

Guide to the Care of the Patient with **Ischemic Stroke**

AANN Reference Series for Clinical Practice



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Preface

To meet its members' needs for educational tools, the American Association of Neuroscience Nurses (AANN) has created a series of guides to patient care called the *AANN Reference Series for Clinical Practice*. Each guide has been developed based on current literature and built upon evidence-based practice.

The purpose of this document is to provide nurses with a tool that will assist them in delivering the optimum-quality and patient-focused care for individuals suffering an ischemic, cerebral infarct. A stroke, or "brain attack," can be a devastating insult to the brain. Stroke is the leading cause of disability in the United States. Because nurses are frequently the professionals who see the full impact of the stroke and have specific skills that can alter the outcome of a patient's recovery, it is important that they have a useful resource that is easy to use and helps drive patient care.

The care of the stroke patient is currently a hot topic in the healthcare industry because of the availability of thrombolytics in the treatment of acute ischemic stroke. Prior to this treatment option, the care of the stroke patient was limited and frustrating to all involved. AANN has attempted to provide neuroscience nurses with up-to-date materials for high-volume, problem-prone diagnoses like stroke. In 1997, the association published a guideline called *Recommendations for the Nursing Management of the Hyperacute Ischemic Stroke Patient*. This guideline helped translate the latest research into an easy-to-use reference. However, due to the high profile of stroke, new medical, nursing, and rehabilitation treatments are becoming available every day. Neuroscience nurses are faced with the challenge of trying to stay on top of this research to provide optimum care for their stroke patients.

AANN strives to be an organization that leads neuroscience nurses into the future of better patient treatments, interventions, and outcomes. This guide has been created to assist nurses in their pursuit of the best care. The efforts of a few will influence many nurses who will provide better care for their patients based on this document. For those patients, families, and nurses affected, we are grateful for their efforts. Being a part of stroke care in the new millennium will truly be exciting, ever evolving, and gratifying to behold.

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Statement of the Problem

Management and treatment of cerebrovascular disease is a rapidly advancing science. New treatments and strategies for better care of the patient with stroke are appearing faster than they can be learned by healthcare providers. The literature identifies under-utilization of prevention strategies, lack of symptom recognition, and healthcare access issues as significant areas for improvement in stroke care. Multiple authors report the longer the delay between symptom onset and initiation of treatment, the higher the complication, morbidity, and mortality rates associated with stroke (Adams, 2003; Graf, Jahnke, & Zadrozny, 2003).

I. Impact of Stroke

In the United States alone there are approximately 700,000 cases of stroke each year, making stroke the third leading cause of U.S. mortality. About 500,000 of these are first attacks and 200,000 are recurrent attacks. On average, a brain attack occurs every 45 seconds, and every 3.1 minutes someone dies of a stroke. Stroke is a leading cause of serious, long-term disability in the United States. In 2003, Americans will pay about \$51 billion for stroke-related medical costs and lost productivity (American Heart Association, 2003). According to the American Stroke Association (ASA) and the National Stroke Association (NSA), stroke accounts for about half of all patients hospitalized for acute neurological disease. To reduce the number of brain attacks, a coordinated public education effort, an integrated emergency response, and a multidisciplinary treatment team are needed.

Nurses play a crucial role in communicating information about risk and disease. Targeting individuals least likely aware of their risk in the community and providing them with the opportunity to engage in risk-modifying activities and disease recognition are ways to improve stroke care.

Not all hospitals are equipped to manage patients with acute stroke rapidly. Eighty to eighty five percent of strokes are ischemic and can benefit from clot-busting drugs when they are administered within 3 hours. With the availability of rt-PA for use in acute ischemic stroke patients, there is an increased opportunity for improved patient outcomes through reduction in disability following brain attack. The National Institutes of Health NINDS publication, *The Rapid Identification and Treatment of Stroke*, has established less than 60 minutes as the goal for the time from door to treatment decision (Adams et al., 1996; Grotta, 1997). Because “time is brain,” nursing professionals must be knowledgeable about new stroke care standards to manage these patients quickly and appropriately—thus the emphasis on the terminology “brain attack” versus stroke.

II. Etiology

Stroke is a term that describes neurologic changes caused by an interruption of blood supply to a part of the brain. The two main types of cerebrovascular disease are ischemic and hemorrhagic.

A. Ischemic stroke

Ischemic stroke accounts for 80%–85% of all stroke and occurs when there is a reduction or blockage of blood supply to the brain, primarily because of occlusive disease of the blood vessel supplying that territory. Hemorrhagic vascular disease accounts for 15%–20% of strokes and occurs when a blood vessel ruptures.

Ischemic stroke is further classified into large-vessel thrombotic strokes, small-vessel thrombotic strokes, atheroembolic strokes from large arteries to distal branches, and cardioembolic strokes.

The types of ischemic stroke and the frequency of their occurrence are as follows:

- 20% atherosclerotic cerebrovascular disease—hypoperfusion, arteriogenic emboli
- 25% penetrating artery disease—lacunes
- 20% cardiogenic embolism—atrial fibrillation, valve disease, ventricular thrombi, others
- 30% cryptogenic stroke
- 5% other—prothrombic states, dissections, arteritis, drug abuse

B. Hemorrhagic stroke

Hemorrhagic stroke is mainly caused by hypertension, which leads to bleeding in the deep structures of the brain. Less commonly seen, subarachnoid hemorrhage (SAH) occurs from the rupture of saccular aneurysms that form at branching points of the intracranial arteries at the circle of Willis.

III. Supporting Data

Clinical manifestations of brain attack are as follows:

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

Clinical symptoms of a thrombotic stroke can occur in minutes, hours, or over days. This is attributed to the increasing size of the thrombus, with partial then complete occlusion of the artery. Embolic strokes occur suddenly and often without warning. Hemorrhagic stroke occurs rapidly; common presenting symptoms are severe occipital or nuchal headaches, paresthesias, paralysis, and change in level of consciousness.

IV. Pathophysiology of Stroke

Ischemia occurs when the blood supply to a part of the brain is interrupted or totally occluded. Survival depends on the length of time that the brain is deprived of oxygen and metabolites and on the amount of altered brain metabolism. Infarction occurs when the brain tissue suffers irreversible changes. The extent of infarction depends on the size and location of the occluded artery and on the adequacy of collateral circulation to the area. Occlusion produces ischemia in the brain tissue supplied by the affected artery and edema in the surrounding tissue. Ischemia alters cerebral metabolism. Cells in the center of the infarcted tissue, or the core, die almost immediately after stroke onset; this often called the site of primary neuronal injury. A zone of hypoperfused tissue exists around the core. This is referred to as the *ischemic penumbra* and is often an area that may be salvaged during acute intervention. Vessels that augment blood flow to the major circulatory vessels of the brain are called collaterals. Differences in number and size of these vessels explain the variations in severity of manifestations seen in individuals. A cascade of biochemical processes develop within minutes of the cerebral ischemic event. Release of neurotoxins such as oxygen free radicals, nitric oxide, and glutamate often occurs, prompting the development of local acidosis and membrane depolarization with influx of sodium and calcium. This influx results in cytotoxic edema and cell death, and secondary neuronal injury occurs.

Methods/Procedures/Interventions/Education

I. Assessment and Monitoring of a Patient with a Stroke

Neurological assessment of the patient with a stroke is critical to prevent re-injury of brain tissue or to preserve tissue that is viable. Neurological assessment cannot be performed by a monitor or represented by a number.

