Northwest Community EMS System

Continuing Education: Stroke

August, 2011

Susan Wood, RN, EMT-P and Jen Dyer, RN, BS

Epidemiology

A stroke is a sudden, catastrophic event causing a focal neurologic impairment due to interruption of cerebral blood flow, most often caused by occlusion or rupture of an artery that supplies a specific region of the brain. *A stroke is no accident!* To indicate the urgency for immediate emergency care, the terminology used to describe stroke has changed from CVA to *brain attack*.

Incidence

In the US, someone has a stroke every 40 seconds, and someone dies of one every 3-4 minutes. Approximately 795,000 people suffer new or recurrent strokes each year (ASA, 2009), from which 143,000 die, leaving 300,000 survivors disabled. About 4.8 million stroke survivors are alive today. The incidence per population subset doubles each decade after 55.

Morbidity and mortality

From 1995 to 2005, the annual stroke death rate fell 29.7%, and the actual number of stroke deaths declined 13.5%. (CDC, 2010). While death estimates are down by 32%-53% due to prevention, early detection and better management, stroke continues to be the 3rd leading cause of death in all developed nations, only preceded by heart disease and cancer (ASA, 2009). The 30-day death rate is 40-84% after cerebral hemorrhage, and 15-33% after cerebral infarction. In a study of persons > 65 years of age, the 30-day fatality rate for all strokes was 12.6%. For ischemic strokes only, it was 8.1%, and for hemorrhagic strokes, it was 44.6% (AHA, 2009). Stroke is the leading cause of adult disability such as paralysis, loss of speech and memory loss, and the leading diagnosis for transfers from hospital to long-term care.

Cost

The costs are staggering. In 2009, the cost for medical and disability related to stroke was $68.9 billion.

Demographics of stroke

While stroke kills and disables people of all ages, both sexes and all races, prevalence, death and disability rates are higher among African-Americans than Hispanics and Caucasians.

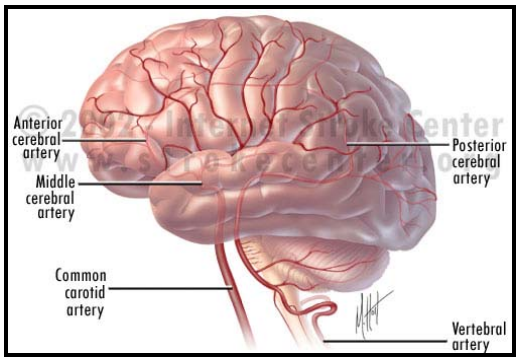
Age of patient

Approximately 50% of strokes that occur in younger patients are hemorrhagic. Only 20-25% are hemorrhagic in older patients.

Gender differences

60% of strokes occur in men. Women are more likely to seriously underestimate their risk of having a stroke. Strokes in women are more likely to be fatal. In 2006, 6 of every 10 deaths due to stroke were in women (CDC, 2010). The AHA reports 96,000 deaths in women and 62,000 deaths in males per year. Women are often older, are more physically frail, or have other health problems that impair recovery.

Vascular supply to the brain



Vascular supply to the brain is unique. There are two systems designed so that interruption in any part will not cause significant loss of flow to tissues served by the healthy circulation. The two systems are called, simply, the anterior and posterior systems. The carotid system serves the anterior part of the brain (frontal, temporal, parietal lobes); the vertebrobasilar system serves the posterior brain (brainstem, medulla, pons, midbrain, cerebellum, occipital lobe, and some parts of the temporal lobe). Both circulations serve the cerebral hemispheres. The Circle of Willis joins the two systems before they enter the brain. When either the posterior or the anterior circulation becomes occluded, collateral circulation may occur through this structure.

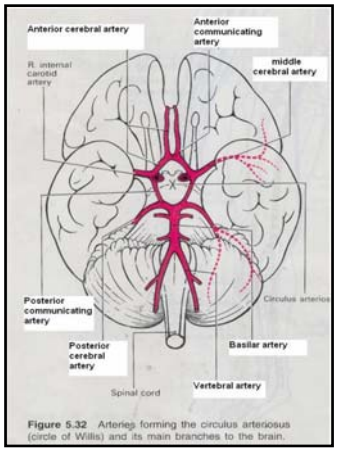
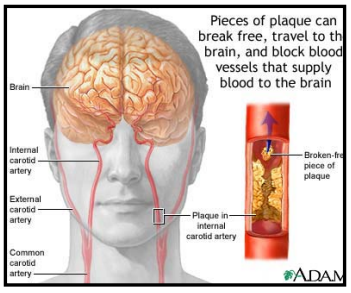


Figure 5.32 Arteries forming the circulus arteriosus (circle of Willis) and its main branches to the brain.

Classifications of stroke – 2 main types

Ischemic stroke **(75%-85%)** are caused by a blood vessel occlusion due to a clot, plaque or embolus.

Thrombotic



Central thrombus: Large-vessel atherothrombotic disease causes 14% of ischemic strokes. Clots form in turbulent flow areas of a large artery, or plaque suddenly obstructs flow, depriving the brain of vital oxygen and nutrients. The clot may partially or completely block the flow of blood.

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Strokes involving carotid artery branches are called **anterior circulation strokes**. They usually involve the cerebral hemispheres. **Posterior strokes:** Involve the vertebral basilar artery distribution.

Small-vessel atherothrombotic or lipohyalinotic disease (*lacunar stroke*): Narrowing or blockage of a smaller penetrating artery accounts for approx. 27% of strokes. Both large and small vessel types cause ↓ tissue perfusion leading to neurotransmitter failure, anaerobic metabolism, cerebral anoxia, and cerebral edema.

Risk factors for ischemic stroke: Smoking, HTN, ↑ cholesterol, diabetes, obesity, & alcohol/drug abuse. Cerebral thrombi commonly occur in patients over age 50 and the risk usually increases with age. These patients may benefit from aggressive reduction of risk factors.

Characteristics: Thrombotic Stroke	
Prodromal warning	Common
Onset during sleep	Sometimes
Development	Gradual
Quick reversal	Possible
Bloody CSF	No
Coma	Rare
Decreased consciousness	Mild
Headache	Mild
Hypertension	Common
Nuchal Rigidity	No
Vomiting	Rare

Embolic stroke

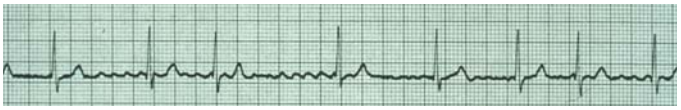
Incidence: 59% of ischemic strokes

Definition: Partial or complete blockage in a cerebral artery from embolic material which is generally composed of cholesterol, plaque, blood, air, or tumor tissue that arose elsewhere in the body (usually in the heart) and migrated to the brain.

S&S: These strokes often occur suddenly, without warning. Symptoms may fluctuate due to the continuing movement of the embolic matter within the blood vessel.

