Clinical Evaluation of the Patient With Acute Stroke

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Relationship Disclosure: Dr. Southerland serves as deputy editor of the *Neurology* podcast and receives research/grant support from the American Academy of Neurology, American Board of Psychiatry and Neurology, Health Resources & Services Administration (HRSA GO1RH27869-01-00), and the National Institute of Neurological Disorders and Stroke (U01 NS069498).

Unlabeled Use of Products/Investigational Use Disclosure: Dr Southerland reports no disclosure.

ABSTRACT

Purpose of Review: This article reviews the clinical evaluation of the patient with acute stroke, including key questions in the focused stroke history, important aspects of the National Institutes of Health Stroke Scale and focused neurologic examination, and the significance of the basic head CT scan in informing a timely treatment decision.

Recent Findings: Advances in both stroke treatment and enhanced diagnostics support an evolving paradigm for acute stroke care, ranging from the prehospital setting to the rehabilitative setting. An international emphasis on best practice strategies promotes efficiency and standardization in stroke systems of care.

Summary: Despite continual changes and augmentations to the field of acute stroke, several fundamentals remain. Central among these is in-depth knowledge of neurovascular anatomy, clinical stroke syndromes, and common mimics, which are foundational to the bedside evaluation of the patient with acute stroke.

Continuum (Minneap Minn) 2017;23(1):40–61.

INTRODUCTION

For each of us privileged to care for patients with acute stroke, it all begins when the pager goes off. From the earliest days of residency, the acute stroke page sets in motion a sequential confluence of anxiety and adrenaline, apprehension and alertness, and, with proper training and experience, the application of acumen and alacrity to whatever stroke scenario we encounter. As the great basketball coach John Wooden wised, ''Be quick, but don't hurry.''

Navigating the oft-tortuous path of an acute stroke alert necessitates the ability to focus one's observational attention, listen intently for key aspects of the history, and remain aware of the team environment in order to facilitate a swift, efficient, and accurate assessment. The latter requires a focused neurologic examination, rapid interpretation of brain imaging, and a thorough knowledge of stroke syndromes and common mimics.

All this must happen rapidly, as ''time is brain,'' in concert with numerous members of the emergency team working toward the same goal. For acute ischemic stroke, ultra-early ''door to treatment'' has become the vanguard.[1,2] Toward this goal, initiatives such as the American Heart Association (AHA)/American Stroke Association (ASA) Get With the Guidelines and Target: Stroke programs seek to effect quality standards in stroke care (**Table 2-1**).[3–6]

Needless to say, acute stroke care is a dynamic practice and demands a nonlinear team-based approach to arrive rapidly at a well-informed diagnosis and treatment decision. This article reviews key aspects along this continuum in the clinical evaluation of the patient with acute stroke.

TABLE 2-1 American Heart Association/American Stroke Association Target: Stroke Best Practice Strategies[a]

▶ **Emergency Medical Services Prenotification**

Emergency medical services providers should provide early prenotification to the receiving hospital when stroke is recognized in the field.

▶ **Stroke Tools**

A stroke toolkit containing a rapid triage protocol, clinical decision support, stroke-specific order sets, guidelines, hospital-specific algorithms, critical pathways, the National Institutes of Health Stroke Scale, and other stroke tools should be available and used for each patient.

▶ **Rapid Triage Protocol and Stroke Team Notification**

Rapid neurologic evaluation should be performed as soon as possible in the emergency department or on the CT/MRI table.

▶ **Single-Call Activation System**

Single-call activation system for the stroke team is defined here as a system in which the emergency department calls a central page operator, who then simultaneously pages the entire stroke team, including notification to ensure rapid availability of the scanner for stroke protocol brain imaging.

▶ **Transfer Directly to CT Scanner**

Guided by prespecified protocols, eligible patients with stroke can, if appropriate, be transported from the emergency department triage area directly to the CT/MRI scanner for initial neurologic examination and brain imaging to determine recombinant tissue plasminogen activator (rtPA) eligibility, bypassing the emergency department bed.

▶ **Rapid Acquisition and Interpretation of Brain Imaging**

At the minimum, the CT scan should be performed within 25 minutes of arrival and interpretation of the CT scan completed within 45 minutes of arrival to exclude intracranial hemorrhage prior to administration of IV rtPA.

▶ **Rapid Laboratory Tests**

When indicated, laboratory tests such as glucose and, for patients in whom coagulation parameters should be assessed because of suspicion of coagulopathy or warfarin treatment, international normalized ratio (prothrombin time)/partial thromboplastin time results should be available as quickly as possible and no later than 30 minutes after emergency department arrival.

▶ **Mix rtPA Ahead of Time**

A useful strategy is to mix drug and set up the bolus dose and 1-hour infusion pump as soon as a patient is recognized as a possible rtPA candidate, even before brain imaging.

▶ **Rapid Access to and Administration of IV rtPA**

Once eligibility has been determined and intracranial hemorrhage has been excluded, IV rtPA should be promptly administered without delay.

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KEY POINT

■ Establishing time of stroke onset, or last known well time, starts the clock on all further decision making for the patient with acute stroke. Confirming last known well time with the patient or a reliable witness, or identifying an associated event, is key to informing accurate treatment decisions going forward.

TABLE 2-1 American Heart Association/American Stroke Association Target: Stroke Best Practice Strategies[a] *Continued from page 41*

▶ **Team-Based Approach**

The team-based approach based on standardized stroke pathways and protocols has proven to be effective in enhancing the number of eligible patients treated and reducing time to treatment in stroke.

▶ **Prompt Data Feedback**

Accurately measuring and tracking the hospital's door-to-needle times, IV rtPA treatment rates in eligible patients, other time intervals, and performance on other stroke performance/quality measures equip the stroke team to identify areas for improvement and take appropriate action.

CT = computed tomography; IV = intravenous; MRI = magnetic resonance imaging.
[a] Data from Fonarow GC, et al, Stroke,[4] *stroke.ahajournals.org/content/42/10/2983.long*; Fonarow GC, et al, JAMA,[5] *jamanetwork.com/journals/jama/fullarticle/1861802*; and Xian Y, et al, Stroke.[6] *stroke.ahajournals.org/content/45/5/1387.long*.

INITIAL HISTORY

First and foremost, acute stroke decision making relies on a brief but accurate history, the leading question of which is, ''When was the patient last seen normal?'' This question serves as a surrogate for time of stroke onset, which starts the clock on all subsequent time windows and management decisions. The question may be modified to, ''When was the patient last seen well?'' to avoid ambiguity over any prior deficits, or ''When was the patient last known well?'' in the case that stroke onset is inferred from unwitnessed events.

Last known well time is often first relayed from a witness or family member to an emergency medical services (EMS) provider, then communicated to the hospital staff through emergency dispatch or at the time of arrival, and then finally communicated to the neurologist representing the acute stroke team. Circling back with the patient or a reliable witness, or identifying an associated event (eg, the patient got up to use the bathroom or made a phone call at a certain time), is important to confirm last known well time and often requires a bit of rapid detective work. It is also good practice to establish the last known well time as a clock time (eg, 8:00 AM) rather than relaying last known well time as ''45 minutes ago'' or ''2 hours ago.'' Using a clock time ensures that all members of the acute stroke evaluation team are operating in the same time window from stroke onset and reduces the probability of calculation errors when estimating stroke onset.

