Acute Ischemic Stroke Management

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**Purpose and Objectives**

The purpose of the “Acute Ischemic Stroke Management” course is to provide evidence-based literature to help prepare nurses for the challenges the acute ischemic stroke patient may present during their emergency department and hospital stays. This course will assist nurses in the early recognition of acute ischemic stroke, and in reviewing cerebral artery anatomy, stroke scales, laboratory values, radiology testing, medications and nursing interventions which are associated with the treatment of this type of patient.

*After successful completion of this course, you should be able to:*

1. Explain the global impact of strokes.
2. Differentiate between a transient ischemic attack, acute ischemic stroke, and hemorrhagic stroke.
3. Identify modifiable and non-modifiable risk factors.
4. Distinguish between the different cerebral arteries.
5. Describe the importance of the head CT scan in diagnosing an acute ischemic stroke.
6. Describe the evidence-based care for the care of the ischemic stroke patient throughout the hospital stay.
7. Identify the risks and benefits of using t-PA.
9. Discuss the future considerations for the management of acute ischemic stroke.

**Introduction**

Due to the debilitating effects of an ischemic stroke, many personal and financial resources are used. The American Stroke Association (ASA) estimated the direct and indirect cost of stroke care and disability in 2010 was 73.7 billion dollars. It may take weeks to several months to get someone to their best functional level. Even then, they may still need assistance. Therefore, it is important for nurses to have a complete understanding of strokes including risk factors, types of stroke, medical interventions, nursing interventions, monitoring, and have an understanding of the continuum of stroke care.

**Glossary of Terms**

**Aphasia:** Lack of language abilities

**Aspiration:** Entry of food or liquid into the airway below the true vocal folds

**Atrial Fibrillation:** Irregular rhythm in which the atria depolarize many times a minute, but do not fully contract

**Cardiac Output:** Amount of blood the heart pumps out of the left ventricle in one minute

**Diplopia:** Double vision

Material Protected by Copyright
Dysarthria: Difficulty in speaking, but able to swallow

Dysphagia: Difficulty or inability to swallow

Dysphonia: Difficulty in speaking, hoarseness

Last Seen Normal (LSN): Is the time when the patient was last seen normal, without neurological deficits

Ptosis: Drooping of the upper eyelid

Tinnitus: Ringing, buzzing sound in the ear

Vasculitis: Inflammation of the blood vessels

Vertigo: Dizziness or lightheadedness

Incidence of First & Recurrent Stroke
Strokes are physically and financially debilitating. According to the American Heart Association strokes have dropped from 3rd to the 5th leading cause of death in the United States (2014).

Each year approximately 795,000 people experience a stroke or suffer from a reoccurring stroke. It is estimated that 137,000 people in the United States die from a stroke each year (American Heart Association, 2014).

Gender
The lifetime risk for women is higher than men. From the ages of 55 to 75 women have a 1 in 5 chance while men have 1 in 6 chance of having a stroke (Go, Mozaffarian, Roger, Benjamin, Berry, Blaha, et al., 2014).

Incidence: Race & Age
The incidence of first-time strokes in African-Americans is two times that of Caucasians. Studies indicate that African-American are at higher risk of acute ischemic stroke (AIS) due to increased prevalence of hypertension, diabetes, obesity and sickle cell anemia.

The American Heart Association (2013) stated, after the age of 55 each decade of life double the chance of having a stroke. While approximately 15,000 people will have their first cerebral infarction before the age of 45.

Incidence: Types of Stroke
Of all the strokes combined, 87% are ischemic and 13% are hemorrhagic strokes. Of hemorrhagic strokes, 10% are an intracerebral hemorrhagic (ICH) strokes and 3% are subarachnoid hemorrhagic (SAH) strokes (Go et al., 2014).

The Brain
The brain is as fragile as it is complex. It requires at least 20% of cardiac output to function properly (Lewis et al., 2011). Interruption to blood flow will cause a neurological event. The greater amount of time the brain is without appropriate blood flow, the worse the outcome.

“Approximately 2 million neurons are lost during each minute of AIS” (Silver & Silver, 2014). The medical team needs to use their astute assessment skills to find the cause of the neurological deficit and treat it appropriately, because time is brain.
Now, let us take a look at the different types of strokes.

**Stroke: Types**
The following are different types of strokes:

1. Hemorrhagic Strokes
   - A. Epidural
   - B. Intracerebral
   - C. Subarachnoid
   - D. Subdural

2. Acute Ischemic Stroke (AIS)
   - A. Transient Ischemic Attack (TIA)

*Please note!*

Transient Ischemic Attacks and Hemorrhagic Strokes will be briefly discussed; however this presentation will focus on the acute care of an Acute Ischemic Stroke.

**Hemorrhagic Stroke: Pathophysiology**
There is an insult to vessel in the cranium which causes the blood to leak within the cranium. The extra blood in the cranium puts pressure and irritates brain tissue and structures. If left untreated, this will cause cell death and/or herniation.

**Hemorrhagic Stroke**
SAH and ICH combined make up 13% of all strokes that are hemorrhagic. It is important for the healthcare provider to be able to recognize the signs and symptoms for this kind of stroke.

