

**American College of Radiology
ACR Appropriateness Criteria®
Cerebrovascular Disease**

Variant 1: Asymptomatic. Structural lesion on physical examination (cervical bruit) and/or risk factors.

Radiologic Procedure	Rating	Comments	RRL*
US duplex Doppler carotid	8	If positive, consider follow-up with CTA or CE-MRA.	○
MRA neck without IV contrast	8	If positive, consider follow-up with CTA or CE-MRA.	○
MRA neck without and with IV contrast	8	CTA and CE-MRA are comparable noninvasive imaging alternatives each with their own advantages and disadvantages.	○
CTA neck with IV contrast	8	CTA and CE-MRA are comparable noninvasive imaging alternatives each with their own advantages and disadvantages.	☼☼☼
CT head perfusion with IV contrast	5		☼☼☼
MRI head perfusion with IV contrast	5		○
MRI head without IV contrast	5		○
MRI head without and with IV contrast	5		○
CT head without IV contrast	5		☼☼☼
CT head with IV contrast	3		☼☼☼
CT head without and with IV contrast	3		☼☼☼
MRA head without IV contrast	3		○
MRA head without and with IV contrast	3		○
CTA head with IV contrast	3		☼☼☼
Arteriography cervicocerebral	2		☼☼☼
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Variant 2:**Carotid territory or vertebrobasilar TIA, initial screening survey.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	9	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered.	○
MRI head without IV contrast	8	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered. Can be useful if there is a contraindication to contrast. MRI is more sensitive than CT for acute infarct.	○
MRA head and neck without IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA. Can be useful if there is a contraindication to contrast.	○
MRA head and neck without and with IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA.	○
CT head without IV contrast	8	Useful to evaluate for acute intracranial pathology. MRI is more sensitive than CT for acute infarct.	☼☼☼
CTA head and neck with IV contrast	8	CTA can be obtained after NCCT.	☼☼☼
US duplex Doppler carotid	5		○
CT head perfusion with IV contrast	5		☼☼☼
MRI head perfusion with IV contrast	5		○
CT head with IV contrast	3		☼☼☼
CT head without and with IV contrast	3		☼☼☼
Arteriography neck	3		☼☼☼
Arteriography cervicocerebral	3		☼☼☼
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Variant 3:**New focal neurologic defect, fixed or worsening. Less than 6 hours. Suspected stroke.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without IV contrast	9	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered. Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. MRI is more sensitive than CT for acute infarct.	☼☼☼
MRI head without IV contrast	8	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered. Can be useful if there is a contraindication to contrast. Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. MRI is more sensitive than CT for acute infarct.	○
MRI head without and with IV contrast	8	Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. MRI head with contrast can be helpful to determine the age of infarct and to evaluate for other causes of symptoms such as tumor or infection.	○
MRA head and neck without IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA. Can be useful in patients with renal failure or contrast allergies.	○
MRA head and neck without and with IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA.	○
CTA head and neck with IV contrast	8	CTA can be obtained after NCCT.	☼☼☼
CT head perfusion with IV contrast	6		☼☼☼
MRI head perfusion with IV contrast	5		○
Arteriography cervicocerebral	5		☼☼☼
CT head with IV contrast	3		☼☼☼
CT head without and with IV contrast	3		☼☼☼
US duplex Doppler carotid	2		○
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Variant 4:**New focal neurologic defect, fixed or worsening. Longer than 6 hours. Suspected stroke.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without IV contrast	8	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered. Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. Can be useful if there is a contraindication to contrast. MRI is more sensitive than CT for acute infarct.	○
MRI head without and with IV contrast	8	Parenchymal brain imaging and CT or MR vascular imaging of the head and neck should be considered. Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. MRI is more sensitive than CT for acute infarct.	○
MRA head and neck without IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA. May be useful in patients with renal failure or contrast allergies.	○
MRA head and neck without and with IV contrast	8	Can be obtained in conjunction with MRI head. Preferred MR vascular imaging of the head and neck includes noncontrast head MRA and contrast-enhanced neck MRA.	○
CT head without IV contrast	8	Noncontrast head CT is often obtained first to assess for hemorrhage or large infarct. MRI is more sensitive than CT for acute infarct.	☼☼☼
CTA head and neck with IV contrast	8	CTA can be obtained after NCCT.	☼☼☼
Arteriography cervicocerebral	6		☼☼☼
CT head perfusion with IV contrast	5		☼☼☼
MRI head perfusion with IV contrast	5		○
CT head with IV contrast	3		☼☼☼
CT head without and with IV contrast	3		☼☼☼
US duplex Doppler carotid	2		○
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Variant 5:**Proven parenchymal hemorrhage (hematoma).**

Radiologic Procedure	Rating	Comments	RRL*
MRI head without and with IV contrast	9	Parenchymal imaging and CT or MR vascular brain imaging should be considered. MRI preferred over CT due to greater range of soft-tissue contrast and superior anatomic detail. Contrast is useful to evaluate for underlying enhancing mass or vascular malformation.	○
MRI head without IV contrast	8	Parenchymal imaging and MR or CT vascular brain imaging should be considered. MRI preferred over CT due to greater range of soft-tissue contrast and superior anatomic detail. Can be useful in patients with contraindications to contrast.	○
MRA head without IV contrast	8	Can be obtained in conjunction with MRI head. Useful to evaluate for underlying vascular malformation.	○
MRA head without and with IV contrast	8	Can be obtained in conjunction with MRI head. Useful to evaluate for underlying vascular malformation.	○
CT head without IV contrast	8	Useful to follow-up hemorrhage evolution and evaluate for complications.	☼☼☼
CTA head with IV contrast	8	Can be obtained after NCCT while the patient is still on the CT scan table to evaluate for underlying vascular malformation.	☼☼☼
MR venography head without IV contrast	7	Can be obtained in conjunction with MRI head. Consider MRV if there is concern for venous thrombosis or vascular malformation.	○
MR venography head without and with IV contrast	7	Can be obtained in conjunction with MRI head. Consider MRV if there is concern for venous thrombosis or vascular malformation.	○
CT venography head with IV contrast	7	Consider CTV if there is concern for venous thrombosis or vascular malformation. CTV can be obtained following NCCT.	☼☼☼
MRA head and neck without and with IV contrast	6		○
Arteriography cervicocerebral	6		☼☼☼
CT head without and with IV contrast	6		☼☼☼
CT venography head and neck with IV contrast	5		☼☼☼
MRA head and neck without IV contrast	5		○
CTA head and neck with IV contrast	5		☼☼☼
CT head with IV contrast	4		☼☼☼
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level1

Variant 6:**Suspected dural venous sinus thrombosis.**

Radiologic Procedure	Rating	Comments	RRL*
MR venography head without and with IV contrast	9	Parenchymal imaging and vascular brain imaging with CT or MR should be considered. With contrast is preferred over MRV without contrast.	○
MR venography head without IV contrast	8	Parenchymal imaging and vascular brain imaging with CT or MR should be considered. Can be useful in the patient with a contraindication to contrast.	○
CT venography head with IV contrast	8	CTV can be obtained while the patient is still on the CT scan table after NCCT and can be obtained rapidly in the emergent setting. Postcontrast image timing can be optimized for evaluation of the intracranial venous structures.	☼☼☼
MR venography head and neck without and with IV contrast	8	Can be obtained in conjunction with MRI head. MRV without and with contrast is superior to MRV without contrast due to problems noncontrast MRV has with slow and turbulent flow. Neck MRV can be useful to evaluate involvement of the neck vessels.	○
CT head without and with IV contrast	7	Can be useful if there is contraindication to MRI. Head CTV with contrast provides superior evaluation of the intracranial venous structures.	☼☼☼
CT head without IV contrast	7	Useful in initial evaluation of symptoms and in follow-up.	☼☼☼
MRI head without and with IV contrast	7	Useful to evaluate for complications of CVT including infarct and hemorrhage; to visualize thrombus; to determine the age of infarct; and to evaluate for other pathologies.	○
MRI head without IV contrast	7	Can be useful if there is a contraindication to contrast. Can evaluate for CVT complications including infarct and hemorrhage and to visualize thrombus.	○
CT venography head and neck with IV contrast	7	CTV can be obtained while the patient is still on the CT scan table after NCCT. Postcontrast image timing can be optimized for evaluation of the venous structures. Can be useful to evaluate involvement of the neck vessels.	☼☼☼
CT head with IV contrast	6		☼☼☼
MR venography head and neck without IV contrast	6		○
Catheter venography cervicocerebral	5		☼☼☼
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

