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Atypical Presentations of Ischemic Cerebrovascular Disease: Part I

Stroke has evolved into brain attack. Now more than ever, the emergency physician must diagnose stroke rapidly and correctly. Although there remains some controversy over the use of thrombolytics and clot extraction, rapid diagnosis is seen as as an important component of care. Emergency personnel from EMTs to the emergency department (ED) staff have learned to recognize a common hemiplegic stroke, but strokes arising from other areas of the cerebral circulation are a lot harder to identify. This two-part series deals with non-hemiplegic cerebrovascular ischemic attacks. The term ischemic attack encompasses both TIA and stroke, as these may be difficult to differentiate early in the course of the disorder. Further, as pointed out in the article, TIA is a risk factor for later stroke in some patients. The first part of this article will deal with vertigo and gait disorders. Part II will cover vision and speech disorders.
—Sandra M. Schneider, MD, FACEP, Editor

Case Presentation

A 62-year-old, right-handed, Caucasian male presents to the emergency department (ED) with a chief complaint of gait instability, slurred speech, and double vision. He is unable to recall when his symptoms began, but his son notes that he sounded normal on the telephone approximately 4 hours previously. On physical examination, the patient required repeated stimulation to cooperate with the examination. He was noted to have dysarthric speech, with pronounced involvement of palatal and lingual syllables. Cranial nerve evaluation revealed a dysconjugate gaze in primary position, with inability to look upward on command. Although motor strength was normal, the patient was unable to perform finger-to-nose testing or heel-knee-shin testing on the right side without considerable dysmetria. A noncontrast head CT was read by the radiologist as "normal."

What would be an appropriate lesion to explain this patient's symptoms, and what would be an appropriate disposition and management plan? The objective of this two-part article is to help arm the emergency physician to be able to answer these questions.

Introduction

Each year, approximately 500,000 Americans suffer from a brain attack. It is the third leading cause of death in the United States, but it is the leading cause of disability, with greater than 30% of survivors demonstrating residual incapacity. The approximate cost in health care and lost productivity was estimated at $45 billion in 2001; without improvements in treatment and prevention, this number can only be expected to increase as the population continues to age.[1]

Approximately 85% of strokes in the United States are ischemic in nature.[2] Over the last decade, a variety of new therapeutic interventions have been approved that are effective in reducing death and disability from cerebral ischemia, including intravenous thrombolysis and various methods for mechanical thrombectomy. These therapies are exquisitely time-sensitive. The diagnosis is more difficult in

Executive Summary

- Stroke is a time-sensitive disorder.
- Vertical nystagmus and dysconjugate gaze suggest a central lesion.
- Wallenberg's syndrome or lateral medullary syndrome presents with dysarthria, vertigo, nausea and vomiting, and loss of pain and temperature sensation over the ipsilateral face and contralateral body.
- Pure sensory stroke occurs from a thalamic lesion and generally involves the face, arm, and leg (rarely fewer than 2 of these).
- Isolated ataxia suggests cerebellar ischemia. Usually there are other cerebellar signs on physical examination.

the setting of transient ischemic attack (TIA), defined as a neurological deficit lasting less than 24 hours.[3] TIA is a harbinger of ischemic stroke, portending a 48-hour stroke risk as high as 8-11% in some patients.[4-6]

As a consequence, early recognition of the signs and symptoms of ischemic cerebrovascular disease is essential to preserve the opportunity to intervene. Most physicians are familiar with the most common symptoms of stroke: sudden loss of motor function on one side of the body, speech difficulties, and gait instability. An added challenge for emergency physicians, however, is properly diagnosing ischemic brain attacks in patients who do not present with these classic symptoms. Patients may present to emergency departments with less specific complaints including dizziness, vague sensory changes, and vision disturbances for which the differential diagnosis may be quite broad. A significant challenge for today's practitioner is identifying which of these patients is suffering from an ischemic brain attack versus a more benign and less time-sensitive process.

Pathophysiology

Of all the organs in the body, the brain is the most dependent upon a continuous blood supply. The brain receives 15% of the cardiac output and is responsible for 20% of total body O_2 consumption, despite accounting for only 2% of body weight. It is not capable of anaerobic metabolism and does not store either glycogen or myoglobin.[7] Consequently, irreversible brain injury can occur when the brain is deprived of O_2 or glucose for more than a few minutes (4-5 in the case of anoxia, 10-15 in the case of hypoglycemia).

Cerebral ischemia occurs when blood flow to a region of the brain falls below the level required to meet the metabolic needs of the tissue. Cessation (or significant reduction) of blood flow precipitates a cascade of events at the cellular level that can result in cell death if perfusion is not restored. Within seconds of onset, energy failure with loss of aerobic glycolysis occurs. The availability of high-energy phosphate compounds declines, with ATP concentration approaching zero after 4 minutes.[8] This is associated with a rise in intracellular calcium ions, obliterating the normal intracellular-extracellular gradient. The influx of Ca++ ions leads to cell depolarization and release of cytotoxic neurotransmitters (primarily glutamate). Furthermore, the anaerobic metabolism results in a local increase in lactic acid concentration. These events collectively produce increased free radical production and cytotoxic edema. The depolarization of the cellular membrane results in activation of phospholipase C, among others, resulting in lipid peroxidation and loss of membrane integrity.[2,8]

In the setting of an arterial occlusion, the degree of ischemia experienced by a particular region of brain tissue depends on the availability of collateral circulation. Collateral connections exist between distal branches of the anterior cerebral artery (ACA) and the middle cerebral artery (MCA) as well as between branches of the middle cerebral artery and the posterior cerebral artery (PCA). (*See Figures 1 and 2.*)

