



## Northwest Community EMSS Paramedic Education Program

# Stroke

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### **OBJECTIVES:**

Upon completion of the class and independent study, each participant will do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Define stroke and explain why they are now called brain attacks rather than a CVA.
2. Cite the incidence and epidemiology of stroke.
3. Differentiate the two main etiologies of stroke into ischemic and hemorrhagic.
4. Compare and contrast the three types of ischemic stroke.
5. Recognize the risk factors and presenting S&S of cerebral aneurysm, subarachnoid hemorrhage and acute intracerebral hemorrhage.
6. Compare and contrast the modifiable and non-modifiable risk factors for stroke.
7. Sequence the impact of disrupted cerebral blood flow and how the brain becomes injured in stroke explaining the importance of salvaging the penumbra.
8. Discuss each link in the stroke chain of survival and explain why these pts are time sensitive.
9. Identify and provide rationale for the EMS resources that must be educated/prepared to identify and/or treat stroke.
10. Explain the five goals of stroke management.
11. Explain the diagnostic importance of information to be obtained in a SAMPLE history for stroke.
12. Sequence the appropriate methods to secure an airway in a patient with a possible stroke.
13. Explain the indications and contraindications (dangers of hyperoxia) for providing supplemental O2 to pts experiencing a possible stroke.
14. Discuss the indications and contraindications for vascular access and IV fluid administration in pts experiencing a possible stroke.
15. Explain the circumstances under which dextrose and midazolam are indicated and contraindicated for pts experiencing a possible stroke.
16. Describe preferred positioning and environmental controls that should be instituted to protect a pt with stroke.
17. Explain the anticipated changes in vital signs in pts with stroke and the thresholds for treatment.
18. Compare and contrast the components, timing, and predictive value of the Cincinnati Prehospital Stroke Scale (CPSS), the Los Angeles Stroke Scale and the Miami Emergency Neurologic Deficit (MEND) exam.
19. Explain the components of a complete pt assessment for stroke.
20. Identify diseases/conditions that must be considered in the differential diagnosis of stroke.
21. Recognize alternate S&S of stroke that may be present with or without alterations to the quick stroke scales or exams.
22. Explain common complications of stroke and their implications for EMS care.
23. State the factors that contribute to poor outcomes in stroke.
24. Differentiate a TIA from a stroke.
25. Explain the importance of calling a stroke alert to the appropriate receiving stroke center.

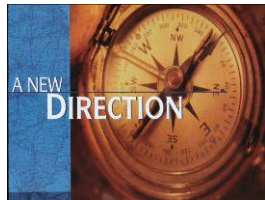
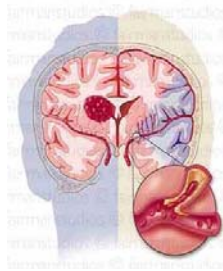


# NWC EMSS Paramedic Education Program

## STROKE – IT'S NO ACCIDENT

### Epidemiology

**Definition:** A stroke is a sudden, catastrophic event causing a focal neurologic impairment due to interruption of cerebral blood flow, most often caused by an 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 was changed years ago from CVA (Cerebrovascular accident) to **brain attack**.

**Do NOT use the term CVA anymore**

**Incidence:** In the U.S., someone has a stroke every 40 seconds and someone dies of one every 3-4 minutes. Approximately 750,000 people suffer new or recurrent strokes each year. About 4.8 million stroke survivors are alive today. The incidence per population subset doubles each decade after 55.

### Morbidity and mortality

On average, someone dies of a stroke every 4 minutes. In 2009, 1 of every 19 deaths in the US was from stroke (*Circulation*, 2013). Due to public education, early detection, and better management, stroke deaths have declined, and rates of appropriate fibrinolytic therapy have increased. Between 1999 and 2009, the annual death rate from stroke fell 36.9%. Stroke now ranks as the *fourth* leading cause of death in the US, down from third place, which it held for decades. Only heart disease, cancer and chronic lower respiratory disease cause more deaths than stroke. Strides in prevention, however, have been offset by growth in the aging population (putting more people at risk).



30-day death rate

- 40-84% after cerebral hemorrhage
- 15-33% after cerebral infarction

Stroke is the leading cause of serious long-term adult disability such as paralysis, loss of speech and memory loss. It is the leading diagnosis for transfers from hospital to long-term care.

**Costs:** The costs are staggering. According to the American Stroke Association, stroke-related medical costs and disability cost Americans about \$73.7 billion in 2010 (AHA, 2013).

### EMS and Stroke

The AHA and the American Stroke Association (ASA) have partnered together to guide stroke care, with the overall goal of minimizing acute brain injury and maximizing patient recovery. The AHA/ASA cites the efforts of EMS systems with recent successes in stroke prevention and improvements in the early hours of stroke care. A quote from the 2010 AHA Guidelines for CPR and ECC states, "The integration of EMS into regional stroke models is crucial for improvement of patient outcomes" (*Circulation*, Nov 2010). So critical is the role EMS and EMS systems play in stroke systems of care, that the AHA/ASA has issued a policy statement and guidelines specific to EMS.

Central to the success of EMS stroke care is commitment to **time-sensitive delivery of care**. The "Implementation Strategies for Emergency Medical Services within Stroke Systems of Care" policy statement provides specific parameters that serve as a measure for EMSS quality, including:

- Dispatch at highest level of care in shortest time possible
- Time elapsed from receipt of call to dispatch is < 90 sec
- Time elapsed from receipt of call to EMS arrival is < 8 min
- Dispatch time is < 1 minute
- Time elapsed from call receipt to EMS unit enroute is < 1 minute
- On-scene time is < 15 min. in the absence of complications (NWC EMSS time goal is 10 min. or less)
- Transport time equivalent to those of AMI or trauma

The primary goals of EMS for the patient with stroke symptoms are timely response, rapid (neuro) evaluation, appropriate stabilization, and expeditious transport to a stroke center according to current guidelines.

### 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:** The most common type of stroke is due to cerebral thrombosis formed from plaques in atherosclerotic vessels. Since atherosclerosis is usually associated with a long history of blood vessel disease, most strokes occur in persons over the age of 65.

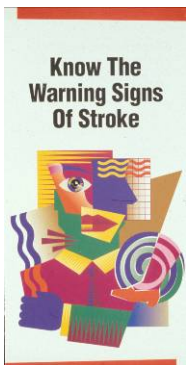


## Stroke

Approximately 50% of strokes in younger patients are hemorrhagic (ruptured congenital aneurysms or AV malformation); only 20%-25% are hemorrhagic in older patients.

**Gender differences:** Forty percent of strokes occur in men (stroke.org, 2013). Women are more likely to dismiss symptoms and to seriously underestimate their risk of having a stroke. Strokes in women are more likely to be fatal. 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.

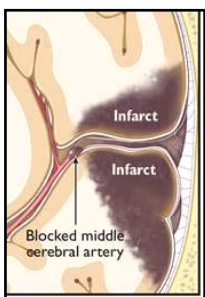
Until the 1990s, care of the stroke patient was largely supportive, focusing on prevention and treatment of respiratory and cardiovascular complications. No specific therapy was available to alter the course and extent of the evolving stroke. Therefore, limited emphasis was placed on rapid transport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



The American Stroke Association, created in 1998, undertook its grassroots initiative, **Operation Stroke**, to educate everyone about stroke warning signs and to increase healthcare emergency and hospital personnel awareness and preparation relative to stroke identification and management. It has since evolved to include a vigorous public awareness campaign and guidelines for all providers across the stroke care

continuum, including emergency dispatch and EMS care providers. Originally intending to strengthen the chain of survival and reduce disability and death, the ASA now assigns itself the "broad mission of prevention, treatment and rehabilitation of stroke" (Stroke, 2010).

