NEUROVASCULAR EMERGENCIES

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ABSTRACT

The diagnosis of a neurovascular accident is multifactorial. It involves recognition of associated risk factors as well as signs and symptoms, which does not always guarantee a correct diagnosis. Depending on the neurovascular event, some injuries become more apparent after the actual neurovascular event occurs. Some signs are acutely severe and immediate while others can be subtle, making neurological injury diagnosis difficult and possibly delayed. Treatment with anticoagulants or thrombolytic agents is discussed, including inclusion and exclusion criteria for administration. The complications of a neurovascular accident are reviewed.
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Statement of Need:
The identification and treatment of a neurovascular accident is critical to prevent further damage. Lifestyle prevention is an essential aspect of nursing care of the patient with associated risk factors.

Course Purpose:
To provide nurses with the needed skills to promote health prevention and to treat neurovascular accidents in all health care settings.
Learning Objectives:
1. Define transient ischemic attack and stroke.
2. Identify treatment priorities for transient ischemic attack.
3. Identify treatment priorities for ischemic stroke.
4. Identify treatment priorities for hemorrhagic stroke.
5. Identify treatment priorities for subarachnoid hemorrhage

Target Audience:
Advanced Practice Registered Nurses, Registered Nurses, Licensed Vocational Nurses, and Associates

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Please take time to complete the self-assessment Knowledge Questions before reading the article. Opportunity to complete a self-assessment of knowledge learned will be provided at the end of the course.
1. **A transient ischemic attack:**
   a. causes temporary neurological dysfunction.
   b. is typically caused by a coagulation disorder.
   c. causes permanent neurological dysfunction.
   d. is very common in children and young adults.

2. **A transient ischemic attack:**
   a. is caused by bleeding into the subarachnoid space.
   b. is considered to be a significant risk factor for stroke.
   c. is characterized by persistent neurological deficits.
   d. does not require the use of neuroimaging.

3. **Treatment priorities for transient ischemic attack includes:**
   a. the use of rtPA and ICP monitoring.
   b. withdrawal of anticoagulants and antihypertensives.
   c. induced hypothermia and maintaining euvolemma.
   d. anticoagulation and antiplatelet therapy.

4. **An ischemic stroke is characterized by:**
   a. bleeding into the subarachnoid space.
   b. temporary neurological dysfunction caused by a thrombus or embolism.
   c. cerebral infarction caused by decreased blood flow.
   d. rupture of small arteries and bleeding into the brain parenchyma.

5. **Treatment priorities for ischemic stroke include**
   a. the use of rtPA and blood pressure management.
   b. ICP monitoring and prophylactic antibiotics.
   c. surgical evacuation of the hematoma and therapeutic hyperglycemia.
   d. monitoring for DCI and re-bleeding
6. A hemorrhagic stroke is characterized by:
   a. decreased blood flow caused by a thrombus or embolism.
   b. rupture of small arteries and bleeding into the brain parenchyma.
   c. bleeding into the subarachnoid space.
   d. hypoperfusion caused by a coagulopathy.

7. Treatment priorities for hemorrhagic stroke include:
   a. administration of rtPA and emergent anti-coagulation.
   b. therapeutic hypothermia and avoiding hyperglycemia.
   c. prophylactic anti-convulsants and prophylactic antibiotics.
   d. monitoring ICP and blood pressure control.

8. Subarachnoid hemorrhage is characterized by:
   a. temporary neurologic dysfunction.
   b. cerebral ischemia that causes an infarct.
   c. bleeding into the subarachnoid space.
   d. hypercoaguability that causes decreased brain perfusion.

9. Treatment priorities for subarachnoid hemorrhage include:
   a. monitoring for rebleeding and DCI.
   b. administration of rtPA and antiplatelet therapy.
   c. the use of hypertonic glucose solutions and factor Xa inhibitors.
   d. anticoagulation with warfarin and carotid endarterectomy.

10. Transient ischemic attacks and strokes are more likely to occur in patients who:
    a. are female, < age 45, and have a normal lipid profile.
    b. are elderly and who have atrial fibrillation and/or hypertension.
    c. are male, < age 50, and have insulin dependent diabetes.
    d. do not smoke, have a normal BNP, and are black.
Introduction

Neurovascular emergencies require timely and accurate assessments and treatments to ensure the best clinical outcomes. This course will give a brief overview of the anatomy of the neurovascular system, describe some of the common neurovascular emergencies, and explore the immediate assessment and treatment recommendations for each type of emergency as well as some of the potential complications care providers for these patients may encounter.

Neurovascular Anatomy

The neurovascular system consists of the brain, spinal cord and associated vasculature. This course will focus mainly on issues of the brain. The anatomy of the brain is complex due its intricate structure and function. This amazing organ acts as a control center by receiving, interpreting, and directing sensory information throughout the body. The brain is made up of many specialized areas that work together.

The cortex is the outermost layer of brain cells. Thinking and voluntary movements begin in the cortex. The basal ganglia are a cluster of structures in the center of the brain. The basal ganglia coordinate messages between multiple other brain areas. The limbic system is a group of structures that controls emotions, emotional responses, hormonal secretions, mood, motivation, as well as pain and pleasure sensations. The limbic system consists of the amygdala, cingulate gyrus, fornix, hippocampus, hypothalamus, olfactory cortex, and the thalamus. The brain is composed of the frontal, occipital, temporal, and parietal lobes as well as the cerebellum and the brain stem. Several layers of tissue including the meninges and the dura surround the brain. The skull (cranium) helps protect the brain from injury.
**Frontal Lobe**

The frontal lobe is located in the anterior part of the brain. It is involved in planning, organizing, problem solving, selective attention, personality and a variety of higher cognitive functions including behavior and emotions. The most anterior portion of the frontal lobe is called the prefrontal cortex. It is very important for the higher cognitive functions and the determination of the personality. The posterior portion of the frontal lobe consists of the premotor and motor areas. Nerve cells that produce movement are located in the motor areas. The premotor areas serve to modify movements.

**Occipital Lobe**

The occipital lobe is in the back of the brain and processes visual information. Not only is the occipital lobe mainly responsible for visual reception, it also contains association areas that help in the visual recognition of shapes and colors. Damage to this lobe can cause visual deficits.
**Temporal Lobe**

The temporal lobes are located on each side of the brain at about the level of the ears. These lobes allow one to differentiate sounds and smells. They also help in sorting new information and are believed to be responsible for short-term memory. The right temporal lobe is mainly involved in visual memory (*i.e.*, memory for pictures and faces) while the left temporal lobe is mainly involved in verbal memory (*i.e.*, memory for words and names).

**Parietal Lobe**

The parietal lobes are located on each side of the brain behind the frontal lobe at the top of the brain. The parietal lobes contain the primary sensory cortex, which controls sensation (touch, pressure). Behind the primary sensory cortex is a large association area that controls fine sensation (judgment of texture, weight, size, shape). Damage to the right parietal lobe can cause visuospatial deficits (*i.e.*, the patient may have difficulty finding his or her way around new, or even familiar, places), while damage to the left parietal lobe may disrupt a patient's ability to understand spoken and/or written language.

**Cerebellum**

The cerebellum is the portion of the brain (located at the back) that helps coordinate movement (balance and muscle coordination). Damage to this area may result in ataxia, which is a problem of muscle coordination. This can interfere with a person's ability to walk, talk, eat, and to perform other self-care tasks.
**Brainstem**

The brainstem is the lower extension of the brain where it connects to the spinal cord. Neurological functions located in the brainstem include those necessary for survival (breathing, digestion, heart rate, blood pressure) and for arousal (being awake and alert).

Most of the cranial nerves come from the brainstem. The brainstem is the pathway for all fiber tracts passing up and down from peripheral nerves and spinal cord to the highest parts of the brain. The brainstem is further divided into the midbrain, medulla oblongata, and the pons.

**Cerebral Vasculature**

The cerebral vascular system is also complex and intricate. The brain receives blood from two sources; the internal carotid arteries (ICAs), which arise at the point in the neck where the common carotid arteries bifurcate, and the vertebral arteries. The internal carotid arteries branch to form two major cerebral arteries, the anterior cerebral artery (ACA) and middle cerebral artery (MCA). The right and left vertebral arteries come together at the level of the pons on the ventral surface of the brainstem to form the midline basilar artery.

The basilar artery joins the blood supply from the internal carotids in an arterial ring at the base of the brain called the circle of Willis. The posterior cerebral arteries (PCAs) arise at this confluence, as do two small bridging arteries, the anterior and posterior communicating arteries. Conjoining the two major sources of cerebral vascular supply via the circle of Willis presumably improves the chances of any region of the brain continuing to receive blood if one of the major arteries becomes occluded.
The major branches that arise from the internal carotid artery - the anterior and middle cerebral arteries - form the anterior circulation that supplies the front part of the brain. These arteries also originate from the circle of Willis. Each gives rise to branches that supply the cortex and branches that penetrate the basal surface of the brain, supplying deep structures such as the basal ganglia and thalamus.

Especially prominent are the lenticuloostriate arteries that branch from the middle cerebral artery. These arteries also supply the basal ganglia and thalamus. The posterior circulation of the brain supplies the posterior cortex, the middle part of the brain and the brainstem. It comprises arterial branches arising from the posterior cerebral, basilar and vertebral arteries.

The pattern of arterial distribution is similar for all the subdivisions of the brainstem. Midline arteries supply medial structures, lateral arteries supply the lateral brainstem and dorsal-lateral arteries supply dorsal-lateral brainstem structures and the cerebellum. Among the most important dorsal-lateral arteries are the posterior inferior cerebella artery (PICA) and the anterior inferior cerebella artery (AICA), which supply specific areas of the medulla and pons. These arteries, as well as branches of the basilar artery that penetrate the brainstem from its ventral and lateral surfaces are especially common sites of occlusion and result in specific functional deficits of cranial nerve, somatic sensory and motor function.
Transient Ischemic Attack

A transient ischemic attack (TIA) is defined as “a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction.”¹ A TIA had traditionally been clinically defined by the temporary nature of the symptoms; a TIA was considered to produce reversible neurologic effects that lasted < 24 hours.² However, it has become clear that for many reasons this definition was inadequate.