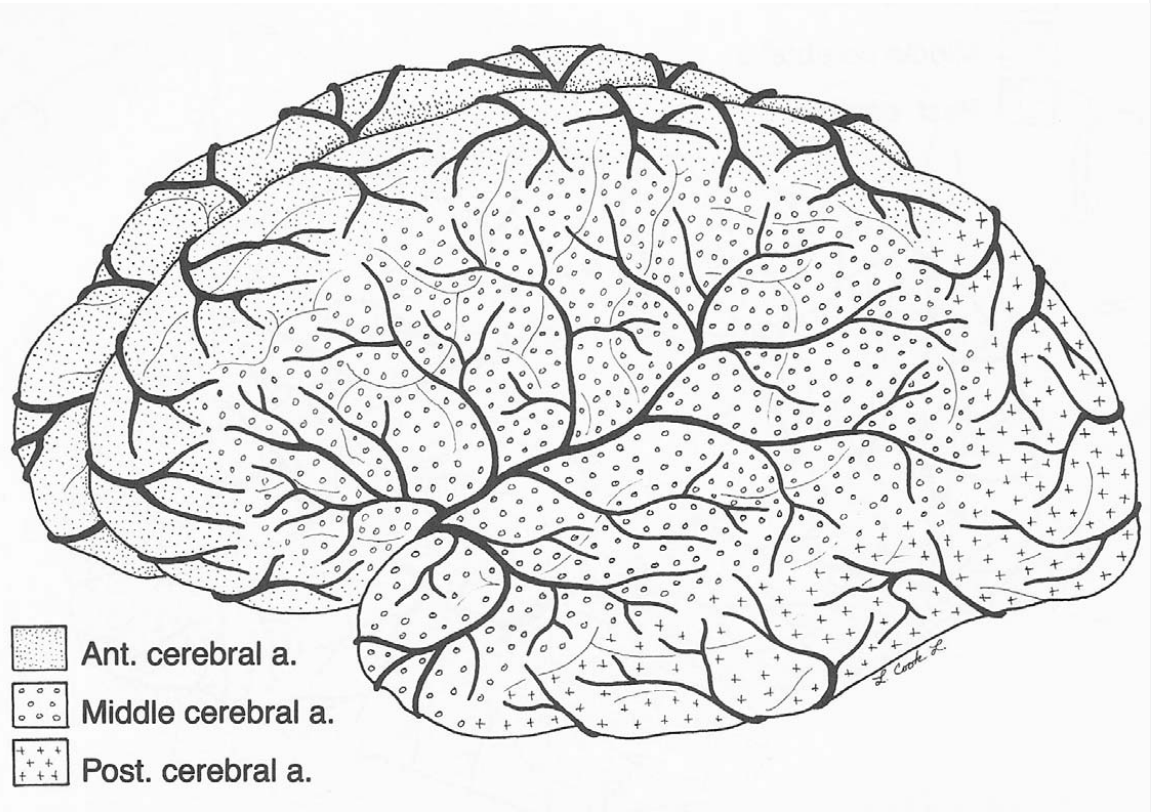
Consequently, in the setting of acute arterial obstruction, there is typically a core of severely affected tissue in the center, which receives its blood supply primarily from the involved artery, surrounded by a ring of less-severely affected tissue that is fed in part by collaterals. This less-severely affected region is called the ischemic penumbra. If the artery is re-canalized before the core region experiences irreversible injury, an infarction may be avoided; however, even if the obstruction remains fixed, some of the tissue in penumbra may remain viable, depending upon the degree of collateral supply.[9] In short, the size of the infarct depends upon both the degree and the duration of ischemia.

Cerebral ischemia may occur in a variety of settings. *Global* cerebral ischemia may occur during periods of severe hypotension. *Focal* cerebral ischemia most commonly occurs following obstruction of an artery. Arterial obstruction can result either from *in situ* thrombus formation or from embolic phenomena. The majority of thrombotic occlusions result from atherosclerosis. Emboli most commonly arise from the heart, especially in the setting of atrial fibrillation, or from large vessels with atherosclerotic plaques. Less commonly, strokes can result from venous obstruction, vasospasm, or arterial inflammation (vasculitis).

Clinical Presentation

Ischemic cerebrovascular disease is characterized by the sudden onset of focal neurological deficits. Classically, the deficit is maximal at onset; however, in rare cases symptoms may fluctuate or "stutter." The patient's deficits result from dysfunction of the

Figure 1: Lateral View of the Left Cerebral Hemisphere



Lateral, somewhat oblique, view of the convexal surface of the left cerebral hemisphere and the paramedian portion of the right cerebral hemisphere showing the anterior, middle, and posterior cerebral arteries and their territories.
Used with permission from: Bogoisslavsky J, Caplan L, eds. *Stroke Syndromes*. New York, NY: Cambridge University Press; 1995.

ischemic brain structures; consequently, the clinical presentation will be dictated by the region of ischemia. An understanding of basic cerebrovascular anatomy (*see Figures 1, 2, and 3*) can be very useful when evaluating patients with neurological complaints. Often, the careful clinician can elicit history or examination findings suggestive of dysfunction of adjacent brain regions in the same vascular territory, so called "co-localizing signs." For instance, a patient presenting with isolated dysarthria is unlikely to have had an ischemic stroke, but the constellation of dysarthria, dysphagia, deviation of the tongue and palate, and facial weakness is suspicious for pontomedullary ischemia.

The following sections provide a description of atypical ischemic brain attack syndromes with a chief complaint-based organization. The article will attempt to highlight features that may help the examiner to differentiate between a stroke and other potential causes.

