time (PT) and partial thromboplastin time (PTT), serum electrolytes and creatinine, fasting blood glucose and lipids, as well as erythrocyte sedimentation rate (ESR.)

**Prognosis**
TIA is a neurologic emergency because patients with TIA are at increased risk of stroke. Studies have shown that TIAs are most likely to occur in the hours and days immediately preceding ischemic stroke. Recognition and urgent evaluation of TIAs can identify patients who may benefit from preventative therapy or from revascularization of large vessels such as the carotid artery.

**Predicting Stroke Risk After TIA**
Methods that can accurately assess the risk of stroke after TIA in individual patients would be useful in triaging patients. A simple assessment called the ABCD² score can be used to identify patients at high risk of ischemic stroke in the first seven days after TIA.

The ABCD² score is tallied as follows:

- Age (greater than or equal to 60 years = 1 point)
- Blood pressure elevation when first assessed after TIA (systolic greater than or equal to 140 mmHg or diastolic greater than or equal to 90 mmHg = 1 point)
- Clinical features (unilateral weakness = 2 points; isolated speech disturbance = 1 point; other = 0 points)
- Duration of TIA symptoms (greater than or equal to 60 minutes = 2 points; 10 to 59 minutes = 1 point; less than 10 minutes = 0 points)
- Diabetes (present = 1 point)
Research estimates the two-day stroke risk determined by the ABCD² score as follows:

- Score 6 to 7: High risk
- Score 4 to 5: Moderate risk
- Score 0 to 3: Low risk

**Stroke**

A stroke is the acute neurologic injury that occurs as a result of the pathologic process of either ischemia or hemorrhage within the brain. Approximately 87% of strokes are due to ischemic cerebral infarction and 13% to brain hemorrhage. The American Heart Association (AHA) estimates that about 780,000 strokes occur each year; 600,000 of these are new strokes and 180,000 are recurrent strokes. In 2007, the overall mortality rate from stroke was 273,000, which makes stroke the third-leading cause of death in the United States. Between 1979 and 2005, the annual number of hospital discharges with stroke as the diagnosis was about 900,000. Direct and indirect costs associated with stroke are estimated to be approximately $65.5 billion. Direct costs are attributed to the initial hospitalization, skilled nursing care, physician and nursing care, medications and durable medical equipment, home health care, and acute rehabilitation. Indirect costs include loss of productivity (loss of future earnings) due to morbidity and mortality and loss of esteem (place in family and society) due to disability.

**Types of Stroke**

The two categories of stroke, hemorrhage and ischemia, are diametrically opposite conditions. Hemorrhage is characterized by too much blood within the closed cranial cavity, while ischemia is characterized by too little blood to supply an adequate amount of oxygen and nutrients to a part of the brain. Each of these categories can be divided into subtypes that have somewhat different causes, clinical pictures, clinical courses, outcomes, and treatment strategies. Stroke is classified into the following subtypes: intracerebral hemorrhage, subarachnoid hemorrhage, and brain ischemia due to thrombosis, embolism, or systemic hypoperfusion.

**Intracerebral Hemorrhage**

Bleeding in intracerebral hemorrhage (ICH) is usually derived from arterioles or small arteries. The bleeding is directly into the brain, forming a localized hematoma that spreads throughout certain predictable pathways. Accumulation of blood occurs over minutes or hours and the neurologic symptoms usually increase gradually over minutes or a few hours. In contrast to brain embolism and subarachnoid hemorrhage, the neurologic symptoms do not begin abruptly and are not maximal at onset. The most common causes of ICH are hypertension, trauma, bleeding disorders, amyloid angiopathy, illicit drug use (mostly amphetamines and cocaine), and vascular malformations. Less frequent causes include bleeding into tumors, aneurysmal rupture, and vasculitis.

**Subarachnoid Hemorrhage**

Rupture of an aneurysm releases blood directly into the cerebrospinal fluid (CSF) under arterial pressure. The blood spreads quickly within the CSF, rapidly increasing the intracranial pressure. Death or deep coma ensues if the bleeding continues. The bleeding
usually lasts only a few seconds, but rebleeding is very common. With causes of SAH other than aneurysm rupture, such as trauma, vascular malformations, bleeding disorders, amyloid angiopathy or illicit drug use, the bleeding is less abrupt and may continue over a longer period of time.

Symptoms of SAH begin abruptly in contrast to the more gradual onset of ICH. The sudden increase in pressure causes a cessation of activity (e.g. loss of memory or focus or knees buckling.) Headache is an invariable symptom and is typically instantly severe and widespread (often referred to as a “thunderclap headache” or “the worst headache of my life”). The onset of the headache may or may not be associated with a brief loss of consciousness, seizure, nausea, vomiting, focal neurologic deficit, or stiff neck. There are usually no important focal neurologic signs unless bleeding occurs into the brain and the CSF at the same time (meningocerebral hemorrhage.) Onset headache is more common in SAH than ICH, whereas the combination onset headache and vomiting is infrequent in ischemic strokes.

*Brain Ischemia – Thrombosis*
Thrombosis generally refers to localized obstruction of an artery. The obstruction may be due to disease of the arterial wall, such as arteriosclerosis, dissection, or fibromuscular dysplasia; there may or may not be superimposed thrombosis. Thrombotic strokes can be divided into either large or small vessel disease. In patients with thrombosis, the neurologic symptoms often fluctuate, remit, or progress in a sputtering fashion.

*Brain Ischemia – Embolism*
Emboli refer to particles of debris originating elsewhere that block arterial access to a particular brain region. Embolic strokes may arise from a source in the heart, aorta, or large vessels. The embolus suddenly blocks the recipient site so that the onset of symptoms is usually maximal at the start. Unlike thrombosis, multiple sites within different vascular territories may be affected when the source is the heart or aorta. Since the process is not local (as with thrombosis), local therapy only temporarily solves the problem; further events may occur if the source of embolism is not identified and treated. Embolic strokes are divided into four categories:

- Those with a known cardiac source
- Those with a possible cardiac or aortic source based on transthoracic and/or transesophageal echocardiographic findings
- Those with an arterial source
- Those with a truly unknown source in which these tests are negative or inconclusive

*Brain Ischemia – Systemic Hypoperfusion*
Systemic hypoperfusion is a more general circulatory problem, manifesting itself in the brain and perhaps other organs. Reduced perfusion can be due to cardiac pump failure caused by cardiac arrest or arrhythmia, or to reduced cardiac output related to acute myocardial ischemia, pulmonary embolism, pericardial effusion, or bleeding. Hypoxemia may further reduce the amount of oxygen carried to the brain. Reduced cerebral blood
flow is more global in patients with systemic hypoperfusion and does not affect isolated regions. Symptoms of brain dysfunction typically are diffuse and nonfocal in contrast to the other two types of ischemia. The neurologic signs are typically bilateral, although they may be asymmetric when there is preexisting asymmetrical craniocerebral vascular occlusive disease.

**Initial Care of Patients With Suspected Stroke**

Phase 1 of stroke care, the emergency or hyperacute phase, encompasses the first three to 24 hours after onset of stroke. This phase generally incorporates the prehospital (activation of emergency medical services [EMS]/9-1-1 and response) and Emergency Department care protocols. The focus is on identifying stroke symptoms and infarct location, assessing the patient for risk of acute and long-term complications, and determining treatment options.

Phase 2 of stroke care includes acute care, which encompasses the period from 24 to 72 hours after onset of stroke. In this phase, the focus is on clarifying the cause of stroke, preventing medical complications, preparing the patient and family for discharge, and instituting long-term secondary prevention modalities. There is considerable evidence that dedicated stroke teams, units and coordinated care improve clinical outcomes in the acute care phase.

Optimal management of the stroke patient in the emergency or hyperacute phase requires an accurate and systematic evaluation that is coordinated and timely. Once a potential stroke is suspected, EMS personnel and nurses must determine the time at which the patient was last known to be well (last known well time). This time is the single most important determinant of treatment options during the hyperacute phase.

The key elements of prehospital care are stabilization of the circulation, airway and breathing (the CABs); identification of signs and symptoms of stroke; establishment or verification of the last known well time; provision of supplemental oxygen to patients with hypoxemia; checking the blood glucose level; avoidance of the administration of glucose-containing fluids (unless the patient is hypoglycemic); rapid initiation of transport (load and go); and delivery of patients to receiving centers capable of rapidly caring for acute stroke.

Recognition of stroke symptoms is an important factor in successful delivery of proven acute therapies. Prehospital assessment tools have been developed to help enhance recognition of stroke symptoms and improve the ability to identify stroke patients in the field. The most common and well-investigated tools are the Cincinnati Prehospital Stroke Scale and the Los Angeles Prehospital Stroke Screen. Newer stroke identification tools include the Face Arm Speech Test (FAST). The FAST is simple and easy to remember tool that assesses four elements of stroke:
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- Facial palsy (noting affected side)
- Arm weakness (noting affected side)
- Speech impairment
- Time of onset

Neurological assessment of the stroke patient should always include the CABs, vital signs, cardiac monitoring during transport, and baseline neurological assessment. Because the field neurological examination will serve as a baseline for assessment of neurological improvement or worsening, the use of a prehospital stroke scale is recommended.

