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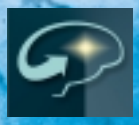

Stroke Best Practices: A Team Approach to Evidence-Based Care

Mark J. Alberts, MD, and J. Donald Easton, MD

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Note: This supplement focuses on diagnosis and management of acute stroke and prevention of secondary vascular events. It does not discuss rehabilitation of patients with recent stroke.

Stroke Best Practices: A Team Approach to Evidence-Based Care

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Reducing the high morbidity and mortality associated with stroke continues to be a major healthcare challenge in the United States. Recent advances in the management and prevention of atherothrombotic events are significant; however, the clinical application of evidence-based "best practices" is lagging in many hospitals across the country. The "Stroke Best Practices" program was designed to assist institutions that lack established stroke centers integrate recent evidence-based recommendations into individualized, in-hospital initiatives to optimize the management of acute stroke, prevent secondary vascular events, and enhance the quality of care for patients with established cerebrovascular disease. Programs that bridge the gap between evidence-based medicine and clinical practice can improve patient outcomes. (*J Natl Med Assoc.* 2004;96:S1–S24.)

Key words: stroke ◆ prevention ◆ management ◆ best practices ◆ hospitals

Each year, approximately 700,000 strokes and 500,000 to one million transient ischemic attacks (TIAs) occur in the United States.[1] The total direct and indirect costs of stroke in 2004 are estimated at $53.6 billion.[1] Of the approximately 4.7 million stroke survivors, 90% suffer from long-term functional limitation and restriction of daily activities.[1] Besides motor or sensory deficits, stroke victims may experience cognitive decline, dementia, and depression.

While recent advances in the management and prevention of atherothrombotic events are significant, the clinical application of evidence-based "best practices" is lagging in many hospitals across the country, including those in the so-called "stroke belt"—a region

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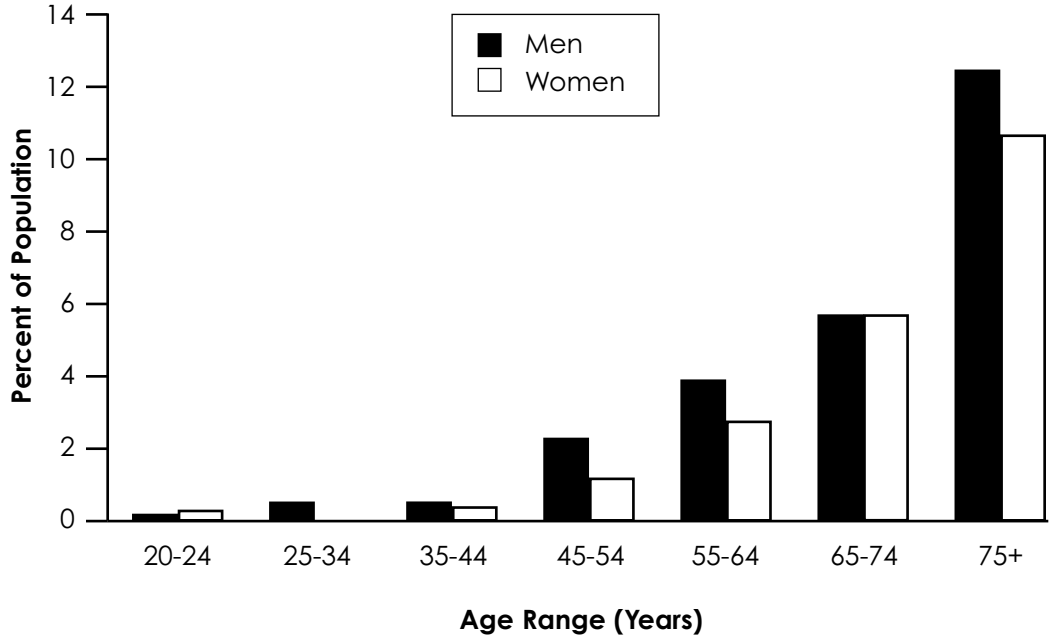
comprising 11 southeastern states with stroke death rates more than 10% higher than the national average.[2]

Programs that implement widely accepted guidelines drawn from evidence-based medicine contribute to the prevention and treatment of cardiac and vascular diseases.[3-9] "Stroke Best Practices: A Team Approach to Evidence-Based Care," a program developed by the Network for Continuing Medical Education (NCME), was designed to help hospitals that do not have established stroke centers integrate recent evidence-based recommendations into customized, in-hospital initiatives for optimizing the management of acute stroke, preventing secondary events, and enhancing the quality of care for patients with established cerebrovascular disease. The program comprised a series of live workshops, held in various regions of the country, and offered continuing medical education credit for physicians and continuing education credit for nurses. Following is a summary of the core content from the program, which includes current prevention and management strategies for acute ischemic stroke.