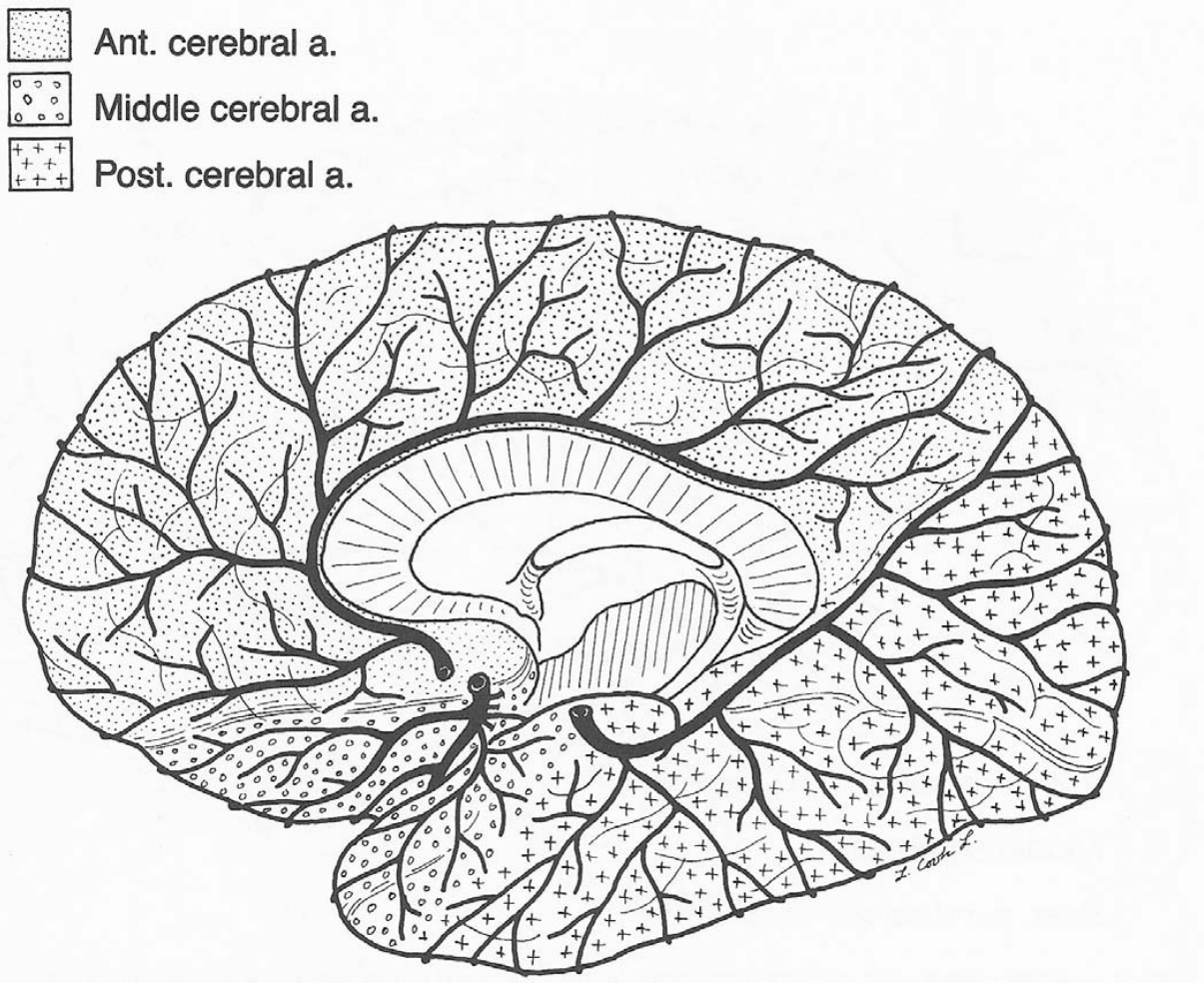
Vertigo and Associated Symptoms

Dizziness is a common complaint among patients seen by emergency physicians. The most common causes of dizziness are peripheral vestibular disorders, but central nervous system disorders must be excluded. The sensation of vertigo is the outcome of many pathological processes causing a mismatch among the visual, vestibular, and somatosensory systems. The areas of the brain that are responsible for these symptoms are the pons, medulla, and cerebellum, which are supplied by branches of the vertebrobasilar system. (*See Figure 3.*)

The history and physical examination findings often are helpful in differentiating central from peripheral causes.

Patients may use the word "dizzy" to describe a wide variety of sensations. Careful questioning may be necessary to determine whether the patient's complaint is more consistent with disequilibrium, vertigo, or presyncopal sensation. Pre-syncope or light-headedness without the sensation of movement is almost always secondary to a reduction in blood flow to the entire brain.[10] While this is suggestive of hypotention, it is not consistent with a focal brain ischemia. However, vertigo, the illusion of movement, is caused by vestibular system dysfunction and can be localized either to the auditory canals or the brainstem.

Figure 2: Sagittal Section of the Right Cerebral Hemisphere



Sagittal section of the right cerebral hemisphere showing the anterior, middle, and posterior cerebral arteries and their territories.
Used with permission from: Bogoisslavsky J, Caplan L, eds. *Stroke Syndromes*. New York, NY: Cambridge University Press; 1995.

Patients may describe a sensation of spinning or tilting in relation to the environment. Peripheral vertigo typically occurs as discrete spells that are sudden in onset; the duration can vary from seconds to hours depending upon the etiology. Spells may be accompanied by hearing loss, tinnitus, or a sense of fullness in the affected ear. Nausea and vomiting may be prominent. Symptoms often are either precipitated or exacerbated by movement, and the patient feels better while lying still.

