Footnotes

In the modern clinical setting the practitioner can order a variety of imaging procedures for the evaluation of stroke patients or patients at risk of stroke. New procedures have improved accuracy and lessened invasiveness but increased the complexity of decision making. When all of the standard imaging techniques are available, a sequential choice must be made to optimize diagnostic yield, reduce the risk of harm to the patient, and minimize cost. Difficulties may arise when the practitioner is limited by the choice of diagnostic methods available or cost-containment policies. These guidelines are intended to update clinicians in the use of diagnostic imaging procedures in patients with acute stroke or at high risk of stroke and to provide insight for their sequential use in a hierarchical order of specificity and practicality. The target audience is physicians who care for patients with stroke, including specialists in neurology, neurosurgery, vascular surgery, internal medicine, emergency medicine, critical care medicine, and family medicine. The panel has endeavored to strike a balance between the tertiary care setting and the community hospital, offering practical recommendations that can be implemented in these settings, where most patients with acute stroke are treated. Advanced technology methods, unproved techniques, and clinical research strategies may be mentioned but are not recommended.

The first section discusses the diagnostic imaging evaluation of the patient with transient ischemia at high risk of stroke. The second section explains the approach to the patient with acute stroke. The third and fourth sections are complementary, dealing with special conditions such as subarachnoid hemorrhage, arteritis, dural sinus and venous thrombosis, arterial
dissection, and spinal cord stroke. Recommendations for procedures in women of child-bearing age and patients intolerant of contrast solutions are also made.

These guidelines should be supplemented by consulting the literature citations provided in the reference section. Ultrasonography of the neck and head has been purposely excluded from the text as it is the subject of another set of practice guidelines. To implement a practical approach while avoiding excessive technical discussion, the panel was composed of a balanced cross-section of clinicians with a special interest in vascular neurology and neuroradiologists with expertise in cerebrovascular imaging. Each member contributed a section, and all participated in editing the final version.

The panel used rules of evidence adapted from the quality of evidence ratings for diagnostic tests developed by the American Academy of Neurology Therapeutics and Technology Assessment Subcommittee* (Table 1) to separate scientific evidence from anecdotal reports or case series. The panel strived for consistency with treatment pathways discussed in other practice guidelines on acute stroke and transient ischemia recently published by the Stroke Council.12

**Rationale for Use of Imaging in Acute Cerebrovascular Disease**

Imaging of the brain and vessels in patients with acute cerebrovascular events has become a routine procedure used in daily clinical practice. However, unless the request for procedures is driven by appropriate clinical questions derived from an adequate clinical assessment, the images obtained may become a hindrance and a dangerous source of confusion rather than a help in treatment of the patient. The clinician should follow two basic pathways when ordering imaging tests in patients with acute cerebrovascular disease:

- Imaging of the parenchyma (brain, spinal cord)
- Imaging of the vessels (extracranial, intracranial)
- Imaging of the brain provides information that can guide patient treatment by
  - Identifying the lesion (is it a stroke?)
  - Determining the type of stroke (ischemic infarct or hemorrhage?)
  - Localizing the stroke (where is it?)
  - Quantifying the lesion (how large is it?)
  - Determining the age of the lesion (how old is it?)

Pertinent answers to these questions help in formulating a diagnosis, devising a plan of action, and advancing a prognosis.

Imaging of the vessels is intended to answer questions about the mechanism of the stroke, whether thrombotic, embolic, or hemodynamic, and the risk of future events by

- Identifying occlusive arterial disease (is there blockage?)
- Localizing the occlusion in extracranial or intracranial vessels (where? carotid? vertebrobasilar? intracranial? extracranial? other?)
- Quantifying the degree of occlusion (how severe?)
Determining the pathology (atherosclerosis? dissection? other?)
Identifying other vascular lesions (malformation, aneurysm, arterial compression, venous thrombosis).

This information will help determine the choice of therapeutic modalities, particularly anticoagulation or thrombolysis or surgical intervention to prevent future strokes.

These practice guidelines have been produced with the goal of helping the clinician select the optimal imaging procedure(s) to be used at the most appropriate time in response to basic questions posed by the clinical formulation. The optimal test is characterized by low risk, prompt availability, diagnostic reliability, and economy of cost.

Future revisions of these practice guidelines may be necessary to accommodate new imaging requirements in response to therapeutic developments, most notably in the fields of thrombolytic therapy, cytoprotective agents, and low-molecular-weight heparins. Imaging identification of cerebral regions at high risk of stroke is a challenge for the immediate future.

**Transient Monocular Blindness and Transient Ischemic Brain Attack**

Transient monocular blindness (TMB) and transient ischemic brain attack (TIA) are focal retinal or brain deficits caused by vascular disease that clear completely in less than 24 hours. Most TIAs have a duration of less than 1 hour, with a median duration of 14 minutes in carotid-distribution ischemia and 8 minutes in vertebrobasilar ischemia. Atherosclerosis of cerebrovascular arteries is the most common cause of transient ischemia in older patients with risk factors for stroke. TIAs may herald cardioembolic strokes in 11% to 30% of instances, and occasionally they may be the result of hypercoagulable states, arterial dissection, arteritis, aneurysm, and arteriovenous malformation. Rarely a transient neurological deficit of nonvascular origin may mimic TMB or TIA, for example, seizures, migraines, tumors, and subdural hematoma, requiring a precise differential diagnosis. Patients who have had a TIA have a risk of stroke estimated at 24% to 29% in the first 5 years after the event. The risk is higher in the first month and highest in patients with hemispheric TIA and carotid stenosis ≥ 70% luminal reduction (40% rate of stroke in 2 years). In contrast, patients of any age with isolated TMB and younger patients with TIA have a generally low risk of stroke.

A TIA should be promptly evaluated to institute therapy as soon as possible to decrease risk of stroke. A standard algorithm for assessment of patients is not universally used, although guidelines have been developed; diagnostic evaluation is influenced by medical history and specific characteristics of the TIA. (See Figure.) The goals of imaging assessment are to identify candidates for specific surgical or medical therapeutic modalities, determine prognosis, and exclude rare nonvascular causes. Diagnostic imaging indications are based on yield of the test, management implications, risk to the patient, and cost. Hospitalization may be advised to keep the patient under medical observation soon after the TIA, perform cerebrovascular arteriography, and expedite the diagnostic assessment, which may include cardiac imaging.

**Procedures**
Imaging of the Brain

Computed Tomography. Computed tomography (CT) of the head has a role in evaluation of patients with transient neurological symptoms. In patients with episodes of TMB or amaurosis fugax caused by retinal ischemia, CT of the head can only provide brain imaging information that is indirectly related to the mechanism of TMB. This includes detection of heavy calcification in the intracranial internal carotid artery (ICA) ipsilaterally, suggesting atherosclerotic plaque formation as a potential source of retinal hypoperfusion or embolism via the ophthalmic artery-central retinal artery route. The observation of associated clinically silent cerebral infarctions reaffirms the presence of vascular pathology. The yield for relevant nonvascular lesions is very low in patients with TMB.6

In patients with TIAs, CT may detect cerebral infarction. Anatomically appropriate acute infarction for symptoms of TIA has been documented in 29% to 34%.7-12 TIA traditionally thought to represent reversible ischemia without brain lesion can actually be the clinical expression of a cerebral infarction with short-lived symptoms.10,13 Silent brain infarction, or infarction without previous history, has been identified in 13% of patients with manifestations of TIA14 and in 47% of patients with TIA and known carotid stenosis, with most silent infarctions occurring on the symptomatic side.15

An additional role for CT in evaluation of patients with recent TIA is the exclusion of other lesions that may simulate stroke. Although uncommon, patients with transient focal neurological deficits suggestive of TIA can have a subdural hematoma,16,17 a brain tumor18 in the corresponding cerebral hemisphere, an arteriovenous malformation (AVM), or, less likely, an aneurysm. Intracerebral hemorrhage (ICH) as the cause of TIA is virtually ruled out in the presence of a fully resolved focal deficit of brief duration (minutes to a few hours), because complete early regression of symptoms is inconsistent with the diagnosis of ICH.19,20

In transient ischemia of the vertebrobasilar territory, the clinical presentation with various combinations of deficits of temporo-occipital, cranial nerves, long tracts, or cerebellar function suggests the need to image the posterior cerebral hemispheres and posterior fossa structures. CT can show posterior territory infarcts, either symptomatic (with TIA) or silent, although with considerably less reliability than in the anterior circulation. Posterior territory infarcts are more likely to be shown in the cerebellum than in the brain stem, since the latter area is poorly visualized with CT as a result of artifact produced by the surrounding bony structures of the posterior fossa.21 As in hemispheric events, there is a negligible chance of finding ICH as a cause of the transient, short, and fully resolved episode of vertebrobasilar TIA. Similarly, posterior fossa mass lesions such as subdural hematoma or tumor will rarely have a TIA-like presentation.