## Classifications of stroke – 2 main types (leaks and plugs)



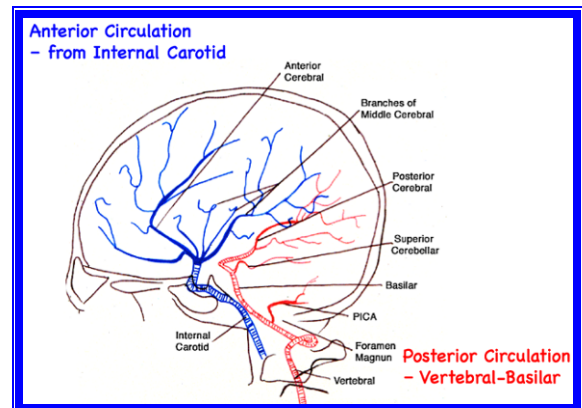
The brain depends on a constant supply of blood. Any blockage threatens this crucial flow. To preserve a continuous blood supply in the event of a blockage, a "system of redundancy" is created by the main arteries: the anterior, middle, and posterior cerebral arteries. They interconnect via communicating arteries to form the structure known as

the Circle of Willis. This unique circulation allows perfusion to continue in the event of blockage in either the anterior or the posterior circulation.

**Ischemic strokes (87%) are caused by a blood vessel occlusion due to a clot, plaque or embolus.** AHA statistics released in 2013 report that for 30-40% of ischemic strokes, no cause is found (*Circulation*, 2013). This type is called *cryptogenic* stroke. The availability of highly effective treatment for prevention of repeat stroke makes identification of etiology extremely important.

## Thrombotic Stroke

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



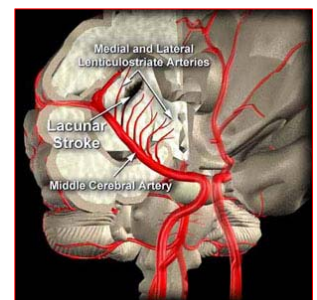
Strokes involving carotid artery branches are called **anterior circulation strokes**. They usually involve the cerebral hemispheres (frontal lobe, anterior parietal lobes, anterior temporal lobes).

**Posterior strokes:** Involve the vertebral basilar artery distribution. The posterior circulation serves crucial structures including the brainstem, cerebellum, and posterior parts of the cerebrum.

## Small-vessel atherothrombotic or lipohyalinotic disease (lacunar stroke):

Beyond the Circle of Willis, collateral circulation is limited, supplied only by vessels in the dura mater & the arachnoid membrane. Blockage or narrowing of a smaller penetrating artery accounts for approximately 27% of strokes. Both large and small vessel types cause reduced tissue perfusion leading to neurotransmitter failure, anaerobic glycolysis, cerebral anoxia, and cerebral edema.

**Risk factors for ischemic stroke:** 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.





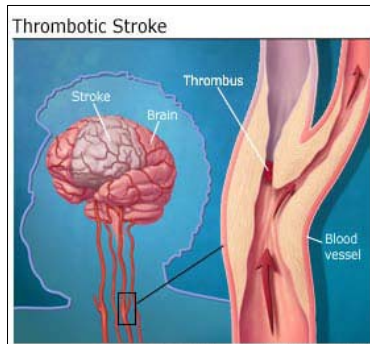
## Thrombotic Stroke

Statistics released in 2012 analyzing stroke mortality between 1990 and 2010, revealed that *in excess of 90%* of stroke deaths are caused by **modifiable** risk factors and are therefore **preventable!** (Lozano et al, 2012).

These include:

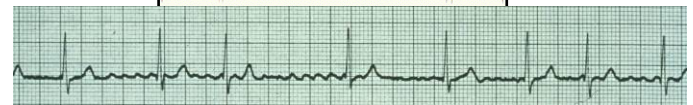
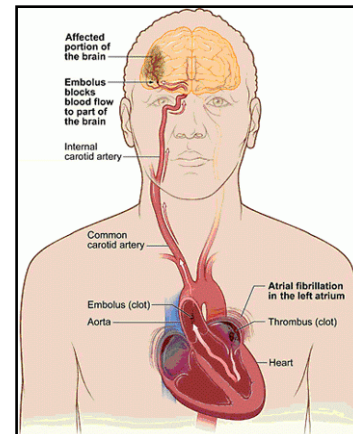
- HTN: ~77% have BP > 140/90 at their first stroke
- Diabetes Mellitus
- Disorders of heart rhythm: Atrial fibrillation, a powerful risk factor, is often asymptomatic and undetected. The older the pt, the greater the risk of stroke from AF (1.5% at ages 50-59, to 23.5% at ages 80-89).
- High blood cholesterol/lipids
- Smoking: Considered to be the most important modifiable risk factor, current smokers have 2-4 times the risk of stroke compared to non-smokers (or those who have quit for 10+ years).
- Physical inactivity: obesity Study results show that mod-. to vigorous-intensity exercise is associated with an overall 35% reduced risk of stroke.

(*Circulation*, Jan. 2013)



## Risk factors for embolic stroke

- Dysrhythmias (**AF** in patients > 60-65 years)



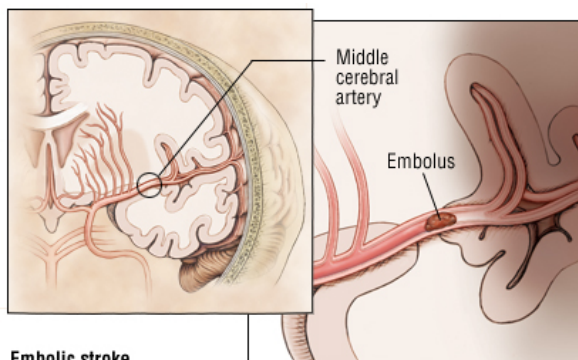
- Pre-existing heart disease: mitral stenosis, valvular vegetation, defective valve prostheses.
- Mural thrombi (most common cause) due to acute MI, cardiomyopathy
- Cardiac chamber lesions (left ventricular aneurysm intracardiac defects with paradoxical embolism (LV and left atrial thrombus with or 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.

## Embolic Stroke

**Definition:** Partial or complete blockage in a cerebral artery from embolic material which is generally composed of cholesterol (usually smaller than 500 micrograms), plaque (usually larger than 500 micrograms), blood, air, or tumor tissue that arose elsewhere in the body (usually in the heart) and migrated to the brain.

Incidence: 59%

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



## Hemorrhagic - 13% of all strokes

### Most unfavorable type of stroke

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

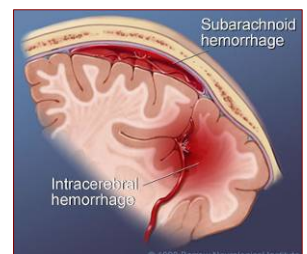
Onset is abrupt. S&S depend on the size and location of the hemorrhage and worsen as more brain tissue is affected.