EMS personnel on the scene should ask the patient’s family or bystanders when the patient was last known to be normal or without neurological deficits (i.e., the last known well time.) Documentation of this report of onset can be helpful in establishing an accurate time of stroke symptom onset. When possible, the information should be obtained directly from the patient. If the patient is unable to give this information, EMS personnel should look to another reliable source for this information. If the time of onset of stroke symptoms is not identifiable, a standard method of time parameters should be used, such as morning (6:00 am to 11:59 am), afternoon (noon to 5:59 pm), evening (6:00 pm to 11:59 pm), and overnight (midnight to 5:59 am). EMS providers must emphasize to families the importance of traveling to the hospital with the patient, particularly if symptom onset is within the time frame for thrombolytic administration and the patient’s language or decision-making capability is compromised. When family members cannot accompany the patient, EMS personnel should document the family’s contact information and provide it to the emergency physician. The current guidelines recommend the use of continuous cardiac monitoring during transport of a suspected stroke patient to determine the presence of cardiac arrhythmias.

Blood pressure should be monitored every 15 minutes, or more often if severe hypertension (systolic blood pressure >200 mm Hg) or relative hypotension (systolic blood pressure <110 mm Hg) is observed during transport. Administration of antihypertensive drugs in the field is not recommended, because induced hypotension carries a possible risk of extending the area of cerebral infarct. Supplemental oxygen should be given to hypoxic patients; in ambulances without oximetry capabilities, oxygen can be administered at low levels, (e.g., 2 to 3 L/min.) If pulse oximetry is available and the patient’s oxygen saturation is >92%, additional oxygen is not needed. Transport with the head of the bed elevated ≈30° may help with oxygenation and may minimize the possibility of aspiration. To decrease the risk of aspiration, the patient should receive nothing by mouth (NPO).

Hypoglycemia, a common stroke mimic, can be identified quickly by measuring blood glucose during transport. Finger-stick tests can be performed if the emergency vehicle is appropriately equipped and personnel are trained. Treatment of severe hypoglycemia should be instituted promptly by EMS personnel. Intravenous access can be established in the field, and non-glucose-containing intravenous fluids can be started if the patient is hypotensive. Establishment of intravenous access should not delay transport.
The EMS providers should give early notification to the ED of the arrival of a potential acute stroke patient. Historic cardiac trials have shown that prearrival notification of the ED enhances rapid diagnostic workup, reducing time between symptom onset and treatment.

Emergency personnel initiate basic triage and care modalities in the field. Once the stroke patient arrives in the ED, patient triage is usually a function of nursing staff. The Emergency Nurses Association and the American College of Emergency Physicians recommend a 5-level Emergency Severity Index as a preferred system for triage in a busy ED. This index puts all stroke patients in the level 2 or “needs immediate assessment” category, the same as for an unstable trauma patient or a critical care cardiac patient.

The ED triage nurse should use specialized checklists, protocols, and other tools to identify stroke patients. Once stroke is confirmed, the nurse uses these procedures and protocols that define who contacts the acute stroke team or appropriate neurological consultant. Emergency nurses should understand that time is critical and be trained in rapid assessment and treatment of stroke patients. Studies have shown that the sooner thrombolytic therapy is started, for patients with ischemic strokes, the greater the benefit.

It is critical that all emergency nurses and other emergency professional staff know that the NIH-National Institute of Neurological Disorders and Stroke (NINDS) benchmark treatment time for acute ischemic stroke (AIS) with intravenous thrombolytics is within 60 minutes of arrival in the ED. In some cases, this time will need to be shortened to successfully initiate thrombolytic therapy within three hours of stroke onset, although there is growing evidence of safety and effectiveness beyond the three hour window from stroke onset. The AHA AIS Writing Committee has issued a Science Advisory stating that some eligible patients may be treated between the three and 4.5 hour window after stroke. The recommendation comes with several caveats and exclusions, however.

As in the prehospital phase, initial patient assessments made by the emergency nurse are based on the principle of assessing the CABs, vital signs, and neurological assessment. The majority of patients with ischemic strokes will present to the ED in a hemodynamically stable condition; however, ischemic strokes involving the posterior circulation can require aggressive airway management, especially if the patient has an altered level of consciousness.

Circulatory collapse or cardiac arrest, although possible, is uncommon in isolated stroke. The occurrence of either may indicate other medical conditions such as acute myocardial infarction, atrial fibrillation, or congestive heart failure. Cardiac monitoring of all suspected stroke patients in the ED helps identify these conditions.

Initial ED documentation of the stroke patient begins with the recording of all information included in the neurological/stroke assessment. Vital signs, including temperature, may be measured frequently as clinically indicated but not less than every 30 minutes while the patient is in the ED. Hyperthermia is associated with poor outcome.
in stroke patients; therefore, it is important to consider treating any fever >99.6°F.

*Early Assessments for Stroke Patients*

Patients suffering from stroke require skilled providers to complete thorough and timely assessments and interventions. This course will discuss specific treatments for different types of stroke later, but will first give an overview of assessments that must be completed for patients with all types of stroke.

*Vital signs*

Parameters of particular concern in patients with stroke include blood pressure, breathing and temperature. The mean arterial pressure (MAP) is usually elevated in patients with an acute stroke. This may be due to chronic hypertension, which is a major risk factor for ischemic stroke. However, an acute elevation in blood pressure often represents an appropriate response to maintain perfusion of the brain. A neuroimaging study with CT or MRI is critical to help guide blood pressure therapy in stroke patients. The observation that the blood pressure frequently rises spontaneously following cerebral ischemia is consistent with this protective hypothesis, although a stress response to the acute event and to hospitalization may also contribute. The hypertensive effect is transient, as blood pressure falls by as much as 20/10 mmHg within 10 days.

The decision to treat elevated blood pressure requires a balance between the potential danger of severe increases in blood pressure, and a possible decline in neurologic functioning when blood pressure is lowered. Blood pressure management in acute stroke remains controversial. Guidelines from the American Heart Association/American Stroke Association published in 2007 suggest that antihypertensive medications should be restarted at approximately 24 hours after stroke onset in patients with preexisting hypertension who are neurologically stable, unless a specific contraindication to restarting treatment is known. However, patients with extracranial or intracranial stenosis may require a slower reduction in blood pressure (e.g. over seven to 10 days after ischemic stroke) as some degree of blood pressure elevation may be necessary to maintain cerebral blood flow to ischemic brain regions. If pharmacologic therapy is given, intravenous labetalol is generally the drug of choice. Specific recommendations for blood pressure management related to thrombolytic therapy will be discussed later.

In patients with ICH and SAH, the approach to blood pressure management must take into account the potential benefits (e.g. reducing further bleeding) and risks (e.g. reducing cerebral perfusion) of blood pressure lowering. Reducing the blood pressure in patients with ICH or SAH may be beneficial by minimizing further bleeding and continued vascular damage. Patients with an intracranial hemorrhage due to ICH or SAH may have increased intracranial pressure (ICP) due to blood within the cranium. Cerebral perfusion pressure (CPP) equals MAP minus ICP. Thus, increases in MAP may be the only means to maintain CPP above 60 to 70 mmHg, the level necessary to maintain brain perfusion. Measuring ICP directly allows blood pressure to be reduced as low as possible while still maintaining the CPP above the conservative level of 60 mmHg.
Intravenous labetalol is generally the first drug of choice if pharmacologic therapy is necessary in the acute phase, since it allows rapid and safe titration to the goal blood pressure. Other first line agents include transdermal nitroglycerine paste and intravenous nicardipine. Intravenous nitroprusside should be considered second line therapy since it carries added theoretical risks of increasing ICP or affecting platelet function, but in fact it is often needed. Sublingual nifedipine should be avoided because it can cause a prolonged and precipitous decline in blood pressure.

Patients with increased intracranial pressure (ICP) due to hemorrhage, vertebrobasilar ischemia, or bihemispheric ischemia can present with a decreased respiratory drive or muscular airway obstruction. Hypoventilation, with a resulting increase in the partial pressure of carbon dioxide, may lead to cerebral vasodilation which further elevates ICP. Intubation may be necessary to restore adequate ventilation and to protect the airway in these patients. This is especially important in the presence of vomiting, which occurs commonly with increased ICP, vertebrobasilar ischemia, and intracranial hemorrhage. Patients with adequate ventilation should have their oxygen saturation monitored. Patients who are hypoxic should receive supplemental oxygen. Supplemental oxygen should not routinely be given to nonhypoxic stroke victims.

Fever may occur in patients with an acute stroke and can worsen brain ischemia. Normothermia should be maintained for at least the first several days after an acute stroke. Hyperthermia may act via several mechanisms to worsen cerebral ischemia including:

- Enhanced release of neurotransmitters
- Exaggerated oxygen radical production
- More extensive blood-brain barrier breakdown
- Increased numbers of potentially damaging ischemic inhibition of protein kinases
- Worsening of cytoskeletal proteolysis

**Positioning, and Oral Intake**

Positioning of the head of the bed must be individualized for each patient. The traditional positioning at 25° to 30° is often used for potentially increased intracranial pressure (ICP), at least until large lobar, ICH, space-occupying lesions or other causes of increased ICP can be ruled out by imaging. Stroke patients with increased ICP and chronic respiratory conditions may need head elevation for maximum oxygenation. The bed should be elevated at least 30° if the patient is at risk of aspiration or airway obstruction due to dysphagia.