The presence of a dilated, tortuous, and calcified basilar artery, also referred to as dolichoectasia, can be readily documented with noncontrast CT scans. Such degree of basilar artery disease is a potential source of TIA and stroke, based on atherosclerotic involvement of the artery leading to symptoms by mechanisms of distal hemodynamic insufficiency, embolism, or basilar branch ischemia.

Contrast-enhanced CT in patients with TIAs can document the presence of incidental lesions
such as AVM, or rarely aneurysm, that are potential contraindications for anticoagulant treatment; in general, however, contrast enhancement is not necessary.

Exclusions: Pregnancy is a relative contraindication, although a shield can be used to diminish the effects of scattered radiation from the highly collimated beam to the head. Patients with allergies to contrast material should be identified.

Magnetic Resonance Imaging. Magnetic resonance imaging (MRI) of the head is used less often than CT for initial evaluation of patients with TIA because it is a more expensive, time-consuming diagnostic modality and is less available. However, MRI has certain advantages. Because of its higher contrast resolution and lack of bone-related artifacts, MRI is more capable of detecting acute and small infarcts, especially those involving the cerebral cortex. The prevalence of white matter hyperintense lesions seen on MRI is high in the general population (13%), increasing with age and the presence of risk factors for stroke. To be considered an infarction, a white matter hyperintense lesion in T2-weighted images should also be seen as hypointense in T1-weighted images. Freedom from distortions by bone artifact gives MRI the advantage of improved resolution of small infarcts in brain stem and cerebellar regions that escape detection by CT.

MRI can provide evidence of absence of the normal "flow voids" in the intracranial ICA, indicative of occlusive disorder at that level, or more proximally in the extracranial ICA, suggesting a cause of TIAs. MRI is better than CT for imaging vessels in the posterior fossa and can demonstrate decreased or absent flow voids of the basilar and vertebral arteries and their branches, suggesting compromised circulation. Findings of vascular ectasia, tortuosity, and stenosis suggest an atherosclerotic cause of TIA. Arterial dissections with subintimal hematoma replacing much of the arterial wall are well identified by MRI. Vascular wall dissection appears as a crescentlike or annular hyperintense profile on the cross-section of T1-weighted images of the vertebral or basilar arteries. The yield of MRI is also superior to that of CT for detection of incidental lesions such as AVM or subdural hematoma. Case reports suggest that MRI may detect isodense subdural hematomas that are unidentifiable on CT.

Despite the superior diagnostic yield of MRI over CT of the head, there is no indication for routine MRI of patients with TMB or TIA, because in general the presence of silent strokes does not change clinical treatment of patients. The panel recognizes that the presence of silent strokes has not been sufficiently evaluated and weighted in the treatment algorithm for symptomatic patients with appropriate carotid stenosis to modify recommendations at this time. Continued vigilance in this area is suggested. Superior identification and localization of symptomatic lesions in carotid or vertebrobasilar territories or vascular anomalies in the occasional patient with transient ischemia of nonatherosclerotic origin may facilitate diagnosis and influence management, but the indication remains subject to individual judgment based on clinical history and other medical circumstances. Diffusion-weighted and perfusion-weighted MRI, although not yet widely available, appear to have high sensitivity and specificity in the early detection of cerebral ischemia; these techniques may be able to differentiate TIA from acute stroke as well as reliably indicate areas at risk.

Limitations of this procedure are its relative unavailability in community hospitals, higher cost, and the claustrophobic reactions of some patients, which impede performance of the test.
Patients who develop claustrophobia or extreme anxiety in enclosed environments cannot tolerate lying still for MRI. This factor, along with metallic foreign bodies and other physical limitations, may exclude up to 14% of patients referred for study. Open MRI units alleviate anxiety in vulnerable patients and, although less powerful than conventional MRI installations, offer a reasonable compromise.

Exclusions: MRI of the head should not be performed in patients with intraorbital or intracranial ferromagnetic fragments, aneurysm clips, otic or cochlear implants, old prosthetic heart valves, pacemakers, and neurostimulators, or when agitation and severe claustrophobia cannot be resolved.

**Imaging of the Vessels**

Various techniques are available to produce images of the neck and cranial vessels that are generally categorized as noninvasive (a contrast agent is not necessary), relatively noninvasive (a contrast agent is administered intravenously), and invasive (a contrast agent is injected intra-arterially). Risk of complications increases slightly with intravenous administration of contrast material and is highest with intra-arterial selective catheterization and administration of a contrast agent. Ultrasonography and magnetic resonance angiography (MRA) are noninvasive, CT angiography and single-photon emission computed tomography (SPECT) are relatively noninvasive, and conventional radiographic angiography with or without selective catheterization is invasive. Ultrasonography, MRA, CT angiography, and SPECT are still evolving, so their roles are changing. Ultrasonography of extracranial and intracranial vessels is the subject of another set of practice guidelines.

**Noninvasive Tests. Magnetic Resonance Angiography.** MRA is a generic name for different approaches and variations of vascular resonance imaging by MRI. MRA refers to the computer-assisted generation of images (angiograms) by MRI created by the contrast of magnetically polarized flowing blood against a stationary magnetically saturated parenchyma. The resulting high-differential signal intensity creates a map that includes anatomic and physiological information. MRA techniques that avoid the risks and limitations of conventional invasive arteriographic methods are in development. Current techniques are based on inflow enhancement (time-of-flight methods) or rely on velocity-induced phase shifts (phase-contrast methods). Time-of-flight acquisitions emphasize vessel morphology, whereas phase-contrast methods provide additional information about velocity, direction of blood flow, and volume flow rates. Slice-by-slice (two-dimensional), volumetric (three-dimensional) acquisitions, or a combination of these allow greater flow sensitivity and/or spatial resolution.

The best images are obtained in normal vessels or those with minimal stenotic lesions, using multiple overlapping thin slabs or three-dimensional-phase contrast techniques that are widely available. Vessels with a large lumen give excellent signals that can be measured and used for MRA display.

Advanced degrees of arterial narrowing will be detected by MRA as luminal narrowing and by an overall drop-off in signal intensity due to associated turbulence as lesions become more severe. There is an inherent bias in interpretation of current MRA images to overestimate degree of stenosis. The image of the true stenotic lumen is reduced by a combination of factors: turbulence that causes countercurrent blood flow, decreasing or negating the signal;
loss or absence of laminar flow that produces better signals than turbulent flow; and volume of flow too small to generate a signal. A small volume of blood moving through a tight stenosis produces little detectable signal; in the most severe stenoses or near-occlusions with scarce flow of blood, a signal may not be detected at all. In 16 studies that were not uniformly blinded in which MRA was compared with carotid angiography and reported in the English literature, the concurrence rate in depicting lesion size ranged from 39% to 98% (Classes II and III). Overestimation of degree of stenosis was frequently noted and accounted for most disagreements (Class II) [36,37].

A meta-analytic review of MRA pooled as a noninvasive test with carotid duplex ultrasonography and carotid Doppler ultrasonography compared with conventional carotid arteriography has shown sensitivities between 0.82 and 0.86, specificities at 0.98, and test-effectiveness measures at or exceeding 3.0 when predicting occlusion [32] (Class II). At ≥ 70% stenosis of the extracranial ICA, these tests have sensitivities of 0.83 to 0.86, specificities of 0.89 to 0.94, and test-effectiveness measures approaching 3.0 [32] (Class II). At 50% stenosis, sensitivity of all three noninvasive tests ranges from 0.85 to 0.93, with a specificity of 0.92 [32] (Class II).

There is satisfactory anatomic correlation with conventional arteriography, but MRA generally overrepresents arterial stenosis, especially in high-grade narrowing [38-42]. Carotid atheromatous ulceration is not reliably visualized with MRA [43]. Research concerning ways to reduce signal loss as a result of stenosis is ongoing; contrast media, for example, might sufficiently increase the signal of flowing blood through stenoses to improve specificity [44]. MRA has the advantage of not being operator dependent.

