

Northwest Community EMSS NCH Paramedic Program **Stroke**

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National EMS Education standard:

Anatomy, physiology, epidemiology, pathophysiology, psychosocial impact, presentations, prognosis, and management of (complex depth, comprehensive breadth) stroke/intracranial hemorrhage/transient ischemic attack.

Assigned readings:

This handout; NW EMSS SOPs (pp. 35-36); Procedure Manual: Neuro Assessment Stroke

Goal: Strengthen participants' ability to assess and recognize strokes and provide appropriate patient care and disposition based on evidence-based stroke management guidelines.

OBJECTIVES:

Upon completion of the assigned readings, class and study questions, each participant will do the following with at least an 80% degree of accuracy and no critical errors:

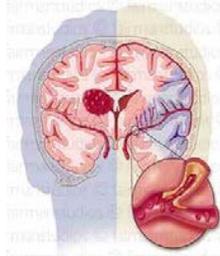
1. Define stroke and cite the incidence and epidemiology of stroke.
2. Differentiate the two main etiologies of stroke into ischemic and hemorrhagic.
3. Compare and contrast the types of ischemic stroke.
4. Explain the impact of modifiable and non-modifiable risk factors for stroke.
5. Sequence the impact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

NWC EMSS CE and NCH Paramedic Program

STROKE – IT'S NO ACCIDENT

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/15.pdf#023](https://www.cdc.gov/nchs/data/hus/15.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 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.



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 that impair their recovery (NINDS, 2016).

Stroke**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.

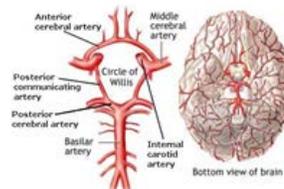
The **common carotid** arteries have two divisions.

- **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**.

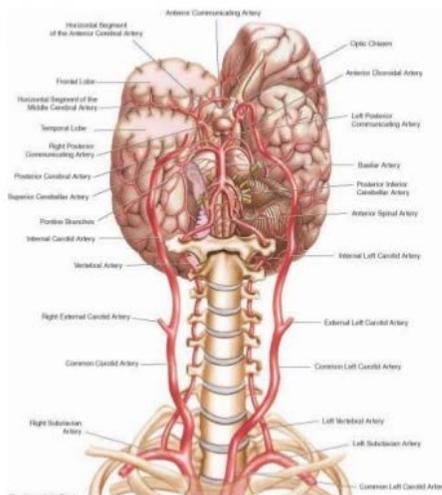
The **Vertebrobasilar arteries** supply the posterior 2/5th of the cerebrum, part of the cerebellum, and the brain stem. Occlusion here can cause severe vertigo, nausea, vomiting, dysphagia, diplopia, visual field loss; gaze palsies, decreased pain and temperature sensation, same side (ipsilateral) cerebellar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

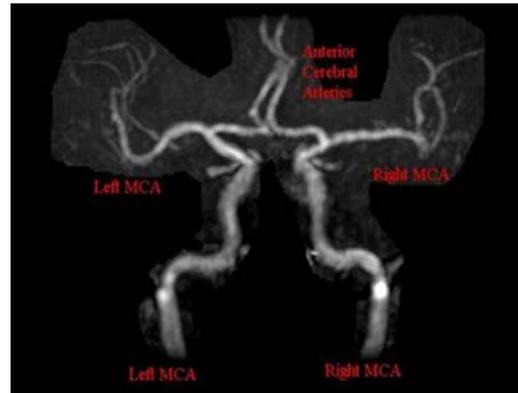
- Anterior communicating artery
- Middle cerebral artery
- Internal carotid artery
- Post. communicating artery
- Posterior cerebral artery



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)

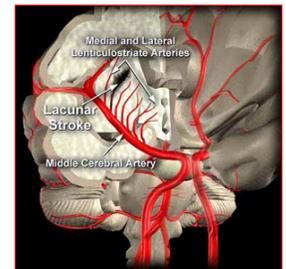
Largest branch of the internal carotid and supplies a portion of the frontal lobe, lateral surface of the temporal and parietal lobes (including the primary motor and sensory areas of the face, throat, hand and arm), and in the dominant hemisphere, the areas for speech. **The MCA is the one most often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



Stroke**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.

Gender difference in the prevalence of HTN

Younger people: HTN more common in men than women. With advancing age, more women than men have HTN. This gender-age difference probably has an impact on the incidence and prevalence of stroke.

Researchers recommend patient-specific HTN treatment, considering care goals, age, menopause, APOE ε4 genotype, metabolic traits, insulin resistance, systemic inflammation, and comorbidities to protect vascular health, and therefore brain health.

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, angiotensin receptor blockers (ARBs), calcium channel blockers, diuretics, and vasodilators (See HF page of SOPs for lists of meds).

- **Heart Disease:** After HTN, the 2nd 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)
- Atheromatous lesions of the ascending **aorta**; 30-70% stenosis of the internal carotid artery, or a clotting abnormality that promotes thrombosis. These patients may benefit from reduction of risk factors.

Heart muscle malformations

Patent foramen ovale (PFO) is a passage or a hole (sometimes 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 increases 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 5th and 6th 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.

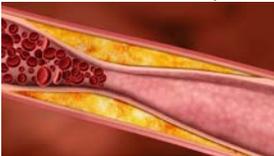
Stroke

- **High blood cholesterol/lipids:** High cholesterol levels contribute to stroke risk. Cholesterol, a waxy substance produced by the liver, 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.

Cholesterol is a lipid (fat-soluble rather than water-soluble). Other lipids include fatty acids, glycerides, alcohol, waxes, steroids, and fat-soluble vitamins A, D, and E. Lipids and water, like oil and water, do not mix. Blood is a water-based liquid, therefore cholesterol does not mix with blood. In order to travel through the blood without clumping together, cholesterol needs to be covered by a layer of protein. The cholesterol and protein together are called a **lipoprotein**.

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 O₂ from hemoglobin, interfering with O₂ 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.



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.

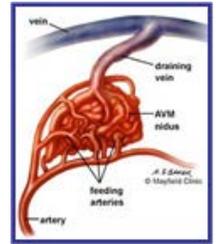


Marijuana smoking: Decreases BP and may interact with other risk factors, such as HTN and cigarette smoking, to cause rapidly fluctuating BP levels, damaging blood vessels.

