

Stroke Management in the Long-Term Care Setting

CLINICAL PRACTICE GUIDELINE

 **PALMed**
POST-ACUTE AND LONG-TERM CARE
MEDICAL ASSOCIATION

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To cite this guideline use: Post-Acute and Long-Term Care Medical Association. Stroke Management in the Long-Term Care Setting Clinical Practice Guideline. Columbia, MD: PALTmed 2011

Preface

This clinical practice guideline (CPG) has been developed under a project conducted by Post-Acute and Long-Term Care Medical Association (PALTmed), the national professional association of medical directors, attending physicians, and others practicing in the long term care continuum. This is one of a number of guidelines undertaken as part of the association's mission to improve the quality of care delivered to patients in these settings.

Original guidelines are developed by interdisciplinary workgroups, using a process that combines evidence and consensus-based approaches. Workgroups include practitioners and others involved in patient care in long-term care facilities. Beginning with pertinent literature searches for articles and information related to the guideline subject, and a draft outline/framework, each group works to make a concise, usable guideline that is tailored to the long-term care setting. Because scientific research in the long-term care population is limited, many recommendations are applied research of older adults and geriatric medicine. Some recommendations are based on the expert consensus opinion of practitioners and geriatric experts in the field.

Guideline revisions are recommended under the direction of the Clinical Practice Guideline Steering Committee. The Steering Committee reviews any PALTmed guidelines that are three years old prior to an annual Steering Committee meeting to determine if the CPG is current. (A thorough literature review is done for each CPG as well to ascertain if the data within is still current.) The PALTmed Clinical Practice Committee Chair selects the guidelines to be revised/created based on 1) the Steering Committee recommendations, 2) data collected, and 3) an assessment of the difficulty of development and relevance to the PALTmed membership. The Board of Directors has final approval. The guideline revision process is similar to the original guideline process, except the workgroup starts with the original guideline (or last revision) as a basis to begin with.

Purpose

PALTmed seeks to develop and revise guidelines that focus on specific concerns and common problems in the long-term care setting. Although other agencies, organizations, and associations have developed a number of guidelines for conditions that occur in elderly and chronically ill individuals, many of these guidelines limit or omit considerations that are unique to the long-term care population.

PALTmed guidelines emphasize key care processes and are created to be used in conjunction with facility-specific policies and procedures to guide staff and practitioner practices and performance. They are meant to be used in a manner appropriate to the population and practice of a particular facility. Guideline implementation may be affected by resources available in the facility, including staffing, and will require the involvement of all those in the facility who have a role in patient care.



Audience

This guideline is intended for the members of the interdisciplinary team in long-term care facilities, including the medical director, director of nursing, practitioners, nursing staff, consultant pharmacist, and other professionals such as therapists, social workers, dietitians, and nursing assistants who care for residents of long-term care facilities.

PALTmed CPGs include many functions and tasks related to recognizing, clarifying, managing, and monitoring various conditions and situations. But the guidelines only sometimes specify who should do these tasks. For example, many disciplines including nursing assistants, licensed nurses, dietitians, and social workers may make and document observations (e.g., that someone does not sleep at night, is more withdrawn, or has a change in usual eating patterns). But only some of them may be qualified to determine the significance of those observations (for example, what is causing the sleeplessness or change in eating patterns). In contrast, practitioners may not be present to make observations, but are trained to analyze the significance and causes of symptoms. Thus, each facility should ensure that tasks are done correctly and by the appropriate interdisciplinary team members. It is important for observers to make and document findings effectively, but they should get appropriate support for interpreting the findings when this is not within the scope of their training or practice.

Assumptions

Guidelines in the long-term care setting should be consistent with fundamental goals of desirable long-term care practice. Operationally, this requirement means that the nursing facility care team systematically addresses (1) each individual's risk factors for a number of diseases and conditions and (2) the adverse consequences of the diseases and conditions on the patient's functioning and quality of life.

However, when nursing facility patients are at or near the end of life, care goals will shift from functional improvement or physical stability to palliation or comfort care. PALTmed guidelines address this transition and provide suggestions for appropriate modification of the patient's care plan.

Long-term care facilities care for a variety of individuals, including younger patients with chronic diseases and disabilities, short-stay patients needing postacute care, and very old and frail individuals suffering from multiple comorbidities. When a workup or treatment is suggested, it is crucial to consider if such a step is appropriate for a specific individual. A workup may not be indicated if the patient has a terminal or end-stage condition, if it would not change the management course, if the burden of the workup is greater than the potential benefit, or if the patient or his or her proxy would refuse treatment. It is important to carefully document in the patient's medical record the reasons for decisions not to treat or perform a workup or for choosing one treatment approach over another.

How to Use These Guidelines

Each guideline includes a narrative portion that covers definition, recognition, assessment, treatment, and monitoring of the condition being addressed. "Recognition" means identifying the presence of a risk or condition. "Assessment" means clarifying the nature and causes of a condition or situation and identifying its impact on the individual. "Treatment" means selecting and providing appropriate interventions for that individual. "Monitoring" means reviewing the course of a condition or situation as the basis for deciding to continue, change, or stop interventions.

Each guideline also includes an algorithm that summarizes the steps involved in addressing the condition. In the algorithm, rectangles signify points where action is to be taken; diamonds indicate points where a decision must be made.

Terminology

We recognize that people who reside in long-term care facilities are “residents”. However, we have used the term “patient(s)” throughout these guidelines because we are addressing individuals within the context of treating a medical condition. In addition, these guidelines also apply substantially to individuals who come to long-term care facilities for short-term care. When referring to pharmaceutical products, we have avoided the use of brand names and refer to classes of drugs whenever possible.





TABLE OF CONTENTS

DEFINITION	1
INTRODUCTION	1
Classifications of Stroke	2
Challenges to the Optimal Management of Stroke in the Long-Term Care Setting	2
<i>Acute stroke</i>	3
<i>Post-stroke</i>	3
Facility Preparedness	3
Importance of Staff Education	4
TABLE 1. Medical Director's Role in Stroke Management in the Long-Term Care Facility	4
Documentation and Decision Making	5
Expected Outcomes From Implementation of Stroke Clinical Practice Guideline	5
Stroke Management Falls Into Three Categories of Urgency	5
RECOGNITION	6
TABLE 2. Potentially Modifiable Risk Factors for Stroke	7
ASSESSMENT	7
TABLE 3. Conditions That Can Produce Signs or Symptoms Similar to a Stroke	8
TABLE 4. Potential Benefits and Risks of Hospital Transfers for Long-Term Care Stroke Patients	9
TABLE 5. Diagnostic Tests for the Initial Hospital-Based Evaluation of Stroke Signs and Symptoms	10
TABLE 6. Elements of a Comprehensive Assessment for a Stroke Patient	12
TABLE 7. Signs and Symptoms of a Possible Swallowing Problem	17
TREATMENT	19
TABLE 8. Strategies to Decrease the Risk of Stroke Complications	20
TABLE 9. Specific Positioning and Handling Techniques to Support a Hemiparetic Arm	22
TABLE 10. Possible Goals of a Stroke Rehabilitation Plan	27
Lifestyle Modifications	28
Hypertension	28
Diabetes	28
Lipids	29
Antiplatelet Therapy to Prevent Thrombotic Stroke	29
Bleeding Risk From Antiplatelet Therapy	30
Atrial Fibrillation	30
Symptomatic Carotid Stenosis	31
MONITORING	32
SUMMARY	33
REFERENCES	33
APPENDIX 1. Modifiable Risk Factors for TIA and Stroke: Interventions, Treatment Goals, and Strategies for Monitoring Adverse Drug Effects	41-43
APPENDIX 2. Quantitative Bleeding Risk Assessment Tool	44
APPENDIX 3. Antithrombotic Baseline Risk/Benefit Assessment Tool	45-46





Stroke Management in the Long-Term Care Setting

DEFINITION

A **stroke** occurs when the local blood supply to the brain is suddenly interrupted, causing brain cell damage and death. The resulting signs and symptoms depend on the location and residual function of the damaged brain areas. Strokes are also called “brain attacks” to emphasize that they are emergencies requiring prompt evaluation and treatment.

INTRODUCTION

Each year, more than 795,000 Americans experience a stroke. About 610,000 of these are first attacks and 185,000 are recurrent attacks. On average across the United States, someone suffers a stroke every 40 seconds, and someone dies from a stroke every 4 minutes.¹

Stroke is also the leading cause of long-term disability in the United States. A population-based 6-month follow-up study of people aged 65 years or more who suffered a stroke showed that 50% had some paralysis on one side of the body (hemiparesis), 35% had symptoms of depression, 30% were unable to walk without some assistance, 26% were dependent in activities of daily living (ADLs), 19% had aphasia, and 26% resided in long-term care (LTC) facilities.² Stroke is a leading cause of hospitalizations for Medicare stroke survivors, and many survivors often require post-acute care for 6 months or more.³ Stroke survivors often require longer stays in LTC facilities in the 5 years following the attack than do people of the same sex and age who have not had a stroke.⁴ The combined direct and indirect costs of strokes in the United States in 2010 were estimated to total \$73.7 billion.¹

Even a transient ischemic attack (TIA) has important implications; in the 90 days following a TIA, between 3% and 17% of patients will have a stroke.^{1,5-8} More ominously, the 1-year mortality rate after a TIA is 25%.^{1,5,9}

Age is the most important risk factor for stroke; the peak incidence of stroke occurs among people aged 80 years or more. With increasing age, the prognosis of stroke worsens, with higher chances of death and discharge to a LTC facility.^{10,11} Despite the negative effects of advancing age on stroke outcome, those over 80 derive the same magnitude of benefit from organized inpatient stroke care as do younger stroke patients.¹²

Classifications of Stroke

Strokes are generally classified as ischemic or hemorrhagic on the basis of their underlying cause. This classification helps to guide both acute therapy and preventive interventions. Of all strokes, 87% are ischemic, 10% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage.¹

Most strokes in older adults are ischemic. An **ischemic** stroke occurs when local blood flow to the brain decreases, causing brain cell death. The decrease in blood flow is usually caused by a blood clot (thrombus) blocking an artery that supplies blood to the brain (i.e., a thrombotic ischemic stroke). Further subclassification of thrombotic ischemic strokes is based on the cause of the arterial blockage (i.e., large-artery atherosclerosis, small-artery vascular occlusion, or cardioembolism).

- ◆ **Large-artery atherosclerosis** involves the large arteries that supply blood to the brain (e.g., aorta, carotid, vertebral, basilar, middle cerebral, anterior cerebral). Arterial clots can form at the site of severe arterial narrowing or with rupture of an atherosclerotic plaque. Treatment and prevention of large-artery strokes focus on controlling processes that promote atherosclerosis (e.g., hypercholesterolemia, hypertension, diabetes, smoking) and reducing the likelihood of thrombus formation (thrombosis), primarily through antithrombotic medications and smoking cessation.
- ◆ Small-artery thrombotic stroke usually involves the penetrating arteries that branch directly off the large arteries at the base of the brain. **Small-artery vascular occlusion** is caused by abnormal thickening of arterial walls resulting from hypertension or diabetes and is usually not directly caused by atherosclerosis. Prevention of small-artery vascular occlusion focuses on controlling hypertension, controlling diabetes, and preventing thrombus formation with medications.¹³
- ◆ A **cardioembolic stroke** occurs when a thrombus (blood clot) originates in the heart and travels (embolizes) to the brain. Thrombus formation within the heart is caused by cardiac abnormalities including atrial fibrillation, prosthetic valves, diseased mitral and aortic valves, and the loss of muscular responsiveness in segments of the heart wall following myocardial infarction (MI). Prevention of cardioembolic strokes focuses on treatment with anticoagulants to reduce thrombus formation or correction of structural abnormalities predisposing to cardiac thrombosis.

A **hemorrhagic stroke** occurs when a cerebral artery ruptures. Common mechanisms of arterial rupture include arterial stiffness or fragility caused by atherosclerosis and hypertension or weakness in the arterial wall resulting from an aneurysm or vascular damage. Other causes of hemorrhagic stroke include bleeding disorders, bleeding associated with a brain tumor, arteritis, cerebral amyloid angiopathy,¹⁴ and trauma.

A **transient ischemic attack** is defined as the occurrence of signs or symptoms of localized brain ischemia that clear completely within 24 hours of onset. Most TIAs last less than 3 hours. Despite resolution of signs and symptoms, imaging reveals cerebral infarction in up to 30% of clinically diagnosed TIAs.¹⁵

Challenges to the Optimal Management of Stroke in the Long-Term Care Setting

Challenges for stroke care in LTC facilities fall into three broad categories:

◆ **Acute stroke**

- ◆ Ensuring prompt recognition, evaluation, triage, and treatment of acute stroke that occurs in the facility;

◆ **Post-stroke**

- ◆ Preventing, recognizing, and treating post-stroke complications;
- ◆ Optimizing post-stroke physical, cognitive, and psychosocial function;
- ◆ Secondary prevention; and

◆ **Stroke prevention**

- ◆ Identifying and treating modifiable stroke risk factors to reduce the risk of future strokes.

Acute stroke

In the LTC setting, the interdisciplinary team may be challenged by the difficult task of assessing and differentiating stroke from other causes of similar symptoms.

Post-stroke

Survivors of an acute stroke who are transferred to a LTC facility are more likely to have dependence in ADLs and instrumental ADLs, have impaired bed mobility, be at risk for nutritional deficits, have pressure ulcers or be at high risk for skin breakdown, or have impaired bladder and bowel continence.¹⁶ Thus, these patients need a comprehensive, interdisciplinary functional assessment that considers their strengths and weaknesses and a treatment plan that addresses risks for post-stroke complications; helps to optimize physical, cognitive, and psychosocial function; and reduces the risk of stroke recurrence.

The interdisciplinary treatment plan should reflect the fact that a stroke is commonly a sign that the patient has generalized vascular disease. The cumulative risk of stroke in the 5 years after an initial stroke is 32% and the cumulative risk of death is 58%, which stresses the need for long-term vigilance and stroke prevention.¹⁷ The Perth Community Stroke Study showed that persons who suffer a stroke are at increased risk for recurrent strokes, cardiovascular events, and death.¹⁸ The study also found that the cause of death among stroke survivors changed over time. In the first 30 days following a stroke, nearly 70% of deaths were related to the initial stroke and its complications. Among patients who survived for 1 year, 41% of subsequent deaths were due to cardiovascular causes (i.e., MI, sudden cardiac death, aortic aneurysm, and claudication); 10% of deaths were related to the initial stroke, and 5% to a recurrent stroke.¹⁸

Thus, a comprehensive assessment for a patient with stroke includes a review of signs, symptoms, and risk factors for both coronary artery disease and peripheral arterial disease. Most importantly, the care team should individualize its approach to stroke evaluation, treatment, and prevention. Ideally, the team and patient should reach shared decisions that consider the patient's physical, cognitive, and psychosocial well-being; prognosis; and goals of therapy. These decisions should consider the benefits, risks, and alternatives to evaluations, tests, and treatments.

Facility Preparedness

LTC facilities should consider developing policies and procedures for responding rapidly to brain attacks. The phrase "time is brain" refers to the fact that hospital-based thrombolysis with tissue plasminogen activator (tPA) is most effective when administered as soon as possible within 3 hours of stroke onset. In properly selected patients aged 80 years or more, tPA can be both safe and effective.¹⁹

In select circumstances, tPA may be given up to 4.5 hours after stroke onset²⁰; however, patients aged over 80 years, those with an international normalized ratio (INR) higher than 1.7, those with a score higher than 25 on the National Institutes of Health (NIH) stroke scale, and those with a history of both diabetes and stroke may not receive tPA after the 3-hour time limit.²¹

Advanced age or residence in a LTC facility should not by themselves be considered contraindications to hospital-based evaluation and treatment of acute stroke. Decisions to evaluate and treat cardiovascular risk factors should be based on patients' choices and practitioners' assessments of the risks and benefits of testing and treatment.

Facilities should also develop and implement approaches to reduce the likelihood of a new acute stroke or other cardiovascular event. Many patients in LTC facilities have a history of stroke or TIA or have multiple risk factors for stroke; yet some patients can obtain important survival and functional benefits from appropriate treatments to reduce stroke and cardiovascular risk. Despite the prevalence of stroke risk factors, many LTC patients may not receive appropriate medications and other interventions to modify stroke risk factors and decrease the chance of stroke recurrence.^{22,23}

Importance of Staff Education

Caregiving staff should be able to identify

- ◆ Patients who show possible signs or symptoms of an acute stroke,
- ◆ Stroke risk factors, and
- ◆ Interventions to control risk factors.

Patient care staff are likely to be among the first to notice changes in a patient. Nursing assistants, nurses, and other caregiving staff should receive training about how to respond and with whom to communicate when they recognize the signs and symptoms of an acute stroke or stroke complications. The medical director plays a key role by helping the facility to identify and use effective education programs, policies and procedures, and clinical approaches for stroke management (Table 1).

TABLE 1

Medical Director's Role in Stroke Management in the Long-Term Care Facility

- ◆ Help the facility to develop the competence of direct patient care staff in recognizing, assessing, and managing acute stroke and its complications
- ◆ Help to establish facility processes for responding to an acute stroke, including establishing an organized and timely process for determining whether to transfer a patient to the hospital or to treat the patient on site
- ◆ Facilitate comprehensive interdisciplinary assessment and treatment for patients who have had a stroke or have stroke risk factors
- ◆ Encourage individualized assessment and treatment plans for stroke survivors that are based on a comprehensive and accurate functional assessment of the resident's strengths, weaknesses, risk factors for deterioration, and potential for improvement
- ◆ Help the facility to identify up-to-date educational programs that address the recognition, evaluation, and prevention of acute stroke
- ◆ Help to educate staff and families about the medical, functional, and psychological issues associated with stroke
- ◆ Help to develop and use outcome and process indicators to measure facility performance in stroke management

Documentation and Decision Making

Decisions about the evaluation and treatment of patients affected by stroke involve complex clinical judgments. For example, patients with advanced illness may be unlikely to benefit from some elements of evaluation, treatment, and monitoring. Other patients may choose treatment goals that emphasize symptomatic relief and maintenance of function but de-emphasize extensive evaluation and aggressive medical interventions.

Ideally, decisions about evaluation and treatment should reflect the autonomous choices of a well-informed patient. If a patient lacks decision-making capacity, this limitation should be documented. If a surrogate decision maker is designated to make decisions on the patient's behalf, the rationale and any supporting legal documentation (e.g., advance directives, durable power of attorney for health care) should be included in the patient's medical record.

