



# Neuroimaging of acute stroke

**AUTHORS:** [Jamary Oliveira-Filho, MD, MS, PhD](#), [Maarten G Lansberg, MD, PhD](#)

**SECTION EDITORS:** [Scott E Kasner, MD](#), [Glenn A Tung, MD, FACR](#)

**DEPUTY EDITOR:** [John F Dashe, MD, PhD](#)

---

All topics are updated as new evidence becomes available and our [peer review process](#) is complete.

Literature review current through: **Jan 2024**.

This topic last updated: **Jul 24, 2023**.

---

## INTRODUCTION

Neuroimaging in the evaluation of acute stroke is used to differentiate hemorrhage from ischemic stroke, to assess the degree of brain injury, and to identify the vascular lesion responsible for the stroke. Multimodal computed tomography (CT) and magnetic resonance imaging (MRI), including perfusion imaging, can distinguish between brain tissue that is irreversibly infarcted and that which is potentially salvageable, thereby allowing selection of patients who are likely to benefit from reperfusion therapy. The use of this technology is dependent upon availability.

Neuroimaging during the acute phase (first 24 hours) of stroke will be reviewed here. Other aspects of the acute evaluation of stroke, the clinical diagnosis of various types of stroke, and the subacute and long-term assessment of patients who have had a stroke are discussed separately:

[Initial assessment and management of acute stroke](#)

[Clinical diagnosis of stroke subtypes](#)

[Overview of the evaluation of stroke](#)

[Approach to reperfusion therapy for acute ischemic stroke](#)

[Spontaneous intracerebral hemorrhage: Pathogenesis, clinical features, and diagnosis](#)

[Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis](#)

[Nonaneurysmal subarachnoid hemorrhage](#)

Ischemic stroke in children: Clinical presentation, evaluation, and diagnosis

Hemorrhagic stroke in children

Stroke in the newborn: Classification, manifestations, and diagnosis

---

## APPROACH TO IMAGING

**Goals of imaging** — Neuroimaging should be obtained for all patients suspected of having acute stroke or transient ischemic attack (TIA) [1]. Brain and neurovascular imaging plays an essential role in acute stroke by [2,3]:

- Differentiating ischemia from hemorrhage
- Excluding stroke mimics, such as tumor
- Assessing the status of large cervical and intracranial arteries
- Estimating the volume of brain tissue that is irreversibly infarcted (ie, infarction core)
- Estimating the extent of potentially salvageable brain tissue that is at risk for infarction (ie, ischemic penumbra)
- Guiding acute interventions, including patient selection for reperfusion therapies (ie, intravenous thrombolysis and mechanical thrombectomy)

**Urgency and scope of imaging** — Because "time is brain" and because imaging provides essential information for selecting treatment, immediate imaging of patients with acute stroke is a priority [4]. Brain imaging is required to exclude the presence of acute hemorrhage, because the management of patients with hemorrhagic stroke is very different from that of patients with acute ischemic stroke.

Neurovascular imaging with CT angiography (CTA) or MR angiography (MRA) is necessary for confirming the presence of large artery occlusion in patients who are potential candidates for mechanical thrombectomy. Neurovascular imaging should evaluate the extracranial (internal carotid and vertebral) and intracranial (internal carotid, vertebral, basilar, and Circle of Willis) large vessels. (See "[Approach to reperfusion therapy for acute ischemic stroke](#)".)

Multimodal CT and MRI can identify acute infarction, large vessel occlusion, infarct core, and salvageable brain tissue and are used to select patients for intravenous thrombolysis and mechanical thrombectomy in later time windows. (See '[Multimodal imaging](#)' below and "[Approach to reperfusion therapy for acute ischemic stroke](#)".)

Brain imaging should not be considered in isolation but rather as one part of the acute stroke evaluation. The approach to imaging may differ according to individual patient characteristics

(eg, time from stroke onset or time last known well, potential candidate for reperfusion therapies) and local availability of stroke expertise and imaging capabilities.

**CT or MRI for initial imaging?** — While CT and MRI can both be used for the initial evaluation of patients suspected of an acute stroke, CT with CTA is the standard imaging modality at most centers.

- **Advantages of CT** – CT is used more often than MRI in acute stroke because of its widespread availability, the rapid scan times, and lower cost [5,6]. The noncontrast CT has excellent test performance characteristics for differentiating ischemic from hemorrhagic stroke.
- **Disadvantage of CT** – Although the noncontrast CT can show signs of early acute ischemic stroke, these signs are very subtle and are often absent in the first hours after ischemic stroke onset (see '[Parenchymal changes on CT](#)' below). Because of that, both the sensitivity and interrater agreement for the assessment of early infarct signs on CT are suboptimal. In one report of 786 patients with ischemic stroke, the sensitivity of noncontrast CT during the first six hours of cerebral ischemia was 64 percent, and local investigators reached only a 40 percent sensitivity [7].
- **Advantages of MRI** – A major advantage of MRI is that DWI is much more sensitive than noncontrast CT for detection of acute ischemic stroke and the exclusion of some stroke mimics. This can be particularly helpful when the diagnosis of stroke is in doubt. For example, the absence of a lesion on DWI can suggest that symptoms are caused by a stroke mimic. In addition, MRI does not expose the patient to radiation.

Standard brain MRI protocols that include conventional T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), and T2\*-weighted gradient-recalled echo (GRE) sequences along with DWI can reliably diagnose both acute ischemic stroke and acute hemorrhagic stroke in emergency settings. MRI with T2\*-weighted GRE and susceptibility-weighted imaging (SWI) is equivalent to noncontrast CT for the detection of acute intraparenchymal hemorrhage and is better than noncontrast CT for the detection of chronic hemorrhage [8-10]. (See "[Spontaneous intracerebral hemorrhage: Pathogenesis, clinical features, and diagnosis](#)", section on 'Brain MRI'.)

- **Disadvantages of MRI** – Compared with CT, shortcomings of MRI are higher cost, limited availability and access (particularly in the emergency setting), patient intolerance or incompatibility, and longer scan completion time. There are numerous potential contraindications to MRI including metallic or electrical implants, devices, and foreign bodies. This issue is reviewed in detail separately. (See "[Patient evaluation for metallic or](#)

electrical implants, devices, or foreign bodies before magnetic resonance imaging" and "Patient evaluation before gadolinium contrast administration for magnetic resonance imaging".)

Despite these drawbacks, a trial of patients with a large vessel occlusion found that 88 percent were able to undergo MRI [11], and a few reports have demonstrated that it is possible to use MRI routinely as the sole neuroimaging screening method prior to intravenous thrombolytic therapy [12,13] or endovascular therapy [14]. In one such study of 135 patients screened with MRI and treated with intravenous thrombolysis, quality improvement processes led to reduced door-to-needle times of  $\leq 60$  minutes [12]. These data suggest that MRI can be used as the only imaging method in select centers with sufficient MRI availability for the evaluation of patients with suspected acute ischemic stroke.

**Multimodal imaging** — Assessment of ischemic brain injury and brain perfusion can be performed with either multimodal CT or multimodal MRI if results are likely to influence treatment decisions, such as mechanical thrombectomy in the late time window (ie, >6 hours from stroke onset or from the time of last known well).

- Multimodal CT includes noncontrast head CT with CTA of the head and neck and CT perfusion (CTP). Multimodal CT improves detection of acute ischemic stroke when compared with noncontrast CT alone [15-18]. In addition, multimodal CT can diagnose large vessel occlusion and estimate the core and penumbra of an acute ischemic stroke [19,20]. After endovascular intervention, both hemorrhage and contrast staining of infarcted tissue can occur. Both conditions have the same appearance on standard noncontrast CT. Dual-energy CT can be used to differentiate the two.
- Multimodal MRI includes MRI of the brain without contrast, high-susceptibility imaging (to exclude hemorrhage), MRA of the head and neck, DWI, and perfusion-weighted imaging (PWI). Multimodal MRI can identify acute infarction, emergent large vessel occlusion, infarct core, and salvageable penumbral brain tissue [21-23].

**Time-based selection of imaging** — It is vitally important to determine the time the patient was last known to be well (ie, at neurologic baseline), because the use of reperfusion therapies for ischemic stroke (intravenous thrombolysis and mechanical thrombectomy) are time- and imaging-dependent. (See "[Approach to reperfusion therapy for acute ischemic stroke](#)".)

- **Time last known well <4.5 hours** – For patients with suspected stroke who present in less than 4.5 hours from time last known well, noncontrast head CT with CTA of the head and neck is the preferred imaging option at most hospitals and is sufficient to exclude

hemorrhage and direct treatment with intravenous thrombolysis. The addition of CTA is required to determine the presence of a large vessel occlusion and eligibility for mechanical thrombectomy [24]. An alternative is MRI with high-susceptibility sequence, DWI, and MRA.

- **Time last known well 4.5 to 24 hours** – For patients with suspected stroke who present within 4.5 to 24 hours of the time last known well (including patients with wake-up stroke and other patients with unknown time of symptom onset), the most common imaging choice is multimodal CT, including noncontrast CT, CTA, and CTP [24]. An alternative is multimodal MRI, if available urgently. Both approaches can determine eligibility for mechanical thrombectomy in the extended 6- to 24-hour time window. In addition, perfusion-core mismatch can be used to select patients with wake-up stroke or unknown symptom onset time but within 4.5 to 9 hours of time last known well, who may benefit from intravenous thrombolysis. (See '[Mismatch and salvageable brain tissue](#)' below.)
- **Time last known well unknown** – For patients presenting with an unknown time of stroke symptom onset and unknown time last known well, the first-choice imaging modality is MRI with DWI, FLAIR, and high-susceptibility sequence [24,25]. This can exclude hemorrhage and determine the presence or absence of a DWI-FLAIR mismatch, which is indicative of stroke onset within 4.5 hours and therefore potential benefit with intravenous thrombolysis. (See '[DWI-FLAIR mismatch](#)' below.)

---

## IMAGING FINDINGS OF ISCHEMIC STROKE

### Assessment of early infarct signs

**Parenchymal changes on CT** — In the setting of hyperacute ischemic stroke, the initial head CT study may show either no evidence of ischemic change or may show early infarct signs, which include the following [26-30]:

- Loss of gray-white matter differentiation in the basal ganglia (eg, obscuration of the lentiform nucleus)
- Loss of insular ribbon or obscuration of Sylvian fissure
- Cortical hypoattenuation and sulcal effacement

Early infarct signs can be subtle ( [image 1](#) and [image 2](#)); both under- and overestimation of early infarct signs are common even in a controlled setting [31]. Studies that have examined the ability of neurologists, neuroradiologists, and general practitioners to recognize early infarct signs have shown modest interrater reliability, particularly among clinicians with more limited

training [32]. Nevertheless, the importance of a noncontrast CT interpreted by an experienced reader as not showing signs of acute ischemia should not be underestimated; it excludes a large infarction with high specificity [7].

In a systematic review involving 15 studies where noncontrast CT scans were performed within six hours of stroke onset, the prevalence of early infarction signs was 61 percent (standard deviation  $\pm$ 21 percent) [26]. The sensitivity of noncontrast CT for signs of brain infarction increases over time from stroke onset.

Note that minor ischemic changes (ie, early infarct signs that involve a relatively small volume of brain tissue) on noncontrast CT are **not** a contraindication to treatment with intravenous thrombolysis, nor is the presence of a hyperdense artery sign (see '[Hyperdense artery sign on CT](#)' below) [1]. While the presence of early infarct signs has been associated with an increased risk of poor functional outcome (odds ratio 3.11, 95% CI 2.77-3.49) [26], an analysis from the National Institute of Neurological Disorders and Stroke (NINDS) trial found that early noncontrast CT signs of infarction were not associated with reduced efficacy of intravenous thrombolysis (tissue plasminogen activator [tPA]) treatment; patients treated with tPA did better whether or not they had early CT signs [33]. (See '[Approach to reperfusion therapy for acute ischemic stroke](#)', section on '[Alteplase](#)'.)

