Acute Ischemic Stroke

By:

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To the Faculty of Washington State University:

The members of the committee appointed to examine the ICNE Research requirements and manuscript of Pat M. Hall find it satisfactory and recommend that it be accepted.

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Pat Hall
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ABSTRACT

Stroke is the third leading cause of death in adults in the United States, behind ischemic heart disease and all forms of cancer. Stroke is also the leading cause of serious, long-term adult disability and the financial burden in lost productivity and health care expenses are estimated at $30 billion annually. Despite the magnitude of the problem, prevention strategies and treatment methods are incompletely used. The American Heart Association developed guidelines for the management of acute ischemic stroke stressing the need for rapid response from the public, emergency medical services and clinicians. Thrombolytics, more specifically, tissue plasminogen activator {TPA(Activase)} has increased interest in treating acute ischemic stroke and demonstrated improved outcomes when given within three hours of onset of symptoms in selected individuals. Whether treated with thrombolytics or not, stroke patients can experience a high incidence of complications due to neurological involvement and are in need of supportive care.
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One of the most common yet serious illnesses is ischemic stroke. It is the third leading cause of death in adults in the United States, behind ischemic heart disease and all forms of cancer (Selman, Tarr, & Landis, 1997). Stroke, also referred to as “brain attack”, occurs 500,000 or more times a year with approximately 158,000 Americans dying in 1995 (Stroke Statistics, 1998). Data collected by the American Heart Association (AHA) indicates that in the United States there is a stroke every minute and a person dies of stroke about every 3 ½ minutes (Stroke Statistics, 1998).

The National Center for Health Statistics (Spotlight on Stroke, 1996) reports that nearly 3 million Americans have suffered a stroke, or 1 per 100 population. The incidence of stroke doubles with every decade after 55 years of age. Five percent of males aged 65 and over and 6% of females in the same age group have suffered a stroke. Stroke rates are 50% higher in African-American men than in white men, and 130% higher in African-American women than in white women. According to information from the United States Department of Health and Human Services (USDHHS,1995), about one-third of the increased risk in African-Americans is attributed to cardiovascular risk factors, another third to factors related to family income, and one-third is unexplained. First-ever strokes account for about 75% of acute events and recurrent strokes for about 25% with a recurrence rate of 7-10% per year which is highest in the first year after a first stroke (USDHHS,1995).

In the Framingham study, recurrence was found to be particularly common for thrombotic stroke and more frequent in men (USDHHS, 1995). The Southeast has the greatest prevalence of cerebrovascular disease and is referred to as the “stroke belt”; the lowest prevalence is in the Northwest. Stroke affects more people every year than any other neurological illness.
Alzheimer's disease comes the closest with 400,000 new cases each year compared to 500,000 new cases of stroke each year (Lyden, 1997).

Stroke is also the leading cause of serious, long-term adult disability in the United States with more than 3,000,000 people in 1991 having survived a stroke (USDHHS, 1995). Furthermore, stroke accounts for more than half of all patients hospitalized for acute neurological disease. Statistics indicate that 31% of stroke survivors needed help caring for themselves, 20% required help walking, 71% had impaired ability to work and 16% had to be institutionalized (USDHHS, 1995). Approximately, 10% of stroke survivors are without disability and are able to function independently.

The financial burden with lost productivity and health care expenses are estimated to be nearly 30 billion annually (USDHHS, 1995, Matcher, 1998). This includes $17 billion in direct health care costs and another $13 billion in indirect costs. In 1996, for people under the age of 65, the average cost of a stroke from hospital admission to discharge was $18,244 with the average length of stay being 6.7 days. Seventy-five percent of stroke patients are able to return home after completing a comprehensive rehabilitation program and their mean duration of survival after the stroke is 7.5 years (Schnell, 1997; Reddy and Reddy, 1997).

Despite the magnitude of the problem both financially and emotionally, prevention strategies and treatment methods are incompletely used. A study by the AHA (Adams, 1994) revealed that nearly two-thirds of persons surveyed could not identify one warning sign of a stroke and there is a perception by the public that stroke is not a medical emergency. Less than one-third of patients with an acute stroke are admitted to the hospital within 24 hours of onset of symptoms and less than 50% are brought to the hospital by emergency transport (Selman, et al.,
1997). Starkman (1996), feels that the public is in denial when it comes to acknowledging that a loved one has experienced a stroke. The public recognizes that the stroke has taken place, but do not want to acknowledge that an acute neurological deficit has occurred. Stroke is many people’s greatest fear.

