



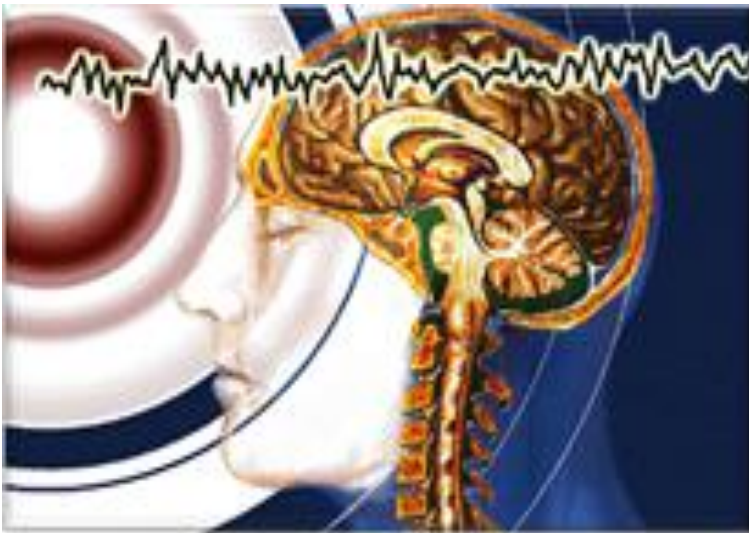
**NORTHWEST  
COMMUNITY  
EMERGENCY  
MEDICAL  
SERVICES  
SYSTEM**

# **NWC EMS System**

## **Continuing Education**

### ***July 2020***

**UNKEYED COMPLETE PACKET**



## ***Seizures and Stroke***

Questions/Comments  
regarding this CE are welcome,  
and should be directed to:  
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## **Knowledge Objectives:**

After completing the class & reading the referenced documents, each participant will do the following with a degree of accuracy that meets or exceeds standards established for their scope of practice without critical error:

### ***Cognitive:***

- Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient with seizure activity.
- Integrate the pathophysiological principles to the assessment of a patient with seizure.
- Differentiate between endocrine emergencies based on assessment and history.
- Correlate abnormal findings in the assessment with clinical significance in the patient with seizures.
- Risk factors for stroke and elements of the PMH that may contribute to stroke
- Medications that patients with high risk for stroke may be taking; especially the various anticoagulants.
- Difference in history and presentation of anterior vs. posterior strokes
- Unique presentation of subarachnoid hemorrhage/aneurysms
- Create a patient with possible stroke with an emphasis on
  - Presentation
  - Mental status exam
  - Vital signs and other specific assessments
  - Limbs: motor and sensory exams
  - Other findings: neck stiffness; vomiting without nausea, SNS abnormalities (sweating on one side)

### ***Affective:***

Appreciate utilization of the SOP decision tree for selecting a receiving hospital: Comprehensive vs Primary stroke center for appropriate patient care.

### ***Psychomotor:***

- Follow along with Dr. Jordan in the proning of a patient (EMS Playbook Appendix C p.2-3).
- Practice modifications to the ETI procedure using the HEPA filter available for your agency (EMS Playbook Appendix E-Adv. Airway).

# Seizures

A seizure is a sudden, uncontrolled episode of high voltage electrical discharge of brain cells in cerebral cortex with associated sensory, motor, and/or behavioral changes. It is usually the result of an increase in extracellular potassium that is not effectively picked up by neuroglia cells (astrocytes). High frequency discharges are usually paroxysmal. Low frequency are usually synchronous.



- **Stats:** 1:200 people have a seizure disorder or 4.5% of the U.S. population; inverse relationship between socioeconomic status and incidence of seizures. Estimated to be 100,000 new cases per year.
- **Age of onset:** Can begin at any age, but majority (75%) experience first seizure before age of 20; 30% develop prior to age 5. Age of the patient at first seizure is frequently related to etiology.
- **Etiology:** 50% of cases have no known cause. Of the other 50%, the following conditions/etiologies may cause instability or irritability of the brain and possibly lead to seizures:

## A. Newborns and infancy

- \_\_\_\_\_ defects
- Developmental \_\_\_\_\_ defects

## B. Childhood

1. Infections, \_\_\_\_\_, and trauma are the most common reasons.
2. First seizure may occur during acute stage of illness or later due to residual damage to some brain cells.
3. Less common causes:
  - a. \_\_\_\_\_
  - b. \_\_\_\_\_
  - c. \_\_\_\_\_
  - d. \_\_\_\_\_
  - e. \_\_\_\_\_

## C. Young adults

1. Head trauma
2. Tumor if first seizure over the age of 20 (40% of patients with brain tumors experience seizures)
3. \_\_\_\_\_, including alcohol and drug withdrawal
4. \_\_\_\_\_ diseases (lupus)
5. Eclampsia

## ➤ Older adults

- A. Stroke
- B. \_\_\_\_\_ trauma (recent or past)
- C. Hypoxia
- D. Hypo\_\_\_\_\_ and other metabolic abnormalities (Ca, K, or manganese)
- E. Brain tumors; primary or metastatic
- F. Vascular disorders
- G. Idiopathic epilepsy
- H. Degenerative neurological disease, Alzheimer's
- I. Abrupt \_\_\_\_\_ withdrawal of \_\_\_\_\_ or chronically used sedatives

Patients can lower their threshold for seizure activity through the following: (able to obtain as part of hx)

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
4. \_\_\_\_\_
5. \_\_\_\_\_
6. \_\_\_\_\_

## International Classifications of Seizures

### Partial Seizures

**Focal Onset:** Named for the area of the brain in which the seizure originates, a partial seizure has a local onset and is traditionally called focal motor or temporal lobe seizures. The \_\_\_\_\_ lobe is the most commonly affected area of the brain



therefore if motor activity occurs; it can appear to affect an isolated portion of the body. If a focal seizure becomes generalized in nature, affecting both sides of the brain, generalized activity may also be observed.

<https://www.youtube.com/watch?v=m4iWcTJW3tU>

As described, partial seizures are associated clinically with focal neurological activity usually the onset is in one specific area of the brain. This localized activity may or may not be associated with an \_\_\_\_\_ of consciousness.

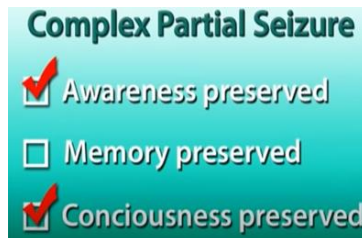
Another division occurs in assessing if the patient



has a level of awareness or impaired awareness during the seizure activity. If a person is completely aware during the focal seizure, this is described as a simple

partial seizure. If either the awareness, memory or consciousness is not preserved, the seizure is classified as complex partial (see SOP, p. 89).

A portion of the time, focal seizures can become generalized affecting a larger part of the body with both tonic and clonic



activity and impaired awareness.

## Generalized Seizures



*Generalized seizures* are traditionally called grand mal or petit mal seizures and the onset of symptoms comes from multiple symmetrical areas of the brain.

Clinical presentation:

The most common form of generalized seizures are characterized by an initial \_\_\_\_\_ of consciousness, \_\_\_\_\_ movements affecting the whole body, and usually tongue biting, incontinence, and mental \_\_\_\_\_.

Generalized tonic clonic seizures are followed by a period of sleepiness, where the patient is minimally

responsive known as a \_\_\_\_\_. This phase may last from a few \_\_\_\_\_ to \_\_\_\_\_

The typical progression of a **generalized tonic clonic seizure (grand mal)**

- Prodrome of ill-defined feelings sometimes present, but no actual aura
- Loss of consciousness
- Tonic phase - Continuous \_\_\_\_\_ tension that makes the body appear stiff. As the muscles of the body become rigid, the simultaneous contractions of the diaphragm and chest muscles may produce a characteristic "epileptic cry". The patients eyes roll up or turn to the side and the tongue may be bitten.
- Hypertonic phase - this involves extreme muscular rigidity and hyperextension of the back.
- Clonic phase - rigidity that alternates with relaxation, appearing as \_\_\_\_\_ motions of the head, arms and legs. The patient may lose bowel and/or bladder control.
- There may be massive autonomic discharge demonstrated by hyperventilation, salivation, tachycardia, urination, defecation, diaphoresis.
- Post seizure coma
- Postictal state: The patient may experience confusion, fatigue, and headache or sore muscles with transient neuro deficits.

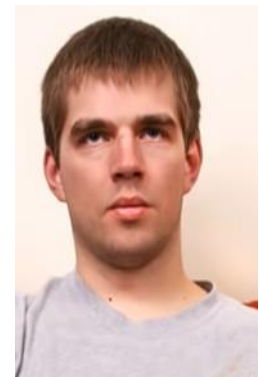


*Absence seizures (traditionally called Petit Mal)*

- Most common onset in children between 4-12 years. Rare to have onsets over age 20.
- Characterized by brief loss of \_\_\_\_\_ of 10 sec. or less during which time the child stares vacantly off into space for a few seconds, neither speaking or hearing what is said, then returns to consciousness \_

\_\_\_\_\_ demonstrating any motor symptoms.

- Motor activity usually consists of automatisms such as eye blinking or mild facial



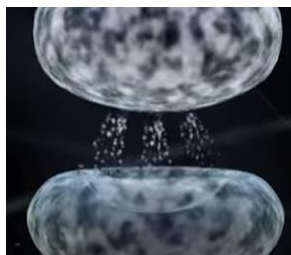


- twitching/lip smacking.
- The patient maintains posture and the seizure is frequently missed because it is so brief and subtle. May occur more than 100 times in a day.
- Generally, absence seizures do \_\_\_\_\_ need to be treated as an emergency. However, these children need prompt and effective treatment, since absence seizures can interfere with response time and learning in school.
- Often confused with complex partial seizures which is unfortunate, as the drugs effective in absence seizures are either not effective at all or only partially effective in controlling partial complex seizures; and the drugs most effective for complex partial seizures are ineffective for absence.



#### Differences:

- Absence is never preceded by an aura
- Absence have abrupt onset and brief duration; seconds rather than minutes
- Absence ends with an instantaneous return to clear consciousness
- Absence is always associated with the EEG abnormality of 3 Hz spike and slow wave discharges which are easily provoked by hyperventilation.



#### Atonic seizures: (drop attacks; astatic; and traditionally called minor motor seizures)

1. Usually start between the ages of 2-5 years
2. Legs give way and patient drops to the ground
3. Last 10 seconds to one minute

4. Prompt recovery with clear mental status and ability to stand and walk
5. Many children wear helmets to prevent injury from the fall

#### Myoclonic seizures (bilateral massive epileptic)

1. Myoclonic jerks are sudden, brief shock-like contractions which may involve the entire body or be confined to the face, trunk, or extremities.
2. May be rapidly \_\_\_\_\_ or relatively isolated.
3. Patients may spill beverages or be propelled out of chairs if jerks are forceful.