Documentation of decisions to forgo assessment or treatment should summarize discussions with the patient, family or surrogate decision maker, and relevant interdisciplinary team members. The medical record should reflect key elements of the decision-making process, including the diagnosis, prognosis, assessment, and treatment options (including refusal of assessment or treatment) as well as the possible benefits and risks associated with these options.

Expected Outcomes From Implementation of Stroke Clinical Practice Guideline

Outcomes that may be expected from the implementation of this clinical practice guideline include the following:

- ◆ Timely recognition of acute stroke,
- ◆ Implementation of appropriate strategies to prevent complications of stroke,
- ◆ Improved monitoring for and recognition of acute complications of stroke,
- ◆ Minimization of acute stroke complications,
- ◆ Improved control of modifiable risk factors for stroke,
- ◆ Improved utilization of appropriate anticoagulant and antithrombotic therapies,
- ◆ Improved quality of life for patients with stroke,
- ◆ Improved documentation of patient choices about assessments and treatments,
- ◆ Decreased readmissions of patients undergoing acute stroke rehabilitation, and
- ◆ Increased percentage of patients admitted for acute stroke rehabilitation who are discharged home.

Stroke Management Falls into Three Categories of Urgency

- ◆ **Acute stroke** is a medical emergency that should be addressed immediately.
- ◆ **Post-stroke** involves care for a patient who has had a stroke recently.
- ◆ **Stroke prevention** involves measures to prevent a first or recurrent stroke.

Certain steps in this guideline are particularly relevant in the context of an acute stroke, whereas others are applicable in the post-stroke or stroke prevention context. These differences are reflected in the text by the use of geometric shapes. (▼ ACUTE STROKE, ● POST-STROKE, ✚ STROKE PREVENTION).

RECOGNITION

Early identification and treatment can help to decrease stroke-related disability and death for appropriately selected patients. For this reason, the signs of acute stroke must be recognized and addressed promptly.

STEP 1 ▼ ACUTE STROKE

Does the patient show signs or symptoms of an acute stroke? Common presentations of an acute stroke include

- ◆ Sudden confusion, difficulty speaking, or difficulty understanding speech;
- ◆ Sudden difficulty seeing out of one eye;
- ◆ Sudden difficulty walking, severe dizziness, or loss of balance or coordination;
- ◆ Sudden numbness or weakness of the face or in an arm or leg, especially if confined to one side of the body; and
- ◆ Sudden severe headache with no other readily identifiable cause.

If at any time a patient displays any of these acute neurological symptoms, go immediately to Step 4, page 7.

Although stroke symptoms develop quickly, caregivers may not witness their onset. For example, a stroke can occur during sleep. Moreover, a stroke does not always cause a dramatic decline in muscle strength, speech, or level of consciousness. A stroke may cause a decline in function even if muscle strength remains normal.

A stroke may cause the new onset of any or all of the following signs and nonspecific symptoms and should be considered in the differential diagnosis if no other apparent cause can be identified:

- ◆ Denial of physical deficits,
- ◆ Difficulty judging distance or depth,
- ◆ Difficulty recognizing or paying attention to one side of the body or environment,
- ◆ Difficulty with new learning,
- ◆ Impulsiveness or poor planning,
- ◆ Poor judgment,
- ◆ Poor safety awareness,
- ◆ Short attention span, and
- ◆ Change in mental status with associated signs and symptoms as listed above.

STEP 2 ● POST STROKE

Has the patient had a previous stroke or a TIA? A patient with a history of stroke or TIA is at high risk for a recurrent stroke. If a newly admitted patient has a history of stroke or TIA, the interdisciplinary team should review available medical records to determine whether the patient has received an appropriate diagnostic evaluation. *If the patient has been evaluated appropriately, go to Step 7. If not, go to Step 6.*

STEP 3 ❖ STROKE PREVENTION

Does the patient have risk factors for stroke? Many LTC patients who have not had a stroke or TIA may have one or more potentially modifiable risk factors for stroke (Table 2).²⁴ Identifying and treating modifiable risk factors is an effective way to reduce the chance of a first stroke or recurrent stroke. *If the patient has not had a stroke but has potentially modifiable risk factors for stroke, go to Step 14.*

TABLE 2
Potentially Modifiable Risk Factors for Stroke

- ◆ Hypertension
- ◆ Atrial fibrillation
- ◆ Hyperlipidemia
- ◆ Diabetes mellitus
- ◆ Estrogen use
- ◆ Carotid artery stenosis
- ◆ Cigarette smoking
- ◆ Heavy alcohol use
- ◆ Inactivity
- ◆ Obesity
- ◆ Sleep apnea

ASSESSMENT

STEP 4 ▼ ACUTE STROKE

Confirm that the patient is suffering an acute stroke.

◆ **Clarify and describe the patient's signs and symptoms.**

Rapidly but thoroughly assess the patient who has signs or symptoms of an acute stroke. Carefully describe the patient's current level of consciousness, cognitive ability, speech, physical function, and physical condition.

It is important to compare the patient's current status with his or her usual (baseline) level of physical, cognitive, and psychosocial function. Many LTC patients already have impairments in physical, cognitive, and psychosocial function caused by previous strokes or other conditions. For patients with pre-existing deficits in physical, cognitive, or psychosocial function, a change in neurological function from baseline may represent a new stroke.

In patients who do not have significant neurological deficits, the following three-step test can help caregivers to quickly and accurately identify the presence of a probable acute stroke:

1. "Show us your teeth." (Observe for facial weakness.)
2. "Close your eyes and raise your arms." (Observe for one-sided weakness.)
3. "Repeat this simple sentence: 'The sky is blue in Cincinnati.'" (Observe for slurred speech, speech deficits, or difficulty understanding speech.)

If this test reveals unilateral facial weakness, unilateral limb weakness, or difficulty with speech that is new or represents a decline from previous function, the patient may be having an acute stroke. Untrained observers using this test have been shown to accurately detect 96% of speech deficits, 97% of cases of one-sided arm weakness, and 72% of cases of facial weakness.²⁵

◆ **Determine whether the patient’s signs and symptoms are caused by a condition that can resemble a stroke.**

Numerous conditions common in the LTC setting can cause signs and symptoms that resemble an acute stroke (Table 3). These conditions usually cause generalized, rather than focal, neurological signs and symptoms. For example, lethargy or confusion that presents without focal loss of strength or sensation may indicate a medical disorder, such as hypoglycemia, hypotension, hypoxia, infection, or an adverse drug reaction.

Urgent vital signs, a finger stick blood-glucose test, pulse oximetry, dipstick urinalysis, and a review of the patient’s drug regimen can quickly determine whether a common condition other than an acute stroke may be contributing to the patient’s signs and symptoms. (See PALmed’s clinical practice guideline on acute change of condition.^a) **If the patient has hypoxia, hypoglycemia, hypotension, or another acute medical condition that may mimic an acute stroke, go to Step 9.**

◆ **Reassess the patient to determine whether symptoms have resolved.**

If the patient’s symptoms do not resolve within 20 minutes, go to Step 5. If the patient’s symptoms are resolving quickly without specific treatment, the symptoms may have been caused by a TIA. A TIA is still considered a brain attack. Patients who have a TIA are at high risk for stroke and should be evaluated promptly and thoroughly (Step 6).

TABLE 3

Conditions That Can Produce Signs or Symptoms Similar to a Stroke

Common

- ◆ Adverse drug reaction
- ◆ Fluid and electrolyte disturbances (e.g., dehydration, hyponatremia)
- ◆ Hypoglycemia
- ◆ Hypotension
- ◆ Hypoxia
- ◆ Systemic infection (e.g., urinary tract infection, pneumonia, sepsis)
- ◆ Vasovagal reaction

Less common

- ◆ Cranial nerve neuropathy
- ◆ Hepatic encephalopathy
- ◆ Hyperglycemia with hyperosmolar state
- ◆ Hypertensive encephalopathy
- ◆ Paresthesia or numbness of unknown cause
- ◆ Parkinsonism
- ◆ Post-seizure paralysis
- ◆ Primary or metastatic brain tumor
- ◆ Recurrent or persistent seizures (status epilepticus)
- ◆ Syncope
- ◆ Subdural hematoma

^a Post-Acute and Long-Term Care Medical Association. Acute Change of Condition in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

STEP 5 ▼ ACUTE STROKE

Decide whether it would be appropriate to transfer the patient to the hospital for further evaluation and treatment. Not all patients experiencing an acute stroke are appropriate candidates for transfer. Hospital transfers for LTC patients with an acute stroke may produce both benefits and risks (Table 4). The practitioner, family or surrogate decision maker, and the patient (if possible) should be involved in deciding whether it is appropriate to transfer the patient to a hospital. The following factors should be taken into consideration:

- ◆ The patient's most recent level of function, including the status of any chronic illnesses;
- ◆ The patient's and family's treatment goals;
- ◆ The patient's wishes, as expressed in advance directives and other care-planning documents or oral discussions;
- ◆ The likelihood of benefit from hospital evaluation and treatment;
- ◆ The availability of emergency services and hospital services to respond in a timely fashion to a brain attack; and
- ◆ The facility's ability to provide timely evaluation along with pertinent management and monitoring for an acute stroke.

Patients with advanced illness may have already chosen to pursue palliative rather than curative care. Other patients who may not be appropriate candidates for hospital transfer include those who have such severe physical, cognitive, or psychosocial disabilities that they are unlikely to benefit from aggressive hospital evaluation, treatment, and monitoring. The facility should have policies and procedures in place for making transfer decisions when the patient's wishes are unknown or unclear. (See PALTmed's clinical practice guideline on acute change of condition.^b)

TABLE 4

Potential Benefits and Risks of Hospital Transfers for Long-Term Care Stroke Patients

Benefits

- ◆ Rapid assessment for conditions that resemble stroke
- ◆ Rapid access to certain diagnostic tests (e.g., computed tomography, magnetic resonance imaging)
- ◆ Rapid stabilization of cardiac, pulmonary, and neurological status
- ◆ Access to aggressive treatment to reverse stroke damage (e.g., thrombolytic ["clot-busting"] medications)
- ◆ Evaluation by teams with expertise in stroke care
- ◆ Access to hospital-based acute stroke units
- ◆ Frequent monitoring for neurological deterioration in the first days after stroke
- ◆ Comprehensive care planning for prevention and rehabilitation following stroke
- ◆ High-intensity rehabilitation services

Risks

- ◆ Deconditioning
- ◆ Delirium
- ◆ Pressure ulcers
- ◆ Use of restraints
- ◆ Use of indwelling urinary catheters
- ◆ Inappropriate medications for frail elderly people, causing adverse drug effects
- ◆ Other adverse iatrogenic events

^b Post-Acute and Long-Term Care Medical Association. Acute Change of Condition in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

STEP 6 ▼ ACUTE STROKE

Perform a diagnostic evaluation for acute stroke. The diagnostic evaluation may take place at the hospital (if the patient has been transferred) or at the LTC facility (if it has been decided not to transfer the patient).

Patients who are not transferred to the hospital for urgent evaluation and treatment may still benefit from selected elements of a diagnostic evaluation conducted in the facility over a period of days or weeks. The same factors for determining the appropriateness of a hospital transfer (see Step 5) may also help caregivers to make decisions about the appropriateness of a complete diagnostic evaluation of the patient with a possible acute stroke. A decision to forgo elements of the comprehensive diagnostic evaluation should be documented in the patient's record in the same way as the decision to forgo a hospital transfer.

Purposes of the diagnostic evaluation include

- ◆ Confirming that the patient's signs and symptoms are caused by a stroke and not by a condition that causes symptoms resembling those of a stroke (see Table 4),
- ◆ Identifying the type of stroke (e.g., thrombotic, embolic, hemorrhagic),
- ◆ Documenting the location and extent of the brain injury,
- ◆ Documenting the functional severity of the stroke,
- ◆ Recognizing conditions that increase the risk of stroke complications,
- ◆ Assessing relevant medical conditions and medications that affect acute stroke treatment and stroke rehabilitation, and
- ◆ Identifying potentially modifiable stroke risk factors.

Table 5 lists recommended and optional diagnostic tests for the initial evaluation of patients presenting to a hospital with stroke signs and symptoms.

TABLE 5

Diagnostic Tests for the Initial Hospital-Based Evaluation of Stroke Signs and Symptoms

Recommended

- ◆ Noncontrast computed tomography (CT) scan of the brain, or magnetic resonance imaging (MRI) of the brain
- ◆ Blood glucose
- ◆ Oxygen saturation (pulse oximetry)
- ◆ Electrocardiogram
- ◆ Blood urea nitrogen (BUN), serum creatinine, electrolytes
- ◆ Markers of cardiac ischemia
- ◆ Complete blood count, including platelets
- ◆ Prothrombin time (PT) and partial thromboplastin time (PTT)

Optional (depending on signs and symptoms and clinical suspicion)

- ◆ Liver function tests
- ◆ Serum ammonia level
- ◆ Blood alcohol level
- ◆ Toxicology screen
- ◆ Arterial blood gas
- ◆ Chest X-ray
- ◆ Electroencephalogram
- ◆ Lumbar puncture

Source: Adams et al, 2007.²⁶

STEP 7 ▼ ACUTE STROKE ● POST STROKE

Perform an interdisciplinary functional assessment. If the diagnostic evaluation confirms the occurrence of a stroke or TIA, perform a broad interdisciplinary assessment of the patient. Transitions from one level of care to another or from one care facility to another should trigger an interdisciplinary assessment. (See PALTmed’s clinical practice guideline on transitions of care.^c) The findings of this assessment should guide decision making about further diagnostic testing, treatment, rehabilitation, prevention, and monitoring (Table 6). The overall goals of a treatment plan based on such an assessment include optimizing the patient’s overall medical status, preserving and improving function, preventing stroke complications, and reducing the risk of recurrent stroke or other vascular events.¹⁶ The assessment should characterize the patient’s

- ◆ Cognitive and psychosocial abilities and impairments, including safety awareness;
- ◆ Physical abilities and impairments, including ability to perform ADLs;
- ◆ Current and expected level of physical endurance;
- ◆ Presence and severity of chronic medical conditions;
- ◆ Risk of stroke complications (e.g., bladder and bowel dysfunction, deep vein thrombosis [DVT], dysphagia, falls, and pressure ulcer); and
- ◆ Presence of stroke complications (e.g., urinary tract infection [UTI], DVT, aspiration, malnutrition, pain, depression, dementia, and skin breakdown).

Complications are common following a stroke. One study found that up to 60% of patients suffered medical complications in the first weeks after a stroke. The most frequent complications were UTI, musculoskeletal pain, and depression.²⁷

The interdisciplinary team should assess the patient for the presence of stroke complications and for factors that increase the risk of complications. The patient’s care plan should include measures to both prevent complications and treat any identified complications. (See Step 10 and Table 8.)

◆ Assess bladder and bowel function.

The prevalence of acute urinary incontinence and bladder dysfunction after a stroke ranges from 32% to 79%. Risk factors for incontinence after a stroke include age over 75 years, cognitive deficits, communication difficulties, disorders of bladder function, dysphagia, immobility from weakness or paralysis, and visual field defects. Incontinence persists in up to 25% of patients and is associated with high rates of institutional care and death.²⁸⁻³⁰

Disorders of bladder function may have multiple causes. It is important to evaluate the patient with incontinence following a stroke to identify modifiable causes of incontinence. Clinicians should assess for urinary retention by using a post-voiding bladder scan or in-and-out catheterization, measure urinary frequency and urinary volume, determine the level of urinary control, and search for signs or symptoms of UTI.¹⁶

Patients with urinary incontinence are often managed initially with indwelling catheters. Catheters increase the occurrence of UTIs, however, and may interfere with rehabilitation and mobility, so the interdisciplinary team should regularly determine whether discontinuation is possible. For further guidance on the assessment and treatment of urinary incontinence in the post-stroke patient, refer to AMDA’s clinical practice guideline on urinary incontinence.^d

Constipation and fecal incontinence also are common after a stroke. Constipation may be caused by immobility, decreased fluid intake, or adverse drug reactions such as those related to the use of

^c Post-Acute and Long-Term Care Medical Association. Transitions of Care in the Long-Term Care Continuum. Clinical Practice Guideline. Columbia, MD.