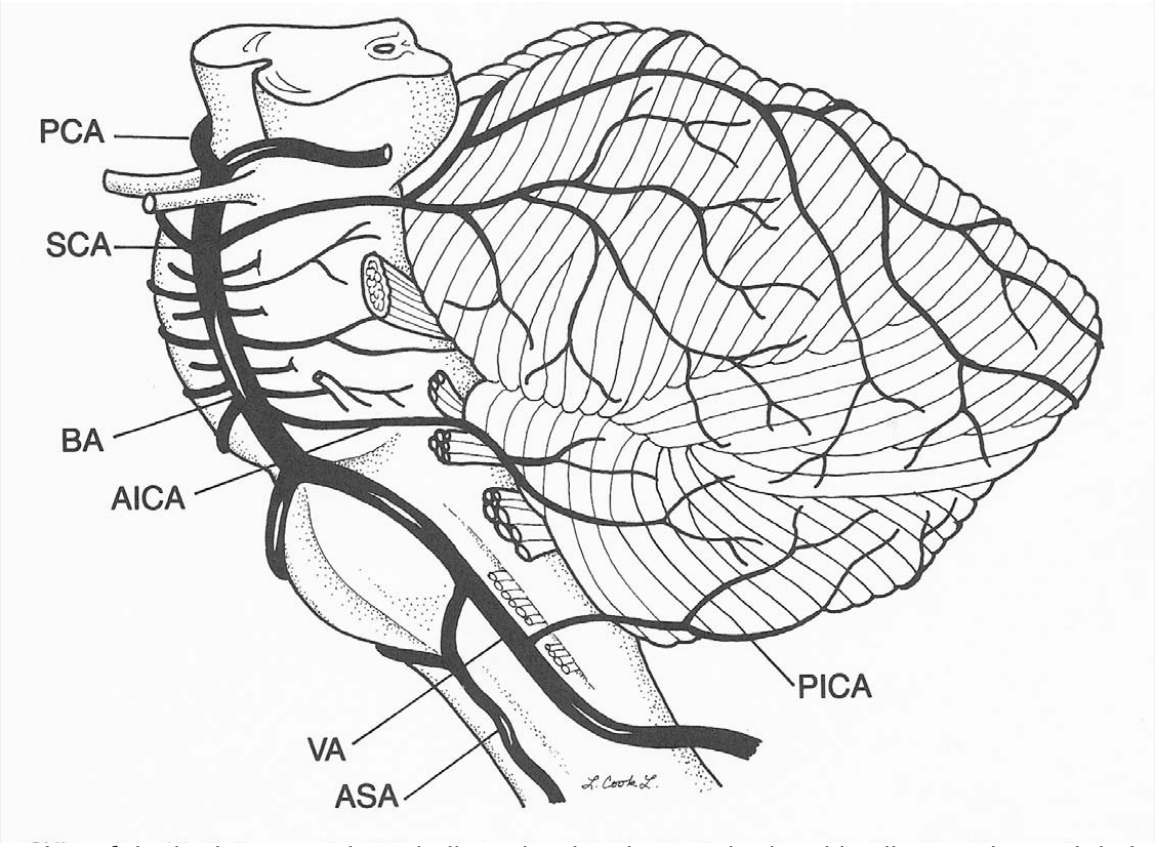
Central vertigo often has a more variable presentation. Patients may describe a sense of tilting or being pushed to one side. The time course and effect of movement are less predictable. Importantly, central vertigo often is accompanied by other signs suggestive of brain dysfunction, as discussed below.

On physical examination, the patient's eye movements, hearing, coordination, gait, and cranial nerve examination should be assessed. Unilateral hearing loss is very suggestive of a peripheral cause, such as labyrinthitis or Meniere's disease. The Dix-Hallpike maneuver can be diagnostic for benign postitional vertigo (BPV), which is one of the most common peripheral causes of vertigo. With the patient in a seated position, the examiner turns the patient's head 45 degrees to one side and then has the patient recline rapidly. The presence of horizontal nystagmus constitutes a positive test. Classically, nystagmus in the setting of BPV has a brief latency, lasts less than 30 seconds, improves with repeated maneuvers, and resolves upon sitting up.[11]

Horizontal nystagmus can be present in patients with central or peripheral causes of vertigo, but vertical nystagmus is indicative of brainstem pathology. The presence of dysconjugate gaze, either at rest or during smooth pursuit, is highly suggestive of a central cause as well. Dysarthria, hoarse speech, dysphagia, and facial asymmetry may be signs of cranial nerve dysfunction and are consistent with a central etiology.

Patients with vertigo from any

Figure 3: Oblique View of the Brainstem and Cerebellum



Oblique view of the brainstem and cerebellum showing the vertebral and basilar arteries and their branches. VA = vertebral artery, ASA = anterior spinal artery, PICA = posterior inferior cerebellar artery, AICA = anterior inferior cerebellar artery, BA = basilar artery, SCA = superior cerebellar artery, PCA = posterior cerebral artery.
Used with permission from: Bogoisslavsky J, Caplan L, eds. *Stroke Syndrome*s. New York, NY: Cambridge University Press; 1995.

cause may complain of gait instability. The gait evaluation may demonstrate veering to one side in either central or peripheral cases; in the latter, the patient tends to veer toward the side of the affected ear. However, a broad-based, unstable gait in a patient with vertigo is concerning for cerebellar pathology. Other physical signs associated with cerebellar pathology include dysmetria and dysdiadochokinesia. See Table 1 for other examination techniques.

The vestibular system is distributed throughout the medulla and lower pons, near structures associated with cranial nerves V, IX, and X, sympathetic autoregulation, and the cerebellar system, among others. Consequently, vertigo resulting from an ischemic event is highly likely to be accompanied by co-localizing signs related to dysfunction of these nearby structures.