In patients with vertebrobasilar ischemia, vascular imaging may identify the source vessel of ischemic attacks such as the subclavian artery, extracranial or intracranial vertebral arteries, basilar artery, or their branches. A specific surgical intervention analogous to carotid endarterectomy as a verified therapy in the posterior territory does not exist; therefore, precise imaging and measurement of vascular stenoses as obtained with conventional carotid arteriography has not reached the level of importance attained in carotid disease. In this setting a technique such as high-quality MRA that provides a vascular overview [45] of the extracranial and intracranial circulations is acceptable for evaluation of vertebrobasilar ischemia. Various MRA techniques produce anatomic images of the vertebral and basilar arteries and their main branches, including the posterior inferior cerebellar, superior cerebellar, and posterior cerebral arteries; the anterior inferior cerebellar artery appears with less consistency. An associated overview of the carotid arteries and the main intracranial branches can be obtained at the same time. Depending on vascular tortuosity and field placement, some vessel segments may not be seen on MRA because of planar exclusion, not disease. In the special case of fibromuscular dysplasia, MRA lacks the capability to distinguish this condition from long segmental atherosclerosis or dissection and may not cover the entire vertebrobasilar and subclavian system.

The agreement of MRA with conventional carotid arteriography in evaluating intracerebral vascular pathology reaches a mean of only 62% [36] (Classes II and III), which is less than with extracerebral carotid pathology.
MRA alone is often not sufficient for study and analysis of blood flow and blood vessel anatomy. When MRA is combined with duplex ultrasound, sensitivity and specificity improve but still result in misclassification of 3% of patients showing negative noninvasive test results but carotid stenosis $\geq 70\%$ on carotid angiography. Misclassification occurs in 9% of patients with occlusion on noninvasive test results but some degree of luminal patency on carotid angiography $^{32}$ (Class II). When MRA and duplex ultrasound findings agree, some practitioners suspend use of radiographic angiography, reserving this technique for disparate results $^{46}$ (Class III). Overestimation of degree of carotid stenosis without concurrent conventional angiographic measurements could lead to an excessive number of surgical procedures.

In summary, the limitations of MRA are relative unavailability, high cost compared with other noninvasive tests, sensitivity and specificity insufficient to establish an indication for carotid endarterectomy, and claustrophobic reactions.

Exclusions: MRA is not indicated for patients with intraorbital or intracranial ferromagnetic fragments, aneurysm clips, otic or cochlear implants, old prosthetic heart valves, pacemakers, and neurostimulators, or when agitation and severe claustrophobia cannot be resolved.

**Relatively Noninvasive Tests.**

**CT Angiography:** High-resolution contrast-enhanced CT scanning of the cervical vessels is a relatively noninvasive procedure that visualizes the arterial lumen and wall, providing information about changes in the carotid artery that might intervene in precipitation of TIA. $^{47}$ A nonionic, water-soluble, radiographic contrast medium is administered in an antecubital vein with a 150 to 200 mL bolus injection spread over the period of the dynamic sequence (average, 3 minutes). Dynamic consecutive 3-mm-thick CT slices are obtained in the axial plane at 3-mm automatic table increments, up to a maximum of 25 slices. Dynamic scans are made from the bottom of the C2 vertebral body to the mid-portion of the C5 vertebral body.

CT angiography of cervical vessels reveals enough vascular detail to be useful as a diagnostic screening method in patients with presumed atherosclerosis of the carotid bifurcation. In patients with TIA and TMB, CT scanning of the cervical carotid vessels is performed with CT scans of the head as a screening test and in serial follow-up evaluations. This is not a standard technique in most centers but could become available in facilities with a high-resolution CT scanner or a newer spiral CT scanner. It may be substituted for ultrasonography $^{48}$ and MRA of the cervical region where ultrasonography is unreliable and MRA not available.

Exclusions: Pregnancy, renal failure, multiple myeloma, congestive heart failure, and allergy to contrast material.

**Single-Photon Emission Computed Tomography:** SPECT is obtained by intravenous injection of a flow tracer or a receptor binding substance tagged with a radionuclide. Using a gamma camera and CT techniques, a three-dimensional image of the distribution of the radionuclide in the brain is acquired. The radiotracer accumulates in different areas of the brain proportionally to the rate of delivery of nutrients to that volume of brain tissue. Perfusion information obtained with SPECT approximates regional cerebral blood flow closely enough to be meaningful in patient care and clinical research studies. $^{49}$ Most routine clinical applications of brain perfusion SPECT rely on images that reflect tracer uptake and retention only. The appearance on SPECT of different areas ranges from "hot" for regions of high photon counts
and therefore high perfusion as the cortex, to "cold" for regions that are not perfused, as the ventricles. Images reflect the regional perfusion in the few minutes after the radiopharmaceutical is injected, regardless of when the patient undergoes scanning.

SPECT can be used to detect ischemic areas after TIA, assess vascular reserve in a particular vascular territory, and differentiate transient epileptic from transient ischemic phenomena. None of these applications is sufficiently widely used in the clinical practice of neurology to provide a recommendation. In patients who are marginal candidates for endarterectomy, the hemodynamic effect of stenosis on cerebral perfusion may be assessed with SPECT. Decreases in cerebrovascular reserve, measured as a diminished dilatory response to CO₂, acetazolamide, or adenosine, may be an indicator of perfusion failure.⁵⁰ A diminished dilatory response suggests that a proximal stenosis in the vascular territory is interfering with perfusion and may become symptomatic. However, no data show a correlation between decreased hemispheric perfusion and subsequent risk of TIA or stroke.

Occasionally the need arises to differentiate ischemia from epilepsy as the cause of a transient neurological deficit. Ischemia causes an area of hypoperfusion on SPECT, whereas during epileptic discharges the activated neuronal pool elicits hyperperfusion. Radiopharmaceuticals that remain stable for at least 6 hours are now available and can be injected into the patient in the electroencephalography laboratory or the patient's room at the time of a clinical event. Afterward the patient can be taken to the nuclear medicine department for scanning.

Exclusions: Pregnancy.

**Invasive Tests.** Radiographic Conventional Cerebrovascular Angiography. Radiographic angiography is the reference standard of the diagnostic effort to identify surgically accessible and remediable carotid lesions. Conventional selective arteriography or digital subtraction arteriography with a 1024×1024 matrix is generally used to image the lumen of cervical and intracranial vessels and visualize the arterial profile. To assess stenosis of atherosclerotic origin, attention is focused on the edges of the lumen at the most narrow segment of the stenosis. Whereas all of the blood is opacified, attenuation occurs within the most narrow segment of the lumen so that the contrast column edge is not marked by a sharp limit. The quality of the final image is determined by operator-related parameters (kilovolt potential [kVp], milliamperes per second [mAs], collimation, equalization of different thickness by masks, and film-screen grid choices).

The North American Symptomatic Carotid Endarterectomy Trial (NASCET)⁵¹ investigators recommend endarterectomy of the symptomatic carotid artery (Class I, type A) in a surgery-tolerant patient when luminal reduction is ≥ 70% on conventional arteriography. Ratios of diameter measurements of the narrowest residual lumen are compared with the normal-appearing carotid profile well beyond the bulb in a measurement made according to the following method:

\[(1−N/D)\times100=\% \text{ stenosis}\]

where N is diameter stenosis, and D, diameter of artery beyond the bulb where the walls are parallel.
The investigators in the European Carotid Surgery Trial (ECST)\(^5\) also recommend endarterectomy of the symptomatic carotid artery (Class I, type A) in a surgery-tolerant patient when luminal reduction on arteriography is \(\geq 70\%\). The method used compares the measurement at the narrowest point with the theoretical diameter of the carotid bulb according to the following equation:

\[
(1-N/E) \times 100 = \% \text{ stenosis}
\]

where \(N\) is diameter stenosis, and \(E\), estimated normal diameter bulb.

Others\(^5\) prefer the use of the common carotid artery diameter (common carotid method) as the denominator:

\[
(1-N/C) \times 100 = \% \text{ stenosis}
\]

where \(N\) is diameter stenosis and \(C\), diameter of common carotid artery.

Approximate linear disparities between the results obtained with the three methods make it possible to convert measurements made by one method to those of another using a mathematical equation\(^5\). Uncertainty remains regarding a surgical indication for categories of luminal reduction \(<69\%\) as measured by the NASCET or ECST methods in patients with symptoms appropriate to the diseased carotid artery. Results of the continuing NASCET and ECST trials must be awaited.