#### Infantile spasms

1. Syndrome that starts between the ages of 3 months to 2 years
2. "Jackknife" or "Salaam" seizures, last for seconds, during which the child's head, neck, and trunk are flexed forward with knees drawn up.
3. Spasms may occur repeatedly during the day
4. Many of these children are mentally retarded
5. Known causes: Hypoglycemia, phenylketonuria, severe anoxic brain damage, and tuberous sclerosis. Etiology is unknown in 50% of cases.

#### Febrile seizures

A febrile seizure is a single, generalized, tonic clonic seizure lasting fewer than 20 minutes that occurs with \_\_\_\_\_ (38° C or higher) in infants and young children without evidence of intracranial infection or any other defined cause (nonfocal neuro exam).



Seizures with fever in children who have had \_\_\_\_\_ nonfebrile seizures, anticonvulsant use, known neurological disease, and history or clinical suspicion of head trauma are excluded from the definition. These seizures tend to be single episode/self-limited.

#### Incidence and epidemiology

A febrile seizure affects 2-5% of all children under the age of 5 years old with the most common age range being between 6 months and 3 years of age. Febrile seizures do occur 3-4 times more often in the male population than in the female population.

## Etiology

A child who has had a febrile seizure has a 33% chance of having \_\_\_\_\_one if less than one year when he had his first. Maturation of the nervous system: (NS) is associated w/ a ↓ excitability of neurons. Maturation occurs more rapidly in females which may explain increased risk in males.

Fever lowers seizure the threshold in everyone as does a rapidly rising temp. Most children have temp of 102.2° F (39° C) or greater at time of diagnosis.

**Rate of rise may be more important than absolute temperature**, although others argue that magnitude of temp is of greater relevance. Associated with a rise in temperature would be the presence of an infection; more over a viral infection. Common viral infections: roseola infantum, influenza A virus, viral pharyngitis, tonsillitis, and viral pneumonia. Possible bacterial infections: salmonella, shigellosis, otitis media, pneumonia, bacteremia, urinary tract infection and bacterial enteritis.

## Long term Treatment

The good news for those who have a febrile seizure is that 2/3 of that population will never have another one, therefore they are self-limiting and do not require long term prophylaxis with medications. Additionally, there is a general risk of 0.5% of the population who has a febrile seizure going on to have epilepsy.

Acquiring the **past medical history** for proper documentation (see SOP p. 89).

1. Patient's history and frequency of seizures
2. Any prescribed medications and compliance in taking them. Amount and time of last dose
3. Recent or past history of head trauma
4. Predisposing illness/disease
5. Recent fever, headache, or stiff neck (possible meningitis)
6. History of ingestion/drug or alcohol abuse; time last used
7. History of diabetes, heart disease, or stroke

## Consider possible etiologies

1. Anoxia/hypoxia
2. Cerebral palsy or other disabilities
3. Eclampsia
4. Stroke/cerebral hemorrhage
5. Trauma/child abuse

6. Anticonvulsant withdrawal/noncompliance
7. Infection (fever, meningitis)
8. Metabolic (hypoglycemia, electrolyte imbalance, acidosis)
9. Toxins/intoxication; ODs, DTs
10. Tumor

## Secondary assessment

Seizure description: presence of an aura, focus of origin (one limb or whole body), simple/complex (conscious or loss of consciousness); partial/generalized; progression and duration; eye deviation prior to or during seizure; incontinence; or oral trauma; or abnormal behaviors (lip smacking)

Duration and degree of mental status changes in postictal period.

## Treatment

1. IMC special considerations:
  - ☐ Clear and protect airway. No bite block. Vomiting/aspiration precautions, suction prn
  - ☐ Protect patient from injury; do not restrain during tonic/clonic movements
  - ☐ Position on side during postictal phase unless contraindicated
  - ☐ If history of generalized tonic/clonic seizure activity: consider need for IV NS TKO
2. If generalized tonic/clonic seizure activity present:  
*Benzodiazepine administration takes precedence over bG determination in pts who are actively seizing*

MIDAZOLAM 0.1 mg/kg IVP/IO (0.2 mg/kg IN/IM) (Max single dose 2 mg) q. 30-60 sec up to 10 mg based on size IVP/IN/IO/IM titrated to stop seizure. If seizures persist: Contact OLMC for additional orders.

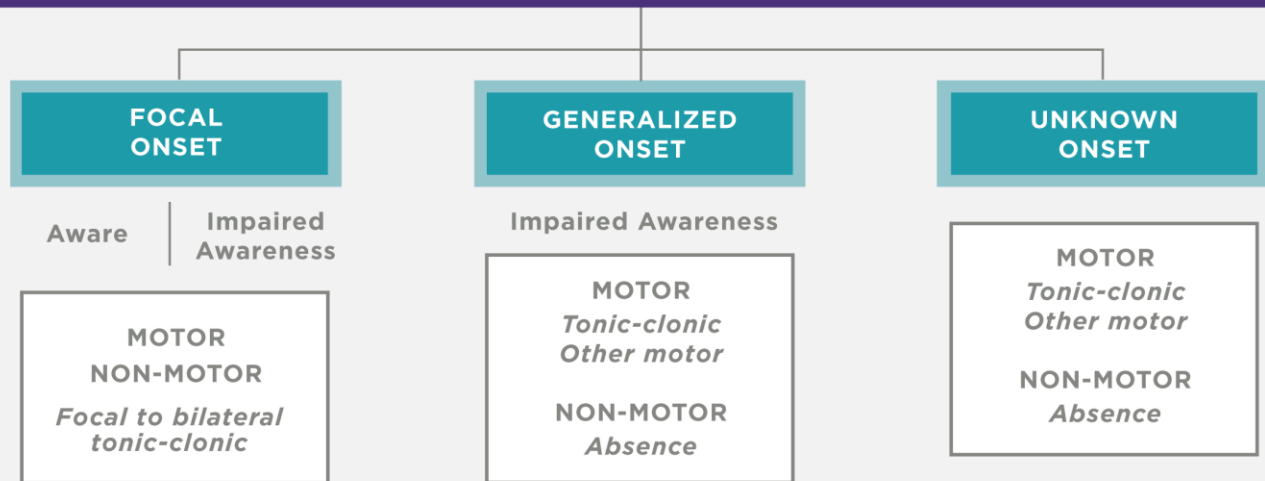
3. Identify and attempt to correct reversible precipitating causes.  
Obtain blood glucose level: If < 70: DEXTROSE or GLUCAGON per Peds Hypoglycemia SOP.

## TREATMENT

When in doubt, call OLMC and explain current symptoms observed and PMHx given to EMS.

## “NEW” CLASSIFICATION OF SEIZURE TYPES BASIC VERSION <sup>1</sup>

*\* from International League Against Epilepsy, 2017*



<sup>1</sup> Definitions, other seizure types and descriptors are listed in the accompanying paper & glossary of terms

<sup>2</sup> Due to inadequate information or inability to place in other categories

## References

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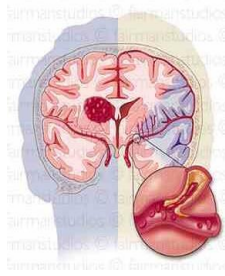
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# Stroke

## Epidemiology

**Broad definition:** A stroke is a sudden event causing 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**. Approx. 80% are preventable and need to be treated with the same urgency as a heart attack with rapid detection and transport to specialty centers similar to a STEMI.

**Do NOT use the term CVA anymore**

## Incidence of stroke in U.S.

- Someone has a stroke every 40 seconds
- > **795,000** each year
- ~ 610,000 are first or new onset
- ~185,00 (nearly one of four) are recurrent
- # of adults who ever had a stroke: 6.3 million
- % of adults who ever had a stroke: 2.6%

## Morbidity and mortality

- Deaths/yr: 133,103 (1 out of every 20 deaths).
- Deaths per 100,000 population: 41.7
- On average, someone dies from stroke every **4 min**
- Cause of death rank: 5 (Only heart disease, cancer, chronic lower respiratory disease, and unintentional injuries cause more deaths than stroke)
- 30-day death rate
  - 40-84% after cerebral hemorrhage
  - 15-33% after cerebral infarction

<https://www.cdc.gov/nchs/fastats/stroke.htm>

Due to public education, early detection, and better management of BP and stroke, deaths have declined, and rates of fibrinolytic therapy and endovascular procedures have increased (CDC, 2014).

These strides have been offset by growth in the aging, diabetic, and obese populations (more people at risk).

Stroke is the leading cause of serious **long-term adult disability** such as paralysis, loss of speech. It is the leading diagnosis for transfers from hospital to rehab and long-term care facilities. Only 10% resolve completely with no lingering S&S.

## Morbidity in stroke survivors

31% need assistance with activities of daily living (ADLs)  
20% need assistance to walk  
16% need placement into assisted living  
33% suffer from depression

## Stroke morbidity factors

- >65 years of age
- Severe cognitive deficit to coma
- Persistent incontinence
- Severe visual/spacial deficits
- Global aphasia
- Co-morbid factors: HF, DM, AMI
- Disability before stroke

**Costs are staggering:** \$34 billion annually, including cost of healthcare services (initial hospitalization; rehabilitation, physician costs, possible readmission), medications, & lost productivity (Mozaffarian et al, 2015).

## Demographics of stroke: Equal opportunity threat

While stroke kills and disables people of all ages, both sexes, and all races, prevalence, death and disability rates vary by race and ethnicity; median household income; urban vs nonmetropolitan locations; and geographic location (mortality inside and outside the Stroke Belt in the Southeastern U.S.). For the latest statistics see:

<https://www.cdc.gov/nchs/data/has/has15.pdf#023>

## Unmodifiable risk factors for stroke: age, gender, race/ethnicity, and stroke family history

**Patient Age:** It is a myth that stroke occurs only in elderly adults. Stroke strikes all age groups from fetuses to centenarians. Older persons do have a higher risk than the general population and that risk increases with age. For every

LIFE ALMOST OVER AT 13



decade after the age of 55, stroke risk doubles; 2/3 of all strokes occur in those >65. People >65 also have a 7 X greater risk of dying from stroke than the general population. The incidence of stroke is increasing proportionately with the increase in the older population

([http://www.ninds.nih.gov/disorders/stroke/detail\\_stroke.htm#1105\\_10](http://www.ninds.nih.gov/disorders/stroke/detail_stroke.htm#1105_10)).

## Gender

### Men:

- 1.25 X higher risk; greater incidence of stroke than women
- Usually younger when they have a stroke and therefore have a higher rate of survival.



### Women:

- Higher mortality from stroke
- More likely to dismiss symptoms and to seriously underestimate their risk.
- Those >30yrs who smoke and/or take oral contraceptives/high levels of estrogen (hormone replacement therapy) are at 22X higher risk.
- Are often older when they have a stroke, are more physically frail, or have other health problems impairing recovery (NINDS, 2016).