Neurological assessment begins the moment a nurse enters the room. A great deal of neurological assessment is done unconsciously by a nurse who is engaged with the patient. First, the nurse watches the patient for the least amount of stimulus that is required to arouse the patient. The patient should be spoken to before he or she is touched. Then the nurse should assess the level of consciousness, cranial nerves II through XII, and basic motor function. Depending on the setting, the neurological examination may be performed once an hour in the intensive care setting, once every 2 hours in a transitional care unit, and once every 4 hours on the ward. Each unit may have different policies. The most important point is to remember that a neurological emergency can occur rapidly, so every contact with a patient with a stroke, whether for an official examination or not, should have the patient's neurological status in mind.

The neurological examination begins with assessing the patient's level of consciousness. Is the patient alert? Does the patient track the speaker with his or her eyes? Does the patient know his or her name, where he or she is, the date, and if possible why he or she is in the hospital. The patient should not be given "yes" or "no" questions. The patient's level of alertness may vary during the day; however, lethargy is one of the first signs that the patient's neurological status is declining. If the patient is intubated, the patient still may be assessed for alertness and orientation. The patient may be given choice answers, preferably with the wrong answer first. The patient may also be asked to hold up two fingers. The patient may be asked to squeeze the examiner's hands; however, to assess for purposeful movement, the patient should be asked to then let go (especially with a good strong grip).

The most important overriding feature of neurological assessment of the cranial nerves and motor ability is symmetry. The patient should have equal abilities on both sides of the body. The first step may be to check the pupils for reactivity. The movements of the eye should be checked by having the patient follow an "H" configuration to assess that the patient is able to move the eyes side to side and up and down. A gross examination of the visual fields may be done by facing the patient straight on and having the patient look at the examiner's nose. Then the examiner uses peripheral vision and holds one or two fingers up in the lower and upper quadrants of the left and right side. The patient must look at the examiner's nose and not directly at the examiner's fingers. The patient's ability to open and close the eyes should be noted, but not necessarily

tested for unless there appears to be a problem. The nasal fold on the patient should be assessed for any facial drooping. The patient may be asked to show the teeth, or give a smile. The patient may be asked to stick out the tongue and move it back and forth. The nurse may then have the patient press the head gently against the nurse's hand to assess for sternomastoid muscle strength. The patient also may shrug the shoulders.

To test for cerebellum function if the patient is unable to walk, the examiner should have the patient first touch the nose. Then the examiner should hold a finger out about arm's length from the patient at varying places. The patient's ability to move the arm from one place to another in a controlled manner should be observed.

The motor function of the patient should then be assessed. The best assessment of motor function is walking. However, this is not always possible. If the patient is comatose, movement may be assessed first through the response to central pain. This should be done by squeezing the shoulder muscles, not by a sternal rub. The patient may withdraw to central pain. The next question is whether the patient is able to localize pain or move away from a particular area that is painful. This may be done by a gentle pinch on the upper arm. If the patient is in better condition neurologically, then the examiner should ask the patient to close the eyes and hold out the hands, as if holding up a pizza, to assess the upper extremity strength. If the patient's arm slowly drifts down, this is a sign of motor weakness and may be the first subtle sign that the patient is deteriorating. Then the patient may be asked to press the arm against the examiner's and, on a 0–5 scale, be given a rating for strength, with 5 being movement against gravity and resistance and 0 being no movement at all. The lower extremities may be assessed by having the patient press on the examiner's foot, or "press on the gas."

The sensory assessment of a patient may be assessed by taking a safety pin and asking the patient to distinguish between sharp and dull on the arms and legs. The alert patient may be asked for any numbness or tingling.

A short but thorough neurological assessment of the stroke patient by an experienced practitioner may only take 5 minutes. As mentioned before, some neurological assessment is done by simply observing the patient and any changes. Was the patient quick to make a joke and now seems apathetic? Subtle differences may mean that areas of the brain are not getting the blood flow because of developing infarction. Walking and talking in the morning and walking and talking in the afternoon is the goal of every stroke patient.

According to the American Stroke Association (ASA) Scientific Statement, "several reliable and well-validated scoring systems have been developed" (Adams et al., 2003). The tool with widespread use is the National Institutes of Health Stroke Scale (NIHSS), or NIH scale (www.ninds.nih.gov/doctors/index.htm). Some other scales frequently used are the Barthel (http://www.neuro.mcg.edu/mcgstrok/Indices/Barthel_Ind.htm) and Rankin scales (<http://www.med.yale.edu/neurol/residency/rankin.html>).

II. Interventions, Troubleshooting, and Patient Problems

A. Emergent evaluation

Time is the most crucial factor in optimally treating an individual who presents with clinical manifestations of a brain attack. The AHA and NSA have prepared consensus statements guiding the initial care of an acute stroke (Adams et al., 2003). The recommendations of care are based on recent research that newly discovered therapeutics must be instituted within the first 3 to 6 hours to have a positive effect on patient outcome.

Acute management of an acute ischemic stroke includes the following steps:

1. Monitor airway and have airway equipment available.
2. Monitor for signs of respiratory compromise and anticipate that patients may require intubation.
3. Titrate oxygen to maintain oxygen saturation greater than 90% by use of pulse oximetry.
4. Establish intravenous (IV) access.
5. Frequently monitor patient's vital signs, neurological deficits, oxygen saturation, and cardiac rhythm.
6. Position patient with head midline and head of bed elevated 30 degrees to decrease risk of aspiration and enhance venous return.
7. Perform an emergent computerized tomography (CT) scan to determine whether patient is a candidate for thrombolytics or other acute interventions.
8. Monitor blood pressure (BP) closely; generally, BP is not treated in ischemic stroke until it is greater than 220/120 mm Hg. Caution should be taken if antihypertensive medication is required; rapid lowering of BP can dramatically decrease cerebral perfusion and worsen the infarction.
9. Treat glucose higher than 150 mg/dl; elevated glucose worsens outcome.
10. Treat temperatures higher than 100°F as because increased temperature worsens outcome.
11. If rt-PA is used, the patient can have an anaphylactic reaction to this drug. There is only a 2% risk of this occurring, but it can be a scary turn of events for the patient, family, and staff. This reaction can come in the form of total airway occlusion due to lip swelling and hives.

Brain attack is a potentially life-threatening event and can have a major effect on the patient and family that will challenge their beliefs and alter the definition of self. Education and support should start in the emergency room by providing a chaplain or minister, managing patient and family privacy, and maintaining modesty, as well as providing adequate pain control for the patient. Maintenance of the family is a desired outcome.

Individuals within the community must be educated on the clinical signs of brain attack and on calling the emergency medical services (EMS) system (911 or other applicable number). EMS personnel must be educated to rapidly recognize

stroke signs and emergently evaluate airway (A), breathing (B), and circulation (C), commonly referred to as the ABCs. It is key to determine the time of onset. Onset should be determined by the last time the patient was known to be well. “Upon awakening” would reflect the time of onset to be when the patient went to bed or if seen during the night up and well. Time of awakening is *not* time of onset if symptoms are present upon awakening. The treatment generally will consist of establishing an IV access, administering supplemental oxygen, and notifying the nearest emergency facility of the expected arrival with a possible brain attack victim. If fluids are initiated, normal saline is the fluid of choice.

Once the patient arrives in the emergency department (ED), personnel must systematically perform necessary evaluations and diagnostic testing to minimize wasted time. Initially ED personnel should evaluate and stabilize the ABCs while taking a brief, comprehensive history. In the initial acute management of the stroke, the goals are to control vital signs, confirm it is a stroke, determine the etiology, prevent decompensation, prevent medical complications, and begin appropriate treatment.