Once the last known well time has been established, clinical diagnostic reasoning begins with a history of presenting symptoms. This first description informs an initial impression of ''stroke or no stroke'' and requires a thorough understanding of clinical stroke syndromes (as discussed later in this article). Symptom chronology is very important to characterizing stroke syndromes. For example, were the symptoms abrupt in onset or more gradual? Stroke tends to be a sudden and discrete event (maximum deficit at onset) compared to common mimickers, such as migraine or encephalopathy, which tend to be more gradual or vague in description.

Exceptions to the maximum deficit at onset rule may be a so-called *stuttering*

transient ischemic attack or a small vessel stroke, from which symptoms may fluctuate or crescendo to maximum deficit over the first 72 hours. While ''rapid improvement in symptoms'' is considered a relative contraindication to IV recombinant tissue plasminogen activator (rtPA), suspicion for stroke should remain high and treatment should remain an option unless the patient completely returns to a nondisabling baseline.

Several other key questions are important to the acute stroke history. A brief review of the patient's past medical history, focusing on vascular risk factors, informs the likelihood of stroke and possible stroke mechanism. If the initial stroke syndrome is unclear, searching for prior nonstroke presentations and common mimics is helpful.

Reviewing potential eligibility for IV rtPA prompts several additional questions. Is the patient taking blood thinners (ie, anticoagulation)? Has he or she experienced any recent hospitalizations, surgery, trauma, bleeding, or other illnesses? Again, it is imperative to verify the brief history with the patient or a close family member and cross-reference the medical record for relevant details as needed. For more information about eligibility criteria for IV rtPA, refer to the article ''Treatment of Acute Ischemic Stroke'' by Alejandro A. Rabinstein, MD, FAAN,[7] in this issue of *Continuum*.

INITIAL EXAMINATION

In the clinical evaluation of the patient with acute stroke, the neurologic examination begins from the first moment that eyes are laid upon the patient. For patients arriving via EMS, this is often as the patient rolls into the hospital on a stretcher or as the patient is stabilized by a team of providers in the emergency department (ED). These initial clinical observations are a critical aspect of the time-sensitive assessment and can help inform the focused neurologic examination thereafter. For example, is the patient awake? Is he or she able to speak or follow commands? Are the eyes open or closed? Is the head or gaze deviated? How are the limbs positioned on the stretcher or bed? Are there purposeful movements?

Patients with acute stroke with a decreased level of consciousness may be intubated in the field upon being ''found down'' or may require rapid intubation on ED arrival because of respiratory distress. In the case of ED intubation, a focused neurologic inventory should be attempted before the patient is pharmacologically sedated and paralyzed. A quick look for pupillary function, gaze deviation, blink to threat, motor tone, and purposeful movements can help formulate an initial impression of the neurologic syndrome.

With the initial observation, it is also essential to take a quick inventory of vital signs. As in any emergency situation, the adage holds true in the patient with acute stroke that airway, breathing, and circulation (the ABCs) always come first. Vital signs can also provide initial clues informing the stroke syndrome. Blood pressure, for example, is usually elevated in acute stroke as the body attempts to autoregulate cerebral perfusion pressure.[8] In a 2003 nationwide survey of 563,704 patients presenting to the ED with a diagnosis of stroke, approximately 70% had a systolic blood pressure of 140 mm Hg or higher, including 77% of patients with ischemic stroke, 75% of patients with intracerebral hemorrhage, and 100% of patients with subarachnoid hemorrhage.[9]

If a patient is potentially eligible for IV rtPA but his or her blood pressure is higher than 185/110 mm Hg or if a high suspicion for hemorrhagic stroke exists (eg, sudden onset of severe headache), preparations should be made for

KEY POINTS

- Symptom chronology is an important feature to help distinguish acute stroke from common stroke mimics. Stroke tends to be abrupt and maximal at onset, with the exception of stuttering transient ischemic attacks or small vessel strokes that may fluctuate in intensity in the acute period.
- Patients with acute stroke with decreased level of consciousness or respiratory distress may require rapid intubation. Prior to sedation, rapid assessment of pupillary function, gaze deviation, blink to threat, motor tone, and purposeful movements can help formulate the neurologic syndrome.

KEY POINTS

- Patients in atrial fibrillation with focal neurologic deficits should be assumed to have cardioembolic ischemic stroke until proven otherwise. Inquiring about anticoagulation and medication compliance in the acute stroke history is essential to informing an appropriate treatment decision.
- The National Institutes of Health Stroke Scale is biased toward left hemispheric and anterior circulation strokes. Therefore, careful vigilance should be employed when assessing stroke severity in patients with nondominant, right hemisphere, brainstem, or isolated cerebellar strokes to guide treatment.

lowering blood pressure in accordance with current treatment guidelines (eg, asking the pharmacist or nurse to prepare IV labetalol or a nicardipine drip to be immediately ready following head CT).[10]

In addition to assessing blood pressure, identifying the cardiac rhythm via ECG or telemetry informs the acute stroke presentation. Patients presenting in atrial fibrillation with focal neurologic deficits should be assumed to be having a cardioembolic stroke until proven otherwise.

The National Institutes of Health Stroke Scale

The most important caveat regarding the National Institutes of Health Stroke Scale (NIHSS) is to recognize that it is not an adequate substitute for a comprehensive neurologic examination. The NIHSS is, however, a highly reliable and valid screening assessment for the rapid evaluation of a patient with acute stroke.[11] The 11-item scale measures consciousness, orientation, visual fields, gaze, language fluency and comprehension, speech, sensory loss and neglect, motor strength, and limb ataxia. Validated for use by neurologists and nonneurologist providers and nurses, the scale can easily be completed in less than 10 minutes and serves as an initial measure of stroke severity ranging from 0 (no deficits) to 42 (maximum score). The NIHSS has no minimum score that would exclude eligibility to receive IV rtPA, and patients with mild but nonetheless disabling symptoms should be offered therapy.[12] Additionally, eligibility for endovascular therapy has recently been established for appropriate patients with an NIHSS score of 6 or higher and the presence of a large vessel occlusion.[13]

Of additional note, one should be aware of biases within the NIHSS. For instance, dominant (left) hemispheric strokes score approximately 4 points higher than nondominant (right) hemispheric strokes, reflecting the impact of aphasia on the neurologic assessment.[14] Similarly, for a given NIHSS score, the volume of infarction is greater for nondominant, right hemisphere than dominant left hemisphere strokes.[15] Additionally, the NIHSS may underestimate posterior circulation stroke deficits compared to anterior circulation stroke deficits.[16] Patients presenting with small brainstem or cerebellar strokes may have a low or even 0 NIHSS score, and careful vigilance should be employed to determine eligibility for acute treatment in this population.[17]

LABORATORY DATA

In the process of transitioning the patient with acute stroke from a focused neurologic examination to an initial head CT, the traditional approach is to obtain a quick blood draw for laboratory testing. Importantly, eligibility criteria for IV rtPA require platelet count greater than 100,000/μL, prothrombin time less than 15 seconds, and partial thromboplastin time within normal limits. However, the 2013 AHA/ASA guideline on the early management of patients with acute ischemic stroke suggests that only a finger-stick blood glucose is absolutely required before initiation of IV rtPA.[10] A prominent exception to this allowance applies for patients on warfarin or with known hematologic abnormalities, for whom pretreatment coagulation profile and complete blood cell count is necessary. Other laboratory tests, such as electrolytes, renal function, and troponins, are suggested in the acute stroke evaluation but should not delay the transition to head CT or treatment with IV rtPA. Additionally, obtaining vascular access for blood draws may delay the transition to head CT;

obtaining proper access during emergency transport and implementing point-of-care laboratory testing are potential methods to help reduce door-to-CT time.