Despite this, there has been an increase in public awareness of stroke risk factors and the need for changes in lifestyle, such as cessation of cigarette smoking, decreasing alcohol and caloric intake, controlling blood lipids and diabetes mellitus. The reduction of stroke mortality and morbidity over the past 25 years is largely due to hypertension control, atherosclerosis prevention, therapy for cardiac disease to eliminate embolic sources, and surgical therapy for stroke prevention (Aminoff, 1998; National Stroke Association, 1998).

Risk factors are divided into those that are potentially modifiable and those that are not. Modifiable risk factors are: transient ischemic attack (TIA), hypertension, diabetes mellitus, atrial fibrillation, left ventricular hypertrophy, and cigarette smoking. The paramount modifiable risk factor for stroke is hypertension and is especially strong for levels above 160/95 mm Hg (USDHHS, 1995). Cigarette smoking increased the risk of stroke by about 50% in both sexes and all age groups, and the risk was directly related to the number of cigarettes smoked per day. Heavy consumption of alcohol, cocaine use, and obesity have also been linked to an increase of stroke. Prior stroke, age, sex(male), race, and family history are nonmodifiable factors. Increasing age is the strongest fixed factor with 72% of strokes occurring in people 65 years or older (USDHHS, 1995).

**Pathophysiology**

The pathogenesis of stroke can be divided into two broad categories: ischemic and
hemorrhagic. Approximately 80 to 85% of strokes are ischemic, with the remainder (17%) being hemorrhagic (Selman, et al.,1997; National Stroke Association, 1997). About two-thirds of hemorrhagic strokes are intracerebral hemorrhages commonly due to poorly controlled hypertension and the remaining third are subarachnoid hemorrhages, caused most often by aneurysmal rupture (Frankel & Kothari, 1997).

Approximately, two-thirds of ischemic strokes are caused by in situ thrombotic occlusions in either large or small vessels most often caused by atherosclerosis (Frankel & Kothari, 1997). Thrombosis may occur anywhere along a carotid artery or its branches, with a frequent site being at the bifurcation of the common carotid into the internal and external carotid arteries.

Thrombotic stroke is a common stroke in diabetics patients.

About one-third of ischemic strokes are embolic, with the clot traveling through the arterial circulation system until it lodges in a vessel too small to allow passage, which in turn, blocks blood flow. Embolic events usually stem from cardiac sources such as atrial fibrillation, endocarditis, and artificial cardiac valve replacement. Less commonly, the clot forms at the carotid bifurcation and either blocks blood flow or gives rise to emboli that lodge in cerebral vessels, most often being the middle cerebral artery (Rordorf, Koroshetz, Copen & Cramer, 1998). The incidence of cerebral embolism increases after age 40. (Schnell, 1997).

Under stroke conditions the brain is perfused with blood at the expense of other less vital organs. When blood flow is blocked to a part of the brain, there is a limited amount of time before irreversible tissue injury occurs. Hypoxia (inadequate oxygenation) can cause cerebral ischemia. The longer the duration of ischemia, the greater the likelihood of irreversible injury. The extent of ischemia is not uniform throughout all portions of the brain. Severe reduction of
blood flow can kill brain cells within minutes, whereas, a relatively mild decrease in blood flow can be tolerated for hours without permanent injury.

Normal cerebral blood flow (CBF) is 55 ml./100 g./minute (Aminoff, 1998). Lyden, Rapp, Babcock, & Rothrock (1994) state that after a vascular occlusion, a portion of the ischemic brain will survive for hours around the core of the ischemic zone where CBF is near zero. The neurons in the penumbra, an area surrounding the ischemic core that is receiving blood from collateral circulation, may not function when the CBF is between 12 and 18 ml./100g/minute, but may survive up to a few hours and be potentially recoverable. The ischemic penumbra may survive if early reperfusion is restored. Within the area of the ischemia is a core that contains cells that are highly dependent on the occluded artery and this area probably cannot be salvaged (Steiner & Hacke, 1998).

The severity of symptoms experienced after the stroke depends on the size and number of collateral vessels. The earlier perfusion is restored to the ischemic penumbra, the greater the likelihood that the injury will be reversible. According to Heiss, Grond and Thiel (1997), much of the tissue initially below the conventional viability threshold can survive if sufficient reperfusion is achieved. However, most tissue that remains hypoperfused will become necrotic and some tissue may become necrotic, despite good initial flow and reperfusion. The amount of edema that takes place may also lead to neurological changes that sometimes resolves within hours to days.