Respiratory failure can occur when there is brain stem involvement or increased intracranial pressure (ICP). Respiratory rate, lung auscultation, and continuous oxygen saturation monitoring should assess the patient’s respiratory status. Supplemental oxygen of 2–4 liters should be used if the patient is unable to maintain an O₂ saturation of greater than 90%. Arterial blood gases (ABGs) are indicated if the patient is unable to maintain an O₂ saturation of 90%. Emergent intubation may be necessary before the stroke outcome is known. The family should be advised and assisted in making decisions concerning length of intubation once the outcome is more evident. Stroke patients can have small brain stem lesions that may lead to difficulty in swallowing and controlling secretions. In this particular case, the patient should be intubated to prevent the risk of aspiration, which can lead to further complications such as pneumonia and atelectasis.

The patient’s cardiac rhythm must be evaluated and treated if abnormalities are found. Cardiac arrhythmias can lead to decreased cardiac output and decreased cerebral perfusion pressure (CPP). A cardioembolic stroke should be suspected and further investigated if the patient’s rhythm is atrial fibrillation and the patient is not therapeutically anticoagulated or not on anticoagulation therapy. An electrocardiogram (ECG) should be performed in the emergency room to rule out any myocardial ischemia. Myocardial infarction (MI) is the third leading cause of death in an acute stroke patient and it is the leading cause of death 30 days following the occurrence of a transient ischemic attack (TIA). ECG changes that mimic myocardial ischemia, such as peaked T waves, are not uncommon in SAH and reflect an anterior circulation hemorrhage. Acute stroke patients should be monitored by telemetry during the first 24 hours of care to detect potentially life-threatening arrhythmias.

A brief history and physical should determine the time and mode of symptom onset, progression of symptoms, recent prior vascular events (i.e., TIA, stroke, or MI), patient's vascular risk factors (i.e., hypertension, atrial fibrillation, smoking, diabetes), recent surgeries, and current medications. A neurological examination, brief medical examination, and emergent CT scan of the brain must be done as soon as the ABCs are stabilized. The patient's symptoms, neurological examination, and medical examination should help in determining the mechanism of stroke and compromised vascular territory. A CT scan without contrast of the brain rapidly excludes hemorrhagic strokes.

B. Stroke clinical manifestations

Symptoms seen with stroke vary with the location and extent of the occlusion. The more anterior the ischemic lesion, the more likely it will produce symptoms associated with motor or speech functions, whereas with more posterior lesions, the impairment will more likely be sensory and visual-field impairments. The middle cerebral artery syndrome is the most common of all cerebral occlusions. Symptoms of the specific occlusions are described below.

1. Anterior circulation

Internal carotid artery symptoms include the following:

- paralysis of the contralateral face, arm, and leg
- sensory deficits of the contralateral face, arm, and leg
- aphasia if the dominant hemisphere is involved
- hemianopia, ipsilateral episodes of visual blurring, or temporary blindness (amaurosis fugax, blindness of one eye)
- carotid bruit.

Anterior cerebral artery symptoms include the following:

- contralateral hemiparesis or hemiplegia of the foot and leg (Footdrop is a common finding.)
- sensory loss over the toes, foot, and leg
- mental status impairment—confusion, amnesia, perseveration, personality changes such as apathy or flat affect
- abulia (inability to make decisions or perform voluntary acts).

Middle cerebral artery symptoms include the following:

- contralateral hemiparesis or hemiplegia of the face and arm (The leg is spared or has less deficit than the arm.)
- contralateral hemisensory in same area
- contralateral hemianopia (Left hemisphere has right visual field cuts and right hemisphere has left visual field cuts.)
- If in left hemisphere, more likely to have aphasia and difficulty in reading, writing, or calculating
- If in right hemisphere, more likely to have neglect of left visual spaces, extinction of left sided stimuli, and spatial disorientation.

2. Posterior circulation

Vertebral-basilar system symptoms include the following:

- hemiplegia/hemiparesis or quadriplegia/quadruparesis
- ipsilateral numbness and weakness of face
- dysarthria and dysphagia
- vertigo, nausea, and dizziness
- ataxic gait and clumsiness
- diplopia, homonymous hemianopia, nystagmus, conjugate gaze paralysis, and ophthalmoplegia
- akinetic mutism (locked-in syndrome when the basilar artery is occluded).

Posterior cerebral artery symptoms include the following:

- homonymous hemianopia, cortical blindness, lack of depth perception, peripheral visual field loss, visual hallucinations
- memory deficits
- perseveration and dyslexia
- thalamic or subthalamic involvement results in diffuse sensory loss, mild hemiparesis, and intentional tremor
- brain stem involvement, resulting in pupillary dysfunction, nystagmus, and loss of conjugate gaze.

Posterior inferior cerebellar artery (referred to as Wallenberg's syndrome) symptoms include the following:

- dysarthria, dysphagia, and dysphonia
- nystagmus
- cerebellar signs—unsteady gait, vertigo, and ataxia
- ipsilateral Horner's syndrome
- nausea and vomiting
- contralateral loss of pain and temperature, balance loss on affected side, and pain and temperature loss on the ipsilateral face.

3. Brain stem/cerebellum

Symptoms include the following:

- motor or sensory loss in all four limbs, crossed signs
- limb or gait ataxia
- dysarthria
- dysconjugate gaze, nystagmus, bilateral visual field defects.

4. Lacunar syndromes

Symptoms include the following:

- pure motor, only involves muscle strength deficits on one side
- pure sensory, involves touch, pain, vibration, or heat or cold deficits on one side

- clumsy hand dysarthria, involves garbled speech and clumsiness on one side
- ataxic hemiparesis, involves motor incoordination on one side.

Physicians must rule out other disorders that can mimic a stroke such as brain tumors; migraine headache with visual transient neurological deficits; or metabolic abnormalities, especially hyperglycemia or hypoglycemia. Isolated vertigo or dizziness are seldom TIAs or a stroke symptom but may be a result of Meniere's disease. It is important to evaluate for associated vascular disorders and perform further diagnostic testing after the emergent evaluation and treatment of the stroke has been completed.

C. Neurological and medical examination

Using the NIH stroke scale, the examiner can perform a comprehensive neurological examination; it can be used in all areas of the hospital. The NIH scale is used as a standardized measure of neurological function and stroke severity. The NIH scale is a scored, assessment tool that identifies neurological deficits that include level of consciousness (LOC), LOC questions, LOC commands, gaze abnormality, visual loss, facial weakness, motor weakness in arm and leg, limb ataxia, sensory loss, aphasia, dysarthria, and extinction or inattention. The tool provides a systematic, thorough assessment of stroke neurological deficits and can be used as a measure of patient outcomes. Studies have shown that an increase or decrease in the stroke score by 4 points indicates important changes (Goldstein, 1994). If the patient has a change in LOC, the examination should include checking pupil size and reactivity. Muscle tone testing and reflex testing, along with additional cognition testing, can be done after the CT is completed. If the patient has a decreased level of consciousness, the Glasgow Coma Scale (GCS) can be used to evaluate patients. Although the GCS score is widely used, it was developed to reflect traumatic injury and does not represent stroke well. A copy of the NIH scale can be retrieved at http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf. A copy of the GCS can be retrieved at <http://www.ssgfx.com/CP2020/medtech/glossary/glasgow.htm>.

A medical examination should include auscultation of the heart, lungs, and carotid arteries to evaluate for murmurs, crackles, and bruits. Palpation of carotid and peripheral pulses should be done to evaluate circulation. The head and neck should be inspected for signs of trauma or nuchal rigidity. Nuchal rigidity, ocular hemorrhage, coma, and papilledema maybe indicative of SAH, infections, tumors, or metabolic abnormalities. A complete physical examination can be done after the CT scan.

D. Laboratory evaluation

Immediate blood work should include complete blood cell count (CBC) with differential, platelet count, prothrombin time (PT), partial thromboplastin time (PTT), electrolytes, creatinine, blood urea nitrogen (BUN), and a blood glucose. The patient's lipid profile will need to be assessed at some point but is not necessary during the acute workup. Routine full chemistry, urinalysis, and cardiac enzymes should be done only if there is an indicated need. If the cause of the stroke cannot be established based on initial laboratory and radiographic evaluation, optional laboratory tests can be performed to screen for unusual causes of stroke.