- **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. It's possible that increased familial risk for stroke is due to environmental factors, such as a common sedentary lifestyle or poor eating habits, rather than heredity.



- **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)



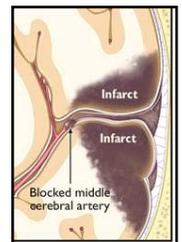
Previous stroke or transient ischemic attack (TIA) also raises the risk of (recurrent) stroke.

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 O₂ (perfusion) or when they are damaged by sudden bleeding into or around them. Unchecked, ischemia ultimately leads to **infarction** (death of brain cells).

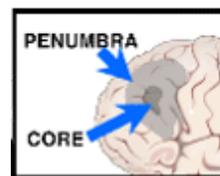


When blood flow is interrupted, some brain cells die quickly (within minutes), while others remain at risk for death. These damaged cells make up the **ischemic penumbra** and can linger in a compromised state for several hours. Collateral circulation to the penumbra occurs but is hard to predict by 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.

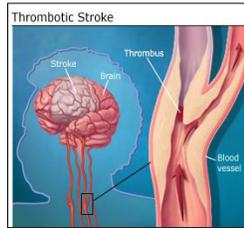
AHA reports that for 30-40% of ischemic strokes, no cause is found (*Circulation*, 2013). This type is called a **cryptogenic** stroke.



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.

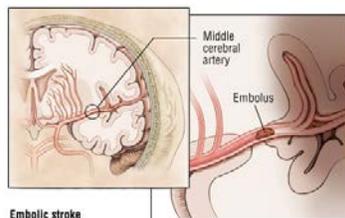
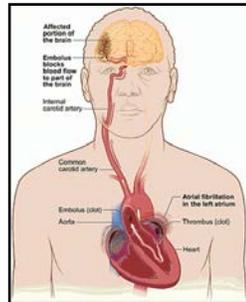
Both large and small vessel occlusions cause reduced tissue perfusion leading to neurotransmitter failure, anaerobic glycolysis, cerebral anoxia, and cerebral edema.

**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.

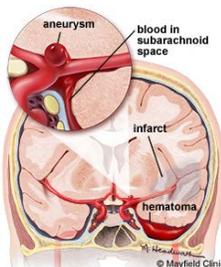
Onset is abrupt. S&S depend on the size and location of the hemorrhage and worsen as more brain tissue is affected.

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).

The most common and potentially treatable cause of secondary neurological injury in this population is **delayed ischemic deficit (DID)**. As the name implies, this phenomenon is fundamentally a reduction of cerebral blood flow (CBF) and O₂ delivery below critical ischemic thresholds, occurring days after the onset of hemorrhage. Three inter-related physiological processes appear to be involved in the reduced O₂ delivery: severe narrowing of intracranial arteries (arterial vasospasm), intravascular volume depletion and a loss of normal autoregulatory function in the distal circulation. DID occurs in up to 40% of patients surviving SAH. One third of these patients will die from this phenomenon and another third will be left with permanent and severe disability (Bradely, 2015).

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

S&S can range from a mild headache with or without meningeal irritation to severe headache (worse headache of their life present in ~80% of pts) and nonfocal findings with or without a dilated pupil. Worsening S&S include mild alteration in neuro exam, including AMS, obviously depressed level of consciousness or focal deficit and finally, a patient that is either posturing or comatose.

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.



ICH most commonly occurs in the cerebral lobes, periventricular white matter or basal ganglia (40-60%), thalamus (20%), the pons, and the cerebellum.

Acute neurologic deterioration results from early hematoma growth, peri-hematoma injury, and obstructive hydrocephalus. Delayed deterioration is usually due to edema and toxic effects of blood on the brain tissue.

Stroke



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 ≥ 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 ~ 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

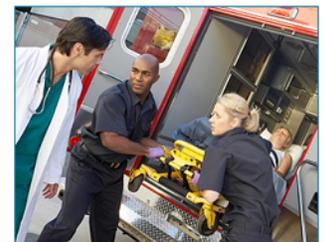
Until the 1990s, care of patients experiencing stroke was largely supportive, focusing on treatment of respiratory and cardiovascular complications while the effects of the stroke became complete. No specific therapy was available to alter the course and extent of the evolving physical losses. Thus, 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.

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

- 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.

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.

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.

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

EMS 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:

Stroke

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

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

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 \leq 10 minutes).

Time barriers at the hospital:

- Time to treatment area
- Time to physician assessment
- Time to CT (goal \leq 25 min.)
- Time for CT interpretation (goal \leq 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.

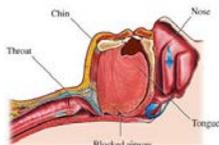
Disposition: Outcomes are best when patients are admitted directly to and receive care in a dedicated stroke unit, where recovery and rehab can begin immediately. Hospitals without full stroke care capabilities should have official transfer agreements with hospitals offering a full complement of stroke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

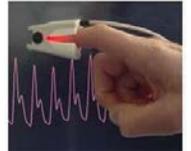
See Stroke SOP: IMC special considerations p.35

○ **Support ABCs as needed**

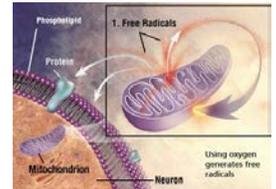
- Ideally, avoid sedating these patients
- Airway obstruction is a major problem, especially if pt loses consciousness. Paralysis of the muscles of the throat, tongue, or mouth can lead to partial/complete upper airway obstruction. Saliva may pool in throat and be aspirated.
- Aspiration is a serious 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 O₂ sats can be maintained at 94%, BLS may be sufficient.
- Assess **ventilatory status (ETCO₂)/ gas exchange (SpO₂)** at least q. 15 min. Give O₂ if SpO₂ < 94% or O₂ 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 O₂/BVM prn.

The AHA does not recommend use of O₂ in a patient with suspected stroke who has adequate oxygenation and SpO₂ readings (94%) to help prevent reperfusion release of free oxygen radicals and vasoconstriction.