## Anatomy and Physiology

**Cerebral blood supply** is the most complex circulatory system in the body. The brain is 3 lbs of tissue but the most perfusion-sensitive organ. It receives ~20% the cardiac output and consumes 25% of available glucose.

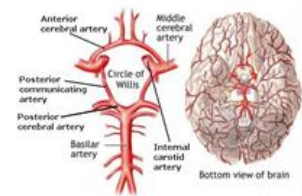
### Main blood vessels supplying the brain:

Blood is supplied to the brain, face, and scalp via two major sets of vessels: the right and left **common carotid** arteries and the right and left **vertebral** arteries.

The **common carotid** arteries have two divisions.

- **External carotids** supply face and scalp with blood.
- **Internal carotids** supply blood to most of the anterior portion of the cerebrum (frontal lobe, anterior parietal lobes and temporal lobes).
- Patients with anterior circulation disruption may present with unilateral paralysis or numbness on the side opposite to the obstruction, language disturbance, visual disturbance, and/or monocular blindness. These strokes may also be referred to as carotid or **anterior strokes**.

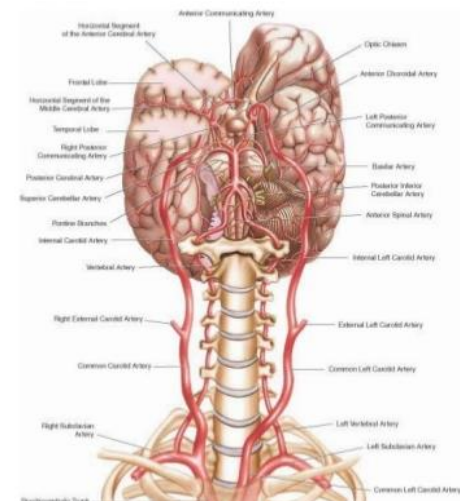
The **Vertebrobasilar arteries** supply the posterior 2/5<sup>th</sup> of the cerebrum, part of the cerebellum, and the brain stem. Occlusion here can cause severe vertigo, nausea, vomiting, dysphagia, diplopia, visual field loss; gaze palsies, decreased pain and temperature sensation, same side (ipsilateral) cerebellar ataxia, loss of 2 point discrimination (**posterior stroke**).



**Circle of Willis** connects the two systems before entering the structures of the brain. Designed so disruption of any part will not cause significant loss of blood flow to tissues (Bledsoe, 2017)

- Anterior communicating artery
- Middle cerebral artery
- Internal carotid artery
- Post. communicating artery
- Posterior cerebral artery

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

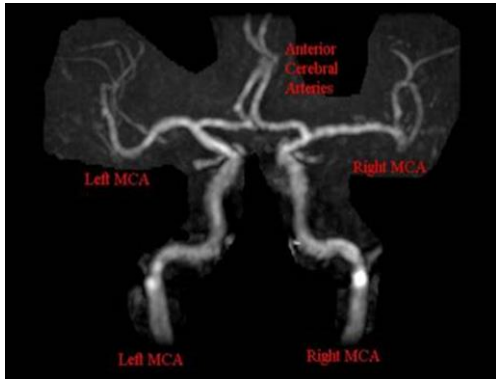


From this circle, other arteries (anterior cerebral (ACA), middle cerebral (MCA), and posterior cerebral (PCA)) arise and travel to all parts of the brain.

### Anterior Cerebral Artery (ACA)

The ACA extends upward and forward from the internal carotid artery. It supplies the frontal lobes, (controls logical thought, personality, and voluntary movement, especially of the legs). Stroke in the ACA results in leg





weakness opposite to the side of brain injury. If both anterior cerebral territories are affected, profound mental symptoms may result (akinetic mutism).

### Middle Cerebral Artery (MCA)

Largest branch of the internal carotid and supplies a portion of the frontal lobe, lateral surface of the temporal and parietal lobes (including the primary motor and sensory areas of the face, throat, hand and arm), and in the dominant hemisphere, the areas for speech. **The MCA is the one most often occluded in stroke.**

### Posterior Cerebral Artery (PCA)

In most people, the PCAs stem from the basilar artery but sometimes originate from the ipsilateral internal carotid artery. The posterior arteries supply portions of the temporal and occipital lobes. Infarction occurring in the territory of the PCA, is usually secondary to embolism from lower segments of the vertebral basilar system or heart.

### Lenticulostriate Arteries

Beyond the Circle of Willis, collateral circulation is limited, supplied only by vessels in the dura mater & arachnoid membrane. Small, deep penetrating arteries (lenticulostriate arteries) branch from the MCA. Occlusions of these vessels or penetrating branches of the Circle of Willis or vertebral or basilar arteries are referred to as **lacunar strokes**. ~27% of strokes are lacunar. High incidence in pts with chronic HTN (<http://www.strokecenter.org/professionals/brain-anatomy/blood-vessels-of-the-brain/>).

**Risk factors for stroke:** Need careful patient history

While you can't control some risk factors like

heredity, age, gender, and ethnicity (see demographics of stroke), >90% of stroke deaths are caused by **modifiable** risk factors due to patient lifestyle choices and are considered **preventable**.

- **Hypertension:** Persistently high BP >140/90 leads to a diagnosis of HTN. Hypertension disrupts the structure of cerebral blood vessels, promotes atherosclerosis, and impairs vital cerebrovascular regulatory mechanisms. These vascular changes increase the susceptibility of the brain to ischemic injury.

Based on the latest data available, 32.6% of US adults ≥20 yrs of age have HTN, which represents ~80 million people. African American adults have the highest prevalence of HTN in the world. Among non-Hispanic black men and women, the age-adjusted prevalence of HTN was 44.9% and 46.1%, respectively (Mozaffarian et al, 2016).

**Of all the risk factors, HTN is the most powerful.** Stroke risk is 4-6 X higher in those with HTN; ~77% have a BP >140/90 at their first stroke.

The impact of HTN on total stroke risk decreases with age. Factors other than HTN play a greater role in overall stroke risk in elderly adults. For those without HTN, the absolute risk of stroke increases over time until around age 90, when the absolute risk becomes the same as for people with HTN.



**Antihypertensive medications** can decrease a person's risk and incidence of stroke by 38% and decrease the stroke fatality rate by 40%. **Common anti-HTN agents** include beta-blockers, angiotensin converting enzyme (ACE) inhibitors, antiotensin receptor blockers (ARBs), calcium channel blockers, diuretics, and vasodilators (See HF page of SOPs for lists of meds).

- **Heart Disease:** After HTN, the 2<sup>nd</sup> most powerful risk factor for stroke, especially if pt. has **atrial fibrillation**.





In AF, the left atrium beats up to four times faster than the rest of the heart. This leads to an irregular flow of blood and the occasional formation of blood clots (mural thrombi) that can leave the heart and travel to the brain, causing a stroke.

AF may be asymptomatic and undetected, affects as many as 2.2 million Americans, and increases risk of stroke by 4-6%. About 15% of patients have AF before they experience a stroke. AF is more prevalent in older age groups. Unlike HTN and other risk factors that have a lesser impact on the absolute risk of stroke with advancing age, **the influence of AF on total risk for stroke increases powerfully with age** (1.5% at 50-59 to 23.5% at ages 80-89). In people >80, AF is the direct cause of one in four strokes.

- **Heart valve disease/malfunction:** Mitral stenosis, mitral annular calcification, valvular vegetation, and defective valve prostheses can double risk for stroke.
- **Cardiac chamber lesions** (LV aneurysm, intracardiac defects with paradoxical embolism (LV and left atrial thrombus with or without mitral stenosis))
- **Heart muscle malformations**  
**Patent foramen ovale (PFO)** is a passage or a hole (called a "shunt") in the heart wall separating the two atria. Blood clots are usually filtered out by the lungs, but PFO could allow emboli to bypass the lungs and go directly through the arteries up to the brain, potentially causing a stroke. Atrial septal aneurysm (ASA), a congenital malformation of heart tissue, is a bulging of the septum or heart wall into one of the atria. PFO and ASA frequently occur together and therefore amplify the risk for stroke. Two other heart malformations that seem to increase the risk for stroke for unknown reasons are left atrial enlargement and LV hypertrophy.  
People with left atrial enlargement have a larger than normal left atrium. Those with LV hypertrophy have a thickening of the LV wall.
- **Cardiac surgery** to correct heart malformations or reverse the effects of heart disease: Strokes occurring in this situation are usually the result of surgically dislodged plaques from the aorta that travel to the arteries in the neck and head, causing stroke. Cardiac surgery ↑ risk of stroke by ~1%.

- **Diabetes Mellitus (DM):** People with DM have 3X the risk of stroke as it accelerates atherosclerosis. This risk is highest in the 5<sup>th</sup> and 6<sup>th</sup> decades and decreases after that. Like HTN, the relative risk of stroke from DM is highest for men at an earlier age and highest for women at an older age. People with DM may also have other contributing risk factors that can amplify the overall risk for stroke. Ex: prevalence of HTN is 40% higher in diabetics. Control of hyperglycemia can reduce risk of microvascular complications and overall risk of stroke.

- **High blood cholesterol/lipids:** High cholesterol levels contribute to stroke risk. Cholesterol contributes to the production of hormones and vitamin D and is an integral component of cell membranes. The liver makes enough cholesterol to fuel the body's needs and this natural production alone is not a large contributing factor to atherosclerosis, heart disease, or stroke. The danger from cholesterol comes from high levels that contribute to the risk of atherosclerosis and thickening of the arteries.

There are two kinds of cholesterol, commonly called the "good" and the "bad." Good cholesterol is **high-density lipoprotein, or HDL** (healthy cholesterol); bad cholesterol is **low-density lipoprotein, or LDL** (lethal cholesterol). Together, these two forms make up a person's total serum cholesterol level. A level of <200 mg/dL is generally considered safe unless one is at high risk for heart attack or stroke, while a level of >240 is considered dangerous and places a person at risk for heart disease and stroke.

Most cholesterol is in the form of **LDL** that circulates

through the bloodstream, picking up excess cholesterol and depositing it where it is



needed (production and maintenance of cell membranes). But when too much is present, the body cannot handle the excessive LDLs, which build up along the inside of arterial walls, hardens, and turns into arterial **plaque**, leading to **stenosis and atherosclerosis**.

Plaque blocks blood vessels, can rupture, and contributes to the formation of blood

clots. A person's LDL level should be <130 mg/dL to be safe. LDL levels between 130 and 159 put a person at a slightly higher risk for atherosclerosis, heart disease, and stroke. An LDL >160 puts a person at great risk for a heart attack or stroke.