The 24-hour time limit in the clinical definition of a TIA was arbitrary. The use of duration of symptoms as a dividing line that could be used to distinguish between a stroke and a TIA was misleading. The emphasis on time implied that a short duration of symptoms was equivalent to a decreased risk for neurological damage, but a TIA that lasts for only a few minutes can cause harm.
A TIA *can* cause cerebral infarctions, albeit small in size.² Ovbiagele, *et al.*, (2002) found that one-third of neurological episodes classified as a TIA by the traditional definition would be considered to be an infarction if a diffusion-weighted MRI scan had been done.³ Central nervous system (CNS) infarction is defined as brain, spinal cord, or retinal cell death caused by ischemia and confirmed by on pathologic, neuro-imaging, and/or clinical evidence of permanent injury.⁴

**Statistics**

Transient ischemic attacks are acknowledged to be very common,²,⁵ but the exact incidence of TIAs is not known. Depending on the source, each year anywhere from 200,000 to 5 million Americans each year have a TIA,⁵,⁶ and it appears that because of under-reporting the true number of TIAs is higher.⁷-⁹ Men have a higher risk of having a TIA than do women, and the risk of having a TIA increases with age.⁴,¹⁰

Transient ischemic attack is unusual in young adults age less than 50. The risk profile for TIA in this age group is often quite similar to that of older adults, *i.e.*, atherosclerosis, hypertension, dyslipidemia, *etc.*¹¹,¹² However, young adults are more likely to have less common risk factors for TIA such as a coagulation disorder.¹³

Transient ischemic attack in children is very unusual. Factors associated with TIA in children include sickle cell disease, congenital heart disease, moyamoya, recent stroke, and migraine.¹⁴
**TIA Etiology and Pathophysiology**

A transient ischemic attack is caused by focal brain, spinal cord, or retinal ischemia. This temporary decrease or cessation of blood flow to a specific area is the end result of a pathologic process; a TIA is not a disease in and of itself.\(^\text{15}\) There is a wide variety of diverse clinical conditions that can cause a TIA.\(^\text{15,16}\) The processes by which they cause ischemia can be usefully divided into three categories.\(^\text{6}\)

*Thrombosis or obstruction*

Atherosclerosis is the most common cause of obstruction that leads to a TIA; and, the carotid arteries, intracranial arteries, and vertebral arteries (major arteries located in the neck) are common sites of atherosclerosis that can cause a TIA. Hypertension is also a risk factor for TIA but a pathologic process called *lipohyalinosis* (thickening of the walls of the arterioles) causes the obstruction. Vascular obstruction and TIA can also be caused by arteritis (inflammation of the blood vessels), dissection of a vessel, and sympathomimetic drugs, such as cocaine. Additionally, fibromuscular dysplasia, a non-atherosclerotic, non-inflammatory condition that causes abnormal growth within the wall of the affected artery, may lead to TIA.\(^\text{17}\)

*Embolism*

An embolism causing TIA can be from a proximal area such as the aorta, carotid arteries, or the heart; or, it may be from the peripheral circulation. Cardiomyopathy, myocardial infarction, valvular heart disease, or a prosthetic heart valve can be the source of an embolism. Hypercoaguable states, which are typically caused by cancer, genetic condition, or infections, may also cause TIAs.\(^\text{15,18}\)
Decreased systemic perfusion

Decreased systemic perfusion caused by hypotension or heart failure can cause transient brain ischemia.\(^6,19\)

**TIA Risk Factors**

Risk factors for a TIA may be non-modifiable or modifiable, and there are other potential risk factors identified with TIA.

- **Non-modifiable risk factors:**
  Non-modifiable risk factors for TIA include age over 55, male gender, African American ethnicity, low birth weight, and a family history of stroke.\(^6,7\)

- **Modifiable risk factors:**
  Atrial fibrillation, diabetes mellitus, dyslipidemia, high alcohol intake, hypertension, obesity, patent foramen ovale, physical inactivity, and smoking are considered to be modifiable risk factors for TIA.\(^6,20-25\)

- **Potential risk factors:**
  - Acute respiratory and urinary infections, alcohol abstinence, drug abuse, hyperhomocysteinemia, high lipoprotein levels, hypercoaguability, infections with certain organisms, migraine with aura, and sleep-disordered breathing.\(^6\)

**TIA Signs and Symptoms**

The signs and symptoms of a TIA typically are seen in the patient’s behavior, gait, memory, movement or muscle function, memory, and
Patients may present with or have a history of aphasia, ataxia, diplopia, hemiparesis, sensory loss, muscle weakness, or vision loss. The signs and symptoms are often focal because the ischemia is caused by decreased perfusion to a specific area and a specific blood vessel or vessels; and transient, usually last between 30 and 60 minutes. The signs and symptoms and their duration are not accurate indicators of the absence of an infarction. In addition, the patient who is having a TIA will have symptoms of rapid onset, no prior history of similar symptoms, and he/she will not have non-specific symptoms. These three factors are considered highly suggestive for a TIA.

A focal sign, often called a focal deficit, is a neurological finding that indicates compromised function or damage to a specific area of the brain. This compromised function or damage then causes a decreased function in a specific part of the body. Examples of focal deficits are aphasia, blurred vision, facial droop, or weakness in one arm or leg. An example of a global sign or deficit would be loss of consciousness.

Unfortunately most patients who have had a TIA are examined by a health care professional after their symptoms have resolved. It has been estimated that ≤ 7% of patients who have had a TIA are examined when their symptoms are at the worst level. Approximately one half of all patients who have a TIA do not know the signs and
symptoms and do not tell their primary care provider about the episode.  

Transient ischemic attack and stroke have many similarities, and distinguishing between them requires a good knowledge base. Key differences between the two include:

- a stroke can cause global and focal deficits
- the signs and symptoms can be severe and life-threatening
- the duration of the signs and symptoms of stroke are typically much longer than that of a TIA
- irreversible tissue death is relatively common
- a stroke can be caused by intracranial hemorrhage
- treatment of a stroke often requires the use of specific therapy to preserve tissue and function

Conditions That Mimic TIA

Transient ischemic attack is a clinical diagnosis and it can be a difficult one to make. There is no confirmatory test, and the patients are often examined after the event and when they are asymptomatic. Research has shown that experienced neurologists can often disagree about whether or not a patient is having a TIA. There can be significant differences of opinion between emergency room physicians and neurologists when making the diagnosis of TIA, and that TIA is often misdiagnosed.

This difficulty is further increased by conditions that mimic TIA. There are many medical conditions that can be and often are misdiagnosed as TIA. The most common of these mimics are migraine aura, seizures, and syncope: Table 1 provides more examples. Many of
these are seldom encountered, very complex, or both, and it is beyond the scope of this module to explain each one. Interested readers are recommended to pursue further study found in the list of footnotes at the end of this study.

Table 1: Transient Ischemic Attack Mimics\textsuperscript{26,32,36}

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Cerebral amyloid angiopathy</td>
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<tr>
<td>Compressive myelopathy</td>
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<tr>
<td>Conversion disorder</td>
</tr>
<tr>
<td>Encephalopathies of hepatic, pulmonary or renal origin</td>
</tr>
<tr>
<td>Functional disorder</td>
</tr>
<tr>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Metastatic or primary tumor</td>
</tr>
<tr>
<td>Migraine aura</td>
</tr>
<tr>
<td>Paroxysmal symptoms associated with multiple sclerosis</td>
</tr>
<tr>
<td>Peripheral vestibular disorder</td>
</tr>
<tr>
<td>Pressure or position-related nerve compression</td>
</tr>
<tr>
<td>Seizures</td>
</tr>
<tr>
<td>Spinal dural arteriovenous fistulas</td>
</tr>
<tr>
<td>Structural brain lesions</td>
</tr>
<tr>
<td>Subdural hematoma</td>
</tr>
<tr>
<td>Syncope</td>
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<tr>
<td>Transient global amnesia</td>
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</tbody>
</table>

The number and complexity of conditions that mimic TIA makes the task of diagnosing a TIA even more daunting. However, there are characteristics of the TIA mimics that can be used to distinguish between these conditions and a TIA.\textsuperscript{6,36}
• **Age:**
  A TIA is unusual in children and young adults.

• **Negative symptoms:**
  Negative symptoms means that there is decreased function or loss of a function, and negative symptoms are more common in TIAs, less so in TIA mimics.

• **Onset:**
  The onset of a TIA is very sudden. In many of the TIA mimics the onset is gradual.

• **Progression:**
  A TIA starts and progresses quickly and then the signs and symptoms gradually decrease in intensity. This slope of progression is not seen for many of the TIA mimics.

• **Duration:**
  A seizure will typically last about five minutes, syncope for a few seconds, and a migraine aura may last for hours.

• **Symptom characteristics:**
  Amnesia or confusion, bladder and/or bowel incontinence, isolated sensory symptoms such as pins and needles, and tonic-clonic motions are indicative of a TIA mimic.\(^6\)

The *ABCD2 clinical prediction rule* can also be used to distinguish a TIA from a mimic or a non-cerebrovascular cause of neurological signs and symptoms. Several authors have found that the ABCD2 can be used to accurately discriminate between a TIA from these other conditions. The lower the ABCD2 score the less likely it is that the patient is having or has had a TIA.\(^{37,38}\) The ABCD2 will be discussed in more detail later in the study module.
Evaluation and Treatment

A TIA should be considered a medical emergency. Between 10%-15% of all patients who have had a TIA will eventually develop a stroke,\(^\text{39}\) usually within 90 days of the TIA.\(^\text{40}\) Approximately 50% of these strokes happen within 48 hours of the occurrence of the TIA.\(^\text{6,40,41}\) In addition, people who have had a TIA have an increased risk for myocardial infarction, unstable angina, or a ventricular arrhythmia within 90 days of a TIA.\(^\text{42-44}\) A timely diagnosis of TIA and starting preventative treatments has been shown to significantly reduce the early occurrence of stroke and the 90 day occurrence of stroke after a TIA.\(^\text{16,45,46}\)

Evaluation

Evaluation of the patient who is having or is suspected to have had a TIA should include the following tests and procedures.\(^\text{6,15,16,40,47}\) Some of these are important for the immediate evaluation, and others pertain to the evaluation of risk factors and for making decisions about preventative therapies.