The lateral medullary syndrome, also known as Wallenberg syndrome, occurs secondary to infarction of the lateral part of the medulla including the vestibular nuclei. The syndrome typically results from an occlusion of the posterior inferior cerebellar artery but can occasionally be secondary to vertebral artery occlusion. Patients generally present with dysphagia and dysarthria; vertigo, nausea, vomiting; ipsilateral ataxia; and loss of pain and temperature sensation over the ipsilateral face and contralateral body. An ipsilateral Horner's syndrome may be present due to involvement of descending sympathetic tracts. An individual patient may manifest only a portion of the described symptoms and signs. Other neurological symptoms that may accompany vertigo secondary to brainstem ischemia include hoarseness and facial paresis, secondary to involvement of the rostral medulla; diplopia or nystagmus; and gait ataxia, which can be seen with involvement of the caudal medulla or cerebellum.[12]

Summary Points

- It is important to classify what type of "dizziness" the patient is experiencing.
- Patients complaining of "dizziness" should have their examination include eye movements, hearing, coordination, gait, and cranial nerves.
- Vertical nystagmus or dysconjugate gaze implies a central neurologic lesion.
- Vertigo from central ischemia usu-

Table 1: Specific Physical Examination Maneuvers and Their Significance

Romberg maneuver (The patient stands with feet together and eyes closed. The test is positive for impaired proprioception if swaying begins upon eye closure and resolves when the eyes are opened again.)	Proprioception (joint position)
Tuning fork	Vibratory
Finger-nose, heel-shin (dysmetria)	Coordination (localized to cerebellum)
Rapid alternating movement (dysdiadochokinesia)	If arrhythmic then cerebellar If asymmetric slowing usually subtle finding for unilateral weakness
Deep tendon reflexes	Hyperreflexic — Upper motor neuron injury Decreased — Lower motor neuron injury
Dix-Hallpike maneuver	Positive in peripheral vertigo
Tandem gait	Proprioception (cerebellar)

ally has co-lateralizing signs that include paralysis, vision changes and ataxia.

Acute Sensory Changes

The perception of sensation is an integration of multiple peripheral nerve afferent systems and the central brain interpretation of those signals. Sensation can be broken down into different modalities. The classic differentiation of sensation is superficial (light touch, pain, and temperature), deep (position sense and vibration), and discrimination (stereognosis and two-point discrimination). The spinothalamic tract, lateral trigeminothalamic tract, and posterior columns are among the many pathways that transmit to the medial thalamic structures. The thalamus is thought to be the central relay area for the incoming sensory information, as the signals are transmitted to the appropriate areas of the cerebrum that interpret and act upon the sensory information.

Patients may present with complaints of changes in sensation. Often the word "numb" is used. This can be used to describe loss of sensation, "anesthesia," or the addition of sensation (pins-and-needles, tingling) or "paresthesia." It can be difficult for the patient to accurately make this distinction between negative and positive symptoms on history or physical examination. The list of possible diagnoses for this broad and common complaint of "numbness" is extensive. A partial listing is found in Table 2.

Determining the proper index of suspicion for a brain attack in the setting of this common complaint is important and can be challenging. When the sensory loss is associated with other classic brain attack symptoms like weakness and dysarthria, the index of suspicion for stroke is obvious. The challenge is determining when to suspect stroke in the setting of only changes in sensation without other neurologic symptoms.

About 2% of those who experience a brain attack will have one that is exclusively sensory in nature.[13] The distribution of the sensory loss can be quite variable, as well as which types of sensation are affected. There are dozens of stroke syndromes that correlate to very specific areas of the thalamus and other sensory relay areas of the brain. They may result in anesthesia or paresthesia in distributions that can vary from the hemi-body to isolated lateral trunk.[14] Most commonly, the sensory involvement shows a face-arm-leg or arm-face distribution. Only rarely is one of these three body areas affected in isolation.[13] Thus, typically a pure sensory stroke (PSS) should be suspected only in the presence of isolated sensory disturbances involving at least two body areas.

PSS is suggestive of thalamic stroke but also has been described in association with pontine, mesencephalic, and subcortical strokes. The thalamus is supplied by branches of the posterior cerebral artery that feed off the vertebrobasilar artery.

Isolated thalamic injury is possible. Thalamic syndrome (Dejerine-Roussy) is an ischemic event in which there may be a total loss of sensation to half of the body. It can involve any or all of the sensory modalities and may result in allodynia, which is hypersensitivity without sensory loss. This is usually in a hemi-body distribution and, if the lesion is isolated to the thalamus, can present with no paralysis or other neurologic symptoms.

Brainstem ischemia can cause other patterns of sensory loss, but the presence of paresthesias will not be an isolated finding. These patients should have obvious deficits that include weakness and possibly altered level of consciousness, as discussed in the section about dizziness.

Summary Points

- The differential diagnosis for

Table 2: Differential Diagnosis for Paresthesias/Abnormal Sensation

Central neurologic	Ischemic brain attack Multiple sclerosis Tumor Seizure Spinal cord injury Guillian-Barre syndrome
Metabolic	Diabetes Hyperventilation Hypothyroidism
Toxicologic	Heavy metals Antibiotics Chemotherapy
Malnutrition	Chronic alcoholism B12 deficiency
Trauma	Neuropraxia
Inflammatory diseases	Autoimmune Chronic wounds Vasculitis
Infectious	Lyme disease Human immunodeficiency virus (HIV) Leprosy Tertiary syphilis
Genetic disorders	Inherited peripheral neuropathies Porphyria

isolated sensory changes and paresthesias is vast.

- Pure sensory strokes usually involve the face, arm, and leg distribution and very rarely involve fewer than two of these areas.
- In a sensory stroke without other focal neurologic deficits, the site is most commonly the thalamus.

