Basic Neuroanatomy and Stroke Syndromes

Joseph R. Pare, MD[a], Joseph H. Kahn, MD[b,*]

KEYWORDS

• Stroke • Stroke syndromes • Neuroanatomy and stroke
• Pathophysiology and stroke

KEY POINTS

- Middle cerebral artery occlusion in dominant hemisphere causes aphasia and contralateral motor and sensory deficits.
- Vertebrobasilar occlusion may present with dizziness and ataxia, then progress to quadriplegia and coma.
- Carotid artery dissection, a cause of stroke in young people, may present with Horner syndrome.
- Cerebral venous thrombosis may present as headache, seizure, or coma.

INTRODUCTION TO STROKE

Acute stroke care is a cornerstone of emergency medicine practice owing to its significant morbidity and mortality. There are an estimated 795,000 strokes annually and 6.4 million American stroke survivors.[1] Stroke is the third leading cause of death nationally, and accounts for a calculated 134,000 deaths annu\ally.[1]

Of those people affected, it is helpful to be aware of the significant disparities within the field of stroke. Black and Hispanic/Latino Americans have a higher incidence of all types of stroke when compared with Caucasians. Studies report stroke incidence is nearly twice as high for blacks as compared with whites.[2] Additionally, stroke should not solely be considered a disease of the elderly, and again racial disparities are most evident among young adults.[2] Acute stroke can present at any age and it is important to be familiar with the evaluation and treatment of stroke to provide timely care.[3] The National Institute of Health Stroke Scale (NIHSS) helps physicians objectively evaluate stroke patients (**Table 1**). This article presents an overview of basic information on neuroanatomy, pathophysiology, and stroke syndromes.

[a] Department of Emergency Medicine, Boston Medical Center, 1 Boston Medical Center Place, Boston, MA 02118, USA; [b] Medical Student Education, Department of Emergency Medicine, Boston Medical Center, Boston University School of Medicine, 1 Boston Medical Center Place, Boston, MA 02118, USA
* Corresponding author.
E-mail address: jkahn@bu.edu

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Table 1
National Institutes of Health stroke scale score

1a. Level of consciousness	0 = Alert; keenly responsive 1 = Not alert, but arousable by minor stimulation 2 = Not alert; requires repeated stimulation 3 = Unresponsive or responds only with reflex
1b. Level of consciousness questions: What is the month? What is your age?	0 = Both answers correct 1 = Answers 1 question correctly 2 = Answers 2 questions correctly
1c. Level of consciousness commands: Open and close your eyes Grip and release your hand	0 = Performs both tasks correctly 1 = Performs 1 task correctly 2 = Performs neither task correctly
2. Best gaze	0 = Normal 1 = Partial gaze palsy 2 = Forced deviation
3. Visual	0 = No visual loss 1 = Partial hemianopia 2 = Complete hemianopia 3 = Bilateral hemianopia
4. Facial palsy	0 = Normal symmetric movements 1 = Minor paralysis 2 = Partial paralysis 3 = Complete paralysis of 1 or both sides
5. Motor arm 5a. Left arm 5b. Right arm	0 = No drift 1 = Drift 2 = Some effort against gravity 3 = No effort against gravity; limb falls 4 = No movement
6. Motor leg 6a. Left leg 6b. Right leg	0 = No drift 1 = Drift 2 = Some effort against gravity 3 = No effort against gravity 4 = No movement
7. Limb ataxia	0 = Absent 1 = Present in 1 limb 2 = Present in 2 limbs
8. Sensory	0 = Normal; no sensory loss 1 = Mild-to-moderate sensory loss 2 = Severe to total sensory loss
9. Best language	0 = No aphasia; normal 1 = Mild to moderate aphasia 2 = Severe aphasia 3 = Mute, global aphasia
10. Dysarthria	0 = Normal 1 = Mild to moderate dysarthria 2 = Severe dysarthria
11. Extinction and inattention	0 = No abnormality 1 = Visual, tactile, auditory, spatial, or personal inattention 2 = Profound hemi-inattention or extinction
Total score = 0–42	

Adapted from National Institutes of Health. National Institute of Neurological Disorders and Stroke. Available at http://www.ninds.nih.gov/disorders/stroke/strokescales.htm.

Quality Measures and Goals of Care

The American Heart Association (AHA) launched a large campaign to decrease stroke mortality by 25% from 1996 to 2006. In 2006, mortality at the 10-year goal had fallen by 18.4%, and in 2008 the 25.0% reduction goal was met.[1] Key recommendations for stroke centers have been made by the AHA to aid physicians in attaining these goals. This includes specially trained professionals with availability of services, such as surgical and endovascular procedures and intensive care units. Recommendations also include having computed tomography (CT), magnetic resonance imaging (MRI), and angiography available, as it is important for providing accurate and swift diagnoses to expedite care. It is the AHA's opinion that it is critical that a stroke service exist within a stroke center. The AHA recommends that this service work to improve physician collaboration, institute organized standardized care, and evaluate performance measures, all with the goal of improving the delivery of care and patient outcomes.[4,5]

Terminology

Acute stroke can be classified as either hemorrhagic or ischemic. Hemorrhagic strokes are those that result in disruption of the luminal integrity of the blood vessel. Factors that may result in hemorrhage include but are not limited to hypertension, vascular malformations, trauma, and neoplasm.