E. Radiographic evaluation

A CT without contrast to rule out hemorrhage is immediately performed once the patient is stabilized. All subsequent therapy and medical management depend on the results of the CT scan. An ischemic stroke or edema generally does not show up on the CT scan for 12–24 hours unless the patient has had a large infarction. Intracerebral hemorrhage greater than 1 cm can immediately be identified in the parenchyma. CT scans diagnose more than 95% of SAHs, indicating an aneurysm when blood is visualized in the subarachnoid space. Repeat CT scans are rarely needed during hospitalization unless there is clinical deterioration in the patient.

A chest radiograph should be obtained in the ED or once the patient is admitted to the stroke center or neuroscience unit. A baseline chest film is necessary to rule out pneumonia because it is the second most common cause of death and to evaluate the size of the heart.

Magnetic resonance imaging (MRI) is generally not indicated for emergency diagnosis of a stroke. The MRI is time-consuming, and difficult to perform on a seriously ill patient. It is more expensive and has no advantage over CT in diagnosing the etiology of an early stroke. Many institutions obtain an MRI 24 hours after the initial stroke to “localize” the stroke. This procedure is costly and rarely contributes to the management of a stroke. MRI is indicated if pathology other than stroke is suspected or if better visualization of the posterior fossa is warranted.

Arteriography is indicated if blood is seen in the subarachnoid space on the CT scan for definitive diagnosis of an aneurysm and its anatomical location. A decision on treatment of the aneurysm can be made at that time on whether the patient is a candidate for ballooning, coil placement, or surgical clipping. Invasive testing arteriography may be performed emergently if the patient is within 3–6 hours of the onset of stroke symptoms. Arteriography is used to diagnose stenosis or acute vascular thrombotic occlusions of large and small blood vessels in the head and neck. Acute revascularization with thrombolytics can restore blood supply and minimize the size of the infarct and immediately improve the neurological deficit if a radiologist specializing in neurointerventional procedures is available. Angiography also allows for clot retrieval in

centers that can perform this procedure. If there is an underlying stenosis producing the ischemic event, a cerebral balloon angioplasty can be performed. If the patient is past the 3–6 hour window of opportunity, the arteriography may be scheduled at a more optimal time. At that point, arteriograms are performed to evaluate whether the patient has a stenosis of the carotid artery requiring surgery.

The noninvasive tests for nonhemorrhagic stroke are discussed here for completeness. Carotid duplex scanning is the standard test used to initially screen for cervical internal carotid stenosis. Differentiation between 95% and 100% occlusion is not possible, but demonstration of stenosis exceeding 60% is highly accurate. If a high-grade stenosis is demonstrated, a cerebral angiography should be done before an carotid endarterectomy is performed. Prior to an angiography, it should be determined whether the patient is a surgical candidate. The angiography is the best tool to accurately evaluate the surface characteristics of the artery. Individuals are considered at high risk for stroke if ulcerated plaques are identified.

The transesophageal echo (TEE) should be included in the workup if the source of stroke is suspected to be cardioembolic or the ECG is inconclusive. Patients with cardiac history, recent MI, and atrial fibrillations are at high risk of having an atrial thrombi. A TEE is sensitive for detecting apical thrombi as well as atrial septal defects or patent foramen ovals that may be the cause of the stroke. Before the TEE is performed, it must be determined whether the patient is a candidate for long-term anticoagulation or a surgical candidate for treatment of any cardiac lesions. The transthoracic echocardiography (TTE) is sensitive only to ventricular thrombi and generally used in stroke patient only to evaluate ventricular function.

F. Acute pharmacological management of ischemic strokes

An acute brain attack must be viewed as an evolving, dynamic process. The stroke event occurs suddenly, but the ischemic tissue resulting in an infarct evolves over a period of time. Recent research has shown there is a window of opportunity to salvage brain tissue. This new information has led researchers to develop new treatments that may halt the progression of ischemic tissue to infarcted tissue. Three new distinct strategies have emerged in an effort to acutely treat stroke: urgent revascularization to restore perfusion to ischemic tissue, efforts to protect neurons from the ischemia, and efforts to block the cascade of reperfusion injury. Prior to the recent research, pharmacological therapy focused only on using anticoagulants to prevent further thrombotic events or antiplatelet therapy to prevent the formation of thrombus.

G. Antithrombolytic therapy

Research has shown that the majority of strokes occur as a result of vascular occlusion. Angiography studies have shown that 80% of acute strokes have a vascular occlusion by a thrombus. The percentage of thrombus found 24 hours

after the acute stroke decreases to 20%. The embolic source is thought to originate as a thrombus in a proximal site. Therefore, acute revascularization restoring perfusion to the ischemic tissue should minimize or completely halt the neurological deficits.

Clinical trials have proven the beneficial use of intravenous recombinant tissue plasminogen activator (rt-PA) for acute stroke treatment. The only drug currently approved by the Federal Drug Administration (FDA) in the acute treatment of stroke is alteplase (Activase). Two clinical trials enrolling 624 patients who were given rt-PA within 90 minutes and 180 minutes of the acute onset of stroke symptoms. Compared to patients treated with a placebo, patients receiving rt-PA were at least 30% more likely to have no disability or minimal disability at 3 months. A symptomatic intracerebral hemorrhage occurred in 6.4% of the patients within 36 hours of the acute stroke. Mortality was 21% in the placebo group, compared to 17% in the rt-PA group (NINDS rt-PA Stroke Study Group, 1995).

Tissue-type plasminogen activator (t-PA) converts plasminogen into plasmin on the surface of the clot. t-PA is clot specific and circulating plasminogen is not usually altered. However, if large doses are given (greater than 100 mg), specificity is lowered and a systemic lytic state can occur. The half-life of rt-PA is 5–7 minutes, and the liver clears the drug. First, the stroke team must first identify whether the patient is a candidate for thrombolytic therapy. A thorough history must be taken from the patient and family to evaluate for any criteria that would exclude the patient from receiving t-PA.

Inclusion criteria for thrombolytic therapy are as follows:

- age 18 years or older
- CT negative for hemorrhage and early signs of infarct
- clinical diagnosis of ischemic stroke causing a measurable persistent neurological deficit beyond isolated sensory deficit
- time of symptom onset well established to be 180 minutes or less before treatment would begin
- BP < 185/110 mm Hg at time of treatment.

Exclusion criteria of thrombolytic therapy are as follows:

- evidence of intracranial hemorrhage on noncontrast head CT
- only minor or rapidly improving stroke symptoms
- high clinical suspicion of SAH even with normal CT
- active internal bleeding (e.g., gastrointestinal bleeding or urinary bleeding within last 21 days)
- known bleeding diathesis, platelet count <100 000/mm³
- patient has received heparin within 48 hours and had an elevated activated partial thromboplastin time (greater than upper limit of normal for laboratory)
- recent use of anticoagulant (e.g., warfarin sodium) and elevated prothrombin time >15 seconds or INR >1.5

- within 3 months of intracranial surgery, serious head trauma or previous stroke or within 14 days, major surgery or serious trauma, recent arterial puncture at noncompressible site, lumbar puncture with 7 days
- history of intracranial hemorrhage, arteriovenous malformation, or aneurysm
- witnessed seizure at stroke onset
- recent acute myocardial infarction
- on repeated measurements, systolic blood pressure > 185 mm Hg or DBP > 110 mm Hg at time of treatment, requiring aggressive treatment to reduce blood pressure within these limits.

