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Comprehensive Overview of Nursing and Interdisciplinary Care of the Acute Ischemic Stroke Patient A Scientific Statement From the American Heart Association

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I schemic stroke represents 87% of all strokes.¹ As worldwide initiatives move forward with stroke care, healthcare providers and institutions will be called on to deliver the most current evidence-based care. The American Heart Association/American Stroke Association (AHA/ASA) charged a panel of healthcare professionals from several disciplines with developing a practical, comprehensive overview of care for the patient with acute ischemic stroke (AIS). This article focuses on educating nursing and allied healthcare professionals about the roles and responsibilities of those who care for patients with AIS.

Nurses play a pivotal role in all phases of care of the stroke patient. For the purposes of this article, the writing panel has defined 2 phases of stroke care: (1) The emergency or hyperacute care phase,^{2,3} which includes the prehospital setting and the emergency department (ED), and (2) the acute care phase, which includes critical care units, intermediate care units, stroke units, and general medical units.

Stroke is a complex disease that requires the efforts and skills of all members of the multidisciplinary team. Nurses are often responsible for the coordination of care throughout the continuum.^{4–9} Coordinated care of the AIS patient results in improved outcomes, decreased lengths of stay, and decreased costs.¹⁰

In developing this comprehensive overview, the writing panel applied the rules of evidence and formulation of strength of evidence (recommendations) used by other AHA writing groups¹¹ (Table 1). We also cross-reference other AHA guidelines as appropriate.

Overview of Stroke

It is important that nurses understand the burden of stroke as a public health issue in the United States. This will guide them in developing appropriate skills to care for AIS patients and to educate patients and families about secondary stroke prevention.

Epidemiology of Stroke

The AHA estimates that \approx 780 000 strokes occur each year; 600 000 of these are new strokes, and \approx 180 000 are recurrent strokes.¹ Eighty-seven percent are ischemic strokes, 10% are intracranial hemorrhages (ICH), and 3% are subarachnoid hemorrhages (SAH). In 2007, the overall mortality rate from stroke was 273 000, which makes stroke the third-leading cause of death in the United States.¹ Between 1979 and 2005, the annual number of hospital discharges with stroke as the diagnosis was \approx 900 000.¹ Direct and indirect costs associated with stroke are estimated to be approximately \$65.5 billion.¹ Direct costs are attributed to the initial hospitalization, skilled nursing care, physician and nursing care, medications and durable medical equipment, home health care, and acute rehabilitation. Indirect costs include loss of productivity (loss of future earnings) due to morbidity and

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⁽Stroke. 2009;40:2911-2944.)

Table 1. Applying Classification of Recommendations and Levels of Evidence

	CLASS I	CLASS IIa	CLASS IIb	CLASS III
	Benefit >>> Risk Procedure/Treatment SHOULD be performed/ administered	Benefit >> Risk Additional studies with focused objectives needed IT IS REASONABLE to per- form procedure/administer treatment	Benefit ≥ Risk Additional studies with broad objectives needed; additional registry data would be helpful Procedure/Treatment MAY BE CONSIDERED	Risk ≥ Benefit Procedure/Treatment shou NOT be performed/adminis tered SINCE IT IS NOT HEL FUL AND MAY BE HARMFO
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	 Recommendation that procedure or treatment is useful/effective Sufficient evidence from multiple randomized trials or meta-analyses 	 Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from multiple randomized trials or meta-analyses 	 Recommendation's usefulness/efficacy less well established Greater conflicting evidence from multiple randomized trials or meta-analyses 	 Recommendation that procedure or treatment is not useful/effective and may be harmful Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	 Recommendation that procedure or treatment is useful/effective Evidence from single randomized trial or nonrandomized studies 	 Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from single randomized trial or nonrandomized studies 	 Recommendation's usefulness/efficacy less well established Greater conflicting evidence from single randomized trial or nonrandomized studies 	 Recommendation that procedure or treatment is not useful/effective and may be harmful Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	 Recommendation that procedure or treatment is useful/effective Only expert opinion, case studies, or standard of care 	 Recommendation in favor of treatment or procedure being useful/effective Only diverging expert opinion, case studies, or standard of care 	 Recommendation's usefulness/efficacy less well established Only diverging expert opinion, case studies, or standard of care 	 Recommendation that procedure or treatment is not useful/effective and may be harmful Only expert opinion, case studies, or standard of care
Suggested phrases for writing recommendations!	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	is not recommended is not indicated should not is not useful/effective/beneficial may be harmful

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as gender, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use. A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Even though randomized trials are not available, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

†In 2003, the ACC/AHA Task Force on Practice Guidelines developed a list of suggested phrases to use when writing recommendations. All guideline recommendations have been written in full sentences that express a complete thought, such that a recommendation, even if separated and presented apart from the rest of the document (including headings above sets of recommendations), would still convey the full intent of the recommendation. It is hoped that this will increase readers' comprehension of the guidelines and will allow queries at the individual recommendation level.

mortality and loss of esteem (place in family and society) due to disability.

