American College of Radiology
ACR Appropriateness Criteria®

Clinical Condition: **Cerebrovascular Disease**

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

Radiologic Procedure	Rating	Comments	RRL*
US carotid duplex	8	May need to confirm with second non-invasive study.	None
MRA neck	8		None
CTA neck	8		Low
MRI head without and with contrast	5	Consider perfusion if stenosis found.	None
CT head without and with contrast	5	Consider perfusion if stenosis found.	Low
US transcranial Doppler	3		None
MRI head without contrast	3		None
MRA head	3	May be useful if stenosis found.	None
CT head without contrast	3		Low
CTA head	3	May be useful if stenosis found.	Low
INV arteriography neck	2		IP
INV arteriography head and neck	2		IP
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 2: **Carotid territory or vertebrobaslar TIA, initial screening survey. (In these tables a TIA is the report of an historical transient ischemic event by the patient or other witness. The acute neurological deficit in progress must be treated as an acute stroke and can only be considered a TIA in retrospect if it resolves without intervention.)**

Radiologic Procedure	Rating	Comments	RRL*
MRI head with or without contrast	8	Consider perfusion if stenosis found. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
MRA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
CT head	8	Consider perfusion if stenosis found. Primarily to rule out hemorrhage. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
CTA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
US carotid duplex	6		None
US transcranial Doppler	3		None
INV arteriography neck	3		IP
INV arteriography head and neck	3		IP
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

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

Radiologic Procedure	Rating	Comments	RRL*
MRI head with or without contrast	8	Consider perfusion if stenosis found. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
MRA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
CT head	8	Consider perfusion if stenosis found. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
CTA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
INV arteriography neck	5	If intra-arterial therapy is considered.	IP
INV arteriography head and neck	5	If intra-arterial therapy is considered.	IP
US carotid duplex	2		None
US transcranial Doppler	2		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 4: **New focal neurologic defect, fixed or worsening. Three to 24 hours.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head with or without contrast	8	Diffusion especially valuable. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
MRA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
CT head	8	For perfusion according to institutional protocols. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
CTA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
INV arteriography neck	6	If intra-arterial therapy is considered.	IP
INV arteriography head and neck	6	If intra-arterial therapy is considered.	IP
US carotid duplex	2		None
US transcranial Doppler	2		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			***Relative Radiation Level**

Clinical Condition: **Cerebrovascular Disease**

Variant 5: **New focal neurologic defect, fixed or worsening. Greater than 24 hours.**

Radiologic Procedure	Rating	Comments	RRL*
MRI head with or without contrast	8	Diffusion especially valuable. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
MRA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	None
CT head	8	For perfusion according to institutional protocols. Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
CTA head and neck	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment not unreasonably delayed.	Low
INV arteriography neck	6	If intra-arterial therapy is considered.	IP
INV arteriography head and neck	6	If intra-arterial therapy is considered.	IP
US carotid duplex	2		None
US transcranial Doppler	2		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			***Relative Radiation Level**

Clinical Condition: **Cerebrovascular Disease**

Variant 6: **Risk for unruptured aneurysm. Positive family history.**

Radiologic Procedure	Rating	Comments	RRL*
MRA head	8	MR preferred if treatment is not unreasonably delayed.	None
CTA head	8	Noncontrast CT obtained routinely at the same time. MR preferred if treatment is not unreasonably delayed.	Low
MRI head with or without contrast	6		None
MRA neck	3		None
CT head	3	Obtained with CTA.	Low
CTA neck	2		Low
US carotid duplex	1		None
US transcranial Doppler	1		None
INV arteriography neck	1		IP
INV arteriography head and neck	1		IP
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			***Relative Radiation Level**

Clinical Condition: **Cerebrovascular Disease**

Variant 7: **Clinically suspected subarachnoid hemorrhage (SAH), not yet confirmed.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without contrast	9		Low
CT head without and with contrast	5		Low
MRI head with or without contrast	4		None
MRA head	4		None
INV arteriography neck	2		IP
INV arteriography head and neck	2		IP
MRA neck	2		None
CTA head	2		Low
CTA neck	2	For treatment planning.	Low
US carotid duplex	1		None
US transcranial Doppler	1		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 8: **Proven SAH by lumbar puncture or imaging.**