**Hemorrhagic Stroke: Incidence**
A hemorrhagic stroke is defined as a bleed which occurs directly in the brain. The mortality of this type of stroke is higher than ischemic stroke. It carries with it a 30-day mortality rate of 32-52% half of these deaths occur within two days of stroke onset (Rordorf & McDonald, 2011).

**Hemorrhagic Stroke: Causes and Symptoms**
Causes of hemorrhagic stroke include, but are not limited to uncontrolled hypertension, vascular malformation,
The signs and symptoms of a hemorrhagic stroke include a very painful intense headache, nausea, and vomiting.

**Transient Ischemic Attack (TIA)**

TIAs are precursors to or a warning sign of an acute ischemic stroke. TIAs have the same symptoms as a stroke including hemiparesis, slurred speech, confusion, difficulty walking and dizziness.

**Definition:**

*National Institute of Neurological Disorders and Stroke (NINDS) define TIAs as a transient episode of neurological deficit which occur when the blood flow in the brain is briefly interrupted. Symptoms are the same as a stroke, usually last less than an hour, but can persist up to 24 hours (NINDS, 2014).*

**Transient Ischemic Attack: Causes and Diagnostic Tools**

A TIA can be caused by circulating microemboli which temporarily block blood flow to a part of the brain; or it can be caused by a temporary blockage of the carotid arteries (Lewis, Dirksen, Heitkemper, Bucher & Camera, 2011).

TIAs do not show up on a Computed Tomography Scan (CT scan) or a Magnetic Resonance Imaging (MRI) (Lewis et al., 2011).

**Significance of Transient Ischemic Attack**

Patients experiencing TIAs must acknowledge the importance of their diagnosis and know the warning signs of a stroke, as 15% of TIAs lead to AIS within three months and 12% will die within the first year (Easton et al., 2009). The greatest risk for AIS is within the first week following a TIA (Furie, Kasner, Adams, Albers, Bush, et al., 2011).

**Stroke Risk After TIA:**

Risk factors that increase the risk of having a AIS after a TIA are: greater than 60 years of age, diabetes mellitus, speech and motor difficulties during the TIA, weakness during the TIA and duration of the TIA lasting longer than 10 minutes (Silver & Silver, R. 2014).

**Ischemic Stroke**

Ischemic strokes are caused by inadequate tissue perfusion following a partial or complete blockage of the artery (Lewis et al., 2011).
Ischemic Stroke: Pathophysiology

When the blood supply to brain in interrupted, the occluded vessel results in ischemia and edema in the surrounding tissue. This may cause a worsening of their symptoms.

During a stroke the brain cells and tissues in the center of the infarction die immediately. The cells which surround the center can be saved if perfusion to that part of the brain is restored in a timely manner.

However, as the result of brain cells and tissue dying, the ischemic event causes even more damage and edema. This will continue unless the blood flow can be restored.

Pugh, Mathiesen, Meighan, Summers & Zrelak, 2009

Acute Ischemic Stroke: Risk Factors

Risk factors are divided into two categories:
1. Non-Modifiable Risk Factors
2. Modifiable Risk Factors

From health screenings to discharge instructions it is imperative for patients to acknowledge the difference between modifiable and non-modifiable risk factors.

Non-Modifiable Risk Factors

Age
Two-thirds of all strokes occur in patients greater than 65 years of age.

Sex
Women have more strokes and die from strokes than men. Additionally, women who have a natural menopause before 42 years of age have twice the risk of having an AIS compared to women who have natural menopause after 42 years of age.

Race
African Americans have a higher stroke rate than Caucasians.

Hereditary
A family history of stroke increases the risk of a stroke (Go, et al., 2014 & Lewis et al., 2011).

Modifiable Risk Factors

Hypertension
Approximately 77% of patients experiencing their first stroke had a blood pressure of greater than 140/90.

Diabetes
Diabetes increases the risk of AIS in all ages.

Dyslipidemia
There has been an association between AIS and total cholesterol but this is not found in all studies.

Obesity
Abdominal obesity increases risk of stroke.

Physical Inactivity
Many studies have shown a consistent relationship between physical activity and a reduction in the risk for a
Cigarette Smoking

Current smokers are 2 to 4 times at risk of having AIS. Additionally, second-hand/environmental smoke exposure has been identified as a risk factor.

Sleep Apnea

Studies are showing there is an increased risk of AIS with patients with sleep apnea (Go et al., 2014, Lewis et al., 2011 & Roger et al., 2011).

Definition of Atrial Fibrillation

Atrial fibrillation is comprised of many disorganized impulses originating in the atria (the top chambers of the heart). The atrial rate can be 350-600 beats per minute (Lewis et al., 2011).

If the atria do not fully contract, the blood does not move through the heart normally and becomes stagnant resulting in a high potential for clots formation.

Risk Factor:

It is not clear if atrial fibrillation (AF) can be prevented; however, it is a major contributor to acute ischemic strokes.

Atrial Fibrillation as a Risk Factor

Paroxysmal, persistent and permanent AF increases the risk for AIS 5 fold (Go et al., 2014). Atrial fibrillation is responsible for up to 15-20% of all ischemic strokes (CDC, 2010). Additionally, AF can be asymptomatic, leaving patients unaware that deadly clots are forming in their heart (Roger, et al., 2011).