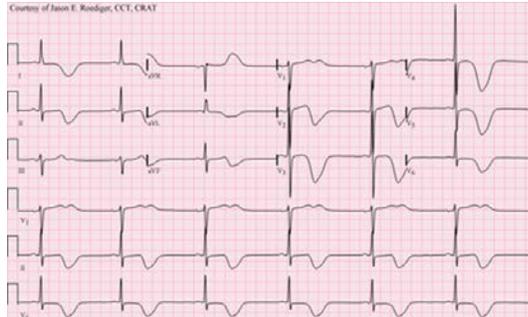
- **Seizure/vomiting precautions;** suction only as needed. Use gentle technique, as trauma to tissues may later lead to significant bleeding if patient is treated with fibrinolytics. Use caution to avoid vagal stimulation during suctioning, as increases in ICP may worsen the effects of stroke.
- **Positioning:** Can affect O₂ 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.

Stroke

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 enroute

- **Disability/mental status** exam: See p.12
If AMS: Consider possible causes of AMS using the AEIOU-TIPs mnemonic in the SOP p. 26.
- **If generalized tonic/clonic seizure activity:** Observe and record seizure activity per SOP p. 37
MIDAZOLAM 2 mg increments **IVP/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 ≥ 90 (MAP ≥ 65) unless contraindicated
If hypovolemic, elderly, debilitated, chronic dx (HF/COPD); and/or on opiates or CNS depressants: ↓ total dose to 0.1 mg/kg.

Observe/record the following **if seizure activity present:**

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

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



Hyperglycemia may mimic stroke symptoms, especially in the elderly

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 SpO₂ monitor for rate alone). Assess for carotid **bruits** if skilled in that procedure. HTN and bradycardia signal ↑ ICP; reassess frequently!



Accurate BP readings are essential. Take once manually in each arm before hooking up the automated cuff. BP may be elevated due to underlying HTN, a stress reaction to the stroke, increased ICP, or a physiological response to decreased brain perfusion. BPs often return to normal without antihypertensive treatment. Lowering the BP can be harmful, as it can decrease the cerebral perfusion pressure (CPP) and worsen the stroke.

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 O₂ 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.

Stroke

History (SAMPLE)

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?

S: S&S/Chief complaints (OPQRST) - Ask about:

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

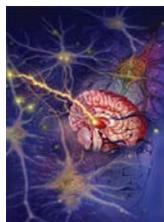
Vertigo: Sense of room spinning around the patient that persists at rest; may be accompanied by nystagmus. Differentiate from light-headedness. Common symptom in posterior strokes as well as a number of nonvascular diseases therefore at least one other S&S of stroke should also be present.

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

Sudden **confusion**, trouble understanding

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
- 3.5 - 6 hours
- > 6 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⁺⁺ Channel Blockers; Other anti-hypertensives; none

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

Anticoagulants/antiplatelet drugs

<p>“Anticoagulants” warfarin / Coumadin apixaban / Eliquis argatroban dabigatran / Pradaxa desirudin / Iprivask edoxaban / Savaysa enoxaparin / Lovenox fondaparinux / Arixtra *full dose LMW heparin Lepirudin / Refludan rivaroxaban / Xarelto</p>	<p>Antiplatelet agents NOT considered an “anticoagulant” for stroke SOP</p> <p>Aspirin clopidogrel / Plavix ASA/dipyridamole / Aggrenox prasugel / Effient ticagrelor / Brilinta ticlopidine (Ticlid)</p>
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*Low molecular weight heparin

- dalteparin Fragmin
- enoxaparin Lovenox
- tinzaparin Innohep

Diabetic drugs; Insulin; Oral agents; Other subcutaneous/injectable agents; None

Antidepressants; cocaine and other vasoconstrictors, e.g. amphetamines: PCP (Phencyclidine AKA angel dust, ozone, wack, rocket fuel) – increases BP; oral contraceptives; hormone replacement therapy (HRT)

Stroke

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: Advanced imaging techniques identify small ischemic brain lesions in up to 20% of pts whose symptoms subsided and who were diagnosed w/ TIA (based on cessation or short duration of stroke symptoms within 24 hours). Up to 15% of strokes are preceded by a TIA that is ignored by over half of pts whose symptoms resolve spontaneously. Prevalence increases with age. Urgent follow-up is recommended for TIA pts to begin anticoagulation and management of risk factors.
Following TIA:
 - 12% experience stroke within next 30 days
 - 3-17% have stroke within 90 days
 - 25% die within a year
- 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).

Mental status overview

- 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?

Assess and score GCS.

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

GCS ≤ 8 + new onset S&S of stroke is a candidate for a Comprehensive stroke center if transport time ≤ 30 min.

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 Brocca'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).



Stroke

Listen for difficulty pronouncing the following:

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

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.

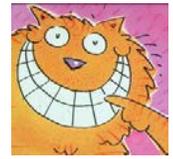
Characteristics showing statistically significant differences between patients with and without speech disturbance and patients were age, blood pressure, history of HTN, arrhythmia and DM, habit of tobacco and alcohol, and paresis.

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

- **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

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

Smile/facial asymmetry (CN VII):

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 7th nerve damage results in total ipsilateral hemi-facial paralysis and they cannot wrinkle their forehead on one side.



Bell's Palsy



Stroke

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

Assess for vision disturbances

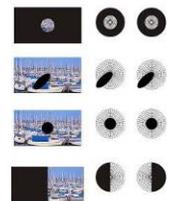
- **(CN II) Monocular blindness:** Sudden, painless vision deficit in one eye due to a central retinal artery occlusion (CRAO) may involve loss of all or part of the vision. Described as a curtain dropping, fog, grayout or blackout of vision. The involved eye is on the same side as the diseased/obstructed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



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

- Lose ability to see in one or more **fields of vision.** With one hemisphere disease, neither eye sees the contra-lateral environment (*hemianopsia*).



Stroke

- The involved visual field loss is opposite to the side of the diseased artery.

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

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



MOTOR assessment overview

- **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 balance, coordination (ataxia)

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.