Risk factors for embolic stroke

- Pre-existing heart disease: mitral stenosis, valvular vegetation, defective valve prostheses.
- Mural thrombi (most common cause) due to acute MI, cardiomyopathy
- Dysrhythmia / atrial fibrillation



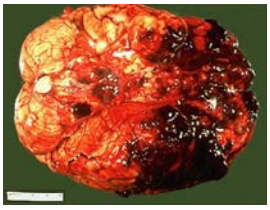
- Cardiac chamber lesions (left ventricular aneurysm)
- Intracardiac defects with paradoxic embolism (LV and left atrial thrombus with or without mitral stenosis)
- Atheromatous lesions of the ascending aorta; 30-70% stenosis of the internal carotid artery, or a clotting abnormality that promotes thrombosis. These patients may also benefit from reduction of risk factors.

Characteristics: Embolic Stroke	
Prodromal warning	No
Onset during sleep	Rare
Development	Sudden
Quick reversal	Possible
Bloody CSF	Rare
Coma	Rare
Decreased consciousness	Mild
Headache	Mild
Hypertension	Possible
Nuchal Rigidity	No
Vomiting	Rare

Hemorrhagic - 15-20% of all strokes

Most unfavorable type of stroke.

Incidence: Affects 37,000-52,000 people/yr in US. Most common in the elderly, middle aged, African Americans, Asian (particularly Japanese), and those with HTN.



Onset is abrupt. Signs & symptoms depend on the size and location of the hemorrhage, and worsen as more brain tissue is affected.

Two forms

Aneurysms, arteriovenous (AV) malformations (5%) and other vascular hematomas may bleed, producing a **subarachnoid hemorrhage** (SAH) around the brain and spinal cord. Especially prevalent in 35-65 yr olds. SAH accounts for about 7% of all strokes, afflicting approximately 30,000 Americans annually.

Characteristics: SAH	
Prodromal warning	Rare
Onset during sleep	Rare
Development	Sudden
Quick reversal	Possible
Bloody CSF	Yes
Coma	Common
Decreased consciousness	Moderate
Headache	Severe
Hypertension	Common
Nuchal Rigidity	Yes
Vomiting	Sometimes

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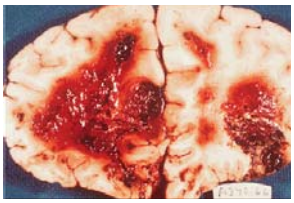
Spontaneous intracerebral hemorrhage (ICH)

Small, deeply penetrating arteries into brain tissue (parenchyma) are susceptible to loss of elasticity w/ HTN and are easily ruptured. ICH most commonly occurs in the cerebral lobes, periventricular white matter or basal ganglia (40-60%), thalamus (20%), the pons, and the cerebellum. Acute neurologic deterioration results from early hematoma growth, peri-hematoma injury, and obstructive hydrocephalus. Delayed deterioration is usually due to edema.



The expanding mass of blood can project 2-3 cm into the brain tissue and can grow to the size of a golf ball, plum or larger. This expansion can be caused by continued bleeding, blood-brain barrier breakdown, or formation of a local coagulopathic state (Badjatia & Rosland, 2005). The mass causes pressure on cerebral tissues and nerves leading to death of neurons.

The hematoma also has the potential to disturb normal intracranial dynamics causing a sudden rise in intracranial pressures, tissue compression, displacement, and herniation from mass effect (Presciutti, 2007). Cerebral spasm contributes to further ischemia.

Characteristics: ICH	
Prodromal warning	No
Onset during sleep	Rare
Development	Gradual or sudden
Quick reversal	No
Bloody CSF	Common
Coma	Common
Decreased consciousness	Severe
Headache	Severe
Hypertension	Not always
Nuchal Rigidity	Sometimes
Vomiting	Sometimes

ICH risk factors

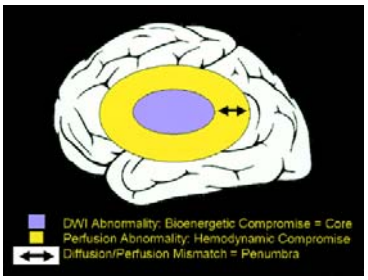
- Uncontrolled hypertension (primary & most easily modifiable risk factor) (Mayer & Rincon, 2005)
- Advancing age
- Cerebral amyloid angiopathy
- Neoplasms (tumors) with fragile blood vessels
- Trauma
- Vascular anomalies such as AV malformations and aneurysms
- Coagulation disorders; sickle cell disease, hemophilia
- Collagen-vascular disease
- Venous thrombosis
- Drugs: Especially anticoagulants, amphetamines (vasculitis), cocaine, oral contraceptives (See SAMPLE hx for details).
- Excessive alcohol intake
- Septic emboli, infective endocarditis, infected valve prosthesis

Disruption to cerebral blood flow (CBF) in stroke

For brain cells to function normally, they must have a constant supply of glucose and oxygen. Interruption in the supply results in an environment of inadequate energy supply (ischemia), forcing the cell to resort to an alternate method of energy production (anaerobic metabolism). The degree of tissue damage is dependent on the duration, severity, and location of the ischemia.

Humans can withstand 5-10 minutes of complete occlusion before brain damage is irreversible. Not all strokes involve complete occlusion of flow. Anaerobic metabolism produces byproducts that are toxic to the cells. Cell membranes become compromised and stop functioning, and the resulting tissue damage causes fluids to accumulate (swell). Because the cranium (skull) is a rigid container, the space taken up by the fluid creates pressure on the cells, as they "give" to accommodate the fluid collection. Nerve structures and vasculature are compressed, and blood flow is restricted. The result is ischemia, eventual irreversible cell damage, and cell death. Early edema (minutes to hours) is due to failure of the sodium and calcium channel pumps in the cells. Sodium accumulates in the cells (intracellular edema), so fluid flows in in an attempt to maintain osmotic equilibrium. As a result, free radicals are released, causing cell damage or death. Over the next hours and days, fluid begins leaking out of the damaged cells, causing increasing extracellular fluid volume.

There are two major zones of injury in stroke. The core *ischemic zone* represents an area of severe ischemia (less than 10-25% normal flow). It is comprised of necrotic neurons and supporting tissue. Core necrosis cells will probably die no matter what is done.



The ischemic ***penumbra*** is a rim of mildly to moderately ischemic tissue between normally perfused tissue and the infarct (core ischemic zone). Cerebral blood flow at critically low levels results in electrical silence of the affected cells (e.g. manifested as paralysis). Collateral blood flow to the ischemic penumbra may be sufficient to preserve brain, but not enough to sustain normal function until perfusion is restored. This tissue, while ischemic, may still be viable for several hours due to supply from collateral arteries. The extent of penumbra is determined by the number and patency of the collateral arteries. This is the target area for pharmacologic therapy and interventional procedures.

Blood pressure in patients in the early stages of stroke is often elevated. This elevated pressure maintains cerebral perfusion and abrupt decreases in BP could enlarge the area of the initial infarction.

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During stroke, autoregulation of BP is defective, so circulation to the brain is dependent on mean arterial pressure (MAP). Why blood pressure rises is still under debate. The "Cushing reflex" has been suggested as the mediator of elevated BP in stroke. However, this reflex usually occurs in situations where intracranial pressure is increased. Typically, elevations in BP occur very early in stroke, when intracranial pressure has not yet increased. A more likely explanation is the presence of a relationship between the vascular occlusion and elevated BP. A study reported in the journal *Stroke* demonstrated that reestablishment of flow in the previously occluded vessel was associated with spontaneous reduction in BP. (Stroke: 2005; 36:268).