Classification of Acute Ischemic Stroke

Ischemic stroke is further divided into three types of strokes:

1. Thrombotic
2. Embolic
3. Decreased Perfusion

Thrombotic Stroke: Causes

Plaque and cholesterol build-up on vessel walls are viewed by the body as an injury. As a result of this build-up, the body forms clots at the “injured” site. The combination of the plaque and the clots reduce the diameter of the cerebral vessel and ultimately decrease blood flow to the brain.

Albertson & Sharma, 2014

Thrombotic Stroke: Risk Factors

Risk factors specifically associated with thrombotic strokes are:

- Atherosclerosis
- Coagulopathies
- Increased platelets
- Vasculitis

Berhheisel, Schlaudecker & Leopold, 2011
Decreased Perfusion

The brain receives approximately 15-20% of cardiac output so it is easy to see how a disruption in blood flow, for even a short amount of time, can be catastrophic. Strokes caused by hypotension or decreased perfusion results from severe carotid stenosis, cardiogenic shock after a myocardial infarction or arrhythmia (Albertson & Sharma, 2014).

Embolic Stroke: Causes and Risk Factors

Embolic strokes are caused when a clot wedges into a narrowing of an artery or the clot diameter is larger than the artery.

The heart is the primary source of clots that travel to the brain (Bader, 2009).

Risk factors specifically associated with embolic strokes are:

- Dysrhythmias
- Myocardial infarction
- Bacterial endocarditis
- Enlarged heart
- Heart failure with an ejection fraction less than 30%
- Rheumatic mitral and/or aortic valve disease
- Prosthetic valves
- Particulate emboli such as in IV drug use

Finnie, 2014

Acute Ischemic Stroke

Signs & Symptoms

Signs and symptoms of AIS are unique, and are often subtle in nature. The patient may feel a sudden weakness or numbness in the face, arm or leg, especially on one side of the body.

Your patient may develop sudden confusion, difficulty speaking or understanding what is being spoken. They may become frustrated easily and may become tearful or emotional.

Other signs are difficulty in walking, falling and an inability to get back up. Finally, a severe headache or sudden trouble seeing in one eye or both eyes should prompt the patient to seek emergent medical attention.

Jauch et al., 2010

Mimics

In assessing the patient, it is important to rule out other pathophysiology which may mimic AIS. Some of the differential diagnoses that the healthcare provider may rule out are hemiplegic migraines, seizures (postictal stage), hypoglycemia, syncope and psychogenic disorders.

Cerebral Arteries

The four cerebral arteries which will be discussed are:

1. Middle Cerebral Artery
2. Anterior Cerebral Artery
3. Posterior Cerebral Artery
4. Vertebral-Basilar Artery

The carotid arteries which are found in the neck will also be discussed.

Cerebral Arteries: Middle Cerebral Artery
The middle cerebral artery (MCA), which branches off the carotid artery, supplies approximately 80% of brain's blood supply and is the most common occlusion site (Albertson & Sharma, 2014 & Tocco, 2011).

A patient with an MCA stroke may exhibit neurological deficits such as facial asymmetry, unilateral arm and hand weakness; difficulty speaking, and garbled speech (Tocco, 2011).

Patients with strokes caused by an occluded (MCA) are at a higher risk for intracranial pressure. Intracranial pressure (ICP) peaks about four days after the stroke (Pugh Mathiesen, Meighan, Summers & Zrelak, 2009).

**Did You Know?**
Recent studies have shown that patients who have had a stroke affecting the MCA may have greater blood flow to their brain if their head of bed is laid flat. Though before doing this, physicians need to weigh the potential neurological benefits to the low positioning against the risk of the patient aspirating (Pugh, Mathiesen, Meighan, Summers & Zrelak, 2009).

On the left side of the picture below is a scan of an MCA ischemic stroke. On the right, the color yellow represents the parts of the brain to which the MCA supplies blood.

The Anterior Cerebral Artery (ACA) supplies the color of the brain which is designated in red. The Posterior Cerebral Artery (PCA) supplies the color of the brain which is designated in green.

![Image provided with permission by Robin Smithuis.](http://www.radiologyassistant.nl/en/483910a4b6f14, n.d.)

Cerebral Arteries: Anterior Cerebral Artery
The anterior cerebral artery (ACA) supplies the anterior and medial portion of the frontal and parietal lobes.

The ACA rarely is the primary site for strokes.

If a stroke occurs here, assess for signs and symptoms of sensory loss, lower extremity weakness, behavioral abnormalities, and incontinence (Tocco, 2011).
Cerebral Arteries: Posterior Cerebral Artery
The Posterior Cerebral Artery (PCA) supplies the medial occipital lobe, inferior, and medial temporal lobes. Signs and symptoms of a PCA stroke are visual disturbances or complete loss of vision, contralateral sensory loss and an inability to recognize familiar faces (Tocco, 2011).

Image provided with permission by Robin Smithuis

Cerebral Arteries: Vertebral-Basilar
Ischemia effecting the vertebral-basilar circulation influences the function of the cerebellum, brain stem or both.

Signs and symptoms of cerebellar strokes include deficits in balance and coordination, dizziness, nausea, vomiting, headache, and slurred speech (Tocco, 2011).

Brain stem strokes are rare and the mortality rate is high. Some of the signs and symptoms correlated with this location of ischemia are hemiparesis, quadriplegia, double vision, and abnormal respirations.

These patients often require intensive care treatment and mechanical ventilation support.