^d Post-Acute and Long-Term Care Medical Association. Urinary Incontinence in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

TABLE 6
Elements of a Comprehensive Assessment for a Stroke Patient

Domain	Indicators
Cognitive abilities	<ul style="list-style-type: none"> ◆ Memory ◆ Problem solving ◆ Safety awareness ◆ Interpersonal socialization ◆ New or progressive cognitive impairment (dementia)
Communication abilities	<ul style="list-style-type: none"> ◆ Auditory comprehension ◆ Spoken expression ◆ Reading and writing ability
Mood	<ul style="list-style-type: none"> ◆ Depression, anxiety
Hydration and nutritional status	<ul style="list-style-type: none"> ◆ Ability to consume fluid, calorie, and protein requirements
Feeding, chewing, and swallowing abilities	<ul style="list-style-type: none"> ◆ Delayed or difficult feeding ◆ Chewing or swallowing disturbances ◆ Dysphagia ◆ Aspiration
Functional abilities (i.e., activities of daily living)	<ul style="list-style-type: none"> ◆ Oral and facial hygiene ◆ Grooming ◆ Dressing (upper and lower body) ◆ Toileting and toilet hygiene ◆ Urinary and fecal continence <ul style="list-style-type: none"> • Bladder management • Bowel management ◆ Bathing ◆ Transfers <ul style="list-style-type: none"> • Toilet, tub, and shower transfers • Transfers between level surfaces • Transfers between uneven surfaces ◆ Ambulation <ul style="list-style-type: none"> • Wheelchair propulsion • Stair climbing
Physical condition	<ul style="list-style-type: none"> ◆ Stamina and endurance ◆ Motor or sensory deficits ◆ Painful conditions ◆ Fall risk ◆ Skin integrity
Status of chronic illnesses that can affect function and rehabilitation	<ul style="list-style-type: none"> ◆ Cardiac, pulmonary, musculoskeletal, neurological, psychological, metabolic, or malignant conditions
Presence of stroke complications	<ul style="list-style-type: none"> ◆ Deep vein thrombosis ◆ Infection (e.g., aspiration pneumonia, urinary tract infection) ◆ Skin ulcer ◆ Depression ◆ Musculoskeletal pain or dysfunction

anticholinergic medications. Fecal incontinence occurs in up to 40% of patients following an acute stroke and persists at 6 months in up to 20% of stroke survivors. Fecal incontinence is more likely to persist in patients with urinary incontinence, sensory or visual neglect, or dependence in toileting. Anticholinergic medications may predispose to fecal impaction and urinary overflow incontinence. One study showed that an algorithmic approach to post-stroke constipation or fecal incontinence could improve symptoms of bowel dysfunction.³¹ Fecal incontinence that persists for more than 3 months after a stroke is associated with an increased risk of LTC placement and death.³²

◆ **Assess cognitive status.**

It is important to assess cognitive status in stroke survivors because stroke can cause or exacerbate cognitive decline and dementia. Approximately 10% of stroke survivors have dementia before their first stroke, and 10% will develop new dementia as a result of their first stroke.³³ At least 30% of stroke survivors will fulfill the diagnostic criteria for dementia, and an even greater proportion will have less severe cognitive impairments.^{28,34,35} Post-stroke dementia is more common in people aged 65 years or more and in those with a lower level of education, a history of stroke, and greater cognitive and physical impairment at the time of the acute stroke.^{36,37} Even among stroke survivors in whom no dementia is found during the first 3 months after the stroke, dementia continues to occur at an average rate of 8% to 9% per year of follow-up.^{38,39} Stroke recurrence is an important determinant of incident or worsening dementia among stroke survivors, which highlights the importance of risk factor reduction to reduce recurrent stroke.^{33,40}

Cognitive decline following a stroke increases with advancing age. More than 70% of post-stroke patients aged over 75 years showed some cognitive impairment, compared with 45% of those aged 65 years or less.³⁴ Despite the high incidence and progressive increase in the prevalence of dementia following a stroke, some improvement in global cognitive function can be seen in up to 50% of stroke patients.³⁸ Treatment with donepezil can maintain or improve cognitive function in patients with vascular dementia.⁴¹

Post-stroke dementia may adversely affect rehabilitation, independence in ADLs, and risk of injury. In addition, death is three times as likely among stroke survivors who have dementia as among those who do not have dementia.³⁹ The method of cognitive assessment will depend on the abilities of the patient and the experience of the interdisciplinary team. A useful tool is the Brief Interview for Mental Status (BIMS) as incorporated in the MDS (MDS 3.0).

◆ **Assess chronic comorbid conditions.**

The practitioner should review all medical and psychological conditions that can affect the patient's cognition, physical function, and physical endurance. Comorbid medical conditions are common among stroke survivors aged 65 and older. Among one Medicare cohort of stroke survivors, 74% had hypertension, 38% had cardiac arrhythmias, 30% had diabetes, 23% had congestive heart failure, 23% had dementia, and 23% had fluid or electrolyte abnormalities.⁴² Concentrate on optimizing the treatment of comorbid medical conditions, controlling disabling symptoms, and minimizing the adverse effects of medications.

Assessment for previously unidentified coronary artery disease and peripheral arterial disease is important because the conditions are prevalent and are leading causes of death among stroke survivors.

◆ **Assess communication abilities.**

Between 17% and 38% of stroke patients experience a sudden decline in speech and language abilities.⁴³ Speech therapy by a speech-language pathologist produces important gains in communication abilities and should begin as soon as possible after stroke occurrence.¹⁶ For patients with communication deficits, a speech-language assessment should include a comparison of pre-stroke and post-stroke communicative ability. Pre-stroke abilities may be estimated anecdotally by asking caregivers and family members how well the patient was able to communicate before the stroke.

A complete speech and language assessment will assess

- ◆ Auditory comprehension (including auditory attention and memory),
- ◆ Visual comprehension (including visual attention and memory),
- ◆ Oral motor structure and function (including palate and voice),
- ◆ Verbal expression (including thought organization and reasoning), and
- ◆ Written expression (including thought organization and reasoning).

◆ **Assess risk for deep vein thrombosis.**

The most important stroke-specific risk factor for DVT or pulmonary embolism (PE) is reduced mobility, characterized by inability to ambulate independently. In studies of DVT after stroke, the number of clinically evident DVTs ranged from 0.2% to 10%, but the number of asymptomatic DVTs detected by ultrasound, fibrinogen scanning, or MRI may reach 80% among those with a paralyzed limb and with increasing stroke severity.^{44,45} DVT is most likely to occur in a paralyzed limb and is often asymptomatic, especially when it occurs below the knee. Symptomatic DVT of an immobile limb may present with increased circumference, edema, redness, warmth, or pain.

The most critical complication of DVT is PE. PE may present with painful breathing, shortness of breath, anxiety, change in mental status, hypoxia, hypotension, rapid respiration, or rapid heart rate, but half of fatal PEs present with sudden death.⁴⁶ PE can occur at any time after a stroke, but most will occur in the first month.⁴⁶ In the first months after a stroke, PE is responsible for 13% to 25% of early deaths,^{18,47} despite a relatively low incidence (1% to 5%) of PE in this population.^{44,48}

◆ **Assess risk for depression.**

Studies across multiple settings show that one in three stroke survivors suffers from depression.⁴⁹ A recent study of stroke survivors aged 70 and older found that the frequency of depression was not influenced by age, gender, or hemisphere affected.⁵⁰ Mortality is two to three times higher in patients with post-stroke depression than in stroke patients who are not depressed.⁵¹

Although the physical and cognitive effects of a stroke might be expected to contribute to an overestimation of the prevalence of depression, the criteria for depression of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*,⁵² apply equally to stroke patients.⁵¹

The most prevalent symptoms in post-stroke depression are anxiety-related and vegetative symptoms (e.g., weight loss, lack of energy, poor sleep, loss of libido). Compared with depressed patients who have not had a stroke, patients with post-stroke depression are more likely to show emotional instability and variation in mood throughout the day and are less likely to experience guilt and suicidal ideation.⁵³

Other conditions may present with signs and symptoms that mimic those associated with depression. For that reason, it is important to assess the post-stroke patient who has depressive

symptoms for other causes of those symptoms. Appropriate screening tools for depression include the 10-item Geriatric Depression Scale (GDS), the Cornell Scale for Depression in Dementia (CSDD), and the Patient Health Questionnaire 9 (PHQ-9), the last of which is included in MDS 3.0. (See PALMed's clinical practice guideline on depression.^e)

◆ **Assess risk for dysphagia and aspiration.**

Safe and effective swallowing is essential for adequate hydration and nutrition. **Dysphagia** is defined as difficulty with or discomfort during swallowing. Dysphagia can result in aspiration, a condition associated with saliva, liquids, or food spilling from the pharynx into the lungs. Dysphagia may also contribute to malnutrition, which adversely affects stroke recovery. Dysphagia occurs in an estimated 22% to 65% of patients following an acute stroke.^{54,55} Up to 25% of stroke patients in LTC may have dysphagia.⁵⁶

Many medications, as well as conditions other than stroke, can cause swallowing problems.^{57,58} Signs and symptoms of a possible swallowing problem are listed in Table 7. The physician should evaluate and rule out medical causes before, or in conjunction with, evaluation by other disciplines. Because aspiration after a stroke can be asymptomatic,⁵⁹ it is prudent to perform an assessment for dysphagia when a stroke patient is identified or transferred to a facility. In one study, 11% of patients with post-stroke dysphagia were not identified in the acute-care hospital but were discovered by formal assessment after transfer to a rehabilitation unit.⁶⁰

For the patient with an acute stroke, a screening clinical evaluation of swallowing may be the appropriate initial assessment for dysphagia. This evaluation should be performed by an appropriately trained practitioner, nurse, or speech therapist and should document the patient's

- ◆ Level of consciousness;
- ◆ Ability to follow commands;
- ◆ Gross strength and coordination of muscles of the face, mouth, and tongue; and
- ◆ Ability to swallow 3 ounces of water without drooling, coughing, or choking.

Any abnormalities detected in the screening clinical examination may prompt consideration of a formal dysphagia assessment by a speech therapist. If a patient with an acute stroke does not cough when swallowing 3 ounces of water, the likelihood that a formal videofluoroscopic examination will find even mild aspiration is only 20%.⁶¹

A formal dysphagia assessment by a speech therapist may include as many of the following components as indicated:

- ◆ Comparison of pre- and post-stroke swallowing status;
- ◆ Prognosis for oral feeding;
- ◆ Oral motor structure and function (including palate and voice) at rest and with volitional movement;
- ◆ Oral stage of swallowing, including management of own secretions as well as performance with food and drink of different bolus sizes and consistency;
- ◆ Pharyngeal stage of swallowing, including dry swallow upon command as well as with presentations of food and drink of different bolus sizes and consistency; and
- ◆ Impact of postural and other therapeutic techniques on swallowing function.

Factors that are identified during the initial swallowing evaluation or that become apparent dur-

^e Post-Acute and Long-Term Care Medical Association. Depression in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

ing the course of treatment may lead to a recommendation for an instrumental assessment such as

- ◆ Modified barium swallow (videofluoroscopic examination),
- ◆ Fiberoptic endoscopic evaluation of swallowing, or
- ◆ Flexible endoscopic evaluation of swallowing with sensory testing.

Observational evidence suggests that programs designed to effectively diagnose and treat aspiration following acute stroke can reduce rates of aspiration pneumonia in the acute-care setting.⁵⁹ It is important to remember that in addition to abnormal swallowing, age over 65 years, dysarthria, aphasia, increased stroke severity, and cognitive impairment are independent predictors of post-stroke pneumonia, and that these risk factors cumulatively increase pneumonia risk.⁶² Nevertheless, most stroke experts endorse the assessment and treatment of patients who have acute stroke-related swallowing abnormalities.^{16,59} Instrumental evaluation and treatment of dysphagia and aspiration may not be appropriate for all patients, however. It is important to decide whether to perform an instrumental assessment in discussion with the patient, family or surrogate decision maker, and relevant interdisciplinary team members. Key elements of the decision-making process should be documented. The documentation should reflect the patient's treatment goals, which should be based on his or her understanding of the likely diagnosis; the prognosis for recovery of function; the expected likelihood of complications from dysphagia or aspiration; and a review of the benefits, risks, and alternatives to instrumental assessment and therapeutic interventions.

◆ **Assess limbs for range of motion, motor and sensory deficits, and painful conditions.**

Up to 80% of stroke patients have weakness in a limb (i.e., hemiparesis).⁶³ Limb weakness can lead to deconditioning, loss of function, spasticity, pain, and DVT. Limb mobility should be assessed and optimized as soon as possible after an acute stroke. If voluntary motion is not possible, the care team should focus on trying to preserve passive range of motion and increase strength.

Complications of limb immobility can present in several ways. Patients may complain of musculoskeletal pain with motion or ADLs, show a decline in physical abilities, develop increased tone or contractures, or exhibit swelling or skin discoloration in a limb. Any of these signs or symptoms should prompt the interdisciplinary team to consider further evaluation to determine the contributing causes. A painful limb may have a wide variety of causes other than the acute stroke (e.g., arthritis, gout, neuropathy, fracture). (See PALTmed's clinical practice guideline on pain management in the LTC setting.^f)

Two causes of pain associated with hemiplegic stroke—limb spasticity and shoulder dysfunction—are important because they can have significant detrimental effects and because preventive treatments may reduce their occurrence or severity.

Spasticity. Immediately after a hemiplegic stroke, the patient may experience a period of limb weakness and flaccidity characterized by clumsiness, lack of tone, and lack of movement. As the brain recovers from a stroke, the flaccid limb may recover muscle tone. **Spasticity** is the occurrence of abnormally increased muscle tone with exaggerated reflexes and increased resistance to passive movement. If spasticity is severe, the patient may experience pain with range of motion, involuntary muscle contractions, or painful muscle spasms.

Spasticity in an upper extremity may present with a loss of finger dexterity, a fistled hand, a flexed wrist, a flexed elbow with the palm held up, or an adducted shoulder. Spasticity in the leg may

^f Post-Acute and Long-Term Care Medical Association. Pain Management in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

TABLE 7

Signs and Symptoms of a Possible Swallowing Problem

- ◆ “Wet” or “gurgly” voice
- ◆ Weak cough
- ◆ Noisy breathing or chest congestion
- ◆ Drooling
- ◆ Coughing or clearing throat when eating or drinking
- ◆ Slow eating
- ◆ Decreased food consumption
- ◆ Stopping eating because of fatigue; signs of excessive effort involved in eating
- ◆ Pocketing food in mouth
- ◆ Difficulty keeping food or liquid in mouth
- ◆ Difficulty chewing
- ◆ Frequent episodes of pneumonia
- ◆ Difficulty swallowing pills
- ◆ Complaints of food sticking in the throat

present with an extended knee, hip adduction, a foot pointed and turned inward, limited range of motion, or difficulty with transfers and ambulation.

Problems that can result from spasticity include pain, joint contractures, difficulty performing ADLs, and difficulty with hygiene as a result of contracted postures. Spasticity may be increased by conditions such as UTI, constipation, pressure ulcers, or braces that cause discomfort.⁶⁴

Shoulder dysfunction. Because shoulder weakness and dysfunction may have multiple causes, it is prudent for the physical therapist and other members of the interdisciplinary team to assess shoulder function and create care plans to prevent shoulder dysfunction.⁶⁵ Because shoulder dysfunction may occur during recovery of muscle tone and voluntary movement, the team should be vigilant for the new onset of pain or dysfunction in the shoulder and arm.

◆ **Assess nutrition and hydration status.**

Optimal nutrition and hydration are important for patients undergoing stroke rehabilitation. Up to 80% of stroke survivors who reside in LTC facilities need some feeding assistance, and 30% are described as having poor food intake or appetite.⁵⁶

Challenges to adequate nutrition and hydration in stroke survivors include difficulty maintaining proper body position; dependency in eating that prolongs eating time; and behaviors such as drowsiness, distractibility, and resistance to feeding. For further guidance on ensuring adequate nutrition and hydration in the post-stroke patient, refer to PALTmed’s clinical practice guideline on altered nutritional status.⁸

⁹ Post-Acute and Long-Term Care Medical Association. Altered Nutritional Status in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.



◆ **Assess skin integrity and risk for pressure ulcers.**

Pressure ulcers are another common complication of stroke. Stroke-related risk factors for skin breakdown include

- ◆ Paralysis and immobility,
 - ◆ Decreased level of consciousness or comatose state,
 - ◆ Loss of sensation,
 - ◆ Muscle spasticity,
 - ◆ Bowel or bladder incontinence, and
- ◆ Malnutrition (related to dysphagia).

A thorough assessment may identify other risk factors for pressure ulcers. (See PALTmed’s clinical practice guideline on pressure ulcers^h and the companion volume on pressure ulcer therapy.ⁱ)

◆ **Assess risk for falls and fractures.**

A stroke can result in multiple risk factors for falls, predisposing the patient to additional injury. Stroke-related risks for falling include impairments in

- ◆ Ability to communicate;
 - ◆ Alertness and attention;
 - ◆ Balance and coordination;
 - ◆ Muscle strength;
 - ◆ Peripheral sensation;
 - ◆ Proprioception;
 - ◆ Safety awareness, including lack of awareness of the presence of paralysis or functional limitations (anosognosia); and
- ◆ Visual field.

For additional risk factors for falls, refer to PALTmed’s clinical practice guideline on falls and fall risk.^j Despite a lack of clear data to confirm efficacy in LTC settings, a reasonable approach to reducing fall risk includes an interdisciplinary assessment and multifactorial interventions. For instance, prudent medication management; therapy to improve strength, balance, and mobility; and care to optimize other chronic medical conditions reduce fall rates in hospitalized patients, so they may reduce the fall risk for individual LTC patients.⁶⁶ The only evidence-based intervention shown to reduce falls after stroke is vitamin D supplementation.⁶⁷

Falls can result in fractures, especially in patients with osteoporosis. Because many LTC patients have osteoporosis and because stroke-related immobility can accelerate bone loss, consider appropriate dietary and pharmacologic measures to prevent or treat osteoporosis. (See PALTmed’s clinical practice guideline on osteoporosis.^k)

STEP 8 ▼ ACUTE STROKE ● POST STROKE

Summarize the patient’s condition. When a patient is admitted to the facility with a history of stroke, it is appropriate to review all relevant information that has been identified thus far, which may include the following:

^h Post-Acute and Long-Term Care Medical Association. Pressure Ulcers in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

ⁱ Post-Acute and Long-Term Care Medical Association. Pressure Ulcer Therapy Companion. Columbia, MD.

^j Post-Acute and Long-Term Care Medical Association. Falls and Fall Risk in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

^k Post-Acute and Long-Term Care Medical Association. Osteoporosis and Fracture Prevention in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

- ◆ The severity of the stroke (NIH stroke scale score less than 6 associated with good recovery, greater than 16 associated with severe disability and high mortality)⁶⁸;
- ◆ The type of stroke (e.g., large-artery atherosclerosis, small-artery vascular occlusion, cardioembolic, hemorrhagic, TIA, unknown);
- ◆ The patient's cognitive abilities, including communication ability, decision capacity, and safety awareness;
- ◆ The patient's nutritional status and findings of a recent nutritional assessment;
- ◆ The patient's physical abilities, particularly the ability to perform ADLs;
- ◆ Stroke complications (e.g., dysphagia, aspiration, incontinence, paralysis, skin breakdown);
- ◆ Mood and behavioral problems;
- ◆ Severity and prognosis of comorbid conditions (e.g., coronary artery disease, congestive heart failure, diabetes, dysphagia, chronic obstructive pulmonary disease, dementia);
- ◆ Modifiable risk factors for recurrent stroke;
- ◆ Nonpharmacologic and pharmacologic treatments prescribed to enhance rehabilitation, relieve symptoms, prevent or treat stroke complications, and reduce the risk of stroke recurrence;
- ◆ An estimation of the patient's prognosis for recovery, development of stroke complications, or further decline in function;
- ◆ Documentation of any decisions to forgo diagnostic testing, rehabilitation, or preventive therapy; and
- ◆ Advance care plans, including the patient's goals of therapy and preferences for life-sustaining treatments.