The optimal position for the head of the bed has not been identified. Recent studies have suggested that positioning of the head of the bed can facilitate an increase in cerebral blood flow and maximize oxygenation to cerebral tissue. A study using transcranial Doppler technology found that the head-flat position maximized blood flow to the brain. Further studies on head positioning of the stroke patient need to be completed; if the patient has a lower risk of increased ICP and is not at risk for aspiration, the head-down position has been shown to be beneficial.
When significant hemiparesis is present, positioning on the paretic side may be more desirable to allow the patient to communicate and to prevent aspiration. Stroke patients are more prone to atelectasis as a result of immobility regardless of position. The patient’s neck should be kept straight, airway patency maintained, and slumped sitting avoided to prevent hypoxia.

Patients should be kept NPO to prevent aspiration; this includes no oral medications, until ability to swallow can be assessed. Emergency nurses may be trained to perform a bedside swallowing assessment to establish whether the patient can safely receive oral intake and swallow ED medications such as aspirin. If swallowing is impaired, medications can be administered rectally, intravenously, or by nasogastric tube.

**Laboratory Assessments**

An electrolyte imbalance can sometimes produce stroke-like symptoms. A comprehensive metabolic panel indicates fluid and electrolyte status. The blood and hemostatic system can be assessed by a complete blood count (CBC) with platelet and coagulation studies such as prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), and fibrinogen. Urinalysis assesses renal function and coexisting urinary tract infection (UTI). Emergent laboratory specimens should be labeled STAT to expedite processing, if the patient is in the time window to receive thrombolytics. All patients with suspected stroke should also have the following studies upon admission to the ED:

- Electrocardiogram
- Cardiac enzymes and troponin
- Lipid profile

The following additional laboratory studies may be appropriate in selected patients:

- Liver function tests
- Toxicology screen
- Blood alcohol level
- Pregnancy test in women of child-bearing potential
- Arterial blood gas if hypoxia is suspected
- Lumbar puncture if SAH is suspected and head CT is negative for blood
- Electroencephalogram (EEG) if seizures are suspected
- Chest radiography and blood cultures if fever is present
- Type and cross match in case fresh frozen plasma (FFP) is needed to reverse a coagulopathy if ICH is present

Blood glucose levels should also be checked as soon as possible, even if it was done previously in the field. Hyperglycemia, generally defined as a blood glucose level > 126 mg/dL is common in stroke patients and is associated with poor functional outcome. Hyperglycemia may augment brain injury by several mechanisms including increased tissue acidosis from anaerobic metabolism, free radical generation, and increased blood
brain barrier permeability. On the other end of the spectrum, hypoglycemia can cause focal neurologic deficits mimicking stroke, and severe hypoglycemia can cause neuronal injury. It is important to check the blood sugar and rapidly treat if necessary. Normoglycemia is the desired goal.

*Intravenous Access*
Ideally, two to three intravenous sites should be established if the acute stroke patient will receive thrombolytic therapy. One site is used for administration of intravenous fluids, another for administration of thrombolytic therapy, and the third for administration of intravenous medications. Diagnostic laboratory blood specimens should be drawn before intravenous fluids are started. Collection of specimens before the patient undergoes imaging allows simultaneous processing of both laboratory and imaging data and facilitates rapid turnaround.

*Intravenous Fluids*
Glucose can have detrimental effects in acute brain injury of all types. Therefore, intravenous solutions with glucose (such as D<sub>5</sub>W [dextrose 5% in water]) should be avoided in stroke patients in the ED. An infusion rate that maintains normovolemia (75 to 100 mL/h) can help facilitate normal circulating blood volume. Stroke patients often present in a hypovolemic state, which may produce hypotension and cerebral hypoperfusion. In these instances, intravenous fluid boluses may be administered, with concomitant close evaluation of the patient’s cardiovascular response.

*Past Medical History*
It can be challenging and time-consuming to obtain a stroke history in the emergency phase. As part of the stroke team, emergency nurses can play a key role in helping obtain a pertinent clinical history from the stroke patient and family members. Important questions to ask include:

- Time patient last known well (will be used as presumed time of onset)
- Time symptoms were first observed (if different from time last known well)
- Was anyone with patient when symptoms began? If so, who?
- History of diabetes?
- History of hypertension?
- History of seizures?
- History of trauma related to current event?
- History of myocardial infarction or angina?
- History of cardiac arrhythmias? Atrial fibrillation?
- History of prior stroke or TIA?
- What medications is patient currently taking? Is patient receiving anticoagulation therapy with warfarin?

*Neurological Assessment*
The patient’s account of his or her neurologic symptoms and the neurologic signs found on examination can give a wealth of information about the location of the process occurring in the patient’s brain. A thorough neurological assessment is a very important
aspect of the stroke patient’s care. Neurological status should be assessed frequently and any changes should be reported to the treating physician immediately. Care providers should know that the three most predictive examination findings for the diagnosis of stroke are facial paresis, arm drift/weakness, and abnormal speech. One of the most important and telling assessments is the patients’ level of consciousness. A decrease in level of consciousness is often one of the first signs of deterioration a patient will exhibit.

There are several standardized scales used to assess neurologic function including the Glasgow Coma Scale (GCS), the FOUR Score and the National Institutes of Health Stroke Scale (NIHSS).

The Glasgow Coma Scale is based on a 15 point scale for estimating and categorizing the outcomes of brain injury on the basis of overall social capability or dependence on others. GCS was initially used to assess level of consciousness after head injury, but the scale is now used by first aid, EMS, physicians and other members of the healthcare team as being applicable to all acute medical and trauma patients. The test measures the motor response, verbal response and eye opening response with these values:

**I. Motor Response**
- 6 - Obeys commands fully
- 5 - Localizes to noxious stimuli
- 4 - Withdraws from noxious stimuli
- 3 - Abnormal flexion, i.e. decorticate posturing
- 2 - Extensor response, i.e. decerebrate posturing
- 1 - No response

**II. Verbal Response**
- 5 - Alert and Oriented
- 4 - Confused, yet coherent, speech
- 3 - Inappropriate words and jumbled phrases consisting of words
- 2 - Incomprehensible sounds
- 1 - No sounds

**III. Eye Opening**
- 4 - Spontaneous eye opening
- 3 - Eyes open to speech
- 2 - Eyes open to pain
- 1 - No eye opening

The final score is determined by adding the values of I+II+III. This number helps medical practitioners categorize the patients’ condition related to survival, with a lower number indicating a more severe injury and a poorer prognosis. Generally patients with scores from three (the lowest possible score) to eight are said to be in a coma.
The FOUR Score is a clinical grading scale designed for use by medical professionals in the assessment of patients with impaired level of consciousness. "FOUR" in this context is an acronym for "Full Outline of UnResponsiveness." The FOUR Score is a 17-point scale (with potential scores ranging from 0 to 16). Decreasing FOUR Score is associated with worsening level of consciousness. The FOUR Score assesses four domains of neurological function: eye responses, motor responses, brainstem reflexes, and breathing pattern.

To assess using the FOUR Score, one would use verbal and tactile stimulation to assess:

**Eye Response**
- 4 – Eyelids open or opened, tracking or blinking to command.
- 3 – Eyelids open but not tracking.
- 2 – Eyelids closed but opens to loud voice, but is not tracking.
- 1 – Eyelids closed but opens to pain, but is not tracking.
- 0 – Eyelids remain closed with pain.

**Motor Response**
- 4 – Thumbs up, fist, or peace sign to command.
- 3 – Localizing to pain.
- 2 – Flexion response to pain.
- 1 – Extensor posturing.
- 0 – No response to pain or generalized myoclonus, status epilepticus.

**Brain Stem Reflexes**
- 4 – Pupil and corneal reflexes present.
- 3 – One pupil wide and fixed.
- 2 – Pupil or corneal reflexes absent.
- 1 – Pupil and corneal reflexes absent.
- 0 – Absent pupil, corneal, and cough reflex.

**Respirations**
- 4 – Not intubated, regular breathing pattern.
- 3 – Not intubated, Cheyne-Stokes breathing pattern.
- 2 – Not intubated, irregular breathing pattern.
- 1 – Breaths above ventilator rate.
- 0 – Breaths at ventilator rate or apnea.

The final score is expressed as a group, rather than a total, for example (E3, M2, B3, R4) with lower scores representing a lower level of consciousness and a poorer prognosis.
The National Institute of Health Stroke Scale (NIHSS) is a standardized method used by physicians and other health care professionals to measure the level of impairment caused by a stroke.

The NIH stroke scale serves several purposes, but its main use in clinical medicine is during the assessment of whether or not the degree of disability caused by a given stroke merits treatment with thrombolytic therapy. Another important use of the NIHSS is in research, where it allows for the objective comparison of efficacy across different stroke treatments and rehabilitation interventions.