**Early Recognition and Treatment**

Prior to 1995, acute stroke was largely considered an unfortunate medical problem requiring supportive care and monitoring. The National Institute of Neurological Disorders and Stroke (NINDS) study (1995), reported on the use of tissue plasminogen activator (TPA
(Activase}) for acute stroke management. With this report, there has been a change in the philosophy of most of the medical community toward the urgency of swift treatment.

Pepe (1997), addressed the need for a Chain of Recovery at the National Symposium on Rapid Identification and Treatment of Acute Stroke. The key links include: 1) immediate identification of stroke symptoms and appropriate reaction by bystanders (or the patients themselves), 2.) early access to emergency medical services (EMS), 3.) rapid EMS response, treatment, and evacuation to designated centers capable of immediately providing definitive diagnosis and treatment of stroke, 4.) early communication to alert the specialty receiving center, ensuring preparation and immediate mobilization of resources for the stroke patient, 5.) rapid diagnosis and intervention at those designated receiving centers, 6.) specialized treatment and evaluation of complications, precipitating factors, and accompanying conditions and 7.) appropriate rehabilitation, when applicable.

The AHA under the guidelines for the management of patients with acute ischemic stroke (1996), also stress the need for rapid response that includes the entire community. The recognition of stroke occurs at three levels: the public, nonphysician EMS, and clinician. The successful outcome begins with the signs of stroke recognized by the patient or bystanders. This requires a wide public awareness of the signs and symptoms of stroke and the immediate contact of the EMS. The EMS personnel should be instructed in rapid recognition, evaluation, treatment, and transport of patients with stroke. A baseline assessment and quick notification of the hospital can save valuable time. The clinician should suspect a stroke whenever there is a sudden onset of focal neurological signs. Initial evaluation should assess the patient's airway, ventilation, and circulation with a neurological exam that should be completed in 5 to 10 minutes. Emergent
evaluation is needed to confirm the cause of the stroke, provide information regarding the possible reversibility, give clues about possible etiology, predict likelihood of immediate complications and begin appropriate treatment.

**Guideline for Clinical Decision Making**

Established guidelines are important tools for minimizing delays and enhance appropriate treatment. The AHA stroke council and American Academy of Neurology jointly developed guidelines with the emergence of thrombolytics in the treatment of acute ischemic stroke. Although details of treatment protocols vary across the nation, many centers are devising strategies for rapid diagnosis and institution of appropriate therapy. Just as every hospital has a plan for heart attack, every hospital needs a plan for stroke. These guidelines are meant to expedite and improve the care given but cannot be considered effective if those in the health care profession are not open to implement the care.

The earlier the treatment is started after the onset of stroke symptoms the better. Therefore, when an individual develops symptoms, the “911” emergency telephone system should be called immediately for transport and evaluation at a medical facility. There should not be delay to see if the symptoms resolve on their own or to notify relatives or private physician for advice. The AHA has stepped up it’s campaign to make the public aware of the need to know the warning signs of stroke (Table 1) and to act quickly.

Part of the problem in the delayed treatment of a stroke patient is the subtle clinical presentation. Most stroke patients do not evoke the same level of anxiety or action as one experiencing an acute myocardial infarction or trauma. Lack of response and anxiety applies not only to the lay person witnessing the symptoms, but also those in the medical field who are not
educated to current medical practices. Prehospital providers can have a profound influence on the patient’s outcome by reducing the time required to deliver the patient to a medical facility (Adams, 1996).

Emergency medical system (EMS) personnel should be knowledgeable of the rapid recognition of the signs and symptoms of an evolving stroke (Table 2), treatment and transport to a medical facility. The baseline assessment should only take a few minutes.

According to Adams (1996), intravenous access, supplemental oxygen and cardiac monitoring should begin during transport by the EMS. Notification of the medical facility should take place with information regarding patient assessment, time of stroke occurrence, and estimated time of arrival. This enables the staff at the medical facility to assemble a “stroke team” and meet the patient on arrival. This “stroke team” should consist, at a minimum, of the emergency room physician or nurse practitioner who initially assesses the patient, an emergency nurse who will initially care for the patient, a consulting or admitting physician (usually a neurologist) who will provide long-term care, radiology personnel (technician and radiologist) for cranial computed tomography (CT) and possible magnetic resonance imaging (MRI), and laboratory personnel. Some facilities encourage a family member to ride with the EMS personnel to facilitate quick access to the patient’s medical history and events that occurred prior to the stroke.

