CME Geriatrics
The Brain
MSSM MOUNT SINAI SCHOOL OF MEDICINE
Series editors: Jesse Weinberger, MD, and Deborah B. Marin, MD

Stroke and TIA

Prevention and management of cerebrovascular events in primary care

Jesse Weinberger, MD

Stroke is a major cause of death and disability in the United States. Control of risk factors—particularly hypertension, diabetes, elevated serum lipids, and atrial fibrillation—can significantly reduce the incidence of stroke. Platelet antiaggregant therapy has a role in primary and secondary stroke prevention. Patients with transient ischemic attacks presenting with carotid stenosis >70% can be managed surgically, whereas those with less stenosis can be treated with platelet antiaggregant therapy. Acute stroke is a medical emergency. Thrombolytic therapy with tissue plasminogen activator within 3 hours of event onset can significantly improve outcomes in selected ischemic stroke patients. Patients with intracerebral hemorrhage usually present with acute onset of identifiable neurologic deficits.

Weinberger J. Stroke and TIA: Prevention and management of cerebrovascular events in primary care. Geriatrics 2002; 57(Jan):38-43.

Stroke is the third leading cause of death in the United States and is the leading cause of neurologic disability. In general, stroke is a disease of older persons. The incidence in persons under age 65 is less than 2 per 1,000 annually. But the incidence increases with age for men and women respectively: 4.6 and 3.8 per 1,000 for ages 65 to 74; 9.4 and 7.4 for ages 75 to 84.[1] Persons with a history of transient ischemic attacks (TIAs) and atrial fibrillation are at increased risk for stroke.

This article reviews the pharmacologic and surgical approaches for reducing the risk of stroke and managing emergent events. Included are discussions of management of atrial fibrillation and TIA. The article also explores the role of antithrombotic therapy in stroke prevention and management, and management of acute ischemic stroke—within the 3-hour treatment window—with thrombolytic therapy. A brief section on hemmorrhagic stroke includes a discussion of subarachnoid hemorrhage. A table summarizes the management considerations and interventions for patients at high risk for stroke; subsequent tables summarize antiaggregant treatment for stroke prevention and management of carotid stenosis.

Dr. Weinberger is professor, department of neurology, The Mount Sinai School of Medicine, New York, and director, division of neurology, North General Hospital, Manhattan, New York. Dr. Weinberger has no real or apparent conflicts of interest related to the content presented here.

Etiology

Ischemic strokes, caused by occlusion of an artery, account for 80 to 85% of cerebrovascular events, whereas hemorrhagic strokes, caused by a ruptured artery, account for 15 to 20%.[2]

There are three main causes of ischemic stroke:

- atherosclerotic disease of large extracranial and intracranial vessels
- occlusion of intracranial vessels by emboli from a cardiac source (cardioembolic stroke)
- and small vessel intracranial occlusive disease resulting from hypertension and diabetes.

To help prevent stroke occurrence, the most important contribution the internist or family practitioner can make is to help older patients modify controllable risk factors, particularly hypertension, diabetes, and serum lipids. The practitioner also should recognize when a patient is experiencing warning signs of impending stroke or TIA and refer patients to a neurologist for appropriate management.

Patients should understand that stroke requires emergency medical treatment within 3 hours of onset for

best outcomes. Patients who suspect they are having a stroke should call 911 and proceed directly to a hospital emergency department, preferably where they can be met by their primary care physician. Within the 3-hour window, a neurologist (preferably a stroke specialist) should be consulted immediately to help determine whether thrombolytic therapy is appropriate.

Patient assessment

All older patients should be evaluated for stroke risk factors (table 1). A careful history and physical examination should be performed. In addition to hypertension, diabetes, and smoking, other significant risk factors for stroke include atrial fibrillation, carotid artery stenosis, and history of TIA. Patients should be asked if they have ever experienced warning signs of stroke, such as transient weakness of the right face, arm and leg; numbness in the extremities; slurred speech; or unilateral vision loss.