The NIH stroke scale measures several aspects of brain function, including consciousness, vision, sensation, movement, speech, and language. A certain number of points are given for each impairment uncovered during a focused neurological examination. A maximal score of 42 represents the most severe and devastating stroke. Current guidelines as of 2008 allow strokes with scores greater than 4 points to be treated with thrombolytics.
The level of stroke severity as measured by the NIH stroke scale scoring system:

- 0 = no stroke
- 1-4 = minor stroke
- 5-15 = moderate stroke
- 15-20 = moderate/severe stroke
- 21-42 = severe stroke

In addition to these standardized tools, the care provider should frequently assess the patients’ orientation, pupillary response, movement (both gross and fine) and sensation in all four extremities as well as the cranial nerves. The following chart lists the 12 cranial nerves and how to assess them:

| Nerve            | Classification | Major functions                                                                 | Assessment                                                                 |
|------------------|----------------|---------------------------------------------------------------------------------|                                                                           |
| I Olfactory      | Sensory        | Smell                                                                           | Have patient identify a familiar scent with eyes closed (usually deferred.) |
| II Optic         | Sensory        | Vision (acuity and field of vision); pupil reactivity to light and accommodation| Have patient read from a card or newspaper, one eye at a time. Test visual fields by having patient cover one eye, focus on your nose, and identify the number of fingers you’re holding up in each of four visual quadrants. |
| III Oculomotor   | Motor          | Eyelid elevation; most EOMs (extra-ocular movements); pupil size and reactivity  | Check pupillary responses by shining a bright light on one pupil; both pupils should constrict. Do the same for the other eye. To check accommodation, move your finger toward the patient’s nose; the pupils should constrict and converge. Check EOMs by having patient look up, down, laterally, and diagonally. |
| IV Trochlear     | Motor          | EOM (turns eye downward and laterally)                                          | Have patient look down and in.                                           |
| V Trigeminal     | Both           | Chewing; facial and mouth sensation; corneal reflex (sensory)                   | Ask patient to hold the mouth open while you try to close it and to move the jaw laterally against your hand. With patient’s eyes closed, touch her face with cotton and have her identify the area touched. In comatose patients, apply drop of saline |
A thorough neuro assessment should also include an evaluation of motor function. Since you will be assessing the ability to move on command, the patient must be awake, willing to cooperate, and able to understand what you are asking him or her.

With the patient in bed, assess motor strength bilaterally: Have the patient flex and extend her arm against your hand, squeeze your fingers, lift her leg while you press down on her thigh, hold her leg straight and lift it against gravity, and flex and extend her foot against your hand. Grade each extremity using a motor scale like the one below.
+5 - full ROM, full strength

+4 - full ROM, less than normal strength

+3 - can raise extremity but not against resistance

+2 - can move extremity but not lift it

+1 - slight movement

0 - no movement

As part of the motor assessment, also check for arm pronation or drift. Have the patient hold her arms out in front of her with his or her palms facing the ceiling. If you observe pronation—a turning inward—of the palm or the arm or the arm drifts downward, it means the limb is weak.

Assess motor response in an unconscious patient by applying a noxious stimulus and observing the patient's response to it. Another approach is central stimulation, such as sternal pressure. Central stimulation produces an overall body response and is more reliable than peripheral stimulation for this purpose. The reason: In an unconscious patient, peripheral stimulation, such as nail bed pressure, can elicit a reflex response, which is not a true indicator of motor activity.

If you use central stimulation, however, do so judiciously because deep sternal pressure can easily bruise the soft tissue above the sternum. One can also squeeze the trapezius muscle, this is often preferred because it's less traumatic. Supraorbital pressure is another option for central stimulation. Do not, however, use it on patients with facial fractures or trauma.

It is important for a thorough neurologic examination to be completed (as much as possible) before the patient transfers off to a test or is sedated and intubated to establish a baseline to serve as a reference point for later exams and to help guide treatment.

Imaging Studies
In the evaluation of the acute stroke patient, imaging studies are necessary to determine the type of stroke (ischemic vs. hemorrhagic) and they are useful to assess the degree of brain injury and to identify the vascular lesion responsible. Some advanced Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) technologies are able to distinguish between brain tissue that is irreversibly infarcted and that which is potentially salvageable, thereby allowing better selection of patients who are likely to benefit from therapy.

Computed Tomography
CT is still considered the gold standard in acute stroke treatment, but recent technology
has led to more sophisticated multimodal approaches in stroke imaging with CT evaluation. CT perfusion and CT angiography provide a map of cerebral blood volume, cerebral blood flow, and mean transit time. These studies identify the ischemic core and brain regions that can guide the decision for further interventional treatment. CT scans are faster than MRI, usually taking only minutes, and will immediately show a hemorrhage. An ischemic event, however, will take longer to be detectable on CT (usually about 24 hours.) The main advantages of CT are widespread access and speed of acquisition. In the initial phase of stroke care, a non-contrast head CT is usually ordered to exclude or confirm hemorrhage; it is highly sensitive for this indication. A non-contrast head CT should be obtained as soon as the patient is medically stable.

CT angiography (CTA) is performed by administering a rapid bolus of standard IV CT contrast dye through a large bore IV line in the antecubital fossa. Certain types of scans are timed to capture the arrival of that dye in the brain. Dye can be seen in the great vessels on the CT images; these serve as data for three dimensional computer reconstructions of the circle of Willis and extracranial cerebral arteries.

**Magnetic Resonance Imaging**

MRI demonstrates evidence of ischemic injury to the brain earlier than CT for all ischemic stroke subtypes. Magnetic resonance angiography (MRA) is a useful noninvasive procedure for evaluating extracranial and intracranial vessels. Newer multimodal technologies (e.g. diffusion-weighted imaging and perfusion-weighted imaging) have further increased the sensitivity or MRI. MRI does take longer that CT, however (20 to 90 minutes) and patients have to be able to lie flat and still for the duration of the test.

**Ultrasonography**

Carotid duplex scanning is the standard ultrasound test initially used to screen for cervical internal carotid stenosis. Demonstration of stenosis > 60% is highly accurate; however, differentiation between severe (95%-99%) and 100% occlusion is not completely reliable. This noninvasive exam can be performed relatively quickly and at the patient bedside.

**Cerebral angiography**

Cerebral angiography is the best tool to accurately evaluate the surface characteristics of a stenosed artery and is considered the “gold standard” for measuring the degree of stenosis of a cervical or cephalic artery. This diagnostic procedure provides images of the blood vessels in the brain and/or head. The test is performed to find blocked or leaking blood vessels. This test can help to diagnose such conditions as the presence of a blood clot, fatty plaque that increases the patient's risk of stroke, cerebral aneurysm or other vascular malformations.

A cerebral angiogram requires that a special dye be injected into the arteries of the head or brain. Under the direction of an expert physician, this procedure is done by inserting a catheter through a blood vessel, (most often the femoral artery) all the way up to the head and/or brain. When the catheter is in the correct position the dye is then injected. At this
point the cerebral angiogram can generate the images of the blood vessels. After the procedure, the nurse will perform frequent measurement of vital signs, neurological assessment, femoral and pedal pulse checks, and groin checks. The radiologist should be notified if a groin hematoma or change in velocity of the pedal pulse develops. Post-procedure activity is restricted according to the closure device used and the patient’s status. This typically means the patient must stay on flat bedrest, keeping the leg that was accessed straight, for two to four hours.

Transesophageal and Transthoracic Echocardiography
All patients with ischemic stroke or TIA should undergo a comprehensive assessment of cardiovascular risk to identify those with the highest likelihood of morbidity and mortality due to unrecognized coronary heart disease or the presence of a cardioembolic source of stroke. Transthoracic echocardiography is excellent for identifying ventricular sources, such as a dyskinetic ventricular wall segment, whereas transesophageal echocardiography excels at identifying atrial and aortic sources, such as patent foramen ovale (PFO) or aortic arch atherosclerosis. A transesophageal echocardiogram is sensitive for detecting apical thrombi and atrial septal defects or patent foramen ovale.

Transthoracic echocardiography is the less invasive of the two procedures and is the most commonly ordered initial test to evaluate for a cardioembolic source of stroke. During a transesophageal echocardiogram, the nurse must monitor the patient closely (usually under moderate sedation) and position the patient to decrease risk of aspiration if nausea or vomiting occurs.

Treatment of Hemorrhagic Stroke
Subarachnoid hemorrhage (SAH) and intracerebral hemorrhage account for about 13% of all strokes. This following section of this course will focus on treatment of SAH, but treatment for IPH is largely similar. Most aneurysmal SAH occur between 40 and 60 years of age; however your children and the elderly can also be affected. Risk factors for SAH include cigarette smoking, hypertension, moderate to heavy alcohol use, and certain genetic risk factors.

A patient presenting with SAH should be admitted to an intensive care unit (ICU) for continuous hemodynamic monitoring. Patients are often given stool softeners, kept at bedrest or minimal activity and given mild analgesia to diminish hemodynamic fluctuations and lower the risk of rebleeding. Care providers should be wary of keeping patients on bedrest for too long however, the activity status should be assessed daily and patients should get out of bed as soon as is safe to do so to avoid complications such as pneumonia, deep vein thrombosis (DVT), pulmonary embolism, and pressure sores.