Conventional arteriography is more satisfactory than any of the noninvasive tests in meeting the diagnostic criteria for imaging of extracranial and intracranial vessels: identification of occlusive arterial disease, localization of the occlusion, quantification of degree of occlusion, determination of pathology, and identification of other vascular lesions. Conventional arteriography fails to demonstrate some vascular mural changes that may intervene in the development of clinical manifestations, such as intraplaque hemorrhage and thrombus attached to the arterial wall. Mural hemorrhage has been associated with plaque growth and increased degree of plaque-induced stenosis of the arterial lumen.\(^5\) Intraplaque hemorrhages have also been directly implicated in precipitation of ischemic cerebral events through a reduction in flow when the plaque protrudes into the lumen and as a result of the breakdown of the luminal surface with release of embolic material\(^5\) (Class III). Mural changes may be identified with duplex ultrasound and CT arteriography.

As stenosis approaches the near-occlusion category, the diameter of the ICA beyond the stenosis becomes reduced, making the ratio of measurement fallaciously low.\(^5\) In these circumstances it is important to assess the presence of intracranial collaterals (or contrast dilution from collaterals), the delay of intracranial arterial filling in comparison with the external carotid scalp branches, and any obvious poststenotic reduction in the internal carotid lumen. In the NASCET analysis such stenoses were arbitrarily designated as 95\%, reflecting both the degree of near-occlusion and the fact that a precise measurement calculation could not be made.

Patients with TMB or TIA are candidates for cerebrovascular arteriography when there is reasonable evidence of carotid atherosclerotic disease that warrants consideration of
endarterectomy. Evidence of luminal reduction ≥ 70% on ultrasound, MRA, or CT arteriography is usually a strong indication for carotid arteriography in symptomatic patients who can tolerate and consent to surgery. Some stenotic lesions measured at 50% to 69% by noninvasive techniques may show luminal reduction ≥ 70% on conventional angiography, making them eligible for endarterectomy. Conversely, occlusion on noninvasive testing may correlate with a residual hairline lumen on arteriography in as many as 9% of patients (Class II). The distinction between occlusion and hairline patency is important because endarterectomy is not performed on total occlusions of uncertain age. Most of the benefit of using noninvasive testing to screen for disease accrues to patients with negative noninvasive test results because the alternative is to proceed to arteriography for all patients, a pathway to be discouraged because of the increased cost and risk.

Arch aortography with an arterial catheter seldom produces images accurate enough to substitute for selective studies of the carotid artery in the neck; in addition, cranial views are limited. On the other hand, the time for examination is shorter than in selective angiography, and the risk for embolization may be reduced. Arch aortography may assist in evaluation of aortic atherosclerosis. Intravenous digital angiography is of insufficient quality to warrant use of this technique.

Arteriography also is required to evaluate intracranial stenosis or occlusion. Other noninvasive, though less precise, diagnostic modalities (MRA, transcranial Doppler ultrasound) are available to evaluate intracranial vessels. Precise definition of intracranial atherosclerotic stenosis may only be done by selective intra-arterial cerebral arteriography, but the diagnosis does not modify initial medical therapy for TIAs as delineated by most physicians, so that the test is often not pursued for this indication.

Selective subclavian and/or vertebral radiographic angiography remains the reference standard procedure for imaging of vessels in vertebrobasilar occlusive disease. The edges of arteries that are diseased, whether from stenotic or occlusive atherosclerosis, dissection, or arterial dysplasias, can be delineated more sharply than by current MRA techniques. However, because specific interventions are not common for reconstruction of the arterial wall in stenoses of the vertebrobasilar circulation, the holoangiography MR approach, if done well, is sufficient in most cases (level of evidence III, grade C) (Class III, type C). Endovascular therapeutic techniques such as angioplasty for elimination of arterial stenosis or lysis of intraluminal clot with thrombolytic substances are innovative therapies that depend on preliminary angiography; it is as yet uncertain whether MRA allows identification of intraluminal clot that could be treated similarly. These techniques are investigational, and data are not available to make clinical recommendations.

Multiple safeguards should be used to minimize potential complications of arteriography. Patients should be well hydrated before the procedure. The guidewire should not remain in the catheter more than 90 seconds. With a judicious protocol that includes reasonable precautions and careful hook-up to a mechanical contrast injector, serious embolic complications can be kept to less than 1%, provided that ongoing patient volume is sufficient to maintain a uniform quality effort. In the Asymptomatic Carotid Atherosclerosis Study (ACAS), the arteriographic complication rate for cerebral infarction was 1.2%. Angiography teams that perform few cerebral studies work at a lower vigilance level than usual for teams performing a
substantial amount of neuroangiography.

When factors beyond diagnostic reliability are tabulated, conventional radiographic arteriography loses ground vis-à-vis other noninvasive diagnostic tests and combinations in situations in which surgical reconstruction of the cervical carotid artery is being considered. Clinicians may want to consider prompt availability, cost, and risk in addition to diagnostic reliability. The panel has relied heavily on diagnostic reliability in formulating the recommendations for imaging of cerebral vessels but acknowledges that these factors may play a role in setting indications for a diagnostic test(s). The diversity of quality, availability, timeliness, and cost of tests should be weighed in the decision-making process by each individual center.

Exclusions: Coagulopathies, dehydration, renal failure, multiple myeloma, and advanced femoral-aortic atheroma may severely increase risk associated with arteriography.

**Recommendations**

**Imaging of the Brain**

- There is general agreement that patients with manifestations suggestive of hemispheric TIA should receive a CT scan of the head in the initial diagnostic evaluation to exclude a rare lesion such as a subdural hematoma or brain tumor responsible for symptoms (Class III, type C). CT may reveal an area of brain infarction appropriate to TIA symptoms in 29% to 34% of patients, a finding that may influence subsequent management, especially the timing of an eventual carotid endarterectomy (Class III, type C). CT of the head has only a limited role in evaluation of patients with TMB (Class III).

- Despite a slight advantage of MRI over CT in detection of brain infarction appropriate to hemispheric symptoms of ischemia, substitution of MRI for CT in initial evaluation of patients with TIA is not warranted. The panel recognizes that this is a subject of considerable dissension. MRI may be considered when a CT scan fails to substantiate the clinical diagnosis or if additional diagnoses require confirmation or exclusion (Class III). There is also advantage in identifying lesions such as subdural hematoma that may be isodense with surrounding parenchyma on CT imaging as well as in AVMs that rarely present with hemispheric TIAs (Class III, type C).

- CT of the head has a limited role in evaluation of patients with vertebrobasilar TIAs, as subdural hematoma or brain tumor are not known to present with transient symptoms resembling posterior circulation ischemia (Class III). CT can detect areas of appropriate cerebellar or, less commonly, brain stem infarction, a finding that in selected instances may alter clinical management. In addition, CT may show evidence of severe atherosclerotic disease in the vertebrobasilar system, such as dolichoectasia of the basilar artery, as a potential mechanism of TIAs (Class III).

- The routine use of MRI in evaluation of patients with vertebrobasilar TIAs is not justified (Class III) vis-à-vis general management, despite its advantages over CT in detection of lesions potentially related to the mechanism of posterior circulation TIAs, such as atherosclerotic tortuosity, stenosis, or occlusion of the basilar artery (Class III). Occult brain infarction is better identified by MRI in the vertebrobasilar territory, a finding that may provide additional information about the source of the TIA.
**Imaging of the Vessels**

A noninvasive screening technique is indicated as an initial diagnostic test in most patients with TIA, in particular for the study of vessels involved in causing symptoms of carotid hemispheric or retinal ischemia. Many specialized centers use carotid duplex or Doppler ultrasonography, a technique discussed in another set of practice guidelines. MRA provides noninvasive imaging of extracranial carotid, vertebrobasilar, and major intracranial vessels but leads to overestimation of degree of arterial stenosis so that its role in evaluation of patients with TIA has limitations (Class II). There is general agreement, however, that high-quality MRA provides a sufficient vascular overview of the extracranial and intracranial circulations for independent evaluation of vertebrobasilar ischemia. Contrast-enhanced CT scanning of the cervical vessels with helical methodology, in particular, images the arterial wall as well as the lumen and may be helpful as a screening tool in centers where it is available (Class III).