TREATMENT

STEP 9 ▼ ACUTE STROKE

Treat medical conditions that may mimic an acute stroke. If the assessment identifies an acute medical condition such as hypoglycemia, hypotension, or hypoxia, treat that condition immediately. If this treatment begins to reverse the patient's neurological symptoms within 20 minutes, nursing staff should notify the attending physician and discuss the need for further evaluation or treatment. *If symptoms suggestive of a stroke persist after 20 minutes, go back to Step 5.*

The initial evaluation may reveal findings that can complicate neurological injury or can cause symptoms of a stroke. For example, blood glucose greater than 400 mg/dL, systolic blood pressure greater than 220 mm Hg, diastolic blood pressure greater than 110 mm Hg, systemic infection, and fluid or electrolyte disturbances can cause symptoms similar to stroke. If these findings exist, notify the attending physician to discuss the appropriateness of further evaluation and treatment. (See AMDA's clinical practice guideline on acute change of condition.¹)

STEP 10 ▼ ACUTE STROKE ● POST STROKE

Develop and implement a care plan to identify and address stroke-related complications. Table 8 lists several common stroke complications and some preventive interventions that may be considered as part of a plan to prevent stroke-related complications.

¹ Post-Acute and Long-Term Care Medical Association. Acute Change of Condition in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

TABLE 8
Strategies to Decrease the Risk of Stroke Complications

Complication	Potential Preventive Interventions
Pneumonia	<ul style="list-style-type: none"> ◆ Optimize mobility ◆ Optimize cardiac and pulmonary conditions ◆ If significant dysphagia identified, modify diet and liquid consistency ◆ Rehabilitate swallowing ◆ Optimize body position when eating or drinking ◆ Consider pneumococcal immunization ◆ Consider influenza immunization ◆ Optimize oral hygiene
Urinary tract infection	<ul style="list-style-type: none"> ◆ Optimize mobility as soon as possible ◆ Treat modifiable causes of incontinence ◆ Identify and treat urinary retention ◆ Remove indwelling catheter
Deep vein thrombosis	<ul style="list-style-type: none"> ◆ Optimize mobility ◆ Consider prophylactic anticoagulation therapy
Pressure ulcer	<ul style="list-style-type: none"> ◆ Optimize mobility ◆ Minimize pressure, shear, moisture ◆ Optimize nutrition
Depression	<ul style="list-style-type: none"> ◆ Optimize function ◆ Treat pain ◆ Provide emotional support and appropriate activities
Spasticity or contracture	<ul style="list-style-type: none"> ◆ Ensure stretching and range-of-motion exercises ◆ Apply splinting ◆ Consider antispasmodic medication
Shoulder displacement	<ul style="list-style-type: none"> ◆ Provide limb support ◆ Transfer carefully ◆ Rehabilitate strength

◆ **Address risk for pneumonia.**

Some medications and comorbid conditions (e.g., gastroesophageal reflux, absence of gastric acid, poor dentition) can increase the risk of aspiration pneumonia. It may be prudent to review medications and optimize oral health in an effort to control the risk of aspiration pneumonia.⁶⁹ Pneumococcal and influenza immunizations may provide additional benefit by preventing specific types of acute pneumonia, thereby optimizing pulmonary status.

◆ **Address risk for deep vein thrombosis.**

The rate of DVT after stroke is similar to the rate seen after major surgery,⁷⁰ and most studies show higher rates of DVT with increasing stroke severity and immobility. Because DVT is frequently asymptomatic, and because up to half of all fatal PEs lack a symptomatic prodrome,⁴⁶ it is prudent to implement prophylactic treatment for several weeks rather than to monitor to detect symptomatic DVT or PE in at-risk patients. Treatment options include intermittent pneumatic compression

devices, low-dose unfractionated heparin (LDUH), low molecular weight heparin (LMWH), and warfarin.^{16,71}

For patients with restricted mobility (e.g., those unable to walk unassisted, unable to ambulate 50 feet, or with dense paresis of a leg), clinicians should prescribe either LDUH or LMWH.⁷¹ LDUH can reduce the incidence of PE to 0.5%.⁷² A meta-analysis of thromboembolism prophylaxis after stroke estimated the number needed to treat (NNT) for prevention of one thromboembolic event. For LDUH (5000 units 2 to 3 times daily), the NNT ranged from 2 to 10, whereas for LMWH the NNT ranged from 1 to 4.⁷³

The PREVAIL study compared 10 days of prophylactic therapy with the LMWH enoxaparin to LDUH in stroke patients unable to walk unassisted.⁷⁴ The main outcomes included both asymptomatic and symptomatic venous thromboembolism (VTE; DVT plus PE), PE, and DVT, as well as intracranial and extracranial bleeding. The two regimens were equally efficacious at preventing symptomatic VTE, and death rates were similar in the two groups. Enoxaparin treatment reduced the incidence of all VTE (symptomatic plus asymptomatic) to 10%, whereas LDUH reduced all VTE to 18% (NNT = 13). Intracranial hemorrhage was markedly higher among patients with an NIH stroke scale score of 14 or higher (12.5%) than among those whose scores were less than 14 (6%), but intracranial hemorrhage rates were similar in the two treatment groups. These data suggest that LDUH reduces symptomatic VTE as well as enoxaparin with similar rates of bleeding complications.⁷⁴

For patients at high risk for hemorrhage because of anticoagulation (e.g., platelet count less than 100,000, uncontrolled hypertension [greater than 180/100], creatinine clearance less than 30 ml/min, or recent trauma or surgery), clinicians may consider physical methods of VTE prevention. Unfortunately, meta-analysis of trial evidence does not show a statistical benefit from either graduated compression stockings or intermittent pneumatic compression devices.⁷⁵ A recent trial compared thigh-length stockings to below-knee stockings for DVT prevention in patients unable to walk independently to the bathroom after stroke.⁷⁶ The study did not show a reduction in VTE with thigh-high stockings, but did find a higher risk for DVT with below-knee compression stockings. This may suggest that clinicians should avoid compressive stockings, especially below-knee stockings, for immobile stroke patients.

The optimum duration of VTE prophylaxis is unknown. Fatal DVT and PE continue to occur for up to 120 days after a stroke,⁴⁶ but the incidence is highest in the first month.⁷⁷ No evidence exists to support long-term anticoagulation in patients who are likely to be immobile indefinitely.

◆ **Address risk for pressure ulcers.**

Although it is debatable whether all pressure ulcers are preventable,⁷⁸ it is prudent to consider modifying pressure ulcer risk factors that are identified by a careful clinical assessment. (See PALMed's clinical practice guideline on pressure ulcers^m and the companion volume on pressure ulcer therapy.ⁿ)

◆ **Address risk for shoulder displacement and pain.**

Nearly one third of patients have shoulder pain in the year following a stroke.⁷⁹ Adverse effects of shoulder pain include persistent difficulties with ADLs, impaired rehabilitation, and decreased quality of life.⁷⁹ The exact cause of shoulder pain may be difficult to determine. MRI studies of painful shoulders after a stroke may show rotator cuff tears, tendonopathies, and atrophy, but these

^m Post-Acute and Long-Term Care Medical Association. Pressure Ulcers in the Long-Term Care Setting. Clinical Practice Guideline.

Columbia, MD. ⁿ Post-Acute and Long-Term Care Medical Association. Pressure Ulcer Therapy Companion. Columbia, MD.

abnormalities do not always correlate with pain severity.⁸⁰ Subluxation of the shoulder is believed to contribute to shoulder pain and disability. Despite the common use of slings and other supportive devices to protect the weak post-stroke shoulder, no data exist to confirm that these practices decrease pain or improve function.⁸¹

Absent compelling trial data to guide prevention and treatment, prudent practice would include an interdisciplinary assessment and individualized treatment plans with regular assessment of efficacy. On the basis of each individual's responses, treatments that reduce pain and improve function should continue, but ineffective or burdensome treatments should be discontinued. Examples of expert recommendations for the weak, painful shoulder include the use of staff education and the use of slings to prevent injury, corticosteroid injection when capsulitis or tendonitis exists, and trials of modalities such as ice, heat, massage, and range of motion exercises.¹⁶

Table 9 describes specific positioning and handling techniques to support a hemiparetic arm at rest and when moving the limb through the range of motion.

TABLE 9

Specific Positioning and Handling Techniques to Support a Hemiparetic Arm

◆ **Positioning for Support**

- ◆ Standing:
 - Sling or other device as recommended by physical or occupational therapist.
- ◆ Sitting:
 - Support arm under elbow with lap board, arm tray, or pillow.
- ◆ Lying:
 - When patient is lying on his or her side, place a pillow to support the arm.
 - When patient is lying supine, place a pillow or rolled towel under the arm.

Be aware that as a result of decreased sensory awareness, the patient may fail to notice if the affected arm falls from the chair or bed that is supporting it.

◆ **Correct Handling Techniques**

- ◆ Do not pull or tug on the arm during dressing, bathing, or transfers.
- ◆ Perform full pain-free range of motion exercises during dressing and bathing twice per day.

◆ **Address spasticity and contracture of a paretic limb.**

A 6-month follow-up study of ischemic stroke patients with limb paresis found that 15% had moderately severe spasticity and that, overall, 42% manifested some degree of spasticity.⁸² A Swedish study showed that, in the first year following a stroke, overall costs (primarily resulting from hospital care) for patients with spasticity are four times the costs for those without stroke-related spasticity.⁸³

◆ **Address risk for urinary tract infection.**

UTI risk can be reduced by avoiding indwelling urinary catheters and by minimizing or eliminating urinary retention. Noncatheterized patients with a history of recurrent UTIs may derive some modest benefit from 500 mg of cranberry extract or judicious use of prophylactic antibiotics.^{84,85} Although evidence does not support other interventions to reduce UTI occurrence, reasonable practice would include attention to perineal hygiene and avoidance of constipation.

STEP 11 ▼ ACUTE STROKE ● POST STROKE

Develop and implement an interdisciplinary treatment plan that treats stroke complications.

When the assessment identifies complications of stroke, implement appropriate curative, restorative, or palliative treatment on the basis of a shared decision that reflects the patient's wishes and treatment goals.

◆ **Treat deep vein thrombosis.**

For guidance on the treatment of DVT in the post-stroke patient, refer to the American College of Chest Physicians (ACCP) guidelines on antithrombotic therapy.⁸⁶

◆ **Treat depression.**

Post-stroke depression should be identified and treated as early as possible. Patients with post-stroke depression who are treated within the first month following an acute stroke have more rapid and more lasting recovery of ADL function.⁸⁷ Treating post-stroke depression may also decrease mortality.⁸⁸

Treating stroke-related depression with antidepressant medication improves recovery of cognitive and physical abilities and reduces mortality.⁵¹ (See AMDA's clinical practice guideline on depression.^o) Studies support the efficacy of nortriptyline, fluoxetine, and citalopram for the treatment of post-stroke depression, but it is unknown whether all antidepressants have similar beneficial effects.^{51,88,89} It is prudent to begin antidepressant treatment with medications that have been proven effective in high-quality research studies.^{51,87,89,90} Fluoxetine may be preferred because it is effective for treating not only post-stroke depression, but also the post-stroke syndromes of excessive or inappropriate laughing, crying, or both (emotional incontinence), and anger proneness.⁹¹

An evolving issue is the place for antidepressants for prevention of post-stroke depression and depression-related disability. A meta-analysis showed that fluoxetine reduced the incidence of post-stroke depression, improved neurological recovery, and improved independence.⁹² Another study showed that escitalopram reduced the incidence of post-stroke depression.⁹³

◆ **Treat dysphagia.**

Dysphagia caused by a stroke can lead to aspiration, which may increase the risk of aspiration pneumonia. Dysphagia may also result in decreased intake of food and fluids, which can lead to altered nutritional status and fluid and electrolyte imbalance. Most recommendations favoring dysphagia treatment following an acute stroke are based on observational studies that report a decreased incidence of aspiration pneumonia attributed to aspiration assessment and treatment.^{16,59} A recent trial showed that swallowing therapy with adjusted dietary consistencies reduced pneumonia, death, and institutionalization among dysphagic acute stroke patients treated in a hospital setting.⁹⁴

The main goals of dysphagia treatment after an acute stroke are to try to reduce the risk of aspiration and to optimize nutrition and hydration by improving the ability to chew and swallow. Treatment strategies to reduce aspiration risk include compensatory strategies that optimize posture during eating, speech therapy to improve strength and coordination of oropharyngeal muscles, and dietary modifications.⁵⁸

Unfortunately, no interventions have been conclusively proven to prevent aspiration pneumonia among stroke patients with dysphagia and aspiration.^{95,96} Most studies of dysphagia do not assign

^o Post-Acute and Long-Term Care Medical Association. Depression in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

patients to nontreatment or observation groups. Many studies fail to report on the crucial outcomes of aspiration pneumonia, malnutrition, functional status, and death. Additionally, any benefits attributed to early dysphagia treatment are confounded by the fact that swallowing may return to normal in up to 80% of patients within 1 month of an acute stroke.⁹⁷

Moreover, 60% to 80% of patients with dysphagia who are followed prospectively do not develop aspiration pneumonia. In other words, most patients who aspirate do not develop aspiration pneumonia.⁹⁸ This pattern suggests that aspiration is a necessary but insufficient risk factor for pneumonia. Co-occurring dependence in feeding, chronic lung disease, congestive heart failure, gastroesophageal reflux, and a bedbound state may be more important risk factors for aspiration pneumonia than aspiration alone.^{55,98}

Treatments that are based on the identification of swallowing abnormalities could plausibly reduce the frequency or severity of aspiration and thus decrease aspiration pneumonia. Most such treatments, however, have not been proven effective in clinical trials. For example, common compensatory strategies, such as chin-tuck, positioning, dietary modifications, and thickened liquids, may improve some symptoms or findings on videofluoroscopy, but these intermediate outcomes do not necessarily translate into reductions in the incidence of aspiration pneumonia. In addition, modified diets and thickened liquids have potential adverse effects, which include weight loss, dehydration, and reduced quality of life.

The data to support informed choices about the assessment and treatment of LTC patients with stroke-related dysphagia are even more problematic. Patients in LTC often suffer from multiple comorbid conditions that can contribute to pneumonia, dehydration, malnutrition, and functional decline. Patients and their families should be aware that no treatments have been proven effective at preventing aspiration pneumonia in frail elderly people in LTC.⁹⁵

This lack of high-quality data does not mean that assessment and treatment of stroke-related dysphagia in LTC has no benefit. Rather, it means that an informed choice by patients and families should include discussion of the limitations of current knowledge when considering the benefits and risks of evaluation and treatment of dysphagia.

◆ **Treat painful musculoskeletal conditions.**

Musculoskeletal pain is common after a hemiplegic stroke. Nonpharmacologic treatments for stroke-related musculoskeletal pain may include positioning, use of splints and supports, and passive range of motion exercises. The decision to treat pain with medication, as well as the choice of medication, should consider the causes of the pain, the patient's comorbid medical conditions, and risks for adverse medication effects. For further guidance on the pharmacologic treatment of pain, refer to AMDA's clinical practice guideline on pain management in the LTC setting.^P

Limb spasticity and shoulder pain are two common stroke-related pain syndromes. Treatment measures for stroke-related shoulder pain are the same as those for prevention (see *Shoulder dysfunction*, page 18.)

Treatment of spasticity. Not all spasticity is detrimental; a mild increase in tone may allow a patient to transfer or stand. The decision to treat spasticity depends on its severity and effect on domains such as ADLs, ease of care, hygiene, and contractures.⁶⁴ The goals of treating spasticity should be explicit and should be tailored to the patient's condition and treatment goals. Common approaches to spasticity include stretching, passive range of motion, and splinting.⁹⁹

^P Post-Acute and Long-Term Care Medical Association. Pain Management in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

Nonpharmacologic treatment options for spasticity include

- ◆ Minimizing noxious stimuli (e.g., infection, impaction, pain);
- ◆ Positioning to minimize contractures (e.g., use of pillows or towel rolls, application of splints);
- ◆ Stretching and range of motion exercises (slow, gentle, pain-free stretching); and
- ◆ Instruction in self-directed range of motion exercises, especially stretching of fingers, elbows, and shoulders.

Pharmacologic or surgical treatment of spasticity may be required if

- ◆ Pain is increasing,
- ◆ Contractures are developing,
- ◆ The patient's ability to perform hygiene practices and other ADLs is impaired, and
- ◆ The patient cannot be maintained in positioning devices.

The choice of pharmacologic agents (e.g., baclofen, dantrolene, tizanidine) to treat spasticity is based on patient characteristics and the adverse effects profiles of the relevant medications.⁶⁴ Although stroke patients may derive less benefit from pharmacologic treatment than patients with spasticity due to other disorders, a trial of therapy with attention to predetermined outcomes and adverse medication effects may be warranted.⁹⁹ Because these agents may be associated with somnolence, which may simulate clinical decline, they should be used at the lowest doses and for the shortest possible time.

Dantrolene works directly at the muscle and may be titrated weekly as needed, from an initial dose of 25 mg daily to a maximum of 100 mg 4 times daily. Periodic testing of liver enzymes is suggested to detect adverse hepatic effects.^{99,100} Baclofen acts centrally, affecting GABA-B receptors, and may be titrated every 2 to 3 days from an initial dose of 5 mg 3 times daily to a maximum of 15 mg 3 times daily. Lower doses should be used in the presence of renal dysfunction; therefore, periodic assessment of serum creatinine and estimated glomerular filtration rate is recommended.^{99,100} Tizanidine affects the central α -adrenoreceptors and may be titrated every 2 to 3 days from an initial dose of 2 to 4 mg daily to a maximum of 12 mg 3 times daily. Practitioners should monitor for sedation and hypotension before and after dose increases and should check creatinine and liver enzymes at baseline; 1, 3, and 6 months; and then periodically.^{99,100}

Patients with contractures or spasticity that cannot be controlled may benefit from referral to a rehabilitative medicine specialist for evaluation and treatment. These patients may be candidates for injections with botulinum toxin.

◆ **Treat altered nutrition and hydration.**

For guidance on the treatment of altered nutrition and hydration in the post-stroke patient, refer to PALTmed's clinical practice guidelines on altered nutritional status⁹ and dehydration and fluid maintenance.^f

◆ **Treat infections.**

For guidance on the treatment of infections in the post-stroke patient, refer to PALTmed's clinical practice guideline on managing infections in the LTC settings⁵ or to the Infectious Disease Society of America's guidelines on the treatment of infection in LTC facilities.¹⁰¹

⁹ Post-Acute and Long-Term Care Medical Association. Altered Nutritional Status in the Long-Term Care Setting. Clinical Practice Guideline.

Columbia, MD. ^f Post-Acute and Long-Term Care Medical Association. Dehydration and Fluid Maintenance in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

⁵ Post-Acute and Long-Term Care Medical Association. Managing Infections in the Long-Term Care Setting. Columbia, MD.