- A comprehensive neurological and cardiovascular examination.
- An MRI scan if possible. An MRI scan is the preferred choice because this mode of scanning can detect small areas of ischemia that a CT scan cannot.\(^\text{6}\) Diffusion-weighted MRI is the mode of choice.
- Vascular imaging techniques, \textit{i.e.}, angiography, echocardiography, and ultrasound can be used to detect lesions and obstructions in the heart or the vasculature that may be the cause of the TIA.
- A 12-lead electrocardiogram and continuous cardiac monitoring. Long-term portable cardiac monitoring should be considered for the detection of atrial fibrillation.
- Cardiac enzymes, complete blood count, serum glucose, hemoglobin A1c, or an oral glucose tolerance test, serum electrolytes and creatinine, erythrocyte sedimentation rate, pulse oximetry, lipid profile, and coagulation studies.
- Screening tests for the unusual causes of TIA or TIA mimics should be done on a case-by-case basis.
- Screening for obesity and sleep apnea.

Evaluation of the patient with the ABCD2 clinical prediction rule is recommended. The ABCD2 uses five parameters to make an estimation of the risk of stroke within 2, 7, 30, and 90 days of a TIA.

**Table 2: The ABCD2 Scale**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Points</th>
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<tbody>
<tr>
<td>Age: &gt; 60 years</td>
<td>1 point</td>
</tr>
<tr>
<td>Blood pressure: &gt; 140 mm Hg systolic or &gt; 90 mm Hg diastolic</td>
<td>1 point</td>
</tr>
<tr>
<td>Clinical factors: Unilateral weakness with or without speech impairment</td>
<td>2 points</td>
</tr>
<tr>
<td>(1 point for speech impairment without weakness)</td>
<td></td>
</tr>
<tr>
<td>Duration of symptoms: &gt; 60 minutes</td>
<td>2 points</td>
</tr>
<tr>
<td>1 minute to 59 minutes</td>
<td>1 point</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1 point</td>
</tr>
</tbody>
</table>

The score can be from 0-7. A score of 0-3 is considered to represent a 1% risk for stroke within 48 hours; a score of 4-5 is considered to represent a 4.1% risk for stroke within 48 hours; and, a score of 6-7 is considered to represent a 8.1% risk for stroke within 48 hours. The
ABCD2 has been evaluated and found to be a reliable tool,\textsuperscript{15} but it has also been shown to have limits; the accuracy of the ABCD2 may depend on the population it is applied to and the experience of the clinician who uses it.\textsuperscript{16,48,49} Extensions of the ABCD2, the ABCD3 and ABCD3-1, have been developed\textsuperscript{50} and, although they have shown promise, they have not yet been validated for widespread use.\textsuperscript{51}

\textit{Treatment}

Assessment and stabilization of airway, breathing, and circulation (the ABCs) are the first treatments. If these are stable then the diagnostic work-up can proceed. The next step is to determine whether or not the patient needs to be hospitalized. Clear and universally accepted guidelines for making this decision are not available.\textsuperscript{16,52} The ABCD2 has been used for this purpose but it is not clear if it is effective.\textsuperscript{6}

Treatment of a TIA is essentially concerned with stroke prevention and treating the cause or causes of the TIA with the use of medication, surgery, and lifestyle modifications. The specific therapies used are discussed below.

\textbf{Anticoagulation:}

If the embolism was caused by non-valvular atrial fibrillation the patient should be anti-coagulated with warfarin, apixaban (Eliquis), dabigatran (Pradaxa), or rivaroxaban (Xarelto) to prevent embolism.\textsuperscript{6,47} The choice of which drug to use should be made on a case-by-case basis. If the patient is unable to take one of these anticoagulants, she/he should be prescribed aspirin.\textsuperscript{6,47} Clopidogrel (Plavix) can be used with aspirin or used alone if the use of aspirin is contraindicated.\textsuperscript{6,47,47}
Aspirin and clopidogrel have been shown to be effective at preventing stroke in patients who have atrial fibrillation, but not as effective as warfarin.\textsuperscript{47} Combining warfarin and antiplatelet therapy does not provide a decrease in the risk for stroke but does increase the risk for bleeding.\textsuperscript{47} It is not clear when anticoagulation should be initiated in this patient population.\textsuperscript{47}

There are also specific guidelines for anti-coagulation when a TIA has been caused by an aortic or mitral valve problem, cardiomyopathy, myocardial infarction, patent foramen ovale, or a prosthetic heart valve. These will not be discussed here in detail but in most cases anticoagulation is recommended.\textsuperscript{47}

Antiplatelet therapy:

Antiplatelet therapy is designed to prevent embolization or thrombus caused atherosclerosis, and if the TIA was caused by large or small vessel atherosclerosis the patient should be started on antiplatelet therapy rather than anticoagulant therapy.\textsuperscript{6,47} Antiplatelet therapy has been shown to be effective at preventing stroke after a TIA:\textsuperscript{47} aspirin, aspirin with dipyridamole (Persantine), clopidogrel alone are the drugs of choice.\textsuperscript{6,47} The choice of which drug to use should be made on a case-by-case basis.

Antiplatelet therapy should be started within 24 hours of the TIA.\textsuperscript{47} Antiplatelet therapy is also recommended for patients who have a significant degree of intracranial atherosclerosis, patent foramen ovale, or aortic arch atheroma.\textsuperscript{47}
Hypertension:

Antihypertensive therapy should be started if the patient who has had a TIA has an established systolic blood pressure ≥ 140 mm Hg or an established diastolic blood pressure ≥ 90 mm Hg.47 Antihypertensive therapy provides clear benefits.6 The target blood pressure is not clear nor is the optimal drug regimen, but there is some evidence that the angiotensin converting enzyme (ACE) inhibitors alone or with a diuretic, or an angiotensin receptor blocker (ARB), are the best choice.6,47

Life style modifications that can lower blood pressure such as limited alcohol consumption, a diet low in fat and high in fruits and vegetables, physical exercise, and salt restriction should also be initiated.47

Carotid endarterectomy, carotid angioplasty and stenting:

Carotid endarterectomy and carotid angioplasty and stenting have been successfully used in patients who have a significant degree of carotid artery stenosis, and these procedures are often recommended for secondary stroke prevention.6,47

Dyslipidemia:

Patients who have had a TIA that is thought to be caused by atherosclerosis and have a low-density lipoprotein cholesterol (LDL-C) level of ≥ 100 mg/dL should be started on statin therapy.47 This recommendation applies to all patients who fit those criteria whether or not there is evidence of other ASCVD. Lowering of LDL-C is
considered to be an important part of preventing secondary stroke.\(^6,47,53\)

Diabetes:

There are no large-scale studies that have shown that secondary prevention of stroke can be accomplished by treating either the pre-diabetes state or diabetes mellitus.\(^47\) However, there are many other benefits to treating diabetes. The 2014 guidelines for the prevention of stroke in patients with stroke and transient ischemic attack state:

"Use of existing guidelines from the ADA (American Diabetic Association) for glycemic control and cardiovascular risk factor management is recommended for patients with an ischemic stroke or TIA who also have DM or pre-DM."\(^47\)

Life style factors:

Life style modifications such as limited alcohol consumption, a diet low in fat and high in fruits and vegetables, physical exercise, salt restriction, smoking cessation, and weight loss if appropriate should also be initiated.\(^47\) The evidence for the effectiveness of these changes in the prevention of secondary stroke is not vigorous but they have obvious benefits aside from this.

There is a high prevalence of obstructive sleep apnea in people who have had a TIA or stroke. Evaluation for obstructive sleep apnea should be considered.
Other screening tests:

Homocysteine is an amino acid and elevated homocysteine levels have been observed in patients who have had a stroke. Antiphospholipid syndrome is an autoimmune disorder that causes peripheral thrombus formation. However, routine screening for hyperhomocysteinemia and antiphospholipid antibodies is not recommended.\textsuperscript{47}

Screening for hypercoaguability disorders should be done on a case-by-case basis and should be considered if the patient is age 45 or younger, has a TIA of undetermined origin, has a history of cancer or a clotting disorder, has multiple arterial or venous occlusions, or a family history of thrombosis.\textsuperscript{13,47}

**Stroke**

A stroke is a disease process that interrupts blood flow to the brain and causes cerebral infarction.\textsuperscript{54-56} Stroke and TIA are in many ways the same. They are both caused by a decrease in blood flow. The patient having a stroke or a TIA experiences neurological signs and symptoms, and they are each caused by an underlying chronic, pathological disease process. A stroke and a TIA are essentially similar processes and they could be viewed as separate points on a continuum and as symptoms of a disease rather than a disease.

Strokes, however, by definition involve tissue death - cerebral infarction. Neurons are highly sensitive to hypoperfusion and the resulting decrease and/or loss of oxygen and glucose. The normal cerebral blood flow is approximately 50-50mL/100 grams of brain tissue every minute. If the flow decreases to 20-40 mL/100 grams, neurological dysfunction will be observed and if the flow decreases to
10-15 mL/100 grams there will be irreversible tissue damage. The consequences of a stroke can be relatively mild. Unlike a TIA, however, many people who have suffered a stroke have permanent disabilities. In addition, intracranial bleeding causes some strokes whereas TIAs are not.

**Classification of Stroke**

Strokes can be categorized in several ways and there are several stroke classification schemes that have been developed that can be used to optimize treatment and prevention. However, all strokes are essentially ischemic or hemorrhagic and, as with a TIA, the pathogenesis of a stroke is thrombotic, embolic, or hypoperfusion. A reasonable way to view strokes is that an ischemic stroke is caused by decreased blood flow; and, intracerebral bleeding in the brain itself or in the subarachnoid space causes a hemorrhagic stroke. The majority of ischemic and hemorrhagic strokes have easily identifiable and common etiologies such as atherosclerosis, atrial fibrillation, or hypertension. Some stroke etiologies however are unusual and seldom seen, *i.e.*, strokes caused by hypercoagulability disorders, and, for some patients, the cause of the stroke cannot be determined or there may be several reasons the stroke has occurred; these are usually called *cryptogenic strokes*.

A commonly used classification scheme for ischemic strokes is *TOAST*, which stands for *Trial of Org 10712 in Acute Stroke Treatment*. The TOAST scheme considers the cause of the stroke, and the five TOAST ischemic stroke categories are: 1) Large
artery atherosclerosis; 2) Cardiac embolism; 3) Small vessel occlusion; 4) Stroke of another undetermined cause, and; 5) Stroke of undetermined cause.