Ischemic strokes can then be labeled as either a transient ischemic attack (TIA) or an acute ischemic stroke (AIS). The definitions of these terms have been updated because of the historic inconsistency of their use within the medical field. The AHA/ACC (American College of Cardiology) published a scientific statement in 2009 defining a TIA as "a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction."[6] Previous terminology had defined TIA as any stroke syndrome with resolution of symptoms within 24 hours. Since that time, research has shown that despite resolution of symptoms, in 30% to 50% of patients, there is neuronal death seen on diffusion-weighted MRI.[6] The newer definition is aimed at clarifying between neuronal death and dysfunction. As a convention, stroke now implies neuronal death, whereas TIA refers to neuronal dysfunction that has not resulted in tissue loss owing to ischemia. TIA and stroke are generally a result of luminal obstruction leading to insufficient blood flow.

NEUROANATOMY

For this article's purpose, the brain is divided into 4 areas to help define structure and function. This article focuses on the cerebrum, diencephalon, cerebellum, and brain stem. These 4 structures are critical to brain function and are important to understand when correlating a clinical evaluation to corresponding neurologic imaging.

Cerebrum

The cerebrum can be divided into 3 structures for simplicity: cortex, basal ganglia, and limbic system. The cerebral cortex is generally divided into the 4 standard lobes of the brain: frontal, parietal, occipital, and temporal. The frontal lobe is located at the anterior portion of the brain and controls skeletal muscle movement (the motor cortex) and behavioral expression. The parietal lobe is posterior to the frontal lobe and houses the sensory cortex and some optic radiations. Optic radiations carry sensory input from the eyes for visual interpretation, and damage in this area can be clinically correlated by the loss of superior contralateral vision. The temporal lobe is associated with smell and hearing. Damage to optic radiations that pass through this lobe can result in loss of inferior contralateral vision. Optic radiations travel to the occipital lobe, and

therefore this lobe is primarily responsible for vision. Any damage to the cerebral cortex may result in clinical presentations with corresponding loss to these anatomic functions.[7]

The basal ganglia primarily coordinate movement and are composed of the caudate, putamen, and globus pallidus. The limbic system is composed of the amygdala, cingulate gyrus, and the hippocampus. The amygdala and cingulate gyrus are important in memory and emotion, whereas the hippocampus is responsible for memory and learning. These structures are located internal to the cerebral cortex and closer to the core of the brain.[7]

Diencephalon

This portion of the brain is the area between the brain stem and the cerebrum. This portion is composed of the thalamus, hypothalamus, and pituitary and pineal glands. The thalamus is critical to sensory function, as almost all sensory information passes through this portion of the brain before being directed to the cerebrum. The thalamus receives input from structures such as the basal ganglia, limbic system, and the cerebellum. The thalamus also receives input from the cerebral cortex so that feedback can be relayed. As such, the thalamus is connected to all the major areas of the brain.[7]

The hypothalamus controls important functions to everyday life, such as modulating hunger and thirst, including certain autonomic and endocrine functions. The pineal and pituitary glands are important endocrine structures that modulate many hormones in addition to helping with the sleep-wake cycle.[7]

Cerebellum

The cerebellum is located adjacent to the brain stem. This portion of the brain is primarily responsible for aiding motor function. The cerebellum does this by coordinating rapid alternating movements, balance, and position sense. Disease of the cerebellum may present with dysfunction of speech, tremor, or ocular findings, such as nystagmus.[7] Cerebellar disease may also be found with examination findings of abnormal gait, or abnormal findings on finger-to-nose or heel-shin testing.

Brain Stem

The brain stem is generally categorized into 3 structural components: medulla, pons, and midbrain. The brain stem is the most primitive portion of the brain, and is responsible for many critical neurologic functions. One example of this is the reticular formation that extends throughout the brain stem. This portion of the brain is important in controlling many critical body functions, such as breathing, blood pressure, and alertness. The brain stem is also where nearly all of the cranial nerves arise and are listed in the following sections based on anatomic location.[7]

Medulla

The medulla is the most caudal portion of the brain stem and ends at the level of the foramen magnum where the spinal cord begins. Cranial nerves (CN) that arise from this portion of the brain include the hypoglossal (CN XII), glossopharyngeal (CN IX), vagus (CN X), and portions of the accessory nerve (CN XI). The medulla also contains important neurologic pathways. Somatosensory tracts pass through the medulla and are those nerve fibers that relay peripheral sensation to the brain for interpretation. In converse, the corticospinal tracts transmit information from the cerebellum to the spinal cord and also pass through the medulla. The pyramids are a ventral structure of the medulla where the corticospinal tracts traverse the midline, decussate, and then transmit impulses to the opposite side of the body. It is the process of

decussating, or crossing the midline, that results in the right or left side of the brain being responsible for sensation or movement on the opposite side of the body.[7]

Pons

The pons is located between the medulla and the midbrain. The abducens (CN VI), facial (CN VII), vestibulocochlear (CN VIII), and trigeminal nerves (CN V) are located at the level of the pons. This portion of the brain acts as a message center between the cerebellum and the cerebrum.[7]

Midbrain

The final portion of the brain stem is the midbrain. This is the most cephalic portion of the brain stem and acts primarily in coordination of eye movement in addition to reflexes associated with hearing and vision. As such, the optic (CN II), oculomotor (CN III), and trochlear nerves (CN IV) arise here.[7]

VASCULAR NEUROANATOMY

The brain requires about 20% of the body's oxygen supply and approximately 15% of the cardiac output. The circulation of the brain can be divided into anterior and posterior components. The carotid arteries give rise to the anterior circulation, and the vertebral arteries comprise the posterior circulation (**Fig. 1**). Together the anterior and posterior circulations merge to form the Circle of Willis. The Circle of Willis is a circular ring where several major blood vessels arise to provide cerebral blood flow.