Risk Factors for Stroke

Nonmodifiable risk factors for stroke include age, gender, race/ethnicity, and heredity.[10] Stroke prevalence rises progressively with age; 12.5% of men and 10.7% of women aged 75 or

Figure 1. Prevalence of Stroke by Age



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older have experienced at least one stroke (Figure 1).[1] Annually, approximately 40,000 more women than men have a stroke.[1] In 2001, the incidence of new and recurrent attacks was 373,000 for women compared with 327,000 for men.[1] More women than men die of stroke each year because women live longer than men. In 2001, women accounted for 61.4% of U.S. stroke deaths.[1] Non-Hispanic black females, the most susceptible population, have more than twofold the prevalence of stroke of white females (3.2% versus 1.5%). Blacks, in general, have almost twice the risk of whites of a first-ever stroke[1] and are more than twice as likely as whites to die of a stroke.[11]

Modifiable risk factors for stroke are divided into lifestyle factors, such as cigarette smoking, excessive alcohol intake, physical inactivity, obesity (body mass index >30 kg/m^2), oral contraceptive use, and drug abuse, and medically or surgically treatable conditions, such as hypertension, dyslipidemia, ischemic or hypertrophic heart disease, atrial fibrillation, diabetes (types 1 and 2), carotid artery disease, and hematologic disorders (e.g., elevated red-blood-cell count, sickle cell anemia).[10,12] Stroke is highly preventable, as suggested by the analysis in Table 1 of the effects of intervention and lifestyle modification.[13]

Risk Stratification for Acute Ischemic Stroke

About 80% of strokes are ischemic (atherothrombotic or embolic), 13% are caused by intracerebral hemorrhage (ICH), and 7% result from subarachnoid hemorrhage.[14] Rapid differentiation between ischemic and hemorrhagic stroke is imperative because the therapies are markedly different.[15]

Noncontrast-enhanced computed tomography (CT) of the brain parenchyma is the imaging modality of choice.[15] Atherothrombotic and embolic strokes (cardioembolic, artery-artery, or cryptogenic) appear on the scans—often after several hours—as areas of hypodensity.[16] Intraparenchymal and subarachnoid hemorrhagic strokes are seen as areas of hyperdensity.

Table 1. Modifiable Risk Factors and Preventable Strokes

	Percent Exposed	Relative Risk	Population-Attributable Risk (Percent)	Projected Strokes Prevented*
Hypertension	56	2.7	49	360,000
Smoking	27	1.5	12	90,000
Atrial fibrillation	4	3.6	9.4	69,000
Heavy alcohol consumption	7	1.7	4.7	34,000

*Based on 731,000 strokes.

Adapted with permission from Gorelick PB. Stroke prevention. An opportunity for efficient utilization of health care resources during the coming decade. *Stroke*. 1994;25:220-224.

Magnetic resonance imaging (MRI) of the brain may be considered for initial diagnostic imaging at qualified centers; however, MRI is limited by the potential difficulty in reliably identifying acute intracranial hemorrhage.[15] Other potential limitations of MRI in the acute setting include cost, lack of availability, and patient contraindications (e.g., claustrophobia, cardiac pacemakers, metal implants).[15] However, diffusion-weighted MRI is exquisitely sensitive to early ischemic changes, even if small or in the brain stem.[17,18]

Other Tests

All suspected stroke patients should be examined clinically to detect head trauma and other possible causes of symptoms. Blood pressure measurement is important because blood pressure may be acutely elevated in stroke patients, even in those without a prior history of stroke. The patient's history may be revealing. Embolic strokes generally produce abrupt neurologic dysfunction that is maximal from the onset, while atherothrombotic strokes have a more gradual or intermittent onset.[16] A 12-lead electrocardiogram (ECG) is mandatory because acute myocardial infarction (MI) and atrial fibrillation produce embolic stroke; mitral stenosis and prosthetic valves are among the other potential sources of emboli.[15,19] Carotid Doppler ultrasonography, magnetic resonance angiography, CT angiography, and cardiac echocardiography can add to the diagnostic information.[15] Test selection should be tailored to the individual patient and the clinical setting.[15] Routine blood

Table 2. Diagnostic Testing for Suspected Acute Ischemic Stroke

Immediate Testing	*Routine Blood Tests*
Brain CT	Blood glucose
Brain MRI (may be considered at qualified centers)	Electrolytes
Electrocardiogram	Complete blood count with platelets
	Prothrombin time
Ancillary Imaging	Activated partial thromboplastin time
Cardiac echocardiography (either TTE or TEE)	Renal and hepatic function
Carotid Doppler ultrasonography	*Additional Tests*
Magnetic resonance angiography	Lipid profile
CT angiography	Homocysteine
Chest x-ray	Hemoglobin A_{1c}
	hs-CRP
	ESR
	Urinalysis