Assessment also should include:

- regular blood pressure checks at the time of routine physical examination
- Auscultation of the neck and chest to check for carotid artery bruit and heart murmur (suggestive of valvular heart disease)
- electrocardiography, which can identify cardiac arrhythmias such as atrial fibrillation (a cause of cardioembolic stroke)
- serum lipid profiling
- questions about smoking history.

Atrial fibrillation and anticoagulation

Cardiogenic embolization, particularly when associated with nonvalvular atrial fibrillation, is a significant stroke risk factor in older persons. Risk of stroke from untreated nonvalvular atrial fibrillation is 5% per year in patients over age 70.[3]

Anticoagulation with warfarin (Coumadin) in these patients can reduce stroke risk by 70%, whereas platelet antiaggregant therapy using aspirin reduces the risk by 20%.[4] Patients under age 70 with atrial fibrillation but no other associated heart disease can get effective stroke prophylaxis with aspirin, 325 mg/d. Stroke prophylaxis guidelines for patients over age 70 who have nonvalvular atrial fibrillation recommend warfarin administration that achieves a target INR of 2.5 (or within the range of 2.0 to 2.9).[4]

Patients over age 85 are at greater risk for complications of cerebral hemorrhage when anticoagulated with warfarin, therefore their target INR should be kept close to 2.0. In patients with atrial fibrillation and mitral stenosis and those with mechanical cardiac valve replacement, anticoagulation should achieve a target INR range of 3.0 to 3.5.[5]

Anticoagulation with warfarin is beneficial as stroke prophylaxis during the first 3 months after an MI; it is particularly useful in patients with large anterior wall infarction and akinetic wall segments.[6] (For the study cited, age was not an independent risk factor.)

Carotid artery bruit. Patients with asymptomatic carotid artery stenosis can be identified by auscultation of a carotid artery bruit near the angle of the jaw. Whether management of patients with asymptomatic carotid artery stenosis should consist of surgical or medical therapy requires further study. Table 2 summarizes the management of patients with carotid stenosis. In a controlled trial involving asymptomatic patients with >60%

Table 1 Ischemic stroke: Checklist for patient management

Review risk factors
- Hypertension
- Diabetes
- Poor diet; no exercise
- High serum cholesterol
- Smoking
- Atrial fibrillation
- Carotid stenosis (TIA history)

Patient assessment
- History and physical exam
- Is patient high-risk? (eg, has one or more of the risk factors above)
- If high-risk, check for TIA history or TIA risk (auscultation, Doppler studies)
- Rule out atrial fibrillation
- Primary prevention
- Patient education regarding stroke symptoms, signs

Risk-factor management
- Maintain normotension
- Tight glycemic control
- Cholesterol lowering
- Diet modification; exercise
- Antithrombotic therapy (platelet antiaggregant therapy)

Primary/secondary prevention
- Pharmacologic management of cardiac risk factors (eg, atrial fibrillation, post MI)
- Antithrombotic therapy (platelet antiaggregant therapy, anticoagulation)

Surgical intervention*
- Endarterectomy
- Stent placement

* Consider for select patients

Source: Prepared for Geriatrics by Jesse Weinberger, MD

Table 2 Stroke prevention in patients with carotid stenosis

Patient	Degree of stenosis	Treatment
Asymptomatic (identified by carotid artery bruit)[†‡]	<60%	Antiaggregant therapy*
	>60%	Consider endarterectomy
History of TIA[†§]	0 to 40%	Antiaggregant therapy*
	50 to 69%	Antiaggregant therapy*; endarterectomy in selected cases
	70 to 99%	Endarterectomy (or stenting in high-risk patients), unless significant contraindications exist

TIA: Transient ischemic attack
* See table 3
† Stenosis as measured by duplex Doppler sonograph
‡ For patients who meet criteria of Asymptomatic Carotic Atherosclerosis Study (ACAS)
§ For patients who meet criteria of North American Symptomatic Carotid Endarterectomy Trail (NASCET)