The AHA recommends that once the patient has arrived, an eyewitness account (if possible) of the stroke should be obtained to establish the time and mode of symptom onset, which is crucial to treatment. Further patient information should also include history of prior seizures, evidence of trauma, infection, diabetes, illicit drug use, and other significant medical
history. Initial management of patients with suspected stroke should include a thorough physical and neurological examination. Respiratory and cardiac function also need to be assessed and supported if appropriate. A complete metabolic panel (CMP), complete blood count (CBC), prothrombin time (PT), partial prothrombin time (PTT), and bedside glucose need to be obtained along with a CT scan.

Bock, at the National Symposium on Rapid Identification and Treatment of Acute Stroke (1997) discussed the paradoxial situation that exists where the victim who might gain the most from aggressive diagnostic and therapeutic interventions is left alone to silently extend their damage while others, who have poorer prognoses and less to gain, are given the benefit of expedited care. All the more reason to recognize subtle changes mentioned by the patient or family member and act quickly.

It is important to recognize various stroke “mimics” during the initial emergency evaluation. These can include: recent seizures, suicide gestures/efforts, conversion disorders, cocaine or amphetamine use, SAH despite a normal CT scan, transient global amnesia, systemic infection, and toxic/metabolic encephalopathy (Selman, et al. 1997). Other neuromedical problems that can be mistaken for stroke include migraine complicated by hemiparesis, brain tumor, carpal tunnel syndrome associated with hand numbness, and brachial plexopathy with arm weakness (Starkman, & Dobkin, 1995). Table 3 lists the information necessary for emergent evaluation. Table 4 provides information on evaluation of diagnostic tests.

**Diagnostic Testing**

An emergency CT scan is absolutely necessary for patients suspected of having a stroke because of the subsequent therapeutic decisions depend on the results. CT is the modality of
choice and is usually requested to exclude SAH. It is widely available, allows for close monitoring, and is tolerated by gravely ill patients. National Stroke Association (1998) reports that a CT scan does not show definitive changes of cerebral infarction for 24 to 48 hours after onset, but subtle signs of ischemia may appear within 3 hours. It can define almost all intracerebral hematomas >1 cm. in diameter and more than 95% of subarachnoid hemorrhages. The findings require a high-quality CT scanner and must be carefully reviewed by a radiologist.

The MRI has some advantages over a CT scan, however, it is not widely available. There is better contrast resolution of all parenchymal structures, significantly better sensitivity to detect abnormal tissues and show evidence of ischemic stroke sooner than a CT scan. Demaerel (1996), states the advantage of MRI is better visualization of small white matter, brainstem and cortical infarcts. It also plays an important role in excluding more unusual causes of stroke, such as vasculitis, arterial dissection, and multiple sclerosis.

A study in Radiology (Sorensen, 1996) reports that the thirty minute long MRI may be the fastest way to fully evaluate patients with neurological change by providing details of flow patterns in the Circle of Willis, extent and location of any acute ischemic tissue changes or perfusion abnormalities. Disadvantages include difficulty monitoring seriously ill patients, time needed to perform the procedure and motion artifacts in patients unable to follow commands to remain still, claustrophobia, and those with any implanted metal (pacemakers).

Two new MRI techniques that use diffusion-weighted imaging and perfusion imaging appear to be more sensitive in depicting infarcted tissue by changes in water mobility and providing information regarding poorly perfused vascular territories with indication on how much brain tissue is at risk (Demaerel, 1996; Caplan, 1998). Focal cerebral ischemia can be detected
earlier with the diffusion-weighted and perfusion imaging than with a CT scan or with the traditional MRI (Sorensen, et al, 1996; Everdingen, Grond, Kappelle, Ramos, & Malie, 1998)).

If the clinical presentation, laboratory data and results of both CT scan and MRI are consistent with the diagnosis of acute ischemic stroke, the indications, contraindications and relative contraindications for thrombolytic therapy need to be considered. In general, thrombolytics expedite clot lysis and restore circulation which may limit the extent of brain injury and improve the outcome of the stroke. In the late 1960's and early 1970's intracranial bleeding was a frequent complication and the therapy was abandoned. At that time, there was no CT diagnostics, consequently many patients were treated that may have had SAH and the 3 hour time frame was not instituted (Adams, et al, 1996).