The diagnosis and evaluation of ischemic and hemorrhagic stroke will be discussed together. Treatment of ischemic and hemorrhagic stroke will be discussed in separate sections.

Statistics

As with all major public health problems the statistics on stroke are quite sobering. These are itemized below.

1. Approximately 795,000 people each year in the United States have a stroke.\(^57\)
2. Of those 795,000 approximately 610,000 will have their first stroke and 185,000 will have had a prior stroke.\(^57\)
3. Eighty-seven percent of strokes are ischemic, 10% are hemorrhagic, and 3% are subarachnoid.\(^57\) The figures are approximate.
4. The incidence of stroke is higher in women than in men and higher in African Americans than in white Americans.\(^57\)
5. Stroke is a significant cause cognitive deficits and functional impairment in people who are ≥ 65 years of age.\(^58\)

Although stroke is a significant cause of morbidity and mortality, the death rate from stroke has been steadily decreasing.\(^31,59\) Stroke was for many years the third leading cause of death in the Unite States, but it is now the fourth leading cause of death.\(^59\) A recent (2014) review noted that although there are many possible causes for this decrease, \textit{i.e.}, the extensive use of statin drugs, more effective
therapy for atrial fibrillation, better control of hypertension is probably the primary reason why fewer people are dying from stroke.\textsuperscript{59}

\section*{Pathophysiology and Causes of Ischemic Stroke}

An ischemic stroke is caused by narrowing of a cerebral blood vessel, the narrowing being the result of a thrombus, an embolism, or hypoperfusion.\textsuperscript{60,61} The \textit{thrombotic} causes of ischemic stroke are:\textsuperscript{60} 1) Atherosclerosis; 2) Vasculitis; 2) Arterial dissection; 4) Polycythemia; 5) Hypercoaguable states, and; 6) Infections such as HIV and tuberculosis.\textsuperscript{31,60}

The \textit{embolic} causes of ischemic stroke are:\textsuperscript{60} 1) Valvular vegetation; 2) Mural thrombi; 3) Cardiac tumors; 4) Paradoxical embolism from a patent foramen ovale or other malformation; 5) Fat emboli; 6) Particulate emboli from intravenous drug use; 7) Septic emboli, and; 8) arterial-arterial emboli. Hypoperfusion that causes a stroke is usually the result of cardiac failure.\textsuperscript{60}

If the decrease in cerebral blood decrease is complete and persists for 4-10 minutes there will be irreversible damage and tissue death. Brain tissue that is not perfused does not receive oxygen and glucose. Without oxygen and glucose the mitochondria cannot product adenosine triphosphate (ATP), and if ATP is not produced the cells will die.\textsuperscript{31} The decrease in blood flow that occurs during an ischemic stroke also causes cell death by secondary mechanisms of injury, apoptosis, inflammation, and oxidative stress; these are collectively called \textit{reperfusion injury}.\textsuperscript{61-64}
Risk Factors for Ischemic Stroke

Non-modifiable

Age, low birth weight, race, and family history of stroke increase the risk for stroke.\textsuperscript{58} The risk for stroke increases significantly after age 55. Low birth weight ($\leq 2500$ grams) has clearly been associated with an increased risk for stroke,\textsuperscript{65,66} but it is not know if low birth weight is a cause for stroke.\textsuperscript{58}

African Americans and to a lesser degree Hispanic Americans suffer disproportionately from stroke and from morbidity and mortality caused by stroke.\textsuperscript{58,67,68} These effects are primarily, but not completely, explained by a higher incidence in these populations of modifiable risk factors, \textit{i.e.}, diabetes, dyslipidemia, and hypertension.\textsuperscript{58,68} A family history of stroke has been estimated to increase the risk of stroke by approximately 30\%.\textsuperscript{69,70}

Stroke is uncommon in adults under age 55. In this population non-modifiable and modifiable risk factors are certainly common, but if a young adult has a stroke uncommon causes of stroke should be part of the differential diagnosis list. For example, hypercoaguable states that can cause stroke are more common in younger adults and these include antiphospholipid syndrome, antithrombin III deficiency, essential thrombocytosis, Factor V Leiden (resistance to activated protein C) heparin induced thrombocytopenia, protein C or S deficiency, acquired or congenital, hyperhomocysteinemia, polycythemia vera, prothrombin gene mutation, and sickle cell anemia.\textsuperscript{61}
Modifiable

Atrial fibrillation, cigarette smoking, diabetes mellitus, diet and nutrition, dyslipidemia, hypertension, obesity and body fat distribution, other cardiac conditions, physical inactivity, and sickle cell disease are important risk factors for stroke. Of these, the evidence for an increase risk for stroke is strongest for atrial fibrillation, cigarette smoking, dyslipidemia, hypertension, physical inactivity, and sickle cell disease. Atrial fibrillation is described as “...a potent and treatable risk for embolic stroke.” Atrial fibrillation confers a five-fold increase in risk for stroke, and a stroke caused by atrial fibrillation is typically more severe and disabling than a stroke not caused by atrial fibrillation.

Cigarette smoking doubles the risk for ischemic stroke, this level of risk is almost identical for people exposed to second-hand smoke. During a four year period the number of strokes attributed to smoking was estimated to average almost 160,000. Dyslipidemia is considered to be a major risk factor for stroke and for recurrent stroke. Hypertension is a significant risk factor for stroke. Lackland et al., (2014) stated:

“Epidemiological studies have shown that elevated blood pressure is the most important determinant of the risk of stroke. The risk is almost linear, beginning at relatively low levels of systolic blood pressure (SBP) and DBP ... The evidence for the benefits of lower blood pressures and reduced stroke risks is strong, continuous, graded, consistent, independent, predictive, and pathogenically significant for those with and without coronary heart disease.”
Physical activity has long been considered to be a major risk factor for stroke.\textsuperscript{57} Many studies have noted the inverse relationship between level of physical activity and the risk for stroke.\textsuperscript{78-80} Stroke is a major complication of sickle cell disease:\textsuperscript{81,82} Prengler \textit{et al.}, (2002) found that 10\% of all children who have sickle cell disease will have a cerebrovascular event by the age of 20.\textsuperscript{83}

The factors that increase risk for stroke have been identified, but using these for assessing a single patient’s risk for having a stroke is an inexact science. There are stroke risk assessment tools, however, the 2014 American Heart Association/American Stroke Association review on stroke prevention states that, although the effect of individual factors on stroke risk is clearly outlined, the interaction between individual risk factors and how that interaction affects stroke risk is less well understood. Additionally, the effectiveness of risk assessment tools for primary stroke prevention has not been well studied; and, the existing tools do not include all possible risk factors. They do not completely account for age, gender, and race.\textsuperscript{58}

There is no ideal stroke risk assessment tool, but the 2014 review notes that the AHA/ACC CV Risk calculator can help identify patients who may benefit from therapeutic interventions that can prevent stroke.\textsuperscript{58} The AHA/ACC CV Risk calculator uses age, race, total cholesterol, HDL cholesterol, systolic and diastolic blood pressure, presence of diabetes, smoking, and treatment for hypertension to determine the 10 year risk for stroke. The risk

\textbf{AHA/ACC CV RISK CALCULATOR}

A quick online resource for health professionals, available online at:

Signs and Symptoms

The clinical presentation of an ischemic stroke is highly variable and it will depend on the patient’s age, co-morbidities, and the size and location of the affected area. Commonly seen signs and symptoms are outlined in the table below.

Table 3: Signs and Symptoms of Ischemic Stroke

<table>
<thead>
<tr>
<th>Aphasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ataxia</td>
</tr>
<tr>
<td>Decreased or altered consciousness</td>
</tr>
<tr>
<td>Diplopia</td>
</tr>
<tr>
<td>Dysarthria</td>
</tr>
<tr>
<td>Facial droop</td>
</tr>
<tr>
<td>Focal or global deficits</td>
</tr>
<tr>
<td>Hemiparesis</td>
</tr>
<tr>
<td>Visual defects</td>
</tr>
</tbody>
</table>

Hemorrhagic Stroke

Hemorrhagic stroke is caused by an intracerebral bleed or a subarachnoid hemorrhage. This section of the module will discuss intracerebral bleeding and stroke; stroke caused by subarachnoid hemorrhage will be discussed separately. Hemorrhagic strokes, either intracerebral or subarachnoid, are much less common than ischemic strokes but they are much more serious. These strokes are typically
severe, they have a high short-term and long-term mortality rate and they are more disabling than ischemic strokes.

The clinical presentation of a hemorrhagic stroke is essentially the same as that of an ischemic stroke but there are some differences. Intracranial bleeding causes increased intracranial pressure so that alterations in consciousness, headache, nausea, and vomiting are more common in hemorrhagic strokes than in ischemic strokes.\textsuperscript{56} Seizures are also more common in hemorrhagic strokes.\textsuperscript{56} Hypertension is also relatively common. Despite these differences signs and symptoms alone cannot distinguish between a hemorrhagic stroke and an ischemic stroke.

