TABLE 116-4 STROKE MIMICS AND DIFFERENTIAL DIAGNOSIS

COMMON MIMICS
Metabolic encephalopathy (hypoglycemia, hyponatremia, etc.)
Systemic infection
Seizure
Migraine
Brain tumors
OTHER MIMICS
Transient focal neurological symptoms associated with amyloid angiopathy
Positional vertigo
Cardiac events
Syncope
Trauma (especially acceleration-deceleration without evidence of external injury)
Subdural hematoma
Herpes simplex virus encephalitis
Transient global amnesia
Dementia
Demyelinating disease
Cervical spine disease/radiculopathy/fracture
Myasthenia gravis
Parkinsonism
Hypertensive encephalopathy
Conversion disorder
Intoxication/substance abuse

metabolic and infectious causes of neurologic deterioration must be excluded in patients with a history of earlier brain injury before diagnosing a new stroke. Focal signs may also occur with metabolic disturbances in patients without a prior history of stroke.

External signs of injury are usually present in brain *trauma*, but they need not be present after acceleration-deceleration injury, such as from a motor vehicle accident. The most frequent sites of brain contusions are the frontal and temporal poles, which are not typical locations for strokes.

Seizures may occasionally complicate acute stroke, but they may also mimic stroke. Unlike stroke, seizures are often characterized by obtundation, an amnestic state, clonic activity, incontinence, or tongue biting. The postictal deficit, often called a *Todd's paralysis*, resembles stroke and weakness or language and other cortical deficits may occur. The deficits after seizure usually resolve within hours after the seizure, but occasionally persist for up to a week, making the distinction from stroke difficult. Seizures may also develop months or years after an infarct or hemorrhage, and the postictal state in these patients may recapitulate the initial stroke syndrome.

Migraine with persistent aura often mimics stroke or TIA. Aura alone, without headache (i.e., acephalgic migraine), is sometimes experienced by those who previously suffered from migraine with aura. Migraine aura typically produces a visual disturbance that marches across the vision of both eyes as an advancing, enlarging blind spot that takes 20 to 30 minutes to resolve. Subsequent unilateral, pounding headache suggests the diagnosis, but may not occur. Less often, migrainous auras take the form of sensory symptoms. The speed of the march is generally slower than the rapid spread of symptomsin stroke.

As many as 10% of *brain tumors* present with acute transient symptoms reflecting intratumoral hemorrhage or focal seizures. Seizures often precede focal signs. CT scan usually demonstrates an enhancing mass even when symptoms are mild.

TABLE 116-5 EVIDENCE-BASED PRIMARY PREVENTION OF ISCHEMIC STROKE

RISK FACTOR	TREATMENT
Hypertension	Anti-hypertensives
Myocardial infarction	HMG-CoA reductase inhibitors
Hyperlipidemia	HMG-CoA reductase inhibitors
Atrial fibrillation	Anticoagulation (warfarin, other agents)
Diabetes mellitus/vascular disease	ACE inhibitor
Diabetes mellitus type II, obesity	Metformin
Aymptomatic carotid stenosis (60-99%)	Carotid endarterectomy
High vascular risk populations	Antiplatelet therapy

TREATMENT

Stroke prevention and treatment are directed toward: (1) preventing the first stroke (primary prevention); (2) limiting damage from the stroke; (3) optimizing functional recovery following stroke; and (4) avoiding recurrence (secondary prevention). Specific measures for treatment and prevention depend on the patient's risk factors and stroke mechanism. The diagnostic evaluation of the stroke patient dictates optimal therapy.

Primary Prevention of Stroke

Randomized trials have demonstrated that specific interventions prevent first stroke among patients with specific risk factors (Table 116-5). Treatment of hypertension, for example, is associated with up to a 45% reduction in the risk of stroke (Level A). Among patients with atrial fibrillation, the use of warfarin is associated with a 60% to 70% relative reduction in risk of stroke, though younger patients without any accompanying heart disease, hypertension, or diabetes may be managed with antiplatelet agents alone (Level A). Hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, or statins, have been shown in some primary prevention studies, and in studies of patients with heart disease, to reduce the risk of a first stroke as well as that of heart disease (Level A). The effects on stroke risk are more modest than the effects on heart disease, possibly reflecting the greater heterogeneity among causes of stroke compared to heart disease. For patients with asymptomatic carotid stenosis of at least 60%, carotid endarterectomy reduces the risk of stroke, though the effect is much more modest than in symptomatic patients, and the number of patients needed to treat to prevent one stroke is greater. Because many of the large randomized trials of endarterectomy for asymptomatic patients were conducted in the era before the current recommended use of statins and antiplatelet agents, it is no longer clear that surgery is superior to medical therapy. New trials are addressing medical versus surgical treatment.

Antiplatelet therapy is not of established benefit for prevention of a first stroke. In a large primary prevention study, for example, aspirin use was associated with an increased risk of both ischemic and hemorrhagic stroke, despite reducing the risk of ischemic heart disease. However, other studies have shown that aspirin reduces the risk of ischemic stroke among women over the age of 45 (Level B).