Gait Instability/Ataxia

Gait instability is a common complaint encountered in the emergency setting. Patients may present with complaints of difficulty executing motor movements with or without the complaint of focal weakness. Often, this can be manifested as difficulty executing activities of daily living and, commonly, difficulty ambulating. While the noticeable problem can be complaints of walking, ataxia is the gross lack of coordination of muscle movements in any limb or body part from picking up a cup of coffee to simply being able to sit up straight.

The ability to execute coordinated muscle movements relies upon the integration of the musculoskeletal system, peripheral nervous system (both for sensory acquisition and efferent relay of information), and central nervous system (spinal cord, cerebellum, and motor cortex).

The primary motor cortex in the frontal lobe is thought to be the main area responsible for planning of motor function. The somatosensory information acquired from the PNS is integrated in the somatosensory cortex, parietal cortex, and cerebellar pathways. Dysfunction in any of these pathways affects movement by the patient.

While dysfunction of the musculoskeletal, vestibular, PNS, or cerebral cortex may present as incoordination, the most pertinent type of ataxia for the discussion of ischemic brain attack with isolated ataxia is that of the cerebellum. The cerebellum is the only area of the brain that typically presents without focal motor deficits in the setting of an acute brain attack.

While other areas of the brain, including brainstem and internal capsule, may have ataxia as part of their constellation of symptoms, they most likely will have other symptoms, including motor weakness and sensory deficits. The cerebellum is supplied by the PICA, AICA, and superior cerebellar artery. (*See Figure 3.*)

The examination of the patient with ataxia is focused on evaluating those systems necessary to maintain an upright posture. Once it has been established that the patient's complaint cannot be explained by the musculoskeletal or vestibular system, and it has been decided that it is likely neurologic, differentiating the cause as peripheral versus central in nature can be a challenge.

An integral part of an ataxia evaluation should include a musculoskeletal examination to rule out obvious mechanical problems that explain difficulty with walking or using an upper extremity. It is important to consider whether the patient has adequate strength to stay upright.

If weakness is an issue, examination should include looking for upper motor neuron damage (hyperactive reflexes, spasticity, or Babinski sign) versus lower motor neuron damage (diminished reflexes, atrophy, or fasciculation). If the patient is weak, it is essential to assess whether the degree of gait difficulty is fully explained by the weakness.

The clinician can differentiate the cause of the ataxia as being either peripheral sensory or cerebellar ischemia through the use of both

Table 3: National Institutes of Health Stroke Scale

Tested Item	Title	Responses and Scores
1A	**Level of consciousness**	0-alert 1-responds to minor stimulation 2-requires repeated stimulation 3-coma/unresponsive
1B	**Orientation questions (2)** Ask patient to name the month and to state his/her age	0-answers both correctly 1-answers one correctly 2-answers neither correctly
1C	**Response to commands (2)** Ask the patient to open and close His/her eyes, and to make a fist	0-performs both tasks correctly 1-performs one task correctly 2-performs neither
2	**Gaze** Evaluate horizontal gaze	0-normal horizontal movements 1-partial gaze pals 2-complete gaze palsy
3	**Visual fields**	0-no visual field defect 1-partial hemianopia 2-complete hemianopia 3-bilateral hemianopia
4	**Facial movement**	0-normal 1-minor facial weakness 2-partial paralysis 3-complete unilateral palsy
5	**Motor function (arm)** Have patient hold arm extended, palm down Score each arm separately	0-no drift 1-drift before 10s but doesn't hit bed 2-falls before 10 seconds 3-no effort against gravity 4-no movement
6	**Motor function (leg)** Have patient hold leg elevated at about 30° Score each leg separately	0-no drift 1-drift before 5 seconds 2-falls before 5 seconds 3-no effort against gravity 4-no movement
7	**Limb ataxia** Perform finger-nose-finger or heel-knee-shin in all limbs. Score 0 if paralyzed or if patient cannot understand.	0-no ataxia 1-ataxia in 1 limb 2-ataxia in 2 limbs
8	**Sensory** Test pin prick sensation. In obtunded patients look for withdrawal from noxious stimulation. Patients who are comatose are given a 2.	0-no sensory loss 1-mild sensory loss 2-severe sensory loss

(*continued*)

Table 3: National Institutes of Health Stroke Scale (continued)

Tested Item	Title	Responses and Scores
9	**Language** Test naming and evaluate spontaneous speech. Patient may be asked to describe events in a standard picture available from NIH.	0-normal 1-mild aphasia 2-severe aphasia 3-mute or global aphasia
10	**Dysarthria** Score as "untestable" if intubated.	0-normal 1-mild dysarthria 2-severe dysarthria
11	**Extinction or inattention** Evaluate for attention to each side, and for extinction to double simultaneous stimulation. Score 0 if unable to evaluate.	0-absent 1-mild (loss 1 sensory modality) 2-severe (loss 2 modalities)

Adapted from: Anonymous. Educational Stroke Resources for Health Professionals. Bethesda, Maryland: www.ninds.nih.gov (US National Institutes of Health); 2007. Available at http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

historical and physical findings. Historically, the most important component is the time course of onset. If the onset is gradual over a long period of time, it is unlikely to be a stroke. Coexisting symptoms of vertigo, weakness, stiffness or slowness of movement, abnormal movements, cognitive difficulties, or significant changes in behavior can be clues to vestibular, cerebellar, pyramidal, extrapyramidal, or frontal lobe disorders.

One of the most common causes of ataxia is loss of proprioception in the feet. This can be tested by examining vibratory and joint position sense at the great toe. These patients often have worsened symptoms when their vision is obscured, and they usually broaden their base of support. They also have an abnormal Romberg sign (trouble standing with their eyes closed, but improved with eye opening). The patient can improve the ataxia by remaining in physical contact with the wall, which may act as a sensory replacement for the lack of information being relayed from the feet.

