

# Brain Attacks and Acute Stroke Management

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## Objectives

By the end of this educational experience, the nurse will be able to:

Identify the different types of brain attacks

Employ criteria for thrombolytic treatment of brain attacks

Apply information presented to nursing care of brain attacks

## Introduction

The purpose of this educational offering is to give nurses an overview of the different types to Brain Attacks, symptoms of each and treatment. The nurse will also become familiar with the criteria for the use of thrombolytic agents and nursing care regarding thrombolytic treatment.

Stroke is a leading killer in the United States, being third as the cause of death and is ranked first as a cause of disability. Approximately 705,000 strokes occur every year, of these approximately 625,000 are ischemic strokes (Jauch, 2007). However, the rate of use of life saving thrombolytic agents is extremely low, with between 1-11% of patients in the United States receiving this treatment (Saver, 2007).

Stroke is the term that defines a sudden loss of blood supply to brain tissue and corresponding loss of neurological function which maybe or may not be, reversible. There are two classifications of stroke ischemic and hemorrhagic. TIA stands for transient ischemic attack and is a temporary interruption in blood flow to the brain that is often a warning symptom of a stroke.

**TIA**

The classic definition of TIA includes minor neurological dysfunction that lasts 24 hours or less, however, with improvements in medical imaging, thought now is

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changing and medical experts are beginning to believe that symptoms lasting 24 hours are resolved symptoms of true stroke and not TIA. The evolving definition of TIA is now that symptoms will last less than one hour (Goldstein, 2008). The cause of the TIA can be due to diminished perfusion through a partially occluded blood vessel or from an acute thrombotic event.

The incidence rate of TIA is approximately 240,000 per year with up to 15% of strokes preceded by a TIA event. Eleven percent of patients will have a stroke within the 7 days following a TIA. Sadly, many patients fail to seek medical attention following symptoms of TIA despite increased education.

The incidence of TIA is higher in African Americans, higher in men v/s women, and increases with age. Pediatric strokes occur in only 3% of the stroke population and are usually related to different etiologies than that of adult stroke (Goldstein, 2008).

By the time that a patient with TIA reaches the ER physician, symptoms are usually resolved. However, early intervention and treatment in TIA can reduce the risk of subsequent stroke by 88% (Alexander, 2008). It is vital that a thorough history is undertaken, and family or emergency workers be questioned as well, as they may have noticed deficits that the patient was unaware of having. Some of the important points to question are:

- Onset, timing, and duration of symptoms
- Have the symptoms happened previously, are they escalating
- Was movement, thought, or speech impaired
- History of Hypertension or cardiovascular disease
- History of previous stroke
- Family history of heart disease or stroke
- Current medications
- History of seizures
- Recent accidents, trauma, or medical procedures
- Recent infections

TIA's are generally caused by the same events that cause stroke: arteriosclerosis, emboli, atrial fibrillation, and hypercoagulation of the blood. Other less common causes of TIA are drug use, arterial dissection, and arteritis (Alexander, 2008).

Risk factors for TIA are similar to that of stroke and include hyperlipidemia, smoking, elevated homocysteine levels, obesity, and diabetes. Risk can be substantially reduced by treating hypertension and atrial fibrillation, two common modifiable risk factors found in the older age population. Persons under the age of 45 years old, who have stroke or TIA, frequently have no vascular risk factors.

Symptoms of TIA include:

Numbness of the face, legs, or hands with or without weakness. Usually only on one side of the body  
Paralysis  
Visual Changes  
Slurred speech  
Dizziness  
Double vision  
Lack of sight in one half of the visual field of the eye  
Blindness in one eye that goes away  
Difficulty with balance  
Aphasia  
Confusion  
Head pain

Transient blurring or graying of the vision is also another common symptom. Occasionally the line of sight will be shaded. Vertebrobasilar TIAs reflect vestibulocerebellar symptoms such as difficult motor control, dizziness, vertigo, difficult to understand, stumbling speech, vision abnormalities, and motor or sensory dysfunction (Alexander, 2008).

## **Hemorrhagic Stroke**

Hemorrhagic stroke is also called Intracerebral Hemorrhage (ICH) and accounts for approximately 15% of all forms of stroke, however, this type of stroke has a higher mortality rate than a cerebral infarct, with only 20% of patients regaining functional independence. The thirty-day mortality rate is 40-80% with almost half of ICH patients dying within the first 48 hours. These patients have a presentation with symptoms that are similar to those of ischemic stroke, but the patient is more ill. The patient is more likely to have headache, seizures, altered mental status, nausea, vomiting, and elevated blood pressure, however, none of these symptoms is a truly reliable indicator for hemorrhagic stroke (Nassisi, 2008).