Standard treatment includes two IV sites infusing NS or 0.45 NS to keep open at 30–50 cc/hr. All IV lines should be established prior to the initiation of rt-PA infusion. If there is a question whether the patient will be able to void, a Foley catheter should be inserted prior to infusion. Family and patient should be educated on the benefits and possible complications. Once it has been determined that the patient meets the criteria and the standard blood work for a stroke workup is done, systemic thrombolytic therapy is started. The drug must be given within 3 hours of the first sign of the acute stroke. Current dosage is 0.9 mg/kg with maximum dose of 90 mg/kg. Of the total mg of rt-PA, 10% is given by intravenous push, and the remaining 90% is infused over 1 hour. The nurse must assess hourly vital signs and serial neurological assessments. Any acute changes in LOC, changes in pupils, or any other aspect of the neurological assessment is an indication to obtain a CT immediately. In addition to neurological assessment, the nurse must assess for signs of internal bleeding, such as tachycardia, decrease in BP, pallor, or restlessness. The nurse must also evaluate for signs of retroperitoneal bleeding, such as subjective complaints of low back pain, numbness or tingling of the lower extremities, and muscle weakness. All body secretions should be tested for occult blood. The patient and family will likely be very anxious during this time, so constant reassurance and explanation of interventions and assessments can be very helpful in calming down the patient and family.

After the patient is treated with thrombolytic therapy, it is important to avoid any invasive procedures such as catheters, arterial punctures, nasogastric tubes, and automatic BP machines for 24 hours. Patients receiving thrombolytic therapy should be admitted to the intensive care unit and can be transferred to the stepdown unit in 24 hours if no complications occur.

1. Intensive observation (ICU)

- Frequent neurological assessment: LOC, motor and extremity function assessed every 15 minutes during infusion. Continue monitoring for additional 2 hours then every 30 minutes for 6 hours and then hourly until 24 hours after initiation of treatment. Complete NIH scale.
- Vital signs monitored in conjunction with neurological assessment avoid automatic BP cuffs.

2. Blood pressure management (maintain <185/110 mm Hg)

- Labetalol, 10–20 mg IVP over 1–2 minutes. The dose may be repeated and/or doubled every 10 minutes up to 150 mg. Alternatively, following the first bolus of labetalol, an IV infusion of 2–8 mg/min may be initiated and continued until the desired BP is reached.
- Sodium nitroprusside, IV infusion, can be used if satisfactory response is not obtained.
- Nicardipine, 5 mg/hr IV infusion as initial dose, titrating to desired effect by increasing 2.5 mg/hr every 5 minutes to maximum dose of 15 mg/hr.

3. Monitoring for bleeding complications

Major bleeding complications are as follows:

- intracerebral hemorrhage, risk highest during infusion but possible 24–36 hours after treatment
- retroperitoneal hemorrhage
- genitourinary hemorrhage
- gastrointestinal hemorrhage.

Minor bleeding complications are as follows:

- oozing from catheter insertion, venipuncture, or intramuscular sites
- gingival bleeding
- hematuria
- hemoptysis
- superficial hematoma
- ecchymosis
- purpura.

Management of intracranial hemorrhage is as follows:

- if suspected, discontinue rt-PA
- immediate CT scan
- draw blood for PT, aPTT, platelet count, fibrinogen, and type and cross
- prepare for administration of 6–8 units of cryoprecipitate containing factor VIII or 6–8 units of platelets
- alert neurosurgeon, may need to consult hematologist
- decision to treat surgically or medically.

III. Patient Problems

A. Initial treatment concerns

The initial plan for the management of an acute stroke patient is to control vital signs, prevent any deterioration of the patient, and prevent any medical complications of the stroke. Medical complications occurring in the acute stages of a stroke, those shown to worsen the patient's neurological outcome, include respiratory failure, hypertension, hyperglycemia, cerebral edema, and fever. The nurse caring for the patient must coordinate the activities of a interdisciplinary

team to provide high-quality care to the stroke patient. The use of a critical care path and physician order sets can guide the team in managing these complex patients. The critical path and physician order provide a guide to the necessary diagnostic tests to determine the cause of a stroke and initiate the appropriate therapy. The critical care path guides and coordinates the team daily in discharge planning and in preventing complications that can occur with a stroke. Issues addressed on the critical care path include patient assessment, consults needed to optimally provide care, diagnostic tests, medications, treatments, mobility and nutrition needs, bowel and bladder care, deep venous precaution, and the patient and family psychosocial and teaching needed prior to discharge.

Once the patient is admitted to the hospital unit, the vitals signs and neurological assessment should be scheduled every 1–2 hours for the first 8 hours. The patient should be monitored on cardiac telemetry for the first 24–48 hours following a stroke. Telemetry can be discontinued after 24 hours if no arrhythmias have been detected. If telemetry is unavailable, a Holter monitor can be used to evaluate for arrhythmias. Atrial fibrillation is commonly first detected at the time of the stroke. The nurse must monitor for arrhythmias and evaluate the patient during any arrhythmias. Many stroke patients have underlying cardiac problems and are at risk for an acute MI during the acute stages of a stroke. The patient may need a cardiac evaluation by a cardiologist during the acute stages of the stroke.

An oxygen saturation monitor should be used to evaluate the patient's oxygenation. If the patient's oxygen saturation is less than 90%, the patient should be placed on oxygen titrated at 2–4 liters per minute to maintain an oxygen saturation of 90%. Arterial blood gases and a chest film should be obtained if a saturation of $\geq 90\%$ cannot be obtained. In the acute stages of a stroke, patients may not be able to handle secretions and are at high risk for aspiration. Respiratory compromises due to infection or pulmonary edema can be a result of the aspiration. Nurses must frequently auscultate lungs, evaluate for signs of respiratory compromise, and evaluate for signs of dysphagia to prevent the occurrence of aspiration pneumonia. Initial interventions include elevating the head of the bed (HOB), monitoring the patient during oral intake, and obtain a formal swallowing evaluation, if any symptoms of choking are noted. However, remember that patients can silently aspirate, so choking symptoms may not easily observed.

It is important to monitor BP frequently during the acute stages of a stroke. BP is elevated in approximately 40%–80% of all patients with an acute stroke. The brain raises the CPP to enhance blood flow to the damaged tissue. The aggressive use of antihypertensives can decrease the blood flow to the viable tissue surrounding the infarction and worsen the neurological deficits. The elevated BP will generally resolve within the first 48 hours after a stroke. The current American Heart Association guidelines for the acute management of stroke recommend that antihypertensive treatment be initiated for nonthrombolytic candidates only if the systolic BP is greater than 220 mm Hg or the diastolic BP is greater than 120 mm Hg for three consecutive readings 15 minutes apart

(Adams et al., 2003). Blood pressure should be decreased gradually. BP should be lowered to 180–185/105–110 mm Hg in patients with a history of hypertension. In normotensive patients, the BP can be lowered to 160–170/95–100 mm Hg. The overaggressive use of antihypertensive agents or calcium antagonist can decrease CPP dangerously. Vasodilators should not be used because they would increase the ICP as the CPP was increased. Intravenous or oral labetalol, intravenous enalapril (Vasotec), nicardipine (Cardene), or nitroprusside (Nipride) should be used cautiously. Nurses must monitor BP more frequently during the first 24 hours because patients are at highest risk for hypertension. If the BP is elevated, the nurse must evaluate for causative factors such as a full bladder or pain. An effort to provide a quiet room may assist in decreasing the BP. The physician must be notified for antihypertensive medication if the BP remains elevated. The nurse should check BP when the patient is in the lying and sitting positions prior to standing the patient for the first time.