The NINDS study (Marler, 1995), demonstrated neurological improvement after a stroke when given within a three hour onset of stroke symptoms in selected individuals. There was also a significant improvement at 24 hours and at three months. Following the NINDS study, the recommended dose of TPA is 0.9mg/kg (maximum 90 mg). The initial 10% is given as an intravenous bolus over one minute and the remaining infused over 60 minutes. No anticoagulants or aspirin should be given within the first 24 hours after TPA. The AHA, working in conjunction with the American Academy of Neurology, have provided eligibility criteria guidelines listed in Table 5.

Thrombolytic therapy is not without risk. Symptomatic intracranial hemorrhage (ICH) occurred in 6.4% of patients treated within the NINDS-sponsored study (Adams, et al., 1996). However, the overall rate was lower than reported in other studies and despite the hemorrhage, the rate of death or severe disability was less in the actively treated group.

Other thrombolytic agents such as streptokinase and urokinase will lyse clots and have
been used for the treatment of stroke. Streptokinase has been documented to have serious adverse effects and questionable efficacy in treating stroke. Urokinase has been used but generally as a thrombolytic agent delivered via a catheter directly to the clot obstructed artery or vein (National Stroke Association, 1998; Albers, 1997).

**Complications**

The majority of stroke patients should be admitted to the hospital because of the high incidence of complications due to neurological involvement. Emergent supportive care and treatment of acute complications is initiated whether the stroke is ischemic or hemorrhagic in nature. Most stroke patients with the exception of hemorrhagic patients, die from complications related to the stroke, rather than the stroke itself (Starkman & Dobkin, 1995). There are many acute neurological complications that can occur (Table 6) after a stroke. Cerebral edema, seizures and hemorrhagic conversion are three of the most acute complications.

Fifteen percent of ischemic stroke victims develop severe brain edema which peaks at 3 to 5 days and is usually not a problem for the first 24 hours except for those with large cerebral infarctions. Brain edema and elevated intracranial pressure caused by occlusions of major intracranial arteries and large multilobar infarctions commonly result in death within the first week. Brain edema can lead to further extension of the original infarction or additional injury to other areas of the brain with additional neurological damage. Treatment may include the use of osmotic diuretics, hyperventilation, drainage of cerebral spinal fluid, and surgery. Controversial management of cerebral edema is the use of corticosteroids due to the increase of infection, furosemide, mannitol and barbiturates (Adams, et al., 1994; Albers, 1997).

Seizures are most likely to occur within 24 hours of stroke and are usually partial, with or without secondary generalization. Overall prognosis of stroke is not altered with intermittent
seizures, which may reoccur in 20% to 80% of cases. Prophylactic administration of anticonvulsants is not recommended for stroke patients not experiencing seizures. Treatment depends on the type and frequency of seizures with phenytoin, carbamazepine, lorazepam and diazepam most commonly used. (Adams, et al., 1994; USDHSS, 1995; Albers, 1997)

According to Adams, et. al., (1996), the treatment of thrombolysis-related bleeding depends on the location and size of the hematoma, ability to control the bleeding mechanically, risk of worsening the neurological condition or death, interval between drug administration and hemorrhage, and the type of thrombolytic drug given. The location, size and etiology of stroke may influence the bleeding complication. In the NINDS stroke trial (Marler, 1995), 6.4% of those in the TPA group experienced symptomatic brain hemorrhage and approximately half of those patients died. Many persons presenting with a stroke are taking aspirin, ticlopidine, or warfarin which may influence the eligibility for, or success of thrombolytic therapy in the setting of an acute ischemic stroke. There was a higher incidence of hemorrhage in those patients presenting to the emergency room with very severe deficits or early evidence of cerebral edema on their pretreatment CT scan.

Treatment for those suspected of having intracranial hemorrhage include STAT blood draw of prothrombin time (PT), partial thromboplastin time (PTT), hematocrit, hemoglobin, platelet count and fibrinogen. Blood should be typed and crossed. If TPA is being infused, it should be discontinued immediately. If intracranial bleeding is suspect, an emergent CT scan is needed and a neurosurgeon should be consulted. Some active bleeding from intravenous or arterial sites can be controlled mechanically with direct compression.

Hypoxia caused from partial airway obstruction, hypoventilation, aspiration pneumonia and atelectasis can result in anaerobic metabolism and decrease in energy stores which in turn can
increase the extent of brain injury. Maintaining adequate oxygenation is a critical component in the emergent care of a stroke patient. Intubation may be necessary when the level of consciousness deteriorates. Hypoxia should be monitored by arterial blood gases and pulse oximetry.