Non-traumatic ICH is characterized as either primary, secondary, or spontaneous. In primary ICH, there is no correlation to any congenital or acquired lesion, while in secondary ICH a lesion is thought to be directly responsible for the bleed. Spontaneous ICH is unrelated to any traumatic event or surgical procedure (Alexander, 2008).

In ICH, bleeding occurs directly into the brain tissue and is thought to arise from damage to the small cerebral arteries of the brain from hypertension. Intraventricular hemorrhage describes bleeding directly into the ventricles of the brain (Alexander, 2008). However, other causes account for ICH as well such as cocaine abuse and antiplatelet therapy. Certain areas of the brain are more predisposed to ICH such as the thalamus, putamen, cerebellum, and brain stem. As the bleeding progresses,

surrounding brain tissue becomes damaged from the pressure of the developing hematomas (Nassisi, 2008).

ICH is diagnosed definitively by imaging studies, and must be done on an emergent basis using either non-contrast CT or MRI (Nassisi, 2008). Chest x-rays should be obtained to check for co-morbid conditions, as well as laboratory studies for coagulation studies, complete blood count, type and screen and basic electrolyte profile. ECG should be obtained and cardiac monitoring initiated as CVA events and Cardiac events can occur concurrently.

While hemorrhagic stroke is the least treatable of the CVA events, in patients who have less severe bleeds or a realistic prognosis of recovery, blood pressure control is a critical element in the management of hemorrhagic stroke. Grossly elevated blood pressure can lead to further bleeding and hematomas formation, which treatment leading to significant decreases in blood pressure may compromise cerebral perfusion and incite further damage to brain tissue.

The American Heart Association guidelines for treating elevated BP are as follows:

- (1) If systolic BP is  $>200$  mm Hg or MAP is  $>150$  mm Hg, then consider aggressive reduction of BP with continuous intravenous infusion with frequent BP (q5min) checks.
- (2) If systolic BP is  $>180$  mm Hg or MAP is  $>130$  mm Hg and there is evidence or suspicion of elevated ICP, then consider monitoring of ICP and reducing blood pressure using intermittent or continuous intravenous medications to maintain cerebral perfusion pressure  $>60$ - $80$  mm Hg.
- 3) If systolic BP is  $>180$  or MAP is  $>130$  mm Hg and there is NOT evidence or suspicion of elevated ICP, then consider modest reduction of BP (target MAP of 110 mm Hg or target BP of 160/90 mm Hg) with BP checks every 15 minutes. (Nassisi, 2008).

At the present time, there is no targeted therapy for hemorrhagic stroke. Anticlotting factors may be effective at stopping the advancement of bleeding into the brain, but may also lead to clotting in other areas of the body and increase the risk of emboli. Patients that are on anticoagulant therapy, and have elevated INR's should have therapy to reduce the INR by Vitamin K injection, fresh frozen plasma, or clotting factors if the bleeding has not advanced beyond the stage of a viable patient outcome.

## **Complications and Prognosis**

Increased intracranial pressure and brainstem herniation are the most dire complications. Worsening cerebral edema is often prognostic of neurological deterioration within the first 48 hours. For neurological decline within the first three hours, worsening of the hematoma is the most common cause. Twenty-five percent of the patients who are initially alert will have decreased consciousness within the first 24 hours.

The prognosis is variable depending upon the location of the bleed and the size of the hematoma, the larger the hematoma, the worse the prognosis. Patients who have bleeding into the ventricles have a poorer prognostic outcome, as do patients who are on oral anticoagulation.

## **Subarachnoid Hemorrhages**

Subarachnoid hemorrhages do not occur as frequently as ICH. The onset of subarachnoid hemorrhage is usually marked by severe headache of sudden onset that is described as “the worst headache I’ve ever had” frequently accompanied by nausea, vomiting, neck pain, and sensitivity to light. Neurological deficits may be present or may manifest hours to days after the bleeding begins (Alexander, 2008).

Spontaneous subarachnoid hemorrhages can be classified into two forms: those that arise from an aneurysmal rupture, and those that do not. Subarachnoid hemorrhages arising from aneurysmal rupture have a higher mortality rate. Most patients die within two weeks of the event and one-third of those who survive never regain functional independence. Non-aneurysmal subarachnoid hemorrhages are less likely to be fatal and have a much better prognosis for return to independent functioning upon recovery (Alexander, 2008).