**HDL** is beneficial and contributes to stroke prevention. It carries a small percentage of the cholesterol in the blood, but instead of depositing on the inside of artery walls, HDL returns to the liver to unload its cholesterol. The liver then eliminates the excess by passing it along to the kidneys. Currently, an HDL level >35 is considered desirable. High levels of HDL are associated with a reduced risk for heart disease and stroke and low levels (<35 mg/dL), even in people with normal levels of LDL, lead to an increased risk for heart disease and stroke.

A healthy diet and regular exercise are the best ways to lower total cholesterol levels. In some cases, cholesterol-lowering medications are needed.

**Core health behaviors:** (diet, physical activity, smoking, and energy balance)

- **Smoking** almost doubles the risk for ischemic stroke, independent of other risk factors, and increases the risk for subarachnoid hemorrhage (SAH) by up to 3.5%.



Smoking is directly responsible for a greater percentage of total strokes in younger than older adults. Risk factors other than smoking (HTN, heart disease, DM) account for more strokes in older adults.

Heavy smokers are at greater risk than light smokers. Good news! The relative risk of stroke decreases immediately after quitting, with a major reduction seen after 2 to 4 years. It may take several decades for a former smoker's risk to drop to that of a nonsmoker.

Smoking accelerates the rate of atherosclerosis progression by 50%, particularly in those with DM & HTN and increases clotting factors, such as fibrinogen. Smoking also increases the damage that results from stroke by weakening the endothelial wall of the cerebrovascular system. This leads to greater damage to the brain from events that occur in the secondary stage of stroke.

Carbon monoxide in smoke displaces O<sub>2</sub> from hemoglobin, interfering with O<sub>2</sub> delivery to tissues.

- **Physical inactivity; obesity:** Obesity strains the entire cardiovascular system. Pts are likely to have high cholesterol, HTN, & DM. Moderate to vigorous-intensity exercise is associated with an overall 35% reduced risk of stroke.
- **High alcohol consumption:** Generally, an increase in alcohol intake leads to an increase in BP. While heavy drinking is a risk for both hemorrhagic and ischemic strokes, studies affirm that daily consumption of smaller amounts of alcohol provides a protective influence against ischemic stroke by decreasing the clotting ability of platelets (similar to the effects of aspirin). Heavy alcohol consumption may seriously deplete platelets and compromise blood clotting and blood viscosity, leading to hemorrhage. Heavy (or binge) drinking can lead to a rebound effect after the alcohol is purged from the body causing blood viscosity and platelet levels to skyrocket, increasing the risk for ischemic stroke.



- **Cocaine** enhances other risk factors, such as HTN, heart and vascular dx. It decreases relative CBF by up to 30%, causes vasoconstriction and inhibits vascular relaxation. Repeated exposure can lead to blood flow deficits that persist long after use has ended, causing permanent damage. Also causes dysrhythmias rapid HR; and 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.



**Marijuana smoking:** Decreases BP and may interact with other risk factors, such as HTN and cigarette smoking, to cause rapidly fluctuating BP levels, damaging blood vessels.

- **Other drugs of abuse** (amphetamines,

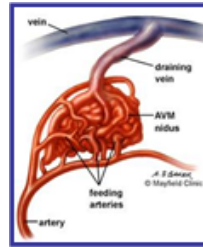


heroin, anabolic steroids; some common legal drugs, such as caffeine, L-asparaginase and pseudoephedrine found in over-the-counter decongestants; and phenylpropanolamine (over-the-counter "diet pills" and cold and flu pills) increase incidence of stroke in those < 40. Many are vasoconstrictors, causing BPs to rise. Ecstasy (MDMA): deaths are related to massive cerebral hemorrhage.

- **Head and neck injuries:** May damage the cerebrovascular system and cause a small number of strokes. Traumatic brain injury may cause bleeding within the brain leading to damage like a hemorrhagic stroke. Neck injury, when associated with spontaneous tearing of the vertebral or carotid arteries caused by sudden and severe extension of the neck, neck rotation, or pressure on the artery, is a contributing cause of stroke, especially in young adults. Often called "beauty-parlor syndrome," referring to extending the neck backwards over a sink for hair-washing. Neck calisthenics, "bottoms-up" drinking, and improperly performed chiropractic manipulation of the neck can also put strain on the vertebral and carotid arteries, possibly leading to ischemic stroke.
- **Infections:** Recent infections may act with other risk factors to add a small risk. The immune system responds to infection by increasing inflammation and the infection-fighting properties of blood. This increases the number of clotting factors leading to an increased risk of embolic-ischemic stroke.
- **Genetic risk factors:** Although there may not be a single genetic factor associated with stroke, genes play a large role in the expression of stroke risk factors such as HTN, heart disease, DM, and vascular malformations.



- **Vascular malformations** may have the strongest genetic link of all risk factors. A vascular malformation is an abnormally formed blood vessel or group of blood vessels. One genetic vascular disease called CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) is a rare, genetically inherited, congenital vascular disease of the brain that causes strokes, subcortical dementia, migraine-like headaches, and psychiatric disturbances.



CADASIL is debilitating and symptoms usually surface around age 45. The exact incidence of CADASIL is unknown. (*Circulation*, Jan. 2013)

Previous stroke or transient ischemic attack (TIA) also raises the risk of (recurrent)

stroke.

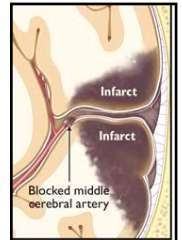
## Classifications of stroke – 2 main types (plugs & leaks)

**Overview:** A stroke occurs when the blood supply to part of the brain is suddenly interrupted (plugs) or when a blood vessel in the brain leaks, spilling blood into the spaces surrounding brain cells (NINDS, 2016).

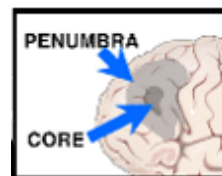


Brain cells become injured (**ischemic**) when they receive inadequate O<sub>2</sub> (perfusion) or when they are damaged by sudden bleeding into or around them.

Unchecked, ischemia ultimately leads to **infarction** (death of brain cells).



When blood flow is interrupted, some brain cells die quickly (within minutes), while others remain at risk for death. These damaged cells make up the **ischemic penumbra** and can linger in a compromised state for several hours. Collateral circulation to the penumbra occurs 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 penumbra may be sufficient to preserve brain, but not enough to sustain normal function until perfusion is restored. With timely treatment these cells can be saved.



Within a few months of the infarction, the necrotic brain cells are reabsorbed by macrophage (WBC) activity, leaving a very small fluid-filled cavity referred to as a lake (or lacune in French) in the injured brain (NINDS, 2016).

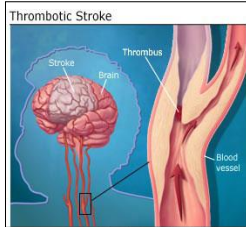
**Ischemic strokes:** 87% are caused by a blood vessel occlusion due to a clot, plaque or embolus.

AHA reports that for 30-40% of ischemic strokes, no cause is found (*Circulation*, 2013). This type is called a **cryptogenic** stroke.



## Thrombotic Stroke

**Central thrombus:** Large-vessel occlusion due to atherothrombotic disease causes 14% of ischemic strokes. A clot forms in turbulent flow areas of a large artery or plaque suddenly obstructs CBF. The clot may partially or completely block CBF.



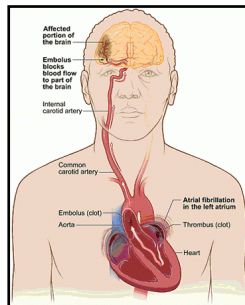
Both large and small vessel occlusions cause reduced tissue perfusion leading to neurotransmitter failure, anaerobic glycolysis, cerebral anoxia, and cerebral edema.

## Embolic Stroke

**Definition:** Partial or complete blockage in a cerebral artery from embolic material, 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 S&S 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

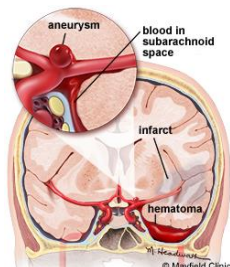
**Incidence:** Affects 37,000-52,000 persons/ yr in US. Most common in middle aged, African Americans, Asian (particularly Japanese), and those with HTN. About 50% of strokes in younger patients are hemorrhagic (ruptured congenital aneurysms or AV malformation); only 20%-25% are hemorrhagic in older patients.

**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

Refers to the nontraumatic presence of blood within the



subarachnoid space from some pathologic process, usually from rupture of a berry aneurysm or arteriovenous malformation (AVM) (Zebian, 2015).

Especially prevalent in 35-65 yr olds, SAH accounts for about 3% of all strokes; afflicting ~30,000/yr in U.S.

Risk factors for SAH: HTN, smoking, heavy alcohol use, use of sympathomimetic drugs and presence of familial genetic predisposition.

Mortality rate reaches as high as 40% within the first week, and ~50% die in the first 6 months (Zebian, 2015).

**Clinical presentation** of a higher grade SAH is "one of the most distinctive in medicine" (Stroke Assoc).

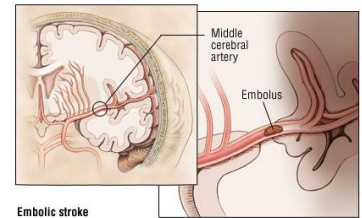
About 20% experience a warning or "sentinel" headache between 2 and 8 weeks before the SAH event. Headache w/ the warning bleed is usually milder than that of the SAH event, and may be accompanied by nausea & vomiting.

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.

Emergent management of SAH, including prehospital care, is critical. EMS is the first medical contact for about 2/3 of SAH patients. An estimated 10-15% die before reaching the hospital. Rapid assessment & transport with advanced notification of the ED is crucial for patients 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.



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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 neuron death.



The hematoma also has the potential to disturb normal intracranial dynamics causing a sudden rise in ICP, 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. Many patients with smaller ICHs will survive if prompt and aggressive medical care is received.



The patient's GCS may decrease  $\geq 2$  points between initial EMS assessments and the ED evaluation. Early deterioration without aggressive intervention is associated with poor long term outcomes (> 75% mortality). Advance Stroke Alert notification correlates with shortened time to evaluation and CT scan.

#### ICH risk factors

- Uncontrolled HTN (primary & most easily modified)
- Advancing age
- Cerebral amyloid angiopathy
- Neoplasms (tumors) with fragile blood vessels
- Trauma
- Vascular anomalies (AVMs and aneurysms)
- Coagulation disorders; sickle cell disease, hemophilia
- Collagen-vascular disease
- Venous thrombosis
- Drugs: Anticoagulants, amphetamines (vasculitis), cocaine, oral contraceptives; excessive alcohol intake
- Septic emboli, infective endocarditis, infected valve prosthesis

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

The brain depends on a constant level of perfusion (**cerebral blood flow** of  $\sim 750$  mL/min or 55 mL/100 gm tissue/minute). One needs a balance of flow and metabolism ( $O_2$  demand & supply). The brain only functions normally if CBF is  $>20$  mL/100 gm of brain tissue/min.