**ASPECTS method** — The Alberta Stroke Program Early CT Score (ASPECTS) was developed to provide a simple and reliable method of assessing and communicating the extent of early ischemic changes on noncontrast CT [34]. ASPECTS has been studied mainly in patients potentially eligible for intravenous thrombolysis or endovascular therapy. The main application of ASPECTS is identifying patients with acute ischemic stroke who have a limited extent of early infarction (eg, an ASPECTS score  $\geq$ 6) and who are therefore likely to benefit from mechanical thrombectomy. (See '[Mechanical thrombectomy for acute ischemic stroke](#)', section on '[Benefit of early \(within 6 hours\) treatment](#)'.)

- **Calculating ASPECTS** – The original ASPECTS is assessed in the middle cerebral artery (MCA) territory. ASPECTS is calculated from evaluation of two standard axial noncontrast CT images: one at the level of the thalamus and basal ganglia, and one just rostral to the basal ganglia ( [figure 1](#) and [figure 2](#)) [34,35]. The score divides the MCA vascular territory into 10 regions of interest that are evaluated on these two axial cuts:
  - Three subcortical regions from the image at the level of the basal ganglia:
    - Caudate (C)
    - Lentiform nucleus (L)
    - Internal capsule (IC)

- Four cortical regions from the image at the level of the basal ganglia:
  - Anterior MCA cortex (M1)
  - Lateral MCA cortex (M2)
  - Posterior MCA cortex (M3)
  - Insular cortex (I)
- Three cortical regions from the image just rostral to the basal ganglia:
  - Anterior MCA cortex (M4)
  - Lateral MCA cortex (M5)
  - Posterior MCA cortex (M6)

ASPECTS is scored on an ordinal scale from 0 to 10, with lower scores indicating more extensive infarction. One point is subtracted for early ischemic change, such as focal swelling or parenchymal hypoattenuation, in each of the 10 defined regions. Therefore, a normal CT has an ASPECTS value of 10 points, while diffuse ischemic change throughout the MCA territory gives a value of zero.

- **Reliability and accuracy** – The inter- and intra-observer reliability of the ASPECTS in early studies was reported as good to excellent [36]. However, in later studies, the interobserver reliability of the ASPECTS was lower, particularly for less experienced readers [37,38]. One other problem with the ASPECTS may be that parenchymal signs on noncontrast CT considered to represent early ischemic change may have different pathophysiologic mechanisms. In particular, there is evidence suggesting that hypoattenuation represents the irreversible infarction core, whereas focal swelling may represent penumbra [39,40].

Rating of the ASPECTS using automated image analysis software may address some of the issues of limited interrater reliability with "manual" ASPECTS scoring [38,41-45].

The ASPECTS is traditionally interpreted on the noncontrast CT. However, greater accuracy for detection of ischemic change and for identifying final infarct volume may be achieved when it is scored on CTA source images or on the contrast-enhanced CT images obtained as part of the CT perfusion (CTP) acquisition [46,47].

- **Predicting functional outcome** – In the initial ASPECTS study, pretreatment noncontrast CT scans from 156 patients with anterior circulation ischemia who were treated with intravenous tPA were prospectively scored [34]. The ASPECTS predicted functional outcome with good sensitivity and specificity (78 and 96 percent, respectively). The prospective Canadian [Alteplase](#) for Stroke Effectiveness Study (CASES) observational



cohort study of 1135 patients treated with intravenous tPA found that each one-point decrement in the baseline ASPECTS was associated with a lower probability of independent functional outcome (odds ratio 0.81, 95% CI 0.75-0.87) [48].

- **Predicting response to thrombolysis** – The ASPECTS of baseline noncontrast CT scans from the NINDS and European Cooperative Acute Stroke Study (ECASS-II) tPA stroke studies was not associated with a statistically significant modification of tPA treatment effect [49,50]. This finding is in agreement with a report from the NINDS cohort, which found that signs of early ischemic change on noncontrast CT were not independently associated with increased risk of adverse outcome after intravenous tPA treatment [33].
- **Selection of patients for mechanical thrombectomy** – Most earlier trials of endovascular therapy have used ASPECTS to exclude patients with extensive early infarct signs. Therefore, 2019 guidelines from the American Heart Association/American Stroke Association (AHA/ASA) recommended treatment with mechanical thrombectomy only for patients with an ASPECTS  $\geq 6$  [1]. However, subsequent trials found that mechanical thrombectomy improves outcomes for patients with acute anterior circulation ischemic stroke due to large vessel occlusion who have a large ischemic core (eg, defined by an ASPECTS 3 to 5 or by a core volume  $\geq 50$  mL). (See "[Mechanical thrombectomy for acute ischemic stroke](#)", section on 'Benefit for large core infarcts'.)
- **Calculating posterior circulation ASPECTS** – The dedicated posterior circulation ASPECTS (pc-ASPECTS), rated on CTA source images, can be used to quantify early infarct signs in patients with posterior circulation stroke. The pc-ASPECTS subtracts one point for each ischemic lesion (right or left) of the thalamus, cerebellar hemisphere, or posterior cerebral artery territory, and two points for each lesion in the mesencephalon or pons [51,52]. A normal pc-ASPECTS has a value of 10 points; lower scores indicate greater extent of infarction.

**Parenchymal changes on DWI** — MRI using diffusion-weighted imaging (DWI) is superior to noncontrast CT for the diagnosis of acute ischemic stroke in patients presenting within 12 hours of symptom onset [53]. DWI can detect abnormalities due to infarction within 3 to 30 minutes of onset [54-56], when conventional MRI and CT images would still appear normal. Studies comparing noncontrast CT, DWI, and fluid-attenuated inversion recovery (FLAIR) have shown that abnormal DWI is a sensitive and specific indicator of ischemic stroke in patients presenting within six hours of symptom onset [57-62]. However, occasional patients with acute ischemic deficits may have a normal DWI. In one retrospective report of 565 patients with acute ischemic stroke, a relevant lesion on DWI was apparent in 518 (92 percent), suggesting that DWI alone may miss an acute stroke in 8 percent of patients [62]. In these cases, follow-up MRI or CT may



confirm an infarct [63,64]. In some of these patients, the stroke was a small brainstem lacunar infarction, and in others, ischemia was seen on perfusion MRI in regions that had not yet become abnormal on DWI [63].

Even in patients with subacute ischemic stroke who delay seeking medical attention, DWI may add clinically useful information to standard MRI. In a prospective observational study of 300 patients with suspected stroke or transient ischemic attack (TIA) and a median delay of 17 days from symptom onset, DWI provided additional clinical information compared with T2-weighted imaging for 108 patients (36 percent), such as clarification of diagnosis or definition of involved vascular territory; this was considered likely to change management in 42 patients (14 percent) [65].

In acute ischemic stroke, failure of the energy-dependent Na-K-ATPase pumps leads to translocation of water from the interstitial to the intracellular space [66]. Intracellular water (cytotoxic edema) cannot diffuse as freely as extracellular water, and this diffusion restriction or reduced diffusivity is readily demonstrated on DWI. In addition to reduced diffusivity, increased T2 relaxation due to vasogenic edema can "shine through" on DWI images, making it difficult to distinguish vasogenic from cytotoxic edema. This quandary can be overcome by comparing DWI with map images of the apparent diffusion coefficient (ADC). The ADC map provides a quantitative measure of the contribution of reduced diffusivity to DWI:

- With acute ischemic stroke associated with cytotoxic edema, decreased water diffusion in infarcted tissue causes increased (hyperintense) signal on DWI and corresponding **decreased** signal intensity on the ADC map image.
- With vasogenic edema, increased DWI signal may occur due to T2 shine-through, but since water diffusion is increased, there is also corresponding **increased** signal on the ADC map image.

## Acute intravascular thrombus

**Hyperdense artery sign on CT** — Hyperdensity of an artery (hyperdense artery sign, also known as hyperdense vessel sign or bright artery sign) on noncontrast CT can indicate the presence of the thrombus inside the artery lumen. This can be visualized on noncontrast CT in 30 to 40 percent of patients with an MCA distribution stroke [29,67]. This finding is highly specific for MCA occlusion and can be observed in proximal MCA occlusions (first branch) as well as in more distal MCA branch occlusions (eg, sylvian dot sign). Similarly, thrombus in the basilar artery can appear as a hyperdensity of that artery on noncontrast CT. Hyperdensity of an artery does not reflect ischemic injury of the brain parenchyma. It is therefore neither time dependent (ie, more prevalent over time) nor directly linked to clinical outcome. This contrasts with early

infarct signs, which reflect parenchymal injury and are therefore time dependent and associated with a worse prognosis. (See ['Assessment of early infarct signs'](#) above.)

**Susceptibility artery sign on MRI** — Susceptibility-weighted MRI imaging (eg, T2\*-weighted gradient-recalled echo [GRE]) is useful for the early detection of acute thrombosis and occlusion involving the MCA or internal carotid artery (ICA) [68-74]. Acute thrombotic occlusion may appear as focal hypointense signal within the MCA or ICA, often in a focal or curvilinear shape; the diameter of the hypointense signal is larger than that of the contralateral unaffected vessel. This finding is called the "susceptibility sign," and it is analogous to the "hyperdense artery sign" described for noncontrast CT.

In a retrospective report of 42 patients with stroke in the MCA territory who underwent MRI at 95 to 360 minutes from stroke onset, a susceptibility sign was found in 30 (71 percent) and its specificity was 100 percent [69]. The overall sensitivity was 83 percent compared with MR angiography (MRA) but varied widely depending on location of the occlusion, from 38 percent for occlusions distal to the MCA bifurcation to 97 percent for occlusions proximal to the MCA trunk.

**Vessel imaging** — Neurovascular imaging with CTA or MRA can evaluate the aortic arch and the extracranial (internal carotid and vertebral) and intracranial (internal carotid, vertebral, basilar, and Circle of Willis) large vessels. It is essential for determining if there is a large vessel occlusion for patients with acute stroke who may be eligible for mechanical thrombectomy, as well as for proper evaluation of the stroke mechanism. (See ["Mechanical thrombectomy for acute ischemic stroke"](#).)

**Presence and location of thrombus** — The head and neck vessel imaging with CTA or MRA can identify thrombus and large vessel occlusion within the territory of the acute ischemic stroke and thereby determine whether the patient may benefit from reperfusion with mechanical thrombectomy. CTA and MRA are also important tools for detecting other vascular lesions that may cause acute ischemic stroke, including arterial atherosclerosis, plaque, stenosis, dissection, vasculitis, fibromuscular dysplasia, and carotid web [75].

For the detection of intracranial large vessel stenosis and occlusion, CTA had sensitivities of 92 to 100 percent and specificities of 82 to 100 percent when compared with conventional angiography [76]. CTA is more accurate for detection of occlusions in larger proximal arteries (eg, emergent large vessel occlusion) compared with smaller distal arteries. The accuracy of CTA for the diagnosis of extracranial carotid stenosis is discussed separately. (See ["Evaluation of carotid artery stenosis"](#), section on 'Computed tomography angiography'.)

For the detection of intracranial large vessel stenosis and occlusion, contrast-enhanced MRA in various studies had sensitivities of 86 to 97 percent and specificities of 62 to 91 percent when compared with digital subtraction angiography [76]. The accuracy of MRA for the diagnosis of extracranial carotid stenosis is discussed separately. (See "[Evaluation of carotid artery stenosis](#)", section on '[Magnetic resonance angiography](#)'.)

**Collateral blood flow** — Collateral blood flow is important because good collateral flow can preserve ischemic brain tissue when the direct supply of blood is blocked by thromboembolism [75]. Good collateral flow is associated with improved outcomes among patients treated with endovascular therapy [77].

- **Collaterals on multiphase CTA** – The pial artery collateral vessels of the brain can be assessed using multiphase CTA ( [image 3](#)), which acquires information about cerebral blood flow in three phases after contrast administration: The first phase consists of conventional CTA with image acquisition from the aortic arch to skull vertex during the peak arterial phase; the second and third phases consist of image acquisition from the skull base to vertex during the peak- and late-venous phases [78].

In the ESCAPE trial, the presence of moderate-to-good pial collateral circulation on multiphase CTA was one of the criteria used to select patients for mechanical thrombectomy [79]. (See "[Mechanical thrombectomy for acute ischemic stroke](#)", section on '[Patient selection](#)'.)