There is general agreement that radiographic arteriography best defines surgically remediable lesions in the accessible, extracranial segment of the carotid artery. Radiographic arteriography is generally recommended for a symptomatic patient when noninvasive tests indicate ≥ 70% occlusion in the appropriate carotid artery and exclusions do not apply. In some instances complete occlusion by noninvasive tests may need confirmation or exclusion by conventional arteriography. The NASCET and ECST have proved the benefit of endarterectomy in patients with symptomatic arterial stenosis with ≥ 70% luminal reduction as measured on cerebral arteriographic images (Class I). Cerebral arteriography may also be required when a diagnosis of dissection, vasculitis, aneurysm, or embolism needs confirmation or exclusion.

**Acute Stroke**

Stroke continues to be a leading cause of morbidity and mortality in the United States. Growing interest in the appropriate management of stroke during the critical early period of evolution along with better organization of healthcare delivery have improved the neurological and functional outcome of stroke victims while reducing mortality and cost. Clinical pathways, practice guidelines, stroke units, and stroke teams have revolutionized the delivery of care to patients with acute stroke, bringing about most of these improvements in clinical outcome. The introduction of powerful diagnostic imaging modalities and aggressive therapeutic protocols has focused attention on the need to streamline diagnostic imaging options while maintaining standards of care that are cost-effective, available, reliable, and necessary. Since 1993 the AHA has included emergent stroke care as part of the cardiopulmonary resuscitation effort and has published practice guidelines for treatment of patients with acute ischemic stroke within the first 24 hours. This section of the guidelines builds on previous AHA guidelines and scientific statements while recognizing that the rapid development of new imaging modalities and the demands of aggressive therapies require that the guidelines be updated periodically to maintain their usefulness. These guidelines provide recommendations for care within the first 72 hours of stroke evolution, when therapeutic intervention is most critical and determines the patient's early prognosis. Recent data indicating therapeutic efficacy of thrombolytic agents when administered intravenously within 3 hours of onset of stroke symptoms emphasize that the time window for evaluation of stroke has become shorter and may require the development of even more efficient imaging techniques and procedures.
Other practice guidelines\textsuperscript{1,62} include information about symptoms and common patterns of neurological abnormalities in stroke, as well as a perspective on requirements for diagnostic imaging in the context of other clinical evaluations, including cardiac imaging.\textsuperscript{63,64} (See Table 2.) As stated in the ACC/AHA guidelines for the clinical application of echocardiography, for cerebrovascular events:

TEE [transesophageal echocardiography] is more sensitive for identifying potential cardiac sources in a patient with an embolic event than TTE [transthoracic echocardiography]. However, if a potential cardiac source is found by TTE in a patient with an embolic event, the additive cost and inconvenience of TEE is probably not warranted. Conversely TEE is uniquely suited for detection of left atrial spontaneous contrast, left atrial thrombi, atrial septal aneurysms, and aortic atheromata. \textsuperscript{65,p872}

**Procedures**

**Imaging of the Brain**

The overriding objectives of the emergent evaluation in acute stroke are to exclude a nonvascular lesion as the cause of the manifestations and to determine whether the stroke is an ischemic infarction or an intracranial hemorrhage. Additional objectives are to localize the lesion, determine its age and extent, and gather documentation on its mechanism. This information will guide the early therapeutic steps and assist in determining an early prognosis. Imaging of the brain should be available, agile, cost-effective, and harmless to the patient.

**Computed Tomography.** CT of the head is indispensable for the emergent evaluation of patients with acute stroke. It is useful for excluding or documenting ICH as the stroke mechanism and to identify other features that directly or indirectly impact the diagnostic evaluation and management of stroke. Use of a third- or fourth-generation CT scanner is recommended.

CT imaging can document several specific features of acute ischemic stroke:

- The anatomic localization and extent of the acute infarct. Detection by CT largely depends on the time between stroke onset and CT examination\textsuperscript{66} and, to a lesser extent, the pale or hemorrhagic character of the infarct\textsuperscript{67} as well as its site and topography (large superficial cortical versus small deep lacunar).\textsuperscript{66} Large cortical surface infarcts are often not documented by CT before 3 hours from onset,\textsuperscript{20} but nearly 60% can be detected by 24 hours.\textsuperscript{68} The diagnostic yield of CT increases progressively, and by 7 days virtually 100% of infarcts that will appear on CT will be documented.\textsuperscript{66} In patients with hemorrhagic infarcts generally due to cerebral embolism or, less commonly, venous infarction, the irregularly hyperdense, "mottled" character of the infarct can at times be demonstrated within 24 hours from stroke onset.\textsuperscript{69}

- Signs of cerebral infarction may be identified within 5 hours from onset of symptoms (even as early as 2 hours\textsuperscript{20}), but they can be subtle.\textsuperscript{71} The most common early signs are an "obscuration" of the lentiform nucleus\textsuperscript{72} and, especially, the loss of the "insular
ribbon." These signs refer to a lack of clear definition of the lentiform nucleus and a loss of the normal white-gray matter distinction in the lateral margins of the insula (the insular ribbon) (Class III). These CT-detected anatomic changes are thought to reflect early ischemic edema in infarcts in the middle cerebral artery (MCA) or ICA distribution.

- Early mass effect in large cerebral hemispheric or cerebellar infarcts can be detected on initial CT performed within hours from onset, in particular in instances of massive infarcts in the MCA territory and, less commonly, in large cerebellar infarcts in the posterior-inferior cerebellar artery distribution. Large MCA territory infarcts often show early effacement of cortical sulci, at times with effacement of the body of the lateral ventricle, before demonstration of the hypodensity of infarction. Posterior-inferior cerebellar artery territory infarcts can show early effacement of cerebellar folia and the ipsilateral quadrigeminal cistern, along with mass effect on the fourth ventricle. These early CT findings (before development of hypodensity) in association with a consistent clinical picture are indicative of massive infarction, a feature that is generally considered a contraindication to immediate use of heparin as an anticoagulant because large infarct size is a risk factor for hemorrhage after embolic cerebral infarction. A similar contraindication may apply to the use of tissue plasminogen activator (TPA) even within the accepted therapeutic window of 0 to 3 hours from stroke onset because early CT signs of massive MCA distribution infarction correlate with poor prognosis, mainly as a result of an increased frequency of hemorrhage into the infarcted tissue. Furthermore, the presence of early signs of mass effect may alert the clinician to the potential development of symptomatic postinfarct edema, and the institution of antiedema measures (diuretic therapy, hyperventilation) may at times be justified.

- Images suggestive of intracranial vascular embolic material can be shown by noncontrast CT on initial presentation with stroke. These can adopt the form of calcified or simply hyperdense structures in a major intracranial artery, most commonly the MCA stem. At times a long, cordlike, hyperdense clot fills the MCA stem (referred to as the hyperdense MCA sign) in the setting of either acute carotid occlusion (where the MCA clot likely represents an artery-to-artery embolus) or, less often, cardiogenic embolism. The demonstration of intracranial images suggestive of clot can, in the appropriate clinical setting, be followed by therapeutic efforts directed at either lysing the clot (by administering intravenous TPA) or preventing further embolization or progression of the thrombus (by anticoagulation with intravenous heparin).

- Demonstration of associated lesions such as AVM or aneurysm, which may contraindicate some forms of acute stroke intervention (anticoagulation, fibrinolysis).

- Documentation of silent cerebral infarcts may provide indirect clues to the mechanism of the presenting stroke by suggesting either a proximal (ie, cardiac, aortic) source of embolism in the event of finding multiple bilateral infarcts in different vascular territories or an ipsilateral carotid source in the face of multiple infarcts limited to the territory of one ICA.

The use of intravenous contrast infusion for CT evaluation of acute ischemic stroke is controversial. There is no evidence that use of contrast increases the yield of CT in patients with acute ischemic stroke, and there is a theoretical concern about promoting cerebral
"toxicity" in the face of an acutely disturbed blood-brain barrier in large infarcts. In the subacute stage 2 to 3 weeks after stroke onset, when the "fogging effect" may obscure the demonstration of cerebral infarction, postcontrast enhancement can be valuable in documenting infarction as it occurs in 60% to 65% of patients at this stage of stroke evolution. The pattern of enhancement is generally homogeneous in deep infarcts, gyral in cortical surface infarcts, and irregular and mottled in large areas of hypodense infarction.