The monitoring of serum blood glucose levels is important in the acute stages of ischemic stroke. Hypoglycemia is rarely the cause of focal neurological deficits but should be treated with one ampule of 50% dextrose if present. Hyperglycemia is frequently seen with an acute stroke. The mechanism is poorly understood, but evidence has shown that hyperglycemia may worsen the cerebral infarct. According to the AHA guidelines, management of the hyperglycemia in stroke patients should be similar to other persons with elevated blood glucose (Adams et al., 2003). More recently, however, the recommendation is to maintain a serum glucose at a maximum level between 140 and 180 mg/dl by using insulin. Also, infusions with solution high in glucose or hypotonic solutions should be avoided.

Patients are at highest risk of increased ICP during the first week following acute stroke. Cerebral edema is more commonly a complication of occlusions of major intracranial arteries and large multilobar infarctions. Patients with increased ICP may show increased lethargy, worsening neurologic deficits, changes in respiratory patterns, and new pupillary changes. The goals in managing brain edema are to reduce ICP while maintaining CPP and to prevent brain herniation from occurring. Immediate treatment includes hyperventilation to decrease the PCO_2 by 5–10 mm Hg, which lowers the ICP by 25%–30%. Hyperventilation should be done only on a short-term basis until other treatments can be initiated. Long-term use of hyperventilation continues to be controversial. Osmotic diuretics such as furosemide or mannitol are recommended. If hydrocephalus is present, drainage of fluid through an intraventricular catheter can rapidly reduce the ICP. The two remaining treatment options for increased ICP are surgical decompression and evacuation of large infarcted area or hemicraniectomy. Large cerebellar infarctions that are compressing the brain stem are treated best with surgical decompression. An evacuation may be done in patients with large hemispheric infarcts, but patients surviving have severe residual neurological deficits. The AHA guidelines do not recommend the use of corticosteroids for treating cerebral edema (Adams et al., 2003).

Signs and symptoms of increasing intracranial pressure are as follows:

- Early signs are decreased LOC, headache, visual disturbances, changes in BP or heart rate, changes in respiratory pattern.
- Late signs are pupillary abnormalities, changes in vital signs more persistent, and changes in respiratory pattern with changes in ABGs.
- After a thorough neurologic assessment has been conducted, the nurse should notify the physician; an emergent CT scan should be performed; and airway, breathing, and circulation should be maintained.

General measures to prevent elevation of ICP include the following:

- HOB elevation should be up 30 degrees or according to physician specification.
- Good head/body alignment prevents increased intrathoracic pressure and allows for ensured venous drainage.
- Good pain control should be provided on a consistent basis.
- Activities should be clustered to prevent overtiring or overstimulating patient.
- Normothermia.

Fever following acute stroke may be due to infection or may be neurogenic. A small increase in temperature worsens the tissue damage when associated with cerebral edema. The patient should be treated with antipyretics agents, and the patient should be evaluated for pneumonia and urinary tract infection and treated accordingly. Current research is studying the use of hypothermia in acute stroke and head injury. At this time, keeping the patient normothermic is the best intervention.

Seizures are a potential complication of stroke and, if not controlled, can be potentially life threatening. Seizures can occur at the time of the acute stroke, during the first few days, or several months after the event. No study has specifically tested the usefulness of anticonvulsant medications in preventing or controlling seizures following stroke. Drugs that have been proven to be of value in preventing seizures of other causes, however, are recommended for patients who have had one or more seizures following stroke. The routine prophylactic administration of anticonvulsants to stroke survivors who have not had seizures is not recommended.

If anticonvulsant medication is required, the nurse must assess the patient's cognitive ability and readiness to learn. The nurse must educate the patient and family about the seizure condition, pharmacological management, and regular medication regimen for treatment of seizures. The patient must be informed to never adjust or take additional medications without consulting the physician. Patients may never experience a seizure or may have a seizure after being discharged from the hospital. Although patients and families should be educated, there is a risk of seizure, and it could occur several months after the stroke.

B. General supportive care of a patient with stroke

Medical and nursing management must focus on prevention of subacute complications of a stroke. Additional complications that can occur include malnutrition, aspiration, bowel or bladder dysfunction, deep vein thrombosis, pulmonary embolism, contractures, joint abnormalities, decubitus ulcers, and depression.

1. Fluid management and nutrition

The management of fluid in the acute stages should be monitored closely to avoid worsening cerebral edema. In general, patients should receive nothing by mouth until swallowing has been assessed. Any signs of aspiration should warrant a more formal evaluation by a speech and language pathologist (SLP). Patients suspected to have swallowing problems should have nothing by mouth until a more thorough evaluation can be completed. Intravenous fluids should either be normal saline (NS) or half-normal saline (0.45 NS) and infused to maintain a normal hydration state for the patient. Nurses must monitor patients for clinically observable signs of dysphagia. Patients at highest risk include those with infarctions in the brain stem, large hemispheric lesions, multiple strokes, or decreased LOC. Clinically observable signs or symptoms of dysphagia include coughing or choking on saliva or food, pocketing of food, garbled speech, facial muscle weakness, delayed or absent swallow reflex, drooling, watery eyes following any intake, or gurgling voice. As mentioned earlier, clinically observable signs of aspiration are not always evident; stroke patients can be “silent aspirators.” A SLP should perform a more thorough swallow test at the bedside to evaluate for signs of aspiration or pocketing of food. Nurses must alert the attending physician when the patient is identified as at risk for aspiration and consult the dietitian to evaluate the patient’s nutritional needs.

A dietitian should evaluate every patient’s nutritional status. Malnutrition has been proven to delay recovery and to increase hospital length of stay. Stroke-related complications such as fever of unknown origin, activity intolerance, and the inability to wean from a mechanical ventilator have occurred in patients not fed for 3 days. Videofluoroscopic swallowing evaluations or modified barium swallowing evaluations have become an important part of stroke care. The swallowing evaluation is important because patients can be “silent aspirators” and be at risk for developing pneumonia due to aspiration. These swallowing tests are performed in the radiology department with a SLP and radiologist present. The patient is asked to swallow different textures of food coated with barium and watched for any aspiration. Following this test, the radiologist and SLP can make recommendations on the type of intake the patient can take in safely, orally.

Patients who are aspirating or are at risk for aspiration with all types of food and liquids should receive nutrition through a soft feeding tube until swallowing is feasible or with a percutaneous endoscopic gastrostomy (PEG) if long-term feeding is anticipated. Patients should be fed as early as possible, in the first 24–48 hours. The dietitian can assist in determining the exact caloric needs

and the correct commercially prepared formula. The dietitian will follow the patient during the rehabilitation and adjust the caloric needs as necessary. Stroke patients rarely need hyperalimentation. Most patients can take an oral diet but may need to be taught special techniques such as head and neck positioning, specified swallowing maneuvers, and changes in consistency of food during the acute phase of the stroke. Weight should be monitored at least two to three times a week to assess for adequacy of nutrition.

A speech therapist will develop a feeding plan to decrease the chance of aspiration. Some basic principles must be remembered. First, patients should be placed in a high Fowler's position, preferably sitting in a chair, for the meal. The patient should be left in the chair for 30 minutes following the meal. Second, mouth care should be done prior to feeding because this can facilitate sensation and the production of saliva, which will facilitate swallowing. Oral care should also be done following eating to observe whether the patient is pocketing food. Food fragments retained in the patient's mouth can lead to aspiration. Third, the patient or care provider should be instructed to place the foods on the unaffected side of the mouth. Fourth, pulmonary status should be assessed following eating. Suctioning apparatus should be kept near the patient at all times for possible use, and the patient should be monitored closely during the first meal. Last, families must be educated on the feeding plan and the required special techniques to decrease the risk of aspiration. Patients should be fed small portions and allowed time to chew and swallow. The chin-tuck method helps to minimize aspiration during swallowing. Allowing a patient to drink thin fluid from a straw while laying flat in bed is one of the most dangerous feeding practices that may occur when a drowsy patient asks for water from a family member. The straws should be removed from the room and the family educated to avoid giving the patient a drink with a straw. Nurses must be conscious of whether the patient has a visual field cut because the patient may only eat the items on one side of the plate. Patients must be instructed to visually scan their meal tray and plate.