Cerebral Arteries: Carotid Artery Disease
The carotid arteries found in the neck also may contribute to an acute ischemic stroke.

Carotid artery disease results from the buildup of cholesterol, atherosclerosis or plaque, in one or both of the carotid arteries causing the artery lumen to narrow resulting in reduced blood flow.

Additionally, a piece of atherosclerotic plaque may break off and migrate into the cerebral circulation causing an embolic stroke.

Image Source: http://en.wikipedia.org/wiki/Carotid
Stroke Scales
Stroke scales assist the nurse and physician to differentiate between the different types of strokes, the severity level, and provide guidance for appropriate treatment.

Stroke scales that will be discussed are the National Institute of Health Stroke Scale (NIHSS), the modified National Institute of Health Stroke Scale (mNIHSS) and the Glasgow Coma Scale.

There are different types of scales used to measure stroke deficits. The most commonly used scale is called the National Institute of Health Stroke Scale (NIHSS).

An example of the NIHSS can be found at:
http://www.nihstrokescale.org/docs/HospitalStrokeScales.pdf

Stroke Scales: NIHSS
The NIHSS is an assessment of deficits concerning level of consciousness, commands, visual fields, facial and limb weaknesses, aphasia, and dysarthria.

It is scored from 0-42; a score of 0 equals no deficits, while a score of 42 exhibits severe deficits. The guidelines for t-PA administration require the physician’s clinical judgment. The nurse or physician completing the NIHSS should be aware that even low scores if not treated appropriately could have long-term complications. For example, a patient who cannot speak may only have a NIHSS of ‘2’, if left untreated this potentially could poor outcome for this patient.

Recommendations for Using the NIHSS
It is recommended to administer the NIHSS upon admission and every 12 hours for the first 24 hours and every 24 hours until patient is discharged (Mink and Miller, 2011). However, depending on the clinical setting the patient is admitted and patient’s ongoing assessments will prompt the frequency of the NIH Stroke Scale.

It is important that the NIHSS is completed the same way each time it is administered. Additional training is necessary for those caring for stroke patients.

For more information and training on the NIHSS visit:
http://www.nihstrokescale.org

Stroke Scales: mNIHSS
The Modified National Institute of Health Stroke Scale (mNIHSS) was derived from the NIHSS. It is recommended the mNIHSS be completed every shift.

According to Meyer and Lyden (2009), the mNIHSS is an improvement over the NIHSS and leads to great accuracy in treating patients.

Stroke Scales: Glasgow Coma Scale
The Glasgow Coma Scale (GSC) assesses eye, motor and verbal responses. The lowest score is a 3, while the highest score is a 15.

The GCS is used concurrently with the NIHSS to evaluate level of consciousness (Mink & Miller, 2011).

Acute Ischemic Stroke: CT Scan
Once the patient has been assessed by the healthcare providers, the next step is to obtain a non-contrast CT scan.
scan to determine if there is a bleed and to see which part of the brain has been affected by the stroke.

A CT scan should be completed within 25 minutes and interpreted within 45 minutes of the patient’s arrival to the Emergency Department (Jauch, Cucchiara, Adeoye, Meurer, Brice, Chan et al., 2010).

When a patient comes into the Emergency Department with stroke symptoms, healthcare providers should work diligently to diagnose the patient for AIS. One of the most important tests is a CT scan of the head.

A head CT scan takes a many images from various angles. Different cerebral arteries each supply particular parts of the brain. It is important to recognize distinct signs and symptoms of a stroke to determine which artery is occluded.

**Purpose of the CT Scan**
The main purpose of the CT scan is to rule out any type of bleed in the brain. Remember: **time is brain**. Therefore, once a CT scan is completed and is negative, the next treatment options must be considered.

**The CT Scan as the Gold Standard**
Although there are several different types of scans which may provide information on the location of an infarct, the CT scan is considered the gold standard for the initial diagnosis of the patient with signs and symptoms of AIS. The CT scan takes less time than a MRI to complete, and is less expensive.

The information provided by the CT scan will highly influence the physician on what medical interventions will be a part of the patient’s plan of care.

**More Info**
The CT scan is the gold standard for identifying a stroke and differentiating between an ischemic stroke and a hemorrhagic stroke (Mink & Miller, 2011).

**Using the CT Scan to Direct Management**
If the CT scan is negative for a hemorrhagic stroke and the patient continues to exhibit signs and symptoms of an acute ischemic stroke, the medical team will evaluate if the patient qualifies for thrombolytic therapy.

**CT Scan: Follow-Up**
Typically, acute ischemic strokes which have occurred in less than 12-24 hours will not show up on a CT scan (Thompson, 2011). Therefore you would expect to see other higher resolution and angiography scans ordered 24 hours after the CT scan.

**Bottom Line:**
It should be noted that the recommendations state a MRI can be utilized to diagnose a stroke. It should be noted that not all hospitals are equipped with MRI capabilities. Therefore, a CT scan is appropriate to rule out hemorrhagic bleed or a large ischemic infarct.

**Acute Ischemic Stroke: Blood Pressure**
As healthcare providers, it is important to note that for the patient who is having an acute ischemic stroke, it is normal for the blood pressure to rise during a stroke. The blood pressure rises due to the opening of the collateral vessels trying to supply blood to the ischemic part of the brain.