INITIAL BRAIN IMAGING

The principal goal of initial brain imaging in the patient with acute stroke is to differentiate hemorrhagic versus ischemic stroke. Of the available modalities, noncontrast head CT is established as a rapidly obtained, highly sensitive, and widely available tool to rule out hemorrhage and inform treatment for acute stroke. Rapid brain MRI offers the additional advantage of being both highly sensitive and specific for ischemic stroke, particularly in cases of suspected stroke mimics,[18] and can adequately rule out hemorrhage on gradient recalled echo (GRE) or susceptibility-weighted imaging (SWI). While either imaging modality is supported in acute stroke guidelines,[10] the generalizability of rapid brain MRI, particularly for smaller and low-access hospitals, remains limited.

To the trained eye, the noncontrast head CT offers additional insights into the patient with acute stroke beyond simply ruling out hemorrhage. Similar to the history and examination, performing an efficient yet accurate assessment of the head CT requires a systematic approach, particularly for the nonradiologist neurologist reading the study at the bedside. For consistency, a top-down or bottom-up approach to all noncontrast head CTs might be utilized, reviewing each slice in sequence to gain a three-dimensional appreciation of the brain. An inside-out approach begins with reviewing brainstem and midline structures, then systematically scanning outward to the cortex through consecutive slices.

Important components of the acute stroke noncontrast head CT for the neurologist to note are listed in **Table 2-2**.[19] In addition to ruling out hemorrhage, important findings include a localizing dense artery sign suggestive of a large vessel occlusion, early ischemic changes suggestive of evolving infarct, or chronic infarcts informing a pattern or risk profile for stroke (**Case 2-1**).

The Alberta Stroke Program Early CT Score (ASPECTS) system is a simple and reliable 10-point scale for evaluating early ischemic changes in acute stroke[20] and is clinically relevant in the evaluation of eligibility for endovascular therapy as supported by recent clinical trial data and updated guidelines.[13,21] For more information on the ASPECTS system, refer to the article ''Treatment of Acute Ischemic Stroke'' by Alejandro A. Rabinstein, MD, FAAN,[7] in this issue of *Continuum*.

If CT findings are ambiguous, it is also helpful to search for any prior brain imaging for quick comparison. Observational studies suggest good interrater reliability between vascular neurologists and neuroradiologists in the interpretation of relevant CT findings in the patient with acute stroke.[22] Nonetheless, after initial treatment decisions are complete, following up with neuroradiology for official interpretation is essential to ensure nothing was missed on the acute read.

STROKE SYNDROMES

In some ways, defining acute stroke syndromes is educated pattern recognition influenced by knowledge and experience. As a seasoned vascular neurologist once put it, ''Does it smell like a stroke or not?'' In other words, does the presentation make neuroanatomic and cerebrovascular sense?

Inherent in this understanding of stroke syndromes is the caveat that stroke is not just one disease. Stroke is the downstream effect of a variety of different mechanisms, physiologic properties, and disease states. In this

KEY POINT

- Evidence-based guidelines suggest the only laboratory test absolutely required prior to initiation of IV recombinant tissue plasminogen activator is a finger-stick blood glucose. Other laboratory tests, such as complete blood cell count and metabolic panel, should not delay head CT or initiation of IV recombinant tissue plasminogen activator. Exceptions include patients taking warfarin or with known hematologic abnormalities, for whom rapid coagulation profiling is warranted. Obtaining proper vascular access in the emergency department may also delay door-to-CT time, so using prehospital access or point-of-care testing may be beneficial.

TABLE 2-2 Key Aspects of the Noncontrast Head CT in the Acute Stroke Evaluation[a]

Finding	Appearance	Significance
Acute hemorrhage	Hyperdensity compared to normal brain parenchyma consistent with blood products	Absolute contraindication to IV recombinant tissue plasminogen activator; requires immediate blood pressure management and often neurosurgical consultation
Early ischemic changes	Indistinct hypodensity with loss of gray-white differentiation, often at the core of infarction (eg, insular ribbon, basal ganglia)	Evidence of evolving infarct; not a contraindication to IV recombinant tissue plasminogen activator; however, extensive early ischemic changes are associated with poor outcome and increased risk of symptomatic hemorrhage
Chronic infarcts	Well-defined hypodensity or encephalomalacia in an arterial territory	Indicative of stroke risk and mechanism, which may inform acute diagnosis and treatment
Dense artery sign	Hyperdensity within an artery (eg, M1, basilar) compared to a normal isodense appearance	Associated with thrombotic large vessel occlusion; M2 dot sign may indicate more distal branch occlusion

CT = computed tomography; IV = intravenous.

Case 2-1

An independent 73-year-old right-handed man developed the sudden onset of confusion, slurred speech, and left-sided weakness. His wife called 911, and initial emergency medical services (EMS) assessment revealed abnormal speech, facial weakness, and left arm drift. He was immediately transported to the nearest primary stroke center, and the EMS crew notified the hospital of the last known well time and the estimated time of arrival. During transport, a point-of-care blood glucose was mildly elevated, IV access was obtained, and rapid ECG was consistent with atrial fibrillation (the patient was not on anticoagulation). A prehospital stroke alert was executed, and upon arrival to the emergency department, the patient was met by the acute stroke team and triaged directly to the CT scanner.

His initial blood pressure was 160/80 mm Hg, his heart rate was 90 beats/min (irregularly irregular), and his National Institutes of Health Stroke Scale score was 13, for disorientation, partial right gaze preference, left hemianopia, left lower facial weakness, dysarthria, left arm weakness, left leg drift, sensory loss, and extinction. Noncontrast head CT revealed a dense artery sign in the distal right M1 trunk and early ischemic changes in the right insular region.

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After reviewing his eligibility criteria for IV recombinant tissue plasminogen activator (rtPA), a premixed bolus and infusion were initiated on the CT table with a door-to-needle time of 19 minutes. The team immediately prepared for a CT angiogram to evaluate for large vessel occlusion and eligibility for endovascular therapy.

Comment. This case presents an ideal state for rapid evaluation of the patient with acute stroke leading to a timely treatment decision. Key elements included awareness of stroke symptoms prompting a call to 911, rapid prehospital management and hospital prenotification, early stroke team notification, premixing of IV rtPA, and direct triage to CT. His stroke presentation is consistent with likely cardioembolism secondary to atrial fibrillation not on anticoagulation. The CT findings of early ischemic changes and a dense artery sign are highly sensitive for a large vessel occlusion, suggesting potential eligibility for endovascular therapy following IV rtPA. His acute syndrome localizes to the right middle cerebral artery territory.

paradigm, stroke is indeed a clinical diagnosis, with cerebral infarction representing the tissue diagnosis and stroke subtype informing the mechanism.