CEREBROVASCULAR DISEASE

Expert Panel on Neurologic Imaging: Michael B. Salmela, MD¹; Shabnam Mortazavi, MD²; Bharathi D. Jagadeesan, MD³; Daniel F. Broderick, MD⁴; Judah Burns, MD⁵; Tejaswini K. Deshmukh, MB, MS⁶; H. Benjamin Harvey, MD, JD⁷; Jenny Hoang, MBBS⁸; Christopher H. Hunt, MD⁹; Tabassum A. Kennedy, MD¹⁰; Alexander A. Khalessi, MD, MS¹¹; William Mack, MD¹²; Nandini D. Patel, MD¹³; Joel S. Perlmutter, MD¹⁴; Bruno Policeni, MD¹⁵; Jason W. Schroeder, MD^{16*}; Gavin Setzen, MD¹⁷; Matthew T. Whitehead, MD¹⁸; Rebecca S. Cornelius, MD¹⁹; Amanda S. Corey, MD.²⁰

The prior version of this document included variants/clinical scenarios covering a broader scope of Cerebrovascular Disease. The variants related to aneurysm, vascular malformations, and subarachnoid hemorrhage were removed and are now covered in the new ACR Appropriateness Criteria[®] topic on “[Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage](#)”. An updated document on stroke and stroke related conditions will be available soon.

Summary of Literature Review

Introduction/Background

Diseases of the cerebral vasculature represent a heterogeneous group of ischemic and hemorrhagic etiologies, which often manifest clinically as an acute neurologic deficit also known as stroke or less commonly with symptoms such as headache or seizures. Stroke is the fourth leading cause of death behind heart disease, cancer, and chronic lower respiratory disease and is a leading cause of serious long-term disability in the United States. Approximately 795,000 people in the United States experience a new or recurrent stroke each year, with 610,000 being their first stroke and 185,000 being recurrent strokes. Eighty-seven percent of strokes are ischemic, 10% are due to intracerebral hemorrhage (ICH), and 3% are secondary to subarachnoid hemorrhage (SAH) [1].

The last 2 decades have seen significant developments in the screening, diagnosis, and treatment of ischemic and hemorrhagic causes of stroke with advancements in computed tomography (CT) and magnetic resonance imaging (MRI) technology, changes in stroke imaging algorithms, and novel treatment devices and techniques such as intravenous tissue plasminogen activator (IV-tPA), carotid artery stenting, intra-arterial therapy (IAT) of ischemic stroke, and endovascular embolization of vascular malformations. These advancements have helped achieve decreases in the annual stroke death rate and number of stroke deaths by 35.8% and 22.8%, respectively, from 2000–2010 [1]. Over the past 2 years multiple randomized controlled trials have conclusively demonstrated the superiority of mechanical thrombectomy when compared to IV-tPA in the treatment of acute ischemic stroke from large vessel occlusive disease [2-5].

Overview of Imaging Modalities

Multiple different imaging modalities can be utilized in the evaluation of cerebrovascular disease, including ultrasound (US), CT, CT angiography (CTA), CT venography (CTV), CT perfusion (CTP), MRI, MR angiography (MRA), MR venography (MRV), MR perfusion, and digital subtraction angiography (DSA). The different imaging modalities all have their own niches in the evaluation of cerebrovascular disease and their own advantages and disadvantages.

¹Research Author, University of Minnesota, Minneapolis, Minnesota. ²Research Author, University of Minnesota, Minneapolis, Minnesota. ³Principal Author, University of Minnesota, Minneapolis, Minnesota. ⁴Mayo Clinic Florida, Jacksonville, Jacksonville, Florida. ⁵Montefiore Medical Center, Bronx, New York. ⁶Children’s Hospital of Wisconsin, Milwaukee, Wisconsin. ⁷Massachusetts General Hospital, Boston, Massachusetts. ⁸Duke University Medical Center, Durham, North Carolina. ⁹Mayo Clinic, Rochester, Minnesota. ¹⁰University of Wisconsin Hospital and Clinic, Madison, Wisconsin. ¹¹University of California San Diego Medical Center, San Diego, California, neurosurgical consultant. ¹²University of Southern California, Los Angeles, California, neurosurgical consultant. ¹³Fairfax Radiology Consultants PC, Fairfax, Virginia. ¹⁴Washington University School of Medicine, Saint Louis, Missouri, American Academy of Neurology. ¹⁵University of Iowa Hospitals and Clinics, Iowa City, Iowa. ¹⁶Walter Reed National Military Medical Center, Bethesda, Maryland. ¹⁷Albany ENT & Allergy Services PC, Albany, New York, American Academy of Otolaryngology-Head and Neck Surgery. ¹⁸Children’s National Medical Center, Washington, District of Columbia. ¹⁹Specialty Chair, University of Cincinnati Medical Center, Cincinnati, Ohio. ²⁰Panel Chair, Emory University, Atlanta, Georgia.

The American College of Radiology seeks and encourages collaboration with other organizations on the development of the ACR Appropriateness Criteria through society representation on expert panels. Participation by representatives from collaborating societies on the expert panel does not necessarily imply individual or society endorsement of the final document.

*The views expressed in this manuscript are those of the author and do not reflect the official policy of the Department of Army/Navy/Air Force, Department of Defense, or United States Government.

Reprint requests to: publications@acr.org

Ultrasound

US is commonly used in screening for carotid artery stenosis and to evaluate for cerebral artery vasospasm after SAH. Advantages of US include its lack of ionizing radiation, low cost, and ability to be performed at the patient's bedside. Disadvantages of US include heavy operator dependence and occasional difficulty in obtaining an adequate acoustic window to visualize the area of interest. Additionally, vascular US is limited in the evaluation of the proximal common carotid and distal internal carotid arteries (ICAs), tandem vascular lesions, near occlusion, and heavily calcified vessels.

Computed Tomography

CT technology continues to improve since its introduction in the early 1970s with improved radiation dose reduction techniques such as iterative reconstruction and tube current modulation as well as reduced scan times secondary to multislice CT technology. CT is used across the spectrum of cerebrovascular diseases. Noncontrast CT (NCCT) of the head is the first-line imaging test to evaluate for intracranial hemorrhage in patients presenting with acute stroke, whether acute ischemic stroke (AIS) or hemorrhagic stroke. CT is also very sensitive in detecting calcification, which can be important in lesion characterization. Contrast-enhanced CT of the head is not commonly used but may be utilized when contrast-enhanced brain MRI cannot be performed. MRI is preferred for evaluation of the brain parenchyma because of the superior soft-tissue contrast resolution of MRI compared to CT.

CTA is versatile and can be used to evaluate for stenosis, occlusion, dissection, vasculitis, aneurysm, or nonaneurysmal vascular malformation in the vessels of the head and neck. CTV can be used in the evaluation of venous sinus thrombosis and vascular malformations. CTP can be used to evaluate cerebrovascular reserve; however, its role in the evaluation of acute stroke remains unproven because of sensitivity to motion, low signal-to-noise ratio, and variation among software packages. CTA, CTV, and CTP all require the use of intravenous (IV) iodinated contrast, which may not be feasible in patients with renal dysfunction or contrast allergy.

Advantages of CT include rapid image acquisition with low susceptibility to motion, ready availability, and relatively high spatial resolution. However, disadvantages of CT include exposing the patient to ionizing radiation and, with the use of IV iodinated contrast material, the risk of contrast allergy and contrast-induced nephropathy.

Magnetic Resonance Imaging

MRI offers superb, unmatched soft-tissue contrast resolution, and it is widely used in the evaluation of cerebrovascular disease. MRI of the brain is usually performed without and with gadolinium-based IV contrast in the evaluation of acute stroke and transient ischemic attack (TIA), and in further evaluation of hemorrhagic brain lesions. A limited noncontrast brain MRI can be performed in the acute stroke patient with diffusion-weighted imaging (DWI), apparent diffusion coefficient (ADC) map, and fluid-attenuated inversion recovery (FLAIR) to evaluate for infarct. Currently, the most common clinical MRI scanners have field strengths of 1.5T and 3T. Increasing field strength leads to increased signal-to-noise ratio, increased susceptibility effects, and increased chemical shift artifact. MRI may be unable to be performed in patients with claustrophobia, morbid obesity, or presence of cardiac device or metal within the patient. Caution should be used when administering IV gadolinium contrast in patients with renal dysfunction or contrast allergy. The American College of Radiology (ACR) documents on MRI safety and contrast media provide more detailed safety information [6,7].