Perfusion is disrupted by changes in blood flow and **cerebral perfusion pressure** (MAP- ICP). Any condition that affects perfusion has a rapid effect on brain function depending on the areas that are impacted (global or focal).

Vascular occlusion or disruption results in decreased  $O_2$  and glucose delivery to the affected cells and increased  $CO_2$  and lactic acid accumulation.

CBF is autoregulated over a wide range of blood pressures (60 to 180 mmHg). It is lost with extremes of hyper or hypotension, severe brain damage with cerebral edema and increased ICP.

When stroke occurs and the brain becomes ischemic, blood flow is regulated by "whatever head of pressure is pushing it" and small changes in MAP and cardiac output can cause drastic alterations in cerebral blood flow.

If flow drops  $<12-20$  mL/100 gm/min, brain cells become electrically silent (as manifested by paralysis) but not dead. **Early mgt is critical to restore perfusion and function.**

#### Chemical controls – very important to EMS

- Excess  $CO_2$  – dilates vessels
- Low  $CO_2$  (hyperventilation) constricts vessels
- Low  $O_2$  – (hypoxia) dilates vessels
- Excess  $O_2$  (hyperoxia) constricts vessels

#### Evolution in stroke care

Until the 1990s, care of patients experiencing stroke was **largely supportive**, focusing on treatment of respiratory and cardiovascular complications while the effects of the stroke became complete. *No specific therapy was available to alter the course and extent of the evolving physical losses.* Thus, limited emphasis was placed on rapid EMS interventions or transport. We also thought oxygen was good and not harmful for everybody!

**THIS HAS CHANGED!** Fibrinolytic drugs and advanced interventional therapies at stroke centers now offer an opportunity to possibly limit

the extent or reverse neurological damage and improve outcomes in select patients with ischemic stroke. It's a whole new era of standards and care!

## Stroke Systems of Care

### Expected benefits

- Improved efficiency of care
- Increased use of acute stroke therapies
- Fewer peri-stroke complications
- Reduced morbidity and mortality
- Improved long-term outcomes
- Reduced health care costs (NMH)

Identification of stroke patients and rapid mobilization of stroke teams is now commonplace in US

- Average door to needle times < 60 minutes
- Opportunities still exist to achieve radical changes (< 20 min)
- Workflow efficiencies are engineered into endovascular interventions at comprehensive centers
- Will continue to improve stroke care as systems approaches are adopted and implemented

The best **team approach** to stroke management **starts with dispatch and EMS** and continues at a hospital within a system of care capable of delivering evidence-based stroke care from the ED to a dedicated stroke unit.

**Table. Stroke Chain of Survival**

<b>DETECTION</b>	Recognition of stroke signs and symptoms
<b>DISPATCH</b>	Call 9-1-1 and priority emergency medical services dispatch
<b>DELIVERY</b>	Prompt transport and prehospital notification to hospital
<b>DOOR</b>	Immediate emergency department (ED) triage
<b>DATA</b>	ED evaluation, prompt laboratory studies, and computed tomography imaging
<b>DECISION</b>	Diagnosis and decision about appropriate therapy
<b>DRUG</b>	Administration of appropriate drugs or other interventions

**Source:** The American Heart Association. The links in the chain of survival [online]. [cited 2008 Feb 20]. Available from Internet: <http://www.americanheart.org/presenter.jhtml?identifier=3012016>.

### Detection

85% of strokes occur at home. Early recognition

depends on patient, family, or other bystanders witnessing the event and recognizing the urgency of the symptoms.

### Barriers: Lack of stroke awareness

Symptoms are often subtle. Many fail to seek help quickly enough to be eligible for fibrinolytic therapy. Mellor et al (2015) found that delays arose from 3 sources:

- Patients' lack of recognition (or perhaps denial) of the significance of their symptoms,
- A decision to first contact primary rather than emergency medical services care, and
- Lack of recognition of the significance of S&S by the initial health care providers.

Efforts should focus on broader public awareness of critical signs of stroke, and specific directives to engage emergency rather than primary services. Public education efforts, such as the FAST campaign, contribute to increased awareness.

### EMS and Stroke

The role of EMS in stroke systems of care is so critical that the AHA & ASA have issued a policy statement and guidelines specific to EMS.

Central to the success of EMS stroke care is a commitment to **time-sensitive delivery of care**.

The "Implementation Strategies for EMS within **Stroke Systems of Care**" policy 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 enroute < 1 min*
- *On-scene time < 15 min. in the absence of complications (NWC EMSS goal is ≤ 10 minutes)*
- *Transport time equivalent to those of AMI or trauma*



### Dispatch (EMD)

Dispatchers should have training in stroke S&S and must recognize and classify potential stroke patients in the same category as AMI and severe trauma. EMDs should give appropriate pre-arrival instructions and dispatch all patients w/ stroke





at the highest level of care in the shortest time possible.

### Delivery

**Primary EMS goals** for patients with stroke symptoms: **timely response, rapid (neuro) evaluation, appropriate stabilization, and expeditious transport to a stroke center** according to current guidelines.

EMS personnel should rapidly and appropriately assess for stroke using a validated 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. Region IX modified the stroke assessment portion of the SOPs to expand on the CSS as it did not detect posterior strokes. The new assessment components more closely align with the MEND scale as we use BEFAST.

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.

### Time-sensitive patient



**TIME IS BRAIN!** In cardiac arrest, the 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, ~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).

**Expedite transport:** EMS personnel must be aware of the possibilities for early stroke interventions at hospitals and appreciate the **need for short scene times**.

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 patient's presentation.

If scene time is longer than 10 minutes, include information in the comments/narrative to support why.

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

**Hospital stroke alert:** Notify the nearest System hospital for OLMC ASAP if S&S of stroke started within the past 6 hours. Patients whose arrival is expected are more likely to receive early CT and appropriate interventional therapy. OLMC 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 evaluated by the stroke team (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

Remove barriers to rapid treatment: prioritize CTs, early read of CT, immediate consult with neurologists, location of fibrinolytics, pharmacists in the ED.

**Drug:** Ideally, tPA should be administered (“door to drug”) in less than 60 minutes after ED arrival. However, it **must** be given within 4.5 hours from the onset of symptoms. 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. New SOPs have a decision tree to transport to a

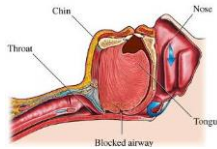
Comprehensive vs. Primary Stroke Center destination.

## Primary assessment/resuscitative interventions

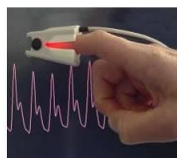
See Stroke SOP: IMC special considerations p.35

### Support ABCs as needed

- Ideally, avoid sedating these patients
- Airway obstruction is a major problem, especially if pt loses consciousness. Paralysis of the muscles of the throat, tongue, or mouth can lead to partial/complete upper airway obstruction. Saliva may pool in throat and be aspirated.
- Aspiration is a serious complication, associated with considerable morbidity and mortality.
- Open the airway with BLS manual maneuvers and secure with nasopharyngeal and/or oral pharyngeal airways as indicated.
- Do not allow a patient to eat or drink until swallowing ability has been assessed by ED staff.
- **If GCS  $\leq 8$  (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 may be sufficient.

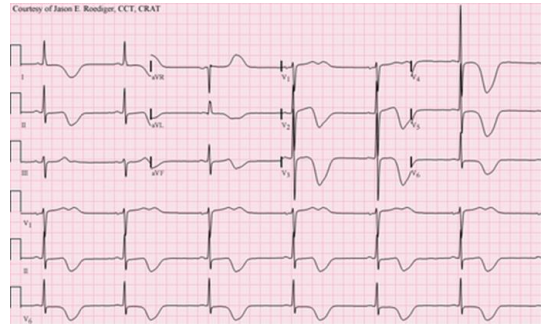


- Assess **ventilatory status (ETCO<sub>2</sub>/ gas exchange (SpO<sub>2</sub>))** at least q. 15 min. Give O<sub>2</sub> if SpO<sub>2</sub> < 94% or O<sub>2</sub> sat unknown;



**avoid hypoxia and hyperoxia.**

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  $\uparrow$  ICP and cardiac and respiratory instability. Patients with hx of cardiac and/or pulmonary disease are at  $\uparrow$  risk for hypoxia. Assist ventilations at 10 BPM prn w/ 15 L O<sub>2</sub>/BVM prn.



- **Seizure/vomiting precautions;** suction only as needed. 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.
- **Positioning:** Can affect O<sub>2</sub> sats, CPP, and ICP. Maintain head/neck in neutral alignment to optimize perfusion and venous outflow from head. Do not use pillows. If SBP > 100: Elevate head of bed 10° - 15°. May need to use lateral immobilization devices to keep head from turning to the side. Do not flex knees or hips to avoid elevating ICP.
- Assess **cardiovascular status/ perfusion adequacy**

- **Monitor ECG; acquire 12L.** 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  $\uparrow$  ICP. Bradycardia w/ AMS and a SBP >90 should NOT be treated with atropine, norepinephrine, or pacing.

**Tachycardia** may indicate hypoxia/hypercarbia.

**A-Fib:** 15% of pts with stroke have AF

**SAH.** Pathological Q waves, ST elevation or depression; prolonged QTc, **wide, large & deeply inverted (neurogenic or cerebral) T waves;** prominent U waves > 1 mm amplitude common causing incorrect suspicion of myocardial ischemia

- **IV usually unnecessary at scene** unless DAI is indicated or hypoglycemic. Avoid multiple attempts. Hospital may ask for lg. (18-20 g) antecubital IV to be placed so pt. can go directly to CT (prepped



for infusion). 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. Excess fluids (IV) will worsen this edema and increase damage to brain tissue.

Excess fluid loading is harmful in stroke; scene times are shortened if IVs are deferred or started

- **Disability/mental status** exam: See p.3  
**If AMS:** Consider possible causes of AMS using the AEIOU-TIPs mnemonic in the SOP p. 27.

- **If generalized tonic/clonic seizure activity:** Observe and record seizure activity per SOP p. 40

**MIDAZOLAM 2 mg** increments **IV/IO** q. 30-60 sec (0.2 mg/kg **IN**) **up to 10 mg** prn titrated to stop seizure. If IV unable and IN contraindicated: **IM** 5-10 mg (0.1-0.2 mg/kg) max 10 mg single dose.

All routes: May repeat prn to a total of 20 mg if SBP  $\geq$  90 (MAP  $\geq$  65) unless contraindicated

If hypovolemic, elderly, debilitated, chronic dx (HF/COPD); and/or on opiates or CNS depressants:  $\downarrow$  total dose to 0.1 mg/kg.