2. Bowel or bladder care

Constipation is the most common bowel problem, and frequently, very little attention is given to bowel care during the acute period. Nurses should assess the patient's premorbid bowel elimination pattern. The nurse must assess for bowel sounds, and abdominal distention and evaluate the patient's fluid intake and hydration status. It is important to determine pre-morbid bowel patterns. If a patient had bowel movement in the morning, it would be ideal to try and duplicate the previous patterns, which may include the use of medications. The patient should be evaluated every 2 days for an impaction. A bowel program can integrate the use of stool softeners, laxatives, suppositories, digital stimulation, and enemas to prevent constipation. The stool softeners should be given daily beginning in the acute phase. A laxative will be necessary if the patient has not had a bowel movement in 2 days. At the end of the second day, it would be ideal to give a laxative that requires 6–8 hours to work, and then attempt

bowel care again in the morning. An enema should be used as a last resort if the laxative, suppository, or digital stimulation is ineffective after day three. The nurse must take the responsibility to request the medications or develop a bowel program protocol or set of orders.

The most common urinary complication is incontinence during the acute phase. Many patients require an indwelling catheter during the acute phase of the stroke. The indwelling catheter should be removed as soon as the patient is medically and neurologically stable to avoid iatrogenic infections. Once the indwelling catheter is removed, intermittent catheterization may be necessary to restrain the bladder. A bladder scanner can be used to evaluate residuals and determine whether catheterization is necessary. Daily intake and output should be monitored, and the patient should be monitored for signs of urinary tract infection. Urinalysis and cultures should be obtained if a urinary tract infection is suspected. The patient should be offered a commode, bedpan, or urinal every 2 hours during waking hours and every 4 hours at night. Patients fall frequently at night when trying to ambulate to the bathroom. The patients should be taken to the bathroom regularly during the night, or patients can be encouraged to use a bedside commode at night to decrease the risk of falls. Also, if there are fluid restrictions, the nurse may encourage higher fluid intake during the day and decrease the amount of fluid intake during the evening prior to bedtime.

Bowel and bladder dysfunction can lead to decreased self-esteem and depression and interfere with the progress of rehabilitation. The nurse must be responsible for evaluating the patient's bowel and bladder routine and coordinating a retraining program to meet the needs of each patient.

3. Deep vein thrombosis

Patients are at risk for development of thrombophlebitis or deep vein thrombosis (DVT) in the weak or paralyzed lower extremity following a stroke. The DVT risk is related to both the paralysis of the leg and the immobility caused by the stroke. The most effective prevention of DVT is early mobilization of the patient. Early mobilization not only decreases the risk of DVT but also lessens the likelihood of major complications such as pneumonia and decubitus ulcers. If the patient is unable to ambulate, passive range of motion or active range of motion can be started during the first 24 hours. Patients should be positioned to minimize the occurrence of dependent edema. In the acute stages, sequential compression devices are generally placed on the legs bilaterally and have been proven to be effective in preventing DVT. Subcutaneous administration of heparin or low-molecular-weight heparins or heparinoids are frequently used if there is no contraindication in using antithrombotic drugs.

4. Mobility and musculoskeletal system

Immobility can lead to contractures, orthopedic complications, atrophy, and nerve pressure palsies. Nursing interventions, including range of motion and positioning techniques, can prevent joint contractures and atrophy. Nurses must assess for special deformities that may be found on the affected side including shoulder adduction; flexion contractures of the hand, wrist, and elbow; external rotation of the hip; and plantar flexion of the foot. Subluxation of the affected shoulder is common. Nurses should take special care to avoid pulling on the affected arm and shoulder when repositioning patients in bed or when moving patients from a lying to a sitting or standing position. Subluxation may not be preventable; however, careful positioning and movement of the affected arm may prevent the development of a painful shoulder-hand syndrome. Nurses can implement passive range of motion in the first 24 hours or instruct patients to perform active range of motion to prevent contractures and other orthopedic complication.

5. Skin care

Stroke patients are at risk for skin breakdown because of the loss of sensation and impaired circulation. Other related complications such as incontinence can accelerate the development of skin breakdown. Patients should be examined for pressure points and massaged when turned. Patients should not be left in the same position longer than 2 hours. Patient's skin must be kept clean and dry, and special mattresses should be used to prevent the development of decubiti. Nurses should take special care when repositioning, turning, or transferring patients to avoid excessive friction or excessive pressure that may lead to skin injury. If the patient will have long-term immobility issues, a progressive turn program may be initiated to increase skin tolerance. Typically, this type of program is not introduced until the rehabilitation setting.

6. Depression

Depression is common with stroke victims. It is not always a result of the patient's sadness over his or her deficits or loss of previous lifestyle, but is actually a result of a chemical and physical change in the brain. Studies have shown that patients with left frontal infarcts are 70% more likely to get depressed than those individuals experiencing similar devastating injuries (Ross & Rush, 1981). The nurse is in an excellent position to notice symptoms of depression and ask the physician to order an antidepressant. Sometimes this depression occurs after hospitalization, so the nurse should educate the patient and family to signs and symptoms of depression.

7. Neurological deficits

Neurological findings during the acute phase and throughout the course of stroke recovery will direct the patient's needs during medical management, direct the rehabilitation efforts, and guide the nursing plan of care. These deficits include altered LOC, cognitive deficits in higher functions, memory, ability to learn, motor deficits, disturbance in balance and coordination,

somatosensory deficits, disorders of vision, unilateral neglect, speech and language deficits, swallowing disorder (dysphagia), and affective disorder.

Disturbances in consciousness are a strong predictor of adverse outcomes after stroke. They are more likely when brain damage is extensive, especially if the brain stem is involved or when increased ICP is present. In these cases, positioning the patient in the lateral or semiprone position will help ensure maintenance of airway. Patients generally require pharyngeal suctioning because they are unable to clear secretions, which increases the risk of aspiration.

Disorders in higher brain functions are common after stroke. The patient's ability to acquire and retain new information can be determined by observing the patient's interaction with other persons; the patient's responses to questions on orientation, current events, and memory of stroke; and the patient's ability to perform arithmetic and simple tests of recall. Communication difficulties may be mistaken for cognitive disorders. A patient's cognitive deficits may interfere with rehabilitation efforts. Nursing interventions should include

- frequent orientation
- activities divided into short steps
- protection from injury
- repeated instructions as necessary
- realistic, attainable goals
- lack of excessive distractions in patient's environment.

Motor deficits reflect the type, location, and extent of vascular lesion. Motor deficits may be isolate or may be associated with sensory, cognitive, or speech deficits. The most common motor deficit is weakness and paralysis, but incoordination, clumsiness, involuntary movements, or abnormal posturing may also be identified. Motor deficits influence activities of daily living and rehabilitation efforts.

Infarctions of the cerebellum or vestibular system can produce disturbances in balance and coordination. If there is incoordination without motor or sensory loss, it is identified as ataxia. Assessing the patient's ability to perform finger-to-nose, heel-to-shin, or alternating movement tests can test ataxia. The patient may exhibit limb, gait, or truncal ataxia.

Somatosensory deficits can be exhibited as numbness, tingling, abnormal sensations (dysesthesia), or excessive reactions to sensory stimuli (hyperesthesia). Profound sensory loss will interfere with the rehabilitation of motor impairments.