Statistics

- Intracranial hemorrhage and stroke are less common than ischemic stroke but hemorrhagic strokes are usually more severe. The fatality rate at one month post-stroke has been reported to be from 35\%\textsuperscript{85} to 50.3\%\textsuperscript{86}
- Approximately 50\% of these deaths happen within two days of the stroke.\textsuperscript{87,88}
- Hemorrhagic stroke is associated with a higher mortality rate and a higher rate of disabilities than ischemic stroke.\textsuperscript{89}

Pathophysiology and Etiology of Hemorrhagic Stroke

An intracerebral hemorrhage is caused by the rupture of small arteries and bleeding into the brain parenchyma.\textsuperscript{90,91} Disruption of blood supply to the affected areas interrupts the delivery of oxygen and glucose, and causes cell death. Because the skull is a closed space the bleeding causes increased intracranial pressure and secondary mechanisms,
which are similar to those caused by an ischemic stroke, will occur after the initial hemorrhage.\textsuperscript{91}

Hypertension is the most common cause of hemorrhagic stroke, cerebral amyloid angiopathy is the most common cause of non-traumatic lobar intracranial hemorrhage in the elderly. Vascular malformations are the most common cause of intracranial hemorrhage in children.\textsuperscript{91} The causes of hemorrhagic stroke are listed in the table below.\textsuperscript{90,91}

\begin{table}[h]
\centering
\begin{tabular}{|l|}
\hline
\textbf{Anticoagulant therapy} \\
\hline
\textbf{Brain tumor} \\
\hline
\textbf{Central nervous system infections} \\
\hline
\textbf{Cerebral amyloid angiopathy} \\
\hline
\textbf{Coagulopathies} \\
\hline
\textbf{Hypertension} \\
\hline
\textbf{Moyamoya} \\
\hline
\textbf{Septic embolism} \\
\hline
\textbf{Sympathomimetic drug abuse} \\
\hline
\textbf{Thrombolytic therapy for myocardial infarction or stroke} \\
\hline
\textbf{Vascular malformations} \\
\hline
\end{tabular}
\caption{Causes of Hemorrhagic Stroke}
\end{table}

A complete discussion of all the causes of hemorrhagic stroke would not be practical here, and this module will focus on four of the most common: hypertension, cerebral amyloidosis, anticoagulant therapy, and thrombolytic therapy.
**Hypertension**

Hypertension is the most common cause of hemorrhagic stroke, and it is also the biggest risk factor for hemorrhagic stroke.\(^{90-92}\) Hypertension has been associated with 60% of all cases of hemorrhagic stroke.\(^{56}\) It has been suggested that hypertension doubles the risk for intracerebral hemorrhage.\(^{93}\)

**Cerebral amyloid angiopathy**

Cerebral amyloid angiopathy (CAA) is a disease that is characterized by the accumulation of amyloid beta peptide deposits in small and medium-sized blood vessels in the brain.\(^{94,95}\) The disease is relatively common in the elderly, both in people with normal neurological functioning and those with dementia. It causes progressive fibrosis and necrosis of the vasculature, and CAA is a common cause of hemorrhagic stroke in the elderly.\(^{96,97}\)

**Anticoagulant therapy**

Anticoagulation with warfarin or other drugs is a widely used therapy for the prevention and treatment of thromboembolism. Anticoagulation therapy is very effective at preventing TIA and stroke, but the use of warfarin carries a significant risk of intracerebral hemorrhage. The risk has been estimated at 1% per year,\(^{98}\) however, it may be higher,\(^{98,99}\) The mortality rate from intracranial hemorrhage and stroke caused by anticoagulation with warfarin has been reported to be 46% - 68%.\(^{98}\)

The risk of intracerebral hemorrhage caused by anticoagulation therapy has been reported to be higher for Asians, blacks, and the
elderly. Additionally, people who have had a stroke or a TIA, have an elevated diastolic blood pressure, or a reduced baseline serum albumin or platelet count are reported to be at higher risk of intracerebral hemorrhage with anticoagulation therapy.\textsuperscript{100} The newer anticoagulants, \textit{i.e.}, dabigatran, rivaroxaban, do not appear to be more likely than warfarin to cause intracerebral bleeding.\textsuperscript{101}

\textit{Thrombolytic therapy}

Thrombolytic therapy with recombinant tissue plasminogen activator (r-tPA) is a highly effective therapy for treating ischemic stroke. Recombinant tissue plasminogen activator can cause bleeding, and the rate of symptomatic intracranial hemorrhage caused by r-tPA given to patients having an ischemic stroke has been reported to be 3.3\% - 7.3\%.\textsuperscript{102-105}

Other causes of hemorrhagic stroke, such as moyamoya disease, are much less common than hypertension or anticoagulant drug therapy; or they are widespread, as with illicit sympathomimetic drug use. However, determining the incidence of stroke is difficult.\textsuperscript{106-107}

\textbf{Risk Factors for Hemorrhagic Stroke}

Risk factors for and causes of hemorrhagic stroke overlap. A condition such as hypertension can be both a risk factor and cause of stroke. Also, risk factors interact and are synergistic.\textsuperscript{108} Risk factors for hemorrhagic stroke include the following:\textsuperscript{56,91}

- Advanced age.
- High intake of alcohol: High intake of alcohol has been clearly and consistently associated with an increased risk for
hemorrhagic stroke.\textsuperscript{109-111} People who drink heavily often have hypertension; this effect and changes in platelet aggregation and fibrinolysis may explain why heavy alcohol consumption increases the risk for hemorrhagic stroke.\textsuperscript{111}

- Hypertension.
- Low serum cholesterol, low serum triglycerides: Low serum cholesterol and low serum triglycerides are inversely related to the risk for, and mortality from, hemorrhagic stroke.\textsuperscript{112-115} Lipid levels may contribute to the risk for, and mortality of, hemorrhagic stroke and mortality by causing necrosis of the arterial walls; and, may reflect poor nutritional status and/or the presence of chronic disease.\textsuperscript{112}
- Previous stroke: A previous stroke increases the risk for intracerebral hemorrhage.\textsuperscript{116}
- Ethnic status: Blacks have a higher risk for hemorrhagic stroke than Caucasions,\textsuperscript{117,118} and in some age groups this risk is five times higher.\textsuperscript{118}

**Signs and Symptoms of Hemorrhagic Stroke**

The clinical presentation of hemorrhagic stroke was previously discussed. It is important to emphasize that signs and symptoms alone are not specific enough to distinguish ischemic from hemorrhagic stroke, but that indications of increased intracranial pressure such as nausea, vomiting, and headache, as well as an altered level of consciousness, and seizures are more common with hemorrhagic strokes and large ischemic strokes.
Evaluation Of The Stroke Patient

This section discusses the evaluation process for a patient suspected of having a stroke.\textsuperscript{56,119,120}

Stabilization of ABCs

The patient should be on continuous cardiac monitoring. The blood pressure and temperature should be closely monitored.

History and Physical Examination

A complete neurological exam and a detailed history should be obtained. The single most important piece of historical information is the time of symptom onset. Stroke scales such as the NIHSS (shown in the chart below) or the Canadian Neurological Scale can be used to determine the severity of the stroke.\textsuperscript{121-122}

\begin{tabular}{|l|l|}
\hline
\textbf{NIHSS Scale} & \\
\hline
\textbf{Instructions} & \textbf{Scale definition} & \textbf{Score} \\
\hline
1a. Level of consciousness: The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation. & 0 = Alert; keenly responsive.  \\
& 1 = Not alert; but arousable by minor stimulation to obey, answer, or respond.  \\
& 2 = Not alert; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped).  \\
& 3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, and areflexic. & \\
\hline
\end{tabular}
**1b. LOC questions:** The patient is asked the month and his/her age. The answer must be correct - there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not "help" the patient with verbal or non-verbal cues.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Answers both questions correctly.</td>
</tr>
<tr>
<td>1</td>
<td>Answers one question correctly.</td>
</tr>
<tr>
<td>2</td>
<td>Answers neither question correctly.</td>
</tr>
</tbody>
</table>

**1c. LOC commands:** The patient is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (ie, follows none, one or two commands). Patients with trauma, amputation, or other physical impediments should be given

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Performs both tasks correctly.</td>
</tr>
<tr>
<td>1</td>
<td>Performs one task correctly.</td>
</tr>
<tr>
<td>2</td>
<td>Performs neither task correctly.</td>
</tr>
</tbody>
</table>
suitable one-step commands. Only the first attempt is scored.

2. Best gaze: Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, pre-existing blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.

0 = Normal.
1 = Partial gaze palsy; gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present.
2 = Forced deviation, or total gaze paresis not overcome by the oculocephalic maneuver.

3. Visual: Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be

0 = No visual loss.
1 = Partial hemianopia.
2 = Complete hemianopia.
3 = Bilateral hemianopia (blind including cortical blindness).
scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and the results are used to respond to item 11.

| 4. Facial palsy: Ask - or use pantomime to encourage - the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or non-comprehending patient. If facial trauma/bandages, orotracheal tube, tape or other physical barriers obscure the face, these should be removed to the extent possible. | 0 = Normal symmetrical movements. 1 = Minor paralysis (flattened nasolabial fold, asymmetry on smiling). 2 = Partial paralysis (total or near-total paralysis of lower face). 3 = Complete paralysis of one or both sides (absence of facial movement in the upper and lower face). |

<p>| 5. Motor arm: The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and | 0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds. 1 = Drift; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = Some effort against gravity; limb cannot get to or maintain (if |</p>
<table>
<thead>
<tr>
<th>6. Motor leg: The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.</th>
<th>0 = No drift; leg holds 30-degree position for full 5 seconds. 1 = Drift; leg falls by the end of the 5-second period but does not hit bed. 2 = Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity. 3 = No effort against gravity; leg falls to bed immediately. 4 = No movement. UN = Amputation or joint fusion, explain:________________ 6a. Left leg 6b. Right leg</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Limb ataxia: This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. 3 = No effort against gravity; limb falls. 4 = No movement. UN = Amputation or joint fusion, explain:________________ 5a. Left arm 5b. Right arm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.

8. Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, "severe or total sensory loss," should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2.

0 = Normal; no sensory loss.
1 = Mild-to-moderate sensory loss; patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched.
2 = Severe to total sensory loss; patient is not aware of being touched in the face, arm, and leg.
| Patients in a coma (item 1a=3) are automatically given a 2 on this item. | 0 = No aphasia; normal.  
1 = Mild-to-moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response.  
2 = Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response.  
3 = Mute, global aphasia; no usable speech or auditory comprehension. |  
| 9. Best language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a=3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands. |  
| 10. Dysarthria: If patient is thought to be normal, an adequate sample  
0 = Normal.  
1 = Mild-to-moderate dysarthria; |  
|
of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN), and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.

| 11. Extinction and inattention (formerly neglect): Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosognosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable. | patient slurs at least some words and, at worst, can be understood with some difficulty.
2 = Severe dysarthria; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric.
UN = Intubated or other physical barrier, explain:________________ |
| 0 = No abnormality.
1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.
2 = Profound hemi-inattention or extinction to more than one modality; does not recognize own hand or orients to only one side of space. |
The following diagnostic tests should be performed on all stroke patients during the evaluation process.⁵⁶,¹¹⁹,¹²⁰

**Table 5: Diagnostic Tests for Stroke Evaluation**

<table>
<thead>
<tr>
<th>Diagnostic Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activate partial thromboplastin time (aPTT)*</td>
</tr>
<tr>
<td>Blood glucose</td>
</tr>
<tr>
<td>Complete blood count</td>
</tr>
<tr>
<td>INR*</td>
</tr>
<tr>
<td>ECG*</td>
</tr>
<tr>
<td>Markers of cardiac ischemia*</td>
</tr>
<tr>
<td>Non-contrast brain CT or brain MRI</td>
</tr>
<tr>
<td>Oxygen saturation</td>
</tr>
<tr>
<td>Prothrombin time*</td>
</tr>
<tr>
<td>Platelet count*</td>
</tr>
<tr>
<td>Renal function tests*</td>
</tr>
<tr>
<td>Serum electrolytes*</td>
</tr>
</tbody>
</table>

*Fibrinolytic therapy should not be delayed for these test results unless:
1) there is clinical suspicion of a bleeding abnormality or thrombocytopenia; 2) the patient has received heparin or warfarin, or; 3) the patient has received other anticoagulants.