Demographics of Stroke

Each year, women experience $\approx 60\ 000$ more strokes than men do.¹ American Indian/Alaskan Native persons have the highest prevalence of stroke (6.7%), followed by persons of multiple races (4.6%). Black men and women have twice the prevalence of stroke as whites (4.0% versus 2.3%).¹ Blacks lead all races or ethnic groups for first-ever stroke. The prevalence of stroke in Hispanic or Latino populations is 3.1% for men and 1.9% for women compared with 2.4% for non-Hispanic white men and 2.7% for non-Hispanic white women.¹ Epidemiologists note the increasing risk of stroke with advancing age, comparable to the risk of Alzheimer's disease. Unless individuals change modifiable risk factors, the large aging American population is faced with rising stroke morbidity and a greater than ever public healthcare burden.⁹

Etiology of Stroke (Stroke Subtypes)

Strokes caused by blocked blood vessels to the brain, or ischemic strokes, lead to cerebral infarction, whereas hemorrhagic strokes caused by ruptured vessels in and around the brain lead to ICH and SAH. The AHA has published guidelines for the medical management of patients with ischemic stroke^{12,13} and strokes caused by ICH¹⁴ and SAH.¹⁵ Nursing and allied health management is not provided in these guidelines and thus is outlined here.

Ischemic strokes are commonly caused by atherosclerotic disease of extracranial or intracranial vessels that circulate blood to the brain. Approximately 20% of ischemic strokes are caused by large-vessel atherosclerosis (extracranial or intracranial segments of carotid or vertebrobasilar arteries), and $\approx 25\%$ of ischemic strokes are due to penetrating artery disease (small-vessel disease) that causes lacunar or subcortical strokes. Another 20% are caused by cardiogenic embo-

lism, most frequently from atrial fibrillation.¹⁶ Approximately 30% of ischemic strokes are termed cryptogenic, for which the exact cause of stroke remains unknown.¹⁶

Hemorrhagic stroke is commonly caused by either primary ICH or SAH. Overall, ICH accounts for $\approx 10\%$ of all strokes and SAH for $\approx 3\%$.¹ Common causes and risk factors for ICH are hypertension (the number 1 cause), bleeding disorders, African-American ethnicity, aging, vascular malformations, excessive use/abuse of alcohol, and liver dysfunction.^{17–20} The primary cause of SAH is a ruptured cerebral aneurysm.

Crossing the Continuum of Care

Phase 1 of stroke care, the emergency or hyperacute phase, encompasses the first 3 to 24 hours after onset of stroke. This phase generally incorporates the prehospital (activation of emergency medical services [EMS]/9-1-1 and response) and ED care protocols. The focus is on identifying stroke symptoms and infarct location, assessing the patient for risk of acute and long-term complications, and determining treatment options.

Phase 2 includes acute care, which encompasses the period from 24 to 72 hours after onset of stroke. In this phase, the focus is on clarifying the cause of stroke, preventing medical complications, preparing the patient and family for discharge, and instituting long-term secondary prevention modalities.

The Emergency or Hyperacute Phase of AIS Care

Optimal management of the AIS patient in the emergency or hyperacute phase of AIS care requires an accurate and systematic evaluation that is coordinated and timely. Once a potential stroke is suspected, EMS personnel and nurses must determine the time at which the patient was last known to be well (last known well time). This time is the single most important determinant of treatment options during the hyperacute phase.

The Nurse's Role

In the prehospital setting, the leading healthcare team member is the emergency medical technician (EMT) or paramedic. Nurses may work as EMTs and paramedics, radio providers of online medical control to EMS personnel from base stations, and educators who teach EMS personnel about stroke and the care of stroke patients.

The key elements of prehospital care are stabilization of the airway, breathing, and circulation (the ABCs); identification of signs and symptoms of stroke; establishment or verification of the last known well time; provision of supplemental oxygen to patients with hypoxemia; checking the blood glucose level; avoidance of the administration of glucose-containing fluids (unless the patient is hypoglycemic); rapid initiation of transport (load and go); and delivery of patients to receiving centers capable of rapidly caring for acute stroke.²¹ When recombinant tissue plasminogen activator (rtPA) was approved as the first acute treatment for AIS, the paradigm of care of the stroke patient shifted, and emergency care of the stroke patient in the field emerged.²² The role of time in determining treatment eligibility and patient outcome has generated a body of literature and knowledge about appropriate care for AIS patients,^{22–35,37–45} from which healthcare providers have developed measures to quickly and easily identify and assess stroke patients.^{46,47} Textbooks and training courses for EMTs and paramedics discuss stroke pathophysiology and identify stroke as a medical emergency. Understanding and recognizing specific stroke symptoms can be challenging.^{22–24,28,47–50} Evaluation of EMS practices has shown that stroke-specific knowledge has been deficient³⁸ but that both overall knowledge²⁸ and identification of stroke symptoms by EMTs and paramedics can be improved with additional stroke-specific education.^{19–25,28,31,47–50}

