Stroke is a medical emergency involving the disruption of blood flow into or through the brain. It may result in significant motor (movement), sensory or cognitive (thought or perception) dysfunction, or even death. It is also commonly referred to as a "brain attack," since immediate recognition and management can reduce the amount of disability or death associated with stroke.

Stroke is the fifth-leading cause of death in the United States. This is down from the 3rd leading cause just a few short years ago but it remains a leading cause of permanent disability and is the leading preventable cause of long-term disability. It also results in billions of dollars in medical costs and lost productivity. According to the American Stroke Association, approximately 795,000 people in the United States suffer a stroke each year, which is approximately one stroke every 40 seconds. Every four minutes, a person will die from a stroke. African-Americans and Hispanic/Latinos have a higher risk of suffering a stroke. Women suffer about 40,000 more strokes per year than men.

While strokes are more often associated with adults and the elderly, children do have strokes as well. Symptoms are often comparable to those reported or seen in adult patients. NEVER forget to consider stroke as a differential diagnosis when caring for a pediatric patient with stroke-like symptoms.
Often, the signs and symptoms of stroke are subtle and go unrecognized for a period of time by the patient and family. Like a heart attack, immediate recognition of the stroke condition and initiation of treatment can reduce the amount of disability and death. This may mean the difference between a patient who suffers significant permanent disability and one who recovers completely or with only minor deficits. There is a narrow window of opportunity (3 – 4 ½ hours) in which fibrinolytic drugs (tPA) can be used to dissolve a clot and restore circulation to the brain tissue. Therapeutic interventions are constantly evolving and improving and invasive surgical procedures including thrombectomies are now considered an acceptable alternative when tPA is contraindicated or has been ineffective and the window of opportunity remains (6 - 8 hours after symptom onset.) It is imperative that EMS personnel be completely aware of and be able to recognize the signs and symptoms of stroke; gain information regarding the time of onset of the first signs and symptoms from family, relatives, friends or bystanders at the scene; initiate stroke treatment; begin rapid transport; and accurately report the stroke findings to the receiving medical facility. Knowing your local hospitals capabilities regarding care of the stroke patient is crucial to transporting your suspected stroke patient to an appropriate facility. Wisconsin EMS Services are required to have a policy on Transport Destination Determination. This policy requirement is intended to assist EMS Providers in determining most appropriate hospital destination for certain patients including acute stroke patients.

Types of Stroke

A stroke is defined as acute impairment of neurologic function that results from a rupture or obstruction of a blood vessel to a specific area in the brain. There are two broad categories of stroke: ischemic and hemorrhagic. Ischemic strokes result from the occlusion of a cerebral artery by a blockage or a clot. Hemorrhagic strokes occur from a cerebral vessel that ruptures, disrupts the blood flow and allows bleeding in and around the brain.

Ischemic strokes

Approximately 80%-85% of all strokes are ischemic. The primary etiology of these strokes is from blockage of a cerebral artery that obstructs blood flow to an area of the brain. The most common underlying cause of ischemic strokes is atherosclerosis. This is the process where fatty deposits collect and line the walls
of vessels. This fat will continue to build up inside the vessel wall and may eventually lead to a blockage at the site of the buildup, or a piece of the fatty plaque can break off and travel through the bloodstream and cause a blockage in a smaller vessel distal to the fatty buildup.
Ischemic strokes are further classified as thrombotic stroke, embolic stroke, transient ischemic attack (TIA), reversible ischemic neurologic deficit (RIND) and hypoperfusion stroke.

**Thrombotic stroke**

A thrombotic stroke results from an acute blockage of a cerebral artery at the site of the buildup of fatty deposits where the internal diameter (lumen) of the vessel is narrowed. This type of clot, a cerebral thrombosis, is often referred to as a "stationary clot," since the site of blockage is at the same site where the clot has formed. Approximately 60% of ischemic strokes are thrombotic.