Source: Prepared with data obtained from references 7 and 8.

stenosis of the carotid, the aggregate risk over 5 years for ipsilateral stroke and any perioperative stroke or death was estimated to be 5.1% for patients who underwent carotid endarterectomy and 11% for patients treated with daily aspirin and medical risk factor management.[7]

Patients in whom asymptomatic cervical bruit is detected can be screened noninvasively with duplex Doppler ultrasonography to assess the degree of carotid stenosis. Patients with severe stenosis (>60%) can be considered for surgical management if there are no significant medical risks to surgery, such as severe coronary artery disease or congestive heart failure. Patients who have moderate stenosis (<60%) or are not surgical candidates should receive platelet antiaggregant therapy with aspirin, ticlopidine (Ticlid), clopidogrel (Plavix), or combination aspirin/dipyridamole (Aggrenox). Diet or statin therapy is recommended for these patients if cholesterol levels exceed 230 mg/dL. Table 3 summarizes medical management with antiaggregant therapy.

Management of TIAs

Patients who experience TIAs and the associated transient symptoms—difficulty with speech, sudden weakness or numbness of the extremities, loss of balance, visual disturbance—are at increased risk for stroke. Without stroke prophylaxis, these patients have a 25 to 50% chance of experiencing a subsequent stroke that causes permanent neurologic deficits.[2] Stroke is most likely to occur in the first 3 months after a TIA.[8]

Aspirin. The original study demonstrating a beneficial effect of aspirin in prevention of stroke for patients with TIA found that a dosage of 650 mg bid reduced the stroke rate by 50%. However, there was no significant reduction in the stroke rate for women in this trial.[9] This prompted several subsequent trials that investigated lower dosages of aspirin (50 to 325 mg/d); results from these showed beneficial reduction of stroke in men and women,[10] although to a lesser extent than the original study. A comparison of individual aspirin trials showed that in patients with TIA, a higher dose of aspirin (at least 975 mg/d) is more effective in preventing stroke than a lower dose.[11]

Aspirin / dipyridamole. The European Stroke Prevention Study underscored the benefits of combination anti-platelet therapy.[3] In this trial, the combination antiplatelet agent aspirin/ dipyridamole, 400 mg/d, yielded the same efficacy in reduction of stroke as 50 mg/d aspirin; in previous trials, aspirin/dipyridamole alone showed no beneficial effect in preventing stroke in TIA patients. Compared with placebo, aspirin/dipyridamole reduced the rate of stroke by 37% in patients with a history of stroke or TIA. The efficacy of aspirin/dipyridamole has not been compared with a higher dosage of aspirin.

Ticlopidine. The antiplatelet agent ticlopidine was developed for patients who cannot tolerate the GI side effects associated with aspirin. Clinical data showed that compared with aspirin, 1,300 mg/d, ticlopidine, 500 mg/d, provided a 12% risk reduction in fatal and nonfatal stroke in patients with a history of TIA.[12] Ticlopidine use, however, was associated with a significantly higher risk of complications (eg, diarrhea, peptic ulceration, gastritis, and GI bleeding) than aspirin. The authors concluded that ticlopidine was somewhat more effective than aspirin in preventing strokes in this population, but that the risks of side effects were greater.

Clopidogrel. The CAPRIE trial showed that long-term administration of clopidogrel, 75 mg/d, a platelet aggregation inhibitor similar to ticlopidine, is more effective than aspirin in reducing the combined risk of ischemic stroke, MI, and vascular death in patients with atherosclerosis.[13] Compared with clopidogrel alone, aspirin, 75 to 325 mg/d, and clopidogrel, 75 mg/d produced a further reduction of 20% in vascular events in patients with acute coronary syndromes.