## **Ischemic Stroke**

Stroke is a general term for loss of perfusion to a specific area of brain tissue, usually accompanied by some degree of neurological dysfunction. Within minutes of the onset of a stroke, the core of the infarction begins to form around the area of least perfusion. Around the core of the infarct lies an area of cytotoxic edema, or metabolically altered tissue. This tissue is salvageable if it is reperfused rapidly enough (Alexander, 2008).

Neurological functioning deteriorates rapidly during ischemic stroke, for each minute of ischemia, 1.9 million neurons, and 14 billion synapses are lost and the brain ages 3.6 years for every hour that passes after the onset of a stroke (Alexander, 2008). Although most stroke victims have some damage, the area of cerebral edema around the core called the penumbral tissue can achieve some degree of recovery if reperfusion is achieved within 3 hours of the beginning of the infarction.

**GENERAL REGIONS OF ISCHEMIC STROKE AND CORRESPONDING NEUROLOGIC DEFICITS**  
**Affected Region** **Common Signs and Potential Sequelae**

Left anterior hemisphere	Aphasia (esp. difficulty reading, writing, and calculating) Right limb weakness and sensory loss Right field visual defect
Right anterior hemisphere	Limb motor weakness or loss Left field visual neglect Unable to determine two-point stimuli on left side
Left posterior cerebral artery	Aphasia (esp. difficulty reading and naming objects) Right visual field defect Occasionally, right-sided numbness
Right posterior cerebral artery	Left limb sensory loss Left-sided neglect Left field visual defect
Vertebrobasilar territory (posterior circulation)	Bilateral vision disturbances and nystagmus Dysarthria and dysphagia Ataxia Dizziness, vomiting, headache No cortical deficits (e.g., aphasia and cognitive impairments)
Caudate nucleus, thalamus, frontal lobe (anterior circulation)	Sudden abnormal behavior
Thalamus (posterior circulation)	Numbness, decreased sensation on face, arm, leg on Same side

(Alexander, 2008)

Ischemic strokes are divided into two types, embolic and thrombotic as determined by the etiology. In addition, they can be further classified into five different sub types according to the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) system (Alexander, 2008).

**FIVE SUBTYPES OF ISCHEMIC STROKE AS CLASSIFIED BY  
THE TRIAL OF ORG 10172 IN ACUTE STROKE TREATMENT (TOAST)**

<b>Classification</b>	<b>Major Characteristics</b>
Large-artery Atherosclerosis (May be an embolus or thrombus)	Greater than 50% stenosis or occlusion of a major brain artery or branch cortical artery Cortical, cerebellar, brain stem, or subcortical infarct >15 mm Cortical or cerebellar dysfunction
Cardioembolism	Cardiac source of emboli

(May be high or medium risk based on evidence of embolism)	Cortical, cerebellar, brain stem, or subcortical infarct >15 mm Cortical or cerebellar dysfunction
Small-vessel (lacuna) Occlusion	Patient presents with lacunar syndrome* Subcortical or brain stem infarcts <15 mm may be detected
Other Determined Cause	May be caused by conditions such as dissection, hypercoagulable states, or sickle cell anemia May have characteristics of any of the other stroke subtypes
Undetermined Etiology	May be any of the following: Two or more causes identified Negative evaluation Incomplete evaluation
*The five classic lacunar syndromes are pure motor hemiparesis, pure sensory stroke, sensorimotor stroke, ataxic hemiparesis, and clumsy-hand dysarthria.	<b>(Alexander, 2008)</b>

### Embolic Strokes

Emboli can arise from either the heart or the arteries. Causes of emboli from the heart include atrial fibrillation, recent MI, prosthetic valves, native valvular disease, endocarditis, mural thrombi, cardiomyopathy, or a patent foramen ovale that allows the passage of venous clots through the heart. Arterial sources can be atherosclerotic plaque from the arterial intima that break loose and become emboli. Embolic strokes tend to be sudden in onset, and previous infarcts or calcified emboli may be noted on diagnostic imaging (Jauch, 2007).

### Thrombotic Strokes

Thrombotic strokes include both large vessel strokes and lacunar strokes. They are due to occlusions of the internal blood vessels or from decreased perfusion to brain tissue such as occurs with prolonged states of hypotension. Other factors can be states of hypercoagulability, sickle cell disease, and arterial dissections.