The signs and symptoms of thrombotic stroke may be progressive. As clot formation progresses, blood flow is reduced to areas supplied by the affected cerebral artery, and ischemia to the brain cells worsens, producing signs and symptoms that may gradually develop and progress. During thrombus formation, as the artery narrows, the surrounding smaller cerebral arteries may begin to dilate in an attempt to deliver more blood to the brain tissue distal to the diseased artery. This collateral circulation, which is similar to that found in the coronary vessels in the heart, may reduce the extent of brain tissue ischemia and death following the stroke.

**Embolic strokes**

Embolic strokes, which account for approximately 40% of ischemic strokes, result from cerebral embolisms--clots or pieces of intravascular material that commonly form in a proximal artery or in the heart and travel through the cerebral circulation until they become lodged in a cerebral blood vessel. An embolism does not have to be a piece of fat or plaque; it can also be an air bubble, tumor or fat tissue. If a piece of material breaks off of a thrombus forming in a vessel and begins to travel downstream, it is referred to as a thromboemboli. This type of clot is often referred to as a "traveling clot," since it is not formed at the site of blockage.

Embolic strokes may present with more sudden onset of signs and symptoms since the blockage is a sudden event and there is no chance for the surrounding cerebral vessels to dilate and produce a collateral circulation effect.
The most common site for thromboemboli formation is in the carotid arteries and in the heart during periods of atrial fibrillation. Atrial fibrillation causes the atria to dilate and blood to stagnate, promoting clot formation.

**Transient Ischemic Attack (TIA)**

A transient ischemic attack (TIA) is a condition where the patient suffers a temporary interruption of blood flow to an area of the brain from either an embolism that arises from another proximal vessel and lodges in a cerebral artery or a disruption in a plaque in an area of atherosclerosis in the vessel. Since the interruption of blood flow is only temporary, there is no permanent neurologic dysfunction or damage associated with the blocked blood flow. The interrupted flow resolves itself after the clot is either dislodged or dissolves.

TIAs produce sudden onset of the same signs and symptoms of stroke; however, the signs and symptoms typically only last for a few minutes to usually no more than one hour. The signs and symptoms of TIA will definitely resolve within 24 hours after the onset. TIAs are often referred to by laypeople as "mini-strokes."

In addition to atherosclerosis and emboli, TIAs may also occur as a result of arterial dissection, inflammation of the arteries (arteritis) and sympathomimetic drugs, such as cocaine.

TIAs are very predictive of impending stroke in patients; thus, they are also referred to as "warning strokes." Approximately 30% of patients suffering TIAs will suffer a stroke in the future. Thus, it is imperative that EMS personnel be aggressive in assessing and managing the patient who has suffered a TIA. Since the signs and symptoms resolve very quickly, the patient may refuse emergency care and transport. Even though prehospital care for a TIA that has resolved is completely supportive, it is extremely important that you educate the patient about the high risk of suffering a true stroke in the near future that may result in permanent disability.

**Reversible ischemic neurologic deficit (RIND)**

Reversible ischemic neurologic deficit (RIND) is similar to a TIA in etiology. It produces the same signs and symptoms of stroke; however, they take longer to resolve—typically 24-72 hours after onset—compared to 24 hours for a TIA.
Likewise, RIND is also referred to as a "mini-stroke" or a minor ischemic stroke and is also a significant predictor of an impending stroke.

**Hypoperfusion stroke**

A hypoperfusion stroke is related to a low-perfusion state in which the entire brain is not receiving an adequate supply of blood through the cerebral arteries. The entire brain becomes ischemic and is subject to brain infarction (death). Unlike thrombotic and embolic stroke, the etiology of hypoperfusion stroke is not due to an isolated event of occlusion of a cerebral artery by a thrombus or embolism that produces focal ischemia and brain tissue necrosis. Instead, hypoperfusion strokes are associated with very poor cerebral blood flow conditions that arise from cardiac arrest, acute myocardial infarction with a decrease in cardiac output from pump dysfunction, and hemodynamically significant cardiac dysrhythmias that create poor perfusion states. Because hypoperfusion strokes affect the entire brain, the signs and symptoms are typically global in nature and don't result in focal neurologic deficits.