MRA of the brain or cervical arteries can be performed without or with contrast, depending on the clinical scenario. Noncontrast MRA techniques include time-of-flight MRA (TOF-MRA), phase-contrast MRA (PC-MRA), and blood oxygenation level-dependent (BOLD) MRV or susceptibility-weighted imaging (SWI). TOF-MRA relies on flow-related enhancement and 3-D TOF-MRA offers excellent spatial resolution. It is commonly used in imaging the circle of Willis in acute stroke. However, TOF-MRA has difficulty evaluating slow and turbulent flow and has poor suppression of background short T1 signal from thrombus. PC-MRA utilizes the difference in phase between moving and stationary blood and offers information on the direction and velocity of moving blood. Additionally, PC-MRA has advantages over TOF-MRA in that PC-MRA is able to evaluate slow flow well and is able to differentiate thrombus with short T1 signal from moving blood. This makes PC-MRA a good candidate for evaluating dural venous sinus thrombosis. However, a drawback of PC-MRA is its long imaging acquisition time. Contrast-enhanced MRA (CE-MRA) utilizes the T1-shortening effects of gadolinium-based IV contrast agents and is less susceptible to image degradation due to slow and turbulent flow. CE-MRA is commonly used in the evaluation of the cervical vessels in the workup of acute stroke and TIA. CE-MRA is also useful in the evaluation of cerebral vascular malformations such as arteriovenous malformations (AVMs) and

dural arteriovenous fistulae (AVFs). Recent advances in 4-D MRA with time-of-arrival maps as well as the utilization of arterial spin labeling techniques shows promise in noninvasive characterization of AVMs and dural AVFs [8-12]. Arterial spin labeling with selective arterial excitation shows great promise, but is not yet mainstream when it comes to the detection and characterization of AVMs [13]. MRV can be used in the evaluation of cerebral venous thrombosis (CVT) and vascular malformations and can be performed without or with IV contrast, similar to MRA, with techniques including TOF, PC, BOLD or SWI, and contrast-enhanced MRV. MR perfusion imaging is similar to CTP and its utility also remains questionable in the evaluation of acute stroke due to sensitivity in patient motion as well as low signal-to-noise ratio.

Digital Subtraction Angiography

DSA remains the gold standard imaging test to evaluate for carotid artery stenosis, cerebral artery aneurysms, vasculitis, and high-flow cerebral vascular malformations. However, DSA is an invasive imaging test with potential complications and is therefore usually not a first-line imaging test except perhaps in patients with acute SAH. However, it remains an integral part in the evaluation and management of patients with cerebrovascular malformations and aneurysms.

Stroke Overview

Transient Ischemic Attack

TIA has traditionally been described as a focal neurologic deficit that resolves within 24 hours. However, up to 50% of patients with the traditional definition of TIA based on the duration of symptoms were found to have infarcts on DWI [14-16]. Subsequently, in 2009 the American Stroke Association proposed a tissue-based definition of TIA as “a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction” [14]. TIA patients are at high risk for future stroke. TIA patients have a 5.3% chance of stroke within 48 hours, 21% of which are fatal [17]. Up to 20% of TIA patients suffer a stroke within 3 months [18-20]. Additionally, the 10-year risk for suffering a stroke, myocardial infarction, or death in a TIA patient is as high as 43% [21,22]. Because of the imminent high risk of future stroke associated with TIA, imaging workup of TIA should be performed as soon as possible.

Stroke

Stroke is a lay term used to describe a sudden-onset neurologic deficit and can be categorized into ischemic and hemorrhagic etiologies.

Ischemic Stroke

Ischemic stroke is responsible for 87% of all strokes and imaging plays a key role in its management [1]. Causes of ischemic stroke include thrombi, emboli, and hypoperfusion (watershed infarcts). The main imaging modalities used in the evaluation of ischemic stroke include head NCCT, CTA of the head and neck, brain MRI, and MRA of the head and neck. The clinical scenario and time since symptom onset determines which imaging modalities are to be used in the workup of ischemic stroke and its underlying causes. Screening for carotid artery stenosis can be performed with duplex US, MRA, or CTA depending on the scenario. Treatment of ischemic stroke has dramatically changed over the last 20 years. In 1996 the U.S. Food and Drug Administration (FDA) approved the use of IV-tPA for thrombolytic therapy of AIS. Subsequently, mechanical thrombectomy/thromboaspiration has also developed.

Treatment of Ischemic Stroke

The goal of ischemic stroke treatment is the salvage of ischemic but yet-to-be-infarcted penumbral brain tissue that surrounds an infarct core. The current FDA-approved treatment for AIS symptoms is IV-tPA within 3 hours of symptom onset, and the recommended initial imaging study is an NCCT to exclude intracranial hemorrhage. However, in 2009 the American Heart Association approved the use of IV-tPA up to 4.5 hours from symptom onset [23]. Interestingly, studies have found that many ischemic stroke patients have large ischemic penumbras up to 24 hours after symptom onset, suggesting the possibility of salvageable tissue at up to 24 hours and the potential for extension of the treatment window beyond 4.5 hours [24,25].

Significant improvements in IAT for AIS have occurred since 1999, with improved revascularization rates now up to 90% or more with current techniques, as opposed to 50% a decade earlier [26-29]. Despite these remarkable developments in IAT, the evidence from 3 large trials, namely, IMS III, SYNTHESIS Expansion, and MR RESCUE [30-32], had led to renewed concerns regarding the additive benefits of IAT over IV-tPA in acute stroke patients. Most recently, however, the data from 4 studies, including MR CLEAN, EXTEND-IA, ESCAPE, and

SWIFT PRIME, have shown that patients who undergo endovascular treatment within 6 hours from symptom onset for anterior circulation, proximal large-vessel occlusion AIS do indeed have significantly better outcomes when compared to patients who are treated with IV-tPA alone, and this is now the standard of care in this situation [2-5].

Hemorrhagic Stroke

Hemorrhagic stroke represents 13% of all strokes, with ICH causing 10% and SAH causing 3% of all strokes [1]. Subdural and epidural hemorrhages are not included in these statistics and are more commonly associated with trauma. Although hemorrhagic strokes are significantly less common than ischemic strokes, hemorrhagic strokes are more severe, with a higher risk of death than ischemic stroke [33]. This increased relative risk declines with time and becomes equal to that of ischemic stroke at 3 months [33]. Numerous underlying etiologies are responsible for these intracerebral and SAHs, including hypertension, amyloid angiopathy, AVMs, cavernous malformations, hemorrhagic neoplasms, cerebral aneurysms, venous sinus thrombosis, dural AVFs, and nonaneurysmal SAH.

Noncontrast head CT remains the initial imaging test of choice in patients presenting with stroke symptoms because of its speed of image acquisition, high sensitivity for acute hemorrhage, wide availability on a 24-hour basis, lack of absolute contraindications, and ease of patient monitoring. Catheter arteriography remains the gold standard imaging test to evaluate for cerebral aneurysm, AVM, and dural arteriovenous fistula. CTA, MRA, and contrast-enhanced brain MRI can provide valuable additional information in the workup of ICH and SAH.

Variant 1: Asymptomatic. Structural lesion on physical examination (cervical bruit) and/or risk factors.

Screening for carotid artery stenosis is an important tool in the prevention of ischemic stroke since carotid artery disease is responsible for 10%–20% of strokes. Studies have shown that treatment of carotid artery stenosis with endarterectomy or carotid artery stenting is effective in reducing the risk of stroke in populations of symptomatic and asymptomatic patients who are selected based on the severity of luminal diameter narrowing in the proximal ICAs and who have access to procedures with reasonable risk profiles [34-39]. Multiple noninvasive imaging techniques have been developed to evaluate for carotid artery stenosis, including duplex US, CTA, and MRA. These noninvasive imaging techniques all have advantages and limitations. They all have high sensitivities and specificities for detecting high-grade treatable carotid artery stenosis [40]. Choosing an imaging modality to evaluate for carotid artery stenosis depends on the clinical scenario as well as patient factors such as age, kidney function, contrast allergies, claustrophobia, presence of morbid obesity, and presence of cardiac devices or metal within the patient.

Duplex US is an effective low-cost method of initial screening for carotid stenosis. However, it is operator dependent and can have difficulty with artifact due to calcified plaque and is limited in the evaluation of near occlusion, tandem lesions, and lesions at the distal carotid and carotid origin [41].