- **Collaterals on FLAIR** – In 85 percent of patients with acute ischemic stroke and MCA occlusion, linear or serpentine hyperintensities in M2 and M3 vessels distal to the site of the occluded vessel can be identified on FLAIR MR images. The extent of these FLAIR-hyperintense vessels correlates with the volume of hypoperfused tissue and is an independent predictor of perfusion-diffusion mismatch [80,81].
- **Collaterals on perfusion imaging** – On MR or CT perfusion-weighted imaging (PWI), collateral blood flow is implied from the size of the core-penumbra mismatch. The larger the mismatch, the better the collaterals. The Tmax hyperintensity ratio is another metric that has been proposed as a measure of collateral status on CT and MR perfusion. It reflects the proportion of tissue that is at risk of infarction, which has an extremely severe delay in contrast [82].

**Mismatch and salvageable brain tissue** — Mismatch is an imaging marker that is used to select patients for mechanical thrombectomy who have salvageable brain tissue. In acute ischemic stroke, the infarct core is considered irreversibly infarcted brain tissue. The surrounding or adjacent ischemic penumbra is brain tissue that is hypoperfused and at risk of

infarction and is therefore potentially salvageable with reperfusion treatment. A mismatch exists when the infarct core is relatively small compared with the larger ischemic penumbra. Mismatch is assessed by imaging, with several different paradigms employed.

**Perfusion-core mismatch** — Perfusion-core mismatch can be assessed with CT or MRI techniques. Both require image processing with automated software. Studies of endovascular mechanical thrombectomy in the late time window (6 to 24 hours after stroke onset) have relied on CTP or multimodal MRI with DWI and PWI to identify patients with small ischemic cores and relatively large penumbra. (See "[Mechanical thrombectomy for acute ischemic stroke](#)", section on 'Patient selection'.)

- **CT perfusion** – CTP quantifies blood flow through the brain using a series of CT scans that are obtained following the injection of an intravenous bolus of iodinated contrast. With this technique, the passage of the contrast through the brain can be evaluated [83,84]. Using perfusion analysis software, maps showing perfusion of the brain are generated. Specifically, analysis of the kinetics of a bolus of iodinated contrast passing through the brain enables estimation of cerebral blood flow, cerebral blood volume, mean transit time of contrast through brain, time to peak, and time to peak of the residue function [84,85]. These maps are useful to estimate the size of the infarction core and the ischemic penumbra. Using perfusion analysis software, it is possible to estimate the size of the infarction core and the ischemic penumbra:
  - The ischemic core is indirectly defined as the region with the most severe perfusion deficit; it is characterized by severely reduced cerebral blood flow by most perfusion software programs. The core region typically also has an elevated mean transit time and decreased cerebral blood volume [25].
  - The ischemic field, defined as the territory that has a reduction in blood flow severe enough to eventually lead to infarction if blood flow is not reversed, is characterized by a significant delay in contrast arrival by most perfusion software programs. Commonly used perfusion parameters to identify the ischemic field include prolonged Tmax, prolonged time to peak (TTP), and prolonged mean transit time (MTT).
  - The ischemic penumbra is the difference between the ischemic field and the ischemic core (ie, the part of the ischemic field that is not already infarcted).
- **PWI and DWI** – PWI can demonstrate ischemic regions of the brain while DWI reveals cytotoxic edema from infarction. The PWI-DWI mismatch refers to the presence of a relatively larger area of ischemia on PWI (ie, the penumbra or territory with critically low

perfusion) relative to a smaller area of irreversible ischemic injury (ie, the infarction core) on DWI ( [image 4](#) and [image 5](#) and [image 6](#)).

PWI quantifies the magnetic susceptibility effect from the passage of intravenously administered gadolinium-based contrast agent. Analysis of the characteristics of the contrast bolus passage through brain tissue can yield maps of the cerebral perfusion, including cerebral blood flow, cerebral blood volume, mean transit time, time-to-peak of the contrast enhancement, and time-to-peak of the residue function.

Aside from PWI, another MRI method to evaluate brain perfusion is arterial spin labeling (ASL). Instead of using an intravascular contrast agent, ASL magnetically labels the blood entering the brain. ASL imaging within 24 hours of stroke symptom onset can depict perfusion defects and mismatches of diffusion and perfusion [86]. In addition, asymmetry of perfusion on ASL appears to correlate with stroke severity and outcome.

In the DEFUSE 3 trial, which studied patients with late-window (6 to 16 hours after the time last known to be well) acute ischemic stroke, either CT or MR perfusion imaging was used to select patients for mechanical thrombectomy [87]. The study demonstrated a benefit of endovascular thrombectomy regardless of whether CT or MRI was used for patient selection, but the benefit was greater for patients selected using PWI compared with CTP. (See "[Mechanical thrombectomy for acute ischemic stroke](#)", section on 'Benefit with a clinical or tissue mismatch defined by imaging'.)

**Clinical-core mismatch** — Using MRI, a clinical-core mismatch (ie, clinical-DWI mismatch) is based on the concept that the neurologic deficits are an expression of both the core infarct and the ischemic penumbra. A mismatch is present if the severity of the neurologic deficits, as measured by the National Institutes of Health Stroke Scale (NIHSS), is greater than that expected from the core infarct, which corresponds to the DWI lesion [88]. An age-adjusted clinical-core mismatch was used to select patients for mechanical thrombectomy in the DAWN trial. (See "[Mechanical thrombectomy for acute ischemic stroke](#)", section on 'Benefit with a clinical or tissue mismatch defined by imaging'.)

**Clinical-ASPECTS mismatch** — Using CT, a clinical-ASPECTS mismatch is present if the severity of the neurologic deficits, as measured by the NIHSS, is greater than expected from the severity of the core infarct, as assessed by ASPECTS [89]. In practice, the mismatch is present for patient with an NIHSS score  $\geq 10$  and an ASPECTS  $\geq 6$ .

**DWI-FLAIR mismatch** — The DWI-FLAIR mismatch refers to evidence of a hyperintense lesion on DWI consistent with acute infarction but no corresponding signal abnormality on the FLAIR images ( [image 7](#) ) [90]. This mismatch indicates that the stroke is relatively acute (ie, within

4.5 hours), since insufficient time has passed for development of hyperintense signal on FLAIR, a sign of vasogenic edema. DWI-FLAIR mismatch has been used in clinical trials to select patients for treatment with intravenous thrombolysis when the time of stroke onset is unwitnessed or unknown [91]. (See ["Approach to reperfusion therapy for acute ischemic stroke"](#), section on 'Benefit with imaging selection of patients'.)

---

## IMAGING OF HEMORRHAGIC STROKE

Acute or subacute hemorrhage on imaging is a contraindication to reperfusion therapy (intravenous thrombolysis and mechanical thrombectomy). Evaluation, diagnosis, and management are reviewed separately according to the location of hemorrhage:

- Intracerebral (see ["Spontaneous intracerebral hemorrhage: Pathogenesis, clinical features, and diagnosis"](#) and ["Spontaneous intracerebral hemorrhage: Acute treatment and prognosis"](#))
  - Intraventricular (see ["Intraventricular hemorrhage"](#))
  - Subarachnoid (see ["Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis"](#) and ["Aneurysmal subarachnoid hemorrhage: Treatment and prognosis"](#) and ["Nonaneurysmal subarachnoid hemorrhage"](#))
  - Subdural (see ["Subdural hematoma in adults: Etiology, clinical features, and diagnosis"](#) and ["Subdural hematoma in adults: Management and prognosis"](#))
  - Epidural (see ["Intracranial epidural hematoma in adults"](#))
- 

## OTHER IMAGING MODALITIES

**Digital subtraction angiography** — Digital subtraction angiography (DSA) is a method of visualizing the cerebral vasculature using selective injections of contrast through a catheter placed in the large arteries of the neck (ie, carotid and vertebral arteries) and head (cerebral arteries).

DSA is rarely performed to triage patients in the setting of acute ischemic stroke for two main reasons. First, it is less available compared with CT angiography (CTA) and MR angiography (MRA). Second, DSA is associated with a risk of stroke, albeit low. The risk of stroke is estimated to be 0.14 to 1 percent, and the risk of transient ischemia is estimated to be 0.4 to 3 percent [92-94].

DSA is an integral part of endovascular therapy procedures (ie, mechanical thrombectomy) for patients with acute ischemic stroke secondary to an occlusion of a large cerebral artery. The increased use of DSA started in 2015 after the publication of five major trials that demonstrated benefit of endovascular therapy up to six hours after stroke onset, and it has further increased since 2018 when two landmark trials were published that demonstrated benefit of mechanical thrombectomy beyond the initial six-hour time window. (See "[Mechanical thrombectomy for acute ischemic stroke](#)".)

DSA remains the gold standard for determining the severity of arterial stenosis and the presence of vasculopathy or vascular malformations [76]. In addition, it provides information about collateral flow and perfusion.

**Ultrasound methods** — Carotid duplex ultrasound (CDUS) and transcranial Doppler (TCD) ultrasound are noninvasive methods for neurovascular evaluation of the extracranial and intracranial large vessels, respectively. Carotid and vertebral duplex and TCD have traditionally been used independently in a predominantly elective, nonacute fashion to evaluate patients with transient ischemic attack (TIA) and ischemic stroke of possible large artery origin.

- **Carotid and vertebral duplex** – Color flow guided duplex ultrasound is well established as a noninvasive examination to evaluate extracranial atherosclerotic disease. This topic is discussed separately. (See "[Evaluation of carotid artery stenosis](#)", section on 'Carotid duplex ultrasound'.)
- **Transcranial Doppler** – TCD ultrasound uses low frequency (2 MHz) pulsed sound to penetrate bone and insonate intracranial vessels of the circle of Willis. Its use has gained acceptance as a noninvasive means to assess the patency of intracranial vessels. In patients with acute stroke, TCD is able to detect intracranial stenosis, identify collateral pathways, detect emboli on a real-time basis, and monitor reperfusion after thrombolysis [95-98]. Major drawbacks include operator dependence, poor acoustic windows (ie, inability to insonate flow in 15 percent of cases), and low sensitivity to flow in the vertebrobasilar arteries.
- **Combined duplex and TCD** – The combination of urgent duplex and TCD has been described in a few small studies. As an example, a study of 150 patients found that the combination of duplex and TCD could be used effectively to detect arterial lesions amenable to interventional treatment [99]. A major limitation of this approach is that many centers are unable to perform these examinations emergently because of the lack of experienced sonographers.



---

## SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Stroke in adults"](#).)

---

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5<sup>th</sup> to 6<sup>th</sup> grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10<sup>th</sup> to 12<sup>th</sup> grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see ["Patient education: Stroke \(The Basics\)"](#))
- 

## SUMMARY AND RECOMMENDATIONS

- **Goals of imaging** – Immediate imaging of patients with suspected acute stroke is a priority because "time is brain" and because imaging provides essential information for selecting treatment. The goals of early neuroimaging are to distinguish ischemia from hemorrhage, exclude stroke mimics, detect signs of early infarction, depict the infarction core and ischemic penumbra, reveal the status of large cervical and intracranial arteries, and help determine patient eligibility for intravenous thrombolysis and mechanical thrombectomy. (See ["Goals of imaging"](#) above and ["Urgency and scope of imaging"](#) above.)
- **Comparison of CT and MRI** – Noncontrast CT of the head is the standard imaging study for early acute stroke evaluation at most centers because of widespread availability, rapid scan times, and sensitivity for intracranial hemorrhage. MRI with diffusion-weighted imaging (DWI) is superior to CT for the detection of acute ischemic stroke and the exclusion of stroke mimics. In addition, MRI reliably detects acute intracranial

hemorrhage. However, MRI is not as readily available for urgent imaging and is more limited by patient contraindications or intolerance. (See '[CT or MRI for initial imaging?](#)' above.)

- **Choosing initial imaging** – For patients who may be eligible for intravenous thrombolysis or mechanical thrombectomy ( [algorithm 1](#)), multimodal CT or MRI can provide crucial information to guide treatment decisions, including:

- Whether there is cervical or intracranial large artery stenosis and occlusion
- The volume of the infarct core that is irreversibly damaged
- The volume of the ischemic penumbra that is salvageable with reperfusion

The selection of initial neuroimaging studies can be guided by the time the patient was last known to be well and by local availability of advanced imaging with multimodal CT and MRI. Specifics are detailed above. (See '[Time-based selection of imaging](#)' above.)