This is a compensatory mechanism which helps the brain to perfuse the ischemic tissue of the brain called the penumbra.
The blood pressure will naturally decrease over the next 24-48 hours (Bader, 2009).

**Penumbra**
The penumbra is the area of reduced blood flow which centers the ischemic area. If blood flow is properly restored within three hours and ischemia stopped, there is decreased chance of neurological damage (Lewis et al., 2011).

![Penumbra Image](http://www.radiologyassistant.nl/en/483910a4b6f14, n.d.)

**Endovascular Interventions**
Select patients with MCA stroke of less than six hours who are not IV t-PA candidates can be considered for intra-arterial fibrinolysis. Mechanical thrombectomy devices remain useful with and without pharmacological fibrinolysis.

Mechanical embolectomy is a procedure where the clot is mechanically removed from the artery. A neuroradiology interventionist must be present to perform this procedure.

The total impact of patient outcomes using mechanical thrombectomy has yet to be determined. The Merci, Penubra System, Solitaire™ FR and Trevo® with the appropriate patient can be useful to restore perfusion to an occluded artery (Stetka & Lutsep, 2013).

**Swallowing Evaluation & Assessment**
After the interventions are completed and the patient is stable, the next thing to do is to evaluate the patient for swallowing. Patients with a neurologic disorder may have significantly reduced sensitivity to aspiration, as indicated by a failure to cough (Logemann, 1998).

All patients should have a swallowing assessment before oral medications or nutrition is initiated (Bernheisel et al., 2011).

**Swallowing Screens: Evidence-Based Practice**
A swallow screen should be quick, low risk, low cost, and accurately identify individuals who require further assessment. A study by Schepp, Tirshwell, Miller and Longstreth (2011) reviewed thirty-five swallowing screens and protocols. The four best screens reviewed took 2-10 minutes to complete. These tests assessed oropharyngeal functions such as speech deficits and asymmetry or weakness of the face, tongue, and palate.
and the patient's ability to swallow water or other liquids (Schepp, Tirshwell, Miller & Longstreth, 2011).

If the patient does not pass the swallowing screen, the patient remains NPO. Further consultation to a Speech Language Pathologist is required.

Swallowing Evaluations
Clinical Evaluation

A clinical evaluation is completed to provide the clinician with data for diagnosis and treatment of dysphagia.

A clinical evaluation is used to provide or expand upon the following information:

- Current medical diagnosis and patient’s medical history
- Current medical status (including nutrition and respiration)
- Patient’s oral anatomy (labial and lingual control, palatal function, and laryngeal control)
- Patient’s respiratory function and its relationship to the swallow
- Patient’s general ability to follow directions
- Patient’s reaction to oral sensory stimulation (taste, temperature, texture)
- Patient’s reactions and symptoms during attempts to swallow

(K. Griffen, 1974; Linden & Siebens, 1980 as cited by Logemann, 1998)

A Speech Language Pathologist may complete a clinical evaluation as well as an instrumental evaluation to further evaluate the swallow.

Instrumental Evaluation

Videoendoscopy, also known as flexible fiberoptic endoscopic examination of swallowing (FEES), is an indirect process to evaluating the function of the swallow. This evaluation is completed using a flexible scope that is inserted into the nose and down to the level of the soft palate. This evaluation does not allow for the observation of key moments during the swallowing process, such as the oral stage and the moment when the pharyngeal swallow is triggered, forcing the Speech Language Pathologist to infer, or indirectly, assess the swallow function. The advantages to using this instrument include the ability to assess sensory awareness, as well as provide biofeedback when completing particular swallow exercises (Logemann, 1998).

Modified Barium Swallow Study (MBSS)

A MBSS consists of capturing the entire swallow (from the oral cavity through to the esophagus) using fluoroscopic images. It is typically recorded with the option to review and store for later comparison should follow-up studies be required. This study has two purposes: to define any abnormalities in anatomy and physiology that may be causing the patient’s symptoms and identify and evaluate treatment options that may allow the patient to immediately resume eating safely and efficiently. This evaluation method not only tells the Speech Language Pathologist whether or not the patient is aspirating, but also provides reasons as to why. (Logemann, 1998).

Management and treatment of dysphagia can include multiple approaches. Treatment can include sensory stimulation, exercise programs, and/or compensatory strategies. Compensatory strategies include postural techniques, modifying volume and rate of food presentation, and changes in food consistency. In more severe cases of dysphagia, the Speech Language Pathologist may recommend that the patient remain NPO until swallowing is considered safe via instrumental evaluation. Recommendations and treatment approaches will vary depending on the location of the lesion and deficits observed (Logemann, 1998).
Acute Ischemic Stroke Management: The First 48 Hours
In the first 48 hours it is important to start addressing other factors that can impact the patient’s recovery. The following conditions and their nursing interventions will be discussed:

- Intracranial pressure
- Vital signs
- Oxygenation
- Blood pressure management
- Hypo-hyperglycemia
- Maintaining normal temperature
- Deep vein thrombosis
- Nutrition
- Genitourinary
- Risk for infections
- Immobility

The First 48 Hours: Intracranial Pressure
Due to the swelling and edema cause by the ischemia, intracranial pressure (ICP) may rise.

Signs and symptoms of ICP include headache, decreased level of consciousness, confusion, aphasia, pupillary changes, and Cushing's triad (hypertension, bradycardia and disordered breathing) (Mink & Miller, 2011).