CTA and MRA are often used for further evaluation of patients with positive duplex US findings. MRA is an excellent screening test since it is noninvasive and does not utilize ionizing radiation. CE-MRA is superior to noncontrast TOF-MRA because it is less affected by slow and turbulent flow, particularly at the carotid bifurcation. The combination of duplex US and CE-MRA is a common choice for carotid artery evaluation [42-44]. Limitations of MRA include difficulty in patients with claustrophobia and the risk of nephrogenic systemic sclerosis with gadolinium contrast agents in specific patients. Also, certain medical devices are not MR safe or require additional evaluation and patient preparation and monitoring during scanning. Retained metal fragments in the patient might also not be considered safe in the MRI scanner. The ACR documents on MRI safety and contrast media provide more detailed safety information [6,7]. CTA has advantages over MRA including its superior spatial resolution, rapid image acquisition, and significantly decreased susceptibility to motion artifacts. CTA is also less prone than MRA to artifacts from calcification and is better able to evaluate slow flow and tandem lesions. In fact, dual-energy CTA has been shown to be helpful in evaluating carotid stenosis in the presence of calcified plaques; however, with this technique, there could be overestimation of high-grade stenosis [45,46]. However, limitations of CTA include radiation exposure to the patient, necessity of IV contrast, and risk of contrast allergy and contrast nephropathy. All of these factors need to be taken into account when deciding which noninvasive imaging modality to use for carotid artery evaluation. If CTA or MRA are inconclusive or if they contradict findings from duplex US, DSA should be performed.

Most of the emphasis of carotid artery imaging has focused on luminal narrowing given its importance shown by trials such as NASCET. However, further characterization of carotid atherosclerotic plaque features such as surface ulceration, plaque rupture, thin fibrous capsule, lipid-rich necrotic core, and presence of inflammation with the help of high-resolution CTA and MRA may provide additional prognostic information [47-49]. These techniques could also possibly be used to follow effectiveness of medical therapy over time [47].

Investigations of cerebrovascular reserve by using CT and MR perfusion with acetazolamide challenge have shown some promising results [50,51]. These methods may identify patients with carotid artery stenosis who are at higher risk for stroke from decreased cerebrovascular reserve and poor collateral circulation.

Variant 2: Carotid territory or vertebrobasilar TIA, initial screening survey.

Imaging evaluation of TIA should be performed as soon as possible because of the associated high risk of future stroke and is ideally performed with imaging of the brain parenchyma including a contrast-enhanced brain MRI along with vascular imaging of the head and neck including TOF-MRA of the circle of Willis and CE-MRA of the cervical vessels [14]. An example of a TIA-protocol MRI includes a contrast-enhanced brain MRI with sequences including DWI, ADC, T1-weighted imaging, T2-weighted imaging, FLAIR, T2*-weighted imaging, or SWI, and postcontrast T1-weighted imaging, as well as TOF-MRA of the circle of Willis and CE-MRA of the cervical vessels. If there is concern for carotid artery dissection, axial fat-suppressed T1-weighted images through the neck should be obtained. This combination of sequences allows for identification of other causes for the patient's symptoms and allows the identification of the presence of infarct and its age [15]. CT and CTA can alternatively be performed if there is contraindication to MRI. However, CT is significantly less sensitive for infarct and parenchymal lesions and CTA requires the use of IV iodinated contrast and exposes the patient to ionizing radiation [16]. If neck CTA or MRA cannot be performed to evaluate the vasculature of the neck, carotid duplex US may be useful. CTP may be used to evaluate for ischemia when MRI is contraindicated or not available [17].

Variant 3: New focal neurologic defect, fixed or worsening. Less than 6 hours. Suspected stroke.

The time period since symptom onset in this variant has changed since the last version of the ACR Appropriateness Criteria on cerebrovascular disease. In the previous version the variants of new focal neurologic deficit included time windows of <3 hours, 3–24 hours, and >24 hours since symptom onset. A major factor in determining the previous time windows was the FDA-approved time window of <3 hours since symptom onset for treatment of acute stroke with IV-tPA. However, recently the data from multiple randomized endovascular studies have shown that patients with large-vessel occlusion (ICA terminus or middle cerebral artery [MCA], M1 segment) who undergo endovascular treatment within 6 hours from symptom onset for anterior circulation AIS do indeed have significantly better outcomes when compared to patients who are treated with IV-tPA alone [2-5]. Therefore, the new variants include time windows of <6 hours and >6 hours.

The goal of imaging in this scenario is to identify hemorrhage and in the absence of hemorrhage to differentiate the extent of permanently damaged infarcted brain tissue (infarct core) from the extent of salvageable but ischemic surrounding brain tissue (ischemic penumbra). NCCT is the first-line imaging study in the workup of acute stroke to rule out intracranial hemorrhage because of its speed, high sensitivity for hemorrhage, and ready availability. The Alberta Stroke Program Early CT (ASPECT) score obtained from a noncontrast head CT can be used to determine the extent of already established ischemic cerebral infarction and the risk of parenchymal hemorrhage following IV-tPA [52,53]. The ASPECT score has been widely used in clinical trials, and it has been repeatedly validated.

After ruling out intracranial hemorrhage and large infarct with NCCT, IV-tPA is used as the first-line treatment for ischemic stroke (in the absence of any of the well-established contraindications for tPA administration). When possible, the next step in the imaging algorithm should be CTA of the head and neck to evaluate for large-vessel occlusion such as the distal ICA or the proximal MCA. The vessels of the neck need to be included to identify atherosclerotic carotid artery disease or dissection with or without tandem occlusion of intracranial arteries. This can be performed immediately after the NCCT without delaying IV-tPA treatment. The importance of identifying large vessel occlusion lies in the fact that IV-tPA has low likelihood of recanalizing these large-vessel occlusions, with only 6% success in recanalizing the distal ICA and 30% success in recanalizing the proximal MCA [54]. This large-vessel occlusion is a possible target for pharmaco-mechanical IAT. At this stage, patients with a distal ICA or proximal MCA clot, National Institutes of Health Stroke Scale (NIHSS) of >8, and an ASPECT score of

>7 are often taken to the angiography suite for further intervention. The role of CTP in this scenario is presently unclear and not well substantiated, leading to a gradual decline in its usage in the setting of AIS [55-61]. CTP images suffer from high noise and low signal-to-noise and contrast-to-noise ratios, as well as exquisite sensitivity to patient movement [58]. Additionally, the results obtained from different commercial software packages can vary widely [57]. Recently, it has been shown that there is no change in patient outcomes when the usage of CTP studies is significantly reduced in the evaluation of acute stroke patients [59].

MRI studies represent an alternative pathway for the workup of acute stroke patients. Noncontrast MRI with susceptibility-weighted sequences is likely to be as sensitive as noncontrast head CT scans in the detection of intracranial hemorrhage [62,63]. However, routine gradient-recalled echo (GRE) studies are unlikely to be similarly sensitive in the detection of ICH [62]. SWI is, however, very sensitive to patient movement and therefore may not be able to entirely eliminate noncontrast head CT in acute stroke patients. The greatest value of MRI at present lies in the information provided by DWI in patients presenting with AIS. The extent of infarcted tissue present on DWI has been shown to have a direct correlation with poor outcomes in stroke patients. In general, if a large core infarct volume (>70 mL) is found on DWI, IAT should not be attempted given the low likelihood of good outcome [64,65]. The risk of hemorrhagic conversion of infarcted brain tissue following IV-tPA administration also increases with increased volume of tissue with restricted diffusion [66]. MRA can be used in a fashion analogous to CTA for the identification of arterial obstruction from thrombosis. In addition, the length of the clot found on MRA or CTA appears to have inverse correlation with the success of reperfusion strategies [67].

MR perfusion can be obtained rapidly and several studies have demonstrated the value of determining the ischemic penumbra in patients presenting with AIS using the diffusion/perfusion mismatch [25,64,68,69]. MR perfusion, similar to CTP, suffers from low signal-to-noise ratio and sensitivity to patient movement. Additionally, concerns regarding usage of gadolinium-based contrast agents are likely to make it unavailable for patients with renal impairment. These drawbacks in the use of perfusion imaging have led to the development of a clinical/diffusion mismatch algorithm in the management of AIS patients [59,70]. A clinically significant clinical/diffusion mismatch indicating an ischemic penumbra is demonstrated by an NIHSS >10 and a DWI lesion <70 mL [59,70]. Patients who present with such a significant mismatch may then be candidates for IV or IAT.