Hyperoxia

The combination of poor perfusion and hypoxemia will worsen and extend ischemic brain injury and has been associated with worse outcomes from stroke. The AHA/ASA recommend that patients with possible stroke should receive supplemental oxygen if O_2 saturation falls below 94% (Class I, LOE C) (Circulation 2010: Part 11: Adult Stroke p. S821). Management of ischemia from stroke is complicated by a paradoxical injury associated with re-oxygenation of previously ischemic cells due to production of oxygen free radicals, which are more prominent in environments of excessive oxygen levels. (Neurosci. 2007, 27(5):1129-38). For this reason, caregivers must be especially vigilant in monitoring oxygenation and judicious in giving supplemental O_2.

TIA

TIA is defined as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia ***without radiologic evidence of infarction*** (CT, MRI, MRA). This differs with the past concept of stroke as simply stroke symptoms that resolved in a number of hours, leaving no deficits. The time limit has been removed due to advances in neuroimaging, which have demonstrated ischemic lesions on up to 20% of CT's and MRI's of patients whose symptoms resolved (Stroke: 2009). Therefore, TIA diagnosis is based on *tissue* rather than time. Symptoms of TIA usually resolve within 60 minutes, but an acute stroke can be of any duration, even "silent" (no symptoms). TIA is considered a strong indicator of possible future major stroke, and the AHA/ASA has outlined aggressive guidelines for management of those patients with TIA.

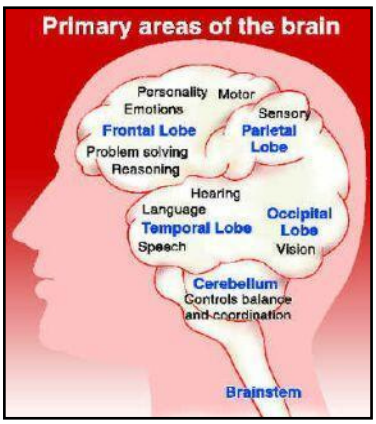
Guidelines suggest that ***TIA should be approached with the same urgency as a confirmed stroke***. Any patient whose potential stroke symptoms have subsided should be *strongly* encouraged to accept transport for prompt medical evaluation. The ASA recommends hospitalization for patients experiencing initial TIA within the past 24-48 hours, during which they should receive a thorough medical evaluation, including same day radiologic imaging, for risk factors and management of those risk factors. For those patients who are not hospitalized, the ASA urges rapid access to urgent (within 12 hours) assessment and diagnostic evaluations for suspected TIA.

Some alarming TIA statistics:

- 200,000 - 500,000 TIA's/yr (AHA 2009)
- 15% of strokes are preceded by a TIA that is *ignored* by > 50% of pts as symptoms resolve spontaneously
- After TIA, 12% experience stroke **w/in next 30 days**
- Following TIA, 3-17% have stroke within 90 days
- 25% of TIA patients die within a year

Characteristics of stroke

The S&S that develop after a stroke or TIA depend on the areas of the brain that are affected and the extent of tissue injury. The areas most commonly affected are the motor and speech centers, vision, and sensory centers.



Barriers to successful stroke care

Symptoms of stroke can be subtle. Less than half of patients call for medical help within 24 hours of symptom onset and a third wait over 2 days! Few patients arrive at the hospital early enough to be eligible for thrombolytic therapy. According to *Stroke* online (May 5, 2011), most healthy people said they would call 911 if they thought they were having a stroke, but research numbers show that few actually do.

One study canvassed 100 inpatients diagnosed with acute stroke in Washington DC. Only 12% had called 911 immediately upon the onset of symptoms. Unfortunately, failure to promptly call 911 results in delayed presentation to definitive care, less opportunity for acute stroke treatment, and, ultimately, greater death from stroke (Medscape Medical News, May 16, 2011).

The AHA and ASA endeavor to increase public awareness of stroke risk factors and stroke warning signs. Studies indicate there is much yet to do. One study revealed that only 38% of those polled in 14 states were aware of stroke warning signs and would first call 911 for anyone they thought might be having a stroke (MMWR, 2008: 57:481-5). Only 60% of actual stroke patients polled between 20003 and 2007 could identify even one stroke risk factor. In a study involving 250,000+ ischemic stroke patients, only 28.3 % arrived in less than 60 minutes from exact last known well times.

The two things that most heavily influenced the prompt arrival were greater stroke deficit severity and **arrival by ambulance versus coming by private vehicle** (Stroke 2010; 41; 1431-1439).

Other barriers to timely care occur once the patient reaches the hospital. These include nursing triage, placement in treatment area, and recognition of significant symptoms. Delay in getting the CT scan, receiving scan results, time spent in consultation with specialty service, time obtaining consent, and prep time for either tPA or interventional care also extend time from symptom onset treatment, and ultimately, negatively impact chances for the best outcome.

Stroke in special populations

Women

Stroke kills twice as many women as breast cancer every year (National Stroke Association, 2010). The NSA has identified stroke symptoms unique to women as compared to the general population. These include sudden onset of: face and limb pain, hiccups, nausea, and general weakness. A 2009 study by the University of Michigan found that women often experience nontraditional symptoms. The most frequent presentations in females with stroke were AMS, meaning confusion, disorientation, and loss of consciousness (Study: Wayne State Univ., Detroit, Michigan).

Young people and children

In a report issued on Feb. 25, 2011, analysts from the CDC noted an increasing risk for stroke among young people (ages 5 to 44), including children and teens. Speculative contributors include risk factors such as obesity, hypertension, and diabetes, which contribute to cardiovascular disease and atherosclerosis as the patient ages. Young adults with stroke are frequently misdiagnosed. The majority of cases are mistaken as inner ear disorders; other misdiagnoses included alcohol intoxication, migraine, and vertigo. Many are later diagnosed with brain stem/cerebellar stroke (ASA International Conference, 2009).

The National Stroke Association reports that on average it takes 12 to 24 hours for adults to get to the hospital after recognizing stroke symptoms. Investigators found that children who had ischemic strokes arrived at a hospital approximately 21 hours after symptom onset (range was six to 48 hours). Arrival times for children with hemorrhagic stroke were shorter, at approximately 12 hours (range 4 – 72 hours). Stroke symptoms in children are often attributed to more common problems – migraine, seizures, or encephalitis (Medscape: Feb. 24, 2011). Contributing to the delay in presentation and diagnosis are the infrequency of stroke in children, the lack of a predominant cause or risk factor for stroke in children, and lack of awareness.

The most common risk factors include sickle cell disease, congenital or acquired heart disease, infections involving the nervous system, and trauma. Signs and symptoms of stroke for children mirror those for adults, in addition to loss of emotional control and changes in mood, cognitive changes or problems with memory, judgment, and problem-solving, behavior changes or personality changes, and improper language or actions. The most common sign of stroke in infants is seizure. Evaluation for stroke is further complicated in the very young and the non-verbal pediatric patient (Nursing Spectrum, March 2011).