Stroke

- **Point location.** Alternately touch patient on face, arms, and legs with the dull and sharp sides of a partially opened paperclip (or end of 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; 3.5 - 6. >6 hours)
- Co-morbid factors (need Comprehensive SC)
 - GCS 8 or less?
 - Sudden, severe headache?
 - Anticoagulant* use within 48 hrs?
 - PMH Intracranial hemorrhage/ aneurysm
- Proximity to nearest stroke center (30 min or less)

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



Stroke

- Shock and CNS hypoperfusion
- Cardiac abnormalities (dysrhythmia, AML, 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
- **All elements leading to hospital selection** (see above p. 15 and SOP)
- **EMS interventions**
- **Patient responses**
- **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

Tissue Plasminogen Activator (tPA)

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

Tissue plasminogen activator (tPA).

Goal of therapy for ischemic stroke: safely maximize functional recovery to pre-stroke baseline by rapidly reperfusing ischemic penumbra. IV-tPA remains the mainstay of acute stroke treatment for patients presenting within 4.5 hours of symptom onset.



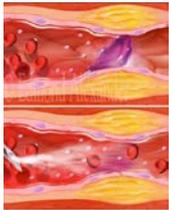
Studies continue to support that time from onset to treatment with tPA is directly related to outcomes in ischemic stroke. The sooner tPA is given, the greater the benefit (Stroke, 2013). Beyond the approved and recommended time windows for tPA use, risk of complications rises.

Stroke

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

Endovascular therapies include intra-arterial tPA and mechanical clot extraction. Profoundly positive results from five endovascular trials published in 2015 mark an extremely encouraging year in the history of stroke care.

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



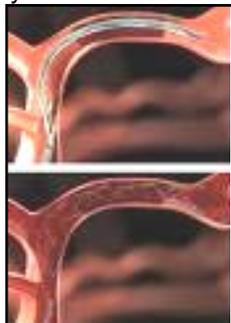
Intra-arterial tPA delivers fibrinolytic medication directly to the thrombus. It can be used for qualified patients up to 6 hours from onset of symptoms (Class I). As is the case with IV tPA, the best clinical outcomes with intra-arterial therapy are likely to be in those patients who receive treatment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

symptom onset as is possible. This therapy can only be done in specialty centers by specially trained interventionalists.

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

The 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
- 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 between the two groups.



Distinctive signs and symptoms of stroke

Weakness or paralysis of the face, arms, or legs, especially on one side of the body; asymmetric smile or mouth drawn to one side; loss of facial expression; possibly a drooping eyelid; one eyelid does not close as tightly as the other

Inability to speak or understand speech (expressive or receptive aphasia) or distorted speech (dysarthria)

Sensory or autonomic symptoms affecting half of the body, such as numbness or abnormal sweat patterns

Visual loss in one half (or one quarter) of the visual field, or dimming or loss of vision (especially in only one eye); constricted pupils; pupils unequal in size or reactivity; double vision

Severe headache with no apparent cause, often accompanied by a stiff neck

Dizziness, confusion, unsteadiness, or falls with no apparent cause; seizures

Drowsiness or decreased consciousness

Other symptoms: nausea or vomiting, respiratory distress, unusually flushed or pale skin, loss of bowel and/or bladder control

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

GCS	Level of Dysfunction	Level of Consciousness	Motor Response	Pupils	Resp	Pulse/ BP
15-8	cortex and cerebrum	Normal to drowsiness, stupor, light coma	Localizes pain withdraws	Normal sluggish & midsize	Normal Cheyne-Stokes	Stable
7-5	upper brainstem	deepening coma	Abnormal flexion or extension	unreactive & enlarging	Cheyne-Stokes Central Neurog. Hyp.	Becoming Unstable
4-3	lower brainstem	profound coma	Abnormal extension; flaccid	fixed & dilated	Failing	Unstable failing

Table from: Shea, J. *Altered States*, Unpublished lecture outline.

Features of clinical situations mimicking Stroke (*Stroke*, 2013)

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
Hypoglycemia	Hx DM, low serum glucose, ↓ LOC
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 tumor	Gradual progression of symptoms, other primary malignancy, seizure at onset
Drug toxicity	Med hx includes Lithium, phenytoin, carbamazepine

Stroke

Prehospital MEND Exam Pocket Card (Side 1)

ASLS Advanced Stroke Life Support
Center for Research in Medical Education

MILLER SCHOOL OF MEDICINE UNIVERSITY OF MIAMI
WWW.ASLS.NET
WWW.CRME.MIAMI.EDU
(305) 243-6491

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

MENTAL STATUS

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

CRANIAL NERVES

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

LIMBS

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

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Left cerebral hemisphere L MCA involvement	Right cerebral hemisphere R MCA involvement
Aphasia: expressive/ receptive or mixed Eyes: deviate to left R visual field deficit R hemiparesis/hemiplegia R hemisensory loss	Neglect (L hemi-inattention) Eyes: deviate to R L visual field deficit L hemiparesis/hemiplegia L hemisensory loss

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

Brainstem or posterior stroke
<p>5 Ds: Dizziness, Diplopia, Dysarthria, Dysphagia (chewing & swallowing), Dystaxia (incoordination) Severe vertigo; nausea, vomiting; Visual field loss; gaze palsies Partial or complete loss of hearing May have difficulty breathing Hallmark: Crossed findings; same side (ipsilateral) ataxia Cranial nerve deficits (ipsilateral) Motor/sensory deficits (contralateral); decreased pain and temperature sensation; loss of 2 point discrimination Clinical findings: asymptomatic to comatose</p>

Alternate Prehospital Stroke Screens

The **MEND exam Prehospital** (© 2005, University of Miami) incorporates the Cincinnati Prehospital Stroke Scale (CPSS) and

selected components of the NIH Stroke Scale (NIHSS) used at the hospital.

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

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

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

Glasgow Coma Scoring	
Strengths	Limitations
<ul style="list-style-type: none"> ■ Simplicity ■ Provides a common language to report neurologic findings based on bedside observations ■ Ability to trend over time 	<ul style="list-style-type: none"> ■ Variation in inter-rater reliability; inconsistent use by EMS and hospital staff ■ If SBP < 80, can't assess GCS with respect to possible outcomes ■ Hypothermia; Hypoglycemia ■ Sedating or paralyzing drugs; Intubated patients ■ Hearing impairments; Language barrier ■ Children younger than 3 years ■ Alcohol/drug intoxication ■ Relationship to mortality differs considerably between blunt & penetrating head trauma

This assessment is not consistently, nor accurately performed by EMS (Bledsoe et al, 2015).