CT = computed tomography; MRI = magnetic resonance imaging; TTE = transthoracic echocardiography; TEE = transesophageal echocardiography; hs-CRP = high-sensitivity C-reactive protein; ESR = erythrocyte sedimentation rate

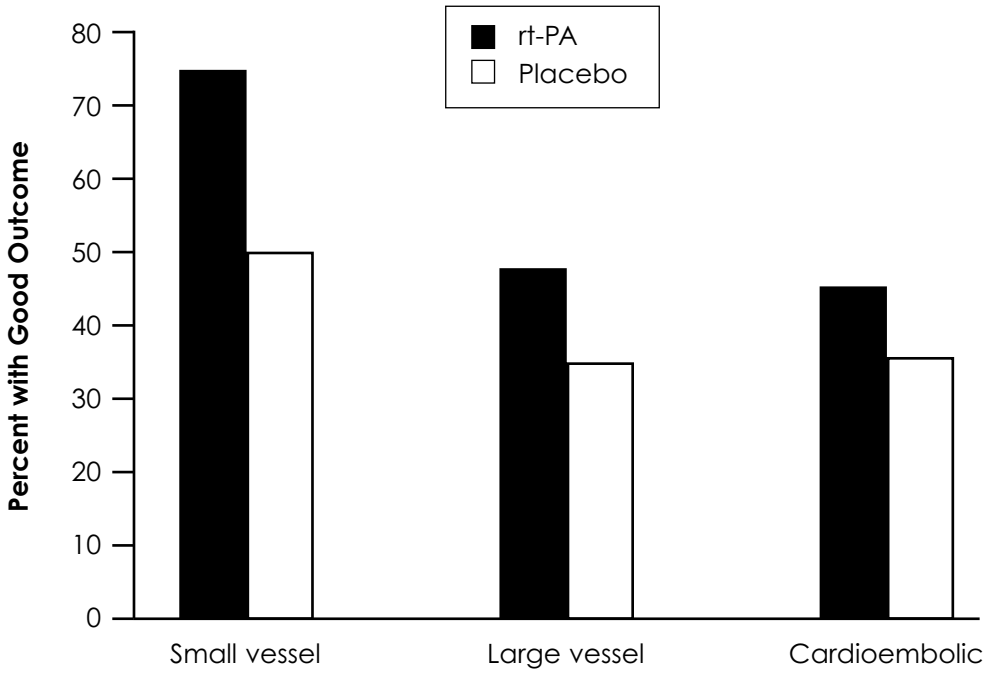
Adapted with permission from Adams Jr HP, Adams RJ, Brott T, et al. Guidelines for the early management of patients with ischemic stroke. A scientific statement from the Stroke Council of the American Stroke Association. *Stroke*. 2003;34:1056-1083.

tests should always be performed to exclude systemic conditions. The key measurements are glucose, electrolytes, complete blood count with platelets, prothrombin time, activated partial thromboplastin time (aPTT), and renal and hepatic function. Measurement of fasting lipids, homocysteine, hemoglobin A_{1c}, high-sensitivity C-reactive protein, and erythrocyte sedimentation rate also is important. Diagnostic studies obtained in patients with suspected acute ischemic stroke are listed in Table 2.

Acute Care of Stroke Patients

Patients with acute stroke or TIAs (if multiple or occurring within the past 48 hours) should be admitted to a stroke unit.[20] Strokes occur in 10% of patients in the first 90 days after a TIA, with half occurring within 48 hours of the TIA.[21] An ideal facility is staffed by a multidisciplinary team that includes a neurologist available 24 hours a day. It should be equipped with stroke care maps, telemetry monitoring, and well-trained nurses. Precautions should be taken to avoid aspiration pneumonia, deep vein thrombosis (DVT), and infection. Prompt access to neurosurgery should also be provided. A meta-analysis of 23 trials with approximately 4,900 participants in the United Kingdom, Sweden, and the United States compared care in a stroke ward with that given by a mobile team or a generic disability service and found a

Figure 2. Efficacy of rt-PA by Stroke Subtype



NINDS rt-PA Stroke Study Group. *N Engl J Med.* 1995;333:1581-1587.

lower risk of death within one year, death or institutionalization, and death or dependency in a stroke ward (unit).[22]