Variant 4: New focal neurologic defect, fixed or worsening. Longer than 6 hours. Suspected stroke.

The time period since symptom onset in this variant has changed since the last version of the ACR Appropriateness Criteria on cerebrovascular disease. In the previous version, the variants of new focal neurologic deficit included time windows of <3 hours, 3–24 hours, and >24 hours since symptom onset. A major factor in determining the previous time windows was the FDA-approved time window of <3 hours for treatment of acute stroke with IV-tPA. However, recently the data from multiple randomized endovascular studies have shown that patients with large-vessel occlusion (ICA terminus or MCA, M1 segment) who undergo endovascular treatment within 6 hours from symptom onset for anterior circulation AIS do indeed have significantly better outcomes when compared to patients who are treated with IV-tPA alone [2-5]. Therefore, the new variants include time windows of <6 hours and >6 hours.

When the patient presents with stroke beyond the therapeutic window of 6 hours since symptom onset, further evaluation is ideally performed with a contrast-enhanced brain MRI along with TOF-MRA of the circle of Willis and CE-MRA of the cervical vessels. If there is concern for carotid artery dissection, axial fat-suppressed T1-weighted images through the neck should be obtained. An example of a stroke-protocol MRI includes a contrast-enhanced brain MRI with sequences including DWI, ADC, T1, T2, FLAIR, T2* or SWI, and postcontrast T1-weighted images, as well as TOF-MRA of the circle of Willis and CE-MRA of the cervical vessels. This combination of sequences allows for identification of other causes for the patient's symptoms and allows the estimation of the age of the infarct. Hyperacute infarct (0–6 hours) will demonstrate restricted diffusion only, which represents cytotoxic edema. Acute infarct (6 hours–3 days) will demonstrate restricted diffusion (cytotoxic edema) and hyperintensity on FLAIR/T2 images (vasogenic edema). Subacute infarct (3–14 days) is characterized by gyral contrast enhancement and is the time period when hemorrhagic transformation is most likely to occur. Vasogenic edema also peaks during the subacute time period at around 5 days, putting the patient at risk for herniation. Restricted diffusion usually resolves during the subacute period. Chronic infarction (>2 weeks) is characterized by volume loss after dead neuronal tissue is removed and replaced by gliosis with subsequent cystic encephalomalacia. In patients with hemorrhage or hemorrhagic conversion of ischemic stroke, the changes in

appearance of blood products over time on T1- and T2-weighted MR sequences are well documented. NCCT and CTA can alternatively be performed if there is contraindication to MRI. However, CT is significantly less capable of providing information regarding the acuity of findings. The role of perfusion imaging in this scenario is currently unclear and in question given the low signal-to-noise ratio, low contrast-to-noise ratio, high sensitivity to patient motion, high interobserver variability, and high intervender software variability. CTP may play a role to evaluate for ischemia when MRI is contraindicated or cannot be performed. Additionally, CTP has shown some promise in identifying patients who may benefit from therapy outside the accepted treatment window [71-74].

Variant 5: Proven parenchymal hemorrhage (hematoma).

There are numerous possible etiologies for cerebral intraparenchymal hemorrhage including hypertension, amyloid angiopathy, coagulopathy, AVM, cavernous malformation, neoplasm, dural venous sinus or cortical venous thrombosis, and dural AVF. If the original noncontrast head CT and clinical history strongly point to a hypertensive cause (basal ganglia location of hemorrhage, patient age >55 years, clinical history of hypertension), it may be reasonable to limit further imaging workup to include follow-up serial imaging studies with noncontrast head CT to evaluate for hemorrhage expansion, herniation, hydrocephalus, and other complications that may require surgical intervention. However, if the etiology of the parenchymal hemorrhage remains in question after initial imaging, further investigation with brain MRI without and with contrast is the imaging study of choice. An example of a brain MRI without and with contrast protocol includes T1, T2, FLAIR, DWI, T2* or SWI, and postcontrast T1-weighted images. If there is concern for a vascular malformation, then MRA of the head should also be performed. MRA can be performed without or with contrast, although contrast-enhanced brain MRA tends to have significant venous contamination. Time-resolved MRA techniques may help identify associated arterial and venous anatomy. MRV of the head may also be useful to help characterize venous drainage in AVMs and dural AVFs. MRV of the head in this situation is best performed with and without contrast. The evaluation for high-flow vascular malformations including AVMs and dural AVFs is further described in the ACR Appropriateness Criteria® topic on “[Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage.](#)” Additionally, if there is concern for CVT, then venous imaging should be performed. The imaging evaluation of CVT is described in Variant 6. Vascular imaging of the neck with MRA and MRV in conjunction with vascular imaging of the head may be useful to evaluate the anatomy, patency, and involvement of the neck vasculature in the initial imaging workup. However, follow-up imaging of the lesion responsible for the parenchymal hemorrhage is often adequately performed with head-only vascular imaging. CE-MRA of the neck is superior to noncontrast MRA due to the difficulties that noncontrast MRA has evaluating the carotid bifurcations and great-vessel origins.

If there is a contraindication to MRI or it is unavailable, then imaging with CT can be utilized, keeping in mind that CT has inferior soft-tissue contrast resolution compared to MRI. CTA and CTV can be utilized for the vascular imaging workup. However, they utilize radiation and require IV contrast, whereas TOF-MRA/MRV and PC-MRA/MRV can be done without contrast or exposure to radiation. Contrast-enhanced head CT can be performed after noncontrast head CT for parenchymal evaluation and possible identification of enhancing neoplasm/metastases. Active bleeding demonstrated by extravasation of contrast in the hematoma on head CTA or contrast-enhanced head CT, known as the “spot sign,” can predict risk of hematoma expansion and risk of in-hospital mortality and poor outcome [75,76]. Subsequent serial follow-up imaging for parenchymal hemorrhage is usually performed with noncontrast head CT to evaluate for hematoma expansion, herniation, hydrocephalus, and other complications that may require surgical intervention.

Nonaneurysmal vascular malformations of the brain can be divided into low-flow and high-flow malformations. High-flow vascular malformations are AVMs and dural AVFs and their imaging evaluation is further described in the ACR Appropriateness Criteria® topic on “[Cerebrovascular Diseases-Aneurysm, Vascular Malformation, and Subarachnoid Hemorrhage.](#)” Low-flow vascular malformations consist of developmental venous anomalies (DVAs) with or without associated cavernomas. DVAs are by themselves benign lesions that are easily detected on the basis of a pathognomonic medusa-head appearance on CTA or MRA [77-79]. Cavernomas, which are sometimes associated with DVAs, can present with intracranial hemorrhage [80,81]. Cavernomas are best diagnosed by noninvasive imaging, particularly with SWI [79,82]. DSA is not recommended for the evaluation of either abnormality except to rule out rare arterialized DVAs. Atypical DVAs are also easily evaluated with SWI, and do not require DSA [78,82,83].

Variant 6: Suspected dural venous sinus thrombosis.

CVT is an uncommon cause for stroke, affecting only about 1%–2% of stroke patients [84]. The patients can present with headaches, seizures, or decreased level of consciousness from ischemic or hemorrhagic stroke [84]. Accurate diagnosis is important since treatment can significantly improve outcomes.

The imaging evaluation for CVT can vary upon the acuity of the patient presentation and the availability of imaging technology. In the acute presentation, NCCT is often performed initially to evaluate for intracranial causes requiring immediate intervention. Signs of CVT on NCCT, including a dense dural venous sinus or cortical vein, are unreliable due to changes in clot density over time as well as hemoconcentration or hemodilution. In fact, NCCT is abnormal in only 30% of patients with CVT [84]. These changes in density of the thrombus can also cause difficulty in visualizing the classic “empty delta” sign of a central filling defect from thrombus within the venous sinus on contrast-enhanced CT. If there is suspicion for CVT after the initial NCCT is performed in the acute setting, CTV can be quickly performed while the patient is still on the CT scan table. In a less acute setting or an acute setting where MRI is readily available without contraindications, contrast-enhanced brain MRI and MRV are often performed for optimal evaluation.