Radiologic Procedure	Rating	Comments	RRL*
INV arteriography neck	8	For treatment planning. As part of cerebral angiography.	IP
INV arteriography head and neck	8		IP
CT head without contrast	8		Low
CTA head	8		Low
MRA head	7		None
MRI head with or without contrast	6		None
MRA neck	6	For future treatment planning.	None
CTA neck	6	For future treatment planning.	Low
US transcranial Doppler	5	For vasospasm.	None
CT head without and with contrast	5		Low
US carotid duplex	1		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 9: **Proven SAH, negative angiogram, follow-up.**

Radiologic Procedure	Rating	Comments	RRL*
INV arteriography head and neck	8		IP
MRI head with or without contrast	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	None
MRA head	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	None
CTA head	8	MR preferred if treatment is not unreasonably delayed.	Low
US transcranial Doppler	5	For vasospasm.	None
INV arteriography neck	5		IP
MRA neck	5		None
CT head without contrast	5		Low
CTA neck	5		Low
CT head without and with contrast	4		Low
US carotid duplex	1		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 10: **Clinically suspected parenchymal hemorrhage (hematoma), not yet confirmed.**

Radiologic Procedure	Rating	Comments	RRL*
CT head without contrast	8		Low
MRI head with or without contrast	7		None
CT head without and with contrast	7		Low
MRA head	4		None
CTA head	4		Low
INV arteriography head and neck	3		IP
MRA neck	3		None
CTA neck	3		Low
INV arteriography neck	2		IP
US carotid duplex	1		None
US transcranial Doppler	1		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

Clinical Condition: **Cerebrovascular Disease**

Variant 11: **Proven parenchymal hemorrhage (hematoma).**

Radiologic Procedure	Rating	Comments	RRL*
MRI head with or without contrast	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	None
MRA head	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	None
CT head without contrast	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	Low
CTA head	8	Combined vascular and cerebral evaluation should be considered. MR preferred if treatment is not unreasonably delayed.	Low
INV arteriography neck	7		IP
INV arteriography head and neck	7	If suspect AVM.	IP
CT head without and with contrast	7		Low
MRA neck	5		None
CTA neck	5		Low
US carotid duplex	1		None
US transcranial Doppler	1		None
fMRI head	1		None
MRI spectroscopy head	1		None
FDG-PET head	1		High
NUC SPECT head	1		High
Rating Scale: 1=Least appropriate, 9=Most appropriate			*Relative Radiation Level

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Summary of Literature Review

Diseases of the cerebral vasculature are often manifested as stroke, a generic term encompassing a range of ischemic and hemorrhagic lesions (Appendix A). There are approximately 700,000 new or recurrent strokes per year in the United States, an average of one every 45 seconds [1]. Stroke is the third leading underlying or contributing cause of death in the U.S. behind heart disease and cancer, accounting for 157,800 deaths in 2003 [1]. The mean age of stroke death in 2002 was 79.6 years, and the overall death rate for stroke is approximately 54%; 8%-12% of ischemic strokes and 37%-38% of hemorrhagic strokes result in death within 30 days [1]. Of all strokes, 88% are ischemic, 9% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage [1]. Significant functional disability is common in nonfatal cases, and stroke is a leading cause of serious, long-term disability in the United States [1]. Between 50%-70% of stroke survivors regain functional independence, but 15%-30% are permanently disabled, and 20% require institutional care at three months after onset. The estimated direct and indirect cost for stroke in the U.S. in 2006 was $57.9 billion [1].