The diagnostic tests listed in the table below, page 45, should be performed for selected patients during the evaluation of a stroke.⁵⁶,¹¹⁹,¹²⁰
Table 6: Diagnostic tests for Stroke Evaluation/Selected Patients

<table>
<thead>
<tr>
<th>Arterial blood gas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood alcohol level</td>
</tr>
<tr>
<td>Chest x-ray</td>
</tr>
<tr>
<td>Electroencephalogram</td>
</tr>
<tr>
<td>Hepatic function tests</td>
</tr>
<tr>
<td>Lumbar puncture</td>
</tr>
<tr>
<td>Pregnancy test</td>
</tr>
<tr>
<td>Thrombin time (TT) and/or ecarin clotting time (ECT)</td>
</tr>
<tr>
<td>Toxicology screen</td>
</tr>
</tbody>
</table>

**Brain and vascular imaging**

Emergency imaging of the brain by CT scan or MRI scan should be done, and it should be done before any specific therapy is started. Non-invasive vascular imaging of the intracranial blood supply is strongly recommended in order to detect large vessel occlusion that may need fibrinolytic therapy or thrombectomy. This procedure should not be done if it would delay the use of rtPA. CT angiography, conventional angiography, MRI-angiography, and Doppler ultrasound can all be used. Assessment of the carotid blood supply can be done, as well.

**Subarachnoid Hemorrhage And Stroke**

Subarachnoid hemorrhage is a bleed into the subarachnoid cavity, the space between the arachnoid membrane and the pia mater. It is an infrequent cause of stroke and most cases are caused by rupture of an aneurysm. A subarachnoid hemorrhagic stroke is a potentially devastating event. The mortality rate is approximately 50%\textsuperscript{124,125}
and, as with other types of hemorrhagic stroke, the 30 day mortality rate is high.\textsuperscript{126}

A stroke caused by a subarachnoid hemorrhage is characterized by the sudden onset of a very severe headache. The headache is usually localized on the same side of the head where the bleeding is occurring. Dizziness, hyponatremia, nausea, and vomiting are often seen and a “sentinel” headache that precedes the subarachnoid hemorrhage by hours or a day is relatively common.\textsuperscript{127,128}

**Causes and Risk Factors** \textsuperscript{129,130}

- Alcohol use
- Amyloid angiography
- Aneurysms
- Cigarette smoking
- Coagulation disorders
- Hypertension
- Infection
- Moyamoya disease
- Neoplasms
- Oral contraceptives,
- Stimulants

There are assessment scales that have been developed for grading the severity of subarachnoid hemorrhage. These are the Hunt and Hess scale, World Federation of Neurological Surgeons (WFNS) scale, Fisher scale, Claasen grading system, and the Ogilvy and Carter scale. The Glasgow coma scale has been used for this purpose, as well. There is no evidence that one is better than another.\textsuperscript{123} The Glasgow coma
scale was not designed for assessment of subarachnoid hemorrhage. The Hunt and Hess (outlined in the table on page 48) is widely used.

Re-bleeding has been reported to occur in 8%-23% of patients.\textsuperscript{131} This will usually happen in the first 24 hours and the risk is especially high in the first six hours.\textsuperscript{123} The risk for re-bleeding can be predicted by: 1) the Hunt and Hess grade; 2) the size of the aneurysm; 3) blood pressure; 4) the presence of a sentinel headache; 5) a longer interval between the onset and when medical care is sought.\textsuperscript{123}

**Hunt and Hess Grading system for subarachnoid hemorrhage.**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Neurologic status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic or mild headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Severe headache, stiff neck, no neurologic deficit except cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsy or confused, mild focal neurologic deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stuporous, moderate or severe hemiparesis</td>
</tr>
<tr>
<td>5</td>
<td>Coma, decerebrate posturing</td>
</tr>
</tbody>
</table>

Evaluation of suspected subarachnoid hemorrhage is essentially the same as for other types of stroke but there are important differences. Headache is often the only presenting symptom of a subarachnoid hemorrhage but headache is a very common complaint and only a small percentage of people who have a headache have a subarachnoid hemorrhage.\textsuperscript{132} Because the morbidity and mortality of subarachnoid hemorrhage are quite high, and re-bleeding relatively common, it is important to quickly and accurately make a diagnosis.
If there is a suspicion that the patient is having a subarachnoid hemorrhage, a non-contrast CT scan of the head should be performed immediately and a lumbar puncture should be performed. The non-contrast CT scan is very sensitive at detecting subarachnoid hemorrhage if the hemorrhage occurred less than 24 hours prior to the exam, but the sensitivity of the CT scan for this purpose decreases with time and lumbar puncture is recommended.

Some authors feel that if the patient has an acute headache, the CT scan of the head is negative, and if the onset of the headache was 6 hours or less prior to the performance of the scan a lumbar puncture is not needed. More recent reviews recommend a lumbar puncture regardless of the CT scan results.

Misdiagnosing a subarachnoid hemorrhage can be potentially catastrophic. A 2013 review of the literature found a rate of misdiagnosis that ranged from 2.5% to 21.7%. The reasons for misdiagnosis are: 1) failure to understand the signs and symptoms associated with subarachnoid hemorrhage; 2) not performing a CT scan or not understanding the limitations of the procedure, and; 3) not performing a lumbar puncture.
Treatment Of Ischemic Stroke

Patients should be admitted to the ICU and have continuous hemodynamic and neurologic monitoring. The following recommendations are from the 2013 guidelines from the American Heart Association and the American Stroke Association (AHA/ASA).

- Stabilization of ABCs

- Patient positioning: If the patient is hypoxic or at risk for airway obstruction, aspiration, increased intracranial pressure, the head of the bed should be elevated at a 15-30 degree angle.

- Oxygen: Many patients who have had a stroke are hypoxic. Supplemental oxygen should be used to maintain an oxygen saturation of greater than 94%. Supplemental oxygen should not be used if the patient is not hypoxic.

- Hyperthermia: Hyperthermia is common after a stroke and it has been associated with poor outcome. If the temperature is greater than 38°C the source should be identified and treated. Patients who are hyperthermic should have their body temperature lowered.

- Blood pressure monitoring: Hypertension is common in patients who have had a stroke; hypotension is uncommon. Maintaining adequate perfusion is important as is avoiding dangerous elevations of blood pressure, but the ideal level of blood pressure for patients who have had an ischemic stroke is not known. The recommendations in Table 7 are from the 2013 AHA/ASA 2013 guidelines for managing ischemic stroke.
Table 7: Blood Pressure Management in Patients with Ischemic Stroke

*Patients who are otherwise eligible for reperfusion therapy but the BP is > 185/110 mm Hg:*

- Labetalol 10–20 mg IV over 1–2 minutes, may repeat 1 time; or
- Nicardipine 5 mg/hr IV, titrate up by 2.5 mg/hr every 5–15 minutes, maximum 15 mg/hr; when desired BP reached, adjust to maintain proper BP limits; or other drugs may be used when needed.

If BP is not maintained at or below 185/110 mm Hg, do not administer rtPA

*Management of BP during and after rtPA or other acute reperfusion therapy to maintain BP at or below 180/105 mm Hg:*

- Check BP every 15 minutes for 2 hours from the start of rtPA therapy, then every 30 minutes for 6 hours, and then every hour for 16 hours

*If systolic BP >180–230 mm Hg or diastolic BP >105–120 mm Hg:*

- Labetalol 10 mg IV followed by continuous IV infusion 2–8 mg/min; or
- nicardipine 5 mg/h IV, titrate up to desired effect by 2.5 mg/h every 5–15 minutes, maximum 15 mg/hr

*If BP not controlled or diastolic BP >140 mm Hg,*

*IV sodium nitroprusside can be used*

- IV hydration:
  Euvolemia is recommended. Hypovolemia decreases perfusion to the organs and hypervolemia can increase cerebral edema and is stressful for the myocardium.
Serum glucose:
Hyperglycemia is common in patients who have had an ischemic stroke and several authors have found an incidence of hyperglycemia to be greater than 40% in this patient population. Elevated serum glucose is common in the acute phase of stroke and may be related to uncontrolled or undetected diabetes mellitus or stress-induced hyperglycemia associated with cortisol and norepinephrine release at the time of insult.

Hyperglycemia has consistently been associated with poor outcome after stroke, but it is not clear if hyperglycemia is a cause of poor outcome or an effect. The 2013 AHA/ASA guidelines recommend: 1) Treating a serum glucose that is < 60 mg/dL; and, 2) “...treat hyperglycemia during acute stroke in a manner that avoids excessive resources, labor, and risk.

It is reasonable to follow the American Diabetes Association recommendation and maintain the blood glucose in a range of 110-180 mg/dL in all hospitalized patients.

Administer recombinant tissue plasminogen activator (rtPA) if indicated.