- **Vessel imaging** – Neurovascular imaging with CT angiography (CTA) or MR angiography (MRA) is essential for confirming the presence of a large artery occlusion in patients who are candidates for mechanical thrombectomy. It is also important for assessing the potential sources of embolism and low flow in ischemic stroke. (See '[Vessel imaging](#)' above.)
- **Imaging the infarct core and ischemic penumbra** – Evaluation of the infarct core and ischemic penumbra with either DWI and perfusion-weighted imaging (PWI) or CT perfusion (CTP) imaging should be performed if the findings are likely to influence treatment decisions, such as mechanical thrombectomy in the late window (ie, >6 hours from the time last known to be well). (See '[Mismatch and salvageable brain tissue](#)' above.)

---

## ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Walter Koroshetz, MD, who contributed to an earlier version of this topic review.

Use of UpToDate is subject to the [Terms of Use](#).

## REFERENCES

1. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early

- Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke* 2019; 50:e344.
2. Molina CA, Saver JL. Extending reperfusion therapy for acute ischemic stroke: emerging pharmacological, mechanical, and imaging strategies. *Stroke* 2005; 36:2311.
  3. Kamalian S, Lev MH. Stroke Imaging. *Radiol Clin North Am* 2019; 57:717.
  4. Puig J, Shankar J, Liebeskind D, et al. From "Time is Brain" to "Imaging is Brain": A Paradigm Shift in the Management of Acute Ischemic Stroke. *J Neuroimaging* 2020; 30:562.
  5. Zerna C, Thomalla G, Campbell BCV, et al. Current practice and future directions in the diagnosis and acute treatment of ischaemic stroke. *Lancet* 2018; 392:1247.
  6. Czap AL, Sheth SA. Overview of Imaging Modalities in Stroke. *Neurology* 2021; 97:S42.
  7. von Kummer R, Bourquain H, Bastianello S, et al. Early prediction of irreversible brain damage after ischemic stroke at CT. *Radiology* 2001; 219:95.
  8. Patel MR, Edelman RR, Warach S. Detection of hyperacute primary intraparenchymal hemorrhage by magnetic resonance imaging. *Stroke* 1996; 27:2321.
  9. Fiebach JB, Schellinger PD, Gass A, et al. Stroke magnetic resonance imaging is accurate in hyperacute intracerebral hemorrhage: a multicenter study on the validity of stroke imaging. *Stroke* 2004; 35:502.
  10. Kidwell CS, Chalela JA, Saver JL, et al. Comparison of MRI and CT for detection of acute intracerebral hemorrhage. *JAMA* 2004; 292:1823.
  11. Simonsen CZ, Yoo AJ, Rasmussen M, et al. Magnetic Resonance Imaging Selection for Endovascular Stroke Therapy: Workflow in the GOLIATH Trial. *Stroke* 2018; 49:1402.
  12. Shah S, Luby M, Poole K, et al. Screening with MRI for Accurate and Rapid Stroke Treatment: SMART. *Neurology* 2015; 84:2438.
  13. Sjølling C, Ashkanian M, Hjort N, et al. Feasibility and logistics of MRI before thrombolytic treatment. *Acta Neurol Scand* 2009; 120:143.
  14. Simonsen CZ, Sørensen LH, Karabegovic S, et al. MRI before intraarterial therapy in ischemic stroke: feasibility, impact, and safety. *J Cereb Blood Flow Metab* 2014; 34:1076.
  15. Ezzeddine MA, Lev MH, McDonald CT, et al. CT angiography with whole brain perfused blood volume imaging: added clinical value in the assessment of acute stroke. *Stroke* 2002; 33:959.
  16. Kloska SP, Nabavi DG, Gaus C, et al. Acute stroke assessment with CT: do we need multimodal evaluation? *Radiology* 2004; 233:79.

17. Hopyan J, Ciarallo A, Dowlatshahi D, et al. Certainty of stroke diagnosis: incremental benefit with CT perfusion over noncontrast CT and CT angiography. *Radiology* 2010; 255:142.
18. Campbell BC, Weir L, Desmond PM, et al. CT perfusion improves diagnostic accuracy and confidence in acute ischaemic stroke. *J Neurol Neurosurg Psychiatry* 2013; 84:613.
19. Tan JC, Dillon WP, Liu S, et al. Systematic comparison of perfusion-CT and CT-angiography in acute stroke patients. *Ann Neurol* 2007; 61:533.
20. Wintermark M, Rowley HA, Lev MH. Acute stroke triage to intravenous thrombolysis and other therapies with advanced CT or MR imaging: pro CT. *Radiology* 2009; 251:619.
21. Althaus K, Dreyhaupt J, Hyrenbach S, et al. MRI as a first-line imaging modality in acute ischemic stroke: a sustainable concept. *Ther Adv Neurol Disord* 2021; 14:17562864211030363.
22. Vilela P, Rowley HA. Brain ischemia: CT and MRI techniques in acute ischemic stroke. *Eur J Radiol* 2017; 96:162.
23. Nael K, Yoo B, Salamon N, Liebeskind DS. Acute Ischemic Stroke: MR Imaging-Based Paradigms. *Neuroimaging Clin N Am* 2021; 31:177.
24. Thomalla G, Gerloff C. Acute imaging for evidence-based treatment of ischemic stroke. *Curr Opin Neurol* 2019; 32:521.
25. Simonsen CZ, Leslie-Mazwi TM, Thomalla G. Which Imaging Approach Should Be Used for Stroke of Unknown Time of Onset? *Stroke* 2021; 52:373.
26. Wardlaw JM, Mielke O. Early signs of brain infarction at CT: observer reliability and outcome after thrombolytic treatment--systematic review. *Radiology* 2005; 235:444.
27. Truwit CL, Barkovich AJ, Gean-Marton A, et al. Loss of the insular ribbon: another early CT sign of acute middle cerebral artery infarction. *Radiology* 1990; 176:801.
28. von Kummer R, Meyding-Lamadé U, Forsting M, et al. Sensitivity and prognostic value of early CT in occlusion of the middle cerebral artery trunk. *AJNR Am J Neuroradiol* 1994; 15:9.
29. Marks MP, Holmgren EB, Fox AJ, et al. Evaluation of early computed tomographic findings in acute ischemic stroke. *Stroke* 1999; 30:389.
30. Wijdicks EF, Diringer MN. Middle cerebral artery territory infarction and early brain swelling: progression and effect of age on outcome. *Mayo Clin Proc* 1998; 73:829.
31. Hacke W, Kaste M, Fieschi C, et al. Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke. The European Cooperative Acute Stroke Study (ECASS). *JAMA* 1995; 274:1017.
32. Wardlaw JM, Dorman PJ, Lewis SC, Sandercock PA. Can stroke physicians and neuroradiologists identify signs of early cerebral infarction on CT? *J Neurol Neurosurg*

Psychiatry 1999; 67:651.

33. Patel SC, Levine SR, Tilley BC, et al. Lack of clinical significance of early ischemic changes on computed tomography in acute stroke. JAMA 2001; 286:2830.
34. Barber PA, Demchuk AM, Zhang J, Buchan AM. Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before thrombolytic therapy. ASPECTS Study Group. Alberta Stroke Programme Early CT Score. Lancet 2000; 355:1670.
35. Pexman JH, Barber PA, Hill MD, et al. Use of the Alberta Stroke Program Early CT Score (ASPECTS) for assessing CT scans in patients with acute stroke. AJNR Am J Neuroradiol 2001; 22:1534.
36. Coutts SB, Demchuk AM, Barber PA, et al. Interobserver variation of ASPECTS in real time. Stroke 2004; 35:e103.
37. Naylor J, Churilov L, Rane N, et al. Reliability and Utility of the Alberta Stroke Program Early Computed Tomography Score in Hyperacute Stroke. J Stroke Cerebrovasc Dis 2017; 26:2547.
38. Prakkamakul S, Yoo AJ. ASPECTS CT in Acute Ischemia: Review of Current Data. Top Magn Reson Imaging 2017; 26:103.
39. Na DG, Kim EY, Ryoo JW, et al. CT sign of brain swelling without concomitant parenchymal hypoattenuation: comparison with diffusion- and perfusion-weighted MR imaging. Radiology 2005; 235:992.
40. Butcher KS, Lee SB, Parsons MW, et al. Differential prognosis of isolated cortical swelling and hypoattenuation on CT in acute stroke. Stroke 2007; 38:941.
41. Herweh C, Ringleb PA, Rauch G, et al. Performance of e-ASPECTS software in comparison to that of stroke physicians on assessing CT scans of acute ischemic stroke patients. Int J Stroke 2016; 11:438.
42. Lee EJ, Kim YH, Kim N, Kang DW. Deep into the Brain: Artificial Intelligence in Stroke Imaging. J Stroke 2017; 19:277.
43. Qiu W, Kuang H, Teleg E, et al. Machine Learning for Detecting Early Infarction in Acute Stroke with Non-Contrast-enhanced CT. Radiology 2020; 294:638.
44. Wolff L, Berkhemer OA, van Es ACGM, et al. Validation of automated Alberta Stroke Program Early CT Score (ASPECTS) software for detection of early ischemic changes on non-contrast brain CT scans. Neuroradiology 2021; 63:491.
45. Mair G, White P, Bath PM, et al. External Validation of e-ASPECTS Software for Interpreting Brain CT in Stroke. Ann Neurol 2022; 92:943.

46. Coutts SB, Lev MH, Eliasziw M, et al. ASPECTS on CTA source images versus unenhanced CT: added value in predicting final infarct extent and clinical outcome. *Stroke* 2004; 35:2472.
47. Parsons MW, Pepper EM, Chan V, et al. Perfusion computed tomography: prediction of final infarct extent and stroke outcome. *Ann Neurol* 2005; 58:672.
48. Hill MD, Buchan AM, Canadian Alteplase for Stroke Effectiveness Study (CASES) Investigators. Thrombolysis for acute ischemic stroke: results of the Canadian Alteplase for Stroke Effectiveness Study. *CMAJ* 2005; 172:1307.
49. Demchuk AM, Hill MD, Barber PA, et al. Importance of early ischemic computed tomography changes using ASPECTS in NINDS rtPA Stroke Study. *Stroke* 2005; 36:2110.
50. Dzialowski I, Hill MD, Coutts SB, et al. Extent of early ischemic changes on computed tomography (CT) before thrombolysis: prognostic value of the Alberta Stroke Program Early CT Score in ECASS II. *Stroke* 2006; 37:973.
51. Puetz V, Sylaja PN, Coutts SB, et al. Extent of hypoattenuation on CT angiography source images predicts functional outcome in patients with basilar artery occlusion. *Stroke* 2008; 39:2485.
52. Tei H, Uchiyama S, Usui T, Ohara K. Posterior circulation ASPECTS on diffusion-weighted MRI can be a powerful marker for predicting functional outcome. *J Neurol* 2010; 257:767.
53. Schellinger PD, Bryan RN, Caplan LR, et al. Evidence-based guideline: The role of diffusion and perfusion MRI for the diagnosis of acute ischemic stroke: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology* 2010; 75:177.
54. Warach S, Gaa J, Siewert B, et al. Acute human stroke studied by whole brain echo planar diffusion-weighted magnetic resonance imaging. *Ann Neurol* 1995; 37:231.
55. Sorensen AG, Buonanno FS, Gonzalez RG, et al. Hyperacute stroke: evaluation with combined multisection diffusion-weighted and hemodynamically weighted echo-planar MR imaging. *Radiology* 1996; 199:391.
56. Li F, Han S, Tatlisumak T, et al. A new method to improve in-bore middle cerebral artery occlusion in rats: demonstration with diffusion- and perfusion-weighted imaging. *Stroke* 1998; 29:1715.
57. González RG, Schaefer PW, Buonanno FS, et al. Diffusion-weighted MR imaging: diagnostic accuracy in patients imaged within 6 hours of stroke symptom onset. *Radiology* 1999; 210:155.
58. Fiebach JB, Schellinger PD, Jansen O, et al. CT and diffusion-weighted MR imaging in randomized order: diffusion-weighted imaging results in higher accuracy and lower