Anterior Circulation

The left and right carotid arteries supply the anterior circulation of the brain. The trunk of the internal carotid travels to the Circle of Willis and divides into the middle and anterior cerebral arteries. The middle cerebral artery (MCA) supplies blood to the parietal, occipital, and temporal lobes, as well as a small portion of the frontal lobe. The lenticulostriate arteries also arise from the MCA, supply the internal capsule and basal ganglia, and are known for their nature of progressive arteriosclerosis leading to stroke. The anterior cerebral artery supplies a small area that is localized to the medial portion of the frontal and parietal lobes.[8]

Posterior Circulation

The left and right vertebral arteries primarily supply the posterior circulation of the brain; however, the anterior spinal artery also vascularizes a small segment of the brain stem. Portions of the vertebral arteries are directly responsible for feeding the most caudal portions of the brain stem. At the level where the medulla meets the pons, the vertebral arteries converge to form the basilar artery. The basilar artery provides

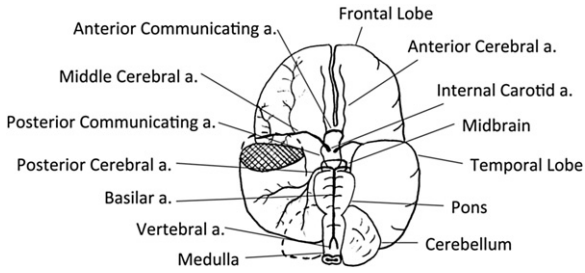

Fig. 1. Basic neuroanantomy.

circulation to the remainder of the brain stem in addition to the cerebellum. It is important to note several arteries that arise from the posterior circulation at the level of the basilar artery that supply the cerebellum, as obstruction of these arteries leads to specific clinical syndromes. Most superiorly is the superior cerebellar artery, then below is the anterior inferior cerebellar artery (AICA), and finally near the level where the vertebral arteries give rise to the basilar artery is the posterior inferior cerebellar artery (PICA). The basilar artery terminates by dividing into the left and right posterior cerebral arteries that comprise the posterior portion of the Circle of Willis and allow for communication with the anterior blood supply (**Fig. 2**).[8]

Venous Circulation

The venous circulation generally does not get as much attention as the arterial system because cerebral venous thrombosis is an uncommon form of stroke. It is important to be familiar with the venous drainage system, however, as occlusion can lead to acute stroke symptoms and significant morbidity and mortality. The superior sagittal sinus, straight sinus, and transverse sinus converge at the confluence of sinuses near the occiput. The transverse sinus communicates with the sigmoid sinus, allowing for venous drainage into the internal jugular vein.[9] Cerebral venous thrombosis is discussed later in this article, as are the common risk factors and locations for venous thrombosis.

PATHOPHYSIOLOGY

As was discussed when covering the difference between stroke types, most strokes are classified as either ischemic or hemorrhagic. The most common type of stroke is ischemic thrombotic stroke; however, low partial pressures of oxygen, hypotension, and impaired oxygen use are all additional physiologic states that can lead to ischemia and neuronal death or dysfunction. Some additional forms of stroke occur as a result of a combination of ischemia and hemorrhage.

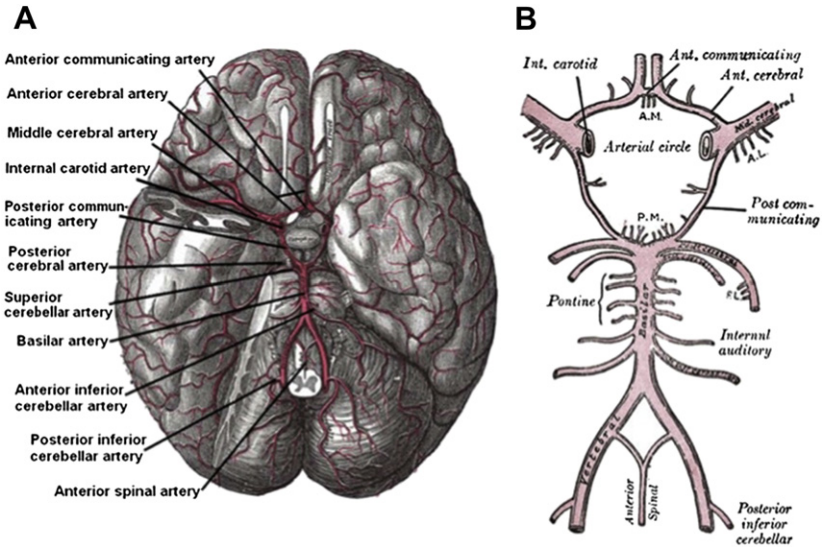

Fig. 2. Circle of Willis. (*A*) Cerebral vasculature. (*B*) Diagram of cerebral vasculature. (*Reprinted from* Gray H. Anatomy of the human body. 20th edition. Philadelphia: Lea & Febinger; 1918.)