- If AMS, seizure activity, or neurologic deficit:** Assess **blood glucose**  
Midazolam administration takes precedence over bG determination in pts who are actively seizing

**If < 70 or low reading: DEXTROSE 10%**  
Do not give D10% without evidence of hypoglycemia.

**If no vascular access: Glucagon** per SOP

- Hypoglycemia = life threat
- Hyperglycemia is harmful

Glucose levels >200 during stroke are associated with poor outcomes. Studies demonstrated that hyperglycemia increased brain water content and edema related to vascular dysfunction (Stroke, 2013).

- Provide comfort and reassurance; establish means of communicating with aphasic patients

- Limit activity; do not allow pt to walk; protect limbs from injury

## SECONDARY ASSESSMENT

### 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  $\uparrow$  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.

EMS should not lower a high BP unless signs of acute MI or LV failure/HF. Severe HTN (SBP > 220 mmHg or DPB >120 mmHg on three repeated measurements made at 15 min intervals) is treated in the hospital.

**Monitor temperature:** 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.

Repeat VS frequently & after each intervention.

**S&S** that develop after a stroke or TIA depend on the area of the brain affected and extent of injury. 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: S&S/Chief complaints (OPQRST) - Ask about:**

Three critical questions must be asked and answered:

1. Did the pt experience a severe headache or seizure at the onset of symptoms?
2. Is the pt taking any type of blood thinner?
3. When was the pt last known well (normal for them)? When did S&S start?



**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 posterior strokes as well as 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**, **vision** deficits

**New onset seizure**

**LAST KNOWN WELL:** Many ischemic strokes occur at night or early morning when the patient is sleeping or just beginning to wake (wake up stroke), so last known well and symptom onset may be difficult to obtain.



**Try your best to determine:**

**Date/Time pt last known to be well** (normal for them)

\_\_\_/\_\_\_/\_\_\_ : \_\_\_  
MM/DD/YYYY

**Time of discovery same as last known well?**

**Date/Time of discovery of stroke symptoms**

\_\_\_/\_\_\_/\_\_\_ : \_\_\_  
MM/DD/YYYY

Unknown

Stress the importance of this info with pt, family

members or caregivers. 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 the pt was last known well (normal for them) as accurately as possible and try to pinpoint when acute S&S began.**

**Categorize symptom duration** to determine destination per transport decision tree

☐ < 3.5 hours

Determine if reported S&S resolved before EMS arrival or in your presence to suspect TIA.

**A: Allergies**

**M: Medications/drugs:**

**Antihypertensives:** ACE Inhibitors; Beta blockers; Diuretics; ARBs; Ca<sup>++</sup> Channel Blockers; Other anti-hypertensives; none

**Cholesterol reducing drugs:** Statin; Niacin; Fibrate; Absorption Inhibitor; none

**Anticoagulants/antiplatelet drugs**

**P: Past Medical History:** Ask about **risk factors** (previously covered) as they may provide clues to presence/type of stroke:

- ☐ None
- ☐ Atrial Fib/Flutter
- ☐ AV malformation, tumor or aneurysm
- ☐ Bleeding 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
- ☐ CAD/Prior MI: Heart/vascular dx (Within 5 yrs after an AMI, 8% men & 11% women will have a stroke)
- ☐ Carotid stenosis
- ☐ Current Pregnancy (or up to 6 weeks post-partum)
- ☐ Depression
- ☐ Diabetes Mellitus

- ☐ Drugs/Alcohol Abuse
- ☐ Dyslipidemia (high cholesterol)
- ☐ Family history of stroke
- ☐ HF
- ☐ HRT (hormone replacement therapy)
- ☐ Hypertension
- ☐ Migraine
- ☐ Obesity/Overweight
- ☐ Previous Stroke
- ☐ Previous TIA
- ☐ Following TIA:
  - 12% experience stroke within next 30 days
  - 3-17% have stroke within 90 days
  - 25% die within a year
- ☐ Previous intracranial or intraspinal surgery, intracranial hemorrhage, or serious head trauma
- ☐ Prosthetic Heart Valve
- ☐ PVD (peripheral vascular disease)
- ☐ Renal insufficiency – chronic (SCr>2.0)
- ☐ Sleep Apnea
- ☐ Smoker

#### Helpful to document patient's baseline function prior to this acute event: (if able)

- ☐ Able to ambulate independently (no help from another person) w/ or w/o device
- ☐ With assistance (from person)
- ☐ Unable to ambulate

L: Last oral intake

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

**Age of pt:** Obtain DOB (age) and patient weight. Age > 55 is an important *unmodifiable* risk factor.

**Hereditary:** Family hx CV disease or stroke; ethnicity.

**Social history: (Core health behaviors):** diet, exercise, alcohol consumption.

#### Prehospital Stroke Screen

See SOP. Clinical S&S of acute stroke vary widely, including findings not picked up by the CSS and often fluctuate based on the type and location of focal ischemia. Need to complete and repeat a sufficient neuro exam to find/suspect posterior as well as anterior strokes. Exam does not take that long and yields important information.

Determine patient's **normal neuro baseline** if possible to detect new onset changes. Also assess for head trauma, infection, meningeal irritation, and vertigo (room spinning).

- Level of consciousness; GCS
- Response to commands: Open/close eyes
- Questions: Age, month (orientation)
- **Speech:** "You can't teach an old dog new tricks." Slurred? Uses wrong words? Mute?

#### Mental status overview

##### Assess and score GCS.

Also assess Orientation X4, memory, affect, behavior, cognition, Hallucinations? Note if sedated.

When onset/LKW is > 3.5 hr with acute S and S of stroke AND transport time is < 15 min longer than to nearest PSC, the patient is a candidate for a Comprehensive stroke center.

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. 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.

#### Speech assessment

Speech abnormality is often one of the first signs of stroke and is generally 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, slurred speech or mispronunciation of words due to disturbances of muscular control (also common with intoxicated persons).



Don't assume lack of speech or response means coma (when scoring GCS). Patient may have receptive or expressive aphasia.

**Motor (expressive) 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. Hold up two objects. Ask them to point to one of them using an arm with good motor strength.

#### Document 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.

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

**Research says:** BMC Neurol. 2013 Jul 15;13:87.

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

**Abstract** 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 cases.

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

**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.

#### Cranial nerves – overview

Other deficits to assess: pupil changes; light or sound sensitivity, deviated uvula; hoarse voice; vertigo/dizziness.

**Smile/facial asymmetry** (CN VII):

- **Facial asymmetry/droop:** smile, show teeth
- **Vision deficits:** loss of visual fields; diplopia
- **Horizontal gaze abnormalities:** dysconjugate gaze, forced or crossed gaze
- Other: pupil changes; light/sound sensitivity, deviated uvula; hoarse voice; vertigo/dizziness

**Exam:** Inspect face for asymmetry, tics, or abnormal movements. Have patient smile, show their teeth, tightly close eyelids, puff out cheeks, and pucker lips. Observe for weakness on one side or flattening of the nasolabial fold. When eyelids are tightly closed, assess if one side does not close as well (shows more lashes than the other).



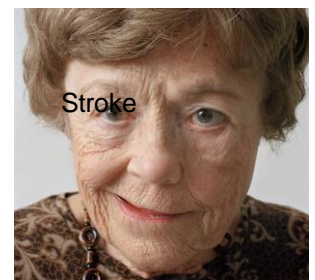
**Normal:** No facial droop; both sides move symmetrically and both eyes close the same

**Abnormal/dysfunction:** Stroke generally presents with weakness/paralysis of the lower face (below the eye) on one side due to bilateral nerve connections to the forehead and eyelids from each hemisphere. The patient has difficulty elevating one corner of the mouth (asymmetric smile), but can lift both eyebrows and wrinkle both sides of the forehead. *Bell's palsy* or peripheral 7<sup>th</sup> nerve damage results in total ipsilateral hemi-facial paralysis and they cannot wrinkle their forehead on one side.



Bell's Palsy

**Any facial asymmetry (new or old), is documented as abnormal on the stroke screen.**



Stroke

**Assess for vision disturbances**

- **(CN II) 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/obstructed 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.

### Pupil size, shape, symmetry, reactivity (CN II & III)

- 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.

### Eye movements (CN III, IV, & VI): gaze abnormalities

Ask pt to hold head still and follow your finger with their eyes as you move it to the extreme gaze limits: Lt & Rt, up & down (trace large H). If pt moves their head, place your finger on their chin to hold head still.

Eyes should move together without stuttering or pain. Nystagmus does not affect a normal score, but should be noted. If either/both eyes are unable to track completely in one direction or another, note assessment as abnormal (Whitehead, 2012).

In a Rt hemispheric stroke, the pt may have difficulty moving their eyes across 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.

**Document best gaze** in the comments section as normal, or describe abnormal findings. Gaze deviation helps localize a stroke. Eyes will look toward the affected side of the brain. Right sided

stroke; eyes look to the right.

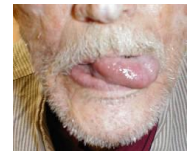


VIII: **Hearing/balance** loss? Sound sensitivity?

**Inspect 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*



- **Unilateral weakness or paralysis (arm drift)**
- **Leg drift:** Open eyes; lift each leg separately
- **Sensory:** Arm/leg (close eyes):point location; sharp/dull; note paresthesias
- **Coordination** – arm/leg Note loss of

### MOTOR assessment overview

#### Arm drift

Weakness, clumsiness, or heaviness. Involves hand, arm, face, or leg, alone or in combination - most commonly the hand and face. 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 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:** Patient can hold position for 10 seconds with no movement or no drift at all

#### Abnormal (new or old)

- Drift: Limb holds 90° (or 45°) briefly, 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 voluntary movement: **Paralysis**

If either frontal lobe has a dysfunction, the opposite (contralateral) side of the body will experience motor loss as 80% of the motor fibers cross in the medulla. If both arms or legs are affected, consider spinal cord lesion although some TIAs/strokes can cause bilateral losses.

### SENSORY deficits

- Assess for sensory loss, tingling, or abnormal sensations 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 to the side of the diseased artery.
- **Point location.** Alternately touch patient on face, arms, and legs with the dull and sharp sides of a partially opened paperclip (or end of monofilament like fish line). Patient should be able to localize where the stimulus is being applied and discriminate if stimulus is dull or sharp. Rate sensory integrity from normal/no loss to total sensory loss/not aware of touch.



### Alterations in sensory function

- Hypalgesia: Decreased sensation
- Analgesia: No sensation
- Hyperalgesia: All touch is painful
- Paresthesia: Alteration in sensation (pins and needles)

**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.

- Test extinction by touching the patient on both sides at once. If they are able to feel only one side, they have parietal lobe disease.
- Rate from no abnormality to profound hemi-inattention.