◆ **Treat pressure ulcers.**

Treatment of pressure ulcers should be guided by approaches to care that incorporate high-quality scientific evidence and expert opinion, such as PALTmed’s clinical practice guideline on pressure ulcers^t and the companion volume on pressure ulcer therapy.^u

STEP 12 ● POST STROKE

Develop and implement a rehabilitation plan to maximize function. After the patient with an acute stroke has been stabilized, the interdisciplinary team can determine the patient’s specific rehabilitation needs. Stroke rehabilitation may help the patient to optimize physical, cognitive, psychosocial, and vocational functioning (Table 10). It is important to develop the rehabilitation plan in collaboration with the patient and family and to individualize each patient’s rehabilitation regimen to reflect the patient’s prognosis, comorbid conditions, and personal goals.

Rehabilitation and restorative therapies may include

- ◆ Occupational therapy to enhance dexterity of the arms and hands;
- ◆ Physical therapy to improve motor strength and promote independence in ADLs;
- ◆ Speech therapy to optimize communication, chewing, and swallowing; and
- ◆ Restorative nursing programs; which could include
 - ◆ Range of motion (passive),
 - ◆ Range of motion (active),
 - ◆ Splint or brace assistance,
 - ◆ Bed mobility,
 - ◆ Transfer,
 - ◆ Walking,
 - ◆ Dressing and/or grooming,
 - ◆ Eating and/or swallowing,
 - ◆ Amputation/prosthesis care, and
 - ◆ Communication.

After completing a comprehensive evaluation, therapists should develop short-term and long-term goals for improving function or compensating for deficits. (See PALTmed’s toolkit on medical necessity for rehabilitative services.^v) Once active physical and occupational therapy have ended, the physical and occupational therapists should work with the restorative nursing staff to develop a plan to optimize the patient’s functional status.

Some patients may not be appropriate candidates for functional improvement or recovery through rehabilitation. For example, patients who have advanced comorbid illnesses or who cannot or will not participate in therapy may be appropriate candidates for palliative care rather than restorative therapy. If the patient, family, and care team determine that rehabilitation is not appropriate because of advanced illness or because improvement in function is unlikely, the decision should be documented in the patient’s medical record.

^t Post-Acute and Long-Term Care Medical Association. Pressure Ulcers in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

^u Post-Acute and Long-Term Care Medical Association. Pressure Ulcer Therapy Companion. Columbia, MD.

^v Post-Acute and Long-Term Care Medical Association. Medical Necessity for Rehabilitative Services Toolkit. Columbia, MD.

TABLE 10

Possible Goals of a Stroke Rehabilitation Plan

Physical Therapy

- ◆ Maintain flexibility of joints and strength of muscles
- ◆ Maintain functional range of motion
- ◆ Improve or maintain balance
- ◆ Improve or maintain gait/locomotion and gait stability
- ◆ Improve or maintain muscle performance (strength, power, and endurance)
- ◆ Improve or maintain quality of movement between and across body segments
- ◆ Improve or maintain highest level of independence and performance of ADLs
- ◆ Reduce risk of falls
- ◆ Develop restorative nursing programs with the plan to optimize or maintain the patient's functional status

Occupational Therapy

- ◆ Improve or maintain strength and range of motion of joints to maximize functional mobility and ADL performance
- ◆ Improve or maintain dining skills through the use of positioning/seating and assistive dining devices
- ◆ Improve functioning and safety through the use of environmental modifications (e.g., adaptive call bell switches, location of bed relative to bathroom, night lights)
- ◆ Promote and maintain safe mobility
- ◆ Use positioning and seating devices to reduce risk of pressure ulcers
- ◆ Develop restorative nursing programs with the plan to optimize or maintain the patient's functional status

Recreational Therapy

- ◆ Optimize ability to socialize according to patient's needs, desires, and interests
- ◆ Address or prevent depression by enabling patient to continue participation in preferred activities, hobbies, and interests
- ◆ Implement therapeutic chores that focus on self-esteem and make the patient feel useful

Speech Therapy

- ◆ Increase auditory and visual comprehension
- ◆ Improve oral motor skills for speech and swallowing
- ◆ Improve swallowing for safe and appropriate oral intake
- ◆ Improve spoken skills for communicating needs and ideas
- ◆ Increase language skills for memory, orientation, problem solving, and reasoning
- ◆ Develop restorative nursing programs with the plan to optimize or maintain the patient's functional status

ADLs: activities of daily living.

STEP 13 ● POST STROKE

Develop and implement a plan for preventing recurrent strokes. One in four strokes occur in patients with a prior stroke,¹³ and the annual risk of recurrent stroke is 7% to 10% per year.¹ Decisions about using interventions to prevent recurrent stroke should be based on the causes of the patient's previous stroke, an assessment of the patient's modifiable risk factors, and the benefits and risks of relevant treatment options.

STEP 14 ✦ STROKE PREVENTION

Address stroke risk factors. Lifestyle changes, diet, exercise, and treatment to address modifiable risk factors can reduce the risk of a recurrent stroke and postpone a first stroke. One study estimated that the expected 24% 5-year cumulative risk of recurrent stroke could be reduced by 80% if patients adopted dietary modifications, exercised, and received antihypertensive medications, aspirin, and a statin.¹⁰² This degree of stroke reduction translates into an NNT of only 5, meaning that achieving these goals in five patients would prevent one stroke. Additional gains could be expected from smoking cessation, glycemic control, and appropriate use of anticoagulants for those with atrial fibrillation.¹⁰² Importantly, a recurrent stroke doubles the risk of death and other vascular complications¹⁷ and increases the likelihood of stroke-related dementia.^{33,40}

The most appropriate preventive treatments depend on the patient's risk factors for stroke, causes of the patient's previous stroke, comorbid medical conditions, and treatment goals. Ideally, the treatment plan will document the presence of advanced comorbid illnesses or individual risk factors that cannot be easily modified and any choices by the patient to forgo treatments intended to reduce risk factors.

If medications are used to prevent a stroke or reduce stroke risk factors, it is prudent to monitor the patient for predictable potential adverse drug reactions. Appendix 1 provides examples of risk factors, interventions, treatment goals, and monitoring strategies for addressing modifiable stroke risk factors.

◆ Lifestyle Modifications

Lifestyle modifications are an integral component of blood pressure control and stroke prevention. The DASH diet, which recommends sodium restriction and a diet rich in fruits, vegetables, and low-fat dairy products, is beneficial for blood pressure reduction.¹⁰³ Patients should stop smoking or using tobacco products and should avoid secondary smoke. Nicotine replacement therapy may be used as part of a smoking cessation program.¹³ Patients who are able should engage in 30 minutes of moderate-intensity physical exercise for 30 minutes one to three times weekly.¹³ Patients should limit alcohol consumption to no more than two drinks daily for men and one drink daily for women.¹³

◆ Hypertension

Hypertension is an important modifiable risk factor for stroke, recurrent stroke, multiple cardiovascular outcomes, and death. A meta-analysis of antihypertensive treatment following stroke showed that antihypertensive medications reduce the risk of recurrent stroke by nearly 3% (from 11.5% to 8.7%), resulting in an NNT of approximately 35.¹⁰⁴ The greatest benefit was seen with the combination of a diuretic with an ACE inhibitor. Even those without a prior history of hypertension should receive antihypertensive medications after a TIA or stroke, if they are appropriate for blood pressure reduction.¹³ No data support an ideal blood pressure goal after stroke or TIA, but benefit is shown by reducing blood pressure by 10/5 mmHg,¹³ and more risk reduction was seen with larger reductions in systolic blood pressure.¹⁰⁴ Although a normal BP is defined as less than 120/80 mmHg, clinicians should base blood pressure goals on patient characteristics, comorbidities, preferences, and ability to tolerate medications.¹³

◆ Diabetes

Diabetes is a risk factor for stroke, and up to 9% of recurrent strokes are attributable to diabetes.¹⁰⁵ It

is reasonable to set a goal for blood sugar control for those patients at risk of initial or recurrent stroke. Deciding on an appropriate target glycosylated hemoglobin (HbA1c) should take into consideration a patient's comorbidities, life expectancy, treatment goals, and preferences. Recent trials of intensive lowering of blood glucose to HbA1c levels of less than 7% have not produced improved composite outcomes of nonfatal heart attack, nonfatal stroke, or death resulting from cardiovascular disease.¹⁰⁶⁻¹⁰⁸ Practitioners should consider basing HbA1c target levels on existing guidelines.¹³ Importantly, the PALTmed clinical practice guideline for diabetes was tailored for the type of patients encountered in LTC.^w

◆ Lipids

A high level of low-density lipoprotein (LDL) cholesterol is a risk factor for stroke, and lowering LDL reduces the risk for stroke.¹⁰⁹ The only trial of statin therapy for secondary stroke showed that 80 mg of atorvastatin daily reduced fatal or nonfatal stroke by 2.2% over 5 years (NNT = 49).¹¹⁰ Those taking atorvastatin also benefited from a similar magnitude of reduction in nonfatal MIs. Whereas this trial showed a slightly elevated incidence of hemorrhagic stroke in those taking atorvastatin,¹⁰⁹ a meta-analysis of statin therapy to lower LDL cholesterol confirmed the reduction in stroke with a variety of statins but did not detect an increase in hemorrhagic stroke.¹¹¹ Clinicians should prescribe intensive statin therapy for those patients with an LDL level of 100 mg/dL or higher, with the goal of reducing LDL by 50% or achieving a target LDL level less than 70 mg/dL.¹³ As with other therapies, decisions about intensive statin therapy and LDL target levels should take into consideration the patient's status, comorbidities, expected longevity, and goals of care.

◆ Antiplatelet Therapy to Prevent Thrombotic Stroke

An antiplatelet agent is indicated for all patients with a TIA or thrombotic stroke that is not caused by a cardioembolism.^{13,71} A meta-analysis of studies comparing placebo to antiplatelet agents administered for an average of 29 months after a thrombotic stroke or TIA showed a 3.8% absolute reduction in the combined events of MI, stroke, and vascular death in patients who received antiplatelet therapy (NNT = 26).¹¹² The NNT was 40 for the prevention of one nonfatal stroke among patients receiving antiplatelet drugs. Adverse effects of antiplatelet therapy include a risk of one to two major extracranial bleeding events for every 1000 patients treated with antiplatelet therapy for 1 year.¹¹²

Appropriate antiplatelet agents for stroke prevention include aspirin, extended-release dipyridamole plus aspirin (ERDP/ASA), and clopidogrel.^{13,71}

Aspirin

Aspirin (50 to 325 mg/day) is the most studied and least expensive antiplatelet agent for secondary stroke prevention.¹³ Aspirin is not effective for primary stroke prevention.¹¹³ A meta-analysis of 10 trials of secondary stroke prevention suggested that after a TIA or stroke, aspirin use results in a 13% reduction in relative stroke risk.¹¹⁴ Debate remains regarding the aspirin dose that is optimal for secondary stroke prevention; similar benefits are shown for doses ranging from 50 to 1500 mg/day.¹¹⁵ Aspirin-induced major gastrointestinal bleeding risk appears to be independent of dose.¹¹⁶

Extended-Release Dipyridamole Plus Aspirin

ERDP/ASA (200/25 mg twice daily) is more expensive than aspirin, but was superior to aspirin alone for stroke prevention in one randomized control trial,¹¹⁷ leading to a preferential recommendation

^w Post-Acute and Long-Term Care Medical Association. Diabetes Management in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.

over aspirin by the ACCP.⁷¹ The 2010 stroke prevention guideline by the American Heart Association and American Stroke Association (AHA/ASA) reviewed additional ERDP/ASA studies and did not recommend it preferentially over aspirin.¹³

Clopidogrel

Clopidogrel is appropriate for patients who are allergic or sensitive to aspirin. For patients with a history of stroke or TIA, clopidogrel was not superior to aspirin for prevention of a subsequent stroke, MI, or death resulting from another vascular cause.¹¹⁸ A trial comparing ERDP/ASA to clopidogrel for secondary stroke prevention found a similar rate of recurrent stroke; the combined outcome of stroke, MI, or vascular death; and hemorrhagic complications for both drugs.¹¹⁹ Thus, it is reasonable to consider aspirin, ERDP/ASA, and clopidogrel equally efficacious as antiplatelet monotherapy. Clinicians and patients may choose among these agents on the basis of clinical factors, cost, and side effect profiles.

Dual Antiplatelet Therapy

Combination therapy with aspirin and clopidogrel should not be used for stroke prevention. Clopidogrel plus aspirin is not superior to clopidogrel alone for stroke prevention, and the combination regimen raises the incidence of serious gastrointestinal bleeding.⁷¹ Combined therapy with clopidogrel and aspirin may be appropriate for patients with a balloon angioplasty or an intracoronary stent.¹²⁰ Thus, if a patient with a TIA or stroke is taking both clopidogrel and aspirin for coronary artery disease, it is reasonable to continue combination therapy according to the cardiac indication.

◆ Bleeding Risk From Antiplatelet Therapy

Because elderly patients with multiple comorbidities are at high risk of bleeding owing to antiplatelet therapy, clinicians may consider prophylactic administration of either a histamine H₂ antagonist (H₂A) or proton pump inhibitor (PPI) to reduce the risk of gastrointestinal bleeding.^{121,122} Despite concerns that PPIs may decrease the effectiveness of clopidogrel and subsequently increase the likelihood of coronary stent thrombosis, MI, or stroke, expert consensus^{121,122} and trial data¹²² support concomitant use of H₂As or PPIs with thienopyridines like clopidogrel to reduce gastrointestinal bleeding rates in high-risk individuals. H₂As may offer some protection from antiplatelet bleeding (up to 25% risk reduction), but PPIs offer greater protection (approximately 50% risk reduction) than H₂As.¹²¹

The strongest risk factor for bleeding caused by antiplatelet agents is a past history of upper gastrointestinal bleeding. Other important bleeding risk factors include advanced age, dual antiplatelet therapy, use of anticoagulants, steroids, nonsteroidal anti-inflammatory drugs, and infection with *H. pylori*. Bleeding risk increases with increasing numbers of risk factors.¹²¹ A quantitative bleeding risk assessment tool can be found in Appendix 2.¹²³

◆ Atrial Fibrillation

Atrial fibrillation increases the risk of stroke four to five fold.¹²⁴ The risk of stroke that is attributable to atrial fibrillation increases with age from 2 per 100 patient years at 60 years of age to 5.3 per 100 patient years at 80 years of age.¹²⁵ The recommendations for anticoagulation to prevent stroke are identical for patients with atrial fibrillation, paroxysmal atrial fibrillation, or atrial fibrillation that has

been converted to sinus rhythm.^{126,127}

Anticoagulation with warfarin is the current standard of care to reduce the risk of stroke caused by atrial fibrillation.^{13,124,126} The ACCP,¹²⁴ but not the AHA/ASA,¹³ offers the option of aspirin rather than warfarin for “low-risk” patients with atrial fibrillation and no other risk factors, but essentially all LTC patients are at a moderate to high risk for stroke, so this option does not apply. Clinicians often prescribe aspirin for patients for whom warfarin is deemed inappropriate. Unfortunately, aspirin may have little or no benefit at reducing the risk of stroke caused by atrial fibrillation.¹²⁴ One new oral anticoagulant, dabigatran, a direct thrombin inhibitor, is not only as effective as warfarin in reducing stroke risk in atrial fibrillation¹²⁸ but also has a lower risk of life-threatening hemorrhagic complications, does not require dose monitoring by laboratory testing as is needed with warfarin, and will likely be incorporated into updated versions of guidelines. Guidelines from the European Society of Cardiology (updated in 2010) recommend dabigatran as an alternative to warfarin therapy in certain situations.¹²⁹

Warfarin reduces the risk of stroke by 66%¹²⁴ and decreases the likelihood of severe disability if a stroke occurs while taking warfarin.¹³⁰ Unfortunately, multiple studies, including those performed in LTC settings,²³ have shown that only about 50% of older people with atrial fibrillation who have a moderate to high risk of stroke and no contraindications receive warfarin.¹³¹⁻¹³⁴ Warfarin may be underused because of cost concerns, the inconvenience of treatment monitoring, and fear of bleeding complications. Practitioners may be more concerned about the possible negative effects of anticoagulant therapy than about the potential benefits of such therapy for older adults with atrial fibrillation. The availability of dabigatran may improve the use of anticoagulation therapy in LTC.

The decision whether to treat a patient with advanced age and multiple comorbidities with an anticoagulant is complicated by the dilemma that those patients at the highest risk of stroke are also the ones at the highest risk of bleeding complications.¹²⁴ Ideally, a shared decision will be reached through discussion with the patient and family about the patient’s prognosis and treatment goals as well as the potential benefits and risks of oral anticoagulant therapy. Such a discussion is important because studies show that atrial fibrillation patients with similar clinical profiles choose different approaches to stroke prevention.¹³⁷⁻¹³⁹

A systematic approach can help guide the practitioner and patient to a patient-centered decision about the use of warfarin and other oral anticoagulants in atrial fibrillation. An evidence-based decision support tool can help to estimate both the risk of stroke and the risk of bleeding caused by anticoagulation (Appendix 3). The first step in this process is to assess whether anticoagulation may improve the quality or length of a patient’s life. For example, patients with short life expectancies resulting from diseases such as metastatic cancer, severe dementia, or advanced chronic illnesses are unlikely to benefit from warfarin therapy. Next, estimate the patient’s risk for stroke and compare it to the risk of treatment-related complications. Estimate stroke risk with the CHADS₂ risk stratification tool.¹⁴⁰ Review the patient’s history for absolute and relative contraindications to warfarin therapy. If the patient is a candidate for warfarin, review the known risks for intracerebral, subdural, and gastrointestinal bleeding. It is important to note that according to data from community studies, a tendency to fall does not create an unacceptably high risk of anticoagulant-related bleeding or subdural hematoma in older adults.¹⁴¹ Finally, check for conditions that may increase bleeding risk. It may be possible to modify some risk factors. For patients with nonmodifiable risk factors, it is prudent to be vigilant for signs or symptoms of bleeding and monitor the INR more frequently.