CT has virtually a 100% yield in diagnosis of acute ICH. Rare exceptions are petechial hemorrhages and isodense acute hemorrhages in anemic patients with very low (<20%) hematocrit. CT has the capability to identify lesion topography and associated features, including mass effect, intraventricular extension, and hydrocephalus. On occasion the differential diagnosis between ICH and heavy parenchymal calcification (in a meningioma, for example) is raised in patients with an acute stroke syndrome. The distinction can generally be made with certainty by measuring the attenuation coefficient of the lesion in Hounsfield units (HU), because fresh blood has values in the order of 50 to 85 HU, whereas calcium deposits have attenuation values in the range of 70 to 200 HU.

The CT features of ICH vary, depending on the time of evolution of the hematoma. In the hyperacute stage (up to 4 hours from onset), the extravasated blood has not yet clotted, producing a collection of irregular hyperdensity within the brain substance with associated local mass effect. The scanning of an ICH in its very early course may show fluid-blood levels, which should be interpreted as a sign of very recent ICH, bleeding into a preexisting cavity (or cyst), or even as bleeding in coagulopathy secondary to thrombolytic therapy. In the acute stage (5 to 72 hours from onset), the ICH becomes denser as a result of clot formation and gradual extrusion of serum producing a relative increase in hemoglobin concentration that results in a higher CT attenuation value. The extruded serum produces the appearance of a thin halo of hypodensity surrounding the hyperdense hematoma.

In addition to these features related to the stage of evolution of the ICH, CT accurately documents its location (deep nuclear structures, subcortical white matter, posterior fossa), as well as the presence of mass effect, ventricular extension, and hydrocephalus. All these factors relate to the potential mechanism of ICH and are important considerations for the diagnostic workup and treatment of patients with ICH as follows:

- Deep basal ganglionic hemorrhage is usually due to hypertension.
- Basal frontal ICH is often due to head trauma or ruptured aneurysm, the latter in particular when associated with blood in the basal subarachnoid space and/or the interhemispheric fissure.
- An acute ICH with marked mass effect and especially a large area of surrounding hypodense edema should raise suspicion of an underlying tumor with superimposed bleeding.
- Lobar hemorrhages can be associated with cerebral amyloid angiopathy (in the elderly), ruptured vascular malformations (in generally normotensive persons), sympathomimetic drug effect (in young and middle-aged patients), or use of thrombolytic or anticoagulant agents.
- Cerebellar hemorrhages with mass effect in the posterior fossa and supratentorial hydrocephalus should be considered for emergency surgical evacuation. 94

- Patients with thalamic hemorrhage and lateral ventricular hydrocephalus may benefit from urgent ventriculostomy. 95

In subdural hematoma with acute strokelike presentation, CT should readily clarify diagnosis by showing an extracerebral hyperdense or hypodense collection, depending on the acute or chronic character of the hematoma, respectively.

To summarize, a noncontrast CT scan of the head should be obtained as part of the initial evaluation of a patient with stroke to document or exclude ICH (near 100% sensitivity) and subarachnoid hemorrhage (96% sensitivity) (Class II) (Tables 3 and 4).

Documentation of ischemic infarction within 3 hours of onset is uncommon (Class II) but more likely in instances of hemorrhagic infarction and massive MCA territory infarctions (Class II). Despite a normal CT scan within hours of stroke onset, follow-up CT 24 hours later or thereafter will show an appropriate area of hypodensity in 60% of cases (Class II).

Exclusions: Pregnancy is a relative contraindication because appropriate shielding can be used to diminish the effects of scattered radiation from the highly collimated beam to the head.

**Magnetic Resonance Imaging.** MRI has a higher yield than CT in the early documentation of infarction, as well as in identification of a hemorrhagic component and the presence of associated features, including mass effect. The use of different combinations of relaxation time (TR) and echo time (TE) result in various MRI sequences that are useful in diagnosis of acute cerebral infarction. They include T1-weighted (short TR, short TE), T2-weighted (long TR, long TE), and "proton-density" (long TR, short TE) sequences. These sequences produce different signal intensity changes depending on the stage of evolution of the cerebral infarct and the presence of some associated features (Tables 5 and 6).

High field strength (1.0 to 1.5 Tesla) magnet MRI is superior to CT in the very early documentation of acute infarction. Echoplanar MR diffusion and perfusion MR may document changes within minutes of the occurrence of stroke. Although changes have been reported as early as 3 hours from stroke onset with conventional high-field MRI, about 15% of patients will show changes in MRI signal after 8 hours and 90% after 24 hours. The changes consist primarily of increased signal in T2-weighted sequences because the T1-weighted sequences have lower sensitivity (about 50%) during the first 24 hours after stroke onset. The findings apply to both large cortical surface infarcts and small deep lacunes in the anterior and posterior circulation, the latter being an area of particularly low diagnostic yield with CT.

Other early MRI signs supportive of the diagnosis of infarction, such as absence of flow void in proton-density sequences of intracranial arteries suggestive of arterial occlusion, can be detected almost immediately after stroke onset. This finding can be further substantiated within the first 24 hours from onset by the appearance of high signal in the affected vessels in the T1-weighted images, indicating the presence of intraluminal clot. Another early MRI sign of recent infarction is effacement of cortical sulci suggesting mass effect, which can be seen in the majority of patients before 24 hours from onset and which generally precedes the development of parenchymal changes in T2-weighted sequences.
MRI may be used to confirm or rule out ICH as the mechanism of an acute stroke. Because of the technical aspects of MRI signal generation, interpretation of findings in acute ICH may at times be difficult, and the diagnosis may not be as straightforward as with CT. MRI may detect areas of hemorrhagic transformation (ie, hemorrhagic infarction) within the infarct earlier than CT. They appear as high-signal changes in T1-weighted sequences, whereas in T2-weighted sequences they are characterized by patchy areas of low signal within the background of typical high-signal intensity infarction. MRI is also more sensitive than CT in documentation of associated lesions such as AVM, aneurism, or old infarcts, the latter especially in the posterior fossa. Contrast-enhancement with gadopentetate dimeglumine, although not necessary for routine evaluation of acute cerebral strokes, can document "slow flow" intravascular enhancement of the affected artery ("intravascular enhancement sign") (Class II) as early as the first hour postictus and parenchymal and meningeal enhancement at 48 hours.

In evaluation of patients with ICH, MRI has the advantage over CT in its capability to date the event accurately and to reveal associated lesions that may suggest the mechanism of ICH. MRI is also superior to CT in detecting petechial hemorrhagic infarction.

Accurate determination of the phase of evolution of an intracerebral hematoma is based on the ability of MRI to detect the presence of various stages of chemical transformation of the hemoglobin molecule. These changes occur in a predictable sequential pattern in the evolution of the parenchymal hematoma. The hemoglobin transformation sequence--oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin--gives specific signal changes on MRI. Each changes the T1 and T2 signal intensity in a predictable manner. These patterns are listed in Table 7.

MRI is also useful for detection of associated lesions that may be related to the mechanism of ICH. When intracranial tumors are the cause of ICH, they produce an atypical pattern of evolution of MRI changes of hemoglobin transformation. These often take the form of multiple simultaneous stages of evolution within the lesion, delayed evolution of signal changes with time, lack of well-defined hemosiderin ring in the late subacute to chronic phase, and persistence of perilesional T2-weighted hyperintensity into the chronic stage, representing edema and/or tumoral infiltration.

Vascular malformations in the vicinity of an acute ICH can be accurately detected by MRI in the form of serpiginous flow voids in the case of AVMs or as an irregular, mottled bright signal lesion with a peripheral hypodense hemosiderin "ring" in cavernous angiomas. Both are best depicted in T2-weighted images. Although some AVMs can be diagnosed equally well by angiography, small malformations and cavernous angiomas often escape angiographic detection, and their presurgical or premortem documentation is possible only with MRI.

In summary, MRI is particularly useful for demonstration of posterior circulation strokes, identification of small hemorrhage, or when dating of the hemorrhage is indicated. It is more sensitive than CT (90%) for detection of cerebral ischemic infarction within the first 24 hours of stroke onset (Class II), for early documentation of hemorrhagic infarction (Class II), and to show early signs of postinfarct brain edema and mass effect (Class II). Limitations of MRI are cost, availability, decreased resolution of early ICH when compared with CT, and claustrophobic reactions.
Exclusions: Patients with intraorbital or intracranial ferromagnetic fragments, aneurysm clips, otic or cochlear implants, old prosthetic heart valves, pacemakers, and neurostimulators. Agitation and severe claustrophobia constitute an exclusion unless resolved.