Optimum management of hypertension following a stroke remains controversial. There are many reasons for an elevated blood pressure: stress from the stroke, a full bladder, pain, underlying hypertension, physiological response to brain hypoxia, or increased intracranial pressure. Once any or all of these problems are taken care of, the blood pressure may move to within normal limits. Lowering of blood pressure should, therefore, be done cautiously because the patient’s neurological status may be compromised with the use of antihypertensive drugs.

Most guidelines recommend minimal to no initial treatment of mild to moderately elevated blood pressure during the first several hours with an ischemic stroke, but more aggressive treatment in patients with intracerebral or subarachnoid hemorrhage (Broderick, 1997). Agents used to treat elevated blood pressure should be those that are easily titrated with a quick onset of action, such as labetalol or low dose enalapril. In the NINDS stroke study, exclusion was made if the systolic blood pressure was >185 mm Hg or diastolic 110 mm Hg or if aggressive treatment was needed to reach those limits.

Hypotension is rarely a problem in stroke patients and may signify dehydration, diminished cardiac output or arrhythmia. Correction of the above problems during the first few hours of a stroke are optimal. The use of intravenous fluids, vasopressors, colloid solutions, and cardiac medications may be needed (Adams, et al., 1994; Albers, 1997; Broderick, 1997).

According to the AHA guidelines for the management of patients with acute ischemic stroke (1994), hyperglycemia can be a response to a serious brain injury and it is not certain if the
elevated glucose would make the stroke worse. Some studies have correlated poor outcome after a stroke with an elevated blood glucose. However, hypoglycemia can produce focal symptoms that may mimic stroke (Albers, 1997). Both hyperglycemia and hypoglycemia should be treated. NINDS exclusion criteria for receiving TPA is a glucose <50 mg/dl or > 400 mg/dl.

Acute myocardial infarction occurs concomitantly with stroke onset in up to 3% of patients and atrial fibrillation in 18%. Fewer than 2% of patients with acute myocardial infarction develop ischemic strokes and 85% of such strokes occur within the first month (Starkman & Dobkin, 1995).

Supportive Care

All patients admitted for stroke should be considered at risk for neurological worsening with approximately 25% of patients deteriorating during the first 24 to 48 hours after admission (Adams, et al 1994). Changes in neurological function are often hard to predict, therefore, stroke patients should be admitted to a unit that has nursing personnel trained to promptly recognize subtle changes in neurological status. Supportive care should include facilitating measures both medical and surgical, observe changes that may prompt interventions, prevention of complications, preventative measure of future strokes, and rehabilitation to restore neurological function (Aminoff, 1998).

Assessment of vital signs and neurological status should take place frequently during the first 24 hours. The patient is usually placed on bed rest, however, early mobilization will decrease the chance of pneumonia, deep vein thrombosis, pulmonary embolism, contractures, and decubitis ulcers. Frequent turning and passive range of motion can be started during the first 24 hours.

These patients are at high risk of aspiration, choking, excessive coughing and vomiting. Oral intake should be held for at least 24 to 48 hours (Schnell, 1997). Assessment of the ability to
swallow should take place to decrease the possibility of aspiration. Pneumonia is the leading cause of death due to aspiration (Starkman & Dobkin, 1995). Patients with infarctions in the brain stem, multiple strokes, large hemispheric lesions, or depressed consciousness are at greatest risk of aspiration (Adams, et al., 1994).

Malnutrition is of concern if the patient is unable to sustain nutrition. If necessary, a feeding tube or gastrostomy tube may be inserted to implement caloric intake and administration of medication. Further patient care includes keeping the head of the bed raised 30 degrees, pulmonary toileting, encouragement of coughing and postural drainage. Vital signs should be followed with special attention given to the temperature which may signify pneumonia and appropriate antibiotic therapy instituted, along with serial chest Xrays.

Due to immobility and inability to complain of pain, stroke patients are at high risk for deep vein thrombosis (DVT). Starkman & Dobkins (1995), rate the incidence of DVT, as high as 75% up to 2 weeks after a stroke. Pulmonary embolism is the fourth commonest cause of death in patients who have had a stroke, which usually occurs when the DVT involves the vessels proximal to the popliteal vein. Early mobilization, subcutaneous heparin, aspirin, alternating pressure stockings and elastic support stockings are suggested modes of treatment.

Sixty percent of patients that have had a stroke, experience urinary incontinence but only 15% remain incontinent at six months (Ayers, et al., 1995). Secondary septicemia occurs in 5% of stroke patients (Adams et al., 1994). The use of indwelling catheters should be avoided due to the increased incidence of infection.