- interrater variability in the diagnosis of hyperacute ischemic stroke. *Stroke* 2002; 33:2206.
59. Chalela JA, Kidwell CS, Nentwich LM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. *Lancet* 2007; 369:293.
  60. Brazzelli M, Sandercock PA, Chappell FM, et al. Magnetic resonance imaging versus computed tomography for detection of acute vascular lesions in patients presenting with stroke symptoms. *Cochrane Database Syst Rev* 2009; :CD007424.
  61. Brunser AM, Hoppe A, Illanes S, et al. Accuracy of diffusion-weighted imaging in the diagnosis of stroke in patients with suspected cerebral infarct. *Stroke* 2013; 44:1169.
  62. Simonsen CZ, Madsen MH, Schmitz ML, et al. Sensitivity of diffusion- and perfusion-weighted imaging for diagnosing acute ischemic stroke is 97.5%. *Stroke* 2015; 46:98.
  63. Ay H, Buonanno FS, Rordorf G, et al. Normal diffusion-weighted MRI during stroke-like deficits. *Neurology* 1999; 52:1784.
  64. Sylaja PN, Coutts SB, Krol A, et al. When to expect negative diffusion-weighted images in stroke and transient ischemic attack. *Stroke* 2008; 39:1898.
  65. Schulz UG, Briley D, Meagher T, et al. Diffusion-weighted MRI in 300 patients presenting late with subacute transient ischemic attack or minor stroke. *Stroke* 2004; 35:2459.
  66. Beauchamp NJ Jr, Barker PB, Wang PY, vanZijl PC. Imaging of acute cerebral ischemia. *Radiology* 1999; 212:307.
  67. Leys D, Pruvo JP, Godefroy O, et al. Prevalence and significance of hyperdense middle cerebral artery in acute stroke. *Stroke* 1992; 23:317.
  68. Flacke S, Urbach H, Keller E, et al. Middle cerebral artery (MCA) susceptibility sign at susceptibility-based perfusion MR imaging: clinical importance and comparison with hyperdense MCA sign at CT. *Radiology* 2000; 215:476.
  69. Rovira A, Orellana P, Alvarez-Sabín J, et al. Hyperacute ischemic stroke: middle cerebral artery susceptibility sign at echo-planar gradient-echo MR imaging. *Radiology* 2004; 232:466.
  70. Huang P, Chen CH, Lin WC, et al. Clinical applications of susceptibility weighted imaging in patients with major stroke. *J Neurol* 2012; 259:1426.
  71. Halefoglu AM, Yousem DM. Susceptibility weighted imaging: Clinical applications and future directions. *World J Radiol* 2018; 10:30.
  72. Hsu CC, Kwan GNC, Hapugoda S, et al. Susceptibility weighted imaging in acute cerebral ischemia: review of emerging technical concepts and clinical applications. *Neuroradiol J* 2017; 30:109.

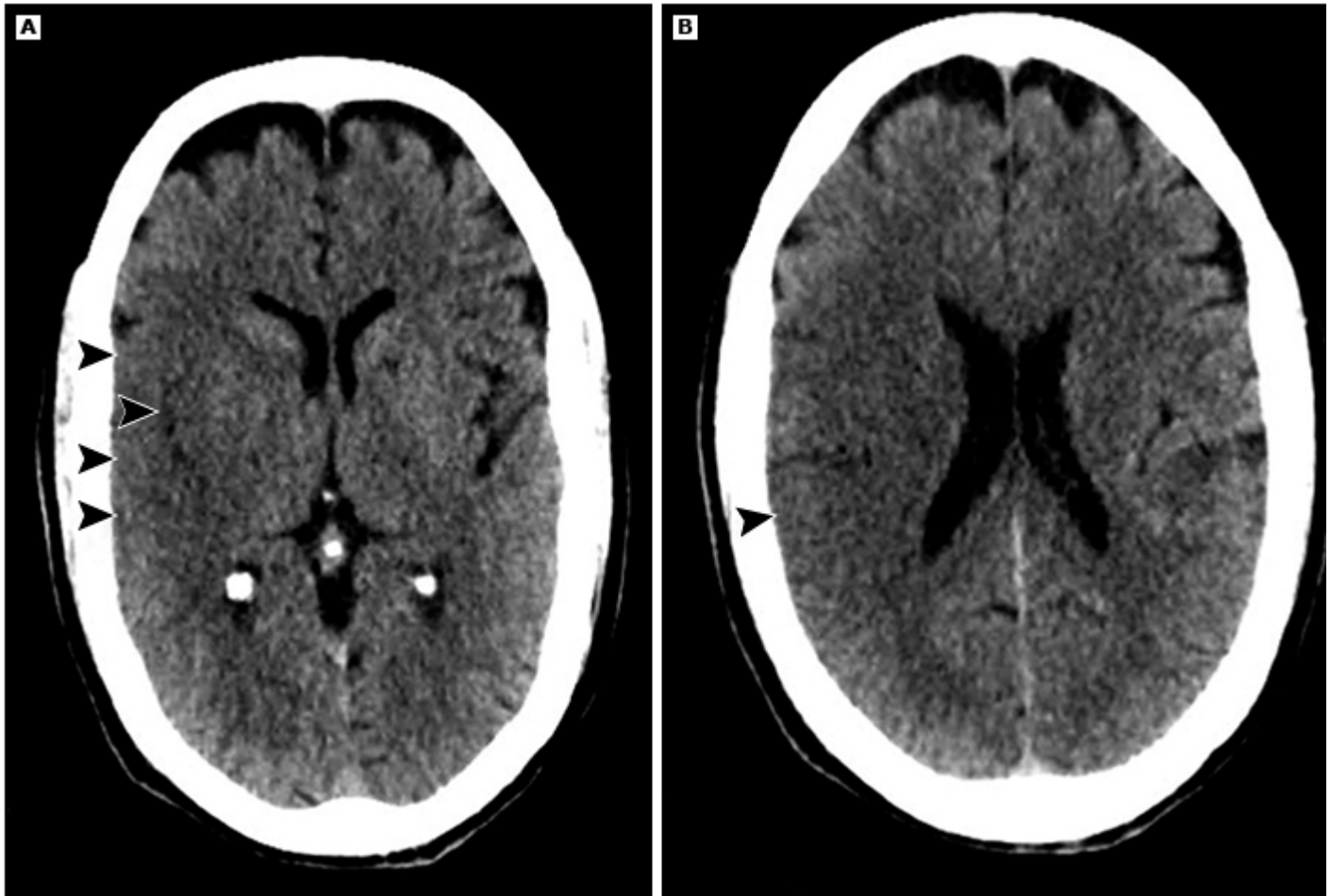


73. Khaladkar SM, Chanabasanavar V, Dhirawani S, et al. Susceptibility Weighted Imaging: An Effective Auxiliary Sequence That Enhances Insight Into the Imaging of Stroke. *Cureus* 2022; 14:e24918.
74. Belachew NF, Dobrocky T, Aleman EB, et al. SWI Susceptibility Vessel Sign in Patients Undergoing Mechanical Thrombectomy for Acute Ischemic Stroke. *AJNR Am J Neuroradiol* 2021; 42:1949.
75. Menon BK. Neuroimaging in Acute Stroke. *Continuum (Minneap Minn)* 2020; 26:287.
76. Latchaw RE, Alberts MJ, Lev MH, et al. Recommendations for imaging of acute ischemic stroke: a scientific statement from the American Heart Association. *Stroke* 2009; 40:3646.
77. Leng X, Fang H, Leung TW, et al. Impact of Collateral Status on Successful Revascularization in Endovascular Treatment: A Systematic Review and Meta-Analysis. *Cerebrovasc Dis* 2016; 41:27.
78. Menon BK, d'Esterre CD, Qazi EM, et al. Multiphase CT Angiography: A New Tool for the Imaging Triage of Patients with Acute Ischemic Stroke. *Radiology* 2015; 275:510.
79. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015; 372:1019.
80. Lee KY, Latour LL, Luby M, et al. Distal hyperintense vessels on FLAIR: an MRI marker for collateral circulation in acute stroke? *Neurology* 2009; 72:1134.
81. Grosch AS, Kufner A, Boutitie F, et al. Extent of FLAIR Hyperintense Vessels May Modify Treatment Effect of Thrombolysis: A Post hoc Analysis of the WAKE-UP Trial. *Front Neurol* 2020; 11:623881.
82. MacLellan A, Mlynash M, Kemp S, et al. Perfusion Imaging Collateral Scores Predict Infarct Growth in Non-Reperfused DEFUSE 3 Patients. *J Stroke Cerebrovasc Dis* 2022; 31:106208.
83. Allmendinger AM, Tang ER, Lui YW, Spektor V. Imaging of stroke: Part 1, Perfusion CT--overview of imaging technique, interpretation pearls, and common pitfalls. *AJR Am J Roentgenol* 2012; 198:52.
84. Vagal A, Wintermark M, Nael K, et al. Automated CT perfusion imaging for acute ischemic stroke: Pearls and pitfalls for real-world use. *Neurology* 2019; 93:888.
85. Heit JJ, Wintermark M. Perfusion Computed Tomography for the Evaluation of Acute Ischemic Stroke: Strengths and Pitfalls. *Stroke* 2016; 47:1153.
86. Chalela JA, Alsop DC, Gonzalez-Atavales JB, et al. Magnetic resonance perfusion imaging in acute ischemic stroke using continuous arterial spin labeling. *Stroke* 2000; 31:680.
87. Albers GW, Marks MP, Kemp S, et al. Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging. *N Engl J Med* 2018; 378:708.

88. Dávalos A, Blanco M, Pedraza S, et al. The clinical-DWI mismatch: a new diagnostic approach to the brain tissue at risk of infarction. *Neurology* 2004; 62:2187.
89. Bousslama M, Bowen MT, Haussen DC, et al. Selection Paradigms for Large Vessel Occlusion Acute Ischemic Stroke Endovascular Therapy. *Cerebrovasc Dis* 2017; 44:277.
90. Thomalla G, Fiebach JB, Østergaard L, et al. A multicenter, randomized, double-blind, placebo-controlled trial to test efficacy and safety of magnetic resonance imaging-based thrombolysis in wake-up stroke (WAKE-UP). *Int J Stroke* 2014; 9:829.
91. Thomalla G, Simonsen CZ, Boutitie F, et al. MRI-Guided Thrombolysis for Stroke with Unknown Time of Onset. *N Engl J Med* 2018; 379:611.
92. Johnston DC, Chapman KM, Goldstein LB. Low rate of complications of cerebral angiography in routine clinical practice. *Neurology* 2001; 57:2012.
93. Willinsky RA, Taylor SM, TerBrugge K, et al. Neurologic complications of cerebral angiography: prospective analysis of 2,899 procedures and review of the literature. *Radiology* 2003; 227:522.
94. Kaufmann TJ, Huston J 3rd, Mandrekar JN, et al. Complications of diagnostic cerebral angiography: evaluation of 19,826 consecutive patients. *Radiology* 2007; 243:812.
95. Alexandrov AV, Demchuk AM, Wein TH, Grotta JC. Yield of transcranial Doppler in acute cerebral ischemia. *Stroke* 1999; 30:1604.
96. Gao S, Wong KS, Hansberg T, et al. Microembolic signal predicts recurrent cerebral ischemic events in acute stroke patients with middle cerebral artery stenosis. *Stroke* 2004; 35:2832.
97. Saqqur M, Shuaib A, Alexandrov AV, et al. Derivation of transcranial Doppler criteria for rescue intra-arterial thrombolysis: multicenter experience from the Interventional Management of Stroke study. *Stroke* 2005; 36:865.
98. Sharma VK, Wong KS, Alexandrov AV. Transcranial Doppler. *Front Neurol Neurosci* 2016; 40:124.
99. Chernyshev OY, Garami Z, Calleja S, et al. Yield and accuracy of urgent combined carotid/transcranial ultrasound testing in acute cerebral ischemia. *Stroke* 2005; 36:32.