Figure 2. National Institutes of Health Stroke Scale

Category	Score	Time	Score
1a. Level of Consciousness (LOC) (Alert, drowsy, etc.)	0 = 1 = 2 = 3 =	Alert Drowsy Stuporous Coma	
1b. LOC Questions (Month, age)	0 = 1 = 2 =	Answers both correctly Answers one correctly Incorrect	
1c. LOC Commands (Open/close eyes, make fist & let go)	0 = 1 = 2 =	Obeys both correctly Obeys one correctly Incorrect	
2. Best Gaze (Eyes open - pt follows examiner's fingers or face)	0 = 1 = 2 =	Normal Partial gaze palsy Forced deviation	
3. Visual (Introduce visual stimulus/threat to pt's visual field quadrants. Cover 1 eye and hold up fingers in all 4 quadrants.)	0 = 1 = 2 = 3 =	No visual loss Partial hemianopsia Complete hemianopsia Bilateral hemianopsia	
4. Facial Palsy (Show teeth, raise eyebrows and squeeze eyes tightly shut.)	0 = 1 = 2 = 3 =	Normal Minor Partial Complete	
5a. Motor Arm - Left (Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue.)	0 = 1 = 2 = 3 = 4 = NT=	No drift Drift Can't resist gravity No effort against gravity No movement Amputation, joint fusion (Explain)	
5b. Motor Arm - Right (Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue.)	0 = 1 = 2 = 3 = 4 = NT=	No drift Drift Can't resist gravity No effort against gravity No movement Amputation, joint fusion (Explain)	
6a. Motor Leg - Left (Elevate extremity to 30 degrees and score drift/movement. Count to 5 out loud and use fingers for visual cue.)	0 = 1 = 2 = 3 = 4 = NT=	No drift Drift Can't resist gravity No effort against gravity No movement Amputation, joint fusion	
6b. Motor Leg - Right (Elevate extremity to 30 degrees and score drift/movement. Count to 5 out loud and use fingers for visual cue.)	0 = 1 = 2 = 3 = 4 = NT=	No drift Drift Can't resist gravity No effort against gravity No movement Amputation, joint fusion (Explain)	
7. Limb ataxia (Finger to nose, heel down shin)	0 = 1 = 2 =	Absent Present in one limb Present in two limbs	
8. Sensory (Pin prick to face, arms, trunk, and legs -compare sharpness side to side, or no feeling at all.)	0 = 1 = 2 =	Normal Partial loss Severe loss	
9. Best Language (Name items, describe picture, and read sentences. Don't forget glasses if they normally wear them.)	0 = 1 = 2 = 3 =	No aphasia Mild to moderate aphasia Severe aphasia Mute	
10. Dysarthria (Evaluate speech clarity by pt reading or repeating words on list.)	0 = 1 = 2 = NT	Normal articulation Mild to moderate dysarthria Near to unintelligible or worse Intubated or other physical barrier	
11. Extinction and Inattention (Use information from prior testing or double simultaneous stimuli testing to identify neglect. Face, arms, legs and visual fields.)	0 = 1 = 2 =	No neglect Partial neglect Complete neglect	
NT= Not Testable acceptable as noted above			
TOTAL SCORE:			

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- National Institutes of health (NIH): www.health.nih.gov
- The National Institute of Neurological Disorders and Stroke (NINDS): www.ninds.nih.gov/disorders/stroke/stroke.htm
- National Stroke Association (NSA): www.stroke.org
- Stroke education for EMS professionals: <http://www.strokeawareness.com/ems4stroke>
- <http://www.neurocriticalcare.org/science-research/subarachnoid-hemorrhage>



Stroke Risk Scorecard

Each box that applies to you equals 1 point. Total your score at the bottom of each column and compare with the stroke risk levels on the back.

RISK FACTOR	HIGH RISK	CAUTION	LOW RISK
Blood Pressure	■ >140/90 or unknown	■ 120-139/80-89	■ <120/80
Atrial Fibrillation	■ Irregular heartbeat	■ I don't know	■ Regular heartbeat
Smoking	■ Smoker	■ Trying to quit	■ Nonsmoker
Cholesterol	■ >240 or unknown	■ 200-239	■ <200
Diabetes	■ Yes	■ Borderline	■ No
Exercise	■ Couch potato	■ Some exercise	■ Regular exercise
Diet	■ Overweight	■ Slightly overweight	■ Healthy weight
Stroke in Family	■ Yes	■ Not sure	■ No
TOTAL SCORE	■ High Risk	■ Caution	■ Low Risk



Risk Scorecard Results

High Risk ≥3: Ask about stroke prevention right away.

Caution 4-6: A good start. Work on reducing risk.