Clopidogrel, 75 mg/d, is now preferred over ticlopidine for treatment of patients with TIA who cannot tolerate aspirin or who have recurrent vascular events while on aspirin therapy. Ticlopidine, 250 mg bid, is still an effective option, but biweekly laboratory testing must be used for the first 3 months of administration to rule out hematologic and hepatic side effects. Ticlopidine administration is associated with neutropenia (2% of pa-

tients), hepatitis (0.4%), and thrombocytopenic purpura (1 case per 1,600 to 5,000).

Patients in whom carotid artery stenosis is not the cause of TIA should undergo transesophageal echocardiography to rule out a source of emboli from fibrin clots in the heart. Patients with TIA who have patent foramen ovale, atrial septal aneurysm, or dilated cardiomyopathy with a ventricular clot must be treated with warfarin rather than with platelet antiaggregant therapy. Platelet antiaggregant therapy is ineffective in preventing further fibrin clots from forming in the heart, whereas warfarin prevents cardioembolic stroke in these patients.[2] When a cerebral ischemic event is not caused by a cardioembolic source, warfarin confers no beneficial effect in prevention of stroke over aspirin.[14]

Carotid stenosis. Approximately one-half of patients with TIA exhibit >50% stenosis at the bifurcation of the cervical carotid artery. The definitive test for measuring the degree of carotid stenosis is catheter angiography, but this procedure is associated with a 1% incidence of stroke or death in patients with atherosclerotic or cerebrovascular disease.

Imaging. Noninvasive imaging of the carotid bifurcation can be performed with duplex Doppler ultrasonography, magnetic resonance angiography (MRA), or CT angiography. Compared with catheter angiography, which is considered the definitive method for identifying the degree of carotid stenosis, duplex Doppler ultrasonography and MRA have a 90% accuracy rate. Combining the two imaging approaches achieves accuracy virtually on par with catheter angiography.

Patients with TIA are usually initially examined with duplex Doppler ultrasonography. If results show stenosis >70% or if the examiner does not consider the results to be definitive, MRA is performed to confirm the duplex Doppler ultrasonography findings.[15]

MRA cannot be performed on patients with pacemakers or who are claustrophobic; these patients should undergo CT angiography. Although this procedure also yields 90% accuracy, the process requires injection of a large volume of contrast dye, so it may be contraindicated in patients with congestive heart failure or renal disease.

Table 3 Antiaggregant therapy for prevention of stroke*

Agent	Dosage
Aspirin	50 mg/d to 650 mg bid
Ticlopidine (Ticlid)	200 mg bid
Clopidogrel (Plavix)	75 mg/d
Aspirin/dipyridamole (Aggrenox)	25/100 mg bid

*For patients at risk of stroke and not scheduled for surgical intervention

Source: Prepared with data obtained from references 3, 9, 10, 12, 13.

Surgical intervention

Endarterectomy. Patients with carotid stenosis >70% may benefit from carotid endarterectomy. At centers where the complication rate for endarterectomy is <2%, the intervention can reduce the risk of subsequent stroke from 25 to 5% in patients on aspirin therapy.[16]

Patients exhibiting <50% carotid stenosis do not benefit from carotid endarterectomy and instead should be on medical therapy with aspirin, clopidogrel, or aspirin/dipyridamole. Some patients exhibiting carotid stenosis between 50 to 70% may benefit from endarterectomy rather than medical therapy; they should be referred to a stroke neurology consultant.

Stent placement. For older patients who are not endarterectomy candidates, carotid artery stent placement is an option. The risk of stroke during the procedure is similar to that of carotid endarterectomy (2 to 8% depending on the center), but the systemic complications are reduced.[17]

Management of acute ischemic stroke

An acute ischemic stroke is a medical emergency, and patients need immediate attention. When possible, the primary care physician should collaborate with the stroke team on emergency intervention. Acute medical care includes stabilizing a patient's cardiopulmonary status and determining whether the stroke was accompanied by an acute MI or respiratory distress.

Blood pressure should be carefully monitored. If the patient is hypotensive, raising the blood pressure may improve the condition. In general, hypertension associated with acute stroke is not treated so that perfusion to the ischemic area will not be reduced unless the mean arterial pressure is >140 mm Hg. Even in this scenario, blood pressure is reduced gradually, usually with low doses of IV labetalol HCl (Normodyne). Acute reduction of the blood pressure can worsen the neurologic symptoms.[18]

Patient assessment. CT should be among the first tests performed on patients suspected of having a stroke. Results will rule out a hemorrhagic event or reveal early signs of permanent infarction, which can increase the risk of hemorrhagic complications.

The stroke neurologist should use the NIH Stroke Scale to quantify the severity of the event. Patients in good condition (a score <5) will not benefit from thrombolytic therapy; patients with severe strokes (a score of >25) are at high risk for hemorrhagic complications and should not receive thrombolytic therapy. Thrombolytic therapy is used only in patients with no hemorrhage or signs of early infarction on the initial CT performed within 3 hours of the event.

tPA. Thrombolytic therapy with tissue plasminogen activator (tPA) should be administered to patients with acute ischemic stroke who are medically stable and can be treated within 3 hours of stroke onset. Throm-

bolytic intervention improves the patient's likelihood of returning to excellent status 3 months after the event (21% for untreated patients, 31% for treated patients).[19] Moreover, age is not a limiting factor for administration of tPA. The risk of death from tPA-induced hemorrhage is 3%, but the intervention has not been shown to increase overall mortality. Hemorrhagic complications were higher in patients over age 85, but the rate was not statistically significant. [19]

Tissue plasminogen activator must be used judiciously and precisely to avoid hemorrhagic complications and death. This requires consultation with the stroke neurologist. Tissue plasminogen activator can be administered if:

- prothrombin time, partial thromboplastin time, and platelet count are not elevated
- the patient has no history of recent surgery or any other illness that could result in significant systemic bleeding.

If all criteria are met, IV tPA is administered at a rate of 0.9 mg/kg over 1 hour with 10% given as an initial bolus. Thrombolytic therapy can also be administered intra-arterially, increasing the therapeutic window to 6 hours, although not without the risk of hemorrhagic complications.

Patients who arrive after the 3-hour window should be evaluated to determine the etiology of the ischemic stroke. Patients who have experienced a stroke and have known cardioembolic sources—particularly atrial fibrillation—are at risk for recurrent stroke within 7 days of the initial event and should undergo prophylactic anticoagulation.

Anticoagulation

To avoid hemorrhage into the area of infarction, anticoagulation is not initiated until 48 hours after stroke onset. Initial treatment consists of administration of heparin with a target PTT of 50 to 70 (a bolus is not given). Alternatively, low-molecular weight heparin (enoxaparin [Lovenox]), 1 mg/kg, can be administered subcutaneously. Parenteral anticoagulation with oral warfarin therapy is maintained until a therapeutic INR of 2.0 to 3.0 has been achieved. In patients with massive infarction and edema, anticoagulation may be deferred until the edema resolves.[18]

Anticoagulation is effective in only a select group of ischemic stroke patients and should be used with care in older persons. Anticoagulation is necessary to prevent recurrent cardioembolic stroke from intracardiac fibrin thrombus due to atrial fibrillation, patent foramen ovale, dilated cardiomyopathy, or valvular heart disease. Anticoagulation may also provide some benefit for patients with large vessel occlusive disease (eg, carotid or basilar artery occlusion) who are having progressive symptoms.[20]

Ischemic stroke patients not undergoing anticoagulation should receive acute platelet antiaggregant therapy with aspirin, which improves outcomes by 1%.[21] Given that 500,000 strokes occur annually in the United States, this is not insignificant improvement. Combining a bolus of clopidogrel, 75 mg, with aspirin, 81 or 325 mg, is 20% more effective than clopidogrel alone in preventing ischemic events in patients with unstable angina and may also be more beneficial in improving outcomes in stroke patients.[22]

Management of hemorrhagic stroke

Patients with intracerebral hemorrhage usually present with acute onset of neurologic deficits including:

- weakness of one side of the body associated with severe headache
- progressive depression of the level of consciousness.

Cerebral hemorrhage in the deep structures of the brain—caused by hypertension—is more common in younger patients; lobar hemorrhage in the cortex—caused by amyloid angiopathy—is more common in older adults.

Swift achievement of normotension (and avoidance of hypotension) should be the goal for patients who present with elevated blood pressure. If CT results reveal increased intracranial pressure or massive shift of brain structures, the patient should be treated with IV mannitol, 100 grams, administered over 1 hour. If the patient needs ventilatory assistance, mechanical hyperventilation should be used.

Steroid medications have not been proven to be effective in intracerebral hemorrhage. Surgical removal of intracerebral hematomas has not been beneficial except in the case of cerebellar hemorrhage.

Patients presenting with acute vertigo, headache, and imbalance with progressive lethargy should be evaluated immediately with CT to rule out cerebellar hemorrhage. In patients with lobar hemorrhage, brain MRI should be performed to rule out an underlying tumor, arteriovenous malformation, or cavernous angioma.[18]

Subarachnoid hemorrhage (SAH) occurs when there is rupture of a saccular aneurysm at branch points in the circle of Willis or as result of arterio-venous malformations. Patients present with severe headache and varying degrees of altered mental status or focal neurologic deficit. CT of the brain is performed to document the hemorrhage. If SAH is strongly suspected, lumbar puncture is performed if CT results are negative. Patients are treated with nimodipine (Nimotop), 60 mg, every 4 hours orally or by feeding tube.[23]

Patients are graded by the Hunt-Hess scale to determine their clinical condition. Patients with headache alone who are awake and alert are considered grade I. Patients with mild drowsiness or confusion or minimal focal deficits such as hemiparesis are grade II. Grade I and II patients can be considered for neurosurgical management to clip the aneurysm and prevent rebleeding. Grade III to V patients have increasing degrees of alteration of state of consciousness or focal deficit. These patients have poor outcomes with surgical management, and surgery is usually deferred unless their clinical condition improves.

Cerebral angiography should be performed to document the source of the bleeding. If an aneurysm is identified, surgical therapy within 24 hours after onset provides the most effective prevention of rebleeding without encountering ischemic consequences of vasospasm. After 24 hours, the risk for complications associated with surgery increases, and it is usually better to manage the patient conservatively for 14 days prior to surgical clipping of the aneurysm.[24]

Conclusion

Modification of controllable risk factors plays a significant role in reducing the risk of stroke. Physicians should work closely with patients to help precipitate behavioral and lifestyle changes and to encourage the use of appropriate pharmacologic therapies. Antithrombotic therapy should be used to reduce stroke risk in patients with atrial fibrillation and history of TIA. Patients with a history of TIA should undergo assessment for carotid stenosis and be considered for platelet antiaggregant and surgical therapy.

Swift intervention for acute ischemic stroke significantly improves post-stroke prognosis. Thrombolytic therapy with tPA must occur within 3 hours of event onset, although intra-arterial administration can increase the intervention window to 6 hours. Patients who have experienced a stroke and have known cardioembolic sources must be carefully managed to prevent recurrent stroke. Patient education also should be included in stroke prevention efforts.