**Classification of ischemic stroke by supplying vessel**

Ischemic strokes can be further classified by the vessel and the area of the brain supplied by that respective blood vessel. The anterior portion of the brain blood supply originates from the carotid arteries. The anterior circulation is responsible for perfusing about four-fifths of the brain tissue. Occlusion of the carotid artery typically will disrupt blood flow to the cerebral hemispheres. The posterior area of the brain is supplied by the vertebrobasilar artery, which makes up the remaining one-fifth of brain perfusion. An occlusion to the vertebrobasilar artery or its branch will usually involve the brainstem, cerebellum (coordination) and/or vision. The presentation of the patient will vary based on which vessel was occluded and what area of the brain becomes ischemic and eventually infarcted.

**Pathophysiology of thrombus formation in ischemic stroke**

The concept of a ruptured plaque typical of the myocardial infarction patient leading to vessel occlusion is also true of the ischemic stroke; however, the occlusion is occurring in a cerebral vessel instead of a coronary vessel. Fatty deposits inside the cerebral vessel lead to fatty streaks. The fatty streaks promote the formation of an atheroma, which is the buildup of atherosclerotic plaque inside the vessel. The atheroma hardens and causes narrowing of the diseased
artery. The atheroma becomes inflamed, ulceration occurs and the plaque ruptures inside the vessel. The body views the internal rupture as an injury to the vessel and begins the cascade of physiologic events to clot the injured artery. This is actually a protective process that ends up occluding the cerebral artery and blocking the blood supply to the distal area of the brain, leading to ischemia and eventually infarction.

If the occlusion occurs at the site of the thrombus formation, it becomes a thrombotic stroke. This process explains the progressive nature of the signs and symptoms seen in thrombotic stroke. A piece of the atheroma can break off, travel distally in the cerebral artery or a branch until it becomes lodged and creates an embolic stroke.

**Hemorrhagic Strokes**

A hemorrhagic stroke is due to a rupture of a cerebral vessel, with bleeding occurring into brain tissue or areas surrounding the brain. Approximately 17% of all strokes are hemorrhagic in nature. When a vessel ruptures, the blood leaks from the vessel, accumulates in the brain and causes the brain tissue to become compressed. Brain damage from a ruptured vessel may result from direct trauma to the brain cells, the compression of the brain from increasing intracranial pressure, release of chemical mediators, spasm of local blood vessels, loss of blood flow distal to the ruptured cerebral vessel, and edema formation from the expanding blood and its compressive effects.

Two common causes of a ruptured vessel leading to hemorrhagic stroke are an aneurysm and arteriovenous malformations (AVM). An aneurysm is a weakened area in a blood vessel that balloons out. It may continue to weaken and eventually rupture and bleed into the brain or its surrounding tissue. An AVM is an abnormal formation of blood vessels that diverts blood away from the brain tissue and connects the arteries directly to the veins. The abnormal vessels of AVMs are weakened and dilate over a period of time. The vessels are prone to rupture from the high pressure contained within the arteries. AVMs are most often due to congenital defects and are not easily detected prior to rupture. AVMs may be found within brain tissue or within the subarachnoid space in the meningeal layers above the brain tissue.
CEREBRAL HEMORRHAGE

Cerebral artery

Intracerebral hematoma

Ruptured cerebral artery

Diagram showing cerebral hemorrhage with labels for cerebral artery, intracerebral hematoma, and ruptured cerebral artery.
Types of hemorrhagic stroke

There are two major types of hemorrhagic stroke: intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). An ICH is caused by a cerebral vessel that ruptures and bleeds directly into the brain tissue. The ruptured vessels are usually small arterioles that have been damaged over time, typically by chronic hypertension. In a SAH, the vessel ruptures into the subarachnoid space located above the actual brain tissue.

Aneurysms are more often the cause of SAH, whereas AVMs are less likely the etiology. When an aneurysm ruptures, it bleeds into the subarachnoid space at the systemic arterial pressure. This produces the sudden onset of severe and dramatic signs and symptoms. Intracerebral hemorrhage is more common than SAH. Both ICH and SAH carry a higher acute mortality rate than ischemic stroke.