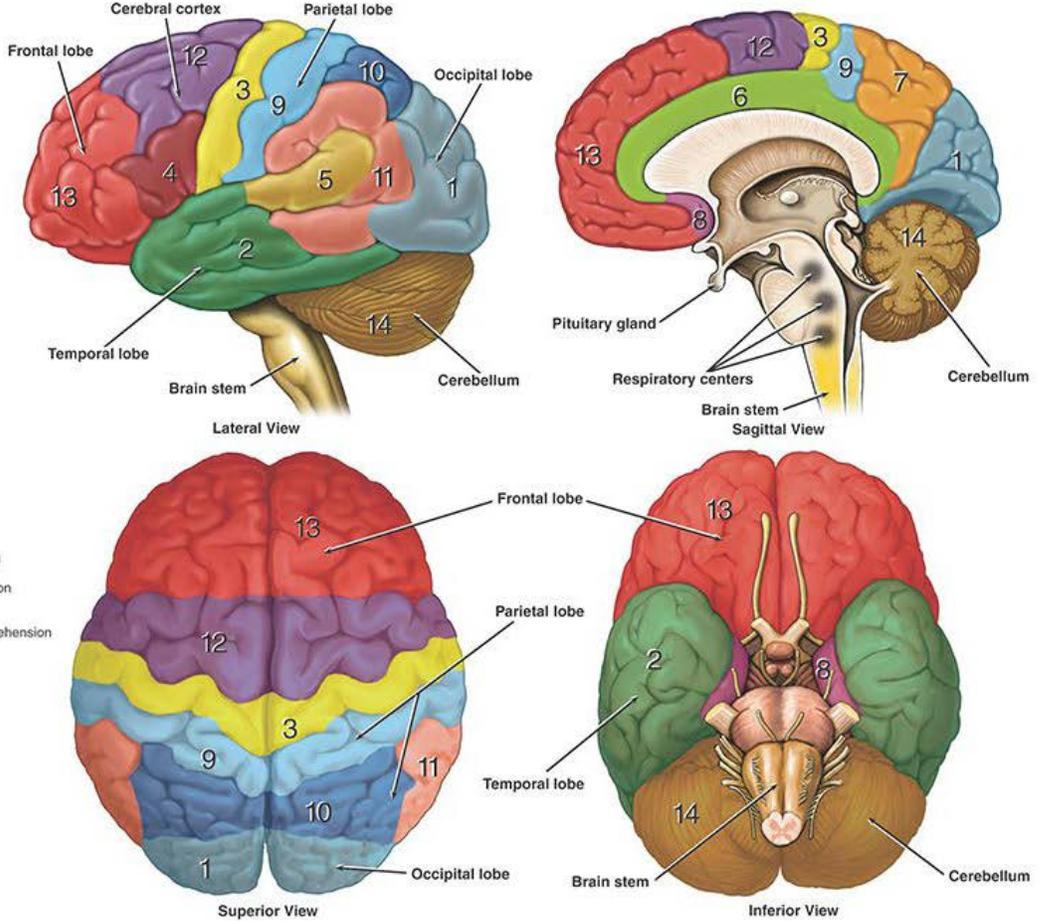
Low Risk 6-8: You're doing very well at controlling stroke risk!

Functional Areas of the Cerebral Cortex

- 1 **Visual Area:**
Sight
Image recognition
Image perception
- 2 **Association Area**
Short-term memory
Equilibrium
Emotion
- 3 **Motor Function Area**
Initiation of voluntary muscles
- 4 **Broca's Area**
Muscles of speech
- 5 **Auditory Area**
Hearing
- 6 **Emotional Area**
Pain
Hunger
"Fight or flight" response
- 7 **Sensory Association Area**
- 8 **Olfactory Area**
Smelling
- 9 **Sensory Area**
Sensation from muscles and skin
- 10 **Somatosensory Association Area**
Evaluation of weight, texture, temperature, etc. for object recognition
- 11 **Wernicke's Area**
Written and spoken language comprehension
- 12 **Motor Function Area**
Eye movement and orientation
- 13 **Higher Mental Functions**
Concentration
Planning
Judgment
Emotional expression
Creativity
Inhibition

Functional Areas of the Cerebellum

- 14 **Motor Functions**
Coordination of movement
Balance and equilibrium
Posture





EFFECTS OF STROKE

www.stroke.org/effects

CLINICAL TRIALS offer access to investigational treatments not yet available to the public.

To find stroke clinical trials currently enrolling, visit: www.stroke.org/clinicaltrials

STROKE IN THE UNITED STATES



Stroke is a leading cause of serious, long-term disability in the United States.

PHYSICAL EFFECTS

VISION
 Vision issues can lead to balance and posture problems, disorientation, trouble focusing or a reduced blink rate.

SLEEP
 Sleep-related breathing disorders, changing sleep patterns and insomnia can develop.

74% of stroke survivors face some form of **PAIN**.

80% of stroke survivors experience **HEMIPARESIS** or one-sided weakness.

70% of stroke survivors have increased **FATIGUE**.

40-60 PERCENT

of stroke survivors develop the lack of ability to control bladder/bowel movements



DYSPHAGIA
Paralysis of the throat muscles that can disrupt the swallowing process.



SPASTICITY
Muscles become tight and stiff, making movement difficult or uncontrollable.



FOOT DROP
Foot doesn't lift correctly when walking. Result of nerve damage.

COGNITIVE/EMOTIONAL EFFECTS

6.8 MILLION

adult stroke survivors in the U.S.

MEMORY PROBLEMS
Can lead to confusion, getting lost in familiar places and difficulty following instructions.

PSEUDOBULBAR AFFECT
Sudden and uncontrollable episodes of crying or laughing.

DEPRESSION
About 1/3 of stroke survivors experience post-stroke depression.

APHASIA
Impairs a person's ability to use and comprehend language.

QUALITY OF LIFE EFFECTS

80 PERCENT FINANCIAL
Percent of people affected by stroke who are worried about their financial future.

DRIVING
Many stroke survivors lose their ability to drive.

SAFETY
Injuries from falls are among the most common post-stroke complications.

RETURN TO WORK
About 1/2 of stroke survivors are able to return to work.

PEDIATRIC STROKE

Teenagers, children, newborns and unborn babies can have strokes too. Some effects of stroke more common in pediatric stroke survivors are:

- SEIZURES
- EPILEPSY
- CEREBRAL PALSY

Learn more at www.stroke.org/pedstroke

Save
Print
PDF
CAD
EKG
(0) Messages
Close

All Times
Timeline
Worksheets
Vitals
Monitor
Assessmen
Procs
IV/IO

Pertinent & PMH

Medical & Surgical History:

For NO PAST MEDICAL HISTORY Click the Circle on the Right

Other PMH:

PMH Obtained From (Mult):

Patient	Family	Repeat Patient Record
Health Care Personnel	Medical Records or Alert Card	Bystander/Other

Alcohol / Drug Use Indicators:

Patient Admits to Alcohol Use	Patient Admits to Drug Use	Smell of Alcohol on Breath
No Indicated drug/alcohol use		
Alcohol Container(s) at Scene	Drug Paraphernalia at Scene	Positive Level Quantified / Documented

Advance Directives:

Written DNR / POLST	None	Other Healthcare Advanced Directive Form
Not Applicable		
Verbal Order - DDM / Guardian	Living Will	Other