These traditional delineations have been reevaluated in the age of advanced imaging, where stroke can be diagnosed by the presence of infarction on brain MRI (ie, restricted diffusion) even in the absence of a persistent stroke syndrome. However, according to a 2013 AHA/ASA statement, the definition of ischemic stroke still invokes ''focal cerebral, spinal, or retinal infarction'' in a ''defined vascular distribution.''[23]

Therefore, the ability to localize a defined vascular distribution in the patient with acute stroke remains the first tool in the black bag of the neurologist and fundamentally requires in-depth understanding of neuroanatomic stroke syndromes, as discussed here. Common large vessel stroke syndromes are listed in **Table 2-3**.[24]

Anterior Circulation Syndromes

The anterior circulation encompasses the distribution of the internal carotid artery and its major branches, the anterior cerebral artery (ACA) and middle cerebral artery (MCA). Anterior circulation ischemia accounts for the majority of all strokes.

Internal carotid artery. As the internal carotid artery (ICA) enters the cerebral circulation, it branches into the ACA and MCA. Occlusion of the ICA most often occurs secondary to atherosclerotic plaque and critical stenosis at the level of the cervical bifurcation or as a thromboembolic occlusion of the distal carotid, the so-called *carotid T lesion*. In the scenario of a carotid T lesion, the majority of the ipsilateral hemisphere becomes ischemic and will result in ACA/MCA territory infarction with contralateral hemiplegia unless rapid reperfusion can be established.

Symptomatic ICA stenosis at the cervical bifurcation often manifests as minor stroke or transient ischemic attack secondary to artery-to-artery embolism into the ipsilateral carotid territory. In addition to hemispheric signs and symptoms, carotid stenosis may be heralded by transient central retinal artery occlusion resulting in ipsilateral amaurosis fugax, or transient monocular blindness (the so-called ''shade coming down over the eye''),

TABLE 2-3 Large Vessel Stroke Syndromes (Laterality Assumes Left Hemispheric Dominance)[a,b]

Vascular Territory	Signs and Symptoms
Internal carotid artery	Combined anterior cerebral artery/middle cerebral artery syndromes; ipsilateral monocular visual loss secondary to transient central retinal artery occlusion (amaurosis fugax); branch retinal artery occlusions may present as ipsilesional altitudinal field cuts
Left anterior cerebral artery	Right leg numbness and weakness, transcortical motor aphasia, possibly ipsilesional or contralesional ideomotor apraxia
Right anterior cerebral artery	Left leg numbness and weakness, motor neglect, possibly ipsilesional or contralesional ideomotor apraxia
Left middle cerebral artery	Right face/arm numbness and weakness more than leg numbness and weakness, aphasia, left gaze preference
Right middle cerebral artery	Left face/arm numbness and weakness more than leg numbness and weakness, left hemispatial neglect, right gaze preference, agraphesthesia, astereognosis
Left posterior cerebral artery	Complete or partial right homonymous hemianopia, alexia without agraphia; if midbrain involvement, ipsilateral third nerve palsy with mydriasis and contralateral hemiparesis (Weber syndrome)
Right posterior cerebral artery	Complete or partial left homonymous hemianopia; if midbrain involvement, ipsilateral third nerve palsy with mydriasis and contralateral hemiparesis (Weber syndrome)
Superior cerebellar artery	Ipsilesional limb and gait ataxia
Anterior inferior cerebellar artery	Vertigo and ipsilesional deafness, possibly also ipsilesional facial weakness and ataxia
Vertebral/posterior inferior cerebellar artery	Ipsilesional limb and gait ataxia; if lateral medullary involvement, may have Wallenberg syndrome (refer to **Table 2-5**)
Basilar artery	Pontine localization with impaired lateral gaze, horizontal diplopia and dysconjugate gaze, nonlocalized hemiparesis, dysarthria; ''locked-in syndrome'' with bilateral pontine infarction (intact vertical eye movements, anarthria, quadriplegia)

[a] Data from Eckerle BJ, Southerland AM, Wiley-Blackwell.[24]
[b] The syndromes listed are reflective of classic neuroanatomy and may vary depending on individual variations in the circle of Willis or collateral vascular supply.

or more commonly as a nondescript visual disturbance. When encountered, this syndrome necessitates immediate imaging of the carotid bifurcation to rule out symptomatic carotid artery stenosis for which endarterectomy or stenting is warranted.

Middle cerebral artery. As the primary source of perfusion to the cerebral hemispheres, the MCA is the most commonly involved intracranial artery in the patient with acute stroke. A complete occlusion of the MCA trunk, commonly referred to as M1, often manifests with hemispheric signs and symptoms: aphasia (dominant hemisphere), hemispatial neglect (nondominant hemisphere), ipsilateral gaze preference (frontal eye fields), contralateral brachiofacial paralysis (ie, involving the face/arm more than the leg), and varying degrees of contralateral hemianesthesia. Strokes involving the frontal eye fields create a conjugate deviation of the eyes

to the ipsilateral ischemic hemisphere, ie, ''looking at your stroke.'' If MCA territory ischemia involves the optic radiations or extends to the occipital lobe, patients will also have a contralateral hemianopia. The most classic of the hemispheric stroke syndromes, and perhaps the easiest to recognize, involves the left MCA territory.

Dominant (left middle cerebral artery). The classic left MCA syndrome presents with left gaze preference, right visual field cut, aphasia, and right hemiparesis/hemianesthesia. Determining handedness in patients with stroke is key to defining hemispheric dominance and characterizing stroke syndromes. Only a minority of primarily left-handed individuals are right hemisphere dominant for language. As a majority of the population is left hemisphere dominant for language, the hallmark sign of a left MCA stroke is aphasia. Aphasia, or dysphasia, is best defined as any acquired abnormality of language (spoken, written, or signed) and is classically dichotomized as expressive (motor, nonfluent) or receptive (sensory, fluent). Expressive aphasia ranges from frank mutism to subtle word-finding difficulty. The latter can often be difficult to discern, especially for the non-neurologist evaluating a patient with acute stroke. The NIHSS aphasia cards are a quick and standardized tool to screen for aphasia at the bedside. For example, describing the scene in the cookie theft picture gives an overall impression of fluency, cadence, word volume, and accuracy. The patient who is mildly aphasic will often make paraphasic errors, which are key diagnostic clues in the bedside evaluation of the patient with acute stroke. Examples of paraphasic errors include phonemic (eg, kite instead of key) and semantic (eg, substituting hand for glove). Defining anomia or dysnomia, or the inability to name objects, is also an important hallmark of aphasia. Patients with subtle dysnomia may substitute a description of an object for the actual name, eg, ''that prickly thing'' rather than cactus, or ''that thing you swing in'' rather than hammock.

Receptive aphasia is characterized as an abnormality with language comprehension. Patients with mild receptive aphasias may do well with simple commands, such as ''open your eyes'' or ''close your fist,'' but struggle with complex or multistep commands, such as ''point to the ceiling with your thumb.'' In addition to deficits in language comprehension, neologisms (made-up words) or nonsensical language without impaired fluency are hallmarks of receptive aphasia.

Aphasia is often difficult to distinguish from nonfocal encephalopathy or delirium, particularly for the non-neurologist. A key difference between these two presentations is how attentive the patient seems. Patients with aphasia are often aware of their impairment and visibly frustrated, attempting to converse and follow commands. On the other hand, patients who are encephalopathic or delirious are often inattentive and unaware of their condition, in addition to lacking other focal signs or symptoms. Other features that may further distinguish aphasia and assist in localization in the patient with acute stroke are characterized in **Table 2-4** and **Figure 2-1**. Of additional note, mixed aphasia can result from subcortical strokes, such as so-called *thalamic aphasia*, which has variable features and may result in false localization to the dominant hemisphere perisylvian language areas.