Because of the gravity of stroke's sequelae, considerable effort has been expended to identify risk factors for the disease (Appendix B) and strategies for stroke prevention in high-risk patients [2]. These range from modification of lifestyle to surgical or endovascular intervention. Surgery has been shown effective in altering morbidity of both asymptomatic and symptomatic patients in randomized, prospective clinical trials in which the intent to treat was determined partly by imaging [3-6]. In asymptomatic patients, screening should be undertaken not only by a sensitive, noninvasive (ie, low-risk) test directed at identifying the abnormal cerebrovascular substrate but also with some consideration for identifying those in risk populations with a high prevalence of disease (eg, patients with carotid bruit) [7,8].

Although the diagnostic accuracy of duplex ultrasound (US), computed tomography angiography (CTA), magnetic resonance angiography (MRA) and time resolved contrast-enhanced MRA (CEMRA), are all high for internal carotid artery (ICA) stenoses of 70%-99% [9,10] only US appears to offer cost-effective initial screening. Alternatively, variability in performance (efficacy vs effectiveness) precludes endorsement of its routine use as the sole examination before endarterectomy, and combined use with CEMRA is an increasingly common practice [7,8,11-13]. Multislice CTA is promising but relatively few rigorous studies have been done, and the technique remains limited by large intravenous contrast injection volumes, potential contrast toxicity or reaction, radiation dose, and plaque calcification that may obscure the stenosis. [10,14-16]. It should be noted that although surgical outcome studies have been based on catheter angiography, the possible morbidity of these studies and continuing improvement in noninvasive exams have made invasive studies less common [10,11,13]. Similarly, a variety of imaging strategies may be undertaken in symptomatic cases where the initial studies can be directed toward the brain parenchyma, and a vascular study can be included immediately at the outset. Elevated ischemic stroke risk in patients with chronic carotid stenosis or occlusion can also be identified using single-photon-emission computed tomography (SPECT) and Xenon-CT methods, which show reduced cerebral vascular reserve (CVR) after acetazolamide challenge, or by elevated oxygen extraction fraction (OEF) using ^{15}O-PET (positron emission tomography) [17-19]. Although there is limited experience with MR and CT perfusion methods for this purpose, elevated cerebral blood volume appears to correlate with reduced CVR and increased stroke risk [20,21], and these studies are widely available.

Clinically, stroke is most often characterized by the ictal onset of focal neurologic symptoms due to ischemia or hemorrhage into the brain. Ischemic infarction can be classified into various subgroups based on the mechanism of the ischemia (hemodynamic or thromboembolic) and the pathology of the vascular lesion: atherosclerotic, lacunar, cardioembolic, or indeterminate. The various stroke subtypes differ in cause, frequency, clinical signs, outcomes, and treatment, and are defined by diagnostic evaluation of etiology (ischemic vs hemorrhagic) and

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underlying vasculature. Intracranial hemorrhage can be subdivided into two distinct types based on the site and origin of blood: subarachnoid and intracerebral hemorrhage.

Although stroke is typically acute in onset, occasionally the onset is less immediate and more gradual or stuttering. Differential diagnostic considerations in these cases include atypical migraine, multiple sclerosis, venous occlusive disease, and atypical epilepsy.

Traditionally, if focal neurologic symptoms continue for more than 24 hours, stroke is diagnosed; otherwise, a focal neurologic deficit lasting less than 24-hours has been defined as a transient ischemic attack (TIA). However, this time-based definition of TIA may be inadequate and misleading, potentially leading to inappropriate delays in diagnosis and treatment. A "tissue-based" definition has been proposed that considers all acute focal neurologic deficits as possible infarcts and classifies them as acute ischemic cerebrovascular syndromes (AICS) based on the degree of certainty of tissue ischemic injury, determined primarily by imaging studies [22,23]. Because most transient ischemic neurologic symptoms last for 1 hour or less and 50% or more show tissue injury on MRI diffusion-weighted imaging (DWI) [22,24,25], the TIA Working Group recently proposed a new definition of TIA as "*a brief episode of neurologic dysfunction presumptively caused by focal brain or retinal ischemia, typically lasting less than one hour, without neuroimaging evidence of acute infarction"* [26]. This change reflects the growing emphasis on the earliest possible diagnosis and treatment of acute ischemia and the use of MRI and CT for definitive infarct diagnosis and exclusion of hemorrhage [27]. In addition, because 15% of all strokes are heralded by a TIA and the 90 day risk of stroke after a TIA is as high as 20%, a TIA should trigger an immediate work up for stroke risks and follow-up imaging studies [1,28].

