INTRODUCTION — Transient ischemic attack (TIA) is now defined as a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction [1]. The end point is biologic (tissue injury) rather than arbitrary (24 hours). In keeping with this definition of TIA, ischemic stroke is defined as an infarction of central nervous system tissue.

TIA was originally defined as a sudden onset of a focal neurologic symptom and/or sign lasting less than 24 hours and caused by reversible cerebral ischemia. However, this classic definition of TIA was inadequate for several reasons. Most notably, there is risk of permanent tissue injury (ie, infarction) even when focal transient neurologic symptoms last less than one hour. Furthermore, about one-half of patients with classically defined TIA syndromes (<24 hours in duration) have corresponding appropriate ischemic lesions by brain MRI on diffusion-weighted or perfusion-weighted imaging. (See "Definition of transient ischemic attack", section on 'Relationship of symptom duration and infarction'.)

Thus, the benign connotation of the term TIA has been replaced by an understanding that even relatively brief ischemia can cause permanent brain injury.

Of greater importance, patients with TIA or minor stroke are at increased risk of recurrent stroke, and therefore require urgent evaluation and treatment since immediate intervention may substantially reduce the risk of recurrent stroke.

This topic will review the diagnostic approach and early management of TIA and minor nondisabling stroke.

Other aspects of transient cerebral ischemia are discussed separately. (See "Definition of transient ischemic attack" and "Etiology and clinical manifestations of transient ischemic attack" and "Differential diagnosis of brain ischemia".)

The management of patients hospitalized with acute stroke is reviewed elsewhere. (See "Initial assessment and management of acute stroke".)
INITIAL EVALUATION — Patients who have had a suspected TIA require urgent evaluation due to the high stroke risk associated with TIA [1]. Furthermore, immediate intervention after a TIA may prevent a significant number of strokes. (See 'Prognosis' below and 'Urgent treatment' below.)

The initial evaluation of suspected TIA and minor nondisabling ischemic stroke includes basic laboratory studies that are suggested by the history and physical examination, an electrocardiogram, brain imaging, and neurovascular imaging. Laboratory testing is helpful in ruling out metabolic and hematologic causes of neurologic symptoms, including hypoglycemia, hyponatremia, and thrombocytosis.

Several neurologic disorders give rise to transient focal neurologic symptoms, and these should be considered before establishing a diagnosis of TIA. In addition to TIAs, the most important and frequent causes of discrete self-limited attacks include:

- Seizures
- Migraine auras
- Syncope

Less frequent causes include pressure- or position-related peripheral nerve or nerve root compression that causes transient paresthesias and numbness; peripheral vestibulopathies that cause transient episodic dizziness; and metabolic perturbations such as hypoglycemia and hepatic, renal, and pulmonary encephalopathies that can produce temporary aberrations in behavior and movement. (See "Differential diagnosis of brain ischemia".)

Hospitalization versus ambulatory evaluation — Whether hospitalization is required for TIA evaluation is not clear, but urgent assessment and management is essential regardless of inpatient or outpatient status [1-5].

Possible advantages of hospitalization include facilitated early use of thrombolytic therapy and other medical management if symptoms recur, expedited TIA evaluation, and expedited institution of secondary prevention [5].

The 2009 American Heart Association and American Stroke Association (AHA/ASA) guidelines for the definition and evaluation of TIA state that it is reasonable to hospitalize patients with TIA who present within 72 hours of symptom onset and meet any of the following criteria [1]:

- ABCD² score (table 1) of ≥3
- ABCD² score of 0 to 2 and uncertainty that the diagnostic workup can be completed within two days as an outpatient
- ABCD² score of 0 to 2 and other evidence that the event was caused by focal ischemia

The ABCD² score (ie, ABCD squared, for Age, Blood pressure, Clinical features, Duration of symptoms, and Diabetes) is a simple prognostic assessment tool with moderate predictive accuracy that was designed to identify patients at high risk of ischemic stroke in the first two days after TIA, as discussed later in detail (table 1). (See 'Predicting stroke risk after TIA' below.)