Thrombolytic Therapy for Acute Ischemic Stroke

Because most strokes are caused by thrombotic or embolic occlusion, prompt revascularization is a rational therapeutic strategy.[15] The National Institute of Neurological Disorders and Stroke (NINDS) rt-PA Stroke Study, on which FDA approval of intravenous (IV) recombinant tissue-type plasminogen activator (rt-PA) for acute ischemic stroke is based, demonstrated that patients treated with rt-PA within three hours of onset had a 12% absolute increase in the likelihood of minimal or no disability after three months.[15,23] The overall benefit outweighed the risk of a symptomatic ICH, which occurred in 6.4% of patients who received rt-PA compared with 0.6% of those who received placebo.[23] Intervention after three hours yielded no benefit and increased the risk of ICH. The sooner rt-PA was given within the three-hour time window, the better the outcome.[24] Stroke subtype affected efficacy, with a better outcome in small vessel (lacunar) strokes than in large vessel or cardioembolic strokes (Figure 2).[23]

The recommended dose of rt-PA is 0.9 mg/kg, with a maximum total dose of 90 mg; higher doses increase the risk of ICH.[15] To date, no other thrombolytic agent has been approved for this indication. Unfortunately, only about 2% to 4% of patients with ischemic stroke are being treated with IV rt-PA, although a wide variation (0% to 10%) has been reported.[25] The main reasons for the low rate are patients presenting beyond the three-hour time window, the lack of a medical infrastructure, and concerns among some physicians about the safety and efficacy of rt-PA.

Other Acute Medical Care

Management of blood pressure is important in the management of acute stroke, where a balance needs to be maintained between hypertension control and avoidance of cerebral hypoperfusion.[16] The need for this delicate balance is based on the hypothesis that while a central core of brain tissue undergoes infarction within minutes of severe ischemia, a penumbra of surrounding tissue initially is only subtotally ischemic. Unless ischemia is reversed, the penumbra will progress to infarction in minutes or hours. Because salvage of the at-risk penumbra is the goal of emergency treatment and a fall in systemic blood pressure is reflected in hypoperfusion, it is essential not to lower the patient's blood pressure during the acute period after an ischemic stroke. In general, the blood pressure should not be lowered unless it is >210 mm Hg systolic or >120 mm Hg diastolic. For patients who are to receive rt-PA, the parameters are >185 mm Hg systolic and >110 mm Hg diastolic.[15] Labetalol and nicardipine are the preferred agents, but nitroprusside should be used if the diastolic pressure is >140 mm Hg.[15]

Hyperglycemia should be treated aggressively if present.[15] Excess glucose is metabolized into lactic acid in patients with cerebral ischemia, and the lactic acidosis is extremely toxic to neurons. Studies have shown that outcome worsens as blood sugar levels increase.[26,27] Treatment with IV or subcutaneous insulin is warranted if necessary.

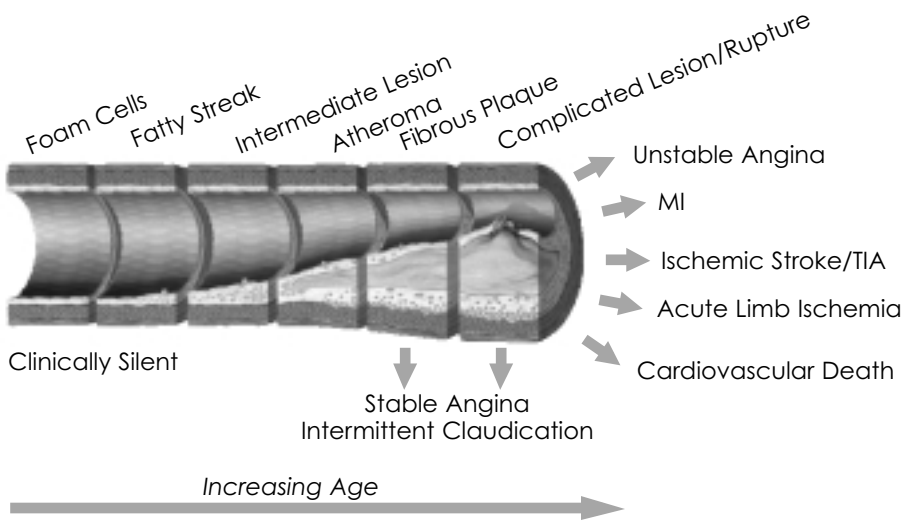
Acute therapy with aspirin within 48 hours is recommended for most patients with ischemic

stroke but should not be given within 24 hours of rt-PA administration.[15] The value of other antiplatelet agents during the acute period has not been proven. Routine emergent anticoagulation with heparin or heparinoids is not recommended because of the high risk of hemorrhagic transformation or ICH, and is contraindicated within 24 hours of rt-PA administration.[15] Subcutaneous administration of heparin or heparinoids is recommended, however, in preventing DVT and pulmonary embolism in bedridden patients with recent stroke.[15]