**Imaging of the Vessels**

Acquiring images of the cervical-cerebral vessels appropriate to acute stroke provides information that might be essential for understanding the mechanism of stroke. Uncovering a tight stenosis, occlusion, dissection, or vascular anomaly may explain the events that led to the stroke. This information is desirable for treatment of the patient after stabilization and should be obtained as early as possible to prevent future strokes. Clinical considerations made in the presence of small strokes and diagnostic techniques recommended are the same ones reported for evaluation of patients with transient ischemia; the advantages and risks are similar except for the variables introduced by the patient's clinical situation. At present there is no routine recommendation for the emergent use of methods that visualize the vessels in acute stroke because no emergent indications are derived from the information obtained. This state of affairs may change in the near future as therapeutic modalities with brief application windows, such as intra-arterial thrombolytic therapy, are introduced for management of acute stroke.\textsuperscript{104,105}

MRA serves as a screening procedure that may help explain the vascular mechanism of strokes and should be considered when that information is clinically relevant and not otherwise available. Compared with conventional angiography, MRA is less costly and much safer but less accurate.\textsuperscript{36} The risks associated with conventional angiography, including vascular damage, ionizing radiation, stroke, and systemic reactions to contrast agents are thus avoided. Furthermore, it may be used when conventional radiography is compromised by organ failures, such as poor renal function, or in multiple myeloma, coagulopathies, absent femoral pulses, and severe, generalized atherosclerosis.

MRA can show stenotic and occlusive lesions of important neck and brain vessels. For large ischemic strokes, surgical reconstruction of vessels is seldom a consideration, and intra-arterial thrombolytic therapy remains an investigational procedure, so angiography by any modality is seldom required in the early stages of stroke.

MRA can show aneurysms\textsuperscript{106} >5 mm in diameter,\textsuperscript{107} but the critical relationship of the neck of the aneurysm(s) with adjoining branches, including small perforators, requires high-resolution radiographic angiography. In subarachnoid hemorrhage conventional angiography is required before surgery, but when the MRA result is negative, conventional angiography is also needed to rule out small aneurysms or to demonstrate small arteriovenous dural fistulas that can drain to pial veins and occasionally present with subarachnoid hemorrhage. For intracranial aneurysms, time-of-flight MRA has higher sensitivity and specificity\textsuperscript{108} compared with phase-contrast MRA.

The limitations of MRA are similar to those for MRI. The intravenous use of gadopentetate dimeglumine, a paramagnetic contrast agent, has been associated with minor renal and general adverse effects in 1% of those receiving it in safety studies involving 13 000 patients.\textsuperscript{109} The estimated anaphylactoid reaction rate including fatal outcome is 1:450 000.\textsuperscript{110} Limitations related to the accuracy of the images obtained have been described in other sections and apply...
here as well.

On the basis of precise anatomic visualization requirements, carotid angiography continues to be the definitive preoperative study for carotid endarterectomy and in patients with arterial aneurysms and AVMs.

In summary, after the emergent phase in patients with acute stroke, MRA serves as a screening procedure for consideration of carotid angiography and serial monitoring of vascular abnormalities. \textsuperscript{111}

At present there is no routine recommendation for performance of conventional radiographic angiography in patients with acute stroke. This recommendation may change should experimental intra-arterial therapeutic interventions prove to be valuable in reversing acute stroke.

SPECT is not widely used in the clinical management of stroke. It may assist in early differentiation of lacunar stroke from cortical stroke when the results of CT and MRI are negative,\textsuperscript{112} and it may have a future supportive role in evaluation of early thrombolysis after ischemic stroke.\textsuperscript{113}

**Recommendations**

- There is general agreement to strongly recommend CT of the head without contrast enhancement as the initial brain imaging procedure in patients with acute stroke (type A).

- A follow-up CT of the head without contrast enhancement 2 to 7 days after stroke onset is recommended when the initial CT scan result is negative and documentation of the presence, location, and extension of the ischemic infarction is needed (type B), or when clinically significant hemorrhagic transformation is suspected.

- MRI of the brain is not recommended for routine evaluation of patients with acute stroke. Despite its imaging advantages, MRI of the brain is not necessary to initiate emergent treatment in the majority of patients with acute stroke; when available, MRI is an appropriate imaging alternative. The panel recognizes that special circumstances may co-occur in some patients (posterior fossa localization, suspicion of dissection, underlying lesion, age of cerebral hemorrhage, uncertain CT image) that drive the need to obtain an MRI image of the head. The decision must be made on an individual basis in accordance with specific clinical situations (type B).

- Imaging of cervicocerebral vessels intended to establish a probable etiology of acute stroke is generally not necessary to initiate emergent management, and the test (ultrasound, MRA, CT angiography, conventional angiography, SPECT) should not delay treatment. When indicated, these procedures should be tailored to specific requirements. Information on carotid or intracranial occlusive disease may guide decisions on cardiac workup, anticoagulation, or carotid endarterectomy for prevention of future strokes. Ultrasound will generally suffice, but conventional radiographic angiography may be occasionally indicated, based on findings of noninvasive screening procedures.
Special Considerations

Subarachnoid Hemorrhage

The initial clinical presentation of subarachnoid hemorrhage (SAH) with focal neurological deficits may be confused with acute stroke. Noncontrast CT scan of the head is the cornerstone of diagnosis of SAH.\textsuperscript{114} It reliably detects SAH at onset and will reveal within 24 hours a high-density clot in the subarachnoid space in 92\% of cases.\textsuperscript{115} The early high rate of positivity of CT in SAH gradually declines to about 50\% 7 days after onset.\textsuperscript{116,117} The usefulness of MRI in diagnosis of SAH is controversial, and there is concern about the ability of MRI to identify subarachnoid blood in the acute stage. Selective catheter cerebral angiography is the recommended procedure for diagnosis of cerebral aneurysm as the cause of SAH.\textsuperscript{62}

Ischemia from vasospasm is a major cause of morbidity and mortality after SAH.\textsuperscript{118} Diagnosis and monitoring of vasospasm is done with transcranial Doppler echocardiography, which assesses the main arteries at the base of the brain. High-resolution conventional angiography is needed to demonstrate the extent and severity of vasospasm, both in major cerebral vessels and more distal vessels.

For further information, the reader is referred to the guidelines for management of SAH published by the AHA Stroke Council.\textsuperscript{62}

Recommendations

- Noncontrast CT of the head is strongly recommended as the initial procedure for diagnosis of SAH (type A).
- Selective catheter cerebral angiography is the recommended procedure for diagnosis of cerebral aneurysm as the cause of SAH (type A).

Arteritis and Other Arteriopathies

The objective of imaging of the brain in patients with cerebral vasculitis is to obtain information on the topography of brain lesions and to depict vascular changes in large and medium vessels.\textsuperscript{119} In general, MRI with MRA is most helpful because MRI is more sensitive than CT in identifying small ischemic lesions\textsuperscript{96} that are common in vasculitis. MRI is also helpful for detecting gliosis in areas of partial ischemia and to date coexisting hemorrhagic disease. MRA can provide screening information on the caliber and patency of proximal large vessels, but precise information should be garnered from conventional radiographic angiography, which offers the advantage of detecting appropriate pathology in medium vessels.\textsuperscript{120}

Small-vessel arteritis (polyarteritis nodosa and systemic lupus erythematosus [SLE]) is not detectable on MRA and often not on conventional angiography. Areas of infarction can be shown on CT or MRI. In SLE, particularly in young patients with hypertension from renal disease, areas of increased T2 signal on MRI or abnormal hypodensity on CT, especially in the posterior circulation, may be related to increased capillary permeability and not cerebral
Infarction. Vanishing areas of ischemia on SPECT with normal MRI have been correlated with transient clinical symptoms in SLE.

Ischemic disease of the brain is one of the hallmarks of the syndrome of mitochondrial encephalomyopathy with lactic acidosis and strokelike episodes (MELAS). The ischemic lesions, often involving the occipital lobes, can be identified on MRI or CT. They appear larger and correlate better with the clinical findings when imaged by SPECT. Angiography is generally negative and does not add to the diagnosis.