Pathophysiology of Stroke

There are two critical elements that brain cells need for normal function: oxygen and glucose. Without these two elements, brain cells begin to malfunction and will eventually die. When an artery becomes occluded from thrombus formation, collateral circulation will assist with the maintenance of blood flow to the areas of the brain distal to the occluded artery. This may prevent a larger area of brain tissue death; however, the area surrounding the dead tissue will continue to receive some blood flow, but may become ischemic from a low blood flow state.
When cerebral blood flow to an area of brain tissue drops below its normal level, it may cause the cells to become "electrically silent." The brain cells are still intact and retain the ability to function; however, they cease transmission of electrical impulses. Thus, the cells are not dead but act as if they are and will not transmit electrical impulses. This causes the patient to present with neurologic deficits such as motor, sensory or cognitive dysfunction. If the blood flow is restored to these ischemic cells, they will become electrically active, continue to function and once again transmit electrical impulses. This may be evident in the patient who initially presents as a severe stroke with significant neurologic dysfunction, who then later regains function of many of the previously dysfunctional areas.

If the cerebral blood flow drops drastically, the brain cells begin to fail. Calcium levels within the cells and potassium levels outside of the cells increase. Due to a reduction of glucose delivery to the cells, the production of energy (ATP) is severely depleted. With the loss of ATP, the sodium/potassium pump fails and allows potassium to remain outside of the cell while sodium is no longer pumped out of the cell. Since sodium attracts water, the cells begin to swell and will eventually rupture and die. This process is known as cytotoxic edema.

The area of the brain surrounding the primary stroke site that continues to receive cerebral blood flow from collateral circulation is termed the ischemic penumbra or ischemic shadow. Since the tissue is receiving a lower cerebral blood flow than normal, again, the brain cells become electrically silent. Irreversible brain cell damage in this area has not yet occurred, and the function of these brain cells can be reversed. This is the area of brain that can possibly be salvaged and limit the extent of the brain injury. Neuroprotective agents are being researched that can protect the ischemic penumbra and preserve the brain cells.

Assessment of the Stroke Patient

The signs and symptoms of stroke may be subtle and unrecognized as significant by the patient, his relatives or bystanders. Simple numbness of the arm may be downplayed as insignificant by the patient for a long period of time, until the signs and symptoms progress to a more severe condition. The patient may then seek EMS; however, several hours may have passed and the chance for reversal of the stroke may be eliminated. It is imperative in your history taking that you attempt to precisely determine time of onset of the first sign or symptom of
stroke, no matter how subtle. This is vital information that must be reported to the receiving medical facility.

General signs and symptoms of stroke include the following:

- Facial droop
- Slurred speech
- Difficulty in speaking (dysphasia) or inability to speak (aphasia)
- Use of inappropriate words (expressive aphasia)
- Numbness to the face, arm or leg—especially on one side of the body
- Headache (may not be severe in ischemic stroke)
- Weakness (paresis) or paralysis (plegia) especially to one side of the body (hemiparesis and hemiplegia)
- Confusion, agitation or other severe altered mental status
- Gait disturbance noted by trouble walking
- Dizziness
- Loss of balance or coordination
- Loss of vision or disturbed vision in one or both eyes
- Inability to understand
- Incontinence

Patients who experience an intracerebral hemorrhage or subarachnoid hemorrhage may present with many of the aforementioned signs and symptoms. ICH and SAH patients typically complain of a sudden onset of the "worst headache" they have ever experienced with pain that may radiate to the face and neck. The headache may be accompanied by nausea, vomiting, intolerance to light and noise, and an altered mental status. These signs and symptoms, especially deterioration in the mental status, may continue to progress as bleeding continues within the brain. Patients with ICH and SAH will typically present with more severe depressed mental status and headache as compared to the ischemic stroke patient.