Nondominant (right middle cerebral artery). As aphasia is the hallmark localizing sign of a left MCA or dominant hemisphere stroke, hemispatial neglect suggests injury to the nondominant hemisphere. Patients with

KEY POINTS

- Strokes involving the frontal eye fields create a conjugate deviation of the eyes to the ipsilateral ischemic hemisphere, ie, ''looking at your stroke.''
- The classic left middle cerebral artery syndrome presents with left gaze preference, right visual field cut, aphasia, and right hemiparesis/hemianesthesia.
- Determining handedness in patients with stroke is key to defining hemispheric dominance and characterizing stroke syndromes. Only a minority of primarily left-handed individuals are right hemisphere dominant for language.
- Listening intently for phonemic or semantic paraphasic errors is important to recognize subtle aphasia in the patient with acute stroke.
- Diagnosing aphasia from other forms of encephalopathy may be distinguished by a patient's level of attentiveness.

KEY POINT

- Mild hemispatial neglect from a right middle cerebral artery stroke can be elicited at the bedside by double simultaneous stimulation, during which the patient extinguishes the contralateral sensory stimulus.

TABLE 2-4 The Aphasias[a]

Type of Aphasia	Fluency	Comprehension	Repetition
Motor/expressive (Broca)	Impaired	Normal	Impaired
Sensory/receptive (Wernicke)	Normal	Impaired	Impaired
Conduction	Normal	Normal	Impaired
Transcortical motor	Impaired	Normal	Normal
Transcortical sensory	Normal	Impaired	Normal
Mixed	Variable	Variable	Variable
Global	Impaired	Impaired	Impaired

[a] Data from Eckerle BJ, Southerland AM, Wiley-Blackwell.[24]

a right MCA stroke can be difficult to diagnose without a directed neurologic examination, as the neglect syndrome often coexists with anosognosia, or unawareness of the deficit. In mild neglect syndromes, testing for extinction by double simultaneous stimulation at the bedside is an essential technique. Identifying tactile neglect is most common, but patients with neglect may exhibit auditory or visual extinction with double simultaneous stimulation as well, the latter being difficult to discern from a frank hemianopia. Motor neglect can also confound true hemiparesis, distinguished

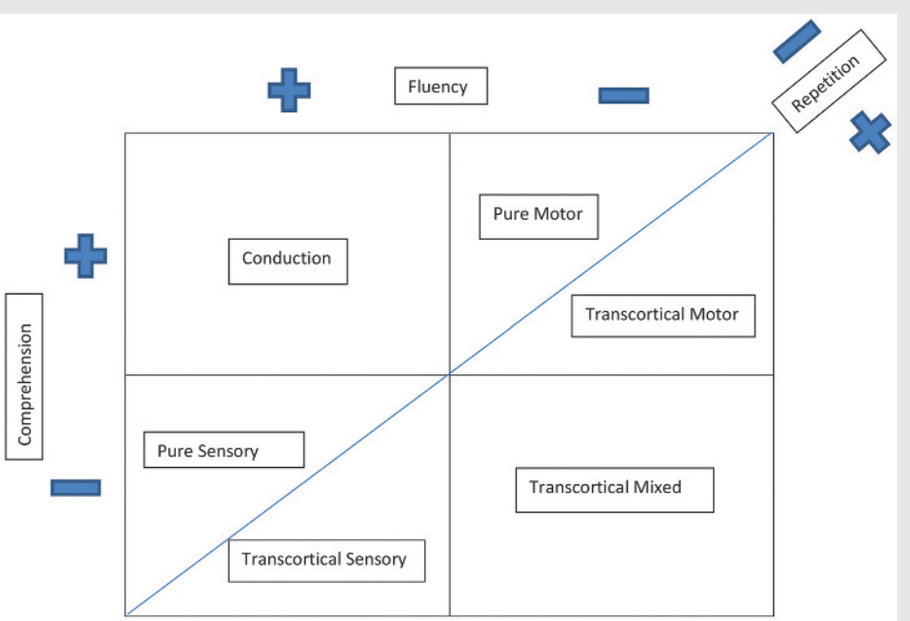

FIGURE 2-1 The graphic aphasia box. Plus symbols indicate intact components of language; minus symbols indicate impaired components of language.

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by checking tone or eliciting movement of the contralateral limb via noxious stimulus. Additional cortical sensory signs of a right MCA stroke include agraphesthesia, astereognosis, and loss of two-point discrimination. Testing for these signs at the bedside can uncover a focal nondominant hemispheric stroke secondary to branch right MCA territory occlusion. It may be difficult to gain consent for treatment from patients presenting with a nondominant hemisphere stroke because of their denial of their symptoms secondary to anosognosia (**Case 2-2**).

Anterior cerebral artery. Isolated ACA territory infarction represents a minority of stroke presentations, most often secondary to a thromboembolus or in situ stenosis. Because of the cortical representation of the motor homunculus, ACA territory strokes classically manifest with contralateral leg weakness more than arm weakness.

Case 2-2

A 77-year-old right-handed woman was at breakfast when she suddenly became confused and fell to the ground with convulsive seizure activity lasting several minutes. Witnesses reported her eyes and head deviated to the left during the episode. In the emergency department, her National Institutes of Health Stroke Scale score was 15, and her examination was notable for mild somnolence, a right gaze preference, decreased blink to threat on the left, and left hemiplegia of the arm and leg. When her arm was presented in her right visual field, she did not recognize it as her own. Head CT demonstrated no acute hemorrhage, with subtle early ischemic change in the right insula and a dense artery M1 sign on the right (**Figure 2-2**).

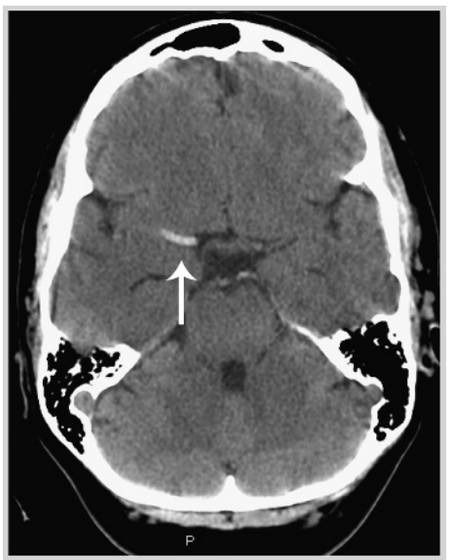

FIGURE 2-2 Head CT of patient in **Case 2-2** showing dense right M1 artery sign (*arrow*).

Continued on page 52

Continued from page 51

When discussing her eligibility for IV recombinant tissue plasminogen activator (rtPA) and endovascular therapy, she was not cognizant of her stroke and refused treatment. Unfortunately, no family member was available for assistance with surrogate decision making. However, when presented with a hypothetical scenario describing her situation if she were having a stroke, she was able to comprehend and endorsed treatment as the appropriate course. The acute stroke team proceeded with IV rtPA followed by CT angiography for eligibility of mechanical thrombectomy.