◆ Symptomatic Carotid Stenosis

Patients who suffer a TIA or stroke typically undergo assessment of their carotid arteries. If studies show stenosis of 50% to 99%, patients should receive optimal medical therapy, including aspirin, blood pressure control, lipid control, and lifestyle modification.¹³ For patients with stenosis greater than 70%, carotid endarterectomy (CEA), in addition to optimal medical therapy, reduces the risk of stroke more than medical therapy alone.^{13,142} For patients with stenosis of 50% to 69%, decisions about CEA hinge on the patient's suitability for surgery and on surgical morbidity and mortality less than 6%. Endarterectomy is not recommend for patients with stenosis less than 50%.¹³

Carotid artery angioplasty and stenting (CAS) is an alternative to CEA. It has the advantages of being less invasive than CEA, and thus may be appropriate for patients with high surgical risk, inaccessible carotid lesions, or prior radiation or surgery performed on the neck. Current stenting procedures use embolic protection devices to capture debris caused by the procedure, and this reduces periprocedural stroke rates.

A recent comparison of CAS and CEA for symptomatic and asymptomatic patients with 50% to 70% stenosis showed no statistically significant difference in a composite end point of stroke, MI, death during the periprocedural period, or ipsilateral stroke within 4 years. For the two endpoints of 4-year rate of stroke and death, CEA was better than CAS. Periprocedural stroke occurred more often with CAS, whereas periprocedural MI occurred more often with CEA.¹⁴³ In that study, CEA was superior to CAS for patients aged 70 years or more. For CAS to be a viable alternative to CEA, CAS periprocedural morbidity and mortality should be under 6%.¹³

MONITORING

STEP 15

Monitor and periodically document the physical, functional, and psychosocial progress of the patient with an old or new stroke. Treatment goals may change as the patient either recovers from the stroke or experiences decline. The interdisciplinary team should regularly re-evaluate both the treatment goals and progress made toward those goals. The team should monitor the continued appropriateness of the treatment plan by taking into consideration the patient's clinical condition and ability to meet treatment goals, as well as the presence of adverse treatment effects.

If the patient is not making progress toward his or her treatment goals or is regressing from a previous level of function, consider reassessing him or her for exacerbations of chronic illnesses, metabolic abnormalities, adverse medication effects, pain, depression, common stroke complications, or new acute stroke. The interdisciplinary team, in collaboration with the patient and family, should determine the appropriateness of evaluating and treating each possible impediment to progress.

STEP 16

Monitor the patient to ensure that modifiable risk factors for stroke are adequately controlled.

Reassess the treatment plan regularly to ensure that modifiable risk factors for a first or recurrent stroke have been controlled to the extent feasible, in accordance with the patient's treatment plan and goals. For example, if risk factors have not been reduced to target levels (e.g., LDL cholesterol less than 100 mg/dL, systolic blood pressure less than 140 mm Hg), adjust the treatment plan appropriately. Alternatively, if the patient's condition or treatment goals have changed, the risk-factor reduction plan should be modified to reflect that change (e.g., less aggressive blood pressure reduction because of postural hypotension resulting in falls).

STEP 17

Monitor the facility's management of stroke and stroke risk factors. The successful prevention and management of stroke depend on staff education and on interdisciplinary assessment and treatment. Facilities may wish to incorporate stroke quality-of-care indicators into their quality improvement process. Possible indicators include

- ◆ Staff awareness of symptoms of acute stroke,
- ◆ Timeliness of response to possible signs and symptoms of acute stroke,
- ◆ Documentation of the patient's care goals,
- ◆ Implementation of interventions to prevent acute complications of stroke,
- ◆ Occurrence of common complications of stroke, and
- ◆ Adequacy of control of modifiable stroke risk factors.

SUMMARY

On average across the United States, someone suffers a stroke every 40 seconds, and someone dies from a stroke every 4 minutes. Stroke is also the leading cause of long-term disability in the United States. Because the risk of stroke increases with age, a large percentage of LTC patients are at risk. High-risk patients can be identified and addressed with a variety of preventive measures.

Patients who have a stroke should be assessed promptly. It is up to the facility and interdisciplinary team leaders to ensure that mechanisms are in place to rapidly identify the patient who is experiencing an acute stroke and to determine—taking into consideration many factors, including the patient's preferences—whether to treat the patient in the facility or transfer him or her to an acute-care setting.

After a stroke, a rehabilitation program devised by the practitioner and interdisciplinary team can optimize function, address problems such as pain and depression, and maximize the patient's quality of life and dignity. The patient's comprehensive care plan and the effectiveness of risk-factor modification strategies should be re-evaluated regularly to ensure that care goals remain appropriate.

REFERENCES

1. American Heart Association, Heart Disease and Stroke Statistics. 2010 Update At-a-Glance. Available at: http://www.heart.org/HEARTORG/General/Heart-and-Stroke-Association-Statistics_UCM_319064_SubHomePage.jsp. Accessed 01/09/12.
2. Kelly-Hayes M, Beiser A, Kase CS, et al. The influence of gender and age on disability following ischemic stroke: The Framingham study. *J Stroke Cerebrovasc Dis* 2003; 12: 119-126.
3. Lee AJ, Huber J, Stason WB. Poststroke rehabilitation in older Americans. The Medicare experience. *Med Care* 1996; 34: 811-825.
4. Leibson CL, Ransom JE, Brown RD, et al. Stroke-attributable nursing home use: A population-based study. *Neurology* 1998; 51: 163-168.
5. Kleindorfer D, Panagos P, Pancioli A, et al. Incidence and short-term prognosis of transient ischemic attack in a population-based study. *Stroke* 2005; 36: 720-723.
6. Lisabeth LD, Ireland JK, Risser JM, et al. Stroke risk after transient ischemic attack in a population-based setting. *Stroke* 2004; 35: 1842-1846.

7. Coull AJ, Lovett JK, Rothwell PM. Population based study of early risk of stroke after transient ischaemic attack or minor stroke: Implications for public education and organisation of services. *BMJ* 2004; 328: 326.
8. Johnston SC, Fayad PB, Gorelick PB, et al. Prevalence and knowledge of transient ischemic attack among US adults. *Neurology* 2003; 60: 1429-1434.
9. Sherman DG. Reconsideration of TIA diagnostic criteria. *Neurology* 2004; 62: S20-21.
10. Bogousslavsky J, Van Melle G, Regli F. The Lausanne Stroke Registry: Analysis of 1,000 consecutive patients with first stroke. *Stroke* 1988; 19: 1083-1092.
11. Sivenius J, Heinonen OP, Pyorala K, et al. The incidence of stroke in the Kuopio area of East Finland. *Stroke* 1985; 16: 188-192.
12. Saposnik G, Kapral MK, Coutts SB, et al. Do all age groups benefit from organized inpatient stroke care? *Stroke* 2009; 40: 3321-3327.
13. Furie KL, Kasner SE, Adams RJ, et al. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2011; 42: 227-276.
14. Gilbert JJ, Vinters HV. Cerebral amyloid angiopathy: Incidence and complications in the aging brain. I. Cerebral hemorrhage. *Stroke* 1983; 14: 915-923.
15. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: A scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. *Stroke* 2009; 40: 2276-2293.
16. Duncan PW, Zorowitz R, Bates B, et al. Management of Adult Stroke Rehabilitation Care: A clinical practice guideline. *Stroke* 2005; 36: e100-143.
17. Hardie K, Hankey GJ, Jamrozik K, et al. Ten-year survival after first-ever stroke in the Perth community stroke study. *Stroke* 2003; 34: 1842-1846.
18. Hankey GJ, Jamrozik K, Broadhurst RJ, et al. Five-year survival after first-ever stroke and related prognostic factors in the Perth Community Stroke Study. *Stroke* 2000; 31: 2080-2086.
19. Ford GA, Ahmed N, Azevedo E, et al. Intravenous alteplase for stroke in those older than 80 years old. *Stroke* 2010; 41: 2568-2574.
20. Hacke W, Kaste M, Bluhmki E, et al. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. *N Engl J Med* 2008; 359: 1317-1329.
21. Del Zoppo GJ, Saver JL, Jauch EC, Adams HP, Jr. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: A science advisory from the American Heart Association/American Stroke Association. *Stroke* 2009; 40: 2945-2948.
22. Quilliam BJ, Lapane KL. Clinical correlates and drug treatment of residents with stroke in long-term care. *Stroke* 2001; 32: 1385-1393.
23. Gurwitz JH, Monette J, Rochon PA, et al. Atrial fibrillation and stroke prevention with warfarin in the long-term care setting. *Arch Intern Med* 1997; 157: 978-984.
24. Ness J, Aronow WS, Ahn C. Risk factors for ischemic stroke in older persons in an academic hospital based geriatrics practice. *Prev Cardiol* 2000; 3: 118-120.
25. Hurwitz AS, Brice JH, Overby BA, Evenson KR. Directed use of the Cincinnati Prehospital Stroke Scale by laypersons. *Prehosp Emerg Care* 2005; 9: 292-296.

26. Adams HP, Jr., del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Stroke* 2007; 38: 1655-1711.
27. Kalra L, Yu G, Wilson K, Roots P. Medical complications during stroke rehabilitation. *Stroke* 1995; 26: 990-994.
28. Patel M, Coshall C, Rudd AG, Wolfe CD. Natural history of cognitive impairment after stroke and factors associated with its recovery. *Clin Rehabil* 2003; 17: 158-166.
29. Brittain KR, Peet SM, Castleden CM. Stroke and incontinence. *Stroke* 1998; 29: 524-528.
30. Nakayama H, Jorgensen HS, Pedersen PM, et al. Prevalence and risk factors of incontinence after stroke. The Copenhagen Stroke Study. *Stroke* 1997; 28: 58-62.
31. Harari D, Norton C, Lockwood L, Swift C. Treatment of constipation and fecal incontinence in stroke patients: Randomized controlled trial. *Stroke* 2004; 35: 2549-2555.
32. Harari D, Coshall C, Rudd AG, Wolfe CD. New-onset fecal incontinence after stroke: Prevalence, natural history, risk factors, and impact. *Stroke* 2003; 34: 144-150.
33. Pendlebury ST, Rothwell PM. Prevalence, incidence, and factors associated with pre-stroke and post-stroke dementia: A systematic review and meta-analysis. *Lancet Neurol* 2009; 8: 1006-1018.
34. Barba R, Martinez-Espinosa S, Rodriguez-Garcia E, et al. Poststroke dementia: Clinical features and risk factors. *Stroke* 2000; 31: 1494-1501.
35. Pohjasvaara T, Erkinjuntti T, Vataja R, Kaste M. Dementia three months after stroke. Baseline frequency and effect of different definitions of dementia in the Helsinki Stroke Aging Memory Study (SAM) cohort. *Stroke* 1997; 28: 785-792.
36. Lin JH, Lin RT, Tai CT, et al. Prediction of poststroke dementia. *Neurology* 2003; 61: 343-348.
37. Madureira S, Guerreiro M, Ferro JM. Dementia and cognitive impairment three months after stroke. *Eur J Neurol* 2001; 8: 621-627.
38. Ballard C, Rowan E, Stephens S, et al. Prospective follow-up study between 3 and 15 months after stroke: Improvements and decline in cognitive function among dementia-free stroke survivors >75 years of age. *Stroke* 2003; 34: 2440-2444.
39. Desmond DW, Moroney JT, Sano M, Stern Y. Incidence of dementia after ischemic stroke: Results of a longitudinal study. *Stroke* 2002; 33: 2254-2260.
40. Ukraintseva S, Sloan F, Arbeev K, Yashin A. Increasing rates of dementia at time of declining mortality from stroke. *Stroke* 2006; 37: 1155-1159.
41. Roman GC, Salloway S, Black SE, et al. Randomized, placebo-controlled, clinical trial of donepezil in vascular dementia: Differential effects by hippocampal size. *Stroke* 2010; 41: 1213-1221.
42. Kind AJ, Smith MA, Frytak JR, Finch MD. Bouncing back: Patterns and predictors of complicated transitions 30 days after hospitalization for acute ischemic stroke. *J Am Geriatr Soc* 2007; 55: 365-373.
43. Kauhanen ML, Korpelainen JT, Hiltunen P, et al. Aphasia, depression, and non-verbal cognitive impairment in ischaemic stroke. *Cerebrovasc Dis* 2000; 10: 455-461.
44. Kelly J, Rudd A, Lewis RR, et al. Venous thromboembolism after acute ischemic stroke: A prospective study using magnetic resonance direct thrombus imaging. *Stroke* 2004; 35: 2320-2325.

45. Warlow C, Ogston D, Douglas AS. Deep venous thrombosis of the legs after strokes. Part I—Incidence and predisposing factors. *Br Med J* 1976; 1: 1178-1181.
46. Wijdicks EF, Scott JP. Pulmonary embolism associated with acute stroke. *Mayo Clin Proc* 1997; 72: 297-300.
47. Kelly J, Rudd A, Lewis R, Hunt BJ. Venous thromboembolism after acute stroke. *Stroke* 2001; 32: 262-267.
48. Langhorne P, Stott DJ, Robertson L, et al. Medical complications after stroke: A multicenter study. *Stroke* 2000; 31: 1223-1229.
49. Hackett ML, Yapa C, Parag V, Anderson CS. Frequency of depression after stroke: A systematic review of observational studies. *Stroke* 2005; 36: 1330-1340.
50. Linden T, Blomstrand C, Skoog I. Depressive disorders after 20 months in elderly stroke patients: A case-control study. *Stroke* 2007; 38: 1860-1863.
51. Robinson RG. Poststroke depression: Prevalence, diagnosis, treatment, and disease progression. *Biol Psychiatry* 2003; 54: 376-387.
52. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (4th edition). 1994. Washington, D.C.: Brandon/Hill.
53. Bogousslavsky J. William Feinberg lecture 2002: Emotions, mood, and behavior after stroke. *Stroke* 2003; 34: 1046-1050.
54. Mann G, Hankey GJ, Cameron D. Swallowing disorders following acute stroke: Prevalence and diagnostic accuracy. *Cerebrovasc Dis* 2000; 10: 380-386.
55. Mann G, Hankey GJ, Cameron D. Swallowing function after stroke: Prognosis and prognostic factors at 6 months. *Stroke* 1999; 30: 744-748.
56. Kumlien S, Axelsson K. Stroke patients in nursing homes: Eating, feeding, nutrition and related care. *J Clin Nurs* 2002; 11: 498-509.
57. Spieker MR. Evaluating dysphagia. *Am Fam Physician* 2000; 61: 3639-3648.
58. Palmer JB, Drennan JC, Baba M. Evaluation and treatment of swallowing impairments. *Am Fam Physician* 2000; 61: 2453-2462.
59. AHRQ. Diagnosis and treatment of swallowing disorders (dysphagia) in acute-care stroke patients. Summary, Evidence Report/Technology Assessment: Number 8, March 1999. Rockville, MD: Agency for Health Care Research and Quality. Available at: <http://www.ahrq.gov/clinic/epcsums/dysphsum.htm>. Accessed 02/04/11.
60. Heckert KD, Komaroff E, Adler U, Barrett AM. Postacute reevaluation may prevent Dysphagia-associated morbidity. *Stroke* 2009; 40: 1381-1385.
61. Mari F, Matei M, Ceravolo MG, et al. Predictive value of clinical indices in detecting aspiration in patients with neurological disorders. *J Neurol Neurosurg Psychiatry* 1997; 63: 456-460.
62. Sellars C, Bowie L, Bagg J, et al. Risk factors for chest infection in acute stroke: A prospective cohort study. *Stroke* 2007; 38: 2284-2291.
63. Barker WH, Mullooly JP. Stroke in a defined elderly population, 1967-1985. A less lethal and disabling but no less common disease. *Stroke* 1997; 28: 284-290.
64. Satkunam LE. Rehabilitation medicine: 3. Management of adult spasticity. *CMAJ* 2003; 169: 1173-1179.
65. Bender L, McKenna K. Hemiplegic shoulder pain: Defining the problem and its management. *Disabil Rehabil* 2001; 23: 698-705.
66. Cameron ID, Murray GR, Gillespie LD, et al. Interventions for preventing falls in older people in nursing care facilities and hospitals. *Cochrane Database Syst Rev* 2010: CD005465.