Rapid and accurate diagnosis of hemorrhage, ischemia, and completed infarction have become paramount in importance to treatment of acute cerebrovascular disease because of the demonstrable benefit (and hemorrhage risk) of acute intravenous and intra-arterial thrombolytic therapy for cerebral ischemia in prospective clinical trials [29-34]. Current clinical practice in the U.S. is limited to the thrombolytic agent recombinant tissue plasminogen activator (rtPA) given intravenously, preferably within 1 hour and no later than 3 hours after symptom onset, following the exclusion of intracerebral hemorrhage by a noncontrast CT scan. However, only 20%-25% of admissions typically arrive at the emergency department within 3 hours of symptom onset and, following appropriate exclusions, successful treatment with rtPA, without symptomatic major hemorrhage, is limited to between 3%-8.5% of ischemic stroke admissions [30,35,36]. The effectiveness of intravenous thrombolysis treatment does not appear to vary by stroke subtype (embolic, atherosclerotic, small-vessel occlusion) [37]. There is insufficient experience in general clinical practice to show that intra-arterial thrombolytic delivery or mechanical clot extraction methods are more effective than intravenous rtPA thrombolysis. Hemorrhagic risk appears to be somewhat higher and organizational complexity may delay treatment delivery and has limited widespread use of these approaches [32,38,39].

Because of the small percentage of acute stroke patients treated within the 3 hour limit, there is growing interest in expanding the treatment time window without increasing hemorrhage risk. A pooled risk-benefit analysis of existing rtPA trials using CT scan exclusion of hemorrhage has suggested that treatment may be safe in some patients out to 4.5 hours after ictus [31]. In addition, several current clinical trials are focused on the use of thrombolytic and neuroprotective agents combined with MRI techniques that identify the "ischemic penumbra," the underperfused yet viable halo of brain parenchyma around or interspersed with the region of completed infarction that is at risk of progressing to infarction. Gadolinium bolus MR perfusion weighted imaging (PWI) measures capillary level tissue blood flow parameters (CBF, CBV, MTT, and TTP) based on the central volume principle, and is being used to identify the volume of tissue with reduced blood flow, which is then compared to the volume of presumed infarcted tissue as indicated by restricted diffusion (reduced ADC on DWI). When the low blood flow tissue volume is larger than the restricted diffusion volume by 20% or more a perfusion-diffusion (PWI-DWI) "mismatch" is said to exist [33]. The low blood flow region outside the restricted diffusion infarct "core" includes underperfused but metabolically stable "oligemic" tissue as well as unstable "penumbral" tissue that may become infarcted if therapy is delayed or ineffective [40,41]. The tissue metabolic status within these zones can be more precisely identified by measuring oxygen metabolism (CMRO2) and the oxygen extraction fraction (OEF, which is elevated and defines "misery perfusion" in the true penumbral zone) using 15O-PET or experimental MRI methods, but these imaging techniques are not likely to be available in general clinical practice in the near future [42,43]. Although imperfect, the MRI PWI-DWI mismatch is being used as a "biomarker" for the treatment decisions at time points from 3-24 hours after ictus in several ongoing thrombolysis and mechanical clot extraction trials [44,45]. Recently published results of two European studies with a new thrombolytic agent, desmoteplase, show successful treatment of patients as late as 9 hours after ictus without increased incidence of symptomatic hemorrhage [46,47].

These results support the intuitively attractive concept of determining treatment based on the physiologic status of the ischemic brain tissue rather than only on the time since symptom onset. However, currently there is insufficient scientific evidence or widespread clinical experience to recommend this diagnostic approach for thrombolytic treatment beyond the 3 hour window after symptom onset [27].