Large Vessel Stroke

Large cerebral vessels predominantly become occluded as a result of either thrombus or embolus. Sites of localized thrombus formation within a vessel generally occur because of the nature of blood flow and anatomy. Most frequently, thrombus can form at the bifurcation of the common carotid artery where it transitions into the internal and external carotid arteries. Additional common sites for thrombus formation include the origin of the MCA, the convergence of the vertebral arteries to become the basilar artery, and the division of the basilar into the posterior portion of the Circle of Willis. All of these sites are prone to the development of arteriosclerosis because of narrowing and changes in the angles of the arising vessels.[10]

Embolisms are frequently caused by cardiac thrombi, which can be seen as a result of wall motion abnormalities after myocardial infarction, valvular disease, or irregular heart rhythms, such as atrial fibrillation. Other sources of emboli can be carotid plaques, venous thrombi that pass through structural cardiac defects, or emboli caused by fat, air, or tumor.[10]

Small Vessel Stroke

Although small vessels can also become occluded owing to the same mechanisms that generally lead to large vessel disease, smaller vessels tend to have other mechanisms for infarction. This disease is generally a result of occlusion caused by arteriosclerosis, but can also be caused by pathology, such as vasculitis. Vasculitis is frequently a result of infection, and the types of infection have changed over time. Immunosuppression has led to opportunistic infections as a more common occurrence. It is important to note that autoimmune forms of vasculitis and cerebral amyloid angiopathy also result in small vessel ischemic strokes.[10]

Hemorrhagic Stroke

Intraparenchymal hemorrhage

Spontaneous intraparenchymal hemorrhages generally present in mid to late life with peak incidence around 60 years of age.[10] Most bleeds are attributable to small vessels as a result of long-standing hypertension. Other etiologies include coagulation disorders, trauma, neoplasms, amyloid angiopathy, vasculitis, vascular malformations, and fusiform aneurysms. Bleeds that occur in the basal ganglia and thalamus are termed deep hemorrhages, whereas bleeds within the cerebral hemispheres are termed lobar hemorrhages. Bleeds within the putamen are the most common, representing 50% to 60% of intraparenchymal hemorrhages.[10]

Aneurysmal subarachnoid hemorrhages

Subarachnoid hemorrhage (SAH) accounts for approximately 5% of all stroke and is estimated to affect about 30,000 Americans annually.[11] SAH is an important disease owing to the significant mortality. In some populations, mortality rates have been documented as high as 45%, and there is significant morbidity among survivors. The most frequent cause of SAH is rupture of a saccular (berry) aneurysm. Saccular aneurysms represent the most common intracranial aneurysms.[10] The etiology of saccular aneurysms is unknown.

Within the United States, risk factors for the development of aneurysmal SAH include hypertension, smoking, and heavy alcohol abuse. Cocaine and phenylpropanolamine, both sympathomimetic agents, have been implicated as risk factors for the development of SAH. Smoking, hypertension, family history of cerebrovascular disease, and female gender (especially postmenopausal) have also been associated with the development of multiple aneurysms.[11] Several genetic syndromes, including

autosomal dominant polycystic kidney disease, neurofibromatosis type 1, Marfan syndrome, and type IV Ehlers-Danlos syndrome, have been linked to aneurysm formation and subsequent rupture.[10]

Vascular Malformations

Vascular malformations significantly increase the risk for stokes. Arteriovenous malformations are the most common form of vascular malformation resulting in hemorrhage. This congenital disease is generally found between the ages of 10 and 30, with seizure or hemorrhage as the presenting symptom. These types of malformations generally arise within the subarachnoid space and are most commonly found within the posterior portions of the MCA territory. Other malformations leading to stroke include cavernous angiomas, capillary telangiectasias, and venous angiomas.[10]

Cerebral Venous Thrombosis

Cerebral venous thrombosis (CVT) is an uncommon and potentially difficult disease to diagnose. CVT has been shown to affect all age ranges and has many possible underlying risk factors. CVT is estimated to account for only 0.5% to 1.0% of all strokes.[12]

There are 2 basic mechanisms that lead to clinical findings suggestive of CVT: (1) patients who have elevated intracranial pressure (ICP) as a result of impaired venous flow and/or (2) those with focal disease from venous ischemia or hemorrhage.[12] Common signs and symptoms of CVT may include headache, papilledema, isolated elevated ICP, focal neurologic deficit, seizure, and/or encephalopathy. Other rare clinical symptoms have also been reported.[12]

Much of the data on CVT comes from a single study of 624 patients. The minimum age for enrollment was 15 years, and the mean age of the affected population was 39, which highlights the degree to which CVT affects younger patients.[13] Predisposing conditions for CVT, in order of prevalence, include but are not limited to oral contraceptives (54.3%), prothrombotic conditions (34.1%), pregnancy/puerperium (21%), and infection (12.3%).[9] There have also been specific anatomic locations that have increased incidence of disease. The most frequent locations are the superior sagittal sinus (62%), transverse sinus (41%–45%), straight sinus (18%), and cortical veins (17%).[9] The American Stroke Association (ASA), in conjunction with the AHA, has published guidelines to aid clinicians in the diagnosis and management of CVT.[9]

STROKE SYNDROMES

AIS results from loss of blood supply to a region of the brain. Occlusion of a vessel can be secondary to a thrombotic or embolic event. Rapid recognition of stroke has become of paramount importance with the increased acceptance and availability of intravenous thrombolysis for the treatment of stroke.