Did You Know?
The current AHA guidelines do not recommend using corticosteroids in the treatment of acute cerebral edema in stroke patients (Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009).

The First 48 Hours: Vital Signs & Dysrhythmias
Vital signs should be taken every 1-2 hours for the first 8 hours. Vital signs may need to be taken more frequently if the patient becomes unstable. Follow your hospital’s policies and procedures.

Regular, intermittent neurological checks are an important assessment tool. Neuro checks should be completed every 15 minutes during the administration of t-PA, and are usually decreased to every 30 minutes for two hours after t-PA is completed, and the patient is stable. After two hours, neurological checks are usually decreased to hourly exams. Always check your facility’s policy and protocol for guidelines on frequency of neurological assessments.

In addition, the patient’s cardiac rhythm should be monitored for 24 hours to assess for any new dysrhythmias. Patients suffering from an acute ischemic stroke are also at high risk for a heart attack.

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

The First 48 Hours: Oxygenation
Unless otherwise contraindicated, as in conditions such as Chronic Obstructive Pulmonary Disease (COPD), oxygen saturation should be kept greater or equal to 92%. If the patient cannot keep their oxygen saturations above the prescribed amount, then the nurse needs to frequently assess their lung sounds for possible
aspiration. The physician may order a chest x-ray or increase their oxygen use.

Another intervention to consider is keeping the patient’s bed elevated 30 degrees or higher if not contraindicated. Other ways to prevent aspiration is to keep the patient on their side and keep their airway clear of secretions.

Pugh, Mathiesen, Meighan, Summers, Zrelak, 2009

The First 48 Hours: Blood Pressure Management
Blood pressure maybe managed with intravenous anti-hypertensives to maintain pressures less than 185/110.

The First 48 Hours: Hypoglycemia & Hyperglycemia
During the acute phases of an acute ischemic stroke it is important to avoid both hypo- and hyperglycemia.

Recent studies have shown that blood glucose levels greater than 200 mg/dl have contributed to poor outcomes. When caring for your patient, try to keep your patient’s blood glucose level less than 140 mg/dl.

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

The First 48 Hours: Free of Fever
Jauch et al., (2010) states a fever over 38.5 C degrees should be treated.

Higher temperatures and increasing tissue damage from the ischemia equates to increased intracranial pressure (Thompson, 2011).

The studies have shown that patients with AIS, who have a fever, have a higher rate of morbidity and mortality (Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009).

The First 48 Hours: Deep Vein Thrombosis
Deep vein thrombosis (DVT) prophylaxis is important due to the high probability stroke patients may not be as mobile as they were prior to the stroke.

Expect the physician to order sequential compression devices or heparin products when appropriate (Bernheisel, et al., 2011).

Do not forget to reposition and ambulate your patients as tolerated.

The First 48 Hours: Nutrition
A nutrition assessment should be completed as soon as the patient is stable. Due to difficulties in swallowing, other means of receiving nutrition are available. A patient can receive nutrition through tube feedings. Using tube feedings for nutrition uses the normal physiological functions such as digestion and absorption.

However, enteral feedings can cause constipation and/or diarrhea. It is important to have a registered dietician be involved in the patient’s nutritional plan.

Total Parenteral Nutrition
Total Parenteral Nutrition (TPN) or Peripheral Parenteral Nutrition (PPN) is another way to give patients nutrition. TPN and PPN are administered intravenously. Even though the patient is getting nutrition, it carries risks such as infection because it needs to be given through a central or peripheral line. Also, it is more
expensive than enteral feedings.

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

**The First 48 Hours: Pressure Ulcer Prevention**
Due to the potential for poor nutrition and mobility, pressure ulcer prevention is imperative. Your facility should already have prevention measures and protocols in place. It is very important to assess your patient’s skin condition every shift. Document your findings and consult a wound care nurse specialist as needed.

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

**The First 48 Hours: Genitourinary**
Constipation is a common bowel problem with those diagnosed with AIS. There are a lot of factors that contribute to constipation such as:

- Immobility
- Not receiving enough IV or PO fluids
- Low fiber diet
- Stress
- Narcotics received for their pain

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

**Genitourinary: Nursing Interventions**
Nursing interventions should include asking the patient what kind of bowel regimen and routine did they have before the stroke. If at all possible, try to replicate the routine in the hospital.

The nurse’s responsibility is to assess for constipation and communicate to the other staff what the patient’s bowel program, alerting the healthcare team to the importance of timed toileting and ensuring proper medications for constipation have been ordered.

Pugh, Mathiesen, Meighan, Summers, & Zrelak, 2009

**Bladder and Bowel Incontinence:** The stroke may have affected the part of the patient's brain that controls the signal for when the bladder and/or bladder is full and needs to empty. This is also referred to as a neurogenic bladder/bowel.

Nursing interventions for this condition is time toileting (every 2-3 hours), decreasing caffeine intake, Kegel exercises and limit fluids in the evenings.

Pugh, Mathiesen, Meighan, Summer, & Zrelak, 2009

**The First 48 Hours: Risk for Infections**
Pneumonia and urinary tract infections (UTIs) remain a great risk to those patients who have suffered an acute ischemic stroke. Due to their immobility and difficulty clearing secretions effectively, preventing pneumonia remains a challenge.