**Recommendations**

- In patients with stroke and suspected cerebral arteritis, MRI is generally recommended (type B), because MRI is more sensitive than CT for identifying small ischemic lesions that, being common in vasculitis, may advance the etiologic diagnosis.
- Conventional angiography is recommended to detect beading, stenosis, or aneurysm, particularly in medium and small cerebral vessels affected by vasculitis (type C).

### Dural Sinus and Venous Thrombosis

This condition may be suspected in patients with encephalopathy of acute or subacute onset who present with ICH, cerebral infarction, headaches, or unexplained seizures. Dural sinus and venous thrombosis is a possible complication of severe dehydration; chemotherapy; use of oral contraceptives; hypercoagulable states, including those associated with pregnancy; and mastoiditis. Cross-sectional imaging with noncontrast CT shows ischemic or hemorrhagic areas distributed almost symmetrically on both sides of the brain as the result of venous hypertension occurring with thrombus in deep or cortical veins or dural sinuses. Compression of ventricles and brain edema may also be observed on noncontrast CT. As many as 25% of instances of cortical vein thrombosis may be overlooked by CT (Class III). Contrast-enhanced CT may show sagittal sinus thrombosis (delta sign), but the false-negative rate is high at 77% (Class III). MRI combined with MRA reliably diagnoses dural sinus, deep vein, and large cortical vein thrombosis. MR obviates bone artifact and shows flow void patency of dural sinuses, deep veins, and large cortical veins. Thrombosed veins usually project a T1-hyperintense signal, whereas dilated collateral nonthrombosed veins appear as prominent signal voids; however, acute and early subacute vascular thrombosis may mimic normal flow in MR images. In acute stages MRI may show blood in deoxyhemoglobin or intracellular methemoglobin phases in T1-weighted sequences appearing as hypointense or hyperintense images. With little additional time, MRA can be added to show the patency status of major dural sinuses, such as the sagittal sinus, transverse sinuses, or deep cerebral veins, in which increased signal manifests normal flow. If available, three-dimensional phase-contrast MRA is also useful for differentiating flow from thrombus formation. Isolated cortical vein thrombosis without major dural sinus thrombosis may be overlooked on MRI and even on conventional angiography, but the diagnosis is suspected by demonstrating abrupt termination of a cortical vein as it advances toward the draining sinus.

Before the advent of MRI and MRA, conventional angiography was the only method for
diagnosis of cortical or dural sinus thrombosis and may still be used when these procedures are not available.

**Recommendations**

- There is general agreement in recommending MRI of the brain when thrombosis of a dural sinus, deep vein, or cortical vein is suspected as the cause of cerebral stroke (type B). Sagittal and coronal T1-weighted images, T2 and gradient echocardiographic axial images, and three-dimensional phase-contrast or time-of-flight MRA are suggested.
- Where MRI and MRA are not available, contrast-enhanced CT of the brain and, in particular, conventional angiography are diagnostic options to be used independently or in combination to advance the diagnosis of deep vein, cortical vein, or dural sinus thrombosis (type C).

**Arterial Dissection**

Arterial dissection is frequently responsible for stroke in young persons. Sudden onset of neck pain and headache in the retro-orbital region or nape of the neck are characteristic of carotid and vertebral dissection, respectively.\(^{128}\) MRI is the initial procedure of choice when arterial dissection is suspected, showing the cross-section of the dissected vessel with a narrow lumen and a thickened wall with high signal on T1-weighted images. On occasion it may be difficult to distinguish the appearance of a thrombus in the vessel from the appearance of dissection. In these instances MRA may be helpful by showing a long stenotic segment with tapered ends typical of arterial dissection. In the cervical ICA, dissection stops abruptly as the vessel enters the foramen lacerum. In 20\% of patients with cervical artery dissection, MRI will not demonstrate a typical abnormality.\(^{129}\) The sensitivity of MRI is higher in internal carotid than in vertebral artery dissection.\(^{129}\) In carotid dissection at the neck, CT angiography or duplex sonography also may be helpful as noninvasive screening procedures.\(^{48,129,130}\) Conventional angiography may be performed to verify the diagnosis if MRI/MRA, CT angiography, or ultrasound evaluation is unclear.\(^{131}\) In young adults radiographic angiography is technically simple and has a low risk.

**Recommendations**

- There is general agreement for recommending CT of the head in early evaluation of young adults with acute stroke (type A).
- When arterial dissection is suspected, MRI/MRA of the head and neck are generally useful to screen for arterial dissection (type C).
- Conventional radiographic angiography may be done if the diagnosis from MRI/MRA is unclear and there is strong suspicion of cervico-cerebral dissection. The procedure is technically simple and low risk in young adults (type C).

**Acute Spinal Cord Stroke**

The diagnosis of spinal cord infarction is suspected in patients with acute myelopathy,\(^ {132}\) often in conjunction with aortic abdominal aneurysm or spontaneous dissection of the aorta. True
spinal stroke may be due to hemorrhage of a spinal cord AVM, ischemic infarction caused by embolus, occlusive arterial disease of the primary radicular feeder(s) to the anterior spinal artery, or acute spinal venous thrombosis. High-resolution MRI is the most appropriate technique in the acute stage \(^{133-135}\) (Class III). As in brain infarction, MRI may show signal changes and some swelling in the cord only after an interval of a few hours. Although the imaging findings may be similar to acute transverse myelitis, the clinical presentation helps to differentiate infarction from myelitis. Spinal MRI is also appropriate for evaluation for the presence of blood and prominent cord vessels that appear in spinal cord AVM and spinal dural arteriovenous fistula. If the presence of prominent spinal vessels is uncertain, good-quality myelography or myelo-CT could be done to advance the diagnosis of AVM.

Selective angiography of spinal cord vessels can be tedious with a low probability of showing important arterial branches to the anterior spinal artery and a good chance of exacerbating ischemia by occluding a stenotic vessel with catheter manipulations. Thus, selective angiography of spinal cord vessels is not recommended in acute spinal stroke. Nonselective aortography does not show the fine details of the spinal cord arterial supply and thus plays no role in evaluation of vascular pathology of the spinal cord.

**Recommendations**

- There is general agreement in recommending MRI for diagnosis of acute spinal stroke (type C).

**Special Recommendations**

**Pregnancy and Women of Childbearing Age**

Thromboembolic stroke in pregnant women or those taking oral contraceptives is well recognized.\(^ {136}\) It should be distinguished from hemorrhagic stroke secondary to ruptured aneurysm or AVM. Thromboembolic infarction can occur at any time during pregnancy and the puerperium, with the highest incidence during the first week postpartum.\(^ {137}\) Cerebral vein thrombosis occurs in 1 to 2 cases per 10 000 childbirths in Western Europe and North America, with the highest incidence during puerperium.\(^ {138}\) Imaging procedures differentiate hemorrhagic from ischemic stroke and may determine the specific etiology.

Imaging studies should be based on neurological indications despite pregnancy. Potential risk of birth defects due to radiation occurs in the first trimester of pregnancy, especially in the first few weeks when the patient may not be aware of the pregnancy. Radiation-protection precautions for the developing fetus should be used whenever the question of pregnancy arises; the lower abdomen can be surrounded by lead aprons, and fluoroscopy of the lower abdomen should be specifically avoided during angiography. CT scanners virtually limit the radiation dose to the tightly collimated slice and the overlapping slice so that more caudal radiation is negligible. MRI produces no radiation, and there is no evidence that the minor heat produced causes damage to the embryo or fetus. The effects of MRI during the first trimester are still unknown, so MRI should be used sparingly with a full explanation to the patient and family members about the need for the study.

**Recommendations**
Use of imaging procedures in pregnancy follows the general rules discussed in previous sections. There are no specific imaging recommendations in pregnancy other than those related to radiation precautions.

**Patients With Allergies to Contrast Solutions**

Among patients receiving ionic, high-osmolality intravascular contrast agents, 5% to 12% will experience some form of adverse reaction,\(^{139,140}\) and in 1 in 1000 administrations the reaction will be life-threatening.\(^{141}\) Low-osmolality, nonionic contrast agents cause fewer adverse reactions. Patients suspected of having an allergy to iodine-containing contrast solutions may receive premedication to prevent anaphylactic reactions. The following protocol is suggested: 50 mg prednisone PO 13 hours, 7 hours, and 1 hour before exam; 50 mg diphenhydramine hydrochloride PO and 300 mg cimetidine hydrochloride PO 45 minutes before exam.

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