Embolic strokes are most commonly caused by embolization of mural thrombus (25% of ischemic strokes) secondary to myocardial infarction, atrial fibrillation, or mitral valve disease, but may also occur when clot forms at the site of an ulcerated plaque in the internal carotid artery or ascending aorta and then embolizes.[9,13] Other cardiac abnormalities predisposing to embolic strokes include patent foramen ovale, atrial septal defects, and atrial septal aneurysm.[9] Embolic events usually cause maximum deficit at onset, and frequently occur while the patient is awake.[9]

Thrombotic strokes generally occur at the site of an ulcerated plaque within a vessel. Thrombotic strokes tend to gradually reach their maximum deficit over a period of hours or days. Thrombotic strokes often occur during sleep, and the patient or family notices the deficits when the patient wakes up in the morning.[9] Risk factors for thrombotic

strokes include atherosclerosis; acquired hypercoagulable states, such as pregnancy, infection, or surgery; and congenital hypercoagulable states, such as protein C and S deficiencies, antithrombin III deficiency, sickle cell disease, factor V Leiden, lupus anticoagulant, and antiphospholipid antibodies.[9,14] Sleep-disordered breathing, such as obstructive sleep apnea, may also be a risk factor for thrombotic stroke.[15,16]

Stroke can follow occlusion of a large vessel, such as the internal carotid artery, anterior cerebral artery, middle cerebral artery, posterior cerebral artery, vertebrobasilar artery, anterior inferior cerebellar artery, posterior inferior cerebellar artery, or superior cerebellar artery (**Table 2**). Stroke may also be caused by the occlusion of a small penetrating artery, referred to as a lacunar infarct.

Ischemic strokes can also be divided into occlusion of anterior or posterior circulation. Anterior circulation infarcts result in frontal, temporal, and/or parietal lobe deficits, whereas posterior circulation strokes affect the occipital lobes, cerebellum, and brainstem. Less commonly, stroke can be secondary to thrombosis of an intracranial venous sinus.

Although intracranial hemorrhage may have a different clinical presentation than ischemic stroke, there is significant overlap of the clinical features. Intracranial hemorrhage usually occurs during waking hours.[9] Intracranial hemorrhage can be classified as intraparenchymal, intraventricular, subarachnoid, subdural, or epidural, based on the location of the bleed. Because the treatment of acute ischemic stroke may include thrombolytics and antiplatelet agents, the identification of intracranial hemorrhage is paramount in differentiating between stroke types. The use of noncontrast head CT can generally exclude intraparenchymal hemorrhage, subdural hematomas, and epidural hematomas, but the exclusion of SAH may require a lumbar puncture if the

Table 2
Partial list of major stroke syndromes

Carotid	Aphasia (dominant hemisphere) or neglect (nondominant hemisphere) Contralateral homonymous hemianopsia Contralateral motor/sensory loss of face, arm, and leg Conjugate ipsilateral eye deviation
MCA	Aphasia (dominant hemisphere) or neglect (nondominant hemisphere) Contralateral homonymous hemianopsia Contralateral motor/sensory loss face/arm > leg
ACA	Apathy, abulia, disinhibition Conjugate eye deviation Contralateral motor/sensory loss leg > arm
PICA	Ipsilateral palatal weakness, Horner syndrome Wallenberg syndrome Ipsilateral limb ataxia Decreased pain/temperature contralateral body
AICA	Ipsilateral deafness Ipsilateral facial motor/sensory loss Ipsilateral limb ataxia Decreased pain/temperature contralateral body
Basilar	Altered consciousness Oculomotor difficulties, facial paresis Ataxia, quadriparesis

Abbreviations: ACA, anterior cerebral artery, AICA, anterior inferior cerebellar artery; MCA, middle cerebral artery; PICA, posterior inferior cerebellar artery.

Data from Goetz CG. Textbook of clinical neurology. 3rd edition. Philadelphia: Elsevier; 2007; and Goldstein JN, Greer DM. Rapid focused neurologic assessment in the emergency department and ICU. Emerg Med Clin North Am 2009;27(1):5.

noncontrast head CT is negative. CT scanning has a sensitivity of 91% to 98% for identifying SAH, and the sensitivity decreases with time.[14] A recent study suggests sensitivity of 100% (97%–100%) if CT scanning is performed within 6 hours of symptom onset.[17] The clinical presentation of SAH is usually different from that of an ischemic stroke. Classically, SAH presents as the sudden onset of a severe headache. The presence of SAH mandates a search for an aneurysm or arteriovenous malformation within the circulation of the brain. Risk factors for intracranial hemorrhage include hypertension, trauma, anticoagulant medications, coagulopathies, stimulant drugs (cocaine and amphetamines), amyloidosis, and brain tumors.[9] Identification and treatment of intracranial hemorrhage is discussed in detail in the article by Caceres and Goldstein elsewhere in this issue.