Rehabilitation

Twenty to thirty percent of stroke survivors are likely to require inpatient rehabilitation (Starkman & Dobkins, 1995). Neurological and functional recovery occurs most rapidly in the
first 1 to 3 months after stroke (USDHHS, 1995). In the Framingham study, improvement in motor strength and performance of self-care functions slowed 3 months after stroke, but continue at a reduced rate throughout the first 12 months, especially in the patients with cerebral infarctions. Stroke rehabilitation begins during the acute hospitalization, as soon as the diagnosis of stroke is established and life-threatening complications are under control. Rehabilitation has been described as "the planned withdrawal of support" in which services are provided when needed and removed when no longer needed (USDHHS, 1995). Rehabilitation should include prevention of further strokes and complications, proper management of general health functions, mobilizing the patient, resumption of self-care activities, emotional support and education for patient and family.

Summary

To optimize the recovery outcome of those with acute ischemic stroke, several steps need to be taken and strengthened by the public and medical personnel. These include: immediate identification of stroke symptoms and appropriate actions, quick access to EMS, rapid EMS response, treatment and evacuation, early communication to the medical facility, rapid diagnosis and interventions, specialized treatment, evaluation of complications, precipitating and accompanying factors, and appropriate rehabilitation when applicable.
References


Lyden, P. D., Rapp, K., Babcock, T., & Roghrock, J. (1994). Ultra-rapid identification, triage,


of ischemic stroke. *American Family Physician, 55*(8), 2655-2663.


CO.


### TABLE 1.

**Warning Signs of Stroke**

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<table>
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<tr>
<td>♦  Sudden weakness or numbness of the face, arm or leg on one side of the body.</td>
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<tr>
<td>♦  Sudden dimness or loss of vision, particularly in one eye.</td>
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<tr>
<td>♦  Loss of speech, or trouble talking or understanding speech.</td>
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<tr>
<td>♦  Sudden, severe headaches with no apparent cause.</td>
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<tr>
<td>♦  Unexplained dizziness, unsteadiness or sudden falls, especially along with any of the previous symptoms.</td>
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TABLE 2.

**Emergency Medical System Personnel Treatment**

- Assure adequate airway
- Monitor vital signs: treat hypotension but not hypertension
- Conduct general assessment: with special attention of recent trauma and cardiovascular abnormalities.
- Conduct neurological examination: Level of consciousness, seizure activity, Glasgow Coma Scale, pupils: size, equality, reactivity, & limb movements

TABLE 3

**Emergent Evaluation Information**

| ♦ Time of onset of symptoms | ♦ Glucose (can be done by finger stick) |
| ♦ Blood tests to include: | ♦ Physical and neurological examination |
| Complete Blood Count (CBC) | ♦ 12-lead electrocardiogram (EKG) |
| Complete Metabolic Panel (CMP) | ♦ Noncontrast brain CT scan |
| Prothrombin Time (PT) | ♦ *MRI (if hemorrhage excluded by CT scan) |
| Partial Prothrombin Time (PTT) | |

*An MRI is not standard practice at many medical institutions, but is included due to new research that is available.*
## TABLE 4

### Evaluation of Diagnostic Tests

<table>
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<th>Sensitivity/Specificity</th>
<th>Time to Results</th>
<th>Patient Teaching and Preparation</th>
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<tbody>
<tr>
<td>CBC</td>
<td>96%</td>
<td>30 minutes to 1 hour</td>
<td>Explain that this test is to evaluate for anemias, infection, to track the progress of various diseases, and to monitor for side effects resulting from acute or chronic use of drugs that may cause blood dyscrasias. No fluid or food restrictions. 5-7 ml. blood sample.</td>
</tr>
<tr>
<td>CMP</td>
<td>96..5%</td>
<td>30 minutes to 1 hour</td>
<td>Explain that this test is to assess renal function and electrolyte balance. No fluid or food restriction. 5 ml. blood sample.</td>
</tr>
<tr>
<td>Prothrombin Time</td>
<td>95%</td>
<td>30 minutes to 1 hour</td>
<td>Explain that this test is to evaluate clotting mechanism. No fasting required. Ask if patient is currently on warfarin. 5-7 ml. blood sample.</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>Low Sensitivity/High Specificity</td>
<td>5-10 minutes</td>
<td>Explain that the test takes 5-10 minutes &amp; requires the patient to lay still in supine position without talking. Electrodes will be placed on arms, legs, and chest and that the patient will feel nothing during the testing</td>
</tr>
<tr>
<td>Computed Tomography Scan (CT Scan)</td>
<td>30% - 79% Sensitivity</td>
<td>30 minutes to 1 hour</td>
<td>Explain that the patient needs to remain still and lay flat. Best to be NPO for several hours prior to test, but may be performed if the patient has eaten. Evaluate the need for premedication if the patient voices concern about claustrophobia.</td>
</tr>
<tr>
<td>Magnetic Resonance Imaging (MRI)</td>
<td>82 - 95% Sensitivity</td>
<td>30 minutes to 90 minutes</td>
<td>Explain that the test does not expose the patient to radiation, all metal objects must be removed. Patient must remain motionless, assess pt. for claustrophobia. Earplugs may be worn due to a thumping noise that may be heard. A microphone allows communication while the test is taking place. There is no food/fluid restriction, best to empty bladder prior to testing.</td>
</tr>
</tbody>
</table>