Although CTV may be as accurate as MRV in the diagnosis of CVT [85], both techniques have their advantages and disadvantages. CTV requires the use of IV iodinated contrast, limiting its use in patients with acute or severe chronic renal failure. TOF and PC-MRV do not require IV contrast and can be used in patients with renal dysfunction. CTV exposes the patient to radiation, and therefore MRV should alternatively be considered in children in order to reduce radiation exposure in this radiation-sensitive population. However, sedation may be required for some children or claustrophobic patients undergoing MRV. CTV is less prone to image degradation due to patient motion than MRV. 3-D reconstruction can be limited in CTV due to difficulty removing bone adjacent to the cerebral dural venous sinuses.

MRI evaluation for CVT optimally consists of brain MRI with and without contrast along with brain MRV with and without contrast. Noncontrast brain MRI may be able to visualize CVT depending on the age of the thrombus, similar to visualizing the evolution of a hematoma on MRI. Brain MRI with and without contrast is useful in evaluating for complications of CVT, including venous infarction, hemorrhage, and edema. Contrast-enhanced brain MRI is useful for determining the age of an infarct, if present, as well as evaluating for other causes of the patient’s symptoms, such as tumor or infection. Brain MRV is ideally performed with and without IV contrast. Noncontrast MRV techniques include 2-D TOF, 3-D TOF, and phase-contrast. Both 2-D and 3-D TOF MRV are limited when there is T1 hyperintense thrombus, which can mimic normal flow. 3-D TOF MRV is limited in areas of slow and turbulent flow which can cause signal loss mimicking thrombus. However, a postcontrast 3-D TOF MRV may help with problem solving in this situation. PC-MRV does not have difficulty evaluating slow flow or differentiating thrombus from normal venous flow. Postcontrast MRV is not limited by slow flow, but T1-hyperintense thrombus may mimic normal flow, so it is important to also evaluate the noncontrast brain MRI and MRV for complete characterization. Subtraction images can also be helpful for the postcontrast MRV to remove the T1-hyperintense thrombus from the MRV images.

Given the current noninvasive imaging options, including MRV and CTV, invasive imaging with catheter venography is seldom needed and can be reserved for when noninvasive imaging options are unavailable or for treatment purposes. Imaging of the neck with MRV, CTV, DSA, or even US may be useful to evaluate the extent of thrombus in the neck. The role of perfusion imaging in this scenario is currently unclear and in question given the low signal-to-noise ratio, low contrast-to-noise ratio, high sensitivity to patient motion, high interobserver variability, and high intervender software variability. However, there have been studies of perfusion imaging showing some promise in the evaluation of CVT, possibly revealing prognostic information and allowing for evaluation of an ischemic penumbra [86].

Summary of Recommendations

- Screening for carotid artery stenosis can be performed noninvasively with duplex US, CE-MRA, or CTA. The combination of duplex US and CE-MRA is a common choice for carotid artery evaluation. DSA is the gold standard for carotid artery evaluation and should be performed if noninvasive imaging is inconclusive or contradictory. Evaluation of cerebrovascular reserve with CT or MR perfusion with acetazolamide challenge may identify patients at higher risk for stroke due to poor collateral circulation.

- Imaging evaluation of TIA should be performed as soon as possible because of the associated high risk of future stroke and is ideally performed with contrast-enhanced brain MRI along with 3-D TOF-MRA of the circle of Willis and CE-MRA of the neck vasculature.
- Noncontrast head CT is the first-line imaging test for acute stroke patients to rule out intracranial hemorrhage and large infarct. When possible, CTA should be the next imaging study after IV-tPA administration in acute stroke patients to evaluate for large-vessel occlusion as a target for IAT. The role of CT and MR perfusion in acute stroke is presently unclear because of low signal-to-noise ratio, sensitivity to patient motion, and varied results between software packages, leading to a gradual decline in its usage, without a significant change in patient outcomes despite its decreased use. Core infarct volume demonstrated by DWI has been shown to predict outcomes in stroke patients, with core infarct volume >70 mL indicative of poor outcomes even with IAT. The importance of the ischemic penumbra in acute stroke has been demonstrated. However, CT and MR perfusion studies can be unreliable, leading to the development of a clinical/diffusion mismatch algorithm. A clinical/diffusion mismatch indicating an ischemic penumbra is demonstrated by a NIHSS >10 and a DWI lesion <70 mL.
- Patients presenting with acute stroke beyond the 6-hour treatment window are ideally evaluated with contrast-enhanced brain MRI along with 3-D TOF-MRA of the circle of Willis and CE-MRA of the neck vasculature. Age of the infarct can be determined with contrast-enhanced brain MRI.
- Intraparenchymal cerebral hemorrhages that do not have the classic clinical history and imaging appearance for hypertensive hemorrhage, should undergo further parenchymal and vascular imaging, ideally with contrast-enhanced brain MRI, MRA, and sometimes MRV. CT, CTA, and sometimes CTV are imaging alternatives.
- Vascular imaging in the evaluation for CVT can be performed with either MRV or CTV and depends on the availability of the imaging modality and after considering the risks and benefits of the use of IV contrast, ionizing radiation, and MRI safety issues. MRV is ideally performed without and with IV contrast due to difficulties that noncontrast MRV has with slow and turbulent flow. CTV requires IV contrast administration and ionizing radiation exposure to the patient. Parenchymal brain imaging in the evaluation of CVT is ideally performed with contrast-enhanced brain MRI, as it is superior to head CT at detecting parenchymal brain complications of CVT including edema and infarct.

Supporting Documents

The evidence table, literature search, and appendix for this topic are available at <https://acsearch.acr.org/list>. The appendix includes the strength of evidence assessment and the final rating round tabulations for each recommendation.

For additional information on the Appropriateness Criteria methodology and other supporting documents go to www.acr.org/ac.

Relative Radiation Level Information

Potential adverse health effects associated with radiation exposure are an important factor to consider when selecting the appropriate imaging procedure. Because there is a wide range of radiation exposures associated with different diagnostic procedures, a relative radiation level (RRL) indication has been included for each imaging examination. The RRLs are based on effective dose, which is a radiation dose quantity that is used to estimate population total radiation risk associated with an imaging procedure. Patients in the pediatric age group are at inherently higher risk from exposure, both because of organ sensitivity and longer life expectancy (relevant to the long latency that appears to accompany radiation exposure). For these reasons, the RRL dose estimate ranges for pediatric examinations are lower as compared to those specified for adults (see Table below). Additional information regarding radiation dose assessment for imaging examinations can be found in the ACR Appropriateness Criteria® [Radiation Dose Assessment Introduction](#) document.

Relative Radiation Level Designations		
Relative Radiation Level*	Adult Effective Dose Estimate Range	Pediatric Effective Dose Estimate Range
○	0 mSv	0 mSv
⊙	<0.1 mSv	<0.03 mSv
⊕	0.1-1 mSv	0.03-0.3 mSv
⊕⊕	1-10 mSv	0.3-3 mSv
⊕⊕⊕	10-30 mSv	3-10 mSv
⊕⊕⊕⊕	30-100 mSv	10-30 mSv

*RRL assignments for some of the examinations cannot be made, because the actual patient doses in these procedures vary as a function of a number of factors (eg, region of the body exposed to ionizing radiation, the imaging guidance that is used). The RRLs for these examinations are designated as “Varies”.

References

1. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics--2015 update: a report from the American Heart Association. *Circulation* 2015;131:e29-322.
2. Berkhemer OA, Fransen PS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015;372:11-20.
3. Campbell BC, Mitchell PJ, Kleinig TJ, et al. Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med* 2015;372:1009-18.
4. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015;372:1019-30.
5. Saver JL, Goyal M, Bonafe A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015;372:2285-95.
6. American College of Radiology. *Manual on Contrast Media*. Available at: <http://www.acr.org/Quality-Safety/Resources/Contrast-Manual>.
7. Kanal E, Barkovich AJ, Bell C, et al. ACR guidance document on MR safe practices: 2013. *J Magn Reson Imaging* 2013;37:501-30.
8. Edjlali M, Roca P, Rabrait C, et al. MR selective flow-tracking cartography: a postprocessing procedure applied to four-dimensional flow MR imaging for complete characterization of cranial dural arteriovenous fistulas. *Radiology* 2014;270:261-8.
9. Gandhi D, Chen J, Pearl M, Huang J, Gemmete JJ, Kathuria S. Intracranial dural arteriovenous fistulas: classification, imaging findings, and treatment. *AJNR Am J Neuroradiol* 2012;33:1007-13.
10. Iryo Y, Hirai T, Kai Y, et al. Intracranial dural arteriovenous fistulas: evaluation with 3-T four-dimensional MR angiography using arterial spin labeling. *Radiology* 2014;271:193-9.
11. Nishimura S, Hirai T, Sasao A, et al. Evaluation of dural arteriovenous fistulas with 4D contrast-enhanced MR angiography at 3T. *AJNR Am J Neuroradiol* 2010;31:80-5.
12. Wu H, Block WF, Turski PA, et al. Noncontrast dynamic 3D intracranial MR angiography using pseudo-continuous arterial spin labeling (PCASL) and accelerated 3D radial acquisition. *J Magn Reson Imaging* 2014;39:1320-6.
13. Telischak NA, Detre JA, Zaharchuk G. Arterial spin labeling MRI: clinical applications in the brain. *J Magn Reson Imaging* 2015;41:1165-80.
14. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. *Stroke* 2009;40:2276-93.
15. Kidwell CS, Warach S. Acute ischemic cerebrovascular syndrome: diagnostic criteria. *Stroke* 2003;34:2995-8.