### Lacunar Strokes

Lacunar strokes represent 20% of all ischemic strokes. "They occur when the penetrating branches of the middle cerebral artery (MCA), the lenticulostriate arteries, or the penetrating branches of the circle of Willis, vertebral artery, or basilar artery become occluded. Causes of lacunar infarcts include microatheroma, lipohyalinosis,



fibrinoid necrosis secondary to hypertension or vasculitis, hyaline arteriosclerosis, and amyloid angiopathy. The great majority are related to hypertension”(Jauch, 2007).

### **Watershed Infarcts**

The infarcts are also known as border zone infarcts, and develop in the most distal portions of the arteries as a result of hypoperfusion, often from prolonged hypotension. They can produce bilateral neurological deficits.

### **Treatment of Ischemic Stroke**

Currently the use of the antiplatelet drug rt-PA is the only approved treatment for ischemic stroke. In order to be effective, tPA must be administered within 3 hours of the beginning of symptoms. This is the time window in which ischemic tissue is stunned and reperfusion may lead to recovery of brain viability. However, most patients present to the ER between 4-24 hours after symptoms have started.

<b>AMERICAN HEART ASSOCIATION RECOMMENDATIONS FOR THROMBOLYTIC THERAPY</b>
<b>Class I Recommendations</b>
Intravenous rt-PA (0.9 mg/kg, maximum dose 90 mg) is recommended for selected patients who may be treated within three hours after the onset of ischemic stroke (Class I, Level of Evidence A). Physicians should review the criteria outlined in the AHA guidelines to determine the eligibility of patients. In addition to bleeding complications, physicians should be aware of the potential side effect of angioedema that may cause partial airway obstruction (Class I, Level of Evidence C).
<b>Class II Recommendations</b>
A patient whose blood pressure can be lowered safely with antihypertensive agents may be eligible for treatment, and the physician should assess the stability of the blood pressure before starting rt-PA (Class IIa, Level of Evidence B). An elevated blood pressure that requires a continuous infusion of sodium nitroprusside may not be sufficiently stable for the patient to receive rt-PA. However, because time is limited, most patients with markedly elevated blood pressure cannot be managed adequately and still meet the three-hour requirement. A patient with a seizure at the time of onset of stroke may be eligible for treatment as long as the physician is convinced that residual impairments are secondary to stroke and not a postictal phenomenon (Class IIa, Level of Evidence C).
<b>Class III Recommendations</b>
The intravenous administration of streptokinase for treatment of stroke is not recommended (Class III Level of Evidence A). The intravenous administration of anurod, tenecteplase, reteplase, desmoteplase,

urokinase, or other thrombolytic agents outside the setting of a clinical trial is not recommended (Class III, Level of Evidence C).

## AMERICAN HEART ASSOCIATION (AHA) RECOMMENDATIONS REGARDING ANTIPLATELET TREATMENT

### Class I Recommendation

The oral administration of aspirin (initial dose is 325 mg) 24 to 48 hours after stroke onset is recommended for treatment of most patients (Class I, Level of Evidence A).

### Class III Recommendations

Aspirin should not be considered a substitute for other acute interventions for treatment of stroke, including the intravenous administration of rt-PA (Class III, Level of Evidence B). The administration of aspirin as an adjunctive therapy within 24 hours of thrombolytic therapy is not recommended (Class III, Level of Evidence A). The administration of clopidogrel alone or in combination with aspirin is not recommended for the treatment of acute ischemic stroke (Class III, Level of Evidence C). However, the panel supports research testing the usefulness of emergency administration of clopidogrel in the treatment of patients with acute stroke. Outside the setting of clinical trials, the intravenous administration of antiplatelet agents that inhibit the glycoprotein IIb/IIIa receptor is not recommended (Class III, Level of Evidence B).

### Classification of Interventions

Class	Definition
I	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective
II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
IIa	The weight of evidence or opinion is in favor of the procedure or treatment
IIb	Usefulness/efficacy is less well established by evidence or opinion
III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful

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### Levels of Evidence

Level	Definition
<b>A</b>	Data derived from multiple randomized clinical trials
<b>B</b>	Data derived from a single randomized trial or nonrandomized studies
<b>C</b>	Consensus opinion of experts

To be considered a candidate for tPA, the patient must meet certain guidelines:

The patient must have a diagnosed ischemic stroke with measurable neurological deficit.

The neurological deficits should not be clearing spontaneously

The neurological deficits should be significant and not isolated

Caution should be undertaken in the treatment of patients with major deficits

Indications of subarachnoid hemorrhage should not be present

Onset of symptoms should not be any greater than 3 hours.