Pugh, Mathiesen, Meighan, Summer, & Zrelak, 2009

**Risk for Infection: Pneumonia**
Up to 35% of patients with a stroke die from pneumonia. Nurses and the healthcare team can assist their patients by encouraging early mobility and aggressive pulmonary care, such as cough, deep breathing and incentive spirometers.
Risk for Infection: Urinary Tract Infections
Another infection healthcare providers need to be cautious of is urinary tract infections (UTIs). The prevalence of UTIs varies in stroke patients. They are usually caused by indwelling catheters used when they are in a critical care unit or the stroke may have caused some changes to their urinary sphincter control.

UTI Prevention
An UTI can be prevented by ensuring the catheter was inserted using sterile techniques and completing perineal care as needed and removing the indwelling catheter as soon as the patient is stable and is able to use a bed pan or bedside commode.

The First 48 Hours: Immobility
Immobility can lead to a number of post-stroke complications such as pressure ulcers, pneumonia and contractures. Frequent turning and range of motion exercises can decrease complications related to immobility and should be implemented as the patient is admitted to the intensive care unit or specialized nursing unit. Occupational and physical therapy should be consulted.

Collaborative Care
The patient who is admitted to the hospital with an acute ischemic stroke will need the assistance of almost every service offered by the hospital. Nurses need to be proactive for their patient to ensure specialized therapies such as speech, occupational, and physical therapies are consulted.

Primary Stroke Center Certification
In 2003, The Joint Commission (TJC) with the assistance from the AHA/ASA launched a program called The Joint Commission’s Primary Stroke Center Certification.

As of January of 2011 there were over 800 certified primary stroke centers in 49 states (The Joint Commission, n.d).

Primary Stroke Centers
The TJC’s goal is to recognize excellence in centers/hospitals where the staff is exceptional in the comprehensive care of stroke patients.

The on-site review of a stroke center is conducted every two years and the certification process is based on evaluation of standards, clinical practice guidelines, and performance measurement activities.

(The Joint Commission, n.d)

Case Study: Mr. Y
It is a Sunday afternoon in the Emergency Department (ED) and you are working the 1100-2300 shift. You have had a steady flow of patients so far and at 1300 you tell your charge nurse you are headed to the cafeteria to get some lunch. While browsing the selections of food, you notice some people gathering around one of the tables in the dining area. You investigate the situation of chaos and find an older African-American gentleman who is markedly overweight staring blankly at you with right sided facial droop and drooling. His wife tells you he is a diabetic and thinks he is just experiencing “low blood sugar.”
Case Study: Calling a Code Stroke
You also notice a pack of cigarettes in his shirt pocket and a prescription bottle of a cholesterol lowering medication on the table.

From the astute assessment skills of his presentation, and noting the risk factors for a stroke (African American, diabetes, cigarette smoking, and treatment for high cholesterol) you proceed to call a Code Stroke. In your facility, Code Stroke has just as much importance as a Code Blue and within a matter of minutes; Mr. Y is in the care of your dedicated Emergency Department physicians and nurses.

Case Study: Initial Management
Mr. Y is laying down with his head of bed (HOB) 30 degrees. You know this is important in decreasing intra-cranial pressure as well as lowering the risk for aspiration.

The ED physician is at the patient’s side and starts to elicit a patient history from the wife. During history taking you are placing him on the EKG monitor, securing the blood pressure cuff and, pulse-oximetry probe.

Case Study: Patient History & Medications

<table>
<thead>
<tr>
<th>Patient History</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>One-pack/day cigarette smoker</td>
<td>Atenolol</td>
</tr>
<tr>
<td>Type II diabetic</td>
<td>Metformin</td>
</tr>
<tr>
<td>High cholesterol</td>
<td>Simvastatin</td>
</tr>
<tr>
<td>Heart attack one year ago</td>
<td>Baby aspirin</td>
</tr>
</tbody>
</table>

Case Study: Neurological Assessment
The patient is alert, his pupils are equal and reactive to light, he cannot perform any task you ask of him, he exhibits moderate sensory loss, has complete right sided facial paralysis, and his right arm and leg do not move.

Case Study: Physical Assessment Data
The cardiac monitor shows a regular rhythm. His BP is 170/80, SpO2 on room air is 93% and a bedside glucose reveals to be 185.

Case Study: Labs
You realize the importance of getting this patient to the CT scan as soon as possible. You are able to draw the appropriate vials of blood before they are ready for your patient in CT.

<table>
<thead>
<tr>
<th>Patient’s Lab Orders</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC – Complete Blood Count</td>
</tr>
<tr>
<td>CMP – Complete Metabolic Panel</td>
</tr>
<tr>
<td>INR – International Normalized Ratio</td>
</tr>
<tr>
<td>PT/INR – Prothrombin Time</td>
</tr>
<tr>
<td>PTT – Partial Prothrombin Time</td>
</tr>
<tr>
<td>Troponin - I</td>
</tr>
</tbody>
</table>
Case Study: The NIHSS & CT Scan
Next you complete the NIHSS and it reflects significant neurological deficits. Within twenty-five minutes Mr. Y is in the CT suite.

Forty-five minutes later, the radiologist calls the results to the Emergency Department physician as well as the neurologist who was consulted. The results are a negative CT scan of the head, no active bleeding noted, and negative for any ischemic areas.