67. Batchelor F, Hill K, Mackintosh S, Said C. What works in falls prevention after stroke?: A systematic review and meta-analysis. *Stroke* 2010; 41: 1715-1722.
68. Adams HP, Jr., Davis PH, Leira EC, et al. Baseline NIH Stroke Scale score strongly predicts outcome after stroke: A report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology* 1999; 53: 126-131.
69. Drinka P. Preventing aspiration in the nursing home: The role of biofilm and data from the ICU. *J Am Med Dir Assoc* 2010; 11: 70-77.
70. Geerts WH, Pineo GF, Heit JA, et al. Prevention of venous thromboembolism: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126: 338S- 400S.
71. Albers GW, Amarenco P, Easton JD, et al. Antithrombotic and thrombolytic therapy for ischemic stroke: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008; 133: 630S-669S.
72. The International Stroke Trial (IST): A randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. International Stroke Trial Collaborative Group. *Lancet* 1997; 349: 1569-1581.
73. Andre C, de Freitas GR, Fukujima MM. Prevention of deep venous thrombosis and pulmonary embolism following stroke: A systematic review of published articles. *Eur J Neurol* 2007; 14: 21-32.
74. Sherman DG, Albers GW, Bladin C, et al. The efficacy and safety of enoxaparin versus unfractionated heparin for the prevention of venous thromboembolism after acute ischaemic stroke (PREVAIL Study): An open-label randomised comparison. *Lancet* 2007; 369: 1347-1355.
75. Naccarato M, Chiodo Grandi F, Dennis M, Sandercock PA. Physical methods for preventing deep vein thrombosis in stroke. *Cochrane Database Syst Rev* 2010: CD001922.
76. Thigh-length versus below-knee stockings for deep venous thrombosis prophylaxis after stroke: A randomized trial. *Ann Intern Med* 2010; 153: 553-562.
77. Viitanen M, Winblad B, Asplund K. Autopsy-verified causes of death after stroke. *Acta Med Scan* 1987; 222: 401-408.
78. Thomas DR. Are all pressure ulcers avoidable? *J Am Med Dir Assoc* 2001; 2: 297-301.
79. Lindgren I, Jonsson AC, Norrving B, Lindgren A. Shoulder pain after stroke: A prospective population-based study. *Stroke* 2007; 38: 343-348.
80. Shah RR, Haghpanah S, Elovic EP, et al. MRI findings in the painful poststroke shoulder. *Stroke* 2008; 39: 1808-1813.
81. Ada L, Foongchomcheay A, Canning C. Supportive devices for preventing and treating subluxation of the shoulder after stroke. *Cochrane Database Syst Rev* 2005: CD003863.
82. Urban PP, Wolf T, Uebele M, et al. Occurrence and clinical predictors of spasticity after ischemic stroke. *Stroke* 2010; 41: 2016-2020.
83. Lundstrom E, Smits A, Borg J, Terent A. Four-fold increase in direct costs of stroke survivors with spasticity compared with stroke survivors without spasticity: The first year after the event. *Stroke* 2010; 41: 319-324.
84. McMurdo ME, Argo I, Phillips G, et al. Cranberry or trimethoprim for the prevention of recurrent urinary tract infections? A randomized controlled trial in older women. *J Antimicrob Chemother* 2009; 63: 389-395.
85. Jepson RG, Craig JC. Cranberries for preventing urinary tract infections. *Cochrane Database Syst Rev* 2008: CD001321.
86. Kearon C, Kahn SR, Agnelli G, et al. Antithrombotic therapy for venous thromboembolic dis-

- ease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008; 133: 454S-545S.
87. Narushima K, Robinson RG. The effect of early versus late antidepressant treatment on physical impairment associated with poststroke depression: Is there a time-related therapeutic window? *J Nerv Ment Dis* 2003; 191: 645-652.
 88. Jorge RE, Robinson RG, Arndt S, Starkstein S. Mortality and poststroke depression: A placebo-controlled trial of antidepressants. *Am J Psychiatry* 2003; 160: 1823-1829.
 89. Andersen G, Vestergaard K, Lauritzen L. Effective treatment of poststroke depression with the selective serotonin reuptake inhibitor citalopram. *Stroke* 1994; 25: 1099-1104.
 90. Dam M, Tonin P, De Boni A, et al. Effects of fluoxetine and maprotiline on functional recovery in poststroke hemiplegic patients undergoing rehabilitation therapy. *Stroke* 1996; 27: 1211-1214.
 91. Choi-Kwon S, Han SW, Kwon SU, et al. Fluoxetine treatment in poststroke depression, emotional incontinence, and anger proneness: A double-blind, placebo-controlled study. *Stroke* 2006; 37: 156-161.
 92. Yi ZM, Liu F, Zhai SD. Fluoxetine for the prophylaxis of poststroke depression in patients with stroke: A meta-analysis. *Int J Clin Pract* 2010; 64: 1310-1317.
 93. Robinson RG, Jorge RE, Moser DJ, et al. Escitalopram and problem-solving therapy for prevention of poststroke depression: A randomized controlled trial. *JAMA* 2008; 299: 2391-2400.
 94. Carnaby G, Hankey GJ, Pizzi J. Behavioural intervention for dysphagia in acute stroke: A randomised controlled trial. *Lancet Neurol* 2006; 5: 31-37.
 95. Loeb MB, Becker M, Eady A, Walker-Dilks C. Interventions to prevent aspiration pneumonia in older adults: A systematic review. *J Am Geriatr Soc* 2003; 51: 1018-1022.
 96. Martino R, Pron G, Diamant N. Screening for oropharyngeal dysphagia in stroke: Insufficient evidence for guidelines. *Dysphagia* 2000; 15: 19-30.
 97. Ramsey DJ, Smithard DG, Kalra L. Early assessments of dysphagia and aspiration risk in acute stroke patients. *Stroke* 2003; 34: 1252-1257.
 98. Langmore SE, Terpenning MS, Schork A, et al. Predictors of aspiration pneumonia: How important is dysphagia? *Dysphagia* 1998; 13: 69-81.
 99. Frontera WR, Silver JK, Rizzo TD Jr, eds. *Essentials of physical medicine and rehabilitation: musculoskeletal disorders, pain, and rehabilitation*. 2nd ed. Philadelphia, PA: SAUNDERS ELSEVIER; 2008.
 100. Epocrates. Online Premium version. Available at: <https://online.epocrates.com>. Accessed 02/04/11.
 101. Smith PW, Bennett G, Bradley S, et al. SHEA/APIC Guideline: Infection prevention and control in the long-term care facility. *Am J Infect Control* 2008; 36: 504-535.
 102. Hackam DG, Spence JD. Combining multiple approaches for the secondary prevention of vascular events after stroke: A quantitative modeling study. *Stroke* 2007; 38: 1881-1885.
 103. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med* 2001; 344: 3-10.
 104. Rashid P, Leonardi-Bee J, Bath P. Blood pressure reduction and secondary prevention of stroke and other vascular events: A systematic review. *Stroke* 2003; 34: 2741-2748.
 105. Hillen T, Coshall C, Tilling K, et al. Cause of stroke recurrence is multifactorial: Patterns, risk factors, and outcomes of stroke recurrence in the South London Stroke Register. *Stroke* 2003; 34: 1457-1463.

106. Duckworth W, Abraira C, Moritz T, et al. Glucose control and vascular complications in veterans with type 2 diabetes. *N Engl J Med* 2009; 360: 129-139.
107. Gerstein HC, Miller ME, Byington RP, et al. Effects of intensive glucose lowering in type 2 diabetes. *N Engl J Med* 2008; 358: 2545-2559.
108. Patel A, MacMahon S, Chalmers J, et al. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. *N Engl J Med* 2008; 358: 2560-2572.
109. Amarenco P, Labreuche J, Lavallee P, Touboul PJ. Statins in stroke prevention and carotid atherosclerosis: Systematic review and up-to-date meta-analysis. *Stroke* 2004; 35: 2902-2909.
110. Amarenco P, Bogousslavsky J, Callahan A, 3rd, et al. High-dose atorvastatin after stroke or transient ischemic attack. *N Engl J Med* 2006; 355: 549-559.
111. Amarenco P, Labreuche J. Lipid management in the prevention of stroke: Review and updated meta-analysis of statins for stroke prevention. *Lancet Neurol* 2009; 8: 453-463.
112. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* 2002; 324: 71-86.
113. Straus SE, Majumdar SR, McAlister FA. New evidence for stroke prevention: Scientific review. *JAMA* 2002; 288: 1388-1395.
114. Albers GW, Tijssen JG. Antiplatelet therapy: New foundations for optimal treatment decisions. *Neurology* 1999; 53: S25-31.
115. Johnson ES, Lanes SF, Wentworth CE, 3rd, et al. A metaregression analysis of the doseresponse effect of aspirin on stroke. *Arch Intern Med* 1999; 159: 1248-1253.
116. Derry S, Loke YK. Risk of gastrointestinal haemorrhage with long term use of aspirin: Meta-analysis. *BMJ* 2000; 321: 1183-1187.
117. Diener HC, Cunha L, Forbes C, et al. European Stroke Prevention Study. 2. Dipyridamole and acetylsalicylic acid in the secondary prevention of stroke. *J Neurol Sci* 1996; 143: 1-13.
118. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. *Lancet* 1996; 348: 1329-1339.
119. Sacco RL, Diener HC, Yusuf S, et al. Aspirin and extended-release dipyridamole versus clopidogrel for recurrent stroke. *N Engl J Med* 2008; 359: 1238-1251.
120. Becker RC, Meade TW, Berger PB, et al. The primary and secondary prevention of coronary artery disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008; 133: 776S-814S.
121. Abraham NS, Hlatky MA, Antman EM, et al. ACCF/ACG/AHA 2010 expert consensus document on the concomitant use of proton pump inhibitors and thienopyridines: A focused update of the ACCF/ACG/AHA 2008 expert consensus document on reducing the gastrointestinal risks of antiplatelet therapy and NSAID use. *J Am Coll Cardiol* 2010; 56: 2051-2066.
122. Bhatt DL, Cryer BL, Contant CF, et al. Clopidogrel with or without omeprazole in coronary artery disease. *N Engl J Med* 2010; 363: 1909-1917.
123. Ducrocq G, Wallace JS, Baron G, et al. Risk score to predict serious bleeding in stable outpatients with or at risk of atherothrombosis. *Eur Heart J* 2010; 31: 1257-1265.
124. Singer DE, Albers GW, Dalen JE, et al. Antithrombotic therapy in atrial fibrillation: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008; 133: 546S-592S.
125. Ruigomez A, Garcia Rodriguez LA, Johansson S, et al. Risk of cerebrovascular accident after a first diagnosis of atrial fibrillation. *Clin Cardiol* 2007; 30: 624-628.
126. Fuster V, Ryden LE, Cannon DS, et al. ACC/AHA/ESC 2006 Guidelines for the Management

- of Patients with Atrial Fibrillation: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation): Developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Circulation* 2006; 114: e257-354.
127. Singer DE, Chang Y, Fang MC, et al. The net clinical benefit of warfarin anticoagulation in atrial fibrillation. *Ann Intern Med* 2009; 151: 297-305.
 128. Connolly SJ, Ezekowitz MD, Yusuf S, et al. Dabigatran versus warfarin in patients with atrial fibrillation. *N Engl J Med* 2009; 361: 1139-1151.
 129. Camm AJ, Kirchhof P, Lip GY, et al. Guidelines for the management of atrial fibrillation: The Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). *Eur Heart J* 2010; 31: 2369-2429.
 130. Hylek EM, Go AS, Chang Y, et al. Effect of intensity of oral anticoagulation on stroke severity and mortality in atrial fibrillation. *N Engl J Med* 2003; 349: 1019-1026.
 131. Stafford RS, Singer DE. National patterns of warfarin use in atrial fibrillation. *Arch Intern Med* 1996; 156: 2537-2541.
 132. Antani MR, Beyth RJ, Covinsky KE, et al. Failure to prescribe warfarin to patients with non-rheumatic atrial fibrillation. *J Gen Intern Med* 1996; 11: 713-720.
 133. Lawson F, McAlister F, Ackman M, et al. The utilization of antithrombotic prophylaxis for atrial fibrillation in a geriatric rehabilitation hospital. *J Am Geriatr Soc* 1996; 44: 708-711.
 134. Albers GW, Yim JM, Belew KM, et al. Status of antithrombotic therapy for patients with atrial fibrillation in university hospitals. *Arch Intern Med* 1996; 156: 2311-2316.
 135. Sudlow M, Thomson R, Rodgers H, et al. The effect of age and quality of life in doctors' decisions to anticoagulate patients with atrial fibrillation. *Age Ageing* 1998; 27: 285-289.
 136. Beyth RJ, Antani MR, Covinsky KE, et al. Why isn't warfarin prescribed to patients with non-rheumatic atrial fibrillation? *J Gen Intern Med* 1996; 11: 721-728.
 137. Thomson R, Parkin D, Eccles M, et al. Decision analysis and guidelines for anticoagulant therapy to prevent stroke in patients with atrial fibrillation. *Lancet* 2000; 355: 956-962.
 138. Gage BF, Cardinalli AB, Owens DK. Cost-effectiveness of preference-based antithrombotic therapy for patients with nonvalvular atrial fibrillation. *Stroke* 1998; 29: 1083-1091.
 139. Man-Son-Hing M, Laupacis A, O'Connor A, et al. Warfarin for atrial fibrillation. The patient's perspective. *Arch Intern Med* 1996; 156: 1841-1848.
 140. Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: Results from the National Registry of Atrial Fibrillation. *JAMA* 2001; 285: 2864-2870.
 141. Man-Son-Hing M, Nichol G, Lau A, Laupacis A. Choosing antithrombotic therapy for elderly patients with atrial fibrillation who are at risk for falls. *Arch Intern Med* 1999; 159: 677-685.
 142. Rothwell PM, Eliasziw M, Gutnikov SA, et al. Analysis of pooled data from the randomised controlled trials of endarterectomy for symptomatic carotid stenosis. *Lancet* 2003; 361: 107-116.
 143. Brott TG, Hobson RW, 2nd, Howard G, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. *N Engl J Med* 2010; 363: 11-23.

APPENDIX 1

Modifiable Risk Factors for TIA and Stroke: Interventions, Treatment Goals, and Strategies for Monitoring Adverse Drug Effects

Risk Factor	Intervention	Treatment Goal	Monitoring
Smoking Use of tobacco products	<ul style="list-style-type: none"> ◆ Provide counseling on the benefits of smoking cessation (IC) 	<ul style="list-style-type: none"> ◆ Smoking cessation ◆ Stop use of tobacco products ◆ Avoid secondhand smoke 	<ul style="list-style-type: none"> ◆ Monitor smoking status
Alcohol consumption	<ul style="list-style-type: none"> ◆ Recommend avoidance of heavy alcohol consumption (more than 5 drinks/d) (IC) 	<ul style="list-style-type: none"> ◆ Limit alcohol consumption to 2 drinks/day (men); 1 drink/day (women) (IIb, B) 	<ul style="list-style-type: none"> ◆ Monitor alcohol consumption
Inactivity		<ul style="list-style-type: none"> ◆ Moderate-intensity physical exercise 30 minutes, 1 to 3 times weekly (IIb, C) 	
Obesity Unhealthy diet	<ul style="list-style-type: none"> ◆ Weight loss as appropriate 	<ul style="list-style-type: none"> ◆ DASH diet (salt restriction, diet rich in fruits, vegetables and low-fat dairy products)^a 	<ul style="list-style-type: none"> ◆ Monitor weight
Hypertension	<ul style="list-style-type: none"> ◆ Encourage lifestyle modification (IIa, C) ◆ Antihypertensive treatment for those with hypertension (IA) ◆ Antihypertensive treatment is reasonable for those without documented hypertension if considered appropriate for BP reduction (IIa, B) ◆ Diuretics or combination of diuretic and ACEI are useful (IA) ◆ Individualize BP medications based on patient characteristics and comorbidities (IIa, B) 	<ul style="list-style-type: none"> ◆ Individualize BP goals based on patient characteristics and comorbidities (IIa, B) ◆ Benefit is seen with reductions of 10/5 mm Hg (IIa, B) ◆ Normal BP is less than 120/80 (IIa, B) 	<ul style="list-style-type: none"> ◆ Check for symptoms of postural hypotension ◆ Measure postural blood pressure, if appropriate ◆ Perform appropriate laboratory testing as indicated (e.g., electrolytes, creatinine, BUN)
Diabetes	<ul style="list-style-type: none"> ◆ Use existing guidelines for glycemic control (IB) ◆ Use existing guidelines for blood pressure reduction (IB) 	<ul style="list-style-type: none"> ◆ Base goals for glucose control on comorbid conditions, presence of diabetic complications, and patient preferences (See PALMed clinical practice guide-line on diabetes^b) ◆ Avoid lowering HbA1c to less than 6.5% in patients with a history of cardiovascular disease or with vascular risk factors 	<ul style="list-style-type: none"> ◆ Monitor according to medications used and goals of therapy
Hyperhomocysteinemia	<ul style="list-style-type: none"> ◆ No evidence exists that lowering homocysteine with folic acid reduces stroke recurrence (IIb, B) 		
Hyperlipidemia	<ul style="list-style-type: none"> ◆ (1) For those without known CHD but with evidence of atherosclerosis or LDL higher than 100, prescribe intensive lipid-lowering therapy (IB) ◆ (2) For those with known CHD or hyperlipidemia, manage according to NCEP III (IA) ◆ (3) For those with low HDL consider treatment with niacin or gemfibrozil (IIb, B) 	<ul style="list-style-type: none"> ◆ (1) Reduce LDL at least 50%, or target LDL to less than 70 (IIa, B) ◆ (2) Achieve National Cholesterol Education Program (NCEP) III targets with lifestyle modifications, diet and medications (IA)^c 	<ul style="list-style-type: none"> ◆ Monitor according to medications used and goals of therapy ◆ Check for muscle pain ◆ Measure liver enzymes periodically ◆ Monitor lipid profile periodically

APPENDIX 1 continued

Risk Factor	Intervention	Treatment Goal	Monitoring
Hormone replacement therapy	<ul style="list-style-type: none"> ◆ Estrogen with or without a progestin is not recommended (III A) 	<ul style="list-style-type: none"> ◆ Discontinue estrogen if currently prescribed 	
Noncardioembolic Ischemic stroke/TIA	<ul style="list-style-type: none"> ◆ Prescribe antiplatelet agents (IA) <ul style="list-style-type: none"> ◆ Aspirin (50-325 mg daily (IA)) ◆ Aspirin 25 mg plus extended release dipyridamole 200 mg twice daily (IB) ◆ Clopidogrel 75 mg daily (IIa, B) ◆ Do not combine aspirin and clopidogrel for routine stroke prevention (III A) ◆ Do not prescribe oral anticoagulants rather than antiplatelet agents (IA) ◆ If a stroke occurs while taking aspirin, no evidence supports increasing the aspirin dose, changing to an alternative antiplatelet agent or combining antiplatelet agents (IIb, C) 		<ul style="list-style-type: none"> ◆ Check for signs or symptoms of bleeding tendency (e.g., bruising, bleeding, petechiae) ◆ Monitor laboratory tests as appropriate (e.g., fecal occult blood testing, hemoglobin, platelet count)
Atrial fibrillation	<ul style="list-style-type: none"> ◆ Consider long-term anticoagulation with warfarin (IA) (Weigh benefits and risks on the basis of risk factors and comorbid conditions) 	<ul style="list-style-type: none"> ◆ INR 2.5 (range 2–3) 	<ul style="list-style-type: none"> ◆ Check for signs of bleeding ◆ Consider testing periodically for fecal occult blood and hemoglobin ◆ Monitor adequacy of anticoagulation
Atrial fibrillation, but with a hemorrhagic contraindication to oral anticoagulation	<ul style="list-style-type: none"> ◆ Prescribe aspirin alone (IA) ◆ Do not prescribe aspirin plus clopidogrel (III B) 		<ul style="list-style-type: none"> ◆ Check for signs or symptoms of bleeding tendency (e.g., bruising, bleeding, petechiae) ◆ Monitor laboratory tests as appropriate (e.g., fecal occult blood testing, hemoglobin, platelet count)
Mechanical cardiac valve	<ul style="list-style-type: none"> ◆ Long-term anticoagulation with warfarin (IB) (Weigh benefits and risks on the basis of risk factors and comorbid conditions) 	<ul style="list-style-type: none"> ◆ INR 3 (range 2.5–3.5) 	<ul style="list-style-type: none"> ◆ Check for signs or symptoms of bleeding ◆ Monitor laboratory tests as appropriate (e.g., fecal occult blood, hemoglobin) ◆ Monitor adequacy of anticoagulation
Bioprosthetic cardiac valve	<ul style="list-style-type: none"> ◆ Consider warfarin (IIb, C) 	<ul style="list-style-type: none"> ◆ INR 2.5 (range 2-3) 	
Cardiomyopathy (EF ≤ 35%) with sinus rhythm	<ul style="list-style-type: none"> ◆ The benefit of warfarin has not been established (IIb, B) 	<ul style="list-style-type: none"> ◆ May consider warfarin (INR 2-3), aspirin 81 mg daily, clopidogrel 75 mg daily, or aspirin 25 mg plus dipyridamole 75 mg twice daily to prevent recurrent ischemic events (IIb, B) 	<ul style="list-style-type: none"> ◆ Check for signs or symptoms of bleeding ◆ Monitor laboratory tests as appropriate (e.g., fecal occult blood, hemoglobin) ◆ Monitor adequacy of anticoagulation
Patent foramen ovale (PFO)	<ul style="list-style-type: none"> ◆ Antiplatelet therapy (IIa, B) ◆ Data do not support equivalence or superiority of anticoagulants compared to aspirin (IIb, B) ◆ Insufficient data exist regarding PFO closure (IIb, C) 		

APPENDIX 1 continued

ACEI, angiotensin-converting enzyme inhibitor; AF: atrial fibrillation; BP: blood pressure; BUN: blood urea nitrogen; CHD: coronary heart disease; DASH: Dietary Approaches to Stop Hypertension; HbA1C, glycated hemoglobin; INR: international normalized ratio; LDL: low-density lipoprotein; TIA: transient ischemic attack.

