strokes, about 87% are ischemic infarctions, 10% primary hemorrhages, and 3% SAHs. Among adults age 35 to 44, the incidence of stroke is 30 to 120/100,000 per year, and for those age 65 to 74, the incidence is 670 to 970/100,000 per year. Stroke incidence rates are approximately twice as high for African Americans as for whites. In northern Manhattan, Caribbean Hispanics had an incidence rate intermediate between that of whites and blacks. Temporal trends in stroke incidence suggest that stroke incidence rates have been declining since 1950; however, disparities in stroke incidence and mortality have increased.

Stroke incidence increases with age, but strokes do occur in young adults and children, and may be missed if the diagnosis is not considered. Although stroke incidence rates are higher for men than women at most ages, among young adults the rates are similar or higher among women, probably related to pregnancy, hormonal contraception, and other hormone-related differences. At older ages, incidence rates among women are again greater, and because women tend to live longer than men, overall about 60,000 more women than men have a stroke each year.

MODIFIABLE RISK FACTORS

Well-established modifiable stroke risk factors include hypertension, cardiac disease (particularly atrial fibrillation), diabetes, hyperlipidemia, cigarette use, physical inactivity, alcohol abuse, asymptomatic carotid stenosis, and a history of TIAs (Table 116-2).

Hypertension is the most powerful modifiable stroke risk factor and is associated with both ischemic and hemorrhagic strokes. Risk of stroke decreases with lower systolic and diastolic blood pressures, and this graded decrement in risk persists down to levels as low as 115/75. There is no clearly defined threshold level below which stroke risk levels off.

Cardiac disease is associated with an increased risk of ischemic stroke. Atrial fibrillation accounts for up to 24% of cerebral infarction in the elderly. Atrial fibrillation (AF) is the most important cardiac cause of embolic stroke, but other cardiac diseases, including valvular heart disease, myocardial infarction (MI), coronary artery disease (CAD), congestive heart failure (CHF), and electrocardiographic evidence of left-ventricular hypertrophy are also associated with stroke risk. Recent evidence also suggests that other atrial abnormalities, such as paroxysmal supraventricular tachycardia, may also increase risk of stroke, even in the absence of atrial fibrillation. Other possible sources of cardiac emboli include patent foramen ovale, aortic arch atherosclerotic disease, atrial septal aneurysms, and valvular strands.

Hyperlipidemia is a stroke risk factor, though its relationship to stroke is more complicated than for heart disease, primarily because of the many types of stroke. Lipid abnormalities, such as elevations in low density lipoprotein (LDL) and decreased levels of high density lipoprotein, are strongly associated with atherosclerotic stroke.

The role of alcohol as a stroke risk factor depends on stroke subtype and quantity consumed. Alcohol consumption has been shown to be a risk factor for both ICH and SAH in a linear fashion, whereas a J-shaped relationship exists between alcohol and ischemic stroke, such that modest consumption (up to two drinks daily in men, and one daily in women) is protective against stroke and heavy consumption (five or more drinks per day) increases risk.

Asymptomatic carotid artery disease, particularly with 75% or greater stenosis, is associated with increased stroke risk (approximately 2% per year). The risk of stroke also depends, however, on the rate of progression of the stenosis, collateral circulation, and the stability of the atherosclerotic plaque.

TIAs are a strong predictor of subsequent stroke. The first several days after a TIA have the greatest stroke risk, with recent series demonstrating a 5% risk at 2 days and 10% risk at 90 days. Patients with transient monocular blindness (*amaurosis fugax*) have a better outcome than those with hemispheric ischemic attacks. The stroke risk after TIA depends on the underlying cause of the ischemia, including the presence and severity of underlying atherosclerotic disease or atrial fibrillation. Age, hypertension, the presence of diabetes, clinical syndromes, including aphasia and hemiparesis, and duration of at least 10 minutes predict patients at higher risk of stroke. Patients with TIA with evidence of infarction on MRI are also at higher risk. Other potential stroke risk factors include migraine, oral contraceptive use, drug abuse, sleep apnea, infection, and inflammation.

TABLE 116-2 STROKE RISK FACTORS

NON-MODIFIABLE RISK FACTORS	Age Sex Race/ethnicity Family history Genetic disorders

WELL-ESTABLISHED MODIFIABLE RISK FACTORS	Hypertension/blood pressure
	Diabetes mellitus/hyperglycemia
	Cardiac disorders
	  Atrial fibrillation
	  Valvular heart disease
	  Recent myocardial infarction
	  Cardiomyopathy/heart failure
	  Bacterial endocarditis
	Hyperlipidemia
	Cigarette smoking
	Carotid stenosis
	TIAs
	Physical inactivity
	Hypercoagulable states (e.g., antiphospolipid antibody syndrome, cancer-associated)
	Alcohol abuse
	Substance abuse (e.g., cocaine, IV drug abuse)

OTHER POTENTIAL RISK FACTORS	Migraine
	Sleep apnea
	Cardiac disorders
	  Paroxymsal supraventricular tachycardia
	  Patent foramen ovale/atrial septal aneurysm
	  Aortic atheroma
	Infections (e.g. varicella zoster virus, influenza)
	Inflammation
	Others

PATHOLOGY

Understanding the pathology of cerebrovascular disease requires an appreciation of the vascular anatomy of the brain, the vascular pathologies that can affect brain vessels, and the response of brain tissue to ischemia and hemorrhage.