Carotid Artery Occlusion

Carotid artery occlusion presents with aphasia if the dominant hemisphere is involved and contralateral neglect if the nondominant hemisphere is affected. There is motor and sensory loss of the face, arm, and leg on the opposite side from the occlusion. The eyes will deviate toward the side of the occlusion, and there may be a visual field deficit on the side opposite from the occluded carotid artery.[18]

Middle Cerebral Artery Occlusion

Occlusion of the MCA will cause global aphasia, if the occlusion occurs in the dominant hemisphere, and contralateral hemispatial neglect, if it occurs in the nondominant hemisphere. Most right-handed people and 70% to 80% of left-handed people have their language centers in the left hemisphere.[18] There will be motor and sensory deficits of the side opposite to the occlusion, involving the face and arm, and to a lesser extent, the leg. There may be a homonymous hemianopia, and conjugate eye deviation toward the side of the lesion.[9]

The MCA divides into superior and inferior divisions. The superior division within the dominant hemisphere supplies the Broca area in the frontal lobe, ischemia of which is responsible for motor aphasia. With Broca aphasia, speech will be halting and poorly articulated.[9] The inferior division of the MCA supplies the Wernicke area in the temporal lobe, ischemia of which causes sensory aphasia. With Wernicke aphasia, speech is fluent but incorrect words and nonsense words may be present, making it difficult to understand the meaning that the speaker is trying to convey.[9]

Anterior Cerebral Artery Occlusion

Occlusion of the anterior cerebral artery will classically cause contralateral motor and sensory deficits, more prominent in the leg than the arm. The face and tongue are usually spared.[19] Lack of concern and disinhibition are frequently present. Incontinence may be present, as well as primitive frontal lobe reflexes (grasp and suck).[9]

Posterior Cerebral Artery Occlusion

Posterior cerebral artery infarcts most commonly occur secondary to embolization.[19] Posterior cerebral artery occlusion frequently presents with a contralateral visual field cut in the form of a homonymous hemianopia. Visual agnosia may be present, as well as disorders of reading, if the PCA occlusion is in the left (dominant) hemisphere.[19] Alexia (inability to read words and sentences) with or without agraphia (inability to write and spell) may be present.[19] Prosopagnosia, or the inability to recognize faces, may also occur with posterior cerebral artery occlusion. There is usually no paralysis. Sensory loss may be present or absent. Aphasia will not be present. Bilateral posterior cerebral artery occlusion may cause blindness.[9,20] Bilateral posterior cerebral artery

occlusions may also cause permanent amnesia, with the inability to form new memories.[19]

Superior Cerebellar Artery Occlusion

Superior cerebellar artery occlusion may classically present with ipsilateral limb dysmetria, ipsilateral Horner syndrome, contralateral loss of pain and temperature sensation, and contralateral weakness of the fourth cranial nerve.[19] This classic presentation is not commonly seen, however.[19]

Posterior Inferior Cerebellar Artery Occlusion

Posterior inferior cerebellar infarcts are the most frequently encountered cerebellar strokes.[19] PICA obstruction may lead to the lateral medullary syndrome (Wallenberg syndrome), consisting of decreased pain and temperature sensation of the ipsilateral face and contralateral trunk and extremities (because of damage to the spinothalamic tract). Other deficits caused by PICA occlusion include Horner syndrome (ipsilateral ptosis, miosis, and anhydrosis), dysphagia, ipsilateral limb ataxia, nystagmus, diplopia, and myoclonus of the ipsilateral palate. These patients are at risk for aspiration.[21]

Anterior Inferior Cerebellar Artery Occlusion

AICA occlusion may present as sudden deafness owing to ischemia of the inner ear causing cochlear dysfunction.[22] AICA occlusion classically presets with vertigo, vomiting, tinnitus, and dysarthria.[19] AICA stroke also causes ipsilateral facial weakness and ipsilateral limb ataxia. Owing to ischemia of the spinothalamic tract, contralateral pain and temperature sensation may be impaired.[18,23]

Vertebrobasilar Artery Occlusion

High-grade occlusion of the vertebrobasilar system causes loss of circulation to the cerebellum, brain stem, thalamus, and occipital lobe. The result is frequently death or major disability, such as coma, quadriplegia, ataxia, dysarthria, cranial nerve dysfunction, and visual deficits.[24] Rarely, basilar artery thrombosis may cause “locked-in syndrome,” in which the patient cannot move or speak but cognition remains intact.[19,24]

Lacunar Infarcts

A lacunar infarct results from occlusion of a deep penetrating artery. These small-vessel occlusions account for 25% of all ischemic strokes. Depending on the region supplied by the occluded penetrating artery, the neurologic findings vary. Lacunar infarcts may present as pure motor hemiparesis, involving the posterior limb of the internal capsule. This syndrome may present as paresis of unilateral face, arm, and leg without sensory findings. Another presentation is ataxic hemiparesis, involving the internal capsule and corona radiata. This syndrome generally will cause ataxia and weakness of one leg. Dysarthria/clumsy hand results from a lacunar infarct involving the pons or internal capsule. This syndrome consists of slurred speech and weakness of one arm. A lacunar infarct in the thalamus may cause a pure sensory stroke. This syndrome will usually cause diminished sensation of unilateral face, arm, and leg. A mixed sensorimotor stroke may result from a lacunar infarct in the thalamus and internal capsule.[9,25,26]