Two common stroke assessment scales with high predictive value used in the prehospital setting are the Cincinnati Prehospital Stroke Scale (CPSS) and the Los Angeles Prehospital Stroke Screen (LAPSS). Either of these scales can be included in your assessment of the stroke patient and reported to the medical facility. Wisconsin has incorporated the CPSS into the EMT curriculum. Our
protocol, M-6 Suspected CVA incorporates the Cincinnati Prehospital Stroke Scale. The Cincinnati Scale assesses for three motor function deficits, facial droop, arm drift and abnormal speech. Patients with 1 of these 3 findings as a new event have a 72% probability of an ischemic stroke. If all 3 findings are present the probability of an acute stroke is more than 85%. A Glasgow Coma Score should also be obtained on the patient. This information is imperative for EMS personnel to collect and report to ensure adequate and aggressive assessment and management of the stroke patient.

**Cincinnati Pre-hospital Stroke Scale**

1. **FACIAL DROOP**: Have patient show teeth or smile.
   - **Normal**: both sides of the face move equally
   - **Abnormal**: one side of face does not move as well as the other side

2. **ARM DRIFT**: Patient closes eyes & holds both arms out for 10 sec.
   - **Normal**: both arms move the same or both arms do not move at all
   - **Abnormal**: one arm does not move or drifts down compared to the other

3. **ABNORMAL SPEECH**: Have the patient say “you can’t teach an old dog new tricks.”
   - **Normal**: patient uses correct words with no slurring
   - **Abnormal**: patient slurs words, uses the wrong words, or is unable to speak

**INTERPRETATION**: If any 1 of these 3 signs is abnormal, the probability of a stroke is 72%.
Table 2. Los Angeles Prehospital Stroke Screen (LAPSS)

<table>
<thead>
<tr>
<th>Screening Criteria</th>
<th>Yes</th>
<th>No</th>
</tr>
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<tbody>
<tr>
<td>1. Age over 45 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. No prior history of seizure disorder</td>
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<td></td>
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<tr>
<td>3. New onset of neurologic symptoms in last 24 hours</td>
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<td></td>
</tr>
<tr>
<td>4. Patient was ambulatory at baseline (prior to event)</td>
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<td></td>
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<tr>
<td>5. Blood glucose between 60 and 400</td>
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</tbody>
</table>

Exam: Look for obvious

<table>
<thead>
<tr>
<th>Facial smile / grimace:</th>
<th>Normal</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Droop</td>
<td>Droop</td>
</tr>
<tr>
<td>Grip:</td>
<td></td>
<td>Week Grip</td>
<td>Week Grip</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No Grip</td>
<td>No Grip</td>
</tr>
<tr>
<td>Arm weakness:</td>
<td></td>
<td>Drifts Down</td>
<td>Drifts Down</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Falls Rapidly</td>
<td>Falls Rapidly</td>
</tr>
</tbody>
</table>

6. Based on exam, patient has only unilateral weakness: Y/N

*Yes (or unknown) to all items above, LAPSS screening criteria met.
If LAPSS criteria for stroke met, call receiving hospital with "code stroke." If not, then return to the appropriate treatment protocol. (Note: the patient may still be experiencing a stroke even if LAPSS criteria are not met.)*

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GLASGOW COMA SCORE

**Eye(s) Opening**

- Spontaneous: 4
- To speech: 3
- To pain: 2
- No response: 1

**Verbal Response**

- Oriented to time, place, person: 5
- Confused/disorientated: 4
- Inappropriate words: 3
- Incomprehensible sounds: 2
- No response: 1

**Best Motor Response**

- Obey's commands: 6
- Moves to localised pain: 5
- Flexion withdraws from pain: 4
- Abnormal flexion: 3
- Abnormal extension: 2
- No response: 1

**Best response**

- Comatose patient: 8 or less
- Totally unresponsive: 3
Stroke Mimics

There are other conditions that can mimic strokes and should be considered when caring for a patient with stroke signs and symptoms. Those mimics can include:

• Hypoglycemia
• Alcohol intoxication
• Drug Overdose
• Migraine
• Cerebral Infections
• Neuropathies (Bell’s Palsy)
• Seizure and Post-seizure
• Epidural hematomas
• Todd’s paralysis
• Metabolic disorders

Some of these can be treated by EMS (hypoglycemia, certain drug overdoses, and seizures). When in doubt, consider the probability of an acute stroke until proven otherwise.