References

1. Weinberger J. Cerebrovascular diseases in the elderly patient. In: Tresch DD, Aronow WS. Cardiovascular disease in the older patient. New York: Marcel Dekker, Inc. (in press).
2. Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P. Antithrombotic and thrombolytic therapy for ischemic stroke. Chest 2001; 119 (1 suppl): 300S-320S.
3. Diener HC, Cunha L, Forbes C, Sivenius J, Smets P, Lowenthal A. European Stroke Prevention Study. 2. Dipyridamole and acetylsalicylic acid in the secondary prevention of stroke. J Neurol Sci 1996; 143(1-2):1-13.
4. Warfarin versus aspirin for prevention of thromboembolism in atrial fibrillation: Stroke Prevention in Atrial Fibrillation II Study. Lancet 1994; 343(8899):687-91.
5. Sherman DG, Dyken ML Jr, Gent M, Harrison JG, Hart RG, Mohr JP. Antithrombotic therapy for cerebrovascular disorders. An update. Chest 1995; 108(4 suppl):444S-456S.
6. Smith P, Arnesen H, Holme I. The effect of warfarin on mortality and reinfarction after myocardial infarction. N Engl J Med 1990; 323(3):147-52.
7. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. JAMA 1995; 273(18):1421-8.
8. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1991; 325(7):445-53.
9. A randomized trial of aspirin and sulfinpyrazone in threatened stroke. The Canadian Cooperative Study Group. N Engl J Med 1978; 299(2):53-9.
10. Antiplatelet trialists' collaboration. Collaborative overview of randomized trials of antiplatelet therapy, 1. Prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of patients. BMJ 1994; 308:81-106.
11. Dyken ML, Barnett HJ, Easton JD, et al. Low-dose aspirin and stroke. "It ain't necessarily so." Stroke 1992; 23(10): 1395-9.
12. Hass WK, Easton JD, Adams HP Jr, et al. A randomized trial comparing ticlopidine hydrochloride with aspirin for the prevention of stroke in high-risk patients. Ticlopidine Aspirin Stroke Study Group. N Engl J Med 1989; 321(8):501-7.
13. A randomised, blinded trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. Lancet 1996; 348(9038):1329-39.
14. Mohr JP, Thompson JLP, Lazar RM, et al. A comparison of warfarin and aspirin for the prevention of recurrent ischemic stroke. Warfarin–Aspirin Recurrent Stroke Study Group. N Engl J Med 2001; 345:1444-51.
15. Weinberger J, Tegeler CH, McKinney WM, Wechsler LR, Toole J. Ultrasonography for diagnosis and management of carotid artery atherosclerosis. A position paper of the American Society of Neuroimaging. J Neuroimaging 1995; 5(4):237-43.
16. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1998; 339(20):1415-25.
17. Qureshi AI, Luft AR, Janardhan V, et al. Identification of patients at risk for periprocedural neurological deficits associated with carotid angioplasty and stenting. Stroke 2000; 31(2):376-82.
18. Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. N Engl J Med 2001; 344(19):1450-60.
19. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med 1995; 333(24):1581-7.
20. Low molecular weight heparinoid, ORG 10172 (danaparoid), and outcome after acute ischemic stroke: A randomized controlled trial. The Publications Committee for the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. JAMA 1998; 279(16):1265-72.
21. Chen ZM, Sandercock P, Pan HC, et al. Indications for early aspirin use in acute ischemic stroke: A combined analysis of 40,000 randomized patients from the Chinese Acute Stroke Trial and the International Stroke Trial. On behalf of the CAST and IST collaborative groups. Stroke 2000; 31(6):1240-9.
22. Yusuf S, Zhao F, Mehta SR, Chrolavicius S, Tognoni G, Fox KK. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. N Engl J Med 2001; 345(7):494-502.
23. Perry HM Jr, Davis BR, Price TR, et al. Effect of treating isolated systolic hypertension on the risk of developing various types and subtypes of stroke: The Systolic Hypertension in the Elderly Program (SHEP). JAMA 2000; 284(4):465-71.
24. Kassell NF, Torner JC, Jane JA, Haley EC Jr, Adams HP. The International Cooperative Study on the Timing of Aneurysm Surgery. Part 2: Surgical results. J Neurosurg 1990; 73(1):37-47.

Turn to page 44 to take the exam

CME Geriatrics Exam

Detach or photocopy this page, place an X in the boxes that correspond to your answers, fill in your name and address, and mail (see address below). Answers must be received by July 1, 2002. A score of at least 80% must be earned to receive CME credit.

Make check for $15 payable to **The Page and William Black Post-Graduate School** and mail it with this exam to Rae Ann Houghton, Geriatrics, 7500 Old Oak Blvd., Cleveland, Ohio 44130. When submitting more than one exam, attach a separate check for $15 to each exam. Documentation of earned credit and the correct answers will be mailed to you. Allow up to 12 weeks for notification.

Accreditation. This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the sponsorship of Mount Sinai School of Medicine. Mount Sinai School of Medicine is accredited by ACCME to provide continuing medical education for physicians. Mount Sinai School of Medicine designates this continuing medical education activity for a maximum of 1 credit in category 1 toward the AMA Physician's Recognition Award. Each physician should claim only those hours that he/she spent in the educational activity.

Faculty Disclosure. It is the policy of Mount Sinai School of Medicine to ensure fair balance, independence, objectivity, and scientific rigor in all its sponsored programs. All faculty participating in sponsored programs are expected to disclose to the audience any real or apparent conflict-of-interest related to the content of their presentation, and any discussions of unlabeled or investigational use of any commercial product or device not yet approved in the United States.

Weinberger J. Stroke and TIA: Prevention and management of cerebrovascular events in primary care. Geriatrics 2002; 57(Jan):38-43.

1. Approximately one-half of patients with TIA exhibit what percentage of stenosis at the bifurcation of the cervical carotid artery.
   - ☐ a. <50
   - ☐ b. >50
   - ☐ c. <40
   - ☐ d. >40
2. Patients with carotid stenosis of what percentage may benefit from carotid endarterectomy.
   - ☐ a. >40
   - ☐ b. >50
   - ☐ c. >60
   - ☐ d. >70
3. Medical therapy for stroke prevention involves which of the following:
   - ☐ a. ticlopidine
   - ☐ b. clopidogrel
   - ☐ c. aspirin
   - ☐ d. all of the above
4. Patients in whom carotid artery stenosis is not the cause of TIA should undergo transesophageal echocardiography to rule out a source of:
   - ☐ a. emboli from fibrin clots in the heart
   - ☐ b. emboli at angle of the jaw
   - ☐ c. emboli from fibrin clots in the lungs
   - ☐ d. none of the above
5. Cardiogenic embolization, particularly when associated with nonvalvular atrial fibrillation, is a significant stroke risk factor in older persons
   - ☐ a. True ☐ b. False
6. Patients under age 70 with atrial fibrillation but no other associated heart disease can get effective stroke prophylaxis with daily intake of:
   - ☐ a. warfarin
   - ☐ b. aspirin
   - ☐ c. vitamins
   - ☐ d. tissue plasminogen activator (tPA)
7. Thrombolytic therapy with tPA should be administered to patients with acute ischemic stroke who are medically stable and can be treated within how many hours of stroke onset:
   - ☐ a. 6
   - ☐ b. 5
   - ☐ c. 4
   - ☐ d. 3
8. To avoid hemorrhage into the area of infarction, anticoagulation is not initiated until how many hours after ischemic stroke onset:
   - ☐ a. 48
   - ☐ b. 36
   - ☐ c. 24
   - ☐ d. 12

In addition to the exam questions, answer the following evaulation questions: (1=strongly agree, 6=strongly disagree)

1. The information presented in this article was useful.
   1 2 3 4 5 6
2. The information presented was fair, objective, and balanced.
   1 2 3 4 5 6

Your name: _____ Degree _____

Address (Street): _____

(City)_____ (State) _____ (Zip)_____

Phone (include area code): _____

Specialty: GP____ FP____ IM____ DO____ Other (specify) _____

Date:_____ Signature:_____

Remove this answer sheet and mail