Stroke Care Evolution: The Stroke Chain of Survival

Until the 1990s, care of the stroke patient was largely supportive, focusing on prevention and treatment of respiratory and cardiovascular complications. No specific therapy was available to alter the course and extent of the evolving stroke. Therefore, limited emphasis was placed on rapid transport or intervention. Fibrinolytic therapy now offers healthcare providers an opportunity to possibly limit the extent of neurological damage and improve outcomes in ischemic stroke patients.

In 1997, the American Stroke Association (ASA) created their grassroots initiative, Operation Stroke, to educate about stroke warning signs and to increase healthcare emergency and hospital personnel awareness and preparation relative to stroke identification and management. It also focuses on strengthening the chain of survival to reduce disability and death.

In May 2009, the Illinois General Assembly passed House Bill 2244, allowing for the creation of stroke systems of care in Illinois. The law identifies hospitals capable of providing emergent stroke care, and directs EMS providers to transport those patients with symptoms of possible stroke to these facilities. The Illinois Hospital Association (IHA) is working with the American Heart Association (AHA), the ASA, and IDPH to create preliminary regulations to guide the creation of this planned legislation.

Official designation of stroke centers will be awarded by IDPH once regulations are established (IHA, 2009). This year (2011), in accordance with Illinois law, Regional Stroke Committees were established within each of the Illinois EMS regions, composed of representatives from each hospital and major discipline (including EMS) that cares for stroke patients. As work progresses within these committees, regional representatives will disperse the information and guidelines to the EMS systems.

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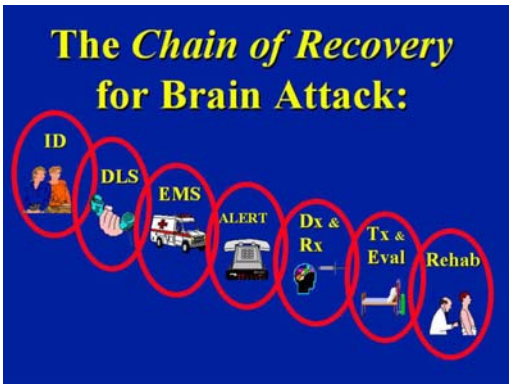
The ASA and AHA have included EMS as an integral part of the stroke system of care. Critical actions identified by the AHA/ASA, according to the Adult Suspected Stroke algorithm include:

- Support ABC's: give oxygen if needed
- Perform prehospital stroke assessment
- Establish time of symptom onset (last normal)
- Triage to stroke center
- Alert hospital
- Check glucose if possible



A team approach to stroke management starts with dispatch and EMS and continues at the hospital – preferably a stroke center. To qualify for fibrinolytic therapy for ischemic stroke, symptoms must be detected within 4 ½ hours, to halt or reverse the cascade of brain damage caused by stroke. This requires rapid identification of stroke S&S and prompt activation of the EMS system.

Only 50% of patients experiencing a stroke arrive at the ED by EMS. Of those that do, their quick arrival represents an advantage if they qualify for time-sensitive treatment. Since 85% of strokes occur at home, this early recognition often depends on the patient, family members, or bystanders. Dispatchers must recognize stroke symptoms. They must classify these patients as they do those with AMI and severe trauma, and provide the callers with pre-arrival instructions if appropriate.

EMS personnel must rapidly and appropriately assess for stroke using a validated stroke tool (Cincinnati Stroke Screen in Region IX). EMS must be aware of the treatment possibilities for early management of stroke and recognize the need for short scene times. EMS' goal is rapid assessment, stabilization, and support of the patient with suspected stroke and transport to an appropriate facility as quickly and safely as possible.

Non-urgent assessments and interventions should be accomplished enroute as much as possible in the interest of minimizing time from symptom onset to definitive care. The NWC EMSS scene time target is 10 minutes or less. If scene time is extended, supporting documentation should be included in the PCR.

The receiving facility should be notified ASAP of the possible stroke patient's pending arrival if onset of symptoms is less than 4½ hours and the patient has at least one abnormal finding that suggests stroke.

Prehospital stroke assessment and care

History (SAMPLE)

Clinical signs and symptoms of acute stroke vary widely and often fluctuate based on the type and location of focal ischemia. Deterioration or improvement can be detected by repeated neuro exams.

S: Common symptoms/chief complaints

Headache of unknown cause: Sudden, excruciating pain, described as the worst of their life or like something exploding in their heads (*thunderclap headache*) is the classic sign of SAH. Look also for decreased level of consciousness, vomiting without nausea, photophobia, and neck rigidity within first 12 hrs. None of these symptoms are common in ischemic strokes.

Weakness, heaviness, or paralysis involving the face or limb(s) alone or in combination (most commonly of the hand/arm and face).

Numbness of face, arms, legs on one side of the body

Speech disturbances

Vertigo: Sense of room spinning around the patient that persists at rest; may be accompanied by nystagmus. Differentiate from light-headedness. Vertigo is a common symptom in a number of nonvascular diseases. Therefore, at least one other S&S of stroke should also be present.

Ataxia: Loss of balance, unsteadiness, stumbling gait, or incoordination on one side

Sudden **confusion**, trouble understanding

Sudden **trouble seeing**

Seizures

A: Allergies

M: Medications: HTN/CV/High cholesterol drugs?

Drugs that place pts at risk for stroke

- Cocaine: Constricts vessels, reducing brain blood flow. Repeated exposure can lead to blood flow deficits that persist long after cocaine use has ended, causing permanent damage. Activates platelets promoting thrombosis; heavy users (6-20 X/wk) have elevated levels of C-reactive protein which is associated with risk of plaque rupture.
- Anticoagulants: ASA, warfarin (Coumadin), Plavix, Pradaxa
- Oral contraceptives: Women who smoke and take BCPs are 22 times more likely to have a stroke.
- **Amphetamines**: Increase BP; inhibit naturally occurring brain factors that help protect against neuronal damage following stroke or trauma.

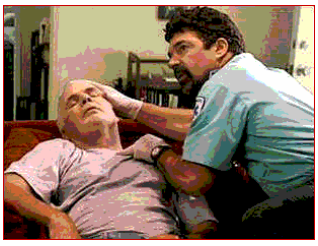
- **PCP** (Phencyclidine also known as angel dust, ozone, wack, rocket fuel) – Increases BP
- **Phenylpropanolamine** (over-the-counter "diet pills" and cold and flu pills) increase incidence of stroke in the under 40 population.
- **Ecstasy** (MDMA): deaths related to massive cerebral hemorrhage.

P: Past Medical History: Ask about **risk factors** as they may provide clues to the type of stroke:

Previous stroke/TIA

Intracranial or intraspinal surgery, serious head trauma, known AV malformation, tumor or aneurysm.

Active internal bleeding or acute trauma



Cardiovascular disease: HTN, (> 140/90); atherosclerosis: Damages arteries over time and can cause them to rupture. HTN causes a 6 fold increased risk.

Dysrhythmias (AF) can cause blood to pool in the heart and form mural thrombi [clots that form on the chamber walls] that can break loose and travel to brain.

AMI; rheumatic heart disease: Within 5 yrs after an AMI, 8% of men & 11% of women will have a stroke.