The 2006 National Stroke Association (NSA) guidelines systematically reviewed, critically evaluated, and updated prior published guidelines for the management of TIA [5]. The following consensus recommendations regarding initial management were proposed, based
mainly on evidence from observational studies and clinical experience:

- The National Stroke Association recommends that hospitalization be considered for patients with a first TIA within the past 24 to 48 hours, and is generally recommended for patients with the following conditions:
  
  Crescendo TIAs
  Duration of symptoms >1 hour
  Symptomatic internal carotid artery stenosis >50 percent
  Known cardiac source of embolus such as atrial fibrillation
  Known hypercoagulable state
  High risk of early stroke after TIA (see 'Predicting stroke risk after TIA' below)

- Patients who need urgent evaluation and are not hospitalized should have rapid access to the following studies:
  
  Brain imaging with head CT and/or MRI
  Neurovascular studies such as CT angiography (CTA), MR angiography (MRA), and/or ultrasound
  Electrocardiogram (ECG)

- All patients with a TIA within the past two weeks who are not hospitalized should undergo investigations within 24 to 48 hours to determine the mechanism of ischemia and subsequent preventive therapy

- Patients who are not admitted should be informed that they need to go to an Emergency Department immediately if symptoms recur

Hospitalization for TIA may be cost effective, particularly for high-risk patients eligible for intravenous (IV) tPA treatment. Supporting evidence comes from a cost-utility analysis of 24-hour hospitalization in the United States for patients diagnosed with recent TIA [6]. The study assumed that only those patients without absolute contraindications for IV tPA treatment would be admitted, and that hospitalized patients who developed a new stroke would be identified and treated with tPA within one hour of stroke onset. The overall cost effectiveness ratio was 55,044 dollars per quality adjusted life year, a value considered borderline cost effective. However, admission was cost effective for patients with a 24-hour stroke risk >5 percent.

**Brain imaging studies** — Brain imaging with CT or MRI is indicated in all patients with suspected TIA or minor stroke as soon as possible, particularly those with symptoms suggestive of hemispheric TIA [1,5]. The 2009 AHA/ASA guidelines recommend neuroimaging within 24 hours of symptom onset and further recommend MRI and diffusion-weighted MR imaging as preferred modalities [1]. Head CT is recommended if MRI cannot be performed.

The presence of a brain infarct on CT or MRI scan located in an area suggested by the anatomy of the TIA or stroke (eg, in the left precentral gyrus or left internal capsule in a patient with transient right arm and leg weakness) identifies a vascular etiology of symptoms.
Many patients whose clinical history and neurologic examination suggest that an attack was transient have infarcts in brain areas appropriate to the neurologic symptoms. (See "Definition of transient ischemic attack", section on 'Relationship of symptom duration and infarction'.)

Infarction is more likely to be identified acutely on MRI than on CT. Diffusion-weighted MRI imaging (DWI) and the apparent diffusion coefficient (ADC) map that is derived from DWI can discriminate tissue injury very early after the onset of ischemic symptoms. DWI reliably confirms whether only ischemia has occurred or if there has also been an element of infarction. These techniques can differentiate stroke from TIA within the first hours after symptom onset. (See "Neuroimaging of acute ischemic stroke".)

Other diseases that mimic TIAs usually can be identified by neuroimaging techniques, although pathological biopsy examination is occasionally needed (eg, temporal artery biopsy or examination of the cerebrospinal fluid). In rare cases, brain biopsy is indicated. (See "Differential diagnosis of brain ischemia".)

**Neurovascular evaluation** — The single most important issue to resolve in the initial evaluation of TIA and ischemic stroke is whether or not there is an obstructive lesion in a larger artery supplying the affected territory. Noninvasive options for evaluation of large vessel occlusive disease include magnetic resonance angiography (MRA), computed tomography angiography (CTA), carotid duplex ultrasonography (CDUS), and transcranial Doppler ultrasonography (TCD). The choice among these depends upon local availability and expertise as well as individual patient characteristics and preference [1]. (See "Neuroimaging of acute ischemic stroke".)

The 2009 AHA/ASA guidelines recommend routine noninvasive imaging of the cervicocephalic vessels as part of the evaluation of patients with suspected TIA [1]. The guidelines note that it is reasonable to obtain noninvasive testing of the intracranial vasculature if knowledge of an intracranial stenosis or occlusion will alter management.

Although noninvasive studies are considered reliable for the exclusion of intracranial stenosis, the 2009 AHA/ASA guidelines conclude that reliable diagnosis of the presence and degree of intracranial stenosis requires evaluation with catheter angiography to confirm abnormalities detected with noninvasive testing [1].