Prehospital Care of the Stroke Patient

It is vital to ensure an adequate airway, adequate ventilation, adequate oxygenation and adequate circulation in the initial assessment. Stroke patients are at an increased risk of loss of airway control and aspiration. Ensure an adequate airway is established and maintained. Protect the patient from aspiration by simply placing him/her in a lateral recumbent position. If vomiting is severe and the airway is severely compromised, an advanced airway device may need to be employed to protect the patient from aspiration. Assess the tidal volume and rate of ventilation. If either the tidal volume or rate is inadequate, immediately begin ventilation at a rate of 12 breaths per minute. Provide supplemental oxygen to the patient with an adequate rate and tidal volume and
via the ventilation device if the patient is being ventilated. Apply a pulse oximeter to monitor oxygen saturation levels. Be sure to immediately respond to declines in oxygen saturation by reassessing the adequacy of ventilation, ventilating if necessary, or increasing the oxygen concentration by switching to a nonrebreather mask or increasing the liter flow. Titrate supplemental oxygen to the lowest level required to maintain oxygen saturation at 94% or greater. Apply the cardiac monitor. Report and document any atrial fibrillation that the patient may experience while in your care. Establish an intravenous line of normal saline. Obtain a blood glucose level. If the patient is confirmed hypoglycemic by the blood glucose reading, administer dextrose up to 25 grams titrated to resolve symptoms. Protect the patient and rapidly transport to the most appropriate medical facility for stroke management.

**Early ED Notification by EMS**

The receiving ED should be notified as soon as possible of the anticipated arrival of a suspected stroke patient. When providing a patient care report via radio/telephone to the ED, EMS should notify the ED of a “Neuro Alert” or “Stroke Alert.” Using plain language has been demonstrated to reduce misunderstandings. Early notification allows the ED the opportunity to assemble the appropriate health care team and to have the CT scanner available immediately upon arrival of the patient. Under most circumstances, the EMS crew and patient will be directed to the CT scanner with perhaps only a momentary stop in the ED. The following patients should be considered as a Neuro Alert/Stroke Alert.

- Suspected stroke/CVA with **new** onset of:
  - One sided weakness
  - Slurred/garble speech
  - Arm drift
  - Unsteady gait
  - Facial droop
- Atypical thought process
- Any of the listed signs and symptoms
  - Facial droop
  - Slurred speech
  - Difficulty in speaking (dysphasia) or inability to speak (aphasia)
  - Use of inappropriate words (expressive aphasia)
  - Numbness to the face, arm or leg—especially on one side of the body
  - Headache (may not be severe in ischemic stroke)
  - Weakness (paresis) or paralysis (plegia) especially to one side of the body (hemiparesis and hemiplegia)
  - Confusion, agitation or other severe altered mental status
  - Gait disturbance noted by trouble walking
  - Dizziness
  - Loss of balance or coordination
  - Loss of vision or disturbed vision in one or both eyes
  - Inability to understand
  - Incontinence

As a final reminder, **ALWAYS** try to establish “last known well time.” Last known well time is the time the patient was last known to be normal or without symptoms. Establishing a “last known well time” is considered by some to be the single most important piece of information that all caregivers should strive to identify. This time is critical for therapy considerations. It is used to determine if a patient is a candidate for certain stroke treatments.