Metabolic diseases/diabetes: Accelerates atherosclerosis. Proper control of hyperglycemia can reduce risk of microvascular complications and reduce the overall risk of stroke.

Abnormal blood lipid levels: Total cholesterol > 200 increases risk. LDL (*lethal* cholesterol) > 130 and HDL (*healthy* cholesterol) < 35 also risk factors.

Obesity: Strains entire cardiovascular system; likely to have high cholesterol, HTN, DM

Blood disorders

- Protein S & C deficiency
- Sickle cell disease: occludes small arteries
- Polycythemia (abnormally large number of RBCs)
- Hemophilia: risk factor (hemorrhagic stroke) regardless of age

Stroke risk factors

Risk factors for stroke	
Modifiable	**Non-modifiable**
High blood pressure	Age
Smoking	Heredity
TIA	Ethnicity
Heart disease	Gender
Diabetes	Prior history of stroke, AMI
Carotid or peripheral artery disease	
Increased RBCs	
Coagulopathies	
Sickle cell disease	
High cholesterol (esp. high LDL, triglycerides)	
Inactivity/obesity	
Alcohol/drug abuse	

L: **Last oral intake**

E: **Events surrounding this incident** (HPI)

E: **Time of onset**: Many ischemic strokes occur at night or early morning when the patient is sleeping or just beginning to wake. They may be unaware of when S&S first started. Stress the importance of this information with patient & family members.

E: **Events surrounding this incident** (HPI)

Onset/progression: abrupt or gradual? Hemorrhagic and embolic are generally abrupt in onset; ischemic may have a "stuttering" progressive onset.

Age of patient: Age > 55 is the most important non-modifiable risk factor.

Hereditary: Family history of CV disease or stroke.

Social history: Excessive alcohol consumption has been linked to HTN. Anti-clotting properties increase the risk of ICH. >2 drinks/day may ↑ risk by 50% as compared to non-drinkers.

Smoking: Accelerates rate of atherosclerosis progression by 50%, particularly in those with diabetes & HTN. Nicotine ↑ HR, BP, and platelet aggregation. Carbon monoxide displaces O_2 from hemoglobin, interfering with O_2 delivery to tissues. Smoking has been confirmed to be the *most important modifiable risk factor* for SAH.

Primary assessment/resuscitative interventions

Airway obstruction can be a major problem, especially if the patient loses consciousness. Paralysis of the muscles of the throat, tongue, or mouth can lead to partial/complete upper airway obstruction. Saliva may pool in the throat and be aspirated. Aspiration of secretions or gastric contents is a serious complication, associated with considerable morbidity and mortality.

Protect **airway** using appropriate positioning and adjuncts.

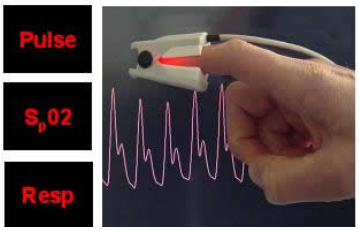
Positioning: Maintain head and neck in neutral alignment to optimize perfusion and venous outflow - may need to use lateral immobilization devices; do not use pillows. If SBP >110 and hemodynamically stable, **elevate head** of stretcher 10°-15° to help decrease risk of aspiration and reduce intracranial pressure. Do not flex knees or hips.

Seizure/vomiting precautions; suction prn. Use gentle technique, as trauma to tissues may later lead to significant bleeding if the patient is treated with fibrinolytics.

If GCS ≤ 8 (must do an accurate GCS!) **assess need for DAI.**

Give lidocaine 1.5 mg/kg as a premed in stroke patients with preserved airway reflexes. See SOPs for actions, indications, etc.

Assess **respirations/ventilatory status/gas exchange (SpO_2 & $EtCO_2$)** q. 15 min depending on degree of stability. Note length and frequency of apnea. Abnormal ventilatory patterns are especially prevalent in comatose patients and usually reflect serious brain dysfunction. Assess for hypoxia. Hypoxia and hypercarbia result from hypoventilation and can contribute to ↑ ICP and cardiac and respiratory instability.



If SpO_2 < 94% or O_2 sat unknown: O_2 by NC or NRM based on patient presentation. Assist ventilations @ 10-12 BPM as needed w/ 15 L O_2/BVM (if advanced airway, 8-10 BPM.)

Hyperoxia causes free radical formation and cerebral vasoconstriction

Assess **cardiovascular status/perfusion adequacy:** Continuously monitor ECG rhythm. Cardiac dysrhythmias may have caused the stroke or may be a side effect of brain injury. Life-threatening dysrhythmias are a potential early complication, particularly of intracranial hemorrhages. A 12 Lead ECG is indicated if the pt has S&S indicative of ACS, and should always be done following syncope.

Bradycardia may indicate hypoxia or ↑ ICP. Bradycardia w/ AMS and a BP >90 SHOULD NOT be treated with transcutaneous pacing or atropine.

Tachycardia may indicate hypoxia/hypercarbia. 15% of stroke pts have **A-Fib.**

If onset of stroke S&S within last 4.5 hours, **DO NOT start an IV on scene** unless need for DAI, seizure activity or hypoglycemia are present. Consider if IN drugs are an option. Hydrate as necessary to maintain CPP but do not give large fluid boluses unless hypotensive. Stroke pts experience cerebral edema as a result of hypoxic tissue damage. Unnecessary/excess fluids (IV) will worsen this edema and increase damage to brain tissue.

Disability/mental status exam: Assess and score the level of consciousness using the **GCS.**

Observe for confusion, agitation. Determine if patient is agitated/consolable or agitated/inconsolable. A patient with ischemic stroke may be drowsy, but rarely unconscious unless the infarct is large.

A patient with a depressed level of consciousness or coma is more likely to have experienced severe brain injury with increased ICP, usually due to a hemorrhagic stroke. Coma is the result of damage to both cerebral hemispheres or to the brain stem.

The most critical patient is comatose or becomes flaccid on the affected side.

AMS: Consider drug OD, sepsis, or severe metabolic abnormalities. Obtain capillary **blood glucose level** on all pts with AMS.

- Hypoglycemia = life threat
- Hyperglycemia is harmful

Maintain euglycemia (AHA, 2010). If < 70 or low reading: **DEXTROSE / Glucagon** per hypoglycemia SOP. Do not give glucose without evidence of hypoglycemia. If glucose > 200 may be less likely to recover from SAH or stroke.

If convulsive activity is present:

Midazolam in 2 mg increments every 30-60 sec IVP/IO (0.2 mg/kg IN) **up to 10 mg IVP/IN/IO/IM** to stop seizures. May repeat to a total of 20 mg if BP > 90.

Short-term use of prophylactic antiepileptic drugs soon after ICH may decrease the risk of early seizures in patients with lobar hemorrhage (IIb, C)(AHA, 2007), but should only be given in response to OLMC orders prior to hospital exam/CT.

Observe and record the following if seizure activity is present:

- Presence of aura
- Focus of origin: one limb or whole body
- Simple or complex (conscious or loss of consciousness)
- Partial/generalized
- Progression and duration of seizure activity
- Eye deviation prior to or during seizure
- Abnormal behaviors (lip smacking)
- Incontinence or oral trauma
- Duration and degree of postictal confusion

Environmental controls

Provide comfort and reassurance; establish means of communicating with aphasic patients. Limit activity; do not allow patient to walk. Elevate side rails and use appropriate stretcher straps. Protect paralyzed limbs.