In many community and academic institutions, education of EMS providers has become a function of the nurse educator, a role that has expanded to the community. The most widely available stroke teaching tool for this purpose²¹ is chapter 9 of the AHA stroke module.^{44,51} Educational videos and other tools are also available for the EMS audience.⁵²

Before beginning an EMS stroke education program, the nurse educator should verify local policies and regulations governing acceptable practice for paramedics and EMTs in that region or state. For example, in some communities, EMS providers are not permitted to perform some recommended practices for acute stroke care, such as determining finger-stick glucose levels and starting intravenous lines. In other communities, higher standards that require specific assessment skills have been developed for EMS to enable them to respond more aggressively to AIS.^{10,27,47}

As a part of their continuing education, EMS personnel must also be provided with accurate information about acute stroke care and treatment capabilities in their community.⁴⁷ EMS units should know which hospitals are equipped to provide specific emergency stroke care, such as those certified by the Joint Commission or their state health agencies.^{10,53–56}

Continuing education of EMS personnel is challenging and requires frequent updates. The nurse educator should keep in mind that a typical EMS provider cares for 4 to 10 stroke patients in a given year.⁴⁴ As a result, field experience may be limited, and reinforcement of knowledge and practice in caring for acute stroke patients will be necessary. One study concluded that the knowledge gained from stroke training decreased by \approx 50% over 1 year⁵⁷; therefore, educational programs about stroke might be repeated from 1 to several times per year. In some states with mandated stroke systems of care, EMS updates in stroke education will be required.²¹ The National Institutes of Health (NIH) proceedings *Improving the Chain of Recovery for Acute Stroke in Your Community* is a useful resource for planning and organizing stroke educational programs.⁴⁷

Education Regarding Prehospital Assessment for Acute Stroke

Although accurate identification of stroke symptoms is a critical success factor in early stroke treatment, the nurse educator needs to include additional aspects of prehospital stroke patient management.^{28,58,59} Recognition of stroke symptoms is an important factor in successful delivery of

Cincinnati Prehospital Stroke Scale ³²	Los Angeles Prehospital Stroke Screen ⁶⁰	Los Angeles Motor Scale ³⁴	Face Arm Speech Test ⁶¹	SNIHSS-565
Facial droop	Facial weakness	Facial weakness	Facial palsy	Right leg motor
Arm weakness	Arm strength	Arm strength	Arm weakness	Left leg motor
Speech	Grip	Grip	Speech impairment	Gaze
Screening criteria (4 items)				Visual fields
	Blood glucose			Language

Table 2. Components of Selected Prehospital Assessment Tools

SNIHSS indicates shortened National Institutes of Health Stroke Scale.

proven acute therapies. Prehospital assessment tools have been developed to help enhance recognition of stroke symptoms and improve the ability to identify stroke patients in the field (Table 2). The most common and well-investigated tools are the Cincinnati Prehospital Stroke Scale and the Los Angeles Prehospital Stroke Screen.^{22,30,32,60} Newer stroke identification tools include the Face Arm Speech Test⁶¹ (Table 3), which is similar to the Cincinnati Prehospital Stroke Scale, and the Melbourne Prehospital Stroke Scale,^{62–64} which is similar to the Los Angeles Prehospital Stroke Screen. Tools to rate stroke severity in the field have been developed, including a shortened version of the NIH Stroke Scale (NIHSS)65 and the Los Angeles Motor Scale.34 If a specific tool has not been identified for use within a community, the nurse educator should review the common signs and symptoms of stroke such as those identified on the Brain Attack Coalition's World Wide Web site (http:// www.stroke-site.org/index.html). These tools may also be used to teach a quick stroke screening evaluation to hospital personnel, because in-hospital strokes also require prompt recognition and action.

EMS personnel have long understood the implications of time to treatment for myocardial infarction and trauma, but the concept of time dependency in AIS is relatively new. The nurse educator must emphasize the importance of obtaining the last known well time.

Stroke education should emphasize that stroke requires high-priority status and that the load-and-go philosophy has now become a part of mainstream acute stroke care in the field.⁴¹ rtPA is the only treatment for AIS approved by the US Food and Drug Administration, and it ideally is administered within 3 hours of symptom onset.^{13,59,66,67} On-site stroke patient assessments should be performed expeditiously, and transport should be initiated as soon as the patient's condition is assessed as stable.^{