In the short time (perhaps 1 hour) it has taken you to read this, 91 people will have suffered a stroke and 15 people will have died from a stroke. For further review, please see attached protocol and EMS Neurologic Checklist.
## Suspected Stroke/CVA

### Initial Medical Care – Special Considerations:
- Titrate Oxygen to bring oxygen saturations to 94% or greater.
- Assist with BVM if patient is not breathing adequately.
- Protect airway, suction as needed.
- If BP is greater than 90 mmHg: elevate head of bed 15-30 degrees.
- Protect paralyzed limbs from injury.
- Keep head neck and spine in neutral alignment. Do not flex neck.
- Complete Neuro/Stroke Alert checklist enroute to hospital.
- IV access
- Obtain and record blood glucose levels. If less than 60 treat per appropriate protocol
  - Adult Diabetic/Glucose Emergencies
  - Pediatric Diabetic/Glucose Emergencies
- History: Length of time of symptoms and LAST KNOWN WELL TIME, (less than 4.5 hours, patient is candidate for intervention).
- Obtain Glasgow Coma Scale and Cincinnati Prehospital Stroke Scale. Note any changes from known baseline.
- If Seizures occur, treat per Seizure protocol.

Note: Bradycardia may be present in those patients due to increased intracranial pressure. Atropine IS NOT to be given if the BP is elevated.

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
<th>Cincinnati Prehospital Stroke Scale</th>
</tr>
</thead>
</table>
| **Eye Opening:**   | **Facial Droop:** have patient show teeth or smile:  
| Spontaneous         | Normal – both sides of face move equally well.  
| In response to speech | Abnormal – one side of face does not move as well as the other side.  
| In response to pain |  
| None                |  
| **Best Verbal Response:** | **Arm Drift:** have patient close eyes and hold both arms out:  
| Oriented conversation | Normal – both arms move the same or both arms do not move at all (other findings, such as pronator grip, may be helpful).  
| Confused conversation | Abnormal – one arm does not move or one arm drifts down compared with the other.  
| Inappropriate words |  
| Incomprehensible sounds |  
| None                |  
| **Best Motor Response:** | **Speech:** have patient say “you can’t teach an old dog new tricks”:
| Obey                | Normal – patient uses correct words with no slurring.  
| Localizes           | Abnormal – patient uses inappropriate words or is unable to speak.  
| Withdraws           |  
| Abnormal flexion    |  
| Abnormal extension  |  
| None                |  

AHC-SM EMS  Approved 7/01/08  Revised 08/01/2012  6/20/2014
Aurora EMS
STROKE/NEURO ALERT CHECKLIST

PATIENT NAME: ________________________ RUN #: ___________ DOB: __________

BASELINE VITALS: B/P: _______  P: _______  R: _______  Oxygen Sat: _______

911 CALL TIME: _______  DATE OF ONSET: _______  LAST KNOWN WELL TIME: _______

TIME AT PT: _______  LEAVE SCENE TIME: _______  ER ARRIVAL: _______

Completed

1. PATIENT WITH SUSPECTED STROKE SYMPTOMS: ____________________________
   WITHIN 4.5 HOURS OF LAST KNOWN WELL TIME!!

   OR  (OBTAIN WITNESS NAME & PHONE NUMBER)

   (DETERMINE IF PT HAS CONTRAINDICATIONS FOR THROMBOLYICS: HEAD
   TRAUMA, SEIZURE AT ONSET, TAKING ANTICOAGULATION, HX OF BLEEDING
   PROBLEMS, POSSIBLE BRAIN HEMORRHAGE)

2. PATIENT WITH SUSPECTED STROKE SYMPTOMS: ____________________________
   GREATER THAN 4.5 HOURS LAST KNOWN WELL TIME!!

   ![ ]

3. A CINCINNATI STROKE SCALE AND GCS HAS BEEN DONE!! __________________________

   ![ ]

4. A BLOOD SUGAR IS DONE AND GREATER THAN 60!! __________________________

   ![ ]

5. INITIATED SUSPECTED CVA PROTOCOL AND MEDS __________________________

   ![ ]

   □ IV  □ Oxygen  □ MONITOR  □ EKG  □ BLOOD SUGAR  □ OTHER: __________________________

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ALL OF THE ABOVE CRITERIA MUST BE CHECKED IN ORDER TO ACTIVATE A "STROKE ALERT" FROM THE FIELD. IF ANY OF THE ABOVE CRITERIA CANNOT BE CHECKED OFF, THEN A "STROKE ALERT" CANNOT BE CALLED IN FROM THE FIELD!!