Rapid assessment, evaluation, and treatment of ischemic stroke is essential. The following Table from the 2013 AHA/ASA guidelines outlines the optimal times for these aspects of stroke care.
### Table 8: Optimal Stroke Care time Limits

<table>
<thead>
<tr>
<th>Step</th>
<th>Time Limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Door to physician</td>
<td>≤10 minutes</td>
</tr>
<tr>
<td>Door to stroke team</td>
<td>≤15 minutes</td>
</tr>
<tr>
<td>Door to CT initiation</td>
<td>≤25 minutes</td>
</tr>
<tr>
<td>Door to CT interpretation</td>
<td>≤45 minutes</td>
</tr>
<tr>
<td>Door to drug (≥80% compliance)</td>
<td>≤60 minutes</td>
</tr>
<tr>
<td>Door to stroke unit admission</td>
<td>≤3 hours</td>
</tr>
</tbody>
</table>

---

**Thrombolytic Therapy**

Thrombolytic therapy with rtPA is the only Food and Drug Administration (FDA) approved therapy for the treatment of ischemic stroke. The use of rtPA has been shown to improve outcomes in patients who have had an ischemic stroke if the drug can be given 4.5 hours or less from the onset of symptoms.\(^{120,143}\) Recombinant tissue plasminogen activator promotes clot dissolution, aka fibrinolysis, by binding to fibrin in a clot and converting plasminogen to plasmin. These drugs, *i.e.* alteplase (Activase®) are enzymes that increase the conversion of plasminogen to plasmin; plasminogen is the inactive precursor to plasmin, and plasmin is an enzyme that breaks down fibrin clots. Recombinant tissue plasminogen activators must be given carefully and correctly. The following guidelines are from the 2013 AHA/ASS guidelines.\(^{120}\) The relative exclusion criteria refer to circumstances in which the benefit of rtPA may outweigh the risks.

The newer anticoagulants are becoming popular. Using rtPA in patients who are have been taking one of these drugs that act by inhibiting factor Xa or thrombin is *not* recommended *unless* laboratory tests such
as aPTT, ECT, INR, factor Xa activity assay, platelet count, and TT time can be measured and are normal or if the patient has not had dose in greater than 48 hours and his/her renal function is normal. The recommendations for inclusion and exclusion criteria are outlined in the Tables on page 54 - 56.

**Table 9: rtPA Inclusion Criteria**

<table>
<thead>
<tr>
<th>Ischemic stroke and measurable neurological deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of symptoms &lt; 3 hours before beginning treatment</td>
</tr>
<tr>
<td>Aged ≥ 18 years</td>
</tr>
</tbody>
</table>

**Table 10: rtPA Absolute Exclusion Criteria**

<table>
<thead>
<tr>
<th>Active internal bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial puncture at non-compressible site in previous 7 days</td>
</tr>
<tr>
<td>Blood glucose &lt; 50 mg/dL</td>
</tr>
<tr>
<td>CT shows multilobar infarction (hypodensity &gt;1/3 cerebral hemisphere)</td>
</tr>
<tr>
<td>History of intracranial hemorrhage</td>
</tr>
<tr>
<td>Intracranial neoplasm, arteriovenous malformation, or aneurysm</td>
</tr>
<tr>
<td>Recent intracranial or intraspinal surgery</td>
</tr>
<tr>
<td>Significant head trauma or a stroke in previous 3 months</td>
</tr>
<tr>
<td>Symptoms suggest subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Systolic BP &gt;185 mm Hg or diastolic &gt;110 mm Hg</td>
</tr>
<tr>
<td>Acute bleeding diathesis, including but not limited to:</td>
</tr>
<tr>
<td>a. Platelet count &lt;100 000/mm3</td>
</tr>
<tr>
<td>b. Heparin received within 48 hours, resulting in abnormally elevated aPTT greater than the upper limit of normal</td>
</tr>
<tr>
<td>c. Current use of anticoagulant with INR &gt;1.7 or PT &gt;15 seconds</td>
</tr>
<tr>
<td>d. Current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevations of laboratory tests such as aPTT, ECR, factor Xa assay, INR, platelet count, and thrombin time</td>
</tr>
</tbody>
</table>
Table 11: rtPA Relative Exclusion Criteria

<table>
<thead>
<tr>
<th>Relative Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction (within 3 months)</td>
</tr>
<tr>
<td>Gastrointestinal or urinary tract hemorrhage within 21 days</td>
</tr>
<tr>
<td>Major surgery or serious trauma within 14 days</td>
</tr>
<tr>
<td>Minor or rapidly improving stroke symptoms</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Seizure and postictal residual neurological impairments</td>
</tr>
</tbody>
</table>

Table 12: Inclusion Criteria: Symptom Onset between 3 - 4.5 Hours

| Additional |
| Ischemic stroke causing measurable neurological deficit |
| Symptoms onset within 3 to 4.5 hours before beginning treatment |

Table 13: Relative Exclusion Criteria: Symptom Onset Between 3 - 4.5 hours

<table>
<thead>
<tr>
<th>Relative Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;80 years</td>
</tr>
<tr>
<td>History of diabetes and prior ischemic stroke</td>
</tr>
<tr>
<td>NIHSS score &gt;25</td>
</tr>
<tr>
<td>Taking an oral anticoagulant regardless of INR</td>
</tr>
</tbody>
</table>
Table 14: Administration of rtPA

1. Infuse 0.9 mg/kg (maximum dose 90 mg) over 60 minutes: give 10% of the dose as a bolus over 1 minute.

2. Admit the patient to an intensive care or stroke unit for monitoring.

3. If the patient develops severe headache, hypertension, nausea, vomiting, or neurologic deterioration, discontinue the rtPA infusion and obtain an emergent CT scan.

4. Measure blood pressure and perform neurological assessments every 15 minutes during and after IV rtPA infusion for 2 hours, then every 30 minutes for 6 hours, then hourly until 24 hours after rtPA treatment.

5. Increase the frequency of blood pressure measurements if systolic blood pressure is >180 mm Hg or if diastolic blood pressure is >105 mm Hg; administer antihypertensive medications as described in Table 7 to maintain blood pressure.

6. Do not place nasogastric tubes, indwelling bladder catheters, or intra-arterial pressure catheters if the patient can be safely managed without them.

7. Obtain a follow-up CT or MRI scan at 24 hours after IV rtPA before starting anticoagulants or antiplatelet agents.

Thrombolysis is an effective treatment for ischemic stroke but it has significant limitations. It can only be given within 3 - 4.5 hours after the onset of symptoms and many patients with ischemic stroke...
present to the hospital well outside of that time frame. Thrombolysis has serious adverse effects; although it has what could be described as a good success rate, there are certainly people who do not respond. Additionally, occlusion of certain cerebral vessels cannot be cleared with rtPA.\textsuperscript{144}

Endovascular therapies such as stenting, intra-arterial fibrinolysis, mechanical thrombectomy, clot aspiration can be used alone for patients who are not eligible for rtPA treatment or used with rtPA if the patient is likely to respond poorly to rtPA, \textit{i.e.}, if the patient has a severe stroke caused by a large occlusion and several clots.\textsuperscript{144-46} At this point the evidence for the effectiveness of these techniques has been described as equivocal,\textsuperscript{147} and there are no trials that directly compare them.\textsuperscript{120}

**Other Aspects of Stroke Care**

Other aspects of stroke care are outlined below according to the recommendations in the 2013 AHA/ASS guidelines.\textsuperscript{120}

**Anticoagulation**

Urgent anticoagulation with heparin or a low molecular weight heparin does not decrease the risk of early neurological deterioration or recurrent stroke is not recommended.

**Platelet Inhibition**

Administering aspirin 24-48 hours after the onset of the stroke is recommended.
**Statins**

If the patient was taking a statin it is reasonable to continue use of the drug.

**Induced Hypothermia**

There is no evidence that induced hypothermia is effective.

**Carotid Endarterectomy**

The use of carotid endarterectomy is not well established.

**Stroke Centers**

The use of specialized stroke centers is recommended.

**Antibiotics**

Prophylactic antibiotics are not recommended. Pneumonia is a serious complication occurring in the first 48 to 72 hours after acute ischemic stroke and accounts for approximately 15% to 25% of deaths associated with stroke. Stroke-associated pneumonia increases length of stay, mortality, and hospital costs. The most common cause of pneumonia is aspiration due to dysphagia.

Urinary tract infections (UTIs) are also common, occurring in approximately 15% to 60% of stroke patients, and independently predict poor outcome. If the patient has pneumonia or a UTI she/he should be treated with the appropriate antibiotics.
**DVT Prevention**

Subcutaneous heparin is recommended to prevent DVT in patients who are immobilized. Aspirin can be used if the use of subcutaneous heparin is contraindicated. External compression devices are another alternative.

**Swallowing Assessment**

A swallowing assessment should be done before liquids, solids, or oral medications are allowed. Nasogastric tubes, PEG tubes, or other devices can be used to provide hydration, medications and nutrition if needed.

**Mobilization**

Early mobilization is recommended. Early mobilization reduces risk of atelectasis, pneumonia, DVT, and pulmonary embolism. Complications from immobility have been found to account for up to 51% of deaths in the first 30 days after ischemic stroke. Immobility can also lead to contractures, orthopedic complications, atrophy, and nerve pressure palsies.

**Indwelling Urinary Catheter**

The most common urinary complication is incontinence, which occurs 30% to 60% of the time in the early recovery period. An indwelling urinary catheter should not be used because of the risk for urinary tract infection.