Case Study: Reviewing Labs & Providing Support
Now you know the CT scan is negative and upon reassessment of the patient, he is still experiencing significant neurological symptoms. His last blood pressure was 175/90.

Knowing this is a scary time, you give emotional support to Mr. Y and his wife. The labs have just resulted. Next, you go and review them with the physician.

### Pertinent Lab Results for Mr. Y

<table>
<thead>
<tr>
<th>Lab</th>
<th>Normal</th>
<th>Mr. Y Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>4300 – 10,800 mm³</td>
<td>10,000 mm³</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>13 – 18 g/100ml</td>
<td>15 g/100ml</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>42 – 52%</td>
<td>45%</td>
</tr>
<tr>
<td>PT/INR</td>
<td>11 – 12.5/0.7 – 1.8</td>
<td>11.5/1.0</td>
</tr>
<tr>
<td>PTT</td>
<td>25 – 38 sec</td>
<td>&lt;30 sec</td>
</tr>
<tr>
<td>Platelets</td>
<td>150,000 – 350,000 mm³</td>
<td>230,000 mm³</td>
</tr>
<tr>
<td>BUN</td>
<td>8 – 25 mg/100ml</td>
<td>22 mg/100ml</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.6 – 1.5 mg/100ml</td>
<td>1.4 mg/100ml</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5 – 5.0 mEq/L</td>
<td>4.0 mEq/L</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 – 145 mEq/L</td>
<td>140 mEq/L</td>
</tr>
<tr>
<td>Glucose (fasting)</td>
<td>70 – 110 mg/100ml</td>
<td>190 mg/100ml</td>
</tr>
<tr>
<td>Troponin-1</td>
<td>0 – 0.1 ng/ml</td>
<td>&lt;0.1 ng/ml</td>
</tr>
</tbody>
</table>

Case Study: t-PA
Since Mr. Y met all the inclusion criteria and his CT scan was negative for a bleed, the neurologist recommended IV t-PA to be ordered.

The physicians discussed their findings and recommendations with Mr. Y and his wife. Mr. and Mrs. Y wanted to proceed with the t-PA infusion. IV t-PA was ordered. A second IV line was placed and a Foley catheter was ordered and placed prior to the t-PA administration. A bolus was given followed by an infusion over an hour.

Case Study: Blood Pressure Management
Mr. Y's blood pressure consistently stayed at 170/80 as the infusion was initiated. Monitoring was completed as per protocol and patient tolerated infusion well without bleeding complications.
Case Study: Swallowing Screen
After his IV t-PA was completed, Mr. Y was transferred to the intensive care unit. There a swallow screen was completed. He did not pass the screen due to coughing when 5mL of water was given to him. An order was placed to the speech language pathologist to further evaluate his swallowing.

Case Study: Acute Rehabilitation
Mr. Y’s neurological status slowly improved and he was able follow commands appropriately. However, he could only move his arm and leg slightly and his facial drooping was still present.

By day three, many therapies had been able to work with Mr. Y and he was able to walk with a walker to his hospital room door and back to bed.

Stroke rehabilitation can start in the acute setting as soon as the patient is medically stable. Initial therapies focus on Speech Language Pathology (SLP), Occupational Therapy (OT) and Physical Therapy (PT). SLP addresses swallowing, speech and cognition. OT targets deficits in function regarding activities of daily living (ADLs), transfers, upper extremity weakness and sensory deficits. PT evaluates and treats deficits in functional mobility, core balance and strength, lower extremity weakness and sensory deficits. All three therapies interact as an interdisciplinary team and share information to develop a safe and effective plan of care.

Case Study: Acute Rehabilitation
Mr. Y was transferred to a rehabilitation facility on day five and continued to make improvements. He was able to be discharged home two weeks later.

Once the patient is medically stable they are able to be admitted to a rehabilitation program or specialized unit. The patient has to be able to tolerate a minimum of three hours of therapy five days a week. This rehabilitative approach focuses on the concept of neuroplasticity, or ‘re-mapping’ new brain pathways, with intense, repetitive and purposeful therapies.

Mr. Y’s had a good outcome because he was at a hospital when his symptoms started and he received t-PA in a timely fashion. Additionally, dedicated nursing care helped keep Mr. Y from acquiring any stroke-related complications. Finally, the acute rehabilitation facility was able to assist Mr. Y regain pre-stroke physical functions.

Future Treatments: Therapeutic Hypothermia
In the case study, Mr. Y received evidence-based care for an acute ischemic stroke. Still researchers continue to conduct studies to improve upon current treatments.

Researchers are discussing how therapeutic hypothermia (TH) is being considered one of the most promising neuroprotective therapies for an acute ischemic stroke.

The goal of TH is to decrease cell death in the penumbra.

Studies have shown TH would generate positive outcomes for the patient suffering an ischemic stroke.

Many elements need to be further researched such as:
- Optimal initiation time
- Cooling and temperature monitoring method
- Target temperature
- Duration of cooling and re-warming

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Future Treatments: Neuroprotective Agents

Research is ongoing to produce a safe neuroprotective treatment during an acute ischemic stroke. To date, clinical trials are still in progress and there are no treatments available that are FDA approved.


References


The Joint Commission www.jointcommission.org.

Tocco, S. (2011). Identify the vessel, recognize the stroke. America Nurse Today. 6(9), 8-11.

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