Patients with cerebellar ataxia tend to compensate by broadening their base of support (normally, the medial side of each foot strikes along a straight line when walking). Examining the patient's posture when he or she is standing with feet together is important. Patients tend to be unsteady in cerebellar ataxia whether they have their eyes opened or closed. Most often, patients have other signs of cerebellar disease, such as speech disturbance.

See Table 1 for a section with specific examination maneuvers and what localizing neurologic area they evaluate.

Hysterical ataxia can be a gait disturbance from a psychological cause. This can be acute onset and have isolated ataxia with no other symptoms, but there are features that can differentiate it from a cerebellar stroke. These patients often "catch themselves" in ways that would suggest higher levels of motor performance than their poor gait would indicate. Astasia-abasia is a term that has been applied to the condition in which the patient lurches wildly and only falls when there is someone or something to break the fall. The wild lurching often requires more coordination that one normally needs to walk.

Another acute cause of ataxia can be carbon monoxide poisoning. These patients can have global neurologic sequelae of confusion, memory deficits, and profound truncal and gait ataxia. These symptoms can improve rapidly with treatment, and the clinical scenario usually differentiates this medical condition from an acute cerebrovascular cause.

Summary Points

- In the setting of ataxia, physical examination of the musculoskeletal system, vestibular, and PNS can help differentiate the cause from a central neurologic event.
- In the setting of an acute brain attack, the only stroke syndrome that will cause isolated ataxia is cerebellar ischemia.
- If a cerebellar stroke is suspected, other cerebellar functional tests (*see Table 2*) can help rule in or out the diagnosis.

Case Conclusion

Now let us return to the case from the introduction of the 62-year-old male who presents with slurred speech, ataxia, and diplopia. Despite the initial head CT read as "normal," it was presumed likely that, based on the clinical presentation, the patient was experiencing brainstem ischemia. The patient was emergently transferred to a primary stroke center. En route, his level of consciousness declined and he was intubated for airway protection.

Further testing confirmed infarctions of the right cerebellum and portions of the midbrain and thalamus,

resulting in residual right-sided hemiataxia, paresis of up-gaze, and hemisensory loss. After emergent treatment, he eventually had a good recovery and was discharged to an inpatient rehabilitation facility.

Part II of this article will deal with other non-hemiplegic stroke syndromes and will discuss management. At that time, the imaging studies of the patient in this case example will be displayed and discussed.

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Physician CME Questions

81. Which of the following findings is consistent with a central cause of vertigo?
    A. Discrete episodes of vertigo that only occur with movement
    B. Presence of vertical nystagmus on physical examination
    C. Loss of hearing and sense of fullness in one ear
    D. Sensation of light-headedness upon standing
    E. Concomitant tinnitus of the ear

82. The ischemic penumbra is:
    A. That area of the brain that is most vulnerable to ischemia
    B. The core of the ischemic insult
    C. An area of brain surrounding the ischemic area that has less severely affected tissue fed by collaterals
    D. The area of a stroke that determines the physical findings

83. A patient presents with vertigo. You perform a Dix-Hallpike maneuver (patient seated with head turned 45 degrees and then rapidly reclines). Which of the following is correct?
    A. The patient has a central lesion if she has horizontal nystagmus.
    B. The patient has a peripheral lesion if she has horizonal nystagmus.
    C. The patient has a central lesion if

Emergency Medicine Reports

CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

she has no nystagmus.
D. The test is only positive if she has nystagmus and vomiting.

84. Which of the following is *not* commonly seen in vertigo from a peripheral cause?
A. nausea and vomiting
B. hearing loss
C. dysmetria
D. sudden onset

85. Which pattern of sensory loss is most consistent with an isolated thalamic stroke?:
A. bilateral foot numbness
B. isolated numbness of the lateral right thumb
C. right sided face, arm, and leg numbness
D. loss of pain and temperature sensation to the right face and left arm and leg
E. total bilateral sensory loss below the umbilicus with sacral sparing

86. Which of the following examination findings suggests isolated cerebellar dysfunction?
A. The patient is standing upright with arms extended. Upon closing of the eyes, the patient begins to sway and becomes unsteady but corrects with eye opening.
B. Incomitant binocular diplopia
C. Left hemiparesis
D. Left hemi-anesthesia
E. Dysmetria (abnormal finger to nose testing)

87. A patient presents with total loss of sensation over one-half of his body. The lesion is likely to be:
A. thalamus
B. pons
C. peripheral neuropathy
D. spinal cord

88. Wallenberg syndrome (lateral medullary syndrome) presents with all of the following *except*:
A. vertigo
B. loss of pain sensation over face
C. loss of vision
D. nausea/vomiting

89. Which of the following areas of the brain are involved in gait?
A. frontal lobe
B. peripheral nervous system
C. cerebellum
D. all of the above

90. The differential diagnosis of ataxia includes:
A. cerebellar stroke
B. hysteria
C. carbon monoxide poisoning
D. all of the above

CME Answer Key

81. B; 82. C; 83. B; 84. C; 85. C; 86. E; 87. A; 88. C; 89. D; 90. D

In Future Issues

Missed Myocardial Infarction

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Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Ischemic Cerebrovascular Disease: Part I

Specific Physical Examination Maneuvers and Their Significance

Romberg maneuver (The patient stands with feet together and eyes closed. The test is positive for impaired proprioception if swaying begins upon eye closure and resolves when the eyes are opened again.)	Proprioception (joint position)
Tuning fork	Vibratory
Finger-nose, heel-shin (dysmetria)	Coordination (localized to cerebellum)
Rapid alternating movement (dysdiadochokinesia)	If arrhythmic then cerebellar. If asymmetric slowing usually subtle finding for unilateral weakness
Deep tendon reflexes	Hyperreflexic — Upper motor neuron injury Decreased — Lower motor neuron injury
Dix-Hallpike maneuver	Positive in peripheral vertigo
Tandem gait	Proprioception (cerebellar)