A focused Doppler and neuroimaging test (eg, MRI or CT angiography) can be used to establish an arterial source of the embolism or low flow. These tests can exclude an arterial source in cases where the symptoms are due to proximal embolism from the heart, aorta, or an unknown source, and in cases where the symptoms are due to small vessel disease. (See "Evaluation of carotid artery stenosis".) The following are important aspects regarding such testing:

- Duplex ultrasound and transcranial Doppler studies require considerable technical skill to perform and an experienced interpreter. They should be used only if there is adequate confidence that the testing and interpretation is reliable.

- Conventional angiography is associated with a small risk of stroke and should be performed by experienced physicians. It should only be considered when the diagnosis is uncertain by noninvasive methods, and when proof of the diagnosis is essential for
proper stroke preventive therapy. As an example, if one of the stroke-producing arterial lesions noted above is suspected but not confirmed by conventional noninvasive Doppler, MRI, or CT methods, then angiography can be considered.

The distinction between artery-to-artery and other (mainly cardiac) sources of embolism can be difficult. Suspicion of the former typically arises once vascular pathology in a large vessel has been identified (eg, with noninvasive testing). Repetitive spells within a single vascular territory are also suggestive of an artery-to-artery source, as is a normal echocardiogram.

The following sections review the diagnostic studies are useful to confirm the origin of transient cerebral ischemia.

**Anterior circulation** — MRI with MR angiography or CT with CT angiography can be used to identify the source of the stenosis when TIA or minor stroke due to low-flow or artery-to-artery embolism is suspected in the anterior cerebral circulation (figure 1 and figure 2). Diffusion and perfusion-weighted MRI imaging can describe areas of the brain that are focally ischemic or infarcted.

Duplex ultrasound and transcranial Doppler can evaluate flow at the bifurcation of the common carotid artery, in the siphon portion of the internal carotid artery, in the ophthalmic artery, and in the middle, anterior, and posterior cerebral artery stems. A positive test rapidly identifies the location of the arterial pathology giving rise to the hemodynamically significant low-flow symptoms. However, a negative test does not rule out such a phenomenon. (See "Evaluation of carotid artery stenosis".)

Extracranial carotid artery evaluation is discussed in greater detail separately. (See "Evaluation of carotid artery stenosis".)

**Posterior circulation** — Patients with symptoms referable to the posterior circulation should have MRA or CTA of the neck. Extracranial vertebral disease is a relatively uncommon source of stroke, but dissection and vertebral origin atherosclerosis can be missed if these vessels are not imaged [7]. The vertebral origins are best imaged noninvasively using MRA with contrast or CTA.

Duplex ultrasound of the proximal vertebral artery or transcranial Doppler insonation of flow in the distal vertebral arteries and in the basilar artery can give reliable clues to the presence of posterior cerebral circulation disease (figure 3). However, the diagnosis is confirmed only by MRA or CTA.

- Vertebral artery stenosis is suspected when duplex ultrasound of mid-vertebral artery flow shows a reduced peak systolic velocity. MR angiography of the aortic arch and CT angiography can identify vertebral origin lesions.

- Distal vertebral lesions and proximal basilar lesions that give rise to embolism can be confirmed by CTA or MRA.

- Transcranial Doppler assessment of flow at the top of the basilar artery may be helpful in identifying emboli there that obstruct flow but may not block it completely. The demonstration of normal flow at the top of the basilar artery does not exclude such a lesion, and CTA or MRA are suggested in this setting if clinical suspicion is high. (See
Rarely, conventional angiography with its risks is necessary to confirm the diagnosis of posterior cerebral circulation disease in order to plan for therapy.

**Intracranial large vessels** — Atherosclerotic stenosis of the major intracranial arteries (carotid siphon, middle cerebral artery, vertebral artery, and basilar artery) is an important cause of ischemic stroke, especially in blacks, Asians, and Hispanics. (See "Intracranial large artery atherosclerosis", section on 'Epidemiology'.)

The gold standard for establishing the diagnosis of intracranial large artery disease is conventional cerebral angiography. Noninvasive studies such as TCD, MRA, and CTA may be used, but noninvasive testing may not be sufficiently reliable to diagnose intracranial arterial stenosis accurately. (See "Intracranial large artery atherosclerosis", section on 'Diagnosis'.)