Care should also be taken when administering analgesics to patients with hemorrhagic stroke and narcotics should be avoided. Nurses and care providers need to be able to accurately assess the patients’ neurologic status at all times to monitor for deteriorations and many analgesics can cause drowsiness. This has the potential to either mask neuro changes or make it impossible for the care provider to differentiate the effects of the medication from a potential change in mental status.
Transcranial Doppler (TCD) ultrasonography measurements are taken and used as a baseline as the patient progresses through treatment; serial TCDs are used to assess for vasospasm, a common complication of SAH. DVT prophylaxis with pneumatic compression stockings is started prior to aneurysm treatment. Subcutaneous unfractionated heparin can be added for DVT prophylaxis after the aneurysm is treated.

The 2006 American Heart Association/American Stroke Association guidelines state that if patients with hemorrhagic stroke are on anticoagulation medications such as warfarin (Coumadin) or clopidogrel (plavix), these should be reversed and discontinued. Appropriate reversal agents include vitamin K, fresh frozen plasma (FFP), or unactivated prothrombin complex concentrate, which is also called factor IX complex.

SAH Grading Scales
SAH is often a devastating event. The appropriate therapy for SAH depends in part upon the severity of the hemorrhage. Level of consciousness on admission, patient age, and the amount of blood on initial head CT scan are the most important prognostic factors for SAH at presentation.

A number of grading systems are used in practice to standardize the clinical classification of patients with SAH based upon the initial neurologic examination and the appearance of blood on the initial head CT. An ideal SAH grading scale would provide the following capabilities:

- Guide management decisions that are influenced by the severity of SAH
- Provide prognosis for clinicians, patients, and family members
- Assist practitioners in their ability to compare individual patients and groups of similar patients regarding studies that examine the impact of new treatments
- Enable practitioners to detect and quantify changes in disease severity while following an individual patient

While a number of SAH grading scales have been proposed, none meets all these requirements or is universally accepted, but we will look at the most commonly used scales.

Hunt and Hess
The grading system proposed by Hunt and Hess in 1968 is one of the most widely used. The scale was intended as an index of surgical risk. The initial clinical grade correlates with the severity of hemorrhage.

- Grade 0: Unruptured aneurysm
- Grade 1: Asymptomatic or mild headache and slight nuchal rigidity (neck stiffness)
- Grade 1a: Fixed neurologic deficit without other signs of SAH
- Grade 2: Moderate to severe headache, stiff neck, no neurologic deficit other than cranial nerve palsy
- Grade 3: Drowsiness or confusion, mild focal neurologic deficit
- Grade 4: Stupor, moderate or severe hemiparesis
- Grade 5: Coma, decerebrate posturing

**World Federation of Neurological Surgeons**
The grading system of the World Federation of Neurological Surgeons (WFNS) was proposed in 1988. It is based on the GCS score and the presence of motor deficits.

- Grade 1: GCS score 15, no motor deficit
- Grade 2: GCS score 13-14, no motor deficit
- Grade 3: GCS score 13-14, with motor deficit
- Grade 4: GCS score 7-12, with or without motor deficit
- Grade 5: GCS score 3-6, with or without motor deficit

**Fisher**
The Fisher scale was devised in 1980 as an index of vasospasm risk (but not clinical outcome) based upon the hemorrhage pattern seen on the initial head CT scan.

- Group 1: No blood detected
- Group 2: Diffuse deposition or thin layer with all vertical layers of blood less than 1mm thick
- Group 3: Localized clots and/or vertical layers of blood 1mm or more in thickness
- Group 4: Intracerebral or intraventricular clots with diffuse subarachnoid blood

**Complications of Hemorrhagic Stroke**
SAH is associated with a high mortality rate. Research has found the average case fatality rate for SAH was 51%. Approximately 10% of patients with aneurysmal SAH die prior to reaching the hospital, 25% die within 24 hours of SAH onset, and about 45% die within 30 days.

A number of additional complications commonly occur in patients who have suffered a SAH including rebleeding, vasospasm and delayed cerebra ischemia, hydrocephalus, increased ICP, seizures, hyponatremia, cardiac abnormalities, and hypothalamic dysfunction and pituitary insufficiency. We will now look at each of these individually.

**Rebleeding**
Most studies have found that the risk of rebleeding is highest in the first 24 hours after SAH, particularly within six hours of the initial hemorrhage. Most rebleeding occurs within the first 72 hours. Factors that may be independent predictors of rebleeding include:

- A high Hunt-Hess grade on admission
- Large aneurysm diameter
- High initial blood pressure
Neurovascular Emergencies, 26

- A sentinel headache preceding SAH
- A longer interval from onset of SAH to admission
- Early ventriculostomy (prior to aneurysm treatment)

Rebleeding is usually diagnosed on the basis of acute deterioration of neurologic status accompanied by appearance of new hemorrhage on head CT scan. Only aneurysm treatment is effective for the prevention of rebleeding, however research has shown some benefit from immediate IV administration of the antifibrinolytic drug tranexamic acid.

**Vasospasm**
Vasospasm causes symptomatic ischemia and infarction in approximately 20 to 30 percent of patients with aneurysmal SAH; it is the leading cause of death and disability after aneurysm rupture. Vasospasm usually begins no earlier than day three after hemorrhage, reaching a peak at days seven to eight. The onset of clinical vasospasm is characterized by a decline in neuro status, including the onset of focal neurologic abnormalities. The severity of symptoms depends upon the artery affected and the degree of collateral circulation.

The calcium channel blocker nimodipine was initially used in patients with SAH to prevent vasospasm. However, despite its vasodilatory effects, there is no convincing evidence that it affects vasospasm. It has been proven to improve outcomes, however and is the standard of care in these patients.

Transcranial Doppler (TCD) sonography is useful for detecting and monitoring vasospasm in spontaneous and traumatic SAH. Velocity changes detected by TCD typically precede the clinical sequelae of vasospasm. Daily recordings offer a window of opportunity to treat patients prior to clinical decline.

**Treatment of Aneurysms**
After an aneurysmal SAH, the patient is at substantial risk of rebleeding. Rerupture of an aneurysm is associated with a mortality that is estimated to be 70%. Aneurysm repair is the only effective treatment to prevent this occurrence. Surgery has been the mainstay of therapy of intracranial aneurysms, however endovascular techniques are becoming more widely used. In the future, gene therapy combined with endovascular techniques may offer improved results for aneurysm treatment.

Surgical management of cerebral aneurysms is an effective and safe procedure with the evolution of microsurgical techniques in the hands of an experienced surgeon. Placement of a clip across the neck of this aneurysm remains the treatment of choice for most aneurysms.
Technological advances have allowed the intraluminal approach to cerebral aneurysms to emerge as a safe and often effective alternative to surgical clipping. The Gubbieli electrolytically detachable coil system was introduced in the early 1990s for the treatment of these lesions. The platinum coil is inserted into the lumen of the aneurysm. A local thrombus then forms around the coils, obliterating the aneurysmal sac. Aneurysms with broad necks, a low neck-to-fundus ratio, distal segment lesions, and a number of giant aneurysms are not amenable to endovascular therapy; surgical therapy is preferred in these circumstances.

_Cerebral Infarction_
Cerebral infarction is a frequent complication of SAH. The most common cause of infarction after SAH is assumed to be vasospasm. Hypovolemia may add to the risk of cerebral ischemia in the setting of vasospasm. Other mechanisms of ischemia include
occlusion (temporary or permanent), of or damage to cerebral arteries during aneurysm surgery, thromboembolism related to turbulent or stagnant aneurysmal blood flow or clip application, and embolism unrelated to SAH.

Hydrocephalus
Hydrocephalus (acute or chronic) is a common complication of SAH. Factors associated with increased risk of hydrocephalus include intraventricular hemorrhage, posterior circulation aneurysms, treatment with antifibrinolytic agents, and a low Glasgow score on presentation. Hydrocephalus after SAH is thought to be caused by obstruction of CSF flow by blood products or adhesions, or by a reduction of CSF absorption at the arachnoid villi.

Hydrocephalus (and increased ICP) are often treated by placing an external ventricular drain (EVD). This allows direct measurement of ICP and the ability to drain excess CSF. The need for long term CSF diversion needs to be assessed on a subacute basis after appropriate treatment of the hemorrhage and aneurysm.

Increased ICP
Patients with SAH may develop increased ICP due to a number of factors, including increased CSF outflow resistance, acute hydrocephalus, hemorrhage volume, reactive hyperemia after hemorrhage, vasoparalysis, and distal cerebral arteriolar vasodilation.

The use of IV mannitol (an osmotic diuretic) or hypertonic saline (1.5%, 3%, and up to 23.5%) is sometimes indicated in patients who have increased ICP refractory to more conservative management such as decreased stimulation, neutral head position and head of bed elevation.

Seizures
Seizures at the onset of SAH appear to be a risk factor for later seizures and a predictor of poor outcome. Patients with a poor grade SAH appear to have a higher incidence of late epilepsy. Patients may require antiepileptic medications for the short-term, or may have to continue them long-term.