Secondary Prevention

Atherosclerotic vascular disease is a generalized and progressive process (Figure 3).[28] Over half of patients who survive a hospitalization for a first stroke and/or cerebrovascular disease are likely to die within five years.[29-31] Although the risk of death is greatest during the first 30 days following a stroke, increased risk of mortality persists for several years. Hardie and colleagues[29] showed that death due to cardiovascular disease is more likely to occur over the long term than is death due to a second ischemic stroke in patients surviving more than one year after a stroke (Figure 4). This may be secondary to the large, diffuse atherothrombotic burden present in this population. The data in Figure 5 show that stroke victims are at greatly increased risk for subsequent events, including death, recurrent stroke, or MI and further suggest that clinical risk, as well as atherosclerotic profile, is predictive of subsequent stroke.[32] This diffuse atherothrombosis was demonstrated in a retrospective analysis of more than 1,800 geriatric patients in a hospital-based geriatrics practice that identified 56% of patients with ischemic stroke as having coexistent coronary artery disease.[33]

Figure 3. Atherothrombosis: A Generalized and Progressive Process

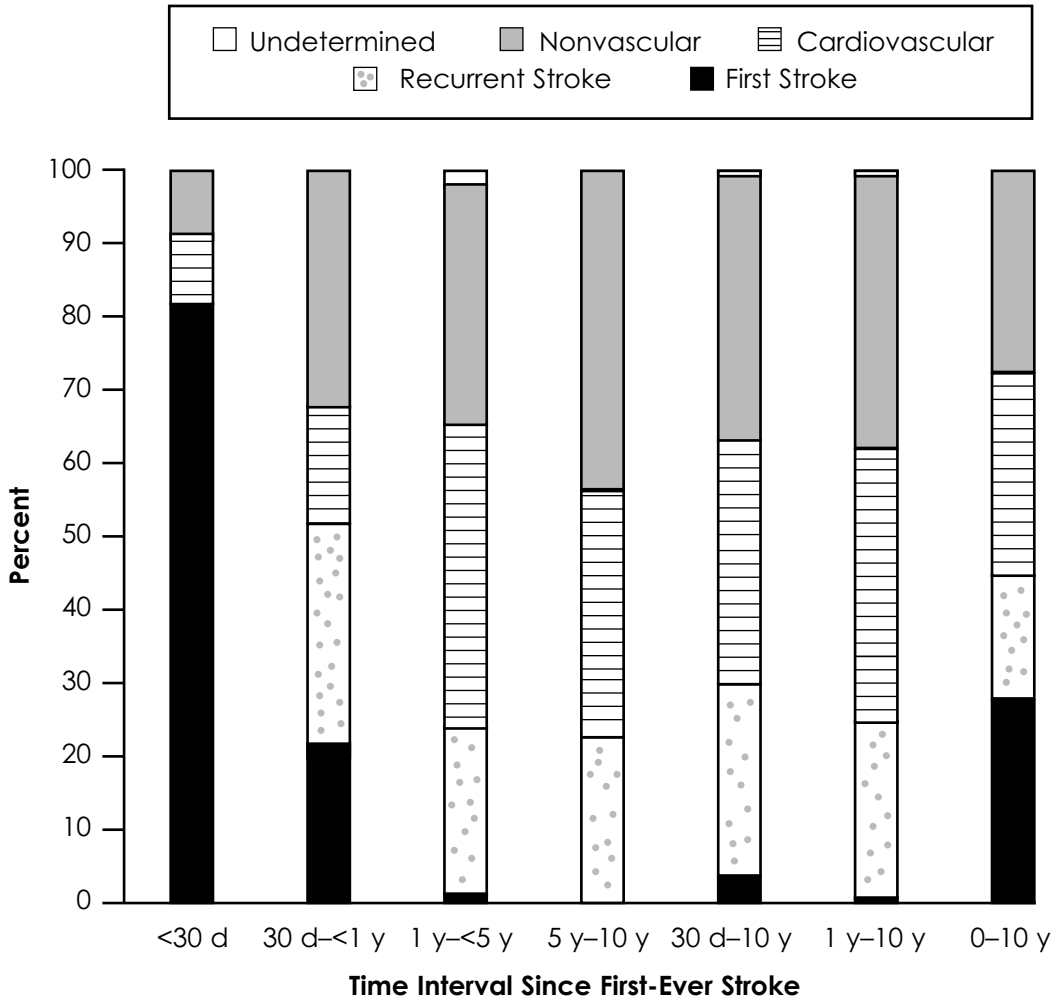


Adapted from Stary HC, Chandler AB, Dinsmore RE, et al. *Circulation*. 1995;92:1355-1374.