With the introduction of CT scanning by Hounsfield in the early 1970s came the ability to acutely assess the brain, subarachnoid, and ventricular spaces noninvasively. Similarly, on the basis of the x-ray attenuation of blood and edema relative to cerebrospinal fluid (CSF) and brain parenchyma, CT is effective in detecting acute hemorrhage into brain parenchyma, the subarachnoid, subdural, or intraventricular spaces, and in distinguishing acute hemorrhage from ischemia/infarction [48-56]. Because of its ready availability and high sensitivity to the presence or absence of acute blood, noncontrast CT historically has been the preferred modality for initial imaging of suspected stroke, but it has lacked a similar sensitivity to acute ischemia and infarction [57].

A recent resurgence in the use of CT for initial stroke evaluation has occurred with the increasing clinical availability of CT perfusion (CTP) and CTA. CTP is acquired by rapid scanning during a bolus intravenous contrast infusion, and blood flow parameters (CBF, CBV, MTT, and TTP) are calculated based on the central volume principle. This has transformed CT into a technique with high sensitivity to cerebrovascular abnormalities and early perfusion deficits, detectable prior to observable low density changes on noncontrast CT [58,59]. Quantitative CTP measurement of cerebral blood flow parameters have been proposed as a means of discriminating between infarct and penumbra [60-65]. These measurements, plus the ability to quickly identify acute hemorrhage and vascular lesions as well as the ubiquitous availability of CT scanners, have been suggested as the key advantages of CT over MRI for acute stroke evaluation. However, the limited volume coverage of CTP (currently restricted to a 2 or 4 cm slab, the width of the detector array), the greater risks of contrast reaction or fluid overload from iodinated contrast materials vs gadolinium, and the lack of a direct measure of cellular viability like diffusion restriction mitigate these advantages over MRI [66].

Alternatively, DWI MRI has been shown to be exquisitely sensitive to acute infarction within minutes of the precipitating ictus [67-69], although tissue with small ADC reductions (eg, 20% below normal) may represent reversible ischemia that does not progress to completed infarct [70]. Additional information obtainable through the combined use of dynamic cerebral blood volume techniques (perfusion imaging, PWI) as well as vascular imaging (MRA) makes MRI an appealing tool for diagnosis and treatment monitoring of acute cerebrovascular disease [70-74]. However, enthusiasm for MRI in the setting of acute stroke has often been stifled by the variable and confounding appearance of hemorrhage on MRI. The recognition and characterization of the MRI findings in intracranial hemorrhage are understandable if one considers: 1) the location, specifically subarachnoid vs intraparenchymal; 2) the oxidative state of hemoglobin and the subsequent breakdown products; 3) the type of imaging pulse sequence used (T1 vs T2, spin-echo vs gradient-echo, conventional spin-echo vs RARE sequences); and 4) the field strength of the machine used to acquire the images [75-79].

Recent experience using T2* (gradient echo) imaging to detect low signal parenchymal hemorrhage and FLAIR scans to detect high signal subarachnoid blood have helped to renew interest in MRI as a first-line modality in patients with acute, focal neurologic deficits [80-84]. Although the presence of small hemorrhages on gradient-echo MRI may better predict hemorrhagic complications of rtPA therapy, there is insufficiently widespread clinical experience to recommend MRI over CT for routine exclusion of intracranial hemorrhage [27]. It is also important to re-emphasize the issue of availability of MRI in the context of the therapeutic window and potential contraindications: patients with pacemakers, cerebral aneurysm clips, ocular foreign bodies, or cochlear implants, and those suffering from claustrophobia or morbid with obesity (>320 pounds) [85].