### Two forms:

#### Subarachnoid Hemorrhage (SAH)

Aneurysms, arterio-venous (AV) malformations (5%) and other vascular hematomas may bleed, producing a (SAH) around the brain and spinal cord.

Especially prevalent in 35-65 yr olds, SAH accounts for about 3% of all strokes; afflicting ~30,000 Americans annually. SAH is associated with mortality rates approaching 45%, and significant morbidity among survivors. Twelve percent die before they can seek medical attention.



## Stroke

Conditions associated with risk for SAH include HTN, smoking, heavy alcohol use, use of sympathomimetic drugs and presence of familial genetic predisposition.

**Clinical presentation** of SAH is “one of the most distinctive in medicine” (ASA). The classic complaint of “the worse headache of my life” is present in about 80% of patients. About 20% experience a warning or “sentinel” headache between 2 and 8 weeks before the SAH event.

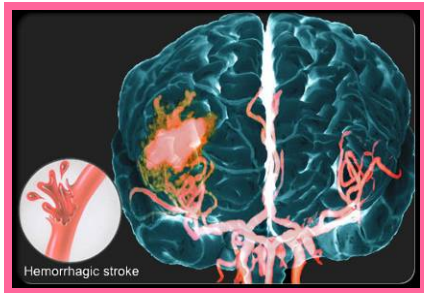
Headache w/ the warning bleed is usually milder than that of the SAH event, and may be accompanied by nausea & vomiting. SAH often occurs during physical exertion or stress. Associated S&S may include nausea &/or vomiting (77%), stiff neck (35%), brief loss of consciousness (53%), and focal neuro deficits. Twenty percent of SAH patients experience seizure(s) within the first 24 hrs after the event.

EMS is the first medical contact for about two thirds of SAH patients. Rapid assessment & transport with advanced notification of the ED is crucial for any patient presenting with more than one S&S for SAH, including headache, change in level of consciousness, or vomiting.

### Spontaneous intracerebral hemorrhage (ICH)

ICH accounts for 10% of all strokes. 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, perihematoma injury, and obstructive hydrocephalus. Delayed deterioration is usually due to edema and toxic effects of blood on the brain tissue.

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. 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. Cerebral spasm contributes to further ischemia.

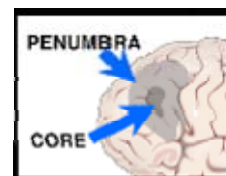
Rapid EMS assessment and transport are crucial because deterioration is common in the first few hours after ICH. Studies indicate that most patients with smaller ICH will survive if aggressive medical care is received. In excess of 20%, the patient's GCS will decrease  $\geq 2$  points in the period between EMS assessment and initial ED evaluation. Early deterioration without aggressive intervention is associated with a high rate of poor long term outcomes ( $> 75\%$  mortality). Advance notification of stroke patient arrival correlates with shortened time to evaluation and CT scan.

### ICH risk factors

- Uncontrolled HTN (primary & most easily modifiable risk factor)
- 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
- Excessive alcohol intake
- Septic emboli, infective endocarditis, infected valve prosthesis

### Disruption to cerebral blood flow (CBF) in stroke

Vascular occlusion or disruption results in decreased  $O_2$  and glucose delivery to the affected tissues and increased carbon dioxide and lactic acid accumulation. CBF is autoregulated over a wide range of blood pressures. When stroke occurs and the brain becomes ischemic, blood flow is regulated by “whatever head of pressure is pushing it” and small changes in BP and cardiac output can cause drastic alterations in cerebral blood flow.



Collateral circulation to the area of stroke occurs (ischemic penumbra) but is hard to predict by looking at the patient. Core necrosis cells will probably die no matter what is done. Collateral blood flow to the ischemic penumbra may be sufficient to preserve brain, but not enough to sustain normal function until perfusion is restored.

Brain only functions normally if blood flow is  $>20$  mL/100 g of brain tissue/minute. If blood flow becomes  $<12$ -20 mL/100 g/min, the brain becomes electrically silent (e.g. as manifested by paralysis) but not dead. Early

**Stroke**

management is critical to restore function.



## Stroke chain of survival



The **team approach** to stroke management **starts with EMS** and continues at a hospital within a stroke system of care capable of delivering acute stroke care in the ED and in a dedicated stroke unit.

### ASA Stroke Chain of Survival

<b>Detection *</b>	Patient/bystander (or EMS) recognition of stroke S&S
<b>Dispatch *</b>	<b>EMS activation; priority dispatch &amp; response</b>
<b>Delivery *</b>	<b>Prompt triage &amp; transport to most appropriate stroke hospital AND prehospital notification</b>
<b>Door</b>	Immed. ED triage to high-acuity area
<b>Data</b>	Prompt ED evaluation, stroke team activation, lab studies, brain imaging
<b>Decision</b>	Dx & determination of most appropriate therapy; discussion w/ pt and family
<b>Drug</b>	Admin. of appropriate drugs/interventions
<b>Disposition</b>	Timely admission to stroke unit, ICU, or transfer

\* denotes EMS role

### Detection

Eighty five percent of strokes occur at home. Early recognition depends on patient, family members, or other bystanders witnessing the event and recognizing the urgency of the symptoms. EDs must aim to diagnose ischemic stroke and begin fibrinolytic therapy within 60 minutes up to 4.5 hrs from onset of symptoms to halt or reverse the cascade of brain damage (salvage neurons) caused by the stroke.

### Lack of stroke awareness



Symptoms are often subtle. Many fail to seek help quickly enough to be eligible for fibrinolytic therapy. Data collected between 2001 and 2004 showed no improvement in symptom to ED arrival time; only one third arrive within 2 hours or less from time of symptom onset.

Patients must be taught to consider any inability to feel, walk, talk, communicate, use part of the body, or see to be a stroke until proven otherwise as strokes can be mild, moderate, or severe in nature.

EMS should be activated as soon as stroke S&S are recognized. Fifty percent of stroke patients arrive at the hospital via EMS. Those who use EMS, arrive faster, which is a major advantage for time-sensitive treatment.

### Emergency Medical Dispatch (EMD)

Dispatchers must recognize classic S & S and classify potential stroke patients in the same category as AMI and severe trauma. EMDs should give appropriate pre-arrival instructions.



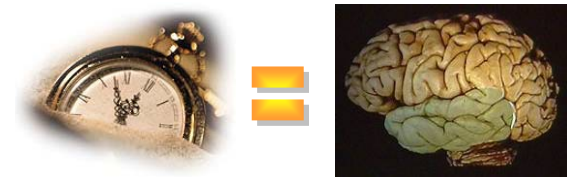
Dispatch for patients w/ stroke S&S should be at the highest level of care in the shortest time possible.

### Delivery

EMS personnel should rapidly and appropriately assess for stroke using a validated stroke tool like the Cincinnati Stroke Scale (CSS), the Los Angeles Prehospital Stroke Screen, or the Miami Emergency Neurological Deficit (M.E.N.D.) exam developed by University of Miami School of Medicine. Currently, Region IX (NWC EMSS) uses the CSS endorsed by The AHA.

The receiving hospital should be notified as soon as possible of impending arrival of a patient w/ stroke S&S. Data confirms that patients whose arrival was expected were more likely to receive fibrinolytic therapy.

### Time-sensitive patient



**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.

For every minute stroke is left untreated, an estimated 1.9 million neurons die. Each hour, in which treatment fails to occur, the brain loses as many neurons as it does in almost 3.6 years of normal aging (Miller, 2012).

EMS personnel must be aware of the possibilities for early stroke interventions at hospitals so they appreciate the **need for short scene times**. The goal is to rapidly assess, stabilize, and transport the patient with a suspected stroke to an appropriate facility as quickly and as safely as possible.

**Stroke**

**Expedite transport:** In the interest of saving time, some EMS actions can be accomplished enroute. Limit assessments/care on scene to those that are urgently needed or clearly indicated by the pt's presentation.

*The NWC EMSS has a scene time target of 10 minutes or less for patients with suspected stroke.*

If scene time is longer than 10 minutes, include information in your narrative to support it!

**Hospital stroke alert:** Notify the nearest System hospital for OLMC ASAP if onset of symptoms is <4.5 hours. Inform them that you are transporting a patient with possible acute stroke. They should either prepare for the patient's arrival or call report to the receiving hospital.

**Door, Data and Decision – Hospitals own this**

Upon arrival at the hospital, the patient should be rapidly placed for immediate evaluation by a physician (goal ≤ 10 minutes).

**Time barriers at the hospital:**

- Time to treatment area
- Time to physician assessment
- Time to CT (goal ≤ 25 min.)
- Time for CT interpretation (goal ≤ 45 min.)
- Time for consultation and decision
- Time for consent
- Time for preparation

These barriers to rapid treatment of stroke patients should be removed (prioritized CTs, location of fibrinolytics).

**Drug:** Tissue plasminogen activator (tPA) is approved by the FDA for the treatment of acute ischemic stroke but **must** be given within 4.5 hours from the onset of symptoms. Ideally, tPA should be administered ("door to drug") in less than 60 minutes after ED arrival. Evidence confirms that the sooner tPA is administered, the more optimal the results.

**Disposition:** Outcomes are best when patients are admitted directly to and receive care in a dedicated stroke unit, where recovery and rehab can begin immediately. Hospitals without full stroke care capabilities should have official transfer agreements with hospitals offering a full complement of stroke care, and patients should be transferred as soon as reasonably possible.

**Physical examination – See SOPs**

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

Explain assessments and care to the patient, even if they cannot respond or communicate.

**History (SAMPLE)****S: Common symptoms/chief complaints (OPQRST)**

**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). **Differentiate generalized from localized (focal) weakness.**

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

**Speech:** Disturbances in word retrieval, word substitution, or inability to speak clearly

**Vertigo:** Sense of room spinning around the patient that persists at rest; may be accompanied by nystagmus. Differentiate from light-headedness. 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

**Vision** deficits

**Seizures**

**A: Allergies**

**M: Medications:** HTN/CV/High cholesterol drugs; anticoagulants/antiplatelet drugs

**Addl. 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.
- **Oral contraceptives:** Women who smoke and take BCPs are 22 X 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:

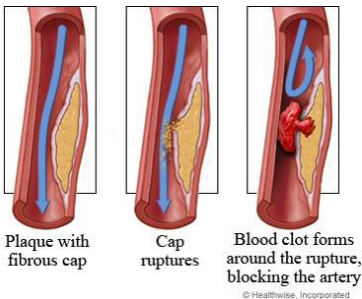
**Previous stroke/TIA:** The definition of Transient Ischemic Attack has changed. TIA is no longer defined by the duration of symptoms but rather the absence of imaging abnormalities in the presence of focal neuro symptoms. Advanced imaging techniques now identify small ischemic brain lesions in up to 20% of pts whose symptoms subsided and who were diagnosed w/ TIA (based on cessation or short duration of stroke symptoms within 24 hours). Notably, 15% of strokes are preceded by a TIA that is ignored by over half of pts whose symptoms resolve spontaneously. Prevalence increases with age.

Urgent follow-up is now recommended for TIA pts, to begin anticoagulation and management of risk factors.

#### Following TIA:

- 12% experience stroke within next 30 days
- 3-17% have stroke within 90 days
- 25% die within a year

**Cardiovascular disease:** HTN, (> 140/90); atherosclerosis:



Damages arteries over time and can cause them to rupture. HTN causes a 6 fold increased risk and 35% prevalence of stroke.

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.

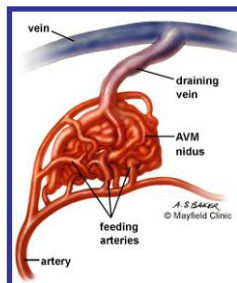
Intracranial or intraspinal surgery

Serious head trauma

Known AV malformation

Tumor or aneurysm

Active internal bleeding or acute trauma



**Metabolic diseases/ diabetes:** Accelerates atherosclerosis. Control of hyperglycemia can reduce risk of microvascular complications and 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 for hemorrhagic stroke regardless of age

**L:** Last oral intake

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

**TIME OF ONSET:** Many ischemic strokes occur at night or early morning when the patient is sleeping or just beginning to wake (wake up stroke), so symptom onset may be difficult or impossible to determine. Stress the importance of this info with pt & family members. Establishing this time may allow treatment for pts who might otherwise be classified as "onset time unknown". This may require "creative questioning" on the part of EMS. Inquire about pre- or post-stroke cell phone use (note call time stamp), or use TV program times to establish onset time. For patients with "wake-up" stroke, attempt to identify a time when they were ambulatory to the bathroom or kitchen or when they were awakened during the sleep period.

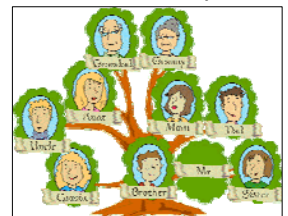
**Use best effort to determine the exact time of symptom onset as accurately as possible.**

If witnesses/family members cannot accompany the patient to the ED, EMS should **obtain their contact information and a call back number** in the event hospital personnel wish to obtain other pertinent information after the patient arrives at the hospital.

**Age of pt:** Age > 55 is the most important *unmodifiable* risk factor.

**Hereditary:** Family history of CV disease or stroke.

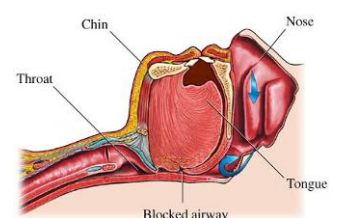
**Social history:** Excessive alcohol consumption has been linked to HTN. Anti-clotting properties increase the risk of ICH. More than 2 drinks per day may ↑ risk by 50% as compared to non-drinkers.



**Smoking:** Accelerates rate of atherosclerosis progression by 50%, particularly in those with diabetes & HTN. Nicotine ↑ HR, BP, and platelet aggregation. Carbon monoxide displaces O<sub>2</sub> from hemoglobin, interfering with O<sub>2</sub> 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



**Stroke**

aspirated.

Aspiration of secretions or gastric contents is a serious complication, associated with considerable morbidity and mortality.

Protect **airway** using appropriate positioning and adjuncts. Do not allow the patient to eat or drink anything until swallowing ability has been assessed by ED staff.

**Positioning:** Patient positioning can affect O<sub>2</sub> saturation, CPP, and intracranial pressure (ICP). New recommendations advise if a pt is not hypoxic and is able to tolerate lying flat, to place them in a supine position. Patients at risk for airway compromise or suspected ↑ ICP should be positioned with their head elevated 15° to 30° (**Stroke**, 2013). Maintain head and neck in neutral alignment to optimize perfusion and venous outflow. May need to use lateral immobilization devices; do not use pillows. Do not flex knees or hips to avoid elevation in ICP.

**Seizure/vomiting precautions; suction prn.** Use gentle technique, as trauma to tissues may later lead to significant bleeding if patient is treated with fibrinolytics. Use caution to avoid vagal stimulation during suctioning, as increases in ICP may worsen the effects of stroke.

**Airway:** The airway should be opened initially with BLS manual maneuvers and secured with nasopharyngeal and/or oral pharyngeal airways as indicated.

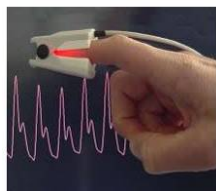
**If GCS ≤ 8** (must do an accurate GCS!) **assess need for DAI.** If airway is patent and O<sub>2</sub> sats can be maintained at 94%, BLS interventions may be sufficient. Give lidocaine 1.5 mg/kg as a DAI premed in patients with suspected stroke who have preserved airway reflexes. See SOPs for actions, indications, etc.

Assess **respirations/ventilatory status/gas exchange (SpO<sub>2</sub> & EtCO<sub>2</sub>)** at least q. 15 min.

Respiratory compromise may occur from aspiration, upper airway obstruction, hypoventilation, and pneumonia. Note length and frequency of apnea. Abnormal ventilatory patterns are especially prevalent in comatose patients and usually reflect serious brain dysfunction. Hypoxia and hypercarbia result from hypoventilation and can contribute to ↑ ICP and cardiac and respiratory instability. Patients with history of cardiac and/or pulmonary disease are at ↑ risk for hypoxia.

**If SpO<sub>2</sub> < 94%:** O<sub>2</sub> by NC or NRM based on patient presentation and response. Assist ventilations at 10-12 BPM as needed w/ 15 L O<sub>2</sub>/BVM prn.

**Hyperoxia causes free radical formation and cerebral vasoconstriction and should be avoided.**



The AHA does not recommend use of O<sub>2</sub> in a patient with suspected stroke who has adequate oxygenation and pulse ox readings to help prevent reperfusion release of free oxygen radicals.

### Assess **cardiovascular status/ perfusion adequacy**



Continuously monitor ECG rhythm. 12 L ECG should be deferred to the ED unless the rhythm is abnormal or the pt is exhibiting cardiac related S&S (AHA, 2010). 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.

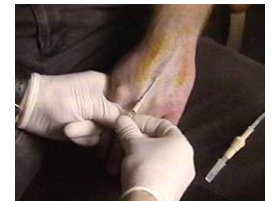
**Bradycardia** may indicate hypoxia or ↑ ICP. Bradycardia w/ AMS and a SBP >90 SHOULD NOT be treated with atropine, dopamine, or transcutaneous pacing.

**Tachycardia** may indicate hypoxia/hypercarbia.

**A-Fib:** 15% of pts with stroke have A-fib that increases stroke risk 5-fold. These strokes are associated with higher likelihood of debilitation and or death. A majority of patients with A-fib are asymptomatic, necessitating more advanced evaluation than simple routine telemetry monitoring during hospitalization (Medscape, 2013).

**Vascular access: IVs by EMS may not be necessary. DO NOT start on scene** unless need for DAI, seizure activity or hypoglycemia are present. Consider if IN drugs are options.

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.



**Fluid loading is harmful in these pts and scene times can be shortened if IVs are deferred or started enroute**

**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

**Stroke**

hemispheres or to the brain stem.

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

**AMS:** Consider possible causes using the AEIOU-TIPs mnemonic in the SOP.

Obtain capillary **glucose level** on all pts with AMS.

- Hypoglycemia = life threat
- Hyperglycemia is harmful



Hypoglycemia may mimic stroke symptoms, especially in the elderly

If < 70 or low reading: **DEXTROSE 10%/ Glucagon** per hypoglycemia SOP. **Do not give glucose without evidence of hypoglycemia.** Glucose levels in excess of 200 are associated with poor outcomes. Animal studies demonstrated that hyperglycemia increased brain water content and edema related to vascular dysfunction (Stroke, 2013).

**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** titrated to pt response. If IV unable and IN contraindicated: IM dose 5-10 mg (0.1-0.2 mg/kg) max 10 mg single dose. May repeat to a total of 20 mg if SBP > 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/record the following if seizure activity 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

**The full neuro exam for a pt with stroke includes 6 key elements:**

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

First three can be obtained by EMS using the Cincinnati Prehospital Stroke Scale during the D portion of the

**primary assessment** in order to make a rapid transport decision. If the CSS is negative and/or stroke is suspected, EMS should complete a more detailed neuro exam as part of the secondary assessment while enroute.



## Stroke

### Stroke Screens

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

To conduct a rapid prehospital evaluation, EMS personnel typically use a validated tool such as the **Cincinnati Prehospital Stroke Scale (CPSS)**, the **Miami Emergency Neurologic Deficit Exam (MEND)**, or the **Los Angeles Prehospital Stroke Screen (LAPSS)**. The NWC EMSS uses the Cincinnati Stroke Scale.

The Cincinnati Prehospital Stroke Scale is used to find 3 of the major findings suggestive of stroke: arm drift, abnormal speech, and facial asymmetry

A simple way to remember the assessment components of the **Cincinnati Stroke Screen** is to cover your XXX.

- Arm drift
- Speech
- Smile

#### Arm drift

Weakness, 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.

With eyes closed, ask pt to lift both arms (palms up) to 45°-90° in front of them and hold for 5-10 seconds. The examiner may need to raise the patient's arms and observe for asymmetrical drift, weakness or flaccidity.



Note: Arm flexors are stronger than extensors; leg extensors are stronger than flexors. If hands pronate or fingers curl, these are subtle signs of weakness.

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

#### Abnormal

- Drift: Limb holds 90° (or 45°), but drifts down before full 10 sec; does not hit bed or other support
- Some effort against gravity; limb cannot get to or maintain 90° or 45°, drifts down but has some effort
- No effort against gravity
- No movement (flaccid)

#### Speech assessment

Often one of the first signs of stroke is an alteration in speech which is characteristic of a **left** hemisphere lesion of any size due to involvement of Broca's area and the frontal lobe motor strip.

Assess their ability to form words. Have the pt repeat a simple phrase like, "You can't teach an old dog new tricks," or have them sing, "Happy Birthday to You".

Assess for difficulty with articulation, phonation, pacing, stuttering, and proper matching of respirations to speech.

Listen for hoarseness. Note slowness or explosiveness. Listen for abnormal speech patterns

### SPEECH DYSFUNCTIONS

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



Listen for difficulty pronouncing the following:

- Groups: F, G, R – most common
- Labials: Lips form B, M, W
- Linguals: Tongue forms L, T, N (CN XII)
- Guttural: G, K

**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! Use effective alternative means of communicating w/ aphasic patients like blinking once for yes or twice for no or writing on a note pad.

#### Score speech

- **Normal:** 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.

**Breaking news:** BMC Neurol. 2013 Jul 15;13:87.

#### Speech disturbance at stroke onset is correlated with stroke early mortality.

**Abstract** -BACKGROUND: Speech disturbance is a common symptom of stroke and is important as a prompt identifier of the event. The frequency of the symptom among each stroke subtype, differences between patients with and without speech disturbance and its correlation to early mortality remain unclear.

RESULTS: Speech disturbance was observed in 52.6% of cerebral infarction (CI), 47.5% of cerebral hemorrhage (CH), and 8.0% of subarachnoid hemorrhage (SAH) cases.

Characteristics showing statistically significant differences between patients with and without speech disturbance and patients were age, blood pressure, history of hypertension, arrhythmia and diabetes mellitus, habit of tobacco and alcohol, and paresis.

Mortality rates of patients with/without speech disturbance were 5.2%/1.2% for CI, 12.5% /4.1% for CH, and 62.5%/ 9.0% for SAH.

**Stroke**

Adjusted hazard ratios were

2.63 (1.14-6.13,  $p = 0.024$ ) in CI,

4.15 (1.41-12.23,  $p = 0.010$ ) in CH, and

20.46 (4.40-95.07,  $p < 0.001$ ) in SAH).

**CONCLUSION: Speech disturbance was frequently observed in stroke patients at the onset** and therefore could be useful to identify the problem at the earliest stage. Hazard ratio for death was higher in stroke patients with speech disturbance than patients without. Speech disturbance is a prompt predictor of stroke early mortality.

**Smile** (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 (CN VII assessment).



**Normal:** Both sides of face move equally well and both eyes close symmetrically

**Abnormal:** If patient can wrinkle both sides of the forehead but has lower facial weakness with an asymmetric smile or one eye that does not close as tightly as the other, suspect a stroke.

If patient can wrinkle *only one side* of the forehead and has facial weakness on that side, consider that this may be Bell's palsy.



Bell's Palsy



Stroke

**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.

**Transport decision: *TIME SENSITIVE PATIENT*:** Once stroke is suspected, minimize scene time to 10 minutes or less. Transport to the nearest stroke center per SOP. Alert nearest System hospital OLMC ASAP with a **stroke alert**.

## Secondary assessment (enroute if stroke suspected)

### Vital signs

Compare rate and quality of peripheral and carotid pulses (do not rely on SpO<sub>2</sub> monitor for rate alone). Assess for carotid **bruits** if skilled in that procedure. HTN and bradycardia signal ↑ ICP; reassess frequently!



**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. BPs 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.



Because prehospital treatment of hypertension without signs of acute MI or left ventricular failure/HF is not recommended, HTN (SBP > 220 mmHg or DPB >120 mmHg on three repeated measurements made at 15 minute intervals) is treated in the hospital setting only.

**Monitor temperature** carefully: Elevated temp (>37.5° C) is often found in patients following intracranial hemorrhage (ICH). Hyperthermia can worsen damage to cerebral tissue and increase O<sub>2</sub> demand.

### Neuro exam (more detailed than CSS)

Clinical S&S of acute stroke vary widely, including findings not picked up in the CSS and often fluctuate based on the type and location of focal ischemia. Need to complete and repeat a more detailed neuro exam enroute.

### Assess for vision disturbances

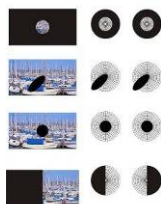
- **Monocular blindness:** Sudden, painless vision deficit in one eye due to a central retinal artery occlusion (CRAO) may involve loss of all or part of the vision. Described as a curtain dropping, fog, grayout or blackout of vision. The involved eye is on the same side as the diseased artery.



- **Diplopia:** Seeing two images. If the eyes don't track together or are not on the same plane, the pt will experience dysfunction in their binocular vision. The pt. may have a sense of bouncing and moving visual images. Cover one eye. Double vision should resolve unless the patient has a dislocated lens, retinal detachment, or high brainstem lesion.

- Blurred/indistinct vision in one/both eyes (new or old?)

- Bilateral loss of the ability to see in one or more **fields of vision**. 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 may be a sign of cranial nerve III (Oculomotor) and midbrain compression.
- Fixed, dilated pupil, ptosis, and eye pulled to the ear in a patient c/o intense headache suggests cerebral aneurysm with loss of CN III function.



- Oval pupils w/ hippus: Usually indicates ↑ ICP and impending brain herniation.

**Hippus:** Pupil rapidly dilates and constricts like it is jiggling up and down when tested for the light reflex. If seen in both eyes, herniation has occurred.

### Extraocular movements (EOMs): Horizontal gaze

Ask pt to hold head still. Have pt follow your finger with their eyes only as you move it to the extreme gaze limits on the Lt and Rt, up and down (large H). If pt moves their head, place your finger on their chin to hold head still. Both eyes should track together without stuttering or pain. The presence of nystagmus does not affect a normal score, but should be noted. If either or both eyes are unable to track completely in one direction or another, mark the test as abnormal (Whitehead, 2012). In a right hemispheric stroke, the pt may have difficulty moving their eyes across the midline to the left (ocular palsy) and may try to move their entire head instead. In a left hemispheric stroke, the pt 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.

Right side: 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 hoarse speech which can indicate dysfunction of CN 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 pt may not recognize own hand and will orient to only one side.

- Touch 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 pt 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

**Sympathetic NS dysfunction:** Look for abnormal sweating patterns, e.g., pt only sweating on one side of their body.

### Use of alternate Prehospital Stroke Screens

The **LAPSS** is a more detailed screen. The examiner must rule out other causes of AMS (seizures, severe hyper or hypoglycemia) and then identify asymmetry (R vs. L) in facial smile/grimace, grip, and arm strength. Asymmetry in any category indicates possible stroke.

The **MEND exam Prehospital** (© 2005, University of Miami) incorporates the Cincinnati Prehospital Stroke Scale (CPSS) and selected components of the NIH Stroke Scale (NIHSS) used at the hospital. (See pg. 17)

The MEND exam arose because the NIHSS is not feasible to perform in the prehospital setting and the CPSS has only a 70% sensitivity to detect stroke in the field. In addition to predicting probability of stroke, it also gauges stroke *severity* and may identify strokes not found using the CPSS.

In the prehospital setting, the CPSS should be performed on scene and Region IX will consider whether the MEND exam should be performed en route.

The MEND exam takes less than 3 minutes. A recent study found it had a 90% correlation with the NIHSS.

### MEND Exam components:

- Level of consciousness (AVPU)
- Speech ("You can't teach an old dog new tricks")
- Questions (age, month)
- Commands (close, open eyes)
- Facial droop (show teeth or smile)
- Visual fields (4 quadrants)
- Horizontal gaze –side to side
- Motor: Arm drift (close eyes, hold out arms)  
Leg drift: (open eyes, lift legs separately)
- Sensory: Arms, legs (Close eyes & touch, pinch)
- Coordination: Arms, legs (finger-nose, heel-shin)

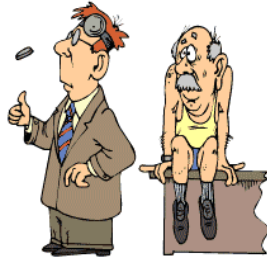
Three critical questions make up the final assessments:

1. When was the pt last known to be normal for them?
2. Did the pt have a seizure or experience a head injury at the onset of symptoms?