Border Zone Infarction Syndromes

Watershed strokes, or border zone infarction syndromes, generally follow periods during which systemic oxygen delivery is impaired, such as with hypoxia or hypotension. There

are a variety of different syndromes attributable to border zone infarction. Anterior border zone infarction syndrome may consist of aphasia and proximal arm weakness, or motor weakness in the lower leg.[9,19] Posterior border zone infarction may cause visual disturbances, such as lateral homonymous hemianopia.[19]

Cerebral Venous Thrombosis

An uncommon cause of cerebral infarction is thrombosis of one of the venous sinuses in the brain. CVT may present as headache, with or without cranial nerve palsies. More severely affected patients may present with seizures and/or coma. Papilledema is frequently present. The incidence of this entity is rising, as it is being recognized more frequently as a result of newer imaging modalities, including CT angiography (CTA) and MRI/MR angiography.[27] The 2011 AHA/ASA statement for physicians on the diagnosis and management of CVT discusses the benefits of CT and CT venogram (CTV) versus MRI and MR venogram (MRV). In summary, CT with CTV is the test of choice in the acute phase of thrombosis. It is noted that MRI and MRV are more sensitive after the acute phase of thrombosis, but the article discusses additional strengths and weakness of each test when used to diagnose CVT.[9]

DEMOGRAPHICS AND RISK FACTORS FOR ISCHEMIC STROKE

Stroke remains the third leading cause of death in the United States, causing 137,000 deaths annually.[28] Age older than 55 is considered a risk factor for stroke, with doubling of the risk for stroke with each decade over age 55.[28] The aging of the US population has contributed significantly to the growth in number of emergency department visits in the United States, with a 19% increase in overall visits from 1995 to 2005, yet the increase in visits by patients aged 65 to 74 was 34% over a similar period.[29] It is anticipated that the aging of the US population will lead to an increase in the number of strokes presenting to emergency departments in the coming years.[28] There is also literature to suggest that strokes are not uncommon in younger patients, and that 10% of all strokes occur in people younger than 50.[30,31] In fact, in a recent prospective study in Switzerland, 14% of strokes over a 6-year period occurred in patients younger than 46 years.[32] Other risk factors for stroke include diabetes, hypertension, hyperlipidemia, smoking, obesity, atrial fibrillation, recent myocardial infarction, sickle cell anemia, vasculitides, fibromuscular dysplasia, and family history.[30,33–36] Recent herpes zoster infection, especially herpes zoster ophthalmicus, may also be a risk factor for stroke.[37]

The epidemic of obesity, diabetes, and hypertension among young Americans may lead to an increase in the incidence of stroke in young people.[38] In addition to the traditional risk factors stated previously, additional risk factors for stroke in people younger than 50 include patent foramen ovale, atrial septal defect, dissection of carotid or vertebral arteries, hypercoaguability, and autoimmune disorders.[31,32] Oral contraceptives, pregnancy, mitral valve prolapse, homocystinuria, polycystic ovary syndrome, cigarette smoking, binge alcohol drinking, and cocaine and amphetamine use may also predispose to stroke in young adults.[9,39,40] In addition to acute ischemic stroke, cocaine and amphetamines may predispose young people to SAH and intracerebral hemorrhage.[9]

Another important cause of stroke in young adults is carotid artery dissection. Carotid artery dissection may be spontaneous or traumatic. Patients with carotid dissection may present with Horner syndrome owing to loss of sympathetic innervation within the carotid sheath. Patients may present with isolated neck pain, and trauma causing dissection may be minimal.[9]

In summary, stroke is a frequent and time-sensitive emergency department presentation. Rapid identification, imaging, neurologic consultation, and treatment of stroke

are essential. Knowledge of the basic neuroanatomy, clinical presentation, and CT findings of various stroke syndromes and hemorrhage aid the emergency physician in diagnosis and management. Application of the NIHSS score (**Table 1**) will allow the emergency physician, in consultation with a neurologist, to administer thrombolytic therapy when appropriate.[41]

REFERENCES

1. Goldstein LB, Bushnell CD, Adams RJ, et al. Guidelines for the primary prevention of stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2010;42:517–84.
2. Cruz-Flores S, Rabinstein A, Biller J, et al. Racial-ethnic disparities in stroke care: the American experience: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42(7):2091–116.
3. Roach ES, Golomb MR, Adams R, et al. Management of stroke in infants and children: a scientific statement from a special writing group of the American Heart Association Stroke Council and the Council on Cardiovascular Disease in the Young. Stroke 2008;39(9):2644–91.
4. Task Force Members, Schwamm LH, Pancioli A, Acker JE 3rd, et al. Recommendations for the establishment of stroke systems of care: recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems. Stroke 2005;36(3):690–703.
5. Alberts MJ, Latchaw RE, Selman WR, et al. Recommendations for comprehensive stroke centers: a consensus statement from the brain attack coalition. Stroke 2005;36(7):1597–616.
6. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease: the American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke 2009;40:2276–93.
7. Silverthorn D. Human physiology: an integrated approach. 4th edition. San Francisco (CA): Pearson/Benjamin Cummings; 2007.
8. Gilman S. Manter and Gatz's essentials of clinical neuroanatomy and neurophysiology. 9th edition. Philadelphia: F.A. Davis; 1996.
9. Saposnik G, Barinagarrementeria F, Brown RD, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42:1158–92.
10. Kumar V. Robbins and Cotran pathologic basis of disease. 7th edition. Philadelphia: Elsevier Saunders; 2005.
11. Bederson JB, Connolly ES, Batjer HH, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the stroke council, American Heart Association. Stroke 2009;40:994–1025.
12. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. Lancet Neurol 2007;6(2):162–70.
13. Ferro JM. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke 2004;35(3):664–70.