Comment. Updated American Heart Association/American Stroke Association stroke guidelines suggest that IV rtPA is ''reasonable in patients with a seizure at the time of onset of acute stroke if evidence suggests that residual impairments are secondary to stroke and not a postictal phenomenon (Class IIa; Level of Evidence C).''[12] In this case, the patient's seizure was the initial manifestation of acute ischemic stroke with a persistent right middle cerebral artery syndrome confirmed by a dense artery sign and early ischemic changes on head CT. Nondominant hemisphere strokes manifesting with anosognosia may present a challenging treatment dilemma in which the patient does not recognize his or her diagnosis. Worrall and colleagues[25,26] suggested that patients with anosognosia are still able to comprehend despite denying their diagnosis; therefore, an ''advance directive approach'' may allow them to consent as their own surrogate by presenting the treatment decision as a ''hypothetical'' discussion. If a patient is deemed to lack capacity and no surrogate is available, acute stroke treatment may proceed via emergency exemption.

ACA strokes may also result in abulia or apathy (lack of will) from ischemia to the anterior cingulate gyrus or caudate head (recurrent artery of Heubner). In addition to ischemic ACA stroke, this syndrome may also manifest from a ruptured aneurysm of the ACA or anterior communicating artery, and, in the extreme form, may result in latent akinetic mutism. A common anatomic variant, in which the right and left ACAs stem from a common A1 trunk, can result in infarction to bilateral ACA territories and present as bilateral leg weakness. As noted, infarction of the ACA territory most often results from carotid occlusion and in conjunction with infarction of the MCA territory.

Posterior Circulation Syndromes

According to AHA/ASA stroke statistics, approximately 20% of incident strokes per year involve the posterior circulation, equating to approximately 70,000 to 100,000 presentations in the United States each year.[27,28] Distinguishing between anterior and posterior circulation syndromes is essential to defining the likely source and mechanism of acute stroke and to guide secondary stroke prevention. Moreover, while compromise of the anterior circulation can produce larger hemispheric strokes, strokes involving the posterior circulation can be equally devastating when involving vital structures in the brainstem and cerebellum (**Table 2-5**).

Brainstem syndromes. Stroke syndromes in the brainstem can be trichotomized into the three anatomic levels: midbrain, pons, and medulla. The midbrain receives circulation from the top of the basilar artery and the posterior cerebral artery (PCA) and is often infarcted via intrinsic perforator (small vessel) occlusion, intrinsic disease in

TABLE 2-5 Common Brainstem Stroke Syndromes[a]

Syndrome	Signs/Symptoms	Localization	Vascular Supply
Weber	Ipsilesional cranial nerve III palsy, contralesional hemiparesis (including the lower face)	Medial midbrain/cerebral peduncle	Deep penetrating artery from posterior cerebral artery (refer to **Table 2-3**)
Benedikt	Ipsilesional cranial nerve III palsy, contralateral involuntary movements (intention tremor, hemichorea, or hemiathetosis)	Ventral midbrain involving red nucleus	Deep penetrating artery from posterior cerebral artery or paramedian penetrating branches of basilar artery
Nothnagel	Ipsilesional cranial nerve III palsy, contralesional dysmetria, and contralesional limb ataxia	Superior cerebellar peduncle	Deep penetrating artery from posterior cerebral artery
Foville	Ipsilateral cranial nerves VI and VII (lateral gaze palsy, upper and lower facial weakness), with or without contralateral hemiparesis	Caudal pontine tegmentum involving the facial colliculus	Pontine perforator branches off the basilar artery
One-and-a-half	Ipsilateral cranial nerve VI (lateral gaze) palsy, bilateral internuclear ophthalmoplegia	Paramedian pons involving the paramedian pontine reticular formation and medial longitudinal fasciculi	Paramedian pontine perforators off the basilar artery
Wallenberg	Ipsilesional facial and contralesional body hypalgesia and thermoanesthesia; ipsilesional palatal weakness; dysphagia, dysarthria, nystagmus, vertigo, nausea/vomiting; ipsilesional oculosympathetic defect (Horner syndrome); skew deviation, singultus	Lateral medulla	Posterior inferior cerebellar artery (should raise concern for disease in parent vertebral artery)
Dejerine	Ipsilesional tongue weakness and contralesional hemiparesis with or without contralesional loss of proprioception and vibratory sense	Medial medulla	Vertebral artery or anterior spinal artery

[a] Data from Eckerle BJ, Southerland AM, Wiley-Blackwell.[24]

the proximal PCA (large artery), or thromboembolus to the top of the basilar artery. The so-called *top of the basilar syndrome* stroke is the most devastating of these events in that it can involve infarction of bilateral midbrain and thalamic territories and cause subsequent injury to the reticular activating system, resulting in coma or death. Bilateral medial thalamic strokes result in a state of depressed level of consciousness or coma and often occur secondary to occlusion of a single thalamoperforator trunk off the top of the basilar artery, known as the artery of Percheron. Other midbrain stroke syndromes may involve oculomotor dysfunction via cranial nerve III

KEY POINTS

- Bilateral medial thalamic strokes result in a state of depressed level of consciousness or coma and often occur secondary to occlusion of a single thalamoperforator trunk off the top of the basilar artery, known as the artery of Percheron.
- When examining patients with stroke who are tetraplegic or appear to be comatose, the examiner must always ensure they are not actually ''locked in'' from bilateral pontine infarction and able to communicate with vertical eye movements or other subtle signs.
- Alexia without agraphia classically results from a left posterior cerebral artery territory stroke causing infarction of the splenium of the corpus callosum and left occipital lobe, leading to a disconnection of visual and language integration.

nuclei (third nerve palsy) or the medial longitudinal fasciculus (internuclear ophthalmoplegia), the corticospinal tracts in the cerebral peduncles (contralateral hemiparesis), cerebellar tracts in the superior cerebellar peduncles (ipsilateral ataxia), or rubral tracts via the red nuclei (ipsilateral tremor).

Similarly, pontine strokes are often the result of small vessel perforator occlusion or intrinsic large artery disease or thrombus in the midbasilar artery. Pontine syndromes often include dysarthria, contralateral hemiparesis via injury to descending corticospinal tracts in the ventral pons, hemianesthesia via ascending sensory tracts, ataxic hemiparesis via crossing cortico-pontocerebellar tracts, or horizontal gaze palsies via involvement of cranial nerve VI nucleus and the paramedian pontine reticular formation, the so-called *lateral gaze center*.

Possibly the most devastating stroke syndrome, locked-in syndrome, manifests from bilateral infarction in the ventral pons, disconnecting the midbrain and supratentorial structures from the rest of the lower nervous system, resulting in complete tetraplegia with a preserved level of consciousness. Importantly, with an intact midbrain, patients with locked-in syndrome can still communicate via blinking or vertical eye movements. Therefore, when examining patients with acute stroke who are apparently comatose, the examiner must always ensure they are not actually awake and able to communicate with eye movements or other subtle signs (**Case 2-3**).

Medullary stroke syndromes. The medullary stroke syndromes are divided by the medial and lateral medulla. The lateral medullary syndrome (Wallenberg syndrome) results from infarction in the posterior inferior cerebellar artery (PICA) territory, often from occlusion of the parent vertebral artery. Classically, lateral medullary stroke presents with components of dysphagia and dysphonia (nucleus ambiguus, vagal and glossopharyngeal nerves), vertigo and disequilibrium (vestibular nuclei), ipsilateral oculosympathetic Horner syndrome (ptosis/miosis, descending sympathetic tracts), ipsilateral face and contralateral body crossed sensory loss of pain and temperature sensation (trigeminal and spinothalamic tracts), and ipsilateral limb ataxia (spinocerebellar tract).