Initiating lifestyle changes can make a difference in reducing the risk of subsequent vascular events.[12] Measures such as smoking cessation, a healthy diet, exercise, weight reduction, and moderate alcohol consumption can help reduce the risk of vascular disease in patients who have experienced a stroke.[12]

Medical and surgical treatments are key components of secondary prevention. They are particularly cost-effective in patients with a history of stroke or TIAs because the absolute risk reductions are substantially higher and the numbers needed to treat are substantially lower than in individuals who have not had an event. The rel-

Figure 4. Histogram Showing Proportion of Patients Dying from Different Causes During Different Time Intervals from Onset of First-Ever Stroke



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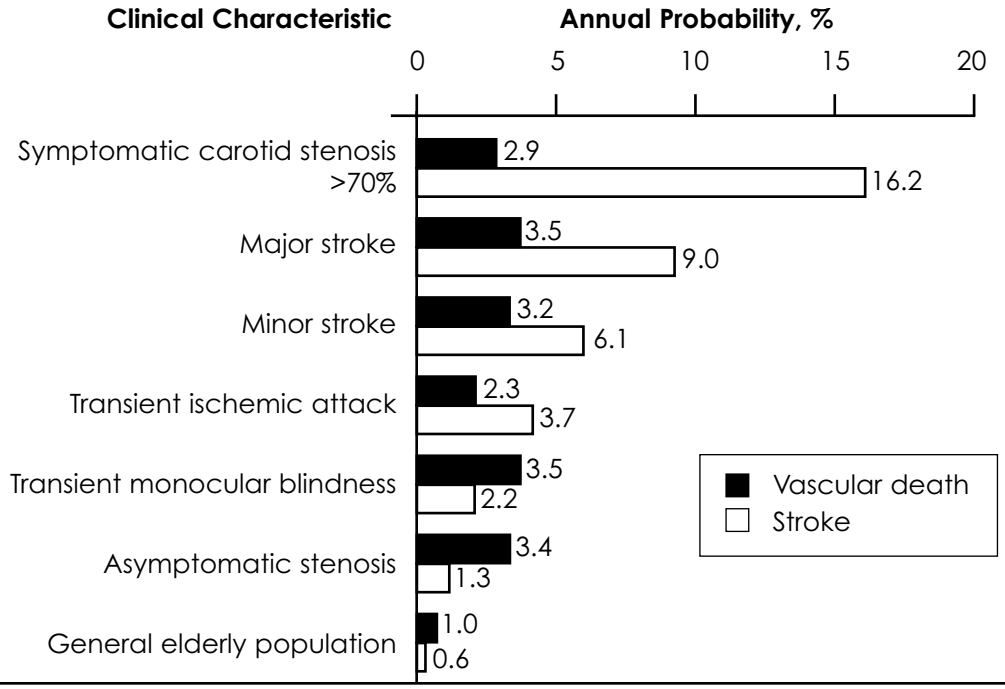
ative risk reduction and the number needed to treat associated with six secondary prevention strategies are summarized in Table 3.[34]

Hypertension is a highly significant risk factor in patients who have had a cerebrovascular event, and antihypertensive therapy addresses the directly proportional relationship between blood pressure level and the risk of stroke.[35] In particular, isolated systolic hypertension in older patients should be treated. However, blood pressure lowering should be gradual to avoid symptoms related to overly aggressive blood pressure reduction, such as cerebral hypoperfusion.[36] The use of an angiotensin-converting-enzyme (ACE) inhibitor, and/or a diuretic, has been shown to reduce recurrent stroke rates.[37-39]

Lowering elevated cholesterol levels is prudent because it helps protect against diffuse vascular disease and decrease the risk of cerebrovascular and coronary events. Aggressive efforts should be made to lower low-density lipoprotein (LDL)-cholesterol levels to <100 mg/dL with statin therapy in patients who have experienced an ischemic stroke or a TIA.[5] The effectiveness of statin therapy in reducing the rate of ischemic (nonembolic) stroke in treated cardiac patients has been demonstrated in the Scandinavian Simvastatin Survival Study (4S trial),[40] the Cholesterol and Recurrent Events (CARE) trial with pravastatin,[41] and the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) trial.[42]

Figure 5. Annual Risk of Stroke or Vascular Death: High-Risk Subgroups



Adapted from Wilterdink JL, Easton JD. *Arch Neurol.* 1992;49:857-863.

Aspirin, the thienopyridines, and a combination of aspirin and extended-release dipyridamole are recommended for secondary prevention of recurrent stroke in patients who have had a noncardioembolic stroke.[19] Aspirin is the most widely studied antiplatelet drug and is the standard of care for secondary prevention of vascular complications after ischemic stroke. The Antithrombotic Trialists' Collaboration summarized the results of antiplatelet trials and found that long-term antiplatelet therapy was associated with a 23% reduction in the risk for nonfatal stroke in patients with a history of stroke or TIA.[43]