**Stroke Prevention Measures**

Stroke prevention measures should be started as soon as possible.
Treatment Of Hemorrhagic Stroke

Many of the treatment concern and goals for hemorrhagic stroke are identical to those of ischemic stroke, i.e., blood glucose, body temperature, and fluid status must be closely monitored, the patient closely observed for complications such as aspiration, DVT, pneumonia, and UTI. Patients should be admitted to the ICU and have continuous hemodynamic and neurologic monitoring. Specific concerns in the management of these patients includes:

- **DVT prevention:**
  Intermittent pneumatic compression should be used to prevent DVT.\(^{148}\)

- **IV hydration:**
  Normal saline should be used for IV hydration. Isotonic fluids should not be used as they can worsen cerebral edema and elevate intracranial pressure. Hypervolemia should be avoided as it can worsen cerebral edema.\(^{148}\)

- **Blood pressure control:**
  Hypertension is common in patients who have had a hemorrhagic stroke,\(^{149}\) and an elevated blood pressure is associated with a poor outcome.\(^{150}\) A 2013 study found that intensive lowering of blood pressure in patients who had a hemorrhagic stroke did not change rates of mortality or severe disability but did significantly improve functional outcomes, and was not associated with an increase in death or serious adverse effects.\(^{151}\) A 2014 review of the treatment of hemorrhagic stroke provided these guidelines for blood pressure management.\(^{149}\)
**Table 15: Blood Pressure Management in Patients with Hemorrhagic Stroke**

<table>
<thead>
<tr>
<th>Blood Pressure Parameters</th>
<th>Management Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP &gt;200 mmHg or MAP &gt;150 mmHg</td>
<td>Consider aggressive reduction of blood pressure</td>
</tr>
<tr>
<td>Systolic blood pressure &gt;180 mmHg or MAP &gt;130 mmHg and evidence or suspicion of elevated ICP</td>
<td>Consider lowering blood pressure to maintain the cerebral perfusion pressure between 61 to 80 mmHg</td>
</tr>
<tr>
<td>Systolic BP &gt;180 mmHg or MAP &gt;130 mmHg but no evidence or suspicion of elevated ICP</td>
<td>Use intermittent or continuous IV infusion of an antihypertensive to lower the MAP to 110 mm HG</td>
</tr>
</tbody>
</table>

- **Intracranial pressure:**
  Increased intracranial pressure (ICP) caused by intracranial hemorrhage can cause further brain injury. In order to avoid an increase in ICP the head of the bed should be elevated 30 degrees and the patient should receive appropriate analgesia and sedation. Glucocorticoids such as dexamethasone should not be used to lower ICP.
Invasive ICP monitoring allows direct measurement of ICP and it appears to be more sensitive to changes in ICP and more effective at preventing complications, i.e., enlargement of a hematoma, than neurological examination and brain scanning.\textsuperscript{152} If ICP needs to be lowered IV infusions of mannitol,\textsuperscript{56,148,153} barbiturate coma,\textsuperscript{56,148} hyperventilation,\textsuperscript{56,148} ventriculostomy,\textsuperscript{56,148,154} and surgical evacuation of the hematoma\textsuperscript{154} are considered reasonable options.

- Seizures:
  Seizures, both clinical (2.7%-17%) and noted on electroencephalography monitoring (28%-31%), are common after intracranial hemorrhage.\textsuperscript{154,155} There is no clear evidence about how seizures affect patient outcome, whether seizures should be treated, or what medications should use to treat them.\textsuperscript{154} A 2014 review noted that “... clinically significant seizures should be treated with anti-convulsants . . .”\textsuperscript{154} but prophylaxis with anti-convulsants, especially phenytoin, is not recommended.\textsuperscript{155}

- Patients receiving anti-coagulants:
  Warfarin should be discontinued if the INR is elevated, IV vitamin K should be administered, and specific vitamin K-dependent factors should be given.\textsuperscript{155} If there is severe bleeding vitamin K, fresh frozen plasma, and prothrombin complex concentrates and recombinant factor VIIa can be given.\textsuperscript{155-158}
Treatment Of Subarachnoid Hemorrhage

Many of the treatment concern and goals for subarachnoid hemorrhagic are identical to those of ischemic stroke, i.e., blood glucose, body temperature, and fluid status must be closely monitored, the patient closely observed for complications such as aspiration, DVT, pneumonia, and UTI.\textsuperscript{130,159,160} Patients should be admitted to the ICU and have continuous hemodynamic and neurologic monitoring. Specific concerns in the management of these patients includes the following conditions.

- **Hyponatremia:**
  Hyponatremia occurs is approximately one-third of all patients who have had a subarachnoid hemorrhage and it is associated with poor outcome.\textsuperscript{160} Hyponatremia is treated by restricting free water consumptions and 1.5% or 2% sodium chloride solutions.\textsuperscript{160} Fludrocortisone or hydrocortisone may help promote diuresis and correct the hyponatremia.\textsuperscript{160}

- **Re-bleeding:**
  Re-bleeding is managed by using anti-fibrinolytic drugs such as tranexamic acid, aminocaproic acid, or surgical clipping and endovascular coiling.\textsuperscript{159,160}

- **Hydrocephalus:**
  Hydrocephalus is defined as an excess of fluid in the ventricles of the brain and it is a relatively common complication of subarachnoid hemorrhage. Symptomatic hydrocephalus is treated making a burr hole in the skull and placing a ventricular drain.\textsuperscript{159,160}
• Seizures:
  Prophylactic anticonvulsants should be considered if the patient has a large hemorrhage that has not yet been surgically corrected.\textsuperscript{159}

• Vasospasm and delayed cerebral ischemia:
  Some patients who have had a subarachnoid hemorrhage will have neurologic deterioration after the initial event. This neurologic deterioration is thought to be caused by arterial vasospasm that reduces cerebral blood flow and in the setting of a subarachnoid hemorrhage it is called delayed cerebral ischemia (DCI).\textsuperscript{160}

  Delayed cerebral ischemia is defined as neurologic deterioration that is not due to other causes or a new infarction that is documented by CT scan more than 72 hours after the hemorrhage.\textsuperscript{160} Prophylactic treatment of DCI includes nimodipine (Nimotop®, a calcium channel blocker), maintaining euvolemia, and lumbar drainage.\textsuperscript{160} If the patient is symptomatic the treatments include induced hypertension, inotropic drugs such as dobutamine and milrinone, transfusions to maintain a hemoglobin level between 8-10 mg/dL, angioplasty, and intra-arterial dilators such as milrinone, nimodipine, nicardipine, and verapamil.\textsuperscript{160}

• Blood pressure management:
  The best treatment for hypertension in a patient who has a subarachnoid hemorrhage is not known. Singer et al., (2013) recommend keeping the systolic blood pressure below 160 mm Hg with enalapril, labetalol, or nicardipine.\textsuperscript{159}
SUMMARY

Transient ischemic attack and stroke are very common neurologic emergencies. A TIA by definition does not cause serious impairment but it is a significant neurological event. Strokes, caused by ischemia or hemorrhage, can be devastating and cause permanent and severe disabilities. Transient ischemic attack and ischemic stroke are caused by thrombus, emboli, or hypoperfusion. Hemorrhagic stroke is caused by rupture of small cerebral arteries, and bleeding into the subarachnoid space causes subarachnoid hemorrhage. Advanced age, ethnic background, and family history are some of the non-modifiable risk factors for TIA and stroke; atrial fibrillation, cigarette smoking, and hypertension are some of the common modifiable risk factors.

Treatment of TIA is primarily supportive, but the patient should receive anti-coagulation and anti-platelet therapy to prevent future TIAs or a stroke. Treatment of an ischemic stroke primarily focuses on clot dissolution using rtPA. Treatment of hemorrhagic stroke focuses primarily on monitoring ICP and preventing cerebral edema, while treatment of subarachnoid hemorrhage focuses on monitoring for elevations in ICP and for the development of DCI. In all of these, TIA, ischemic stroke, hemorrhagic stroke, and stroke caused by subarachnoid hemorrhage, it should be remembered the brain is exquisitely sensitive to lack of blood and oxygen and rapid assessment and treatment is essential to prevent permanent neurological damage.

Please take time to help the NURSECE4LESS.COM course planners evaluate nursing knowledge needs met following completion of this course by completing the self-assessment Knowledge Questions after reading the article. Correct Answers, 69 page.
1. **A transient ischemic attack:**
   a. causes temporary neurological dysfunction.
   b. is typically caused by a coagulation disorder.
   c. causes permanent neurological dysfunction.
   d. is very common in children and young adults.

2. **A transient ischemic attack:**
   a. is caused by bleeding into the subarachnoid space.
   b. is considered to be a significant risk factor for stroke.
   c. is characterized by persistent neurological deficits.
   d. does not require the use of neuroimaging.

3. **Treatment priorities for transient ischemic attack include:**
   a. the use of rtPA and ICP monitoring.
   b. withdrawal of anticoagulants and antihypertensives.
   c. induced hypothermia and maintaining euvoema.
   d. anticoagulation and antiplatelet therapy.

4. **An ischemic stroke is characterized by:**
   a. bleeding into the subarachnoid space.
   b. temporary neurological dysfunction caused by a thrombus or embolism.
   c. cerebral infarction caused by decreased blood flow.
   d. rupture of small arteries and bleeding into the brain parenchyma.

5. **Treatment priorities for ischemic stroke include**
   a. the use of rtPA and blood pressure management.
   b. ICP monitoring and prophylactic antibiotics.
   c. surgical evacuation of the hematoma and therapeutic hyperglycemia.
   d. monitoring for DCI and re-bleeding
6. A hemorrhagic stroke is characterized by:
   a. decreased blood flow caused by a thrombus or embolism.
   b. rupture of small arteries and bleeding into the brain parenchyma.
   c. bleeding into the subarachnoid space.
   d. hypoperfusion caused by a coagulopathy.

7. Treatment priorities for hemorrhagic stroke include:
   a. administration of rtPA and emergent anti-coagulation.
   b. therapeutic hypothermia and avoiding hyperglycemia.
   c. prophylactic anti-convulsants and prophylactic antibiotics.
   d. monitoring ICP and blood pressure control.

8. Subarachnoid hemorrhage is characterized by:
   a. temporary neurologic dysfunction.
   b. cerebral ischemia that causes an infarct.
   c. bleeding into the subarachnoid space.
   d. hypercoaguability that causes decreed brain perfusion.

9. Treatment priorities for subarachnoid hemorrhage include:
   a. monitoring for rebleeding and DCI.
   b. administration of rtPA and antiplatelet therapy.
   c. the use of hypertonic glucose solutions and factor Xa inhibitors.
   d. anticoagulation with warfarin and carotid endarterectomy.

10. Transient ischemic attacks and strokes are more likely to occur in patients who:
    a. are female, < age 45, and have a normal lipid profile.
    b. are elderly and who have atrial fibrillation and/or hypertension.
    c. are male, < age 50, and have insulin dependent diabetes.
    d. do not smoke, have a normal BNP, and are black.
Correct Answers
1. A
2. B
3. D
4. C
5. A
6. B
7. D
8. C
9. A
10. B

Footnotes:


http://www.uptodate.com/contents/spontaneous-intracerebral-hemorrhage-treatment-and-prognosis?source=search_result&search=spontaneous+intracerebral+hemorrhage&selectedTitle=1%7E150


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