Alternatively, a medial medullary syndrome involves ventral structures in the lower brainstem with ipsilateral tongue weakness (hypoglossal nucleus) and contralateral hemiparesis (pyramidal tracts rostral to the decussation). Medial medullary strokes often occur from branch occlusion of the anterior spinal artery, with or without parent vertebral artery disease.

Posterior cerebral artery. Stroke involving the PCA territories is often the downstream result of thromboembolism to the posterior circulation or intrinsic atherosclerotic disease in the PCA. The typical circulation of the PCA supplies the midbrain, thalamus, and occipital and dorsomedial temporal lobe. The hallmark localizing sign of a PCA territory stroke is a contralateral homonymous hemianopia secondary to occipital lobe infarction, absent of hemiparesis or other hemispheric signs that would localize to the MCA territory. Hemianopias emanating from PCA stroke are said to be macular sparing, secondary to redundancy of macular representation in the occipital lobe. Another textbook syndrome involving the PCA territory is alexia without agraphia, which classically manifests from infarction to the splenium of the corpus callosum and left occipital lobe, resulting in a disconnection of visual and language integration.

Case 2-3

A 63-year-old man with multiple vascular risk factors was found down in his home and unable to communicate, prompting a call from his family to 911. On arrival of emergency medical services, he was deemed ''comatose'' and intubated in the field for airway protection. In the emergency department, his blood pressure was 180/100 mm Hg and his initial National Institutes of Health Stroke Scale score was 32; his neurologic examination (off sedation and paralytics) was notable for horizontal gaze palsy, bifacial paralysis, tetraplegia with bilateral extensor posturing, and hyperreflexia with bilateral Babinski signs and triple flexion. On further testing, the patient was consistently able to blink to command and deviate his gaze vertically. Head CT showed no acute changes but was significant for a dense basilar artery sign (**Figure 2-3**). The acute stroke team rapidly prepared for IV recombinant tissue plasminogen activator, followed by endovascular therapy.

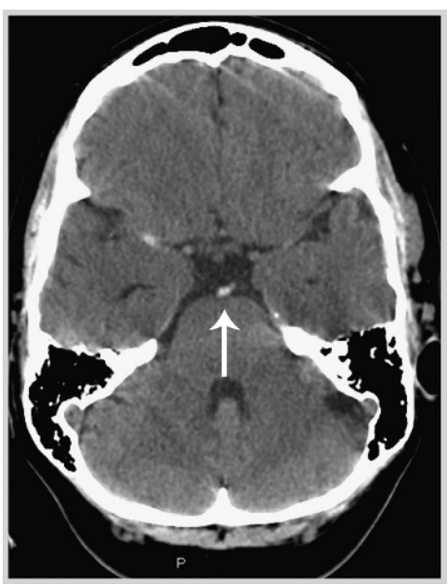

FIGURE 2-3 Head CT of the patient in **Case 2-3** showing dense basilar artery sign (*arrow*).

Comment. Locked-in syndrome is a devastating cerebrovascular condition manifesting from bilateral injury to the ventral pons, often secondary to basilar artery occlusion. With intact midbrain and supratentorial structures, patients are able to communicate via blinking or vertical eye movements and are able to consent for procedures and goals of care. In the acute stroke setting, locked-in syndrome is a neurovascular emergency and requires rapid efforts toward reperfusion therapy. Head CT may confirm the clinical suspicion by presence of a dense basilar sign indicating acute thrombotic occlusion. Acute stroke guidelines for basilar artery occlusions are limited in the evidence base. However, the devastating nature of the syndrome likely warrants early attempts at endovascular therapy and potentially considering treatment beyond standard stroke treatment time windows.

KEY POINTS

- Patients presenting with acute-onset dysequilibrium or gait ataxia should prompt a thorough examination of eye movements, postural stability, and gait to rule out a paramedian cerebellar stroke.
- The HINTS methodology (head impulse test, pattern of nystagmus, and test of skew) helps distinguish central from peripheral vestibulopathy in patients presenting with an acute vestibular syndrome.

Cerebellar arteries. Of the arteries supplying the cerebellum, the largest distribution stems from the PICA. PICA territory strokes often involve the lateral medulla, as described previously, or the ipsilateral cerebellar hemisphere. Cerebellar hemispheric strokes present with ipsilateral limb dysmetria (past-pointing), dyssynergia (incoordination) or intention tremor (widened amplitude at target), impaired check response (rebound with sudden limb deceleration), and dysdiadochokinesia (impaired rapid-alternating movements). Note, cerebellar stroke syndromes involving the paramedian or flocculonodular lobes may be less discrete, apparent only as midline signs of titubation (postural instability with sitting upright) or gait ataxia with ambulating. In this scenario, a patient with acute stroke may actually have an NIHSS score of 0, a reminder that the NIHSS is not a replacement for a thorough neurologic examination. Patients will often report listing to the ipsilateral side.

While less common than PICA territory stroke, occlusion of the anterior inferior cerebellar artery (AICA) results in a characteristic syndrome of sudden vertigo and ipsilateral sensorineural hearing loss. This occurs due to ischemia of the vestibulocochlear nerve and inner ear supplied by the labyrinthine artery. AICA strokes can also include ipsilateral upper and lower facial weakness (cranial nerve VII) due to involvement of the pontomedullary junction. The superior cerebellar artery supplies a smaller segment of the rostral cerebellar hemisphere, and occlusion, from intrinsic arterial disease or thromboembolus, also presents with ipsilateral cerebellar signs.

Acute vestibular syndrome. Between 2 million and 4 million emergency department visits each year are related to dizziness.[28,29] Dizziness or vertigo as a presenting symptom of stroke is a challenging clinical scenario, even for the most seasoned neurologist or stroke specialist, and can be difficult to discern from peripheral vestibular disorders in the absence of other localizing signs, eg, focal limb ataxia or neighborhood signs, such as dysarthria, diplopia, dysphagia, or dysphonia.

In these presentations, neuro-otologic bedside maneuvers can help distinguish central from peripheral causes of vertigo. One set of methods is termed *HINTS*, an acronym for head impulse test, pattern of nystagmus, and test for skew. If the head impulse test is normal, the fast phase of nystagmus changes direction, or a skew deviation is present, it suggests a central process such as posterior circulation stroke with greater than 90% sensitivity.[30] In a 2015 study of patients presenting with acute vertigo and either nystagmus or imbalance, this testing helped risk stratify patients who went on to have a confirmed stroke.[31] Additionally, these bedside tests may actually have greater sensitivity than brain MRI for diagnosing small brainstem and cerebellar strokes in the hyperacute setting[32] and therefore are valuable clinical tools to help guide inpatient versus outpatient management of patients presenting with acute dizziness. Still, these bedside otologic maneuvers may not capture all central processes or causes of stroke in vestibular syndromes and should be considered supplemental to a dedicated history and thorough neurologic examination. **Table 2-6** lists red flags that should heighten concern for stroke in the differential diagnosis for patients presenting with acute vestibular syndrome.