Hyponatremia
Hyponatremia after SAH is common, and is likely mediated by hypothalamic injury. The water retention that leads to hyponatremia is due to increased secretion of antidiuretic hormone (ADH) which may result from either the syndrome of inappropriate ADH secretion (SIADH), or much less often, volume depletion induced by cerebral salt wasting.

Cardiac Abnormalities
Cardiac abnormalities and ECG changes are commonly seen after SAH and appear to be more common and severe in those with more severe SAH. The most frequent ECG abnormalities are ST segment depression, QT interval prolongation, deep symmetric T wave inversions, and prominent U waves. Life-threatening dysrhythmias such as torsades de pointes have also been described, as well as atrial fibrillation and atrial flutter.
Myocardial injury is most likely due to the centrally mediated release of catecholamines within the myocardium due to hypoperfusion of the posterior hypothalamus.

**Treatment of Ischemic Stroke**

The treatment of patients who have suffered an ischemic stroke involves several phases. The goals in the initial phase include:

- Identifying stroke with attention to onset time and nature of symptoms
- Insuring medical stability
- Differentiating acute ischemic stroke from hemorrhage (with CT scan)
- Determining the patient’s eligibility for thrombolytic therapy
- Quickly reversing any conditions that are contributing to the symptoms
- Uncovering the location, size, and vascular territory affected by stroke
- Establishing blood pressure parameters and monitor neurologic status frequently
- Preventing and treating complications
- Determining the etiology and mechanism of stroke
- Initiating secondary stroke prevention

The timely restoration of blood flow using thrombolytic therapy is the most effective maneuver for salvaging ischemic brain tissue that is not already infarcted. There is a narrow window during which this can be accomplished, since the benefit of thrombolysis decreases in a continuous fashion over time. An important aspect of the hyperacute phase of stroke assessment and management is the rapid determination of patients who are eligible for thrombolysis. Research has shown that IV alteplase (recombinant tissue-type plasminogen activator or tPA) improves functional outcome at three months if given within three hours of symptom onset or within three hours of when the patient was last seen normal in cases when the onset is unknown. Recent research has found that in certain cases, alteplase is still beneficial when the window is extended to 4.5 hours after stroke onset. Thrombolytic treatment must be given as soon as possible, rather than near the end of the time window, however.

Prior to tPA treatment, all patients require:

- Confirmation that treatment is commencing within the required 4.5 hour time window
- Confirmation of a persistent, measureable neurologic deficit
- Confirmation that the patient selection criteria are met
- Confirmation that the noncontrast head CT is without hemorrhage
- Confirmation that the blood pressure is within parameters
- Two IV lines, preferable large bore
- Accurate body weight determination

Informed consent should be obtained if possible. Neurologic deficits caused by acute stroke may preclude obtaining informed consent from the patient. However, alteplase is
an FDA approved therapy for acute ischemic stroke and consent is not required as an emergent therapy if surrogate consent is not possible

Because “time is brain” and proper care of stroke patients requires timely action, current guidelines recommend the following in-hospital timeline as a goal for all patients with ischemic stroke who are eligible for tPA:

- Evaluation by a physician – 10 minutes
- Stroke or neurologic expertise contacted (i.e. stroke team) – 15 minutes
- Head CT or MRI scan – 25 minutes
- Interpretation of neuroimaging scan – 45 minutes
- Start of treatment – 60 minutes

Expedited stroke protocols may have the effect of reducing treatment delays and improving patient outcomes. Current and emerging guidelines for the initial care of patients with acute ischemic stroke emphasize coordinated approaches involving rapid assessment and treatment. Specific order sets (standing orders) that address issues such as control of blood glucose, parameters to treat fever, and consultations with other multidisciplinary team members should be developed to expedite patient care. Primary stroke centers are recommended to have succinct, organized stroke care pathways. These clinical pathways improve coordination of acute stroke care and discharge planning, decrease hospital costs, decrease readmission rates, reduce length of hospital stay, and enhance usefulness of outcome measurement and quality improvement.

All patients should be admitted to an intensive care unit or dedicated stroke unit for at least 24 hours of close neurologic and cardiac monitoring. The nurse is responsible for administration of tPA. tPA is packaged as a crystalline powder and reconstituted with sterile water. After reconstitution, the preparation is 100mg total. The alteplase dose is calculated at 0.9 mg/kg of actual body weight, with a maximum dose of 90mg. Ten percent of the dose is given as an IV bolus over one minute, and the remainder infused over one hour. It is advisable to remove any excess tPA from the bottle prior to administration, in order to avoid accidental overdose if the IV pump is inaccurately calibrated.

Per AHA guidelines, important measures during the first 24 hours of tPA treatment include the following:

- Vital signs and neurologic status should be checked every 15 minutes for two hours, then every 30 minutes for six hours, then every 60 minutes until 24 hours from the start of tPA treatment.
- Blood pressure must be maintained at or below 180/105 during the first 24 hours. Patients with blood pressure above these parameters should be treated with IV labetalol or transdermal nitroglycerin paste or IV nicardipine. (If the patient’s initial blood pressure is above 185/110 and remains as such after these medications, tPA should not be administered.)
- Anticoagulant and antithrombotic agents, such as heparin, warfarin (Coumadin)
or antiplatelet drugs, should NOT be administered for at least 24 hours within the tPA infusion is completed.

- Invasive procedures such as venipuncture, catheter placement, and nasogastric tube insertion should be avoided for at least 24 hours.
- A follow-up noncontrast head CT scan should be obtained 24 hours after tPA is initiated if treatment with antithrombotic agents such as aspirin or heparin is planned.

Complications of tPA

Intracranial hemorrhage is the most severe complication of thrombolytic therapy, occurring in approximately six percent of patients. No patient characteristics can be identified at presentation that reliably predict whether a patient will or will not develop a hemorrhage as a result of treatment with tPA. However, patients with very severe strokes and those with evidence of major infarction on head CT may be at an increased risk for intracerebral hemorrhage following treatment.

Intracranial hemorrhage (ICH) should be suspected in any patient who develops a sudden neurologic deterioration, a decline in level of consciousness, new headache, nausea and vomiting, or a sudden rise in blood pressure after thrombolytic therapy is administered, especially within the first 24 hours of treatment. In patients with suspected ICH, the tPA infusion should be discontinued and a stat noncontrast head CT or MRI should be obtained. Blood should be drawn for typing and cross matching, and measurement of prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count and fibrinogen levels.

If ICH is confirmed by CT or MRI, the administration of agents to reverse the effects of thrombolytic and antiplatelet therapy should be considered including 10 units of cryoprecipitate to increase the levels of fibrinogen and factor VIII, and six to eight units of platelets. In patients receiving unfractionated heparin for any reason, one milligram of protamine for every 100 units of heparin given in the preceding four hours should be considered. The physicians will decide on further action in collaboration with other team members such as the consulting neurosurgeon. Facilities that treat patients with thrombolitics should have a hemorrhage algorithm and clinical guidelines to expedite assessment and management of a new ICH.

Another complication of IV tPA administration is systemic bleeding. This usually occurs in the form of oozing from IV catheter sites, ecchymosis (especially under automated blood pressure cuffs), and gum bleeding as well as hematuria and hemoptysis. These complications do not require cessation of treatment. More serious bleeding, such as from the gastrointestinal or genitourinary system, may or may not require discontinuation of tPA depending on the severity. Rarely, patients who suffer stroke after a recent myocardial infarction can develop bleeding into the pericardium, resulting in life-threatening tamponade. Consequently patients who become hypotensive after tPA should be evaluated with urgent echocardiography.

Other Treatment Options
Research has shown that IV alteplase treatment for acute ischemic stroke can be performed safely and effectively via telemedicine in situations where local stroke expertise is not routinely or immediately available. Guidelines published in 2009 by the American Heart Association and American Stroke Association (AHA/ASA) support the use of telemedicine in the acute stroke setting in these situations. The AHA/ASA concluded that telemedicine using high-quality videoconferencing systems in this setting is useful for patient evaluation with NIH stroke scale examination, review of brain CT scans and thrombolysis decision making. As technology advances, so do the opportunities for care of stroke patients. Some organizations are already using robots in conjunction with telemedicine to perform neurologic examinations in areas where stroke experts are not on site.

The use of intra-arterial thrombolysis as an alternative to IV tPA is becoming more widespread. Intra-arterial therapy is based on the principle of delivering thrombolytic therapy at higher concentrations directly into the thrombus. The intra-arterial approach gives stroke patients more treatment options and should be initiated by specially trained interventional radiologists. In selected cases, intra-arterial thrombolysis extends the window of intervention to six hours after the onset of ischemic stroke symptoms. The intra-arterial approach is commonly used for treating stroke due to a large thrombus in the middle cerebral artery (MCA), a life threatening vertebrobasilar stroke in the posterior circulation, and when IV tPA is contraindicated. An emergent cerebral angiogram is required to place the delivery catheter at the site of the thrombus. Combined therapy with IV thrombolysis and then intra-arterial thrombolysis is used occasionally.