No history of head trauma or stroke in the last 3 months

No history of MI in the last 3 months

No history or symptoms of GI bleed in the last 3 weeks

No arterial punctures in a non-compressible site in the last 7 days

No history of surgery within the past 14 days

No prior history of ICH

Systolic BP less than 185, diastolic BP less than 110

No acute trauma

No oral anticoagulant use or INR less than 1.7

For Heparin use in past 48 hours, must have normal APT, and platelet count above 100,000.

Blood glucose greater than 50mg/dL

No seizure with postictal impairments

No evidence of multilobular infarct on CT

Patient and family must understand risks and benefits of therapy (Becker, 2008).

There are multiple reasons that tPA is the ideal treatment for early ischemic stroke. Heparin is only useful in preventing future clotting; it will not dissolve a clot already in progress. Streptokinase carries an unacceptable risk of generalized bleeding, it is not clot specific. However, tPA works by attaching itself specifically to plasminogen already formed in a thrombus (Becker, 2008).

NINDS Recommended Stroke Evaluation Time Benchmarks for Potential Thrombolysis Candidate (Jauch,2007)

<b>Time Interval</b>	<b>Time Target</b>
Door to doctor	10 min
Access to neurologic expertise	15 min
Door to CT scan completion	25 min
Door to CT scan interpretation	45 min
Door to treatment	60 min
Admission to monitored bed	3 h

General Management of Patients With Acute Stroke (Jauch, 2007)

Blood glucose	Treat hypoglycemia with D50 Treat hyperglycemia with insulin if serum glucose >200 mg/dL
Cardiac monitor	Continuous monitoring for ischemic changes or atrial fibrillation
Intravenous fluids	Avoid D5W and excessive fluid administration IV isotonic sodium chloride solution at 50 mL/h unless otherwise indicated
Oral intake	NPO initially; aspiration risk is great, avoid oral intake until swallowing assessed
Oxygen	Supplement if indicated (SaO <sub>2</sub> <90%, hypotensive, etc)
Temperature	Avoid hyperthermia, oral or rectal acetaminophen as needed

Nursing care during the acute period of stroke is vital to the patient's recovery. The nurse should carefully monitor baseline neurological status, and reassess the patient at regular intervals for the first 24-48 hours. Any worsening of neurological symptoms should be immediately conveyed to the physician for further assessment. Monitoring of the patient's blood pressure should begin upon ED arrival and occur no less than every 15 minutes prior to tPA therapy and more frequently as the patient's condition warrants. Signs of increased ICP and possible intercranial bleeding include: hypertension, nausea, vomiting, headache and worsening neurological status. Before initiating tPA infusion, the nurse should validate that a signed consent for treatment is available on the chart per hospital policy and that documentation is present of the patient and family appraisal of the risks and benefits of the procedure per hospital policy.

During tPA infusion, the nurse should evaluate for sudden changes in neurological status and report these to the physician as they may indicate the beginning of intercranial bleeding. The blood pressure must be monitored every 15 minutes for at least 2 hours. Frequent blood pressure assessment is crucial for the following 24 hours. After the first 2 hours, blood pressure should be checked every 30 minutes for the next 6 hours, then hourly for the next 16 hours. The nurse should be alert for the presence of bleeding from puncture sites or IV sites and inform the physician accordingly. The urine should be assessed for the presence of hematuria.

The patient is kept on bed rest, then after 24 hours, a follow-up CT or MRI is performed to evaluate for intercranial bleeding from tPA. If no signs of bleeding are present, then the patient is started on preventative antiplatelet therapy and a program of rehabilitation is started through speech, physical, and occupational therapy as appropriate.

## **Conclusion**

The treatment and care of stroke patients is a specialized area of medicine and nursing whose full scope is beyond the limits of this educational activity. What has been attempted to emphasize is familiarizing the nurse with the different types of brain attacks and acute management of stroke victims in a timely manner to preserve eligibility of the patient for thrombolytic care along with care management techniques for tPA.

There are still challenges to overcome in the battle against the disabling effects of brain attacks, and perhaps the greatest of these challenges is to prevail against patient procrastination in utilizing emergent medical evaluation for stroke symptoms. The number one reason that stroke victims fail to be eligible for thrombolytic therapy is their arrival at the ED outside the therapeutic window of opportunity for thrombolytic therapy. Continuing to educate patients about the time factor involved with stroke will hopefully increase earlier utilization of emergency management services.

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