Source: Furie KL, Kasner SE, Adams RJ, et al; for the American Heart Association Stroke Council, Council on Cardiovascular Nursing, Council on Clinical Cardiology, and Interdisciplinary Council on Quality of Care and Outcomes Research. Guidelines for the Prevention of Stroke in Patients With Stroke or Transient Ischemic Attack. A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 2010; Oct 21. [Epub ahead of print]

- Sacks FM, Svetkey LP, Vollmer WM, et al; DASH-Sodium Collaborative Research Group. Effects on Blood Pressure of Reduced Dietary Sodium and the Dietary Approaches to Stop Hypertension (DASH) Diet. N Engl J Med 2001;344:3-10.
- ^b Post-Acute and Long-Term Care Medical Association. Managing Diabetes in the Long-Term Care Setting. Clinical Practice Guideline. Columbia, MD.
- ^c Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA 2001; 285(19): 2486-97.

Definition of Classes and Levels of Evidence Used in AHA Recommendations

Class I	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
Class IIa	The weight of evidence or opinion is in favor of the procedure or treatment
Class IIb	Usefulness/efficacy is less well established by evidence or opinion
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful
Therapeutic recommendations	
Level of Evidence A	Data derived from multiple randomized clinical trials or meta-analyses
Level of Evidence B	Data derived from a single randomized trial or nonrandomized studies
Level of Evidence C	Consensus opinion of experts, case studies, or standard of care
Diagnostic recommendations	
Level of Evidence A	Data derived from multiple prospective cohort studies using a reference standard applied by a masked evaluator
Level of Evidence B	Data derived from a single grade A study, or one or more case-control studies, or studies using a reference standard

Source: Furie KL, Kasner SE, Adams RJ, et al. Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011; 42: 227-276.

APPENDIX 2
Quantitative Bleeding Risk Assessment Tool

Bleeding Risk Score				
Factor	Points			
Age	45-54 0	55-64 2	65-74 4	75+ 6
Peripheral arterial disease	No 0	Yes 1		
Congestive heart failure	No 0	Yes 2		
Diabetes	No 0	Yes 1		
Hypercholesterolemia	No 1	Yes 0		
Hypertension	No 0	Yes 2		
Smoking	Never 0	Former 1	Current 2	
Antiplatelet agents	No 0	ASA 1	Other 2	Both 4
Oral anticoagulants	No 0	Yes 4		

Risk Stratification by Use of the Bleeding Risk Score

Bleeding risk score total	Two-year risk of serious bleeding (%)	Risk Category
0-6	0.46%	Low risk
7-8	0.95%	Intermediate risk
9-10	1.25%	Intermediate risk
11-21	2.76%	High risk

Adapted from: Ducrocq G, Wallace JS, Baron G, et al; REACH Investigators. Risk score to predict serious bleeding in stable outpatients with or at risk of atherothrombosis. Eur Heart J 2010;31:1257-65.

APPENDIX 3

Antithrombotic Baseline Risk/Benefit Assessment Tool

Patient Name _____ Date _____

Height		Weight		Creatinine	
Estimated GFR		INR		Stool guaiac	
Urine dip for blood		Hemoglobin		Platelet count	
Stool <i>Helicobacter pylori</i> antigen (optional)		Blood glucose			

Estimated Stroke Risk for Patients With Atrial Fibrillation (Adapted from Gage et al, 2001)

CHADS-2 Score	Strokes/10,000 patients/y	1 point for
0	190	Congestive heart failure or ejection fraction <50%
1	280	
2	400	Hypertension
3	590	Age ≥75
4	850	Diabetes
5	1,250	2 points for
6	1,820	

Benefits of Warfarin Therapy (Adapted from Singer et al, 2004)

- Risk of stroke decreased 65%
- Stroke less likely to cause death or severe disability if taking warfarin

Absolute Contraindications to Warfarin (Adapted from Man-Son-Hing and Laupacis, 2003)

- | | |
|--|--|
| <input type="checkbox"/> Current active bleeding | <input type="checkbox"/> Platelet count <50,000 |
| <input type="checkbox"/> Blood pressure consistently >160/90 | <input type="checkbox"/> Noncompliance with medication or monitoring |

Relative Contraindications to Warfarin (Adapted from Man-Son-Hing, Laupacis, 2003)

- | | |
|--|--|
| <input type="checkbox"/> Ethanol ≥2 oz/d ⁴ | <input type="checkbox"/> Nonselective NSAID without gastric cytoprotection (e.g., PPI, misoprostol) ⁴ |
| <input type="checkbox"/> Extreme functional disability | <input type="checkbox"/> Poor short-term prognosis due to malignancy, advanced chronic disease |

Intracerebral Bleeding Risk for Outpatients With Atrial Fibrillation (per 10,000 patients/y) (Adapted from Levine et al, 2004)

No therapy: 10 ICH; 4 will die	Warfarin: 75 ICH; 43 will die
Aspirin: 20 ICH; 5 will die	

Subdural Hematoma Risk in Outpatient Elderly (per 10,000 people/y) (Adapted from Man-Son-Hing et al, 1999)

No therapy: 4 SDH; 2 will die	Aspirin: 8 SDH; 4 will die
Warfarin: 12 SDH; 4 will die	

Risk of Hospitalization for Central Nervous System Bleeding Among Nursing Home Stroke Survivors (Adapted from Quilliam et al, 2001)

Aspirin: 19/10,000 people/y	Warfarin: 33/10,000 people/y
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APPENDIX 3 continued

Antithrombotic Baseline Risk/Benefit Assessment Tool

Relative Risks of Significant Gastrointestinal Bleeding (Adapted from Man-Son-Hing and Laupacis, 2003; 2002)

<input type="checkbox"/> History of active peptic or duodenal ulcer bleeding ³	30% chance of rebleeding in 5 y; 13.5 times excess risk compared to those with negative PMH
	No increased risk if treated for <i>H. pylori</i>
<input type="checkbox"/> Taking NO warfarin, aspirin, NSAID ⁷	RR 1; 117 upper-GI bleed/10,000 people/y (16 people will die)
<input type="checkbox"/> Taking warfarin ⁷	RR 2.4; 280 upper-GI bleed/10,000 people/y (42 people will die)
<input type="checkbox"/> Taking aspirin ⁷	RR 1.2; 140 upper-GI bleed/10,000 people/y (7 people will die)
<input type="checkbox"/> Taking nonselective NSAID ⁷	RR 3.8; 450 upper -GI bleed/10,000 people/y
<input type="checkbox"/> Taking COX-2 selective NSAID ⁷	RR 1.9; 320 upper-GI bleed/10,000 people/y
<input type="checkbox"/> Taking PPI or misoprostol with NSAID ^{7,8}	RR 1.9; 320 upper-GI bleed/10,000 people/y

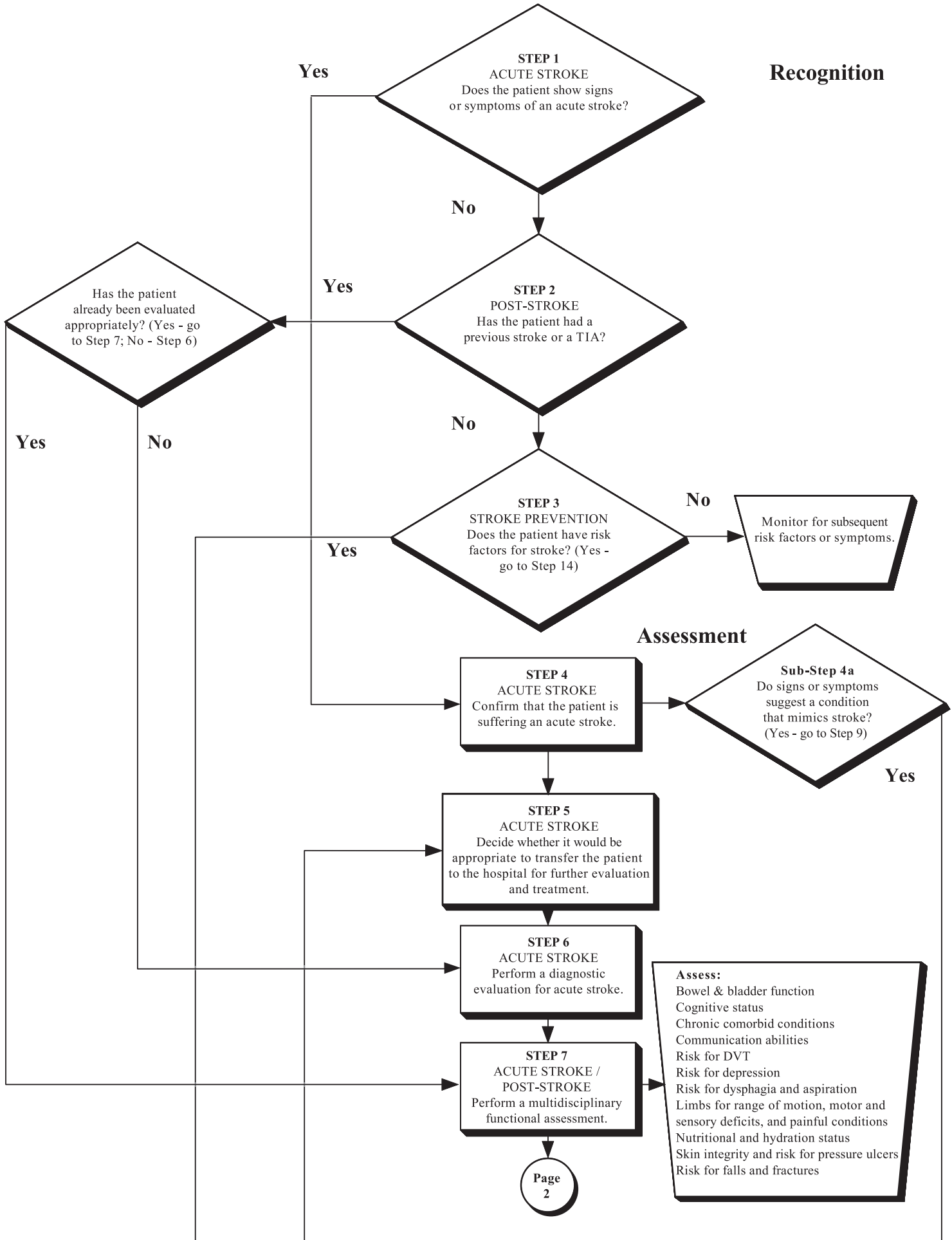
Conditions That May Increase Bleeding Risk and Require More Frequent Monitoring^{5,9} (Adapted from Levine et al, 2004⁵; Beyth et al, 1998⁹)

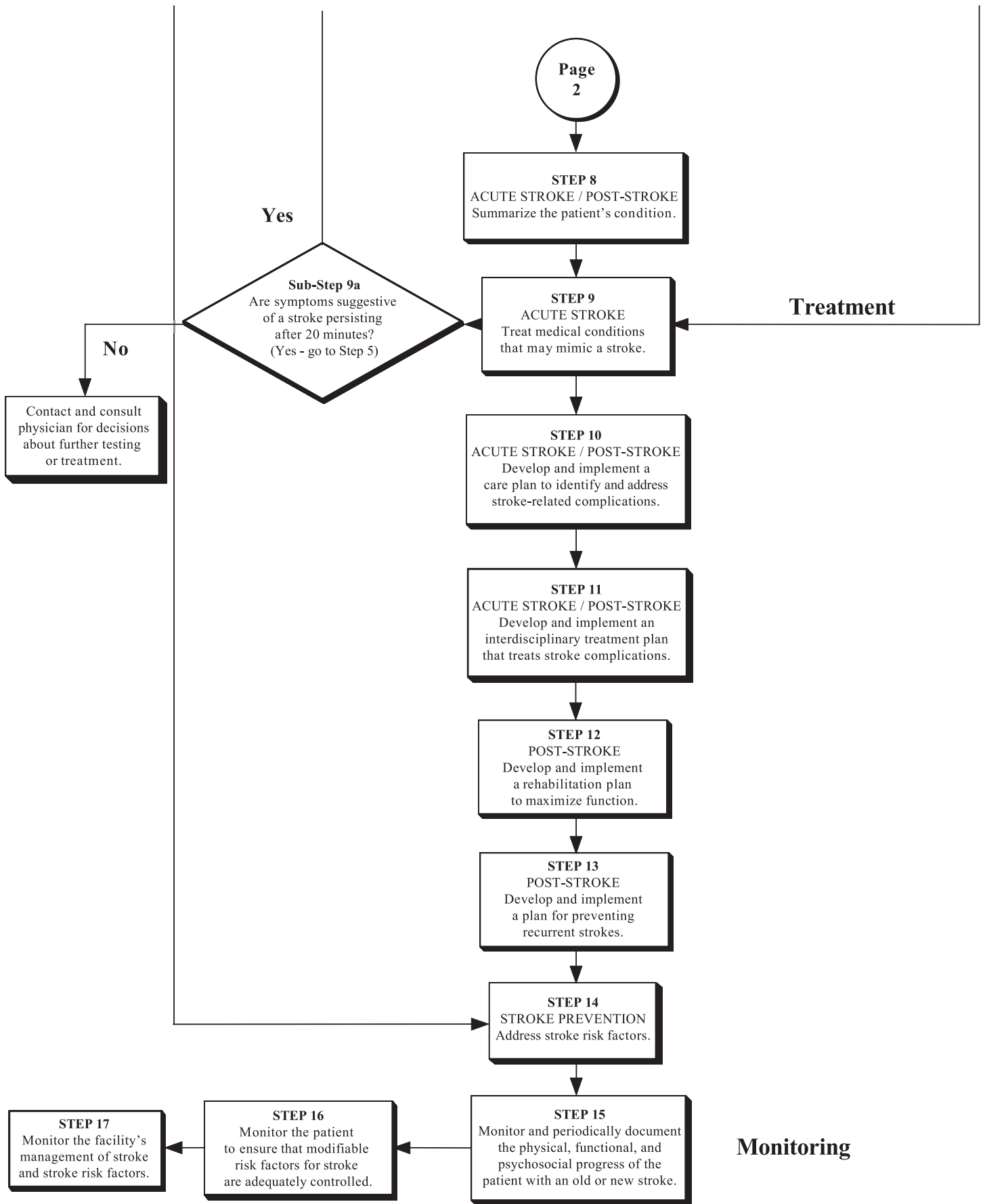
<input type="checkbox"/> Prior stroke ⁴	<input type="checkbox"/> Malignancy ⁴	<input type="checkbox"/> GFR<30 ⁴
<input type="checkbox"/> Liver disease ⁴	<input type="checkbox"/> Malnutrition ⁴	<input type="checkbox"/> Hematocrit <30% ⁸
<input type="checkbox"/> Serum creatinine >1.5 mg/dL ⁸	<input type="checkbox"/> Diabetes ⁸	
<input type="checkbox"/> Age ≥65 ⁸	<input type="checkbox"/> Recent MI ⁸	

COX-2: cyclooxygenase 2 inhibitor; GFR: glomerular filtration rate; GI: gastrointestinal; ICH: intracerebral hemorrhage; INR: international normalized ratio; MI: myocardial infarction; NSAID: nonsteroidal anti-inflammatory drug; PMH: past medical history; PPI: proton pump inhibitor; RR: relative risk; SDH: subdural hematoma; TIA: transient ischemic attack.

Sources: Beyth RJ, Quinn LM, Landefeld CS. Prospective evaluation of an index for predicting the risk of major bleeding in outpatients treated with warfarin. *Am J Med* 1998;105:91-99.
 Gage BF, Waterman AD, Shannon W, et al. Validation of clinical classification schemes for predicting stroke: Results from the National Registry of Atrial Fibrillation. *JAMA* 2001; 285: 2864-2870;
 Levine MN, Raskob G, Beyth RJ, et al. Hemorrhagic complications of anticoagulant treatment: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126: 287S-310S.
 Man-Son-Hing M, Laupacis A. Anticoagulant-related bleeding in older persons with atrial fibrillation: Physicians' fears often unfounded. *Arch Intern Med* 2003; 163: 1580-1586.
 Man-Son-Hing M, Laupacis A. Balancing the risks of stroke and upper gastrointestinal tract bleeding in older patients with atrial fibrillation. *Arch Intern Med* 2002; 162: 541-550.
 Man-Son-Hing M, Nichol G, Lau A, Laupacis A. Choosing antithrombotic therapy for elderly patients with atrial fibrillation who are at risk for falls. *Arch Intern Med* 1999; 159: 677-685.
 Quilliam BJ, Lapane KL, Eaton CB, Mor V. Effect of antiplatelet and anticoagulant agents on risk of hospitalization for bleeding among a population of elderly nursing home stroke survivors. *Stroke* 2001; 32: 2299-2304.
 Singer DE, Albers GW, Dalen JE, et al. Antithrombotic therapy in atrial fibrillation: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126: 429S-456S.
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This is the stroke management in the long-term care setting algorithm to be used in conjunction with the written text of this clinical practice guideline. The numbers next to the different components of the algorithm correspond with the steps in the text.





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