**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.

Test upper extremities by having the pt use one finger to touch their nose and then touch your fingertip (light on an object); perform rapid alternating movements by rapidly pronating and supinating both hands or sequentially touch each finger in rapid succession to the thumb.

To test the lower extremities, have them run the heel of one foot down the shin of the opposite leg. If a stroke is suspected, do not walk the patient or have them stand up to check for balance or ataxia.



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

**Pseudobulbar affect:** Note any sudden outbursts of uncontrollable and inappropriate emotions such as laughing or crying.

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

### Environmental controls

Provide comfort and reassurance. Elevate side rails after ensuring that limbs are clear; protect weak/paralyzed extremities and use appropriate stretcher straps.

**Transport decision:** **TIME SENSITIVE Patient:**

Once stroke is suspected, minimize scene time to ≤10 minutes. Transport to the nearest appropriate stroke



**center per decision tree in SOP.** Alert nearest System hospital OLMC ASAP with a **stroke alert.**

**The selected hospital depends on the following:**

- Acute onset S&S that suggest new stroke
- Patient stability
- Last known well time (<3.5)
- Co-morbid factors (need Comprehensive SC)
- Travel time < 15min to the closest CSC in relation to the PSC.



If witnesses/reliable historian cannot accompany patient to the ED, EMS should **obtain their contact information/call back number** in the event hospital personnel wish to obtain more information after

patient arrival.

**Differential diagnosis of stroke: Consider other causes of presenting S&S ("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 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, prolapsed 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 (numbness) w/ assoc. 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 or "read" context of body language
- Apathy; Pseudobulbar affect

### Medical

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



**EMS/ECRN Documentation:** see screens in appendix

- **Patient demographics:** DOB, gender, weight
- **Chief complaint**  
Note if acute S&S resolved prior to hospital arrival
- Primary assessment/resuscitative interventions
- Vital signs (at least 2 sets; more if unstable)
- **History:** SAMPLE – especially last known well; onset S&S; on anticoagulant?
- **Physical exam findings**
  - GCS; orientation
  - **Speech disturbances;** slurred vs. aphasia
  - Cranial nerve disturbances: vision, visual fields, diplopia, EOMs, gaze palsy (one or both eyes), **facial droop**
  - Motor exam: Weakness/Paresis (**arm drift**)
  - Cerebellar exam: Ataxia; incoordination; vertigo
  - Sensory changes
  - Other neurological signs/symptoms
- **OLMC contact; called stroke alert**
- **Call back number for reliable historian**

### Hospital categorization of stroke etiology

- Large-artery occlusion/atherosclerosis (e.g., carotid or basilar)
- Cardioembolism (e.g., AF/flutter, prosthetic heart valve, recent MI)
- Small-vessel occlusion (e.g., subcortical or brain stem lacunar infarction <1.5 cm)
- Stroke of other determined etiology (e.g., dissection, vasculopathy, hypercoagulable or hematologic disorders)
- Cryptogenic stroke (stroke of undetermined etiology)

**Hospital interventions:** *(Nice to know only for EMS)*

### 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

Patients who do not qualify for IV tPA may still be

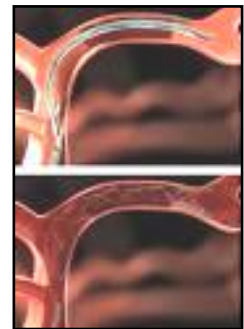
candidates for other, advanced therapies for stroke.

**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 mainstay of treatment has long been tPA. But for people with blood clots in larger arteries, tPA often does not dissolve them completely.

Mechanical thrombectomy is usually done within six hours of acute stroke symptoms, and only after the patient receives tPA. However, new research suggests this procedure can be done safely up to 7.3 hours after S&S begin (Saver et al, 2016). To remove the clot, doctors thread a catheter through an artery in the groin up to the blocked artery in the brain. The stent opens and grabs the clot, allowing doctors to remove the stent with the trapped clot. Special suction tubes may also be used.



Stent retrievers are being used at hospitals across the US, including comprehensive stroke centers and some primary stroke centers certified by the AHA/ASA and the Joint Commission.

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.

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.

Those patients with uncontrolled HTN, internal bleeding, seizures, blood clotting problems, or those on anticoagulants are not candidates for this procedure.

**Picture to puncture:** 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.

Studies have shown that when clot retrieval is performed within eight hours of symptom onset, 60-68% of patients achieved post-procedure revascularization.

Complications from clot retrieval procedures include hemorrhage, internal bleeding, death and recurrence of stroke.

### **Patient outcomes**

Hospitals score patients according to functional outcomes following stroke using the scale below:

#### **Stroke outcome scale**

- 5 Patient bedridden and incontinent
- 3 Needs assistance and is not able to work between the two groups.

- 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**

**'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. A 24-month study done in London, 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 hemorrhage rates

FRONTAL LOBE	
Function	Dysfunction
Inhibitions (social/sexual)	Loss of self-restraint
Input into autonomic function	Autonomic dysfunction in BP/dysrhythmias
Inhibitory effect on spinal cord spasticity	Spastic paralysis if UMN lesion
Repress primitive reflexes like sucking, rooting	Re-expression of primitive reflexes

PARIETAL LOBE	
Function	Dysfunction
<b>Primary sensory interpretation area for opposite side:</b> pain, pressure, touch and vibration; localization of stimuli; position sense (proprioception)	Impaired sensation, loss of two point discrimination; loss of left/right discrimination; loss of double sided stimulation (DSS)
<b>Non-dominant hemisphere:</b> Sensory association area; spatial orientation. <b>Dominant hemisphere:</b> Important role in comprehending symbols: mathematical and those used for non-spoken words.	Astereognosis; general or sensory seizures, neglect syndrome; ↓ proprioception: common in elderly
<b>Sensory speech area</b> - Ability to interpret spoken word; abstraction; interprets proverbs; calculations; test using money	Global dysphasia
Ability to read & write	Construction apraxia
Body image	Distorted body image

TEMPORAL LOBE	
Function	Dysfunction
<b>Primary auditory area/auditory assoc. area;</b> recognize familiar voices or sounds. Integrates sensory data related to vision and hearing.	Auditory hallucinations. Hallucinations triggered by excess release of dopamine. Drugs such as PCP, LSD all cause dopamine surge - as does schizophrenia. Abrupt cessation of alcohol causes dopamine rebound that causes the hallucinations and DTs. Partial complex seizures; most common type.
<b>Dominant hemisphere:</b> <i>Wernicke's area:</i> Ability to organize speech, esp. understanding and responding to verbal input.	Receptive (Wernicke's) aphasia. Global aphasia with hemispheric strokes - both sides.
Interprets and discriminates smells; <i>uncus</i> (medial section)	Aura prior to seizure: funny taste or smell.
Connects w/ frontal lobe, hypothalamus, and limbic system. Anterior lobe: behavior, emotion and personality (through <i>limbic system</i> ). Influences autonomic functions.	Behavior changes. Irritability; agitated angry behavior after head injury. Lose use of language if dominant hemisphere. ↑ sexual activity - limbic system. Dysfunctional feeding activities.
Memory storage - recent and past: <i>Hippocampus</i> (first to go with aging) Right lobe: primarily visual memories Left lobe: names and factual memories	Cells most sensitive to hypoxia. Can't lay down memory so patient develops retrograde amnesia. Drinking causes direct toxicity to those neurons.

OCCIPITAL LOBE	
Separated from cerebellum and brain stem by the tentorium	
Function	Dysfunction
Primary visual processing area; interprets light/dark; shape, shadow & motion; color and interpretation.	Visual agnosia; hemianopsia (loss of visual field); cortical blindness; nerve OK can see, but doesn't know what it is.
Visual association area; L side reads; R side sees, but does not read	Visual hallucinations

**NWC EMSS/NCH Paramedic Program Skill Performance Record**

**Neuro Assessment: Stroke**

Name:	Lab Buddy:
Date:	# attempts:

**Instructions to the participant:** You have 10 minutes to assess the patient, verbalize the prehospital interventions that are indicated and determine the most appropriate receiving hospital (Comprehensive or Primary Stroke Center).

Performance standard	YES	No
* Scene size up/safety; Determine nature of illness; scan environment for clues; apply appropriate BSI		
Determine need for additional assistance		
<b>PRIMARY ASSESSMENT</b>		
<b>*Airway:</b> Assess for impairment and assure patency <input type="checkbox"/> Manual airway maneuvers if needed <input type="checkbox"/> Verbalize if adjuncts are needed for airway access/control (BLS or ALS) <input type="checkbox"/> Aspiration risk? Verbalize seizure/vomiting precautions; suction would be standing by <input type="checkbox"/> Maintain head/neck in neutral alignment; do not use pillows. If SBP > 100: Elevate head of bed 10° - 15°		
<b>Breathing/ventilatory/gas exchange status;</b> assess for impairment <input type="checkbox"/> *Assess for spontaneous ventilations; general rate and pattern (normal, fast or slow) <input type="checkbox"/> *Assess depth; effort/WOB; accessory muscle use <input type="checkbox"/> Assess patient position, adequacy of air movement, symmetry of chest expansion, retractions <input type="checkbox"/> Lung sounds if in ventilatory distress <input type="checkbox"/> *Assess gas exchange; apply SpO <sub>2</sub> monitor; assess for hypoxia, cardiorespiratory or neurological compromise. Note before & after O <sub>2</sub> if able. Note signs of hypoxia <input type="checkbox"/> *Assess ETCO <sub>2</sub> number& waveform if possible ventilatory, perfusion, metabolic compromise. Verbalize if ventilatory assistance is needed w/ BVM		
<b>*Correct hypoxia/assure adequate ventilations per IMC:</b> Target SpO <sub>2</sub> : 94%. <input type="checkbox"/> O <sub>2</sub> if SpO <sub>2</sub> < 94% or O <sub>2</sub> sat unknown <input type="checkbox"/> If ≥94%: NO Oxygen; avoid hyperoxia		
<b>Circulatory status;</b> assess for impairment <input type="checkbox"/> *Pulses for presence, general rate/quality/rhythmicity <input type="checkbox"/> Skin (color, temperature, moisture, turgor) <input type="checkbox"/> *Verbalize need for ECG monitor: rhythm ID and 12 L for evidence of acute/old changes <input type="checkbox"/> *Assess need for immediate IV (DAI, hypoglycemia, hypotension); defer most IV starts to enroute <input type="checkbox"/> Verbalize OLMC may request Ig. bore antecubital IV as CT prep; <input type="checkbox"/> Avoid multiple attempts/excess fluid loading. NS TKO		
<b>Disability: explore causes of AMS</b> <input type="checkbox"/> If generalized tonic/clonic seizure activity: Observe and record per SOP <input type="checkbox"/> *MIDAZOLAM usual dosing for seizures <input type="checkbox"/> * If AMS, seizure activity, or any neuro deficit: Assess blood glucose per System procedure <input type="checkbox"/> * If < 70 or low reading: <b>DEXTROSE</b> / Glucagon per Hypoglycemia SOP		
<b>Exposure/environment</b> <input type="checkbox"/> Discretely undress pt to inspect approp body areas <input type="checkbox"/> Protect pt modesty, maintain body warmth		
<b>SECONDARY ASSESSMENT</b> <b>Vital signs</b> <input type="checkbox"/> *BP/MAP: <input type="checkbox"/> *Pulse: <input type="checkbox"/> Resp: <input type="checkbox"/> Temperature <input type="checkbox"/> Repeat VS frequently & after each intervention. Anticipate HTN & bradycardia due to ↑ ICP. <input type="checkbox"/> Do NOT Rx HTN or give atropine for bradycardia if SBP > 90 (MAP > 65)		