END OF PAGE

Save
Print
PDF
CAD
EKG
(0) Messages
Close

All Times

- Patient >
- Patient Address & Phone >
- Signs & Symptoms >**
- Medications >
- Allergies >
- Pertinent & PMH >**
- Trauma Information >
- ETCO2 Wave Form Shape >
- Stroke Information >
- Impression and Protocols >**
- Destination / OLMC Contact >**
- Transport Details >**
- Narrative >**
- All Delay Entries >**
- Was Patient Transferred? >
- Supplemental Questions >
- Signatures >**
- Refusal Considerations >
- Unreported Monitor Data Only >

Signs & Symptoms

Patient Complaints

+ Add

Arrived at Patient Time: 🕒

Date & Time of Symptom Onset: 🕒

Primary Symptom: ▼ ☰

Associated Symptoms: ☰ ☰

Chief Complaint Anatomic Location: ▼ ☰

Chief Complaint Organ System: ▼ ☰

Work-Related Illness/Injury:

END OF PAGE

- Timeline
- Worksheets
- Vitals
- Monitor
- Assessmen
- Procs
- IV/O
- Meds
- Airway Con
- All

Assessments

Power Tool

Date: Time: 🕒

+ Add New

Delete

Cancel

OK

Skin	Mental Status	Neurological	Head	Face	Eye
Neck	Shoulder	Chest/Lungs	Heart	Abdomen	Pelvis
Hip	Upper Leg	Knee	Lower Leg	Ankle	Foot
Upper Arm	Elbow	Forearm	Wrist	Hand	Back/Spine

Neurological ▼ Details

Normal Baseline for Patient		Not Done		
Normal Speech	Normal Gross Motor	Normal Cerebellar Function	Gait-Normal	Aphagia
Aphasia	Cerebellar Function-Abnormal	Decerebrate Posturing	Decorticate Posturing	Gait-Abnormal
Hemiplegia-Left	Hemiplegia-Right	Postictal	Stroke Symptoms Resolved in EMS Presence	Reported Stroke Symptoms Resolved Prior to
Seizures	Speech Slurring	Strength-Asymmetric	Strength-Symmetric	Tremors
Weakness-Facial Droop-Left	Weakness-Facial Droop-Right	Weakness-Left Sided	Weakness-Right Sided	

Assessments Power Tool

Date: 12/16/2016 Time: 11:38

+ Add New Delete Cancel OK

Skin	Mental Status	Neurological	Head	Face	Eye
Neck	Shoulder	Chest/Lungs	Heart	Abdomen	Pelvis
Hip	Upper Leg	Knee	Lower Leg	Ankle	Foot
Upper Arm	Elbow	Forearm	Wrist	Hand	Back/Spine

Mental Status Details

Normal Baseline for Patient Not Done

Oriented-Event Oriented-Person Oriented-Place Oriented-Time Combative

Confused Hallucinations Pharmacologically Sedated/Paralyzed Unresponsive

Vitals Power Tool

Crew Member:

OK Cancel Delete Repeat Last

Sitting Supine Semi-Fowlers Fowlers Trendelenburg Standing Shock More...

Level of Responsiveness (AVPU)

Alert Verbal Painful Unresponsive

GCS - Eye

Opens Eyes spontaneously Opens Eyes to verbal stimulation Opens Eyes to painful stimulation No eye opening when assessed

GCS - Verbal

Oriented (>2 Years); Smiles, oriented to sounds, follows Confused (>2 Years); Cries but is consolable, Inconsistently Inappropriate words (>2 Years); Inconsistently Incomprehensible sounds (>2 Years); Inconsolable, agitated No verbal/vocal response

GCS - Motor

Obeys commands (>2Years); Appropriate response Localizing pain Withdrawal from pain Flexion to pain Extension to pain No Motor Response

GCS Total

GCS - Qualifier

GCS accurate without influence Patient Chemically Sedated Patient Intubated Patient Chemically Paralyzed Eye Obstruction Prevents Eye Assessment

Blood Pressure Control

/ P

Mean Arterial Pressure


Crew Member

OK
Cancel
Delete
Repeat Last

Human Interp
Computer Interp
Transmission without Interpretation
Transmission with Remote Interpretation

Speech (Stroke)

Normal
Abnormal

Facial Droop (Stroke)

Normal
Abnormal
Left
Right

Arm Drift (Stroke)

Normal
Abnormal
Left Drifts Down
Left Falls Rapidly
Right Drifts Down
Right Falls Rapidly

Stroke Scale Type

Cincinnati
Miami Emergency Neurologic Deficit (MEND)
F.A.S.T. Exam
NIH
Other

! Stroke Scale Score

Normal
Abnormal
Non-Conclusive
⊖

Reperfusion Checklist

No Contraindications to Thrombolytic Use
Possible Contraindications to Thrombolytic Use
Definite Contraindications to Thrombolytic Use
⊖

Save
Print
PDF
CAD
EKG
Messages (0)
Close

All Times

- Patient
- Patient Address & Phone
- Signs & Symptoms
- Medications
- Allergies
- Pertinent & PMH
- Trauma Information
- ETCO2 Wave Form Shape
- Stroke Information**
- ! Impression and Protocols
- ! Destination / OLMC Contact
- ! Transport Details

Stroke Information

Arrived at Patient Time: ⊙

Time the Patient was Last Known Well: ⊙

IF UNABLE TO OBTAIN a TIME click the Circle on the Right

Callback Phone # for someone who has Knowledge about the Stroke Patient

+ Add

Callback Phone # ✕

Callback Phone # for someone who has Knowledge about the Stroke Patient:
8475555555

END OF PAGE

Timeline
Worksheets
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Save
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- ! All Delay Entries >
- Was Patient Transferred? >
- Supplemental Questions >
- ! Signatures >
- Refusal Considerations >

Destination / OLMC Contact

Hospital Pre-Arrival Alert Request

+ Add

What Type of Hospital Pre-Arrival Alert was Requested?:

Stroke
v
☰

Time the "Ambulance or EMS Unit" called for a Pre-arrival Alert:

🕒

Time your Unit Left Scene: 🕒

OLMC Contact Time: 🕒

OLMC Contacted: v ☰

OLMC Log # (if needed):

Destination State:

Destination County:

Destination ZIP Code:

Set From Postal Code

END OF PAGE

- Timeline
- Worksheets
- Vitals
- Monitor
- Assessmen
- Procs
- IV/IO
- Meds
- Airway Con
- All