3. Is the pt taking any type of blood thinner?

**Differential diagnosis of stroke: Consider other causes of presenting signs ("Stroke Mimics")**

- 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 patient (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

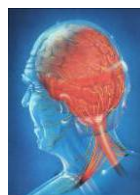


**Complications & Consequences of Stroke**

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:

**Neurological**

- Cerebral edema; ↑ ICP
- Hydrocephalus
- Hemorrhagic transformation
- Seizures
- Balance problems
- Spasticity(causing stiff and awkward movement)



- Contractures (permanent contraction of a muscle due to spasm or paralysis)
- Speech problems
- 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

**Medical**

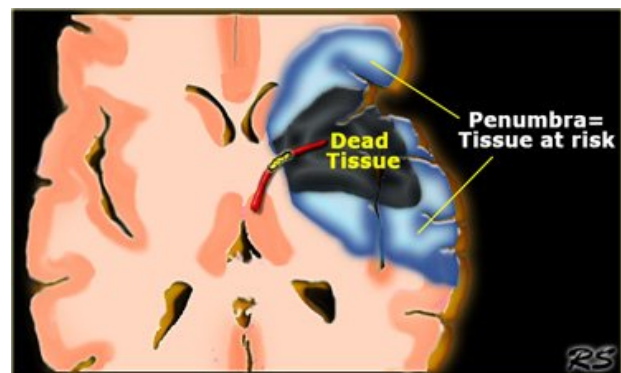
- Dysphagia (problems swallowing), resulting in poor nutrition, aspiration pneumonia
- Hypoventilation
- Atelectasis; ARDS
- Myocardial ischemia, dysrhythmias
- Neurogenic pulmonary edema
- Decubitus ulcers
- DVT/pulmonary embolism
- UTI
- Depression

**Hospital interventions: (Nice to know)**

**Goals of emergency management**

- Support vital functions
- Restore cerebral circulation
- Reduce neurological deficits
- Prevent progression and cell death
- Restore patient to optimal level of pre-stroke function

**Tissue Plasminogen Activator (tPA )**



**TIME IS BRAIN!** 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*

**Stroke**

*penumbra*) does not die for hours. Each hour of ischemia increases the degree of irreversible brain damage.



**Tissue plasminogen activator (tPA)** is the only FDA-approved treatment for acute ischemic stroke. The goal of therapy for ischemic stroke is to safely maximize patient functional recovery to pre-stroke baseline by rapidly reperusing 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 continue to support 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 (Stroke, 2013). Beyond the approved and recommended time windows for tPA use, risk of complications rises.



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).

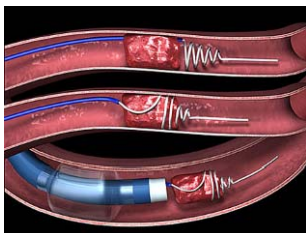
### Interventional stroke care

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

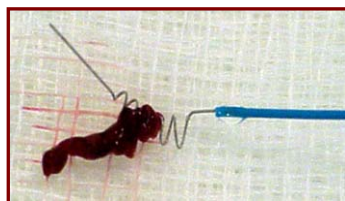
**Intra-arterial tPA** delivers fibrinolytic medication directly to the thrombus. It can be used for qualified patients up to 6 hours from onset of symptoms (Class I). As is the case with IV tPA, the best clinical outcomes with intra-arterial therapy are likely to be in those patients who receive treatment as soon after symptom onset as is possible. This therapy can only be done in specialty centers by specially trained interventionalists.

**Clot retrieval devices:** There are currently four FDA-approved devices for clot disruption or removal.

The Mechanical Embolus Removal in Cerebral Ischemia (MERCi) Thrombectomy System is a looped wire with progressively smaller helical loops, advanced via a small catheter through the clot. As



the catheter is withdrawn, the coiled wire is deployed, which captures the clot for removal.



The Penumbra Thrombectomy System is designed to aspirate clots from large intracranial vessels. With a suction catheter at the proximal end of the clot, the device continuously debulks and aspirates clot debris until the vessel is clear. The device can remove a clot in a matter of minutes, whereas fibrinolytics, even those delivered intra-arterially, may take as long as 2 hours to dissolve a thrombus.



### The Solitaire Flow Restoration



device and the Trevo Retriever device have the unique ability to both immediately bypass the clot and restore flow and retrieve the clot.

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 HTN, 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.

### Patient outcomes

Patients are scored with regards to functional outcome following stroke using the scale below:

#### Stroke outcome scale

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

#### Poor outcomes

- > 65 years old
- Severe cognitive deficit → coma
- Persistent incontinence
- Severe visual/spatial deficits (inattention)
- Global aphasia
- Co-morbid factors: HF, DM, AMI, previous stroke
- Disability before stroke

## Advances and treatments on the horizon

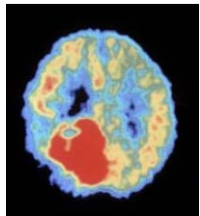
### Tissue vs. Time:

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



**Hypothermia:** Offers potential for delayed depletion of energy stores, reduction of intracellular acidosis, slowed calcium influx into ischemic cells, suppressed production of oxygen free radicals, and limited inflammation.

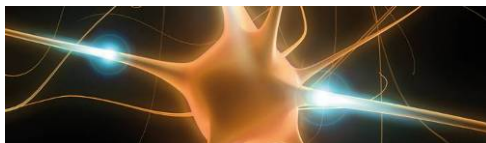


In animal models, if the brain was cooled by as little as 1° C, the size of the stroke was reduced by as much as one half. Hypothermia challenges include determination of most effective target temperature, treatment duration and time window to begin cooling, and development of techniques to induce

cooling that avoid discomfort and shivering.

Much work is yet to be done. Research must identify criteria for patient selection, the therapeutic window for initiation of therapy, the safest and most effective speed, level, technique and duration of hypothermia, and how to minimize complications (hypotension, dysrhythmias, and pneumonia).

**EuroHYP-1**, a current international clinical trial, was begun in early 2012. The trial will test the efficacy of therapeutic cooling on 15,000 ischemic stroke patients, including patients who get tPA and those who do not. Cooling must begin within 6 hours of symptom onset and will be continued for 24 hours. Participants will be evaluated for outcome at 3 months post stroke.



**Neuroprotective agents** refer to therapy that salvages or delays the infarction of ischemic penumbra by directly acting on brain tissue as opposed to reperfusion it.

Cellular-level events in the ischemic cascade include

build-up of calcium within cells, release of excitatory neurotransmitters, generation of oxygen-free radicals, and release of inflammatory mediators. Neuroprotective agents interfere at various points in the ischemic pathway.

Substantial reduction in infarct volume was demonstrated in animal models when neuroprotective agents were delivered within 10-120 minutes of onset of ischemia. This concept is especially significant for prehospital providers. Because these agents will likely be safe and effective in both ischemic and hemorrhagic stroke, they would be initiated as early as possible (possibly being started in the field), prior to brain imaging, fibrinolysis, and or revascularization therapies. Prehospital administration of these agents mitigates one key confounding factor in past trials – failure to treat patients early enough after onset of symptoms.