Secondary assessment

Vital signs

Compare rate and quality of peripheral and carotid pulses (do not rely on SpO_2 monitor for rate alone). Assess for evidence of carotid **bruits** if skilled in that procedure.
Accurate BP readings are essential. Take once manually in each arm before hooking up the automated cuff. BP may be elevated due to underlying HTN, a stress reaction to the stroke, increased ICP, or a physiological response to decreased brain perfusion.

BP's often return to normal without antihypertensive treatment. Lowering the BP can be harmful, as it can decrease the cerebral perfusion pressure (CPP) and worsen the stroke.

HTN needs treatment only for those with SBP > 220 mmHg or DPB >120 mmHg on three repeated measurements made at 15 minute intervals. Prehospital treatment of hypertension without signs of acute MI or left ventricular failure/CHF is not recommended.

Monitor temperature carefully: Elevated temp (>37.5° C) is often found in patients following ICH. Hyperthermia can worsen damage to cerebral tissue and increase O_2 demand.

The full neuro exam of a stroke patient includes 6 key elements:

- ***Time of symptom onset***
- ***Level of consciousness***
- ***Stroke screen***
- Severity of stroke
- Type of stroke
- Location of stroke

The first three of these can be obtained by EMS personnel.

Perform a neuro exam using a Quick Stroke Scale

Performing an extensive neurological examination outside of the hospital is impractical because it delays transport of the patient to an appropriate facility.

To conduct an out-of-hospital evaluation, use a validated tool such as the **Cincinnati Prehospital Stroke Scale** (CSS) or the **Los Angeles Prehospital Stroke Screen** (LAPSS).

A simple way to remember the assessment components of the **CSS** is to cover your *XXX*.

Arm drift
Speech
Smile

Arm drift

Weakness, clumsiness, heaviness, or dysfunction of the arm or hand is tested by **arm drift**. Motor deficits may involve one half of the body (hemiparesis or paralysis) or all four limbs.

Ask patient to lift both arms to 45°-90°, palms up, with eyes closed, in front of them and hold for 5-10 seconds. Score from no drift to no movement.

Normal: Both arms move the same or do not drift at all

Abnormal

- Drift of one arm
- Can't resist gravity
- No effort against gravity
- No movement

Motor losses in stroke: Arm flexors stronger than extensors; leg extensors stronger than flexors.

Speech disturbances

This is often one of the first signs in both types of stroke and is characteristic of a **left** hemisphere lesion of any size due to involvement of Brocca's area and the frontal lobe motor strip. Ask the patient to repeat a simple phrase or sentence. Evaluate speech pacing and clarity by having them repeat a simple phrases like, "You can't teach an old dog new tricks," or have them sing, "Happy Birthday to You".

Dysfunctions

Dysarthria: Imperfect articulation of speech or mispronouncement of words due to disturbances of muscular control so words sound slurred (as is common with intoxicated persons).

Motor aphasia: Inability to express oneself, trouble with word retrieval or selecting correct words, using inappropriate words, or inability to speak.

Receptive aphasia: Inability to understand written, spoken, or tactile speech symbols.

Don't assume lack of speech or response means coma (when scoring with the GCS). Patient may have receptive or expressive aphasia! Design effective alternative means of communicating w/ aphasic patients.

Score Dysarthria from normal to severe

- Normal: Responds/can speak, and is able to repeat the sentence clearly and easily.
- Abnormal: Cannot answer at all, cannot repeat sentence correctly, slurs words, mumbles, or is difficult to understand.

Smile asymmetry (facial palsy): The face can be involved on one side and the limbs on the other. Ask the patient to smile, grimace, show their teeth and tightly close their eyelids.

Normal: Both sides of face move equally well

Abnormal: If patient can wrinkle both sides of forehead but has lower facial weakness with an asymmetric smile or one eye does not close as tightly as the other, suspect a stroke. (Patients w/ Bell's Palsy will *not* be able to wrinkle the forehead on the affected side.)

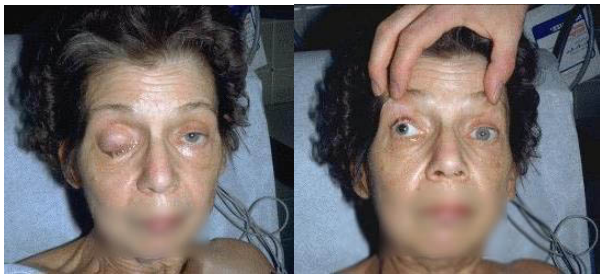
Assess for vision disturbances

- **Monocular blindness**: Sudden, painless vision deficit in one eye - may involve loss of all or part of the vision. Described as a curtain dropping, fog, grayout or blackout of vision. The involved eye is on the same side as the diseased artery.
- **Diplopia**: Seeing two images. Eyes don't track together or are not on the same plane so patient no longer sees "in stereo". May have a sense of bouncing and moving visual images. Cover one eye. Double vision should resolve unless dislocated lens, retinal detachment, or high brainstem lesion.
- Blurred or indistinct vision in one or both eyes.
- Loss of the ability to see in one or more **fields of vision** in both eyes. Involved visual field is opposite the side of the diseased artery.

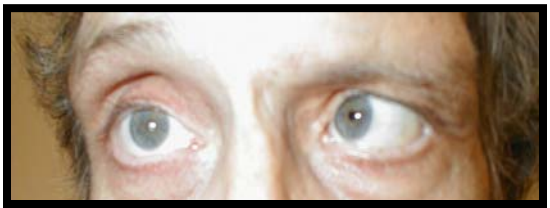
  Testing one eye at a time, introduce a visual stimulus in all visual field quadrants. Score from no loss to bilateral hemianopsia.

Pupil size, shape, equality, symmetry, reactivity

- Unilateral pupil dilation is a sign of brainstem dysfunction due to herniation
- Fixed, dilated pupil, ptosis, and eye pulled to the ear in a patient c/o intense headache suggests ruptured aneurysm with pressure on CN III
- Oval pupils w/ hippus suggest ↑ ICP



Extraocular movements (EOMs): Ocular palsy, dysconjugate gaze



- Have patient follow your finger with their eyes as you move it fully to the left and to the right of his or her visual field. In a right hemispheric stroke, the patient may have difficulty moving their eyes across the midline of the visual field to the left (ocular palsy) and may try to move their entire head instead. In a left hemispheric stroke, the patient may have trouble understanding and executing your verbal instructions.
- Score best gaze as normal, gaze palsy, or fixed deviation. Gaze deviations help localize a stroke.

If in the right hemisphere, the eyes will look toward the unaffected side of the body and the affected side of the brain (right).

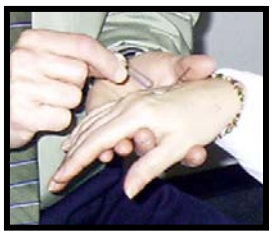
Inspect the back of the throat. Look for deviation of the uvula and listen for hoarseness when the patient speaks which can indicate dysfunction of cranial nerves IX & X (glossopharyngeal and vagus).