Lacunar (Small Vessel) Syndromes

Distinguishing between large and small vessel syndromes in acute stroke has taken on heightened relevance with the recent evidence in favor of

TABLE 2-6 Red Flags Concerning for a Central Process in Patients Presenting With Acute Vestibular Syndrome

- Four Ds: diplopia, dysarthria, dysphagia, dysphonia
- Vertical or direction-changing nystagmus
- Skew deviation
- Normal head-impulse test
- Sudden deafness or tinnitus
- Focal signs (eg, dysmetria, dyssynergia, dysdiadochokinesia)
- Acute neck/occipital pain (ie, rule out vertebral artery dissection)
- The high-risk patient (eg, older age, vascular risk factors, history of stroke or coronary artery disease)

endovascular therapy for patients with large vessel occlusion. Small vessel (ie, lacunar syndromes) indicate occlusion of perforating arteries in the subcortex, brainstem, or cerebellum, often associated with chronic hypertension and diabetes mellitus (Table 2-7). Lacunar infarcts may be clinically silent, as seen on head CT, or result in stroke syndromes involving densely packed white matter tracts with specific localization patterns. The most commonly described small vessel syndromes include pure motor, pure sensory, sensorimotor, ataxic hemiparesis, and the so-called *dysarthria-clumsy hand syndrome*. The various subcortical localizations of these syndromes include the internal capsule, thalamus, basal ganglia, pons, cerebellum, and subcortical white matter (corona radiata). A less common, but striking, small vessel syndrome manifests as hemiballism from infarction in the subthalamic nucleus.

Spinal Vascular Syndromes

Like the brain, the spinal cord has a robust collateral blood supply. The anterior and posterior spinal arteries supply the ventral and dorsal cord, respectively, as branches of the extradural vertebral artery. Strokes referable to the anterior spinal artery can be isolated to penetrating branch occlusions (eg, medial medullary syndrome) or more completely involve the anterior two-thirds of the spinal cord (anterior spinal syndrome), sparing only the sensory tracts of the dorsal columns.

Ischemic vulnerability in the spinal cord is greatest in the midthoracic sections, where a watershed exists between the anterior spinal artery and more robust radicular arteries supplying the lumbosacral enlargement (eg, the artery of Adamkiewicz). In this scenario, a patient often presents with sudden neck or back pain, followed by paraplegia and a spinal sensory loss of pain and temperature below the level of infarction. Spinal shock initially appears as flaccidity and hyporeflexia, followed within days by upper motor neuron signs of spastic paraplegia and hyperreflexia. In addition to atherosclerosis and aortic dissection as common etiologies, anterior cord syndromes can also result iatrogenically during aortic surgery.

STROKE MIMICS

In the clinical evaluation of the patient with acute stroke, the ability to rule

TABLE 2-7 Lacunar Syndromes[a]

Syndrome	Signs/Symptoms	Localization	Vascular Supply
Pure motor	Contralesional hemiparesis	Posterior limb of internal capsule, corona radiata, or basis pontis	Lenticulostriate branches of the middle cerebral artery or perforating arteries from the basilar artery
Pure sensory	Contralesional hemisensory loss	Ventroposterolateral nucleus of the thalamus	Lenticulostriate branches of the middle cerebral artery or small thalamoperforators from the posterior cerebral artery
Sensorimotor	Contralesional weakness and numbness	Thalamus and adjacent posterior limb of internal capsule	Lenticulostriate branches from the middle cerebral artery
Dysarthria-clumsy hand	Slurred speech and (typically fine motor) weakness of contralesional hand	Base of the pons between rostral one-third and caudal two-thirds	Perforating arteries from the basilar artery
Ataxic hemiparesis	Contralesional (mild to moderate) hemiparesis and limb ataxia out of proportion to the degree of weakness	Posterior limb of internal capsule or base of the pons	Lenticulostriate branches of the middle cerebral artery or perforating arteries from the basilar artery
Hemiballismus/hemichorea	Contralesional limb flailing or dyskinesia	Subthalamic nucleus	Perforating lenticulostriate arteries

[a] Data from Eckerle BJ, Southerland AM, Wiley-Blackwell.[24]

out stroke holds equal importance to the ability to rule it in. Many stroke mimics, or chameleons, exist that challenge clinical diagnostic reasoning.[33] **Table 2-8** lists some common stroke mimics. With this challenge, one must rely on a targeted history, neurologic examination, and fundamental understanding of cerebrovascular localization to effectively limit the excessive treatment of false positives or neglected treatment of false negatives. Further, the time-sensitive nature of treatment for acute ischemic stroke does not always allow for a final diagnosis prior to making a treatment decision.

Mimics often present with vague symptoms and neurologic syndromes lacking in defined localization. Some common conundrums when discerning stroke from stroke mimics include multiple localizations (eg, embolic stroke), encephalopathy versus aphasia, postictal syndromes, peripheral neuromuscular disorders or myelopathy with lateralized weakness, complicated migraine aura, and sophisticated somatization disorders. Nonetheless, the risk of treating patients with stroke mimics is very low, and absolute certainty cannot be the threshold to make a vital treatment decision in a time-sensitive setting.[34]

CONCLUSION

The clinical evaluation of the patient with acute stroke is clearly a dynamic process, requiring mastery in the

TABLE 2-8 Common Stroke Mimics[a]

Mimic	Clinical Aspects Differentiating From Stroke
Seizures	Witnessed seizure activity,[b] postictal period (eg, Todd paresis), direction of gaze preference (eyes gaze away from seizure during ictus), history of seizures
Metabolic encephalopathy[c]	Hypoglycemia, electrolyte abnormalities (eg, hypernatremia), hepatic or renal encephalopathy
Toxic encephalopathy[c]	Alcohol or illicit drug exposure, neuro-active or sedating medications
Infectious encephalopathy[c]	Urinary tract infection, sepsis, meningitis/encephalitis, brain abscess
Brain tumor	Gradual onset of symptoms, systemic malaise, may present with seizure at onset
Neuromuscular	Focal weakness or numbness localizing to a spinal level, root, plexus, or peripheral nerve; may be associated with exacerbating trauma or limb compression
Migraine with aura	Gradual onset, preceding aura, throbbing headache with migrainous features, history of stereotypical episodes
Psychogenic	Lack of objective signs, inconsistent examination, neurologic symptoms in a nonvascular distribution, history of similar events

[a] Data from Eckerle BJ, Southerland AM, Wiley-Blackwell.[24]
[b] Embolic cerebral infarcts may manifest with seizure activity as a presenting symptom and should be considered in the differential of new seizure presentation, particularly in patients who are older.
[c] Encephalopathy is often differentiated from stroke by a more gradual and less discrete onset of symptoms. Note that patients with a history of stroke may present with a reactivation syndrome mimicking prior stroke symptoms in the setting of toxic/metabolic/infectious encephalopathy.

focused stroke history, neurologic examination, and diagnostic assessment, all while working through a time-sensitive and team-based environment. Arriving at a correct and timely treatment decision not only warrants proficiency with acute stroke protocol but also requires knowledge of neurovascular anatomy, clinical stroke syndromes, and common mimics (ie, does the case make neuroanatomic and cerebrovascular sense?). With the rapid evolution of acute stroke therapies and advanced diagnostics, the fundamentals of the bedside assessment must continue to be promoted as the foundation of acute stroke care. "Be quick, but don't hurry."

USEFUL WEBSITES

National Institutes of Health Stroke Scale

commondataelements.ninds.nih.gov/Doc/NOC/NIH_Stroke_Scale_NOC_Public_Domain.pdf

National Institutes of Health Stroke Scale Certification Training

nihstrokescale.org

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