As mentioned previously, CT is highly sensitive to the presence or absence of acute blood and has been the mainstay in emergent evaluation of acute cerebrovascular disease. Documented acute subarachnoid or parenchymal hemorrhage are conditions associated with high morbidity and mortality [86,87]. In the case of aneurysmal subarachnoid hemorrhage (SAH), this is partly due to the relatively high rate of early rebleeding. In patients presenting with SAH, early surgery or coiling is offered as a strategy to circumvent this problem, which in turn requires early cerebral angiography [86]. Intra-arterial angiography's sensitivity to cerebral aneurysms is estimated to be greater than 90%; in the setting of acute SAH this figure decreases to slightly greater than 80% [88]. Initially negative studies may require additional angiography at a future time.

Recent clinical practice has shifted toward use of noncontrast CT for SAH detection, followed immediately by CTA for aneurysm detection. Comparisons between CTA and catheter angiography in SAH patients, beginning with single-slice methods [89,90] and more

recently with multislice methods [91-93], have shown overall aneurysm detection sensitivities of 85%-95%, lower for smaller aneurysms, to approximately 50% for those less than 2 mm in diameter. Treatment of intracranial aneurysms following SAH is increasingly based on CTA alone [94,95]. Late appearances of new neurological changes suggestive of post-SAH vasospasm, ischemia, or hydrocephalus are increasingly investigated with transcranial Doppler (TCD) and CT imaging with CTA and CTP, while catheter angiography and SPECT are being used less frequently than in the past [96-102].

Treatment of intracranial aneurysms has evolved in recent years toward more use of endovascular coil embolization in place of or combined with surgical clipping [103,104]. Follow-up of treated aneurysms, clipped or coiled, to identify residual filling, is done definitively with catheter arteriography (DSA) but there is a growing interest in the use of less invasive techniques. CTA is inherently limited for this purpose because of the prominent "star" artifact produced by aneurysm clips and even more by the aneurysm coil mass. Time of flight MRA for this purpose is increasingly popular but is limited by local susceptibility and spin dephasing artifacts from the clip or coils and by turbulent flow and T1 saturation signal loss [105,106]. Dynamic CEMRA using bolus gadolinium injection and short TE elliptico-centric time-resolved acquisitions (eg, TRICKS) produces less susceptibility artifact and dephasing with reduced venous contamination of the arterial signal. However, at this point the limited number of studies are not sufficiently conclusive to recommend routine CEMRA for post-treatment aneurysm follow-up [107-109]. Most of these studies were performed at 1.5T, and experience with aneurysm clips and coils at 3.0T is limited. Before imaging at 3.0T, safety clearance for specific devices should be obtained from published sources or the device manufacturer [85]. In Europe, a prospective randomized multicenter trial comparing clipping and coiling in 2,143 patients with ruptured intracranial aneurysms suitable for both treatments demonstrated that endovascular coiling was more likely to result in independent survival at 1 year than neurosurgical clipping [104].

Because of the cumulative long-term risk of morbidity and mortality from subarachnoid hemorrhage, especially with larger aneurysms (>25 mm in diameter) and the relatively low risks of clipping or coiling unruptured intracranial aneurysms, there may be a clinical role for prophylactic screening [110,111]. Intra-arterial angiography carries the risk of thromboembolic complication and is relatively expensive; MRI and CTA provide less expensive, noninvasive alternatives, although their sensitivity to lesions less than 5 mm in diameter is suspect [90,95,112]. To date, individuals with a history of aneurysm or SAH in a first-degree relative have been considered candidates for screening [113]. Nevertheless, significant gaps in knowledge of the natural history (and thus risk of rupture) of intracranial aneurysms remain. Hence, while screening with MRA or CTA may be appropriate in patients with a positive family history, its impact on patient outcome is questionable [113].