The Merci Retriever was the first device approved for clot retraction in acute ischemic stroke patients who were not candidates for tPA or who failed IV therapy. The Penumbra System was the second retrieval device approved to remove blood clots in patients with acute ischemic stroke. These devices have been used in combination with IV or intraarterial therapy as well. Several other approaches to recanalization with device catheters are available. The EKOS catheter is being trialed in concurrence with intra-arterial low energy ultrasound. Further research is needed to determine its clinical efficacy.

Other investigational methods of reperfusion therapy for acute ischemic stroke include angioplasty and stenting, mechanical clot disruption, and the combined use of fibrinolytics and GP IIb/IIIa antagonists.

In cases of stroke precipitated by a carotid artery occlusion, surgical treatment may be necessary. The proximal internal carotid artery (ICA) and the carotid bifurcation are the locations most frequently affected by carotid atherosclerosis. Progression of plaque formations here may result in luminal narrowing and ulceration this process can lead to ischemic stroke from hemodynamic compromise, embolization or thrombosis. Treatment options for occlusive disease of the ICA include carotid endarterectomy, carotid angioplasty and stenting and medical management.

Carotid endarterectomy (CEA) is a surgical process whereby the surgeon makes a small incision in the neck just below the level of the jaw, the narrowed carotid artery is
The blood flow through the narrowed area may be temporarily rerouted (shunted). Rerouting of the blood flow is done by placing a tube in the vessel above and below the narrowing. Blood flows around the narrowed area during the surgery. The artery is then opened and the plaque is carefully removed, often in one piece. A vein from the leg may be grafted on the carotid artery to widen or repair the vessel. The shunt is then removed, and the artery and skin incisions are closed. Patients are usually monitored in the ICU overnight and are often discharged to home the following day.

In some cases of ischemic stroke, decompressive surgery may be indicated. This involves the removal of a large part of the cranium to allow space for the expansion of swollen brain tissue. The basis of decompressive surgery is to reduce ICP and prevent fatal brain herniation, increase perfusion pressure to the brain that is still salvageable, and preserve cerebral blood flow. Research has shown that in cases of large hemisphere infarction, decompressive surgery reduced mortality rates from 80% to 30%.

**Complications of Ischemic stroke**

Not all patients improve in the initial hours after presentation. Three main types of deterioration are seen in patients with ischemic stroke during the initial days after hospitalization. The first group includes medical complications, infectious processes, cardiovascular compromise, and metabolic abnormalities, which affect the patient systematically and may exacerbate ischemia. These complications usually do not develop on the day of admission. The second group includes brain edema, in the setting of large hemispheric strokes, intracerebral hemorrhage, and seizures. These complications are also usually not present on admission and headache and altered consciousness are the key features. The third type of deterioration is a gradual increase in focal deficits while the patient remains alert and free of medical complications. This type of progression of deficits is often associated with lacunar infarcts.

Medical and nursing management both focus on the prevention of subacute complications of stroke, including malnutrition, aspiration, pneumonia, UTI, bowel or bladder dysfunction, DVT, pulmonary embolism, contractures, joint abnormalities, and skin breakdown. Depression is also common after stroke. Even in specialized stroke units, up to 63% of patients experience one or more complications after acute stroke. Next, we will discuss some of the most common complications.

*Hyperglycemia*

Hyperglycemia in critically ill patients has long been associated with complications.
Infarct expansion, hemorrhagic conversion, and poor clinical outcomes have been reported in the acute ischemic stroke population. Even the benefit of thrombolytics may be reduced. Increased blood glucose provides additional substrate for anaerobic metabolism, which promotes lactic acidosis and free radical production. Elevated serum glucose is common in the acute phase of stroke and may be related to uncontrolled or undetected diabetes mellitus or stress-induced hyperglycemia associated with cortisol and norepinephrine release at the time of insult. In one study, elevated glucose was present in two thirds of acute ischemic stroke patients. Treatment with insulin confers a protective effect in critically ill patients. The American Stroke Association ASA 2007 “Guidelines for the Early Management of Adults With Ischemic Stroke” recommend the use of rapid-acting insulin for a blood glucose level >140 mg/dL.

The nurse should monitor the blood glucose level based on the patient’s glucose level at admission. If blood glucose is >140 mg/dL and the patient has received thrombolytic therapy, it may be prudent to monitor glucose every one to two hours, because there is evidence that these patients are more prone to ICH. Treatment for hyperglycemia may be instituted, depending on individual hospital insulin or oral hypoglycemic treatment protocols. In patients who have not received thrombolysis, glucose may be monitored every six hours in the first 24 to 48 hours and continued if the patient is known to have diabetes. It is also important to evaluate the need for diabetic education whether or not the patient is a known or newly diagnosed diabetic.

**Cerebral Edema**

Cerebral edema is a common complication of large multilobar infarctions. It usually peaks three to five days after acute ischemic stroke and is not a significant problem in the first 24 hours except in patients with large cerebellar infarcts or in younger stroke patients. Young people usually do not have significant cerebral atrophy, thereby allowing no room for swelling. ICP increases as a result of cerebral edema, and monitoring for increased ICP should be part of the ongoing assessment of acute ischemic stroke patients. When invasive ICP monitoring is not available, the nurse must rely on the less accurate clinical signs of increasing pressure, which can include change in level of consciousness, worsening neurological deficits, new pupillary changes, or changes in respiratory patterns. Changes in level of consciousness are an early sign of increasing ICP, whereas pupillary changes are a late sign. Hydrocephalus may also develop as a result of obstruction of the cerebral spinal fluid pathways. The treatment plan goals should be to reduce ICP, maintain cerebral perfusion pressure to prevent worsening cerebral ischemia, and prevent secondary brain injury.

Hypotonic fluids containing excess free water should be avoided in patients who have or are at risk for cerebral edema. The nurse must evaluate the patient for hypoxia, hypercarbia, or hypothermia that may lead to elevated ICP. The head of the bed should be elevated 20° to 30°, the neck should be in a neutral position to facilitate venous drainage, and the airway should be assessed for patency. As ICP increases, the patient’s blood pressure may rise to maintain adequate cerebral perfusion pressure. The use of an aggressive antihypertensive agent with venodilating effects, such as nitroprusside, should be avoided because it can cause cerebral venodilation and can lead to a more elevated
ICP. For cerebellar infarcts and hemorrhages in which hydrocephalus and a generalized increase in ICP are an issue, an ICP catheter is usually not inserted. For large hemispheric infarcts and hemorrhages, herniation rather than generalized increased ICP is the main concern, and ICP monitoring is generally not helpful. ICP treatment may include modest hyperventilation to decrease Pco2 by 5 to 10 mm Hg to produce enough vasoconstriction to temporarily lower ICP. However, hyperventilation is only a temporary measure, and brain perfusion may be compromised as vasoconstriction occurs. Frequent neurological assessments must be done to look for potential changes in brain perfusion.

On the basis of studies conducted primarily among the head injury patient population, it has become clear that some nursing care activities increase ICP transiently in some patients; however, it is not possible to identify any given activity that is uniformly detrimental to patients with increased ICP. Therefore, nurses need to evaluate each patient’s physiological response to routine care.

Osmotic diuretics such as furosemide or mannitol can be used to treat cerebral edema. Intravenous mannitol (0.25 to 0.50 g/kg) administered over 20 minutes can be given every six hours. Serum and urine osmolality should be monitored if mannitol is used. Lasix 40 mg can be used as adjunctive therapy but should not be used long-term. Barbiturates can be used for severe cerebral edema. Continuous electroencephalographic monitoring should be performed if barbiturates are administered. Hypothermia can also be used to treat elevated ICP. Unfortunately, all of these modalities are short-lived and palliative at best. The recommendations for reducing cerebral edema need further study with regard to acute ischemic stroke. Currently, there is no clinical evidence that these measures reduce cerebral edema or improve outcome in patients with ischemic brain swelling.

If hydrocephalus is present, fluid drainage through an intraventricular catheter can rapidly reduce ICP. Surgical decompression (hemicraniectomy) is the most definitive and invasive treatment of massive cerebral edema. Large cerebellar infarctions and hemorrhages that cause direct cerebellar compression of the brain stem are best treated with surgical decompression. Surgical evacuation may be done in patients with large hemispheric infarcts, but survivors typically have severe residual neurological deficits.

Seizures
Seizures are a possible complication of large cortical strokes and can be potentially life-threatening if not controlled. They can occur at the time of stroke, during the first few days after the event, or several months later. No study has specifically tested the usefulness of anticonvulsant medications in preventing or controlling seizures after stroke. Drugs proven to be of value in preventing seizures from other causes, however, are recommended for patients who have had more than one seizure after stroke. Routine prophylactic administration of anticonvulsant drugs to stroke survivors who have not had seizures should be avoided.

Infection
Pneumonia and UTI are frequently seen in the acute phase after stroke. Fever or a change
in level of consciousness should give the nurse a high index of suspicion for infection. Stroke patients frequently present to the hospital with a compromised chest radiograph or a UTI.