14. Marques MA, Murad FF, Ristow AV, et al. Acute carotid occlusion and stroke due to antiphospholipid antibody syndrome: case report and literature review. Int Angiol 2010;29(4):380–4.
15. Johnson KG, Johnson DC. Frequency of sleep apnea in stroke and TIA patients: a meta-analysis. J Clin Sleep Med 2010;6(2):131–7.
16. Ramar K, Surani S. The relationship between sleep disorders and stroke. Postgrad Med 2010;122(6):145–53.
17. Perry JJ, Stiell IG, Sivilotti MLA, et al. Sensitivity of computed tomography performed within six hours of onset of headache for diagnosis of subarachnoid haemorrhage: prospective cohort study. BMJ 2011;343:d4277.
18. Goldstein JN, Greer DM. Rapid focused neurological assessment in the emergency department and ICU. Emerg Med Clin North Am 2009;27(1):1–16, vii.
19. Bogousslavsky J, Caplan LR. Stroke syndromes. Cambridge (United Kingdom); New York: Cambridge University Press; 2001.
20. Helseth E. Posterior cerebral artery stroke. Medscape Reference. March 29, 2011.
21. Khedr EM, Abo-Elfetoh N. Therapeutic role of rTMS on recovery of dysphagia in patients with lateral medullary syndrome and brainstem infarction. J Neurol Neurosurg Psychiatr 2010;81(5):495–9.
22. Lee H, Sohn SI, Jung DK, et al. Sudden deafness and anterior inferior cerebellar artery infarction. Stroke 2002;33(12):2807–12.
23. Lee H, Cho YW. Auditory disturbance as a prodrome of anterior inferior cerebellar artery infarction. J Neurol Neurosurg Psychiatr 2003;74(12):1644–8.
24. Kaye V. Vertebrobasilar stroke. Medscape Reference. March 29, 2011.
25. Sacco S, Marini C, Totaro R, et al. A population-based study of the incidence and prognosis of lacunar stroke. Neurology 2006;66(9):1335–8.
26. Papamitsakis N. Lacunar syndromes. Medscape Reference. December 10, 2011.
27. Lipton RB, Silberstein SD, Dalessio DJ. Wolff's headache and other pain. New York; Oxford (United Kingdom): Oxford University Press; 2001.
28. Fonarow GC, Reeves MJ, Zhao X, et al. Age-related differences in characteristics, performance measures, treatment trends, and outcomes in patients with ischemic stroke. Circulation 2010;121(7):879–91.
29. Xu KT, Nelson BK, Berk S. The changing profile of patients who used emergency department services in the United States: 1996 to 2005. Ann Emerg Med 2009; 54(6):805.e1-7–10.e1-7.
30. De Silva R, Gamage R, Wewelwala C, et al. Young strokes in Sri Lanka: an unsolved problem. J Stroke Cerebrovasc Dis 2009;18(4):304–8.
31. Janssen AWM, de Leeuw FE, Janssen MCH. Risk factors for ischemic stroke and transient ischemic attack in patients under age 50. J Thromb Thrombolysis 2011; 31(1):85–91.
32. Arnold M, Halpern M, Meier N, et al. Age-dependent differences in demographics, risk factors, co-morbidity, etiology, management, and clinical outcome of acute ischemic stroke. J Neurol 2008;255(10):1503–7.
33. Marx JA, Hockberger RS, Walls RM, et al. Rosen's emergency medicine: concepts and clinical practice. Philadelphia: Mosby/Elsevier; 2010.
34. Rae-Grant A, Weiner HL. Weiner and Levitt's neurology. Philadelphia: Lippincott Williams & Wilkins; 2008.
35. Bosnar-Puretić M, Basić-Kes V, Jurasić MJ, et al. The association of obesity and cerebrovascular disease in young adults—a pilot study. Acta Clin Croat 2009;48(3):295–8.
36. Jood K, Ladenvall C, Rosengren A, et al. Family history in ischemic stroke before 70 years of age: the Sahlgrenska Academy Study on Ischemic Stroke. Stroke 2005;36(7):1383–7.

37. Kang JH, Ho JD, Chen YH, et al. Increased risk of stroke after a herpes zoster attack: a population-based follow-up study. Stroke 2009;40(11):3443–8.
38. Kitzman-Ulrich H, Wilson DK, St George SM, et al. The integration of a family systems approach for understanding youth obesity, physical activity, and dietary programs. Clin Child Fam Psychol Rev 2010;13(3):231–53.
39. Parlakgumus HA, Haydardedeoglu B. A review of cardiovascular complications of pregnancy. Ginekol Pol 2010;81(4):292–7.
40. de Groot PC, Dekkers OM, Romijn JA, et al. PCOS, coronary heart disease, stroke and the influence of obesity: a systematic review and meta-analysis. Hum Reprod Update 2011;17(4):495–500.
41. Akins PT, Delemos C, Wentworth D, et al. Can emergency department physicians safely and effectively initiate thrombolysis for acute ischemic stroke? Neurology 2000;55(12):1801–5.