The most common visual disorder is homonymous hemianopia. Visual disorders include visual-field defects, conjugate gaze paralysis, or diplopia. Severe visual disturbances will increase the complexity of rehabilitation.

Unilateral neglect refers to a patient's lack of awareness of a specific part of the body or to the external environment. Unilateral neglect generally occurs with right hemisphere strokes. These patients ignore sensory stimuli in the left half of the environment. Patients with severe neglect will deny the problems occurring as a result of the stroke or may not recognize their own body parts. Patients will ignore items in the left visual field when asked to describe a complex picture and ignore sensory stimuli on the left. Bedside evaluation will find

the patient's head turned toward the right, and the patient will often ignore examiners on the left side. Patients with neglect may only wash or bathe one side of the body. The patient's safety must be the immediate concern of all involved in the patient's care. Patients are at risk of falling because they deny they have motor deficits and are unable to be aware of physical structures that are within their perceptual space.

Communication disorders occur in as many as 40% of stroke patients. Aphasia is most often seen following vascular events in the language-dominant hemisphere and leads to disturbances in comprehension, speech, verbal expression, reading, and writing. Dysarthria and apraxia of speech need to be differentiated from aphasia. Dysarthria may be due to dysfunction of the larynx, pharynx, palate, tongue, lips, or mouth. Dysarthria is an impairment of speech because of slow or weak muscle coordination. Patients may also have apraxia of speech; the patient is unable to program a sequence of volitional movements despite the absence of motor deficits.

Patient/Family Education

For the stroke patient, rehabilitation begins immediately. It can happen at all ages. The risk factors may include hypertension, smoking, diabetes, coronary heart disease, and family history. With every neurological assessment, the brain is stimulated. Families can participate from the moment their loved one survives the immediate admission by speaking to the patient, learning from the nurse and physical therapist about range of motion, the speech therapist about feeding the patient correctly, and the occupational therapist on how to assist with activities of daily living. The brain may take up to 1 year to recover to the fullest ability. Headaches may last up to 6 months, but taper off after the first month. The symptoms of another stroke, such as a severe headache, sudden visual difficulties, sudden speech difficulties, inability to move one side of the body, or numbness and/or tingling on one side of the body, should be watched for. The patient should go directly to the hospital if these symptoms appear. The American Stroke Association and National Stroke Association have excellent Web sites that can be resources for the nurse, patient, and family (see page 32).

Key areas of patient education in the stroke population, their significant others, and care takers are the following:

1. What is a stroke?
2. Is my stroke ischemic or hemorrhagic?
3. Signs and symptoms of a stroke
4. What to do to prevent a future stroke?
 - (a) medications
 - (b) blood pressure management
 - (c) activity
 - (d) diet
 - (e) monitoring and follow-up
5. Common complications are as follows:
 - (a) dysphagia
 - (b) skin breakdown
 - (c) urinary and/or bowel incontinence
 - (d) behavior changes
 - (e) contractures
 - (f) seizures
 - (g) depression
6. What's next?
 - (a) rehabilitation
 - (b) recovery/prognosis

Expected Outcomes

The patient will be free of

- respiratory complications such as aspiration, pneumonia, pulmonary emboli, and anoxia
- increasing neurological deficits
- seizures
- malnutrition
- skin breakdown
- contractures
- communication deficits
- bowel and bladder incontinence, constipation, retention
- deep vein thrombosis
- depression
- injury.

The patient will

- learn compensatory swallowing techniques, remain upright during meals, and alert the nurse of any shortness of breath.
- learn to identify the signs and symptoms of stroke. The patient will also learn how to monitor BP and take the correct medications.
- learn what is a seizure, what safety precautions are necessary, how to treat seizures, what medications to use, and when to seek emergency care.
- learn appropriate dietary modifications, good dietary habits, safe swallowing practices, and the importance of exercise and monitoring for weight gain or loss.
- learn correct positioning techniques in the chair and bed and safe techniques in transferring from bed to chair and so on.
- learn how to perform passive and active range of motion, and apply any splints or braces needed.
- obtain an effective means of communication.
- learn how to maintain the bowel and bladder, including medication, diet, fluid and timing training.
- be able to identify signs and symptoms of a deep vein thrombosis and pulmonary embolism, take the appropriate preventive medication, and understand the importance of activity.
- learn the signs and symptoms of depression and how to seek assistance for treatment.
- learn safety practices related to memory, ambulation, transferring, falling, and visual impairments.

Evidence-Based Practices

- Use of rt-PA in acute stroke
- Brain perfusion
 - (1) Do not lower BP unless it is greater than 220/120 or rt-PA will be administered.
 - (2) Administer IV fluids.
 - (3) Use oral or IV labetalol to reduce blood pressure gradually.
- Maintenance of patient's blood glucose in the 120–140 mg/hl range
- Maintenance of patient's temperature less than 100°F during the first 48 hours following stroke
- Use of CT scan to evaluate the type of stroke
- Use of aspirin to prevent future strokes and coumadin (warfarin) in patients with atrial fibrillation
- Early mobilization
- Subcutaneous anticoagulation and/or compression stockings to prevent DVT with immobile patients
- Anticonvulsants for seizures (not prophylactic)
- Identification of cause of stroke and initiation of secondary prevention measures.

Innovative Practices (Helpful Tips)

- **Bladder scan:** To avoid inserting a Foley catheter or straight catheterizing a patient. Because catheter insertion could cause trauma and this should be avoided following administration of thrombolytics, catheterization should be avoided by scanning the bladder for volume.
- **Intra-arterial thrombolysis:** Patients who do not have successful results from intravenous rt-PA or are outside the 3-hour time window may benefit from intra-arterial administration of thrombolytics. This intervention is currently being studied all over the world, and most institutions have not yet implemented this practice. It requires collaboration and planning between the emergency and radiology departments and their associated physicians.
- **Hypothermia:** Some institutions are lowering body temperature to preserve brain cells and decrease disability; this is a relatively new intervention, but has promising results.
- **Neuroprotective agents:** A great deal of research is being done in this area. No one agent has demonstrated clear success, but there will be future developments in this area.
- **Secondary preventative treatments:** If the stroke patient is not already on these drugs, stroke patients should initiate lipid lowering treatment and ACE inhibitors following emergent stroke care.
- **Trials are being conducted to validate the use of thrombectomy devices** such as concentric mechanical thrombus retriever, which has a 6-hour time window versus the present 3-hour time window with thrombolytics. Also, the Angiojet device sprays saline at the clot before retrieving it.

Documentation

In the ED, documentation should include the following:

- time of onset
- symptoms
- vital signs: BP, pulse rate and rhythm, respiration, oxygen saturation, temperature, and blood glucose
- neurological assessment: NIH scale score, level of physical functioning, LOC, muscle strength
- time of lab tests and EKG
- time of radiological testing: CT scan, chest X ray
- time of thrombolytics
- swallowing evaluation.

Ongoing documentation should include the following:

- neurological assessment: level of physical functioning, cognitive level, muscle strength, and cranial nerve findings. (Some physicians prefer the nurse to describe “what they saw” versus a certain cranial nerve is not functioning.)
- vital signs: BP, pulse rate and rhythm, respirations, oxygen saturation, temperature
- input and output
- swallowing ability
- mechanism of communication
- activity level
- skin integrity
- psychosocial issues
- patient and family education
- discharge planning.

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Web sites

- <http://www.stroke-site.org/index.html>
http://www.neuro.mcg.edu/mcgstrok/Indices/Barthel_Ind.htm
<http://www.med.yale.edu/neurol/residency/rankin.html>
<http://www.ninds.nih.gov/>
<http://www.americanheart.org>
<http://www.strokeassociation.org/>
<http://209.107.44.93/NationalStroke/default.htm>



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