Differential Diagnosis for Paresthesias/Abnormal Sensation

Central neurologic	Ischemic brain attack Multiple sclerosis Tumor Seizure Spinal cord injury Guillian-Barre syndrome
Metabolic	Diabetes Hyperventilation Hypothyroidism
Toxicologic	Heavy metals Antibiotics Chemotherapy
Malnutrition	Chronic alcoholism B12 deficiency
Trauma	Neuropraxia
Inflammatory diseases	Autoimmune Chronic wounds Vasculitis
Infectious	Lyme disease Human immunodeficiency virus (HIV) Leprosy Tertiary syphilis
Genetic disorders	Inherited peripheral neuropathies Porphyria

National Institutes of Health Stroke Scale

Tested Item	Title	Responses and Scores
1A	**Level of consciousness**	0-alert 1-responds to minor stimulation 2-requires repeated stimulation 3-coma/unresponsive
1B	**Orientation questions (2)** Ask patient to name the month and to state his/her age	0-answers both correctly 1-answers one correctly 2-answers neither correctly
1C	**Response to commands (2)** Ask the patient to open and close His/her eyes, and to make a fist	0-performs both tasks correctly 1-performs one task correctly 2-performs neither
2	**Gaze** Evaluate horizontal gaze	0-normal horizontal movements 1-partial gaze pals 2-complete gaze palsy
3	**Visual fields**	0-no visual field defect 1-partial hemianopia 2-complete hemianopia 3-bilateral hemianopia
4	**Facial movement**	0-normal 1-minor facial weakness 2-partial paralysis 3-complete unilateral palsy
5	**Motor function (arm)** Have patient hold arm extended, palm down Score each arm separately	0-no drift 1-drift before 10s but doesn't hit bed 2-falls before 10 seconds 3-no effort against gravity 4-no movement
6	**Motor function (leg)** Have patient hold leg elevated at about 30° Score each leg separately	0-no drift 1-drift before 5 seconds 2-falls before 5 seconds 3-no effort against gravity 4-no movement
7	**Limb ataxia** Perform finger-nose-finger or heel-knee-shin in all limbs. Score 0 if paralyzed or if patient cannot understand.	0-no ataxia 1-ataxia in 1 limb 2-ataxia in 2 limbs
8	**Sensory** Test pin prick sensation. In obtunded patients look for withdrawal from noxious stimulation. Patients who are comatose are given a 2.	0-no sensory loss 1-mild sensory loss 2-severe sensory loss

(*continued*)

National Institutes of Health Stroke Scale (continued)

Tested Item	Title	Responses and Scores
9	**Language** Test naming and evaluate spontaneous speech. Patient may be asked to describe events in a standard picture available from NIH.	0-normal 1-mild aphasia 2-severe aphasia 3-mute or global aphasia
10	**Dysarthria** Score as "untestable" if intubated.	0-normal 1-mild dysarthria 2-severe dysarthria
11	**Extinction or inattention** Evaluate for attention to each side, and for extinction to double simultaneous stimulation. Score 0 if unable to evaluate.	0-absent 1-mild (loss 1 sensory modality) 2-severe (loss 2 modalities)

Adapted from: Anonymous. Educational Stroke Resources for Health Professionals. Bethesda, Maryland: www.ninds.nih.gov (US National Institutes of Health); 2007. Available at http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

Supplement to *Emergency Medicine Reports*, April 13, 2009: "Atypical Presentations of Ischemic Cerebrovascular Disease." *Authors*: **Dag Shapshak, MD**, Assistant Professor of Emergency Medicine, Medical University of South Carolina, Charleston; and **Angela N. Hayes, MD**, Assistant Professor of Neurology, Medical University of South Carolina, Charleston.

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Please take a moment to answer the following questions to let us know your thoughts on the CME program. Fill in the appropriate space and return this page in the envelope provided. **You must return this evaluation to receive your certificate. ACEP members — Please see reverse side for option to mail in answers.** Thank you.

CORRECT ● INCORRECT ⊘ ✓ ✗ ⚠

1. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other _____

After participating in this program, I am able to:	**Strongly Disagree**	**Disagree**	**Slightly Disagree**	**Slightly Agree**	**Agree**	**Strongly Agree**
2. Recognize or increase index of suspicion for specific conditions.	○	○	○	○	○	○
3. Understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed.	○	○	○	○	○	○
4. Apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed.	○	○	○	○	○	○
5. Understand the differential diagnosis of the entity discussed.	○	○	○	○	○	○
6. Understand both likely and rare complications that may occur.	○	○	○	○	○	○
7. The test questions were clear and appropriate.	○	○	○	○	○	○
9. I am satisfied with customer service for the CME program.	○	○	○	○	○	○
10. I detected no commercial bias in this activity.	○	○	○	○	○	○
11. This activity reaffirmed my clinical practice.	○	○	○	○	○	○
12. This activity has changed my clinical practice.	○	○	○	○	○	○

If so, how? _____

13. How many minutes do you estimate it took you to complete this entire semester (13 issues) activity? Please include time for reading, reviewing, answering the questions, and comparing your answers with the correct ones listed. _____ minutes.

14. Do you have any general comments about the effectiveness of this CME program?

_____

I have completed the requirements for this activity.

Name (printed) _____ **Signature** _____

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ATYPICAL PRESENTATIONS OF ISCHEMIC CEREBROVASCULAR DISEASE: PART I

81. A B C D E 82. A B C D 83. A B C D 84. A B C D 85. A B C D E 86. A B C D E 87. A B C D
88. A B C D 89. A B C D 90. A B C D