## GRAPHICS

### Early ischemic changes on noncontrast head CT scan

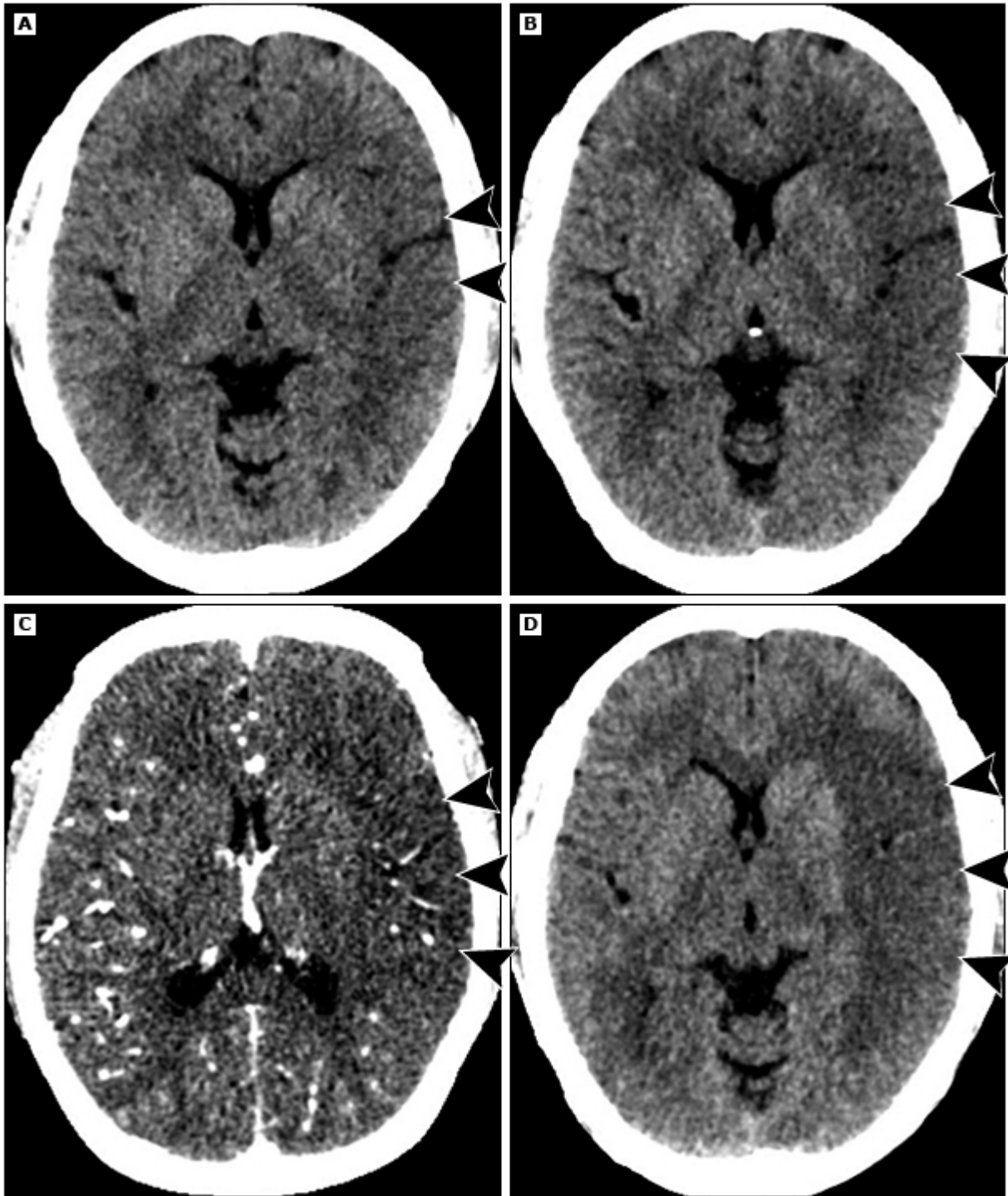


Findings of EIC in a 59-year-old man who presented with acute left hemiparesis. (A and B) NCCT 3.5 hours after symptom onset shows hypodensity and cortical swelling with sulcal effacement. There is loss of gray-white matter differentiation in the right frontal operculum, right temporal operculum, right insular cortex, and right frontoparietal lobes (arrowheads).

CT: computed tomography; EIC: early ischemic changes; NCCT: noncontrast-enhanced computed tomography.

*From: Prakkamakul S, Yoo AJ. ASPECTS CT in Acute Ischemia: Review of Current Data. Top Magn Reson Imaging 2017; 26:103. DOI: 10.1097/RMR.000000000000122. Copyright © 2017. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.*

## Evolution of early ischemic changes on head CT scan



Evolution of early ischemic changes over time in a 35-year-old woman who presented with acute aphasia.

(A) NCCT at one hour after symptom onset shows loss of gray white matter differentiation in the left frontal and temporal operculum and insular cortex and hypodensity in the lentiform nucleus (ASPECTS was 4 points for involvement of insula, lentiform, M1-3, M5).

(B and C) NCCT and CTA-SI at two hours after symptoms onset and IV rtPA administration show more conspicuous hypodensity and mild effacement of cortical sulci compared with NCCT at one hour after symptoms onset.

(D) NCCT at eight hours after symptoms onset shows frank hypodensity in the infarcted areas. The extent of the hypodense areas remains the same but is more conspicuous than prior NCCT studies.

---

CT: computed tomography; NCCT: noncontrast-enhanced computed tomography; ASPECTS: The Alberta Stroke Program Early CT score; CTA-SI: computed tomography angiography source image; IV: intravenous; rtPA: recombinant tissue plasminogen activator.

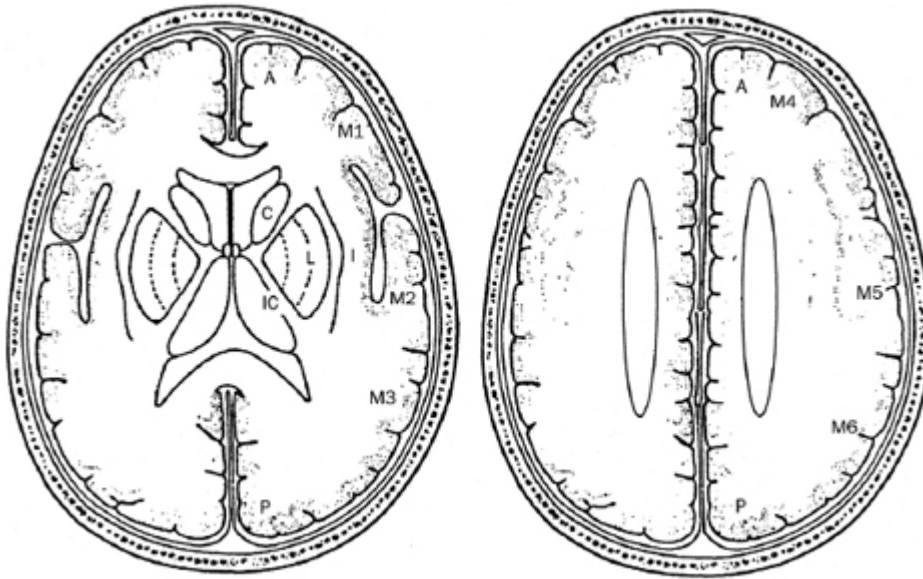
---

*From: Prakkamakul S, Yoo AJ. ASPECTS CT in Acute Ischemia: Review of Current Data. Top Magn Reson Imaging 2017; 26:103. DOI: [10.1097/RMR.000000000000122](https://doi.org/10.1097/RMR.000000000000122). Copyright © 2017. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.*

---

Graphic 121624 Version 2.0

## ASPECTS study form



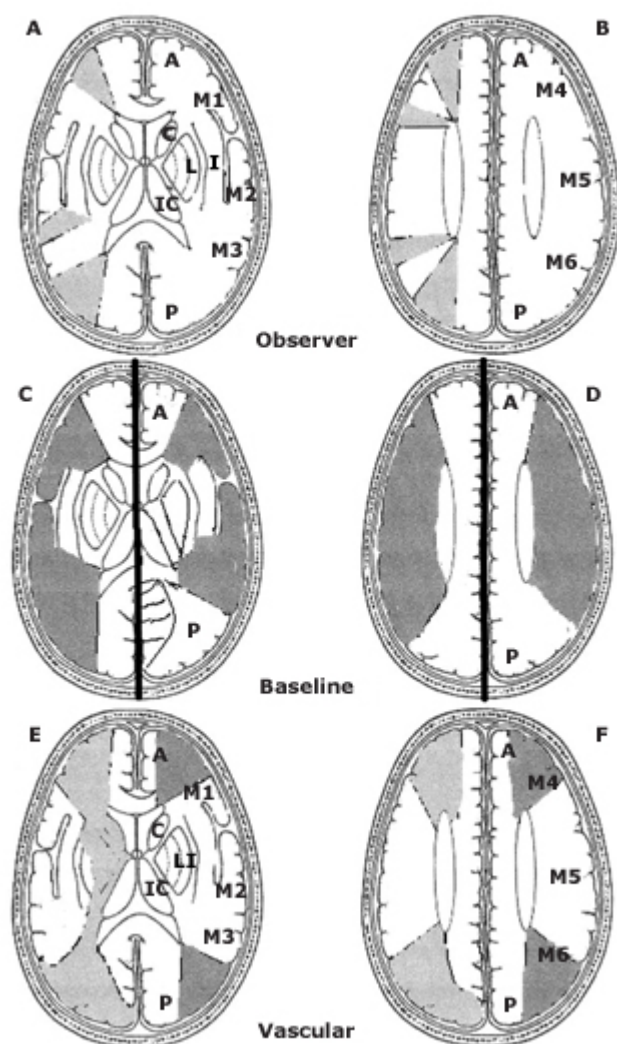
The ASPECTS value is calculated from two standard axial CT cuts: one at the level of the thalamus and basal ganglia (left), and one just rostral to the basal ganglia (right). A: anterior circulation; P: posterior circulation; C: caudate; L: lentiform; IC: internal capsule; I: insular ribbon; MCA: middle cerebral artery; M1: anterior MCA cortex; M2: MCA cortex lateral to insular ribbon; M3: posterior MCA cortex; M4, M5, and M6 are anterior, lateral, and posterior MCA territories immediately superior to M1, M2, and M3, rostral to basal ganglia

---

*Reproduced with permission from: Barber, PA, Demchuk, AM, Zhang, J, Buchan, AM. Validity and reliability of a quantitative computed tomography score in predicting outcome of hyperacute stroke before thrombolytic therapy. ASPECTS Study Group. Alberta Stroke Programme Early CT Score. Lancet 2000; 355:1670. Copyright © 2000 The Lancet.*

---

## ASPECTS study form and MCA variants



**(A and B)** Right hemisphere, observer variations: lower and upper ASPECTS slices show as shaded areas the minimal and maximal variations in size of the cortical areas of the MCA (M1-M6) chosen by six expert observers. Left hemisphere, ASPECTS study form: A = anterior circulation; P = posterior circulation; C = caudate head; L = lentiform nucleus; IC = internal capsule; I = insular ribbon; MCA = middle cerebral artery; M1 = anterior MCA cortex; M2 = MCA cortex lateral to insular ribbon; M3 = posterior MCA cortex; M4, M5, and M6 are anterior, lateral, and posterior MCA territories, respectively, approximately 2 cm superior to M1, M2, and M3, respectively, rostral to basal ganglia. **(C and D)** Cortical MCA area variations with change of baseline. In the right hemisphere, the baseline is parallel to the inferior OML; in the left hemisphere, the baseline is the superior OML. OML=orbitomeatal line. **(E and F)** Normal vascular variations in MCA size on the two ASPECTS slices. The right hemisphere shows the larger normal variations described by van der Zwan\* (light shading). The left hemisphere of each shows the smaller, textbook, variations (dark shading).

### References

\* van der Zwan, A, Hillen, B, Tulleken, AF, et al. Variability of the territories of the major cerebral arteries. *J Neurosurg* 1992; 77:927.



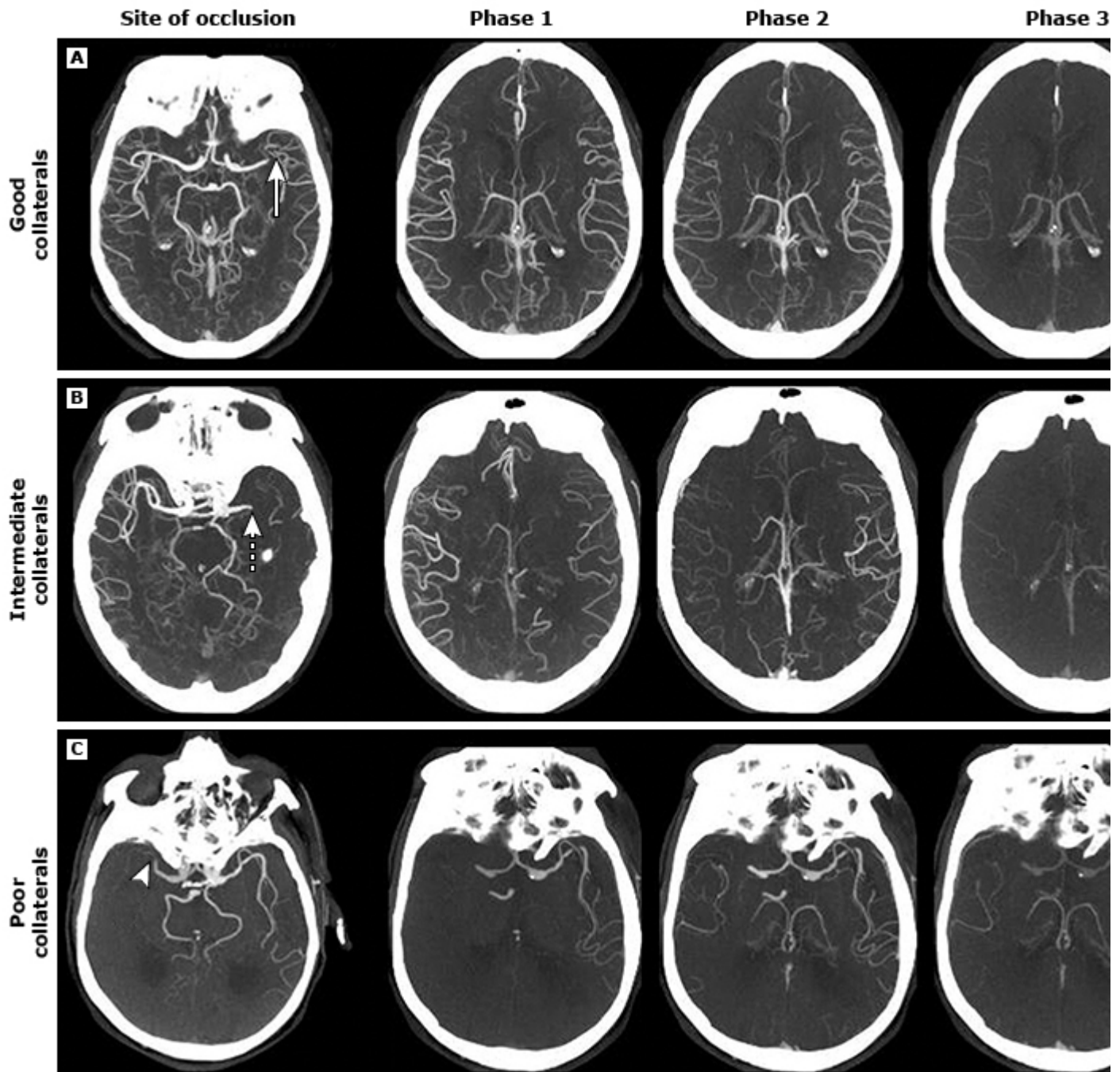
• Osborn, AG. *Neuroradiology*, Mosby, St. Louis 1995.

*Reproduced with permission from: Pexman, JH, Barber, PA, Hill, MD, et al. Use of the Alberta Stroke Program Early CT Score (ASPECTS) for assessing CT scans in patients with acute stroke. AJNR Am J Neuroradiol 2001; 22:1534. Copyright © 2001 American Society of Neuroradiology.*

---

Graphic 63480 Version 1.0

## Multiphase CTA images



(A) Images in a patient with a left M1 MCA occlusion (arrow) and good collaterals (backfilling arteries).

(B) Images in a patient with a left M1 MCA occlusion (dashed arrow) and intermediate collaterals.

(C) Images in a patient with a right M1 MCA occlusion (arrowhead) and poor collaterals (minimal backfilling arteries).

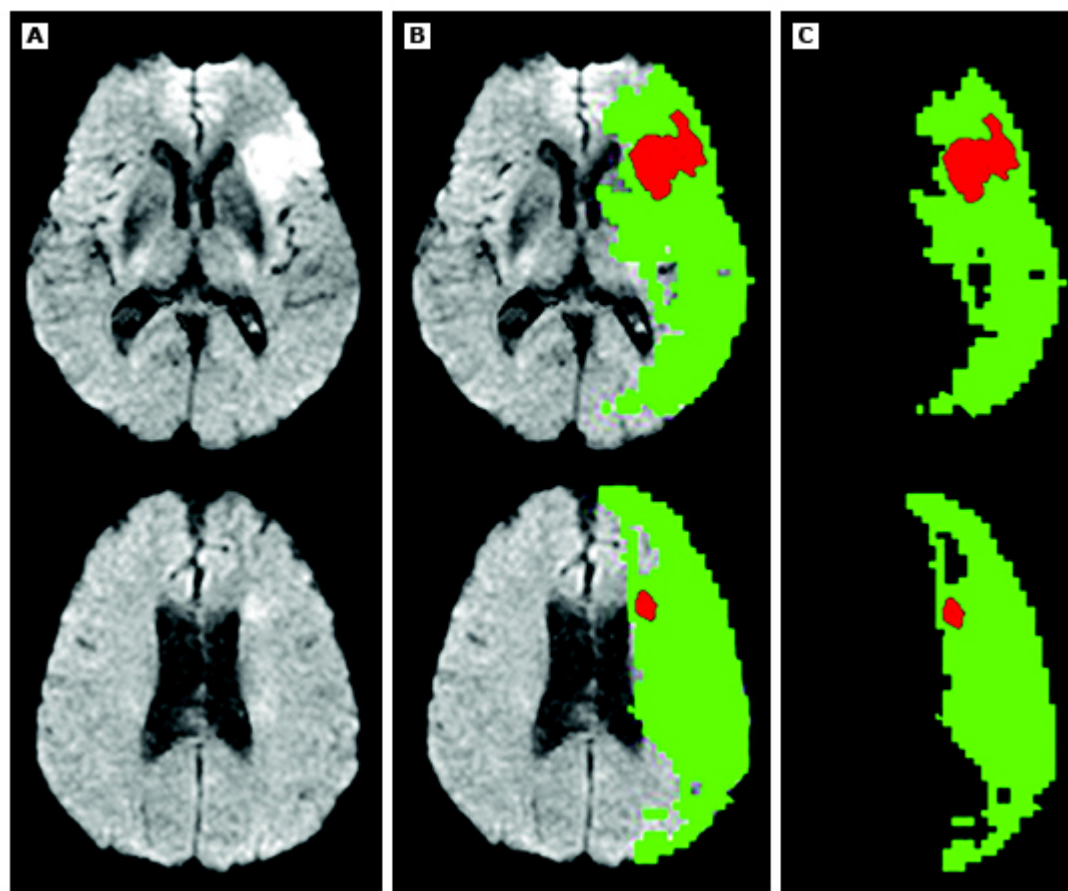
---

CTA: computed tomography angiography.

---



## Classic pattern of DWI-PWI mismatch



Representative case with classic pattern of mismatch.

(A) Diffusion weighted-image (DWI).

(B) DWI abnormal lesion (shown in red) and hypoperfusion lesion (shown in green) superimposed on DWI.

(C) DWI abnormal lesion and hypoperfusion lesion with brain image (DWI) removed.

---

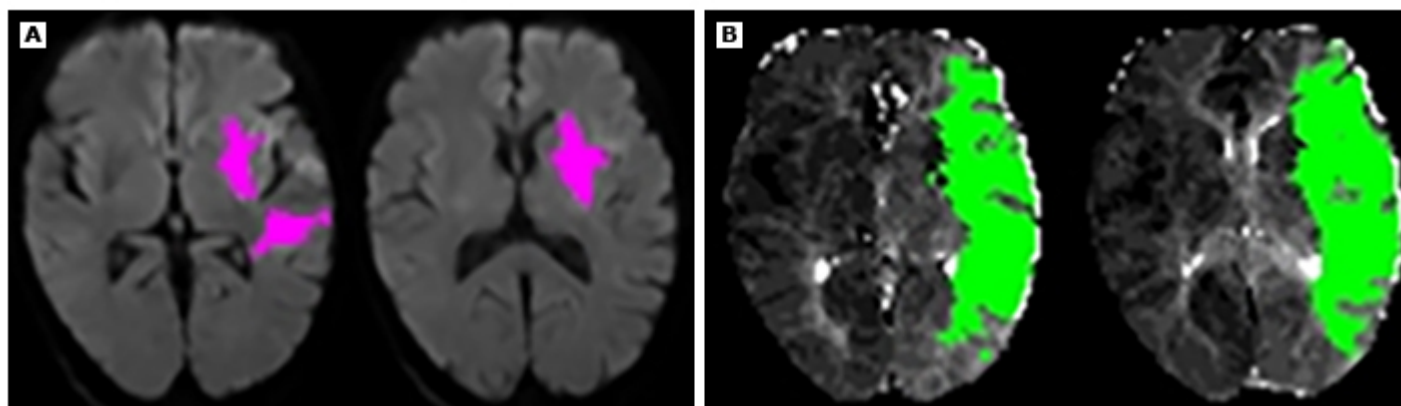
PWI: perfusion-weighted image.

---

*Reproduced with permission from: Ogata T, Nagakane Y, Christensen S, et al. A topographic study of the evolution of the MR DWI/PWI mismatch pattern and its clinical impact: a study by the EPITHET and DEFUSE Investigators. Stroke 2011; 42:1596. Copyright © 2011 Lippincott Williams & Wilkins.*

---

## Brain MRI showing large core-penumbra mismatch on diffusion- and perfusion weighted sequences in a patient with acute ischemic stroke



Diffusion- and perfusion-weighted brain MRI showing a large core-penumbra mismatch in a patient with an acute ischemic stroke. Diffusion-weighted MRI (panel A) indicates the infarction core in red as tissue with apparent diffusion coefficient  $<620$  that measures 17 mL in volume. Perfusion-weighted MRI (panel B) indicates the ischemic penumbra in green as tissue with  $T_{\max} > 6.0$  seconds that measures 139 mL in volume. The mismatch volume is 122 mL and the mismatch ratio is 8.2.

---

MRI: magnetic resonance imaging.

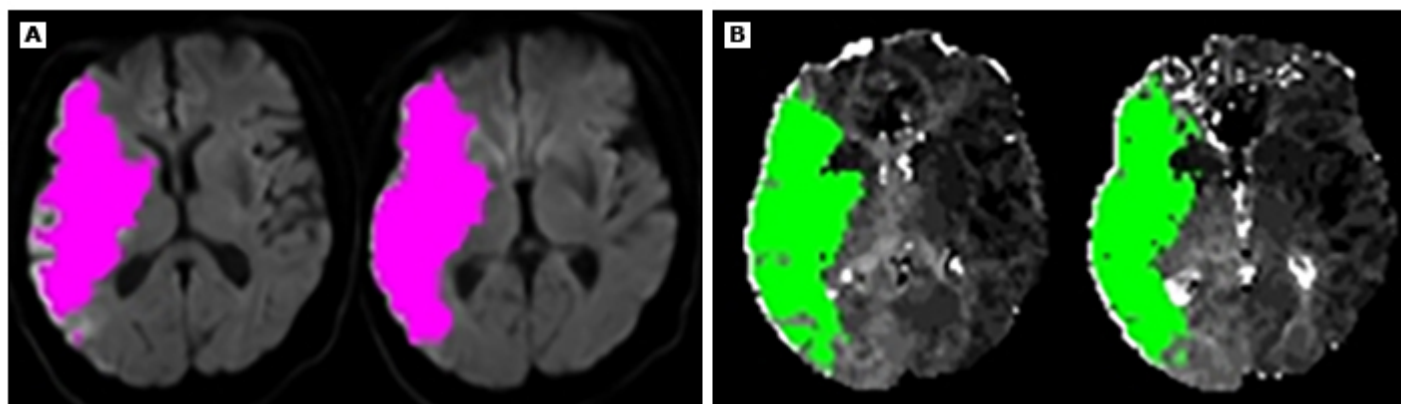
---

*Glenn A Tung, MD, FACP.*

---

Graphic 129431 Version 2.0

## Brain MRI showing a small core-penumbra mismatch on diffusion- and perfusion-weighted sequences in a patient with acute ischemic stroke



Diffusion- and perfusion-weighted brain MRI showing a small core-penumbra mismatch in a patient with an acute ischemic stroke. Diffusion-weighted MRI (panel A) indicates the infarction core in red as tissue with apparent diffusion coefficient  $<620$  that measures 151 mL in volume. Perfusion-weighted MRI (panel B) indicates the ischemic penumbra in green as tissue with  $T_{max} >6.0$  seconds that measures 177 mL in volume. The mismatch volume is 26 mL and the mismatch ratio is 1.2.

---

MRI: magnetic resonance imaging.

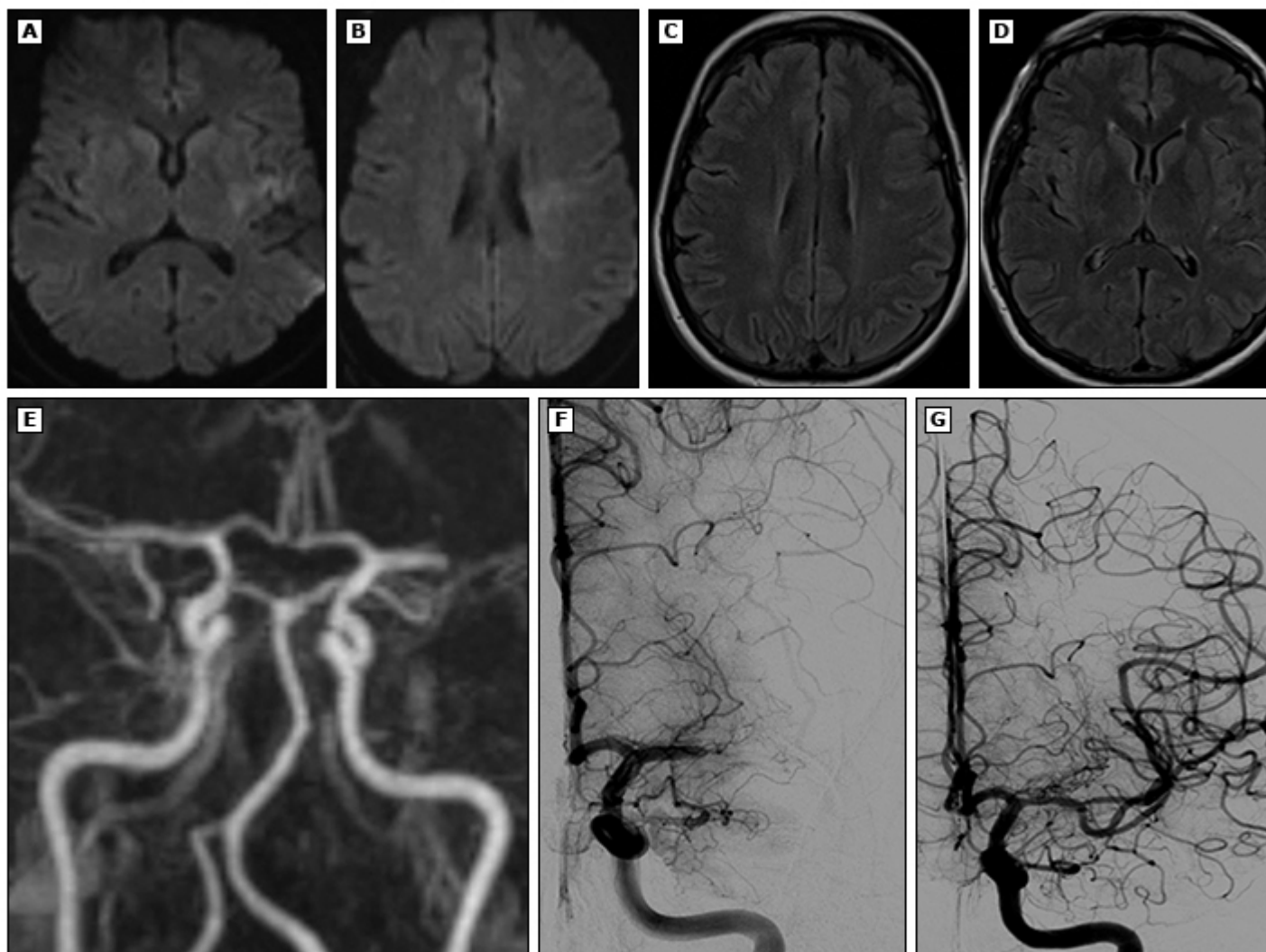
---

*Glenn A Tung, MD, FACR.*

---

Graphic 129434 Version 2.0

## MRI showing DWI-FLAIR mismatch in acute ischemic stroke due to left middle cerebral artery occlusion



A 49-year-old woman presented with right hemiplegia and dysarthria. The patient arrived at the emergency department 72 minutes after symptom detection. Her initial NIHSS score was 13. Brain MRI showed an acute ischemic lesion in the left MCA territory on DWI (A and B) without parenchymal signal changes on FLAIR (C and D). MRA showed occlusion of the M1 segment of the left MCA with collateral blood flow in the distal cerebral artery territory (E). Intravenous thrombolysis was started 140 minutes after symptom detection. On DSA, the left MCA was still occluded on the M1 segment (F) and was recanalized after mechanical thrombectomy with a TICI score of 3 (G). Time from symptom detection to recanalization was 189 minutes (3 hours and 9 minutes). The mRS score at three months was 0.

---

NIHSS: National Institutes of Health Stroke Scale; MRI: magnetic resonance imaging; MCA: middle cerebral artery; DWI: diffusion-weighted imaging; FLAIR: fluid-attenuated inversion recovery; MRA: magnetic resonance angiography; DSA: digital subtraction angiography; TICI: thrombolysis in cerebral infarction; mRS: modified Rankin scale.

---

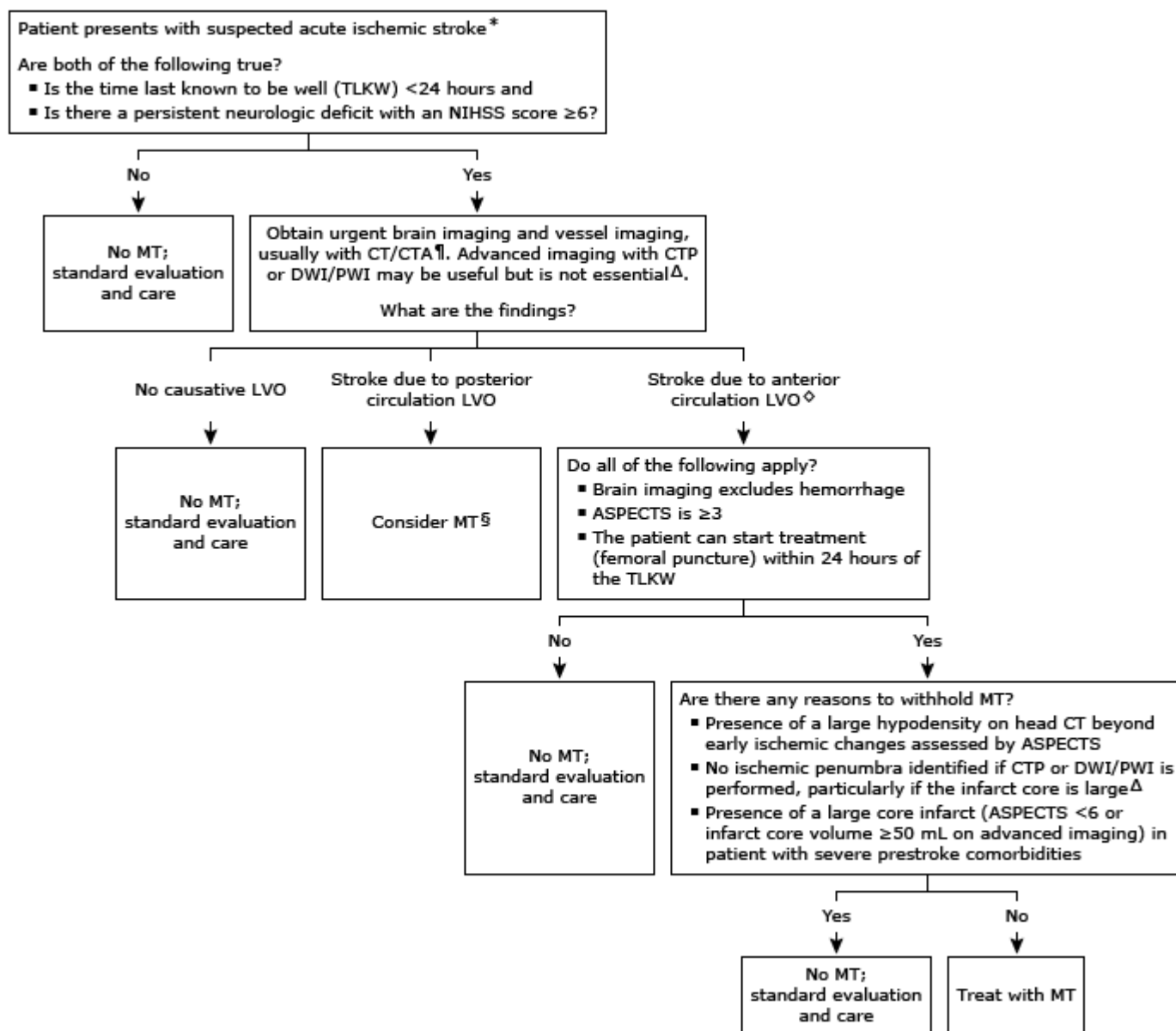


*Used with permission of the American Society of Neuroradiology, from: Mourand I, Milhaud D, Arquizan C, et al. Favorable bridging therapy based on DWI-FLAIR mismatch in patients with unclear-onset stroke. AJNR Am J Neuroradiol 2016; 37:88; permission conveyed through Copyright Clearance Center, Inc. Copyright © 2016.*

---

Graphic 129421 Version 1.0

# Patient selection for mechanical thrombectomy to treat acute ischemic stroke



MT should be performed at a stroke center with appropriate neurointerventional expertise. The TLKW is judged to be the time of stroke symptom onset (if known), or the time the patient was last seen at their baseline for patients with wake-up stroke or those found down. The ASPECTS method is used to assess the extent of ischemic changes on head CT or DWI. Refer to UpToDate topics for a full discussion of MT, including calculation of ASPECTS, NIHSS, and mRS, and other clinical issues pertaining to MT.

ACA: anterior carotid artery; ASPECTS: Alberta Stroke Program Early CT Score; CT: computed tomography; CTA: computed tomography angiography; CTP: computed tomography perfusion; DSA: digital subtraction angiography; DWI: diffusion-weighted magnetic resonance imaging; ICA: internal carotid artery; LVO: large vessel (artery) occlusion; MCA: middle cerebral artery; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging; mRS: modified Rankin Scale; MT: mechanical thrombectomy; NIHSS: National Institutes of Health Stroke Scale; PWI: perfusion-weighted magnetic resonance imaging.

\* All patients with acute ischemic stroke should be simultaneously evaluated for treatment with intravenous thrombolysis; refer to UpToDate topics for details.

¶ Urgent imaging is usually done with CT and CTA, less often with MRI and MRA.

Δ Advanced imaging is not essential but can be used at centers with CTP or DWI/PWI and automated software imaging analysis to determine infarct volume; patients can be selected for MT if they have salvageable brain tissue with a mismatch between a relatively larger area of hypoperfusion (ischemic penumbra) and a smaller area of irreversibly injured brain tissue (infarct core). Refer to UpToDate text for details.

◇ There is intracranial arterial occlusion of the distal ICA, or the M1 or proximal M2 segment of the MCA, as shown by CTA, MRA, or DSA.

§ MT may be a treatment option at expert stroke centers for patients with basilar artery LVO, but benefit is uncertain.