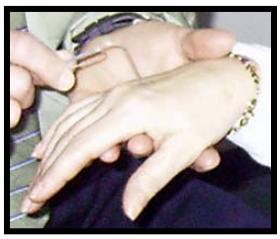
Ask a conscious patient to stick out their tongue and look for deviation. Indicates dysfunction of CN XII (hypoglossal). (*Pearl…Palate away; tongue towards the affected side*

Also assess for head trauma, infection, meningeal irritation, hearing loss, and vertigo (room spinning).

Sensory deficits

- Assess for sensory loss, tingling, or abnormal sensation involving the face, arms, or legs; alone or in combination. Usually occurs simultaneously and on the same side as the weakness. The involved body parts are opposite the side of the diseased artery.
- Test for superficial touch and pain sensation using a sharp vs. dull object (broken cotton swab or paperclip); compare side to side. Score sensory integrity from normal/no loss to severe to total





sensory loss/not aware of touch to face, arm, or leg.

Alterations in sensory function

- Hypalgesia: Decreased sensation
- Analgesia: No sensation
- Hyperalgesia: All touch is painful
- Paresthesia: Alteration in sensation

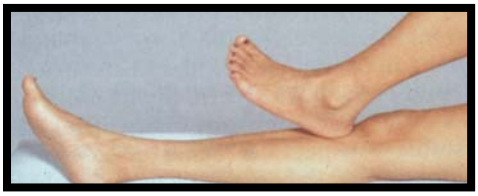
- **Extinction and inattention:** Observe visual, tactile, or auditory inattention to one side or the other. The patient may not recognize his or her own hand and will orient to only one side.

Touch on both sides of body at once. Score from no abnormality to profound hemi-inattention.

Observe for cerebellar dysfunction: The cerebellum is responsible for posture, equilibrium, coordination, fine skilled movements, balance, gait, stride, and arm swing. Assess for incoordination of fine motor movements on one side of the body.

Have the patient rapidly bring individual fingers to thumb in rapid succession (rapid alternating movements) or use one finger to touch their nose and then touch your fingertip (light on an object).

To test the lower extremities, have them run the heel of one foot down the shin of the opposite leg.



Document the presence of ataxia, incoordination, imbalance and/or inability to perform rapid alternating movements or fine skilled movements.

Note any inappropriate affect, such as excessive laughing or crying.

Differential diagnosis of stroke: Consider other causes of presenting signs

- Cervical/head trauma
- Infections (TB, fungal, herpes simplex encephalitis, meningitis)
- Hypertensive encephalopathy
- Intracranial mass
  - Tumors (primary and secondary)
  - Epidural/subdural hematomas
- Seizure disorder with persistent neurological signs (Todd's paralysis.) Tonic clonic seizures can occur simultaneous with hemorrhagic stroke.
- Migraine headaches with persistent neurological signs can mimic SAH but usually appear gradually and classically present with an aura. Basilar artery (brainstem) and hemiplegic migraines (often associated with aphasia if the left hemisphere is involved) may be difficult to differentiate from stroke until angiography is performed. Hemiplegic migraine is more common in young women (20-40) as opposed to older patients (40-70), who are more likely to have atherosclerosis and thrombotic, ischemic strokes.
- Metabolic disturbances
  - Hyperglycemia (DKA/HHNS)
  - Hypoglycemia
  - Post-cardiac arrest ischemia
  - Toxicological cause
  - Endocrine disorder (myxedema)
  - Uremia
- Psychiatric syndromes
- Shock and CNS hypoperfusion
- Cardiac abnormalities (dysrhythmia, AMI, prolapse mitral valve)
- Degenerative disorder (Alzheimer's)
- Cranial arteritis
- Hemiplegia in young women (think of the pill)
- Hemiplegia in postpartum women: think venous infarct

Primary Stroke Centers vs. Comprehensive Stroke Centers (PSC, CSS)

IDPH has collaborated with the JCAHO, Joint Commission on Accreditation of Health Care Organizations, to award hospitals with the designation of "Primary Stroke Center" (PSC). JCAHO is a nationally recognized, independent accrediting agency, which exists to improve health care by evaluating health care organizations and programs. Their endorsement is recognized as a symbol to the public that a particular organization has a strong commitment to providing safe, effective, high quality health care. Standards for designation are based on guidelines established by the Brain Attack Coalition (BAC) and the American Heart and American Stroke Associations (AHA, ASA).

Primary Stroke Centers

As of Jan. 2011, more than 800 hospitals were designated PSC's in 11 states, Illinois being one of them. Stroke center certification recognizes centers that make "exceptional efforts to foster better outcomes for stroke care" (The Joint Commission). Among the required services of PSC's by Illinois law:

- Written transfer agreements w/ hospitals that have neurosurgical expertise
- Administration of thrombolytic therapy
- Administration of medical therapies according to nationally recognized, evidence-based stroke guidelines
- 24/7 Brain imaging available
- 24/7 Lab services (coagulation studies)

Comprehensive Stroke Centers

IDPH and JCAHO have not yet begun assessing hospitals for CSC certification, thus, the NWC EMSS has not implemented changes in transport patterns at the writing of this education. In preparation for the process, the AHA/ASA has issued quality care directives for CSS's. Of interest to EMS, key areas of emphasis for CSS certification are expected to include:

- Personnel w/ expertise in neurosurgery & vascular neurology
- Advanced neuroimaging capabilities (MRI,

cerebral angiography)
- Surgical and endovascular services (clipping and coiling of intracranial aneurysms, carotid endarterectomy, intra-arterial thrombolytic therapy

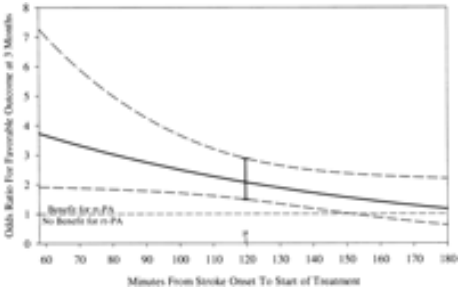
Transport decision

Transport decision: ***TIME SENSITIVE PATIENT*:** Once stroke is suspected, minimize scene time to 10 minutes or less. Transport to the nearest appropriate hospital, providing as much pre-arrival notice as possible.

In the near future, which hospital the patient is taken to *may* depend on time from symptom onset/last known normal. NWC EMSS SOP instructs EMS to take the patient to the nearest *Primary* Stroke Center (PSC) if symptom onset/last know normal is within the last 4.5 hours. Within that time frame, a patient with stroke symptoms is a candidate for IV tPA, provided he or she t meets all remaining criteria for tPA. Symptom onset *beyond* 4.5 hours leaves the patient outside the window of time to safely qualify for IV tPA. Therefore, the appropriate destination for the patient becomes a *Comprehensive* Stroke Center (CSC), ***IF*** that facility is reachable within 30 minutes by ground travel. Delivering the patient to a CSC provides them the opportunity to be evaluated to receive other invasive therapies for stroke, since they no longer qualify to receive IV tPA. For patients whose symptoms began *more than* 8 hours prior to EMS' arrival, transport to the nearest or requested hospital is acceptable.