Save
Print
PDF
CAD
EKG
Messages (0)
Close

All Times

Current Field (MultiSelect)

Reason for Choosing this Destination

Alpha Ascend
Alpha Descend

Select an Item

- Closest Facility
- Patient Choice
- Family Choice
- On-Line Medical Control
- Protocol
- Regional Specialty Center
- Diversion
- Insurance Status/Requirement

Destination / OLMC Contact

NOT TRANSPORTED can be chosen by the Clicking the Circle on the Right

Destination Name: v ☰ 🕒

Reason for Choosing this Destination: ☰ ☰

Type of Destination: v ☰

EMS Transport Method:

Ambulance

Helicopter

Ground - Bariatric

Ground - Wheelchair Van

Fixed Wing

Ground - Other

Hospital Pre-Arrival Alert Request

+ Add

Time your Unit Left Scene: 🕒

OLMC Contact Time: 🕒

- Timeline
- Worksheets
- Vitals
- Monitor
- Assessmen
- Procs
- IV/IO

Stroke

NWC EMSS STROKE ALERT / EXAM PREHOSPITAL CHECKLIST

Dates & Times

Date:	Dispatch time:	Pt contact time:	EMS Departure time:
-------	----------------	------------------	---------------------

Basic DATA

Pt. name		DOB		Gender	
Witness name	Call back number:				
Chief complaint(s)				Time last known well	Military clock: <input type="checkbox"/> < 3.5 hrs or > 6 hrs <input type="checkbox"/> 3.5 – 6 hrs
Glucose		BP	R	L	Pulse
					Resp

HISTORY Present Illness YES NO

Severe headache or seizure at onset?		
Head trauma at onset?		

EXAMINATION – NEW ONSET ✓ IF ABNORMAL

MENTAL STATUS	Level of consciousness: GCS 8 or less?		
	Speech ("You can't teach an old dog new tricks" (expressive/receptive aphasia)		
	Orientation: Answers to questions: Age, month		
	Responds appropriately to commands: open/close eyes		
CRANIAL NERVES	Vision: blurred, diplopia, loss of vision; loss of visual field; photophobia	R	L
	Horizontal gaze: gaze palsy or fixed deviation	R	L
	Facial droop; can't close eyelids tightly	R	L
	Hearing deficit	R	L
	Uvula / tongue deviates	R	L
LIMBS	Motor – Arm drift (close eyes and; hold out both arms for 10 sec)	R	L
	Motor – Leg drift (open eyes and lift each leg separately)	R	L
	Sensory – Face, arm, leg (close eyes – touch each side)	R	L
	Coordination – arm and leg (finger to nose, heel to shin); ataxia; vertigo	R	L
Other	Neck stiffness (cannot touch chin to chest; vomiting)		

PMH	<input type="checkbox"/> None	<input type="checkbox"/> A-Fib/Flutter	<input type="checkbox"/> AVM, tumor, aneurysm	<input type="checkbox"/> Bleeding disorders	<input type="checkbox"/> CAD/Prior MI/Heart/vascular dx
	<input type="checkbox"/> Carotid stenosis	<input type="checkbox"/> Pregnant (or up to 6 wks post- partum)	<input type="checkbox"/> HF	<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"/> Previous TIA:	<input type="checkbox"/> HRT	<input type="checkbox"/> HTN <input type="checkbox"/> Migraine
	<input type="checkbox"/> Obesity	<input type="checkbox"/> Previous stroke	<input type="checkbox"/> Renal failure	<input type="checkbox"/> Previous intracranial surgery/bleed	<input type="checkbox"/> Serious head trauma
	<input type="checkbox"/> *Prosthetic valve	<input type="checkbox"/> PVD		<input type="checkbox"/> Sleep apnea	<input type="checkbox"/> Smoker/tobacco use

MEDS	Anti-hypertensives:	<input type="checkbox"/> ACEI	<input type="checkbox"/> ARB	<input type="checkbox"/> Beta blocker	<input type="checkbox"/> Ca blocker	<input type="checkbox"/> Diuretic	<input type="checkbox"/> Other	
	Cholesterol reducers:	<input type="checkbox"/> Statin	<input type="checkbox"/> Niacin		<input type="checkbox"/> Fibrate	<input type="checkbox"/> Absorption Inhibitor		
	<input type="checkbox"/> Anticoagulant use in 48 hrs:	<input type="checkbox"/> warfarin/Coumadin	<input type="checkbox"/> dabigatran/Pradaxa	<input type="checkbox"/> desirudin/Privask	<input type="checkbox"/> apixaban/Eliquis	<input type="checkbox"/> edoxaban/Savaysa	<input type="checkbox"/> argatroban	<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	<input type="checkbox"/> prasugel/Effient		
	Platelet inhibitors:	<input type="checkbox"/> ASA	<input type="checkbox"/> clopidogrel/Plavix	<input type="checkbox"/> ticagrelor/Brilinta	<input type="checkbox"/> dipyridamole/Aggrenox	<input type="checkbox"/> ticlodipine/Ticlid		
	Diabetic drugs:	<input type="checkbox"/> Insulin	<input type="checkbox"/> Oral agents		<input type="checkbox"/> Other subcutaneous/injectable agents			
	<input type="checkbox"/> Antidepressants – list:							
	<input type="checkbox"/> Cocaine/other vasoconstrictors, e.g. amphetamines: PCP (Phencyclidine AKA angel dust, ozone, wack, rocket fuel)							
	<input type="checkbox"/> Oral contraceptives; hormone replacement therapy (HRT)							
	Others:							

Destination

Nearest hospital	Patient unstable
Nearest SC (Primary or Comprehensive)	<input type="checkbox"/> Onset <3.5 or > 6 hours with acute S&S of stroke; NO co-morbid factors <input type="checkbox"/> Criteria for Comprehensive, but > 30 minutes from closest CSC
Nearest Comprehensive SC	<input type="checkbox"/> Onset 3.5 - 6 hours AND/OR <input type="checkbox"/> Co-morbid factors: GCS ≤8; severe HA; anticoagulant w/in 48 hrs; PMH of ICH/AVM/aneurysm <input type="checkbox"/> Travel time ≤30 min scene to CSC

Stroke alert called to (OLMC hospital)	Time:
Receiving hospital	Time: