Stroke is the third leading cause of death and the leading cause of adult disability in the United States. Roughly one American suffers a stroke every minute, and one American dies of stroke every 3.5 minutes. In 1995, the National Institute of Neurological Disorders and Stroke study showed that intravenous administration of tissue plasminogen activator (tPA) had clinical benefit for a select group of patients with acute ischemic stroke. Most importantly, patients maximally benefit from intravenous administration of tPA if treatment is started as soon as possible within 3 hours of symptom onset.

The narrow therapeutic window of stroke therapy has important implications for emergency medical services. EMS professionals must be proficient in their ability to recognize, assess, manage, treat and transport stroke patients. Decisions made by EMS personnel can affect treatment and contribute to the immediate short-term and long-term outcomes of the patient.

Stroke is a general term that describes injury or death of brain tissue usually due to interruption of cerebral blood flow. The term “brain attack” is used because it compares the physiology of a stroke with that of a heart attack. In both cases, oxygen deprivation causes damage to the affected tissue.

**Classifications of Strokes**

The American Heart Association has defined two primary categories of stroke: ischemic stroke and hemorrhagic stroke.

*Ischemic Stroke*

Approximately 85% of strokes are the ischemic type. An ischemic (or occlusive) stroke occurs when a cerebral artery is blocked by a clot or other foreign matter. This results in ischemia and progresses to infarction, the death of tissues as a result of cessation of blood supply. These strokes are further classified as either thrombotic, embolic, or a result of systemic hypoperfusion.

Thrombotic stroke: A cerebral thrombus is a blood clot that gradually develops in and obstructs a cerebral artery. As a person ages, atheromatous plaque deposits can form on the inner walls of arteries. The buildup causes a narrowing of the arteries and reduces the amount of blood that can flow through them. This process is known as atherosclerosis. Once the arteries are narrowed, platelets adhere to the roughened surface and can create a blood clot that blocks the blood flow through the cerebral artery. This results in brain tissue death. The signs and symptoms of thrombotic stroke develop gradually. This type of stroke often occurs at night with the patient awakening with altered mental status and/or loss of speech, sensory or motor function.
Embolic stroke: A stroke caused by an embolus results when an intracranial vessel is blocked by a foreign substance. This can be a solid, liquid or gaseous material carried to a blood vessel from a remote site. Common sources of cerebral emboli include:

⇒ Atherosclerotic plaques originating from large vessels of the head, neck or heart
⇒ Thrombi that develop on the valves or in the chambers of the heart
⇒ Air embolism from chest injuries
⇒ Fat embolism from long bone injuries

Signs and symptoms of cerebral embolus are similar to those of thrombotic. However, embolic signs and symptoms develop more quickly. Also, they often are associated with an identifiable cause (e.g. atrial fibr).
Systemic hypoperfusion: Systemic hypoperfusion is a more general circulatory problem, manifesting itself in the brain and perhaps other organs. Reduced perfusion can be due to cardiac pump failure caused by cardiac arrest or arrhythmia, or to reduced cardiac output related to acute myocardial ischemia, pulmonary embolism, pericardial effusion, or bleeding. Hypoxia may further reduce the amount of oxygen carried to the brain. Symptoms of brain dysfunction typically are diffuse and non-focal in contrast to the other two categories of ischemic stroke. Most affected patients have other evidence of circulatory compromise and hypotension.

Hemorrhagic stroke

Hemorrhagic strokes account for approximately 15% of all strokes. They are usually classified as being intracerebral (within the brain) or subarachnoid (the space around the outer surface of the brain).

Intracerebral hemorrhage: Bleeding in intracerebral hemorrhage (ICH) is usually derived from arterioles or small arteries. The bleeding is directly into the brain, forming a localized hematoma which spreads along white matter pathways. Accumulation of blood occurs over minutes or hours; the hematoma gradually enlarges by adding blood at its periphery like a snowball rolling downhill. The most common causes are hypertension, trauma, vascular malformations and illicit drug use. Less frequent causes include bleeding into tumors, aneurismal rupture and vasculitis. An intracerebral hemorrhage begins abruptly. In about half of the people, it begins with a severe headache, often during activity. However, in older people, the headache may be mild or absent. Symptoms suggesting brain dysfunction develop and steadily worsen as the hemorrhage expands. Some symptoms, such as weakness, paralysis, loss of sensation, and numbness, often affect only one side of the body. People may be unable to speak or become confused. Vision may be impaired or lost. The eyes may point in different directions or become paralyzed. The pupils may become abnormally large or small. Nausea, vomiting, seizures, and loss of consciousness are common and may occur within seconds to minutes.
Subarachnoid hemorrhage: Rupture of arterial aneurysms is the major cause of subarachnoid hemorrhage (SAH). Aneurysm rupture releases blood directly into the cerebrospinal fluid (CSF) under arterial pressure. The blood spreads quickly within the CSF, rapidly increasing intracranial pressure. Death or deep coma ensues if the bleeding continues. The bleeding usually lasts only a few seconds but rebleeding is common. With causes of SAH other than aneurysm rupture, the bleeding is less abrupt and may continue over a long period of time. Symptoms of SAH begin abruptly, occurring at night in 30 percent of the cases. The primary symptom is sudden severe headache (97% of cases) classically described as the “worst headache of my life.” The headache is lateralized in 30% of patients, predominantly to the side of the aneurysm. The onset of the headache may or may not be associated with a brief loss of consciousness, seizure, nausea, vomiting, focal neurologic deficit, or stiff neck. There are usually not important focal neurologic signs at presentation unless bleeding occurs into the brain and CSF at the same time.

Assessment

Since neuro assessment was covered in the in-station CE during Jan and Feb, we will not be taking time to go over that element here. But do not forget to do your BEFAST assessment. B=Balance, E=Eye movement, F=Facial droop, A=Arm Drift, S=Speech and T=Time.
Management

Because the outcome of some stroke treatments is time dependent, it is important to rapidly identify and transport any suspected stroke patient. The hospital should be notified immediately of a suspected stroke patient and scene time should be kept at a minimum, ideally less than 10 minutes.

Transport position: The primary aim of acute stroke treatment is to restore blood flow to poorly oxygenated brain tissue. Positioning the head at zero degrees or supine, during assessment and transport may increase arterial blood flow through the effects of gravity. Traditional positioning for most acute neurological conditions has been with 30 degree of head elevation to decrease intracranial pressure. However, in ischemic stroke patients, ICP does not peak until 48 hours post-infarction, and increased blood flow may be more beneficial in the acute setting. However, proper assessment and management of airway, breathing and circulation are of primary importance in acute stroke. Stroke patients may need stabilization of airway and transportation with aspiration precautions. Therefore, they should be transported with the head of their bed elevated 10 - 15 degrees. To help prevent an increase in intracranial pressure head and neck should be kept in neutral alignment. Do not use pillows.

Oxygen: Decreases in oxygen saturation can lead to worsening of cerebral ischemia. Current recommendations in literature are to monitor oxygen saturation continuously with a pulse oximeter and treat hypoxia with supplemental oxygen. Because there is no conclusive evidence that supplemental oxygen for those that are not hypoxic causes harm, pre-hospital providers may consider the use of oxygen in stroke for patients who are not hypoxic. However, because the goal of treatment should be to maintain normoxia, supplemental oxygen for those who are not hypoxic should be given at low flow rates, titrating pulse ox to no higher than 99%.

IV Access: Since acute stroke should be treated as a time-dependent emergency, rapid transport to a definitive care facility is of utmost importance. Delays in the pre-hospital setting need to be avoided. One potential time delay relates to the establishment of IV access. A field line may be appropriate when acute resuscitation meds need to be give (e.g. DAI, seizures). Otherwise, transportation should be started immediately and IV access secured en route.

IV Fluids: Research has shown that there is no benefit from routine hemodilution. In patients who are hemodynamically stable, 0.9 Normal Saline should be run at a strict TKO rate. An IV is not necessary at scene unless seizure activity, hypoglycemia, or need for DAI.
**Cardiac Monitoring:** Stroke patients are at risk for adverse cardiac outcomes as a result of an increase in sympathetic tone and the release of catecholamines causing a proarrhythmic state. The increase in tone can also cause heart strain and myocardial infarction. Potential ECG changes include QT prolongation, T-wave flattening or inversion, ST segment alteration and supraventricular dysrhythmias. Given the wide range of cardiac risk factors and complications, continuous three-lead ECG monitoring should be done. Do not use Atropine for Bradycardia if SBP is above 90.

**Blood Glucose Monitoring:** Capillary blood glucose measurement should be obtained as soon as possible. Early hypoglycemic patients can present with focal neurological findings that mimic a stroke. In addition, severe and prolonged hypoglycemia can lead to brain injury, so prompt identification and correction of hypoglycemia is imperative. The identification of hyperglycemic patients is also important. Hyperglycemia is recognized as an independent risk factor for increased morbidity and mortality following stroke. Hyperglycemia worsens cerebral edema, enhances hemorrhagic transformation of the stroke and exacerbates the post-ischemic injury. In summary it is important to check glucose early to identify and treat hypoglycemic patients as well as identify hyperglycemic patients so that early in-hospital intervention can be started.

**Blood Pressure Management:** Blood pressure management in acute stroke has been a controversial topic. In theory, a reduction in blood pressure should prevent additional strokes, reduce further vascular damage, decrease cerebral edema and lessen the chance for hemorrhagic transformation of an ischemic area. However, reducing blood pressure could also reduce cerebral perfusion and lead to enlargement of the area at risk for ischemic injury. Cerebral autoregulation is often disrupted in the setting of ischemia, and cerebral perfusion depends almost entirely on systemic arterial blood pressure. For ischemic stroke, a range of systolic blood pressure (SBP) from 140 to 180 mmHg appears to be optimal and reducing SBP below 140 mmHg or by more than 20 mmHg is detrimental. Current recommendations for blood pressure management in ischemic stroke are to avoid anti-hypertensives in the acute setting unless SBP > 220 mmHg or if medically necessary for treating accompanying conditions such as acute myocardial infarction, decompensated heart failure, aortic dissection, acute renal failure or hypertensive encephalopathy. In hemorrhagic stroke the goal of blood pressure management is to maintain mean arterial blood pressure < 130 mmHg and cerebral perfusion pressure (CPP) > 70mmHg. Because of the importance of not over aggressively treating hypertension in acute stroke, blood pressure management should be done in a controlled manner with continuous assessment and close titration of medications. **Because it is very difficult to closely monitor blood pressures and titrate medications in the prehospital environment, current recommendations for the management of blood pressure in the prehospital environment for potential stroke patients are to not intervene and to let the body autoregulate.**
1. The American Heart Association has defined two primary categories of stroke: _____________ and ______________.

2. Approximately 85% of all strokes are hemorrhagic.
   A. True
   B. False

3. Ischemic strokes are further classified as either ____________, ____________, or _____________________.

4 – 6. Match the type of ischemic stroke with its characteristics.

<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombolic</td>
<td>A. Altered mental status and/or loss of speech, sensory or motor function, develops quickly. Often associated with an identifiable cause (e.g. atrial fib).</td>
</tr>
<tr>
<td>Embolic</td>
<td>B. Symptoms of brain dysfunction typically are diffuse and non-focal in contrast to the other two categories of ischemic stroke.</td>
</tr>
<tr>
<td>Systemic</td>
<td>C. Signs and symptoms develop gradually. This type of stroke often occurs at night with the patient awakening with altered mental status and/or loss of speech, sensory or motor function.</td>
</tr>
</tbody>
</table>

7. The most common causes of intracerebral hemorrhage are hypertension, trauma, vascular malformations and illicit drug use.
   A. True
   B. False
8 – 9  Match the type of hemorrhagic stroke with its characteristic.

<table>
<thead>
<tr>
<th></th>
<th>Intracerebral hemorrhage</th>
<th>A. Begins abruptly. In about half of the people, it begins with a severe headache, often during activity. However, in older people, the headache may be mild or absent. Symptoms suggesting brain dysfunction develop and steadily worsen as the hemorrhage expands. Some symptoms, such as weakness, paralysis, loss of sensation, and numbness, often affect only one side of the body. People may be unable to speak or become confused. Vision may be impaired or lost. The eyes may point in different directions or become paralyzed. The pupils may become abnormally large or small. Nausea, vomiting, seizures, and loss of consciousness are common and may occur within seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Subarachnoid hemorrhage</td>
<td>B. Begins abruptly, occurring at night in 30 percent of the cases. The primary symptom is sudden severe headache (97% of cases) classically described as the “worst headache of my life.” The headache is lateralized in 30% of patient, predominantly to the side of the aneurysm. The onset of the headache may or may not be associated with a brief loss of consciousness, seizure, nausea, vomiting, focal neurologic deficit, or stiff neck. There are usually not important focal neurologic signs at presentation unless bleeding occurs into the brain and CSF at the same time.</td>
</tr>
</tbody>
</table>

10. After returning home from a quick trip to the grocery store (she was gone 30 minutes) a women finds her husband with slurred speech and left –sided weakness. He was acting normally when she left. She calls 911. You arrive within 5 minutes of the call. You find him with the above symptoms. VS BP 198/98  HR 92 RR 18. Pulse Ox 95%. Discuss the important elements of the pre-hospital management of this patient.