EMS providers in the Los Angeles area are involved in a clinical study in which magnesium is being given to pts whose stroke symptom onset is within 2 hours. Estimated data collection date for this trial is June 2013. Early outcomes data were encouraging, citing (1) dramatic early recovery in 42%, good 90 day functional outcome in 75% of pts, and no serious adverse events related to the study agent! (FAST-MAG, 2013).

**'Wake-up' Stroke:** Between 20 - 25% of all strokes occur while patients are asleep. Current guidelines for administration of IV tPA exclude these patients because time of symptom onset must be less than 3 (up to 4.5) hours. Two clinical trials are under way at this time - the WAKE-UP clinical trial in Europe and the Safety of IV Thrombolysis for Wake-up Stroke trial, which is overseen by the University of Texas. The European study will use MRI imaging to identify patients with wake-up stroke whose MRI shows a particular imaging pattern noted consistently in patients whose known time of symptom onset was less than 4.5 hours. Projected study completion date is May 2016. The U of Texas study will administer IV tPA to patients with wake-up stroke following the usual IV tPA criteria except for time of onset. IV tPA must be administered within 3 hrs of awakening.



A 24-month study done in London, England (2009-2010) compared symptom improvement, functional outcome, and incidence of intracranial hemorrhage (ICH) (the most worrisome of adverse events associated with IV tPA) in 2 groups of patients treated with IV tPA. Among 326 patients with ischemic stroke, and 68 patients with wake up stroke, there were no differences in early improvement, functional outcome at 90 days, or intracranial



**Stroke**

hemorrhage rates between the two groups.



**Stroke**

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](http://www.Strokeassociation.org) or call 630-789-9222.


<b>Risk factors for stroke</b>	
<b>Modifiable</b>	<b>Nonmodifiable</b>
<ul style="list-style-type: none"> <li>■ High blood pressure (hypertension)</li> <li>■ Smoking</li> <li>■ TIAs</li> <li>■ Heart disease</li> <li>■ Diabetes</li> <li>■ Carotid or peripheral artery disease</li> <li>■ Increased RBCs (Waldenstrom's macroglobulinemia)</li> <li>■ Coagulopathies</li> <li>■ Sickle cell disease</li> <li>■ High cholesterol (esp. high LDL and high triglycerides)</li> <li>■ Inactivity/obesity</li> <li>■ Abuse of alcohol or drugs</li> </ul>	<ul style="list-style-type: none"> <li>■ Age</li> <li>■ Ancestry (hereditary)</li> <li>■ Ethnicity</li> <li>■ Gender</li> <li>■ Prior medical history (stroke or AMI)</li> </ul>

<b>Distinctive signs and symptoms of stroke</b>
Weakness or paralysis of the face, arms, or legs, especially on one side of the body; asymmetric smile or mouth drawn to one side; loss of facial expression; possibly a drooping eyelid; one eyelid does not close as tightly as the other
Inability to speak or understand speech (expressive or receptive aphasia) or distorted speech (dysarthria)
Sensory or autonomic symptoms affecting half of the body, such as numbness or abnormal sweat patterns
Visual loss in one half (or one quarter) of the visual field, or dimming or loss of vision (especially in only one eye); constricted pupils; pupils unequal in size or reactivity; double vision
Severe headache with no apparent cause, often accompanied by a stiff neck
Dizziness, confusion, unsteadiness, or falls with no apparent cause; seizures
Drowsiness or decreased consciousness
Other symptoms: nausea or vomiting, respiratory distress, unusually flushed or pale skin, loss of bowel and/or bladder control


<b>Characteristics of strokes</b>	<b>Thrombosis</b>	<b>Embolism</b>	<b>Intracerebral Hemorrhage</b>	<b>SAH</b>
Prodromal warning	Common	No	No	Rare
Onset during sleep	Sometimes	Rare	Rare	Rare
Development	Gradual	Sudden	Gradual or sudden	Sudden
Quick reversal	Possible	Possible	No	Possible
Bloody CSF	No	Rare	Common	Yes
Coma	Rare	Rare	Common	Common
Decreased consciousness	Mild	Mild	Severe	Moderate
Headache	Mild	Mild	Severe	Severe
Hypertension	Common	Possible	Not always	Common
Nuchal rigidity	No	No	Sometimes	Yes
Vomiting	Rare	Rare	Sometimes	Sometimes

Features of clinical situations mimicking Stroke ( <i>Stroke</i> , 2013)	
Etiology	History and Exam Findings
Psychogenic	Lack of objective cranial nerve findings, neuro findings in a nonvascular distribution, inconsistent examination
Seizures	Hx of seizures, witnessed seizure activity, postictal period
Hypoglycemia	Hx DM, low serum glucose, ↓ LOC
Complicated Migraine/Migraine with Aura	Hx similar events, preceding aura, headache
Hypertensive encephalopathy	Headache, delirium, significant HTN, cortical blindness, cerebral edema, seizure
Wernicke's encephalopathy	Hx alcohol abuse, ataxia, EOM paralysis, confusion
CNS abscess	Hx drug abuse, endocarditis, medical device implant w/ fever
CNS tumor	Gradual progression of symptoms, other primary malignancy, seizure at onset
Drug toxicity	Med hx includes Lithium, phenytoin, carbamazepine


Prehospital MEND Exam Pocket Card (Side 1)



Center for Research in Medical Education



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**MEND EXAMINATION - PREHOSPITAL**  
Green Boxes Contain Basic Exam (CPSS)

**MENTAL STATUS**

- Level of Consciousness (AVPU)
- Speech: "You can't teach an old dog new tricks"
- Questions (age, month)
- Commands (close, open eyes)

**CRANIAL NERVES**

- Facial Droop (show teeth or smile)
- Visual Fields (four quadrants)
- Horizontal Gaze (side to side)

**LIMBS**

- Motor – Arm Drift (close eyes-hold out arms)  
Leg Drift (open eyes-lift each leg separately)
- Sensory – Arm, Leg (close eyes & touch, pinch)
- Coordination – Arm, Leg (finger-nose, heel-shin)

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Left cerebral hemisphere L MCA involvement	Right cerebral hemisphere R MCA involvement
Aphasia L gaze preference R visual field deficit R hemisensory loss	Neglect (L hemi-inattention) R gaze preference L visual field deficit L hemiparesis L hemisensory loss

Lacunar Syndromes
<ul style="list-style-type: none"> <li>▪ Pure motor</li> <li>▪ Pure sensory</li> <li>▪ Clumsy hand/dysarthria</li> <li>▪ Ataxic hemiparesis</li> <li>▪ Multi-infarct dementia (Cesario &amp; Szost, 2013)</li> </ul>

Brainstem stroke
Posterior circulation 5 Ds: Dizziness, Diplopia, Dysarthria, Dysphagia, Dystaxia Hallmark: Crossed findings Cranial nerve deficits (ipsilateral) Motor/sensory deficits (contralateral) Clinical findings: asymptomatic to comatose

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- National Stroke Association (NSA): [www.stroke.org](http://www.stroke.org)
- Stroke education for EMS professionals: <http://www.strokeawareness.com/ems4stroke>