Pneumonia is a serious complication occurring in the first 48 to 72 hours after acute ischemic stroke and accounts for approximately 15% to 25% of deaths associated with stroke. Stroke-associated pneumonia increases length of stay, mortality, and hospital costs. The most common cause of pneumonia is aspiration due to dysphagia. Immobility and atelectasis can also lead to development of pneumonia. The patient’s airway and oxygenation must be monitored closely; some patients may require endotracheal intubation and mechanical ventilation. Early mobility and good pulmonary care can help prevent pneumonia. Preventive measures in intubated patients include ventilation in a semirecumbent position, positioning of the airway, suctioning, early mobility, and shortened use of intubation, if feasible. Nursing management includes prompt recognition and reporting of fever; the source of the fever should be sought and treatment begun immediately. Early management of nausea and vomiting can help prevent aspiration pneumonia; use of antiemetic medications is warranted in this situation. Suctioning of the airway should be done carefully if increased ICP is present.

UTIs are common, occurring in approximately 15% to 60% of stroke patients, and independently predict poor outcome. The use of an indwelling catheter and changes in sphincter control increase the risk of UTI. Indwelling catheters should be avoided if possible but are often required in the acute phase of stroke. The catheter should be removed as soon as the patient is medically and neurologically stable. Intermittent catheterization may lessen the risk of infection. External catheters, incontinence pants, and intermittent catheterization are alternatives to an indwelling catheter. The patient should be assessed for UTI if there is a change in level of consciousness and no known reason for neurological deterioration. A urinalysis and urine culture should be obtained if UTI is suspected.

**Bowel and Bladder**

Constipation is the most common bowel problem. The nurse should assess the patient’s pre-hospital bowel elimination pattern, bowel sounds, and abdominal distention, if present. The patient should also be evaluated for hydration and impaction. The nurse must request medications if needed or develop a bowel program, which can integrate the use of stool softeners, laxatives, and enemas to prevent constipation early after stroke.

The most common urinary complication is incontinence, which occurs 30% to 60% of the time in the early recovery period. An infarct in the frontal lobe or the pons can lead to incontinence. Voiding problems include neurogenic bladder; hyperreflexia with urge incontinence, urgency, and frequency; and urinary retention with or without overflow incontinence. After the indwelling catheter is removed, intermittent catheterization may be necessary to retrain the bladder. Intermittent catheterization should occur every four to six hours to prevent filling of the bladder beyond 500 mL and to stimulate normal physiological filling and emptying. Intermittent catheterization is recommended if post-void residual urine volume is >100 mL.
Urinary incontinence can increase the incidence of dermatitis, skin breakdown, UTIs, and perineal thrush. Urinary incontinence also interferes with rehabilitation and is a major factor in patients being discharged to nursing homes. Voiding strategies should be incorporated into the daily plan of care. The nurse must initiate a bladder-training program to decrease the number of incontinent episodes. The patient should be offered a commode, bedpan, or urinal every two hours during waking hours and every four hours at night. Neurological deficits may complicate the task of going to the bathroom. High fluid intake during the day and decreased fluid intake in the evening should be encouraged.

**Mobility and the Musculoskeletal System**

Stroke patients may be initially kept on bed rest but should be mobilized when they are hemodynamically stable. Early mobilization reduces risk of atelectasis, pneumonia, DVT, and pulmonary embolism. Complications from immobility account for up to 51% of deaths in the first 30 days after ischemic stroke. Immobility can also lead to contractures, orthopedic complications, atrophy, and nerve pressure palsies. The nurse should monitor the first transfer from bed to an upright position, because some patients may have neurological worsening during movement. Joints on the paralyzed side must be positioned higher than joints proximal to it. The nurse must assess for deformities that may be found on the affected side (e.g., shoulder adduction). Subluxation of the affected shoulder is common, and special care should be taken to avoid pulling on the affected arm and shoulder when repositioning or moving the patient. Nursing interventions, including range-of-motion and positioning techniques, can prevent joint contractures and atrophy.

**Pulmonary Embolism and DVT**

Pulmonary embolism occurs more commonly than is suspected clinically and accounts for a substantial number of deaths after acute ischemic stroke. Stroke patients are at risk for developing DVT, especially as a result of paralysis or impaired mobility. Prevention of DVT is one of the core performance measures for primary stroke center certification, and initiation of prevention has become a quality indicator in several populations. Safe ambulation should be started as soon after stroke as possible. Pneumatic compression devices and compression stockings can be used to prevent pulmonary embolism. Until recently, anticoagulants such as low-molecular-weight heparin and unfractionated heparin were also used. The results of the Prevention of VTE after Acute Ischemic stroke with LMWH enoxaparin (PREVAIL) Trial showed that a 40mg injection of enoxaparin once daily was more effective than 5000 IU of unfractionated heparin twice a day for prevention of DVT in acute ischemic stroke patients. Stroke patients taking anticoagulants should be assessed for bleeding daily.

**Falls**

Falls are a common cause of injury in stroke patients, with hip fractures the most prevalent injury. Hip fractures in the first seven days after stroke are associated with a poor prognosis and have been recognized as a consequence of hemiplegia since the 1950s. Most fractures occur on the paretic side. Patients with right hemispheric infarcts that cause neglect or inattention have the highest fall risk.
Minimization of fall risk is a global responsibility. Nurses must implement fall-prevention programs and educate other staff and family members about risks and fall precautions. These may include identifying patients at risk, use of alarm systems, use of special equipment (e.g., enclosure beds), and placing call buttons and the patient’s belongings near the patient to prevent the patient from reaching for something and risking a fall. Voiding times should be scheduled to prevent falls that occur when a patient tries to go to the bathroom. In some cases, it may be necessary to have a sitter stay with the patient to ensure the patient’s safety.

**Skin Care**

Stroke patients are at risk for skin breakdown because of loss of sensation and impaired circulation, older age, decreased level of consciousness, and inability to move themselves because of paralysis. Related complications such as incontinence can accelerate skin breakdown. Major pressure areas are the heels, sacrum, and lateral malleoli. Patients should be examined for skin breakdown when repositioned and after sitting. Special care should be taken when moving patients to avoid excessive friction or pressure. Patients should not be left in one position for longer than two hours. The skin must be kept clean and dry, and special mattresses should be used where indicated. The Braden Scale is a tool commonly used to predict the risk of the development of pressure ulcers (decubitus). Nursing personnel can use this predictive model to design the care of stroke patients with immobility and those who are at risk for the development of skin breakdown.

**Dysphagia and Aspiration**

Aspiration is frequently a result of dysphagia. Research has shown that approximately half of all aspirations due to dysphagia are “silent” and go unrecognized until there is a pulmonary manifestation or complication. Order sets should include a swallow assessment before oral intake, performed by a nurse; evaluation by a speech language pathologist; and institution of NPO status with intravenous normal saline at 75 to 100 mL/h until evaluation by the speech language pathologist. Swallow assessment requires an evidence-based tool. The Massey Bedside Swallowing Screen is one such tool.

Optimally, swallow assessment is performed soon after the patient’s arrival in the ED. Until then, the patient should remain on NPO status, which means no ice chips, no oral medications, no water, and no exceptions. The nurse should assess swallowing by direct observation, looking for the presence of choking, coughing, a wet voice, a delay in initiating swallow, uncoordinated chewing or swallowing, extended time eating or drinking, pocketing of food, and loss of food from the mouth. When oral intake is authorized, the nurse should follow the speech language pathologist’s recommendations, which include improving the patient’s ability to concentrate while eating with minimal distractions. These assessments and interventions may make the difference between improved recovery and increased morbidity or even mortality.

**Nutritional Compromise**

Fifty percent of patients with severe strokes were reported to be malnourished at two to three weeks after the stroke. Malnutrition was associated with higher complications and
poorer functional outcomes. To avoid nutritional compromise, nutritional intervention should occur no later than three to four days after diagnosis of dysphagia. The Ontario Heart and Stroke Association suggests that early gastrostomy should be considered if it is anticipated that dysphagia will continue beyond six weeks; however, dysphagia resolves in at least 87% of stroke patients.

A dietitian can provide an accurate assessment of nutritional health and nutrient needs. Without adequate nutrition, there is a risk of weight loss, impaired immune system, increased weakness, increased length of stay, and mortality. Nutritional assessment should be performed on the stroke unit at the time of admission and throughout the hospital stay. The simplest but most valuable thing the nurse can do to monitor nutrition is to monitor the patient’s weight and weight change over time and monitor the patient’s dietary intake.

Stroke may compromise the patient’s ability to self-feed, which can impact self-esteem. A pleasant environment that encompasses patience and encouragement fosters hope in the stroke patient. Use of seasonings, the serving of foods at appropriate temperatures, and augmentation of food presentation may enhance appeal and compensate for the patient’s loss of taste or smell. An interdisciplinary approach to treating dysphagia will result in early detection and early intervention to minimize the impact on the patient’s life, family, and healthcare costs.

**Conclusion**
In conclusion, we have discussed TIA, hemorrhagic strokes and ischemic strokes; recommended treatments and possible complications for each as well as basic nursing cares for all neurovascular emergencies. Neurovascular emergencies can arise for a variety of reasons and from a variety of conditions, but all require skilled members of the healthcare team to act quickly and efficiently in a coordinated effort to provide the proper care in order to achieve the best outcomes possible.
References


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