In the second European Stroke Prevention Study (ESPS-2) trial,[44] the combination of extended-release dipyridamole plus aspirin produced a statistically significant 37% reduction in the risk of stroke versus placebo; however, statistically significant reductions were not demonstrated for stroke and/or death, MI, or death versus aspirin. The ongoing European/Australian Stroke Prevention in Reversible Ischaemia Trial (ESPRIT) is comparing the benefits and risks of the combination of dipyridamole plus aspirin with aspirin alone in patients with a TIA or a minor ischemic stroke.[45]

The antithrombotic effects of clopidogrel were compared with those of aspirin in the Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events (CAPRIE) study.[46] Patients treated with clopidogrel demonstrated an 8.7% relative risk reduction for the outcome cluster of ischemic stroke, MI, or vascular death compared with aspirin (P=0.043). In the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial, the combination of clopidogrel with aspirin produced a 20% relative risk reduction for vascular events in patients with acute coronary syndromes compared with aspirin alone.[47] The results of the recent Clopidogrel for the Reduction of Events During Observation (CREDO) trial showed that long-term therapy consisting of clopidogrel plus

Table 3. Secondary Prevention of Stroke by Treatment

Treatment	RRR	NNT (One Stroke/Year)
Antihypertensive	28%	51
Statins	25%	57
Aspirin	28%	77
Thienopyridine versus aspirin	13%	64
Smoking cessation	33%	43
Carotid endarterectomy	44%	26

RRR = relative risk reduction; NNT = number needed to treat to prevent one stroke per year

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aspirin significantly reduced the risk of ischemic events at one year compared with aspirin alone in patients undergoing elective percutaneous coronary intervention (PCI).[48] The Management of Atherosclerosis with Clopidogrel in High-Risk Patients with Recent Transient Ischaemic Attack or Ischaemic Stroke (MATCH) trial is comparing the efficacy of clopidogrel plus aspirin with that of clopidogrel alone for preventing stroke, MI, and death in high-risk patients with a recent TIA or ischemic stroke.[49]

Warfarin is recommended for both primary and secondary prevention of cardioembolic stroke in patients with atrial fibrillation.[19] A meta-analysis of 16 randomized trials of warfarin versus aspirin or placebo in patients with atrial fibrillation indicated that warfarin was associated with a lower risk of stroke.[50] In the Warfarin-Aspirin Recurrent Stroke Study (WARSS), warfarin was not superior to aspirin in patients with a recent history of *noncardioembolic* ischemic stroke for prevention of the combined primary endpoint of recurrent ischemic stroke or death.[51]

Carotid Endarterectomy to Prevent Stroke

Patients with symptomatic carotid stenosis are at high risk for stroke: the risks are 13% per year in those with 70% to 99% stenosis and 7% per year in those with 50% to 69% stenosis.[52] Endarterectomy plus medical therapy produces threefold greater reductions than medical therapy alone in the risk of ipsilateral stroke within five years in patients with symptomatic 70% to 99% stenosis, and the benefit is realized quickly. However, the benefit is diminished in those with 50% to 69% stenosis, and there is no benefit if the stenosis is <50%. The American Heart Association guidelines for carotid endarterectomy point out that there are intraoperative and perioperative risks associated with the procedure.[53] Endarterectomy may be considered for asymptomatic carotid stenosis, provided the stenosis is ≥60%. The perioperative complication rates (morbidity or mortality) should be <6% for symptomatic stenosis and <3% for asymptomatic stenosis.

Preliminary data suggest that balloon angioplasty with stent placement, perhaps with the addition of distal protective devices, may be considered when surgical risks are too high because of congestive heart failure, chronic obstructive pulmonary disease, severe coronary heart disease, or previous carotid endarterectomy, radical neck surgery, or radiation therapy.[54] Randomized trials comparing stenting with carotid endarterectomy in patients with symptomatic carotid stenosis are currently in progress.[55]

Implementing Stroke Best Practices: Providing a Continuum of Care for Enhanced Patient Outcomes

The continuum of care for stroke patients consists of acute care, critical pathways (a.k.a. care maps, protocols) for in-hospital management, and discharge planning. Initiation of secondary prevention while the patient is still hospitalized for the initial ischemic stroke requires the creation of care management teams functioning with preprinted orders and testing per protocol. Several examples of ongoing educational programs are cited below.

AHA "Get with the Guidelines" (GWTG) Stroke Program

The AHA-sponsored GWTG program focuses on care team protocols to ensure that patients with ischemic stroke are treated with the proper medications and discharged appropriately.[9] In the acute stage, emphasis is placed on improving timeliness by obtaining answers to the following questions:

- When did the symptoms begin?
- How long did it take for emergency medical services to arrive after receipt of a call?
- How long did it take for the patient to arrive at the emergency department?
- Is this the patient's first neurologic deficit?
- When was CT scanning begun?
- What was the timing of thrombolytic therapy (if applicable)?