16. Restrepo L, Jacobs MA, Barker PB, Wityk RJ. Assessment of transient ischemic attack with diffusion- and perfusion-weighted imaging. *AJNR Am J Neuroradiol* 2004;25:1645-52.
17. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA* 2000;284:2901-6.
18. Eliasziw M, Kennedy J, Hill MD, Buchan AM, Barnett HJ. Early risk of stroke after a transient ischemic attack in patients with internal carotid artery disease. *CMAJ* 2004;170:1105-9.
19. Kleindorfer D, Panagos P, Pancioli A, et al. Incidence and short-term prognosis of transient ischemic attack in a population-based study. *Stroke* 2005;36:720-3.
20. Wu CM, McLaughlin K, Lorenzetti DL, Hill MD, Manns BJ, Ghali WA. Early risk of stroke after transient ischemic attack: a systematic review and meta-analysis. *Arch Intern Med* 2007;167:2417-22.
21. Clark TG, Murphy MF, Rothwell PM. Long term risks of stroke, myocardial infarction, and vascular death in "low risk" patients with a non-recent transient ischaemic attack. *J Neurol Neurosurg Psychiatry* 2003;74:577-80.
22. van Wijk I, Kappelle LJ, van Gijn J, et al. Long-term survival and vascular event risk after transient ischaemic attack or minor ischaemic stroke: a cohort study. *Lancet* 2005;365:2098-104.
23. Del Zoppo GJ, Saver JL, Jauch EC, Adams HP, Jr. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: a science advisory from the American Heart Association/American Stroke Association. *Stroke* 2009;40:2945-8.
24. Neumann-Haefelin T, Wittsack HJ, Wenserski F, et al. Diffusion- and perfusion-weighted MRI. The DWI/PWI mismatch region in acute stroke. *Stroke* 1999;30:1591-7.
25. Ribo M, Molina CA, Rovira A, et al. Safety and efficacy of intravenous tissue plasminogen activator stroke treatment in the 3- to 6-hour window using multimodal transcranial Doppler/MRI selection protocol. *Stroke* 2005;36:602-6.
26. Fargen KM, Meyers PM, Khatri P, Mocco J. Improvements in recanalization with modern stroke therapy: a review of prospective ischemic stroke trials during the last two decades. *J Neurointerv Surg* 2013;5:506-11.
27. Mokin M, Khalessi AA, Mocco J, et al. Endovascular treatment of acute ischemic stroke: the end or just the beginning? *Neurosurg Focus* 2014;36:E5.
28. Nogueira RG, Lutsep HL, Gupta R, et al. Trevo versus Merci retrievers for thrombectomy revascularisation of large vessel occlusions in acute ischaemic stroke (TREVO 2): a randomised trial. *Lancet* 2012;380:1231-40.
29. Saver JL, Jahan R, Levy EI, et al. Solitaire flow restoration device versus the Merci Retriever in patients with acute ischaemic stroke (SWIFT): a randomised, parallel-group, non-inferiority trial. *Lancet* 2012;380:1241-9.
30. Broderick JP, Palesch YY, Demchuk AM, et al. Endovascular therapy after intravenous t-PA versus t-PA alone for stroke. *N Engl J Med* 2013;368:893-903.
31. Ciccone A, Valvassori L, Nichelatti M, et al. Endovascular treatment for acute ischemic stroke. *N Engl J Med* 2013;368:904-13.
32. Kidwell CS, Jahan R, Gornbein J, et al. A trial of imaging selection and endovascular treatment for ischemic stroke. *N Engl J Med* 2013;368:914-23.
33. Andersen KK, Olsen TS, Dehlendorff C, Kammersgaard LP. Hemorrhagic and ischemic strokes compared: stroke severity, mortality, and risk factors. *Stroke* 2009;40:2068-72.
34. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. *JAMA* 1995;273:1421-8.
35. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. *N Engl J Med* 1998;339:1415-25.
36. Brott TG, Hobson RW, 2nd, Howard G, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. *N Engl J Med* 2010;363:11-23.
37. Grotta JC. Clinical practice. Carotid stenosis. *N Engl J Med* 2013;369:1143-50.
38. Halliday A, Harrison M, Hayter E, et al. 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): a multicentre randomised trial. *Lancet* 2010;376:1074-84.
39. Rothwell PM, Eliasziw M, Gutnikov SA, et al. Analysis of pooled data from the randomised controlled trials of endarterectomy for symptomatic carotid stenosis. *Lancet* 2003;361:107-16.
40. Wardlaw JM, Chappell FM, Best JJ, Wartolowska K, Berry E. Non-invasive imaging compared with intra-arterial angiography in the diagnosis of symptomatic carotid stenosis: a meta-analysis. *Lancet* 2006;367:1503-12.

41. Jahromi AS, Cina CS, Liu Y, Clase CM. Sensitivity and specificity of color duplex ultrasound measurement in the estimation of internal carotid artery stenosis: a systematic review and meta-analysis. *J Vasc Surg* 2005;41:962-72.
42. Barth A, Arnold M, Mattle HP, Schroth G, Remonda L. Contrast-enhanced 3-D MRA in decision making for carotid endarterectomy: a 6-year experience. *Cerebrovasc Dis* 2006;21:393-400.
43. Honish C, Sadanand V, Fladeland D, Chow V, Pirouzmand F. The reliability of ultrasound measurements of carotid stenosis compared to MRA and DSA. *Can J Neurol Sci* 2005;32:465-71.
44. U-King-Im J, Hollingworth W, Trivedi RA, et al. Cost-effectiveness of diagnostic strategies prior to carotid endarterectomy. *Ann Neurol* 2005;58:506-15.
45. Korn A, Bender B, Thomas C, et al. Dual energy CTA of the carotid bifurcation: advantage of plaque subtraction for assessment of grade of the stenosis and morphology. *Eur J Radiol* 2011;80:e120-5.
46. Thomas C, Korn A, Ketelsen D, et al. Automatic lumen segmentation in calcified plaques: dual-energy CT versus standard reconstructions in comparison with digital subtraction angiography. *AJR* 2010;194:1590-5.
47. Tang TY, Howarth SP, Miller SR, et al. The ATHEROMA (Atorvastatin Therapy: Effects on Reduction of Macrophage Activity) Study. Evaluation using ultrasmall superparamagnetic iron oxide-enhanced magnetic resonance imaging in carotid disease. *J Am Coll Cardiol* 2009;53:2039-50.
48. U-King-Im J, Tang TY, Patterson A, et al. Characterisation of carotid atheroma in symptomatic and asymptomatic patients using high resolution MRI. *J Neurol Neurosurg Psychiatry* 2008;79:905-12.
49. Wintermark M, Jawadi SS, Rapp JH, et al. High-resolution CT imaging of carotid artery atherosclerotic plaques. *AJNR Am J Neuroradiol* 2008;29:875-82.
50. Chen A, Shyr MH, Chen TY, Lai HY, Lin CC, Yen PS. Dynamic CT perfusion imaging with acetazolamide challenge for evaluation of patients with unilateral cerebrovascular steno-occlusive disease. *AJNR Am J Neuroradiol* 2006;27:1876-81.
51. Endo H, Inoue T, Ogasawara K, Fukuda T, Kanbara Y, Ogawa A. Quantitative assessment of cerebral hemodynamics using perfusion-weighted MRI in patients with major cerebral artery occlusive disease: comparison with positron emission tomography. *Stroke* 2006;37:388-92.
52. Menon BK, Puetz V, Kochar P, Demchuk AM. ASPECTS and other neuroimaging scores in the triage and prediction of outcome in acute stroke patients. *Neuroimaging Clin N Am* 2011;21:407-23, xii.
53. Vora NA, Gupta R, Thomas AJ, et al. Factors predicting hemorrhagic complications after multimodal reperfusion therapy for acute ischemic stroke. *AJNR Am J Neuroradiol* 2007;28:1391-4.
54. Saqqur M, Uchino K, Demchuk AM, et al. Site of arterial occlusion identified by transcranial Doppler predicts the response to intravenous thrombolysis for stroke. *Stroke* 2007;38:948-54.
55. Dani KA, Thomas RG, Chappell FM, et al. Computed tomography and magnetic resonance perfusion imaging in ischemic stroke: definitions and thresholds. *Ann Neurol* 2011;70:384-401.
56. Deipolyi AR, Wu O, Macklin EA, et al. Reliability of cerebral blood volume maps as a substitute for diffusion-weighted imaging in acute ischemic stroke. *J Magn Reson Imaging* 2012;36:1083-7.
57. Fahmi F, Marquering HA, Streekstra GJ, et al. Differences in CT perfusion summary maps for patients with acute ischemic stroke generated by 2 software packages. *AJNR Am J Neuroradiol* 2012;33:2074-80.
58. Gonzalez RG. Low signal, high noise and large uncertainty make CT perfusion unsuitable for acute ischemic stroke patient selection for endovascular therapy. *J Neurointerv Surg* 2012;4:242-5.
59. Gonzalez RG, Copen WA, Schaefer PW, et al. The Massachusetts General Hospital acute stroke imaging algorithm: an experience and evidence based approach. *J Neurointerv Surg* 2013;5 Suppl 1:i7-12.
60. Kamalian S, Maas MB, Goldmacher GV, et al. CT cerebral blood flow maps optimally correlate with admission diffusion-weighted imaging in acute stroke but thresholds vary by postprocessing platform. *Stroke* 2011;42:1923-8.
61. Konstas AA, Goldmakher GV, Lee TY, Lev MH. Theoretic basis and technical implementations of CT perfusion in acute ischemic stroke, part 2: technical implementations. *AJNR Am J Neuroradiol* 2009;30:885-92.
62. Cheng AL, Batool S, McCreary CR, et al. Susceptibility-weighted imaging is more reliable than T2*-weighted gradient-recalled echo MRI for detecting microbleeds. *Stroke* 2013;44:2782-6.
63. Verma RK, Kottke R, Andereggen L, et al. Detecting subarachnoid hemorrhage: comparison of combined FLAIR/SWI versus CT. *Eur J Radiol* 2013;82:1539-45.
64. Lansberg MG, Straka M, Kemp S, et al. MRI profile and response to endovascular reperfusion after stroke (DEFUSE 2): a prospective cohort study. *Lancet Neurol* 2012;11:860-7.