Parenchymal brain hemorrhage may be associated with underlying vascular malformations such as AVM, pial arteriovenous fistulae, and cavernous malformations in younger patients as well as dural fistulae in older individuals. Diagnosis, assessment of risk for future hemorrhage, and effective treatment planning are all predicated on determination of the size of the underlying lesion, location within the brain parenchyma, pattern of venous drainage, and presence of intranidal aneurysm [114,115]. Acutely, this information is most frequently obtained by intra-arterial angiography, which in more complicated cases may be supplemented by MRI. Although time-resolved ellipticocentric bolus contrast CEMRA techniques with multicoil sensitivity encoding currently have temporal resolution in the 1-2 second range, they do not yet rival catheter DSA arteriography for separation of arterial and venous phases of high-flow AVMs, but may be useful for follow-up of partially embolized lesions [116]. Baseline and follow-up MRI may be appropriate in partially embolized cases or in patients undergoing stereotactic radiosurgery as noninvasive, low-risk means of identifying ischemic complications and assessing response to therapy [117-120].

Assumptions

All patient scenarios should be addressed as though the patients had been referred for imaging following a history and physical examination including neurological, vascular, and ophthalmoscopic exams.

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Appendix A. Definitions

Asymptomatic	Patients with no cerebral or retinal symptoms of vascular disease.
Cerebrovascular Disease (CVD)	All disorders in which an area of the brain is transiently or permanently affected by ischemia or bleeding, or in which one or more blood vessels of the brain are primarily impaired by a pathological process.
Acute Ischemic Cerebrovascular Syndrome (AICS)	Classification of acute neurological dysfunction within 7-days after onset by the degree of certainty of tissue ischemic injury (definite AICS", "probable AICS", "possible AICS", and "not AICS") which is based on the clinical presentation and evidence provided by laboratory and imaging studies.
Transient Ischemic Attack (TIA)	A brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of hemorrhage or acute infarction on CT or MRI.
Stroke	A generic term used to represent any one or all of a group of disorders, including cerebral infarction, intracerebral hemorrhage, or subarachnoid hemorrhage. Characterized by a non-convulsive focal neurologic deficit (FND) >24 hours in duration.
Cerebral (Ischemic) Infarction	An area of coagulation necrosis in brain tissue (i.e. tissue death) due to local anemia resulting from obstruction of the circulation to the area.
Hemorrhagic Infarction	A natural state of brain tissue following recanalization of a previously occluded artery. The bleeding component refers to petechial hemorrhage, single or confluent, usually maximal in cerebral gray matter, but not a true clot of blood.
Parenchymal Hemorrhage	A well-localized clot of blood, often with mass effect. Associated with neurologic worsening and easily recognized signs depending on its location.
Subarachnoid Hemorrhage	Bleeding into the subarachnoid space characterized by the sudden onset of severe headache that is typically dramatic. There may also be rapid alteration of consciousness, or vomiting, or both. Other symptoms in order of severity include minimal headache, nuchal rigidity, fixed neurologic deficits, cranial nerve palsy, drowsiness, confusion, stupor, or coma.

Appendix B. Risk Factors [2]

Characteristics and Lifestyles	
Definite	• Cigarette smoking
	• Alcohol consumption
	• Drug abuse
	• Age
	• Sex
	• Race
	• Familial factors
Possible	• Oral contraceptive use
	• Diet
	• Personality type
	• Geographic location
	• Season
	• Climate
	• Socioeconomic factors
	• Physical inactivity
	• Obesity
	• Abnormal blood lipids
	• Maternal mortality
Disease or Disease Markers	
Definite	• Hypertension
	• Cardiac disease
	• TIA
	• Elevated hematocrit
	• Diabetes mellitus
	• Sickle cell disease
	• Elevated fibrinogen concentration
	• Migraine and migraine equivalents
Possible	• Hyperuricemia
	• Hypothyroidism
Asymptomatic Structural Lesions	
Physical Examination	• Bruit (cervical, orbital, cranial)
	• Retinal emboli
	• Blood pressure differences between arms
	• Reduced pressure on oculoplethysmography
Imaging	• Silent infarction or hemorrhage (MRI, CT)
	• Arteriovenous malfomation, aneurysm, hamartoma
	• Atherosclerosis with arterial stenosis
	• Fibromuscular dysplasia, dissection
Multiple Factors in Combination	