TABLE 5

Eligibility Criteria for Treatment with TPA.

<table>
<thead>
<tr>
<th>INCLUSION CRITERIA</th>
<th>EXCLUSION CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ ≥18 years of age</td>
<td>➢ Rapidly improving or minor symptoms</td>
</tr>
<tr>
<td>➢ Clinical diagnosis of ischemic stroke</td>
<td>➢ CT scan showing evidence of intracranial hemorrhage.</td>
</tr>
<tr>
<td>➢ Measurable neurological deficit</td>
<td>➢ History of intracranial hemorrhage.</td>
</tr>
<tr>
<td>➢ Clearly defined time of stroke onset (≤ 90 min. before treatment or 91-180 min. before treatment)</td>
<td>➢ Seizure at stroke onset</td>
</tr>
<tr>
<td>➢ Baseline CT scan showing no evidence of intracranial hemorrhage</td>
<td>➢ Stroke or serious head trauma ≤ 3 months.</td>
</tr>
</tbody>
</table>
<pre><code>                                                                                   | ➢ Major surgery or other serious trauma ≤ 2 weeks.      |
                                                                                   | ➢ GI or UT hemorrhage ≤ 3 wks.                          |
                                                                                   | ➢ SBP &gt; 185 mm Hg; DBP &gt; 110 mm Hg.                      |
                                                                                   | ➢ Aggressive treatment to lower BF.                      |
                                                                                   | ➢ Glucose &lt; 50 mg/dl or &gt;400 mg/dl                       |
                                                                                   | ➢ Symptoms of subarachnoid hemorrhage.                   |
                                                                                   | ➢ Arterial puncture at noncompressible site or lumbar puncture ≤ 1 week. |
                                                                                   | ➢ Platelet count &lt; 100,000/mm³.                         |
                                                                                   | ➢ Heparin ≤ 48 hrs. associated with elevated aPTT       |
                                                                                   | ➢ Clinical presentation suggesting post-MI pericarditis or acute MI. |
                                                                                   | ➢ Pregnant or lactating females.                         |
                                                                                   | ➢ Currently taking oral anticoagulants with PT &gt; 15 seconds. |
</code></pre>

<table>
<thead>
<tr>
<th>Neurological</th>
<th>Medical</th>
</tr>
</thead>
<tbody>
<tr>
<td>♦ Cerebral edema</td>
<td>♦ Aspiration</td>
</tr>
<tr>
<td>♦ Hydrocephalus</td>
<td>♦ Hypoventilation</td>
</tr>
<tr>
<td>♦ Increased intracranial pressure</td>
<td>♦ Pneumonia</td>
</tr>
<tr>
<td>♦ Hemorrhagic transformation</td>
<td>♦ Myocardial ischemia</td>
</tr>
<tr>
<td>♦ Seizures</td>
<td>♦ Cardiac arrhythmias</td>
</tr>
<tr>
<td></td>
<td>♦ Deep vein thrombosis</td>
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<tr>
<td></td>
<td>♦ Pulmonary embolism</td>
</tr>
<tr>
<td></td>
<td>♦ Urinary tract infections</td>
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<tr>
<td></td>
<td>♦ Decubitus ulcers</td>
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<tr>
<td></td>
<td>♦ Malnutrition</td>
</tr>
<tr>
<td></td>
<td>♦ Contractures</td>
</tr>
<tr>
<td></td>
<td>♦ Stiff joints</td>
</tr>
</tbody>
</table>