



Q: Should patients with TIAs be hospitalized?

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A: Some patients with transient ischemic attacks (TIAs) may need to be hospitalized for specific reasons, eg, to undergo imaging studies or treatment that cannot be done immediately on an outpatient basis or if the TIA symptoms recur or are ongoing at the time of presentation. The important point is that TIAs are not benign and that the causes of TIAs should be urgently diagnosed and treated.

■ TIAs ARE NOT BENIGN

In contrast to strokes, TIAs are widely—and erroneously—assumed to be benign. However, recent research suggests that after a first TIA, as many as one of every five patients suffers a permanent stroke within 3 months, and that in half of these patients this occurs very shortly after the initial TIA. Both the public and medical professionals need to be made more aware of the serious implications of a TIA.

The risk of early recurrence is highest in patients with carotid occlusive disease and lowest in those with stroke related to small-vessel disease (lacunar stroke).^{1,2}

■ NEW DEFINITION OF TIA

The classic definition of TIA is “a sudden focal neurologic deficit lasting for less than 24 hours, of presumed vascular origin, and confined to an area of the brain or eye perfused by a specific artery.”³

This time-based definition emerged in the 1950s and 1960s, long before brain imaging was available.^{3,4} The 24-hour criterion was based on the arbitrary assumption that if the syndrome persists for 24 hours or longer, an injury to the brain parenchyma should be detectable by microscopy.

Modern brain-imaging techniques, in particular magnetic resonance imaging (MRI), have substantially altered this assumption, and clinical, experimental, and imaging data have revealed that a definition based on time alone may not accurately predict the absence of brain injury.⁵

In view of these findings, Albers and the TIA Working Group recently proposed revising the definition to “a brief episode of neurologic dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of acute infarction.”⁶

The proposed new definition of TIA emphasizes that we should view stroke and TIA as on the same spectrum of serious conditions involving brain ischemia. Both are markers of current or impending disability, with the only distinction that TIA offers a much greater opportunity to initiate treatments that can forestall the possible onset of brain infarction.

In addition, the proposed new definition acknowledges that transient ischemic symptoms may cause permanent brain injury, and it encourages the use of neurodiagnostic tests to identify brain injury and its cause in order to permit rapid interventions for acute brain ischemia. The concept is analogous to the distinction between myocardial infarction and unstable angina or non-Q-wave syndrome, and it should be translated into clinical practice accordingly.

■ CURRENT MANAGEMENT: UNCERTAINTIES PERSIST

Neither practitioners nor patients are sure exactly what to do when a TIA occurs. Indeed, data on the management of patients with TIAs are still largely lacking, in contrast to the situation with strokes. Consensus guidelines for the care of patients with a TIA are summarized in **TABLE 1**.¹

Both TIA and stroke are markers of impending disability

**TABLE 1****Consensus guidelines for the care of patients with a transient ischemic attack**

INTERVENTION	AMERICAN HEART ASSOCIATION	NATIONAL STROKE ASSOCIATION
Evaluation		
Timing	Prompt	Within hours of onset of symptoms
Hospitalization	No recommendation	If appropriate imaging studies are not immediately available
Laboratory tests	Determined on the basis of history Used to identify causes of TIA that would require specific therapy, to assess modifiable risk factors, and to determine prognosis	No specific recommendations
Electrocardiography	Recommended	Recommended
Imaging of the head	Computed tomography (CT) in all patients Routine use of magnetic resonance imaging not recommended, owing to higher cost and lower tolerability	No specific recommendation
Imaging of carotids	Prompt ultrasonography, magnetic resonance angiography, or CT angiography	Urgent evaluation (not further specified)
Antithrombotic medications		
If cardioembolic cause	No specific recommendation on short-term use of heparin Long-term oral anticoagulation for patients with atrial fibrillation	Acute anticoagulation can be considered (limited evidence in support)
If noncardioembolic cause	Antiplatelet therapy: aspirin 50–325 mg/day, clopidogrel, ticlopidine, or aspirin plus dipyridamole Anticoagulation not generally recommended	Antiplatelet therapy: aspirin 50–325 mg/day
Carotid endarterectomy	Recommended for good surgical candidates with 70%–99% stenosis and TIA within previous 2 years Consider for patients with 50%–69% stenosis on the basis of clinical features that influence risk of stroke and risk of complications; timing not discussed	Recommended without delay for those with symptomatic stenosis of 50%–99%

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A patient with a TIA has, by definition, already recovered from a focal deficit. On the other hand, symptoms noted during a clinical evaluation may or may not resolve, ie, the symptoms may or may not be transient. Therefore, we should treat every patient with a focal neurologic deficit during the evaluation as having had an acute stroke.

CT of the head for all. In most emergency rooms, standard care for patients with a suspected TIA includes computed tomography (CT) without contrast to rule out hemorrhagic stroke, in addition to basic laboratory tests, and electrocardiography. Patients receive a prescription for aspirin and are discharged to their primary care physician for follow-up.



Universal hospitalization is not feasible. Some argue that since a TIA implies nothing less than an ischemic stroke, all patients should be admitted for prompt diagnosis and initiation of secondary prevention. This may not be feasible, given that the diagnosis of TIA is “soft” and often overused.

Sometimes the clinical scenario dictates admission:

If the TIA has resolved before presentation, the patient would be admitted only if he or she is a candidate for carotid intervention or urgent anticoagulation (see below). Other situations that call for hospitalization are:

- Recurrent stereotypical TIAs
- Ongoing TIA symptoms at the time of presentation, even if they resolve.

Carotid imaging. In view of the data on early risk of stroke after TIA, it may be prudent to order carotid ultrasonography or an equivalent carotid study to investigate for carotid occlusive disease that would prompt admission and urgent intervention.

Anticoagulation for some. Another reason to admit patients with TIA is to begin urgent anticoagulation when arterial dissection, atrial fibrillation, or cardiogenic embolism is suspected. A cost-effective strategy may be an observation unit within the emergency department or a specialized stroke clinic.

■ FUTURE MANAGEMENT: MRI AND MRA

MRI and magnetic resonance angiography (MRA) are the premier diagnostic tools to expeditiously assess patients with TIA. The

diagnostic yield of MRI for TIA and stroke is much higher than that of CT, but the yield depends on the duration of symptoms, the presence of localizing cortical signs, and stroke risk factors such as history of cerebrovascular disease, diabetes mellitus, or atrial fibrillation.

TIA with negative imaging results remains a diagnostic dilemma. Besides faulty clinical impression or misinterpretation of the diagnostic imaging, technical causes of false-negative MRI relate to limited signal-to-noise ratio of current diffusion-weighted imaging pulse sequences, and these will likely improve in the near future. Therefore, the acronym “TIA” may before too long stand instead for “tiny infarct apparent,”⁷ so that no ambiguities would remain about whether to admit a patient with TIA.

The size, location, and number of ischemic lesions on MRI give clues to their cause. MRI can predict stroke related to small-vessel disease, large-vessel disease, and embolism with acceptable accuracy. Additional angiographic MRI sequences can visualize the entire cervicocranial vasculature in a few minutes and enable the clinician to initiate the appropriate next step—either discharge with antithrombotic medication and risk factor management, or admission for urgent carotid revascularization. Patients in whom MRI suggests an embolic cause but normal cervicocranial vasculature may require transthoracic and transesophageal echocardiography to assess for cardiac and aortic sources of embolism. 

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MRI is likely to replace CT for evaluating TIA and stroke