These transport guidelines will be effective once region hospitals are designated by the Joint Commission and IDPH. *Until then*, EMS should continue present patterns of transport for patients suspected of having a stroke.

Tissue Plasminogen Activator (tPA)



TIME IS BRAIN! In cardiac arrest, brain must be resuscitated in minutes to prevent irreversible brain damage (entire brain is not perfused). In most forms of stroke, the patient has *focal ischemia*. The core area must receive resuscitation within minutes, but a large area around the core (*ischemic penumbra*) does not die for hours. Each hour of ischemia increases the degree of irreversible brain damage.

IV tPA is approved by the FDA for the treatment of acute ischemic stroke. The goal of therapy for ischemic stroke is to safely maximize patient functional recovery to pre-stroke baseline, by rapidly reperfusing the ischemic penumbra. The FDA has approved tPA use within 3 hours from onset of symptoms. However, the AHA/ASA supports the longer time window for treatment of 4.5 hours in carefully selected patients. Studies have revealed that time from onset to treatment with tPA is directly related to outcomes in ischemic stroke. The sooner tPA is given, the greater the benefit (Target: Stroke Campaign Manual, AHA, 2010). Beyond the approved and recommended time windows for tPA use, risk of complications rises. The most common of complications is intracranial hemorrhage.

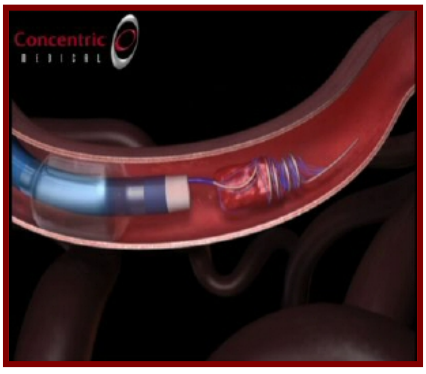
tPA is no different from other medications in that it has potential adverse effects, which makes it essential that a careful consideration is given to exclusion criteria, risks and benefits for use in each individual patient. The most major of its complications is symptomatic intracranial hemorrhage, which occurs at a rate of roughly 5.2%. Other complications include orolingual angioedema (allergic reaction), acute hypotension, and systemic bleeding. (Circulation 2010: Part 11: Adult Stroke).

Interventional stroke care

Patients who do not qualify for IV tPA may still be candidates for other, advanced therapies for stroke.

Intra-arterial tPA delivers fibrinolytic medication directly to the thrombus. It can be used for qualified patients up to 6 hours from onset of symptoms (Class I). This therapy can only be done in specialty centers by specially trained interventionalists.

Clot retrieval devices (MERCI Thrombectomy System and Penumbra Thrombectomy System) actually remove the clot from the occluded vessel. Studies have shown that when clot retrieval is performed within eight hours of symptom onset, 60-68% of patients achieved post-procedure revascularization. Patients eligible for clot retrieval are those with recent ischemic stroke, who can receive treatment within at least 12 (preferably 3-6) hours of the onset of stroke. Those patients with uncontrolled high blood pressure, internal bleeding, seizures, blood clotting problems, or those on anticoagulants are not candidates for this procedure. Complications from clot retrieval procedures include hemorrhage, internal bleeding, death and



recurrence of stroke.

Complications & Consequences of stroke
Stroke affects many body systems. Some stroke sequelae are permanent, some are not. Many impact the patient's ability to be independent, safe and productive. Conditions that patients may experience after stroke:

- Balance problems
- Spasticity(causing stiff and awkward movement)
- Contractures (permanent contraction of a muscle due to spasm or paralysis
- Speech problems
- Dysphagia (problems swallowing), resulting in poor nutrition, aspiration pneumonia
- Depression
- Visual deficits (w/ associated safety concerns)
- Sensory deficits (ex: numbness), w/ associated safety concerns
- Perceptual problems: impaired judgment, lack of awareness of unsafe environment, impaired understanding of surroundings
- Cognitive deficits: learning of new information, problem solving, impulsiveness, decreased attention, distractibility
- Memory problems
- Communication problems
- Personality changes
- Behavior changes/problems: inability to inhibit inappropriate behavior, inability to "read" context of body language
- Apathy

Patient outcomes

Patients are scored with regards to functional outcome following stroke. Below is the scale used**.**

Stroke outcome scale

- 5 Patient bedridden and incontinent
- 3 Needs assistance and is not able to work
- 1 Relatively normal neurologically
- 0 Asymptomatic

The EMS subcommittee of Operation Stroke has prepared educational materials for EMS personnel and the general public. For more information: Contact the American Stroke Association on their web site **at www.Strokeassociation.org** or call 630-789-9222.

The future of stroke in America
A CDC report on showing percentages of the U.S. population w/ specific stroke risks (below) presents an alarming suggestion of potential for stroke occurrence in the future. In 2003, about 37% of adults in America reported having two or more risk factors listed below. The table below shows percentages of US adults with stroke risk factors, for the years 2005 to 2006. (www.dcd.gov/stroke/facts.htm).

Risk Factor	Percentage
Inactivity	39.5 %
Obesity	33.9 %
High Blood Pressure	30.5 %
Cigarette Smoking	20.8 %
High Cholesterol	15.6 %
Diabetes	10.1 %

Advances and treatments on the horizon

An article appeared in the journal *Neurology* in May 2011, reporting that approximately 14% of ischemic strokes occur while the patients sleep (**"wake-up stroke"**). That represents 58,000 ischemic strokes that occurred in 2005. Dr. Jason Mackey, of the department of Neurology at the University of Cincinnati, notes, "It's important to get an accurate time line of stroke symptoms, given that the only proven treatment for ischemic stroke, tPA, must be given within 4 ½ hours of the last time a patient was known to be well." Wake-up strokes are an area of great interest. Currently there is a European trial called WAKE UP, which seeks to determine whether MRI is useful, safe and accurate in determining whether patients with "wake-up stroke" actually fall within the time window of 4 ½ hours. Advanced imaging would be used to determine the presence of brain tissue that is not completely dead/still salvageable to help determine which of these patients might still benefit from tPA.

Another reference to changes in treatment guided by tissue status rather than time comes from Andrew Bivard, in an article published in *Expert Review of Cardiovascular Therapy*: "The logical change that needs to occur in patient selection for reperfusion therapy in stroke is a move from the current time-based method to a tissue-based imaging regime." Efforts are underway to making this change a reality, in the form of:

- clinical trials
- research on the technical aspects of imaging to determine a standard approach that can be applied world-wide
- implementation of infrastructure (i.e.: stroke systems of care) to ensure patients w/ acute stroke receive transport to facilities that provide

the care each patient requires

Another exciting area of study is the use of **hypothermia** for patients with moderate to severe stroke. EuroHYP-1, an international clinical trial, will test the efficacy of therapeutic cooling on 15,000 ischemic (only) stroke patients, including patients who get tPA and those who do not. Cooling must begin within 6 hours of symptom onset and will be e continued for 24 ours. Participants will be evaluated for outcome at 3 months post stroke.