65. Yoo AJ, Verduzco LA, Schaefer PW, Hirsch JA, Rabinov JD, Gonzalez RG. MRI-based selection for intra-arterial stroke therapy: value of pretreatment diffusion-weighted imaging lesion volume in selecting patients with acute stroke who will benefit from early recanalization. *Stroke* 2009;40:2046-54.
66. Sanak D, Nosal V, Horak D, et al. Impact of diffusion-weighted MRI-measured initial cerebral infarction volume on clinical outcome in acute stroke patients with middle cerebral artery occlusion treated by thrombolysis. *Neuroradiology* 2006;48:632-9.
67. Riedel CH, Zimmermann P, Jensen-Kondering U, Stingele R, Deuschl G, Jansen O. The importance of size: successful recanalization by intravenous thrombolysis in acute anterior stroke depends on thrombus length. *Stroke* 2011;42:1775-7.
68. Furlan AJ, Eyding D, Albers GW, et al. Dose Escalation of Desmoteplase for Acute Ischemic Stroke (DEDAS): evidence of safety and efficacy 3 to 9 hours after stroke onset. *Stroke* 2006;37:1227-31.
69. Hacke W, Albers G, Al-Rawi Y, et al. The Desmoteplase in Acute Ischemic Stroke Trial (DIAS): a phase II MRI-based 9-hour window acute stroke thrombolysis trial with intravenous desmoteplase. *Stroke* 2005;36:66-73.
70. Gonzalez RG. Current state of acute stroke imaging. *Stroke* 2013;44:3260-4.
71. Donnan GA, Baron JC, Ma H, Davis SM. Penumbral selection of patients for trials of acute stroke therapy. *Lancet Neurol* 2009;8:261-9.
72. Jovin TG, Liebeskind DS, Gupta R, et al. Imaging-based endovascular therapy for acute ischemic stroke due to proximal intracranial anterior circulation occlusion treated beyond 8 hours from time last seen well: retrospective multicenter analysis of 237 consecutive patients. *Stroke* 2011;42:2206-11.
73. Kim JT, Yoon W, Park MS, et al. Early outcome of combined thrombolysis based on the mismatch on perfusion CT. *Cerebrovasc Dis* 2009;28:259-65.
74. Michel P, Ntaios G, Reichhart M, et al. Perfusion-CT guided intravenous thrombolysis in patients with unknown-onset stroke: a randomized, double-blind, placebo-controlled, pilot feasibility trial. *Neuroradiology* 2012;54:579-88.
75. Delgado Almandoz JE, Yoo AJ, Stone MJ, et al. The spot sign score in primary intracerebral hemorrhage identifies patients at highest risk of in-hospital mortality and poor outcome among survivors. *Stroke* 2010;41:54-60.
76. Wada R, Aviv RI, Fox AJ, et al. CT angiography "spot sign" predicts hematoma expansion in acute intracerebral hemorrhage. *Stroke* 2007;38:1257-62.
77. Lee C, Pennington MA, Kenney CM, 3rd. MR evaluation of developmental venous anomalies: medullary venous anatomy of venous angiomas. *AJNR Am J Neuroradiol* 1996;17:61-70.
78. Ruiz DS, Yilmaz H, Gailloud P. Cerebral developmental venous anomalies: current concepts. *Ann Neurol* 2009;66:271-83.
79. Santucci GM, Leach JL, Ying J, Leach SD, Tomsick TA. Brain parenchymal signal abnormalities associated with developmental venous anomalies: detailed MR imaging assessment. *AJNR Am J Neuroradiol* 2008;29:1317-23.
80. Campbell PG, Jabbour P, Yadla S, Awad IA. Emerging clinical imaging techniques for cerebral cavernous malformations: a systematic review. *Neurosurg Focus* 2010;29:E6.
81. Smith ER, Scott RM. Cavernous malformations. *Neurosurg Clin N Am* 2010;21:483-90.
82. Thomas B, Somasundaram S, Thamburaj K, et al. Clinical applications of susceptibility weighted MR imaging of the brain - a pictorial review. *Neuroradiology* 2008;50:105-16.
83. Jagadeesan BD, Delgado Almandoz JE, Moran CJ, Benzinger TL. Accuracy of susceptibility-weighted imaging for the detection of arteriovenous shunting in vascular malformations of the brain. *Stroke* 2011;42:87-92.
84. Saposnik G, Barinagarrementeria F, Brown RD, Jr., et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2011;42:1158-92.
85. Ozsvath RR, Casey SO, Lustrin ES, Alberico RA, Hassankhani A, Patel M. Cerebral venography: comparison of CT and MR projection venography. *AJR* 1997;169:1699-707.
86. Gupta RK, Bapuraj JR, Khandelwal N, Khurana D. Prognostic indices for cerebral venous thrombosis on CT perfusion: a prospective study. *Eur J Radiol* 2014;83:185-90.

The ACR Committee on Appropriateness Criteria and its expert panels have developed criteria for determining appropriate imaging examinations for diagnosis and treatment of specified medical condition(s). These criteria are intended to guide radiologists, radiation oncologists and referring physicians in making decisions regarding radiologic imaging and treatment. Generally, the complexity and severity of a patient's clinical condition should dictate the selection of appropriate imaging procedures or treatments. Only those examinations generally used for evaluation of the patient's condition are ranked. Other imaging studies necessary to evaluate other co-existent diseases or other medical consequences of this condition are not considered in this document. The availability of equipment or personnel may influence the selection of appropriate imaging procedures or treatments. Imaging techniques classified as investigational by the FDA have not been considered in developing these criteria; however, study of new equipment and applications should be encouraged. The ultimate decision regarding the appropriateness of any specific radiologic examination or treatment must be made by the referring physician and radiologist in light of all the circumstances presented in an individual examination.