Performance standard				YES	No
<b>HISTORY</b>					
<input type="checkbox"/> <b>Attempt to determine baseline status:</b> dementia, pre-existing limitations/deficits, unable to care for self? <b>Chief complaint:</b> <input type="checkbox"/> Severe HA or seizure at onset <input type="checkbox"/> Head trauma at onset?					
<b>Stroke Screen</b>					
<b>B</b>	<b>BALANCE</b> /Coordination – Dizziness/vertigo? Balance problems/incoordination? Unsteady, fall? Assessment: Finger to nose, rapid alternating movements, heel to shin. Note <b>ataxia; tilting to one side, vertigo</b>	R	L		
<b>E</b>	<b>EYES:</b> Vision changes: blurred, diplopia, <b>loss of visual field</b> ; photophobia Eye position; ptosis. Horizontal gaze: <b>gaze palsy</b> or fixed deviation	R	L		
<b>F</b>	<b>FACE:</b> <b>Smile</b> , show teeth; <b>close eyelids</b> , wrinkle forehead Note unilateral weakness/asymmetry:	R	L		
<b>A</b>	Motor – <b>ARM</b> (close eyes and; hold out both arms for 10 sec). Note weakness, heaviness, paralysis. Score as Normal; Abnormal: Drift, some effort against gravity; no effort against gravity/no movement	R	L		
<b>S</b>	<b>SPEECH</b> (Repeat “You can’t teach an old dog new tricks” or sing Happy Birthday <input type="checkbox"/> Expressive/receptive aphasia <input type="checkbox"/> Dysarthria <input type="checkbox"/> Word substitution or retrieval deficits	<input type="checkbox"/> Normal <input type="checkbox"/> Abnormal			
<b>T</b>	<b>TIME last known well</b> /normal pt baseline <input type="checkbox"/> ≤3.5 hrs <input type="checkbox"/> >3.5 hrs up to 22 hrs	Time:			
<b>Other assessments</b>	<b>Level of consciousness:</b> AMS? GCS: E                      V                      M	Total GCS:			
	Orientation: Answers accurately: Name, age, month of year; location, situation	X (1-4)			
	Responds to commands: open/close eyes	Y	N		
	<b>Gross hearing</b> – Note new onset unilateral hearing deficit; sound sensitivity	R	L		
	Say “Ah”, palate rises, uvula midline; Stick out tongue: remains midline (note abnormalities)	R	L		
	<b>Neglect:</b> one sided extinction (visual, auditory, sensory)	R	L		
	<b>Motor:</b> Lift leg. Normal; Abnormal: drift to no effort against gravity	R	L		
	<b>Sensory:</b> Focal changes/deficits (face, arms, legs); paresthesias, numbness	R	L		
	<b>ANS:</b> Sweating only one side	R	L		
Neck stiffness (cannot touch chin to chest; vomiting)					
<b>*History of present illness</b>					
<input type="checkbox"/> Onset (suddenly) <input type="checkbox"/> Provocation/palliation <input type="checkbox"/> Quality <input type="checkbox"/> Region/radiation <input type="checkbox"/> Severity <input type="checkbox"/> Clarifying questions re: assoc. complaints <input type="checkbox"/> Date of birth <input type="checkbox"/> ~Weight					
<b>*Allergies</b> (meds, environment, foods)					
<b>PMH</b>	<input type="checkbox"/> None <input type="checkbox"/> <b>AF/Flutter</b> <input type="checkbox"/> AVM, tumor, aneurysm <input type="checkbox"/> Bleeding disorders <input type="checkbox"/> CAD/Heart dx <input type="checkbox"/> Cancer <input type="checkbox"/> Carotid stenosis <input type="checkbox"/> Pregnant (≤6 wks. post-partum) <input type="checkbox"/> Depression <input type="checkbox"/> Diabetes <input type="checkbox"/> Drug/Alcohol Abuse <input type="checkbox"/> Dyslipidemia <input type="checkbox"/> Family Hx stroke <input type="checkbox"/> HF <input type="checkbox"/> Hormone RT <input type="checkbox"/> <b>HTN</b> <input type="checkbox"/> Migraine <input type="checkbox"/> Obesity <input type="checkbox"/> <b>Previous stroke</b> <input type="checkbox"/> Previous TIA <input type="checkbox"/> Previous intracranial surgery/bleed <input type="checkbox"/> Head trauma <input type="checkbox"/> *Prosthetic valve <input type="checkbox"/> PVD <input type="checkbox"/> Renal failure <input type="checkbox"/> Sleep apnea <input type="checkbox"/> Tobacco use				
<b>MEDS</b>	<b>Anticoagulant use in 48 hrs:</b> <input type="checkbox"/> warfarin/Coumadin <input type="checkbox"/> apixaban/Eliquis <input type="checkbox"/> argatroban <input type="checkbox"/> dabigatran/Pradaxa <input type="checkbox"/> desirudin/Privask <input type="checkbox"/> edoxaban/Savaysa <input type="checkbox"/> enoxaparin/Lovenox <input type="checkbox"/> fondaparinux/Arixtra <input type="checkbox"/> LMW heparin <input type="checkbox"/> lepirudin/Refludan <input type="checkbox"/> rivaroxaban/Xarelto <b>Platelet inhibitors:</b> <input type="checkbox"/> ASA <input type="checkbox"/> clopidogrel/Plavix <input type="checkbox"/> dipyridamole/Aggrenox <input type="checkbox"/> prasugel/Effient <input type="checkbox"/> ticagrelor/Brilinta <input type="checkbox"/> ticlodipine/Ticlid <input type="checkbox"/> Cocaine/other vasoconstrictors, e.g. amphetamines: PCP				
<b>Last oral intake</b>					

Performance standard	YES	No
<b>Event surrounding this incident</b>		
<b>Review of Systems in addition to stroke screen</b>		
<b>Head:</b> DCAP, BLS, TIC		
<b>Chest:</b> DCAP,BLS, TIC; Auscultate breath sounds		
<b>Abdomen/pelvis</b> <input type="checkbox"/> Inspect <input type="checkbox"/> Palpate (guarding, rigidity)		
<b>Extremities:</b> <input type="checkbox"/> Palpate <input type="checkbox"/> Assess equality of peripheral pulses; evidence of trauma		
<b>Skin: Integumentary assessment</b> (integrated above) color (variation), moisture, temp, texture, turgor, lesions/breakdown; hair distribution; nails (clubbing)		
Psychological/social assessment		
<b>Considers stroke mimics</b> (below)		
<b>*Correct paramedic impression:</b> (Acute stroke)		
<b>Verbalize treatment plan</b> <input type="checkbox"/> Provide comfort & reassurance; establish means of communicating with aphasic patients <input type="checkbox"/> *Limit activity; do not allow pt to walk; protect limbs from injury		
<b>Decision tree for transport: Patient presents with S&amp;S new onset stroke</b> <b>*Minimize scene time (&lt; 10 minutes) - transport to the nearest PSC/CSC per Stroke Checklist</b> <input type="checkbox"/> Nearest hospital: Patient unstable <input type="checkbox"/> Nearest SC (Primary or Comp): Onset/LKW (normal baseline) <3.5 hrs with acute S&S of stroke <input type="checkbox"/> Nearest Comprehensive SC : Onset/LKW (normal baseline) >3.5 hrs (up to 22 hrs) with acute S&S of stroke AND Travel time <15 min longer than to nearest PSC <input type="checkbox"/> *Call <b>Stroke Alert</b> to OLMC ASAP if one or more criteria of BEFAST or other assessments is positive		
<b>Critical Criteria - Check if occurred during an attempt</b> <input type="checkbox"/> Failure to initiate or call for transport of the patient within 10 minute time limit <input type="checkbox"/> Failure to take or verbalize body substance isolation precautions <input type="checkbox"/> Failure to determine scene safety before approaching patient <input type="checkbox"/> Failure to voice and ultimately provide appropriate oxygen therapy <input type="checkbox"/> Failure to assess/provide adequate ventilation <input type="checkbox"/> Failure to find or appropriately manage problems associated with airway, breathing, hypoperfusion <input type="checkbox"/> Does Secondary assessment before assessing and treating threats to airway, breathing, and circulation <input type="checkbox"/> Failure to determine the primary problem/accurately do stroke screen and recognize stroke equivalents <input type="checkbox"/> Uses or orders a dangerous or inappropriate intervention <input type="checkbox"/> Exhibits unacceptable affect with patient or other personnel		

***Factually document below your rationale for checking any of the above critical criteria.***

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**Scoring:** All steps must be independently performed in correct sequence with appropriate timing and all starred (\*) items must be explained/ performed correctly in order for the person to demonstrate



competency. Any errors or omissions of these items will require additional practice and a repeat assessment of skill proficiency.

**Rating: (Select 1)**

- ☐ **Proficient:** The paramedic can sequence, perform and complete the performance standards independently, with expertise and to high quality without critical error, assistance or instruction.
- ☐ **Competent:** Satisfactory performance without critical error; minimal coaching needed.
- ☐ **Practice evolving/not yet competent:** Did not perform in correct sequence, timing, and/or without prompts, reliance on procedure manual, and/or critical error; recommend additional practice

CJM 6/19

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Preceptor (PRINT NAME – signature)

Stroke mimics	
Etiology	History and Exam Findings
Psychogenic	Lack of objective CN findings, neuro findings in nonvascular distribution, inconsistent exam
Seizures	Hx of seizures, witnessed seizure activity, postictal period; post-seizure w/ persistent neuro signs (Todd's paralysis) (Tonic clonic seizures can occur simultaneous with hemorrhagic stroke)
Hypoglycemia	Hx DM, low serum glucose, ↓ LOC
Infection	Bell's palsy: Complete hemiparesis of face; can't wrinkle forehead on affected side; TB, fungal, herpes simplex encephalitis, meningitis
Complicated 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 mass	Tumors (primary and secondary); epidural/subdural hematomas: Gradual progression, seizure at onset of S&S
Drug toxicity	Med Hx includes Lithium, phenytoin, carbamazepine