Despite this limitation, we suggest the use of noninvasive methods (CTA, MRA, or TCD) for the initial evaluation of intracranial vessels in patients with the following higher prevalence situations [7):

- Patients of black, Asian, or Hispanic ethnicity
- Patients younger than age 50 without clear cardiac or extracranial source
- Patients with recurrent stereotyped TIAs
- Patients with posterior circulation event and no clear cardiac source
- Patients with preoperative evaluation of collateral circulation before carotid endarterectomy

In most cases, cardiac and extracranial neurovascular imaging should be performed prior to the evaluation of intracranial large arteries, as the pretest probability of these entities is higher in both younger and older patients [7]. In addition, definitive management for most causes of intracranial disease is unclear. (See "Intracranial large artery atherosclerosis".)

**Small vessels** — Small vessels are not directly visible with any of the currently available imaging techniques. However, evaluation of the extracranial carotid artery is recommended for patients with lacunar infarction or suspected small vessel (lacunar) TIA referable to the anterior circulation [7].

Further noninvasive neurovascular studies in the vascular territory appropriate to the symptoms may be considered in the following settings [7]:

- Absence of vascular risk factors
- Clinically atypical lacunar syndrome
- Evidence of a lacune in an atypical territory by imaging
- Typical lacunar syndrome with nonlacunar infarct by imaging

Lacunar infarction and small vessel disease are discussed in detail separately. (See "Lacunar infarcts".)

**Cardiac evaluation** — A possible cardiac source should be considered in patients with embolic TIA or minor stroke caused by embolism. At minimum, such patients should have a standard 12-lead electrocardiogram as soon as possible after symptom onset [1].

**Echocardiography** — Echocardiography is indicated for patients who are candidates for
anticoagulation or for patients who have suspected endocarditis [7]. It is not clearly indicated for patients who require anticoagulation for another reason (eg, for atrial fibrillation) unless there is a suspicion of endocarditis.

Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) methods each have unique advantages.

- TTE is noninvasive, but it can be limited by an inadequate window in large patients.
- TTE is better than TEE for detecting apical left ventricular thrombi and mitral annular calcification in the elderly.
- TTE can provide a better estimate of left ventricular function than TEE.
- TEE is the test of choice for evaluation of aortic atherosclerosis. However, definitive management of this entity is not clear, and some experts argue that it is not essential to make this diagnosis [7]. Suspicion for aortic disease is increased in the setting of multiple arterial territory infarcts without clear cardiac source or a history of peripheral artery disease.
- TEE is superior to TTE for detecting spontaneous echo contrast ("smoke-like" echoes) within the left atrium, a finding which is associated with left atrial thrombi.
- TEE is superior for detecting abnormalities of the interatrial septum (eg, atrial septal defect, patent foramen ovale, and interatrial septal aneurysm) that are associated with thromboembolism.
- TEE is also the best way to visualize the left atrium, including the atrial appendage, which is the main site of thrombus formation.

2009 AHA/ASA guidelines conclude that echocardiography, at least transthoracic echocardiography, is reasonable when no cause for TIA or ischemic stroke has been identified by other aspects of the work-up [1]. Transesophageal echocardiography, useful for identifying patent foramen ovale, aortic arch atherosclerosis, and valvular disease, is reasonable when identification of any of these conditions would alter management.

The use of echocardiography for the detection of cardiac sources of embolism is discussed in greater detail separately. (See "Echocardiography in detection of intracardiac sources of embolism".)

Although transthoracic echocardiography is less sensitive than TEE for most of the disorders associated with cardiac embolism, we recommend TTE with agitated saline contrast as a first test because it is noninvasive and better tolerated. We proceed to TEE if TTE is negative and treatment decisions hinge on identifying one of the possible TEE findings.

**Cardiac monitoring** — Cardiac monitoring is an essential part of evaluation to exclude atrial fibrillation in the setting of embolic TIA or stroke. The 2009 AHA/ASA guidelines consider prolonged cardiac monitoring with inpatient telemetry or Holter monitor to be useful for patients without a clear etiology after initial brain imaging and electrocardiography [1].

Holter monitoring or continuous telemetry may be most useful in patients with a history of
palpitations, paroxysmal atrial fibrillation, evidence of spontaneous echo contrast on TEE, and cryptogenic TIA [7]. Although the electrocardiogram and Holter monitoring can identify patients who have atrial fibrillation, the demonstration of normal sinus rhythm does not exclude intermittent atrial fibrillation. (See "Ambulatory monitoring in the assessment of cardiac arrhythmias".)

**Other** — Blood cultures, an erythrocyte sedimentation rate, or antinuclear antibody testing are indicated if bacterial or nonbacterial endocarditis is suspected.

**Blood tests** — In patients with suspected TIA, the following blood tests should be considered [1,5,7]:

- Complete blood count (CBC)
- Prothrombin time and partial thromboplastin time
- Serum electrolytes and creatinine
- Fasting blood glucose and lipids
- Erythrocyte sedimentation rate (ESR)

Suspicion for blood disorders as potential sources of cerebral ischemia should be raised in the following settings [7]:

- Cryptogenic stroke or TIA
- Age 45 or younger
- History of clotting dysfunction
- Multiple venous and arterial occlusions
- Suspected or confirmed cancer
- Family history of thrombotic events

In these settings, additional blood and coagulation studies should be considered. This topic is discussed elsewhere. (See "Overview of the evaluation of stroke", section on 'Blood tests'.)

**PROGNOSIS** — TIA is a neurologic emergency because patients with TIA and minor stroke are at increased risk of recurrent stroke [8-15]. This risk is illustrated by the following studies:

- A meta-analysis of 11 observational studies published through December 2006 found that the risk of stroke at 2 days, 30 days, and 90 days after TIA was 3.5, 8.0, and 9.2 percent, respectively [13]. In the three studies that used active ascertainment of stroke outcome (ie, face-to-face evaluation by a practitioner at three months rather than use of administrative data), the 2, 30, and 90 day risk of stroke after TIA was even higher (9.9, 13.4, and 17.3 percent, respectively). Similar findings were reported in a meta-analysis of 18 cohorts published through June 2007 [16].

- A prospective observational study of 1380 patients with TIA and 3855 patients with ischemic stroke found that subsequent stroke incidence during the hospital stay was 8 percent for patients with TIA and 7 percent for patients with ischemic stroke [17]. During the first six months after the initial ischemic event, recurrent stroke incidence was 13 percent for both groups. Two percent of patients with TIA died during hospital stay, and 17 percent were dependent at follow-up.

- A well-designed prospective cohort study of 2447 participants from the Dutch TIA trial
found that the risk for major vascular events and stroke was highest shortly after TIA or minor stroke, declined to its lowest point at about three years, and then progressively increased over the remainder of the 10-year follow-up (figure 4) [18]. In contrast, the risk for mortality gradually rose throughout the study. By 10 years, 60 percent had died and 54 percent had experienced new vascular events (stroke and myocardial infarction). Event-free survival was 48 percent. Predictive factors for risk of vascular events and death included age over 65 years, diabetes, claudication, previous vascular surgery, and pathologic Q waves on baseline electrocardiogram.

The urgency associated with TIA derives also from the observation that TIAs are most likely to occur in the hours and days immediately preceding ischemic stroke. As an example, a study that analyzed four cohorts of patients who had recent ischemic stroke found that TIAs occurred most often in the 48 hours prior to the stroke [19]. Another study found that the risk of ischemic stroke occurring within 24 hours of a probable or definite TIA was approximately 5 percent [20]. Of all ischemic strokes during the 30 days after a first TIA, 42 percent occurred within the first 24 hours. This may be an overestimate related to the difficulty distinguishing a single ischemic event (stroke) with fluctuating symptoms from separate events (TIA followed by stroke) within a short period of time. Nevertheless, these observations underscore the high early risk of developing a permanent deficit after transient ischemic symptoms and the importance of urgent assessment, risk stratification, and treatment.

Given this short time window and high risk of stroke — 4 to 10 percent in the first 48 hours after TIA [13] — neurologic evaluation of and intervention for TIA should occur urgently. Furthermore, clinical TIAs associated with evidence of infarction by neuroimaging may be a marker of particularly high risk for ischemic stroke. (See "Definition of transient ischemic attack", section on 'Transient symptoms with infarction'.)

Recognition and urgent evaluation of TIAs can identify patients who may benefit from preventive therapy or from revascularization of large vessels such as the carotid artery. As examples, premonitory carotid territory TIAs occur in approximately 50 to 75 percent of patients with ischemic stroke from extracranial carotid disease [21-23], and vertebrobasilar TIAs are associated with a risk of subsequent stroke or death that is similar to or possibly higher than that seen with carotid TIAs [24]. In addition, the large artery atherosclerosis subtype of TIA appears to be associated with a higher risk of stroke recurrence at 7 and 90 days after TIA than other subtypes (cardioembolism, small vessel disease, undetermined, or other determined cause) [25].

Predicting stroke risk after TIA — Methods that can reliably assess the risk of stroke after TIA in individual patients would be useful for triaging patients.

A simple assessment called the ABCD² score (ie, ABCD squared, for Age, Blood pressure, Clinical features, Duration of symptoms, and Diabetes) can be used to identify patients at high risk of ischemic stroke in the first seven days after TIA [26]. The ABCD² score is tallied as follows:

- Age (≥60 years = 1 point)
- Blood pressure elevation when first assessed after TIA (systolic ≥140 mmHg or diastolic ≥90 mmHg = 1 point)
- Clinical features (unilateral weakness = 2 points; isolated speech disturbance = 1 point;
other = 0 points)
- Duration of TIA symptoms (≥60 minutes = 2 points; 10 to 59 minutes = 1 point; <10 minutes = 0 points)
- Diabetes (present = 1 point)

The ABCD² score was based upon two earlier prognostic scores for TIA — the California score [11] and the ABCD score [27] — and was derived and validated using independent study populations (two derivation and four validation cohorts) from the US (California) and the UK (Oxford) that included 4809 patients with TIA [26]. The unified ABCD² score was a slightly more accurate predictor of stroke risk than either of its predecessors in these populations.

Estimated two-day stroke risks determined by the ABCD² score in the combined derivation and validation cohorts were as follows [26]:
- Score 6 to 7: High two-day stroke risk (8 percent)
- Score 4 to 5: Moderate two-day stroke risk (4 percent)
- Score 0 to 3: Low two-day stroke risk (1 percent)

The ABCD² score was designed to be used in primary care settings to stratify patients according to stroke risk and thus identify those who required emergency assessment by specialists. The ABCD² score identifies a moderate or high risk TIA (score ≥4) with high sensitivity (92 percent).

While the ABCD² score may be a useful clinical tool in primary care settings, it suffers from low specificity, limiting its utility for high risk populations. Its predictive performance is not optimal, and is generally lower in hospital settings compared with population based settings [26,28]. Validation studies of the ABCD² score have been inconsistent, with results ranging from high predictive value to predictions slightly better than chance [29-35].

Other studies have suggested that the ABCD² score is useful for distinguishing a true TIA from TIA mimics [36-39].

There is accumulating evidence suggesting that the findings of acute infarction on diffusion-weighted MRI (DWI) [33,34,40,41] or acute or chronic ischemic lesions on CT [42] after a TIA are important predictors of stroke. The 90 day risk of stroke after an imaging-negative TIA is <1 percent [33,34,40]. In contrast, after an imaging-positive TIA, the corresponding risk appears to be as high as 14 percent.

Risk models that combine information from acute DWI, noninvasive angiography and presumed TIA etiology in addition to the clinical ABCD² score may improve the accuracy of stroke risk prediction after TIA [33,34,43,44]. As an example, the CIP model incorporated diffusion-weighted MRI findings with a dichotomized ABCD² score [33]. The result was improved accuracy, compared with the ABCD² score alone, for stroke risk predictions at both two days and seven days after TIA. The requirement for MRI limits the widespread applicability of advanced risk prediction models.

**URGENT TREATMENT** — The preferred approach to treatment of TIA and ischemic stroke is to determine the pathophysiology of the event so that specific stroke preventive therapy can be prescribed. An overview of the treatment of specific causes of TIA and ischemic stroke is discussed elsewhere. (See "Secondary prevention for specific causes of ischemic stroke and
In addition to specific treatment, accumulating evidence suggests that immediate intervention after a TIA or minor ischemic stroke can reduce the risk of recurrent stroke compared with delayed intervention. This point is illustrated by the following reports:

- The prospective EXPRESS study evaluated the impact of expediting outpatient treatment for TIA or minor ischemic stroke [45]. In order to compare traditional with expedited treatment, the study was conducted in two phases. In phase one, 323 patients were seen in a traditional clinic setting where evaluation required a scheduled appointment and treatment recommendations were made to referring physicians. In phase two, 297 patients were seen in an urgent walk-in stroke clinic without having to arrange an appointment, and treatment was implemented immediately by clinic practitioners. In both phases, treatment of confirmed TIA or stroke was individualized according to patient characteristics, but generally included antiplatelet or anticoagulant therapy, statin therapy, antihypertensive medication, and carotid endarterectomy as required. Although EXPRESS was not a randomized trial, the study was nested in an ongoing population-based study of stroke and TIA, thus minimizing the potential problems of incomplete ascertainment and selection bias that complicate observational studies. The following observations were reported [45]:

  The median delay to assessment in the outpatient clinic was significantly reduced from phase one to phase two (3 days versus <1 day), as was the median delay to first prescription of treatment (20 days versus 1 day)

  The risk of recurrent stroke at 90 days was significantly lower for patients seen in phase two than for those seen in phase one (2.1 versus 10.3 percent; adjusted hazard ratio 0.20, 95% CI 0.08-0.49)

- The observational SOS-TIA study analyzed the rapid assessment of 1085 patients with suspected TIA in a hospital-based clinic with 24 hour access [46]. Patients were evaluated within four hours of admission, and those with a final diagnosis of confirmed or possible TIA (n = 845) received immediate treatment with a stroke prevention program that included antiplatelet or anticoagulant treatment and/or carotid revascularization as appropriate. At 90 days, the observed stroke rate was much lower than an expected stroke rate predicted by the ABCD² scores (1.24 versus 5.96 percent).

The results of this study should be interpreted with caution because of methodologic limitations, including the use of ABCD² scores to predict stroke risk, rather than determination of stroke risk in a control population [47].

Early evaluation and intervention for symptomatic carotid artery disease may be an important aspect of stroke prevention. Supporting evidence comes from a pooled analysis of the NASCET and ECST trials, which found that early carotid endarterectomy (within two weeks of a nondisabling stroke or TIA) significantly improved outcome compared with later surgery [48]. Thus, early identification of symptomatic carotid disease is critical. (See "Management of symptomatic carotid atherosclerotic disease", section on 'Timing of surgery'.)
Given these data, we recommend that appropriate diagnostic evaluation and stroke prevention treatment be implemented without delay, preferably within one day of the ischemic event, for patients who present with TIA or minor ischemic stroke.

**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 5th to 6th 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 10th to 12th 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 information: Stroke (The Basics")
- Beyond the Basics topics (see "Patient information: Stroke symptoms and diagnosis" and "Patient information: Transient ischemic attack")

**SUMMARY AND RECOMMENDATIONS**

- The initial evaluation of suspected TIA and minor nondisabling ischemic stroke includes brain imaging, neurovascular imaging, and a cardiac evaluation. Laboratory testing is helpful in ruling out metabolic and hematologic causes of neurologic symptoms. (See 'Initial evaluation' above.)

- TIA and minor nondisabling ischemic stroke are associated with a high early risk of recurrent stroke. The stroke risk in the first two days after TIA is approximately 4 to 10 percent. The ABCD² score (table 1) may identify patients at high risk of ischemic stroke in this time period. (See 'Prognosis' above.)

- Accumulating evidence suggests that immediate evaluation and intervention after a TIA or minor ischemic reduces the risk of recurrent stroke. For patients who present with TIA or minor ischemic stroke, we recommend that implementation of appropriate diagnostic evaluation and stroke prevention treatment without delay, preferably within one day of the ischemic event (Grade 1B). (See 'Urgent treatment' above.)

- Risk factor management is appropriate for all patients with ischemic stroke or TIA, and most patients should be treated with all available risk reduction strategies. Currently viable strategies include blood pressure reduction, statins, antiplatelet therapy, and lifestyle modification, including smoking cessation. These interventions are discussed in greater detail separately. (See "Secondary prevention of stroke: Risk factor reduction".)

- An overview of the treatment for specific causes of TIA and ischemic stroke is provided separately. (See "Secondary prevention for specific causes of ischemic stroke and transient ischemic attack".)
Some important recommendations apply to select patients with TIA:

For patients with TIA or ischemic stroke of atherothrombotic, lacunar (small vessel occlusive), or cryptogenic type, we recommend treatment with an antiplatelet agent (Grade 1A). This issue and the choice among specific antiplatelet agents (ie, aspirin, aspirin plus extended-release dipyridamole, or clopidogrel) are discussed separately. (See "Antiplatelet therapy for secondary prevention of stroke".)

For patients with atrial fibrillation and a recent ischemic stroke or TIA, we recommend oral anticoagulation (Grade 1A). We recommend aspirin for patients with atrial fibrillation and cardioembolic stroke who have contraindications to anticoagulant therapy (Grade 1B). Prevention of recurrent stroke in patients with atrial fibrillation reviewed in detail elsewhere. (See "Antithrombotic therapy to prevent embolization in nonvalvular atrial fibrillation".)

Selected patients with recently symptomatic carotid stenosis of 50 to 99 percent who have a life expectancy of at least five years are generally treated with carotid endarterectomy. This issue is discussed in detail separately. (See "Management of symptomatic carotid atherosclerotic disease".)

For patients with TIA or ischemic stroke having carotid endarterectomy, we recommend aspirin at a dose of 81 to 325 mg/day started before surgery (Grade 1A). (See "Carotid endarterectomy", section on 'Aspirin'.)

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REFERENCES


3. Lindley RI. Patients with transient ischemic attack do not need to be admitted to hospital for urgent evaluation and treatment: against. Stroke 2006; 37:1139.


**ABCD² score**

The ABCD² score can be used to identify patients at high risk of ischemic stroke in the first two days after TIA. The score is tallied as follows:

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<thead>
<tr>
<th><strong>Age:</strong></th>
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<td>≥60 years</td>
<td>1 point</td>
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<tr>
<td>&lt;60 years</td>
<td>0 points</td>
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**Blood pressure elevation when first assessed after TIA:**

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<td>Systolic ≥140 mmHg or diastolic ≥90 mmHg</td>
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<td>Systolic &lt;140 mmHg and diastolic &lt;90 mmHg</td>
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**Clinical features:**

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<td>Unilateral weakness</td>
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<td>Isolated speech disturbance</td>
<td>1 point</td>
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<td>Other</td>
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**Duration of TIA symptoms:**

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<tr>
<td>≥60 minutes</td>
<td>2 points</td>
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<tr>
<td>10 to 59 minutes</td>
<td>1 point</td>
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<td>&lt;10 minutes</td>
<td>0 points</td>
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**Diabetes:**

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<tr>
<td>Present</td>
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<td>Absent</td>
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Anatomy of the cerebral arterial circulation

Coronal section of cerebral hemisphere

- Anterior cerebral A.
- Middle cerebral A.
- Deep branches of middle cerebral A.
- Anterior choroidal A.
- Posterior cerebral A.

Representation of the territories of the major cerebral vessels.

Middle cerebral artery distribution and signs and symptoms of occlusion

**Signs and symptoms of occlusion**
- Paralysis of contralateral face, arm, and leg; sensory impairment over the same area (pinprick, cotton touch, vibration, position, two-point discrimination, stereognosis, tactile localization, barognosis, cutaneousgraphia
- Motor aphasia
- Central aphasia, word deafness, anomia, jargon speech, sensory agraphia, acalculia, alexia, finger agnosia, right-left confusion
- Apractognosia of the minor hemisphere (amorphosynthesis), anesognosia, hemisomatognosia, unilateral neglect, agnosia for the left half of external space, dressing "apraxia," constructional "apraxia," distortion of visual coordinates, inaccurate localization in the half field, impaired ability to judge distance, upside-down reading, visual illusions
- Homonymous hemianopsia (often homonymous inferior quadrantnopsia
- Paralysis of conjugate gaze to the opposite side

**Structures involved**
- Somatic motor area for face and arm and the fibers descending from the leg area to enter the corona radiata and corresponding somatic sensory system
- Motor speech area of the dominant hemisphere
- Central, suprasylvian speech area and parieto-occipital cortex of the dominant hemisphere
- Nondominant parietal lobe (area corresponding to speech area in dominant hemisphere); loss of topographic memory is usually due to a nondominant lesion, occasionally to a dominant one
- Optic radiation deep to second temporal convolution
- Frontal contraversive field or fibers projecting therefrom

Development of yearly risks over time after TIA or minor stroke

Initial evaluation and management of transient ischemic attack ...

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