After immediate laboratory sampling (e.g., blood glucose), blood pressure measurement, and noncontrast cranial CT, in-hospital management continues with:

- a decision to administer thrombolytic therapy based on the timing of the symptoms and the nature of the insult
- maintenance of appropriate blood pressure, hydration, and airway adequacy
- other medical management (e.g., aspirin, heparin)
- planning for physical, speech, and other adjunctive therapy as necessary

All of the following secondary stroke prevention measures are addressed in the hospital and then followed through appropriately after discharge:

- appropriate antiplatelet therapy
- smoking cessation counseling
- moderation of alcohol consumption
- lipid-lowering therapy
- blood pressure control
- diabetes management
- weight and exercise management
- atrial fibrillation management
- a strategy to assess and treat carotid artery stenosis

The GWTG program advocates mobilizing hospital- and community-based physicians, as well as other healthcare professionals, into a cohesive and motivated team. The program focuses on care team protocols to ensure that patients are treated with and discharged on appropriate medications and with risk-modification counseling. "Champions" from the hospital staff are identified to lead, develop, and mobilize teams to implement treatment and discharge guidelines for stroke patients in their respective institutions.

UCLA Stroke PROTECT Program

The Stroke PROTECT (Preventing Recurrence of Thromboembolic Events through Coordinated Treatment) program[8] developed at the University of California, Los Angeles (UCLA) Medical Center, was designed to reduce the incidence of stroke—and, simultaneously, MI and peripheral arterial occlusion—by integrating proven prevention measures into standard hospital care. It operates at the community level and involves interaction of the patient, family, and primary care physician with the hospital staff. Its elements consist of preprinted orders, simple guidelines, education, discharge treatment, and prospective monitoring of treatment use. All patients admitted with a diagnosis of ischemic stroke or TIA participate to a degree

depending on their stroke subtype. PROTECT has eight goals—four therapeutic and four behavioral—to be accomplished before discharge (Table 4).

The most visible effect of PROTECT has been the greatly increased utilization of statins and ACE inhibitors/angiotensin-II receptor blockers at discharge. Parallels may be drawn with the American College of Cardiology's Guidelines Applied in Practice (GAP) program and UCLA's Cardiac Hospitalization Atherosclerosis Management Program (CHAMP).[6,7] Both of these programs significantly increased utilization of antiplatelets, lipid-lowering drugs, and other appropriate agents and behavior modifications in patients discharged after acute MI. The programs used customized guideline-oriented tools designed to facilitate adherence to evidence-based therapy.[6,7]

Summary

Reducing the high morbidity and mortality associated with stroke continues to be a major healthcare challenge in the United States. Programs that bridge the gap between evidence-based medicine and clinical practice can improve patient outcomes. The "Stroke Best Practices" workshops developed by NCME were designed to assist institutions that lack stroke centers in implementing the latest evidence-based practices for in-hospital stroke management. The importance of postdischarge continuum of care was emphasized throughout the program. Lectures covering the topics addressed in this article were delivered by national experts in neurology, emergency medicine, and vascular surgery. Formal presentations were followed by individual working discussions among healthcare professionals attending from local institutions. Sample pathways and implementation tools from successful stroke programs across the country were shared during breakout sessions and used as templates for the creation of best practices suited to the infrastructure and resources of each hospital. Short-, intermediate-, and long-term goals were set by each institution during the breakout sessions. Each hospital also nominated a "champion" to sustain the momentum of the best practices program and to facilitate follow-up with NCME.

Table 4. Goals of the UCLA Stroke PROTECT Program

Therapeutic Goals	Behavioral Goals
Appropriate hospital initiation and maintenance of:	
• Antithrombotic therapy • ACE inhibitor/ARB • Statin • Thiazide diuretic	• Exercise education • Diet education • Smoking cessation • Awareness of stroke warning signs and the need to call 911

ARB = angiotensin-II receptor blocker

UCLA Stroke PROTECT Program. Available at: http://www.strokeprotect.mednet.ucla.edu.

By employing a team approach and implementing stroke best practices, institutions without stroke centers may use evidence-based best practices to bridge the gap between guidelines and clinical practice to optimize the management of acute stroke, prevent secondary vascular events, and enhance the quality of care for patients with established cerebrovascular disease.

Outside the hospital, primary care physicians are the first line of defense against stroke. Valuable, evidence-based interventions, such as identifying and treating atrial fibrillation and carotid stenoses, rushing stroke patients to rt-PA therapy within the three-hour time window, and ensuring that patients are treated in a stroke unit, can be easily implemented in clinical practice. Evidence also supports the need for rigorous blood pressure control and appropriate antiplatelet and statin therapy. In addition, all patients should be counseled about lifestyle modification for stroke prevention and instructed to dial 911 if they experience the warning signs of a stroke. If these important stroke prevention measures are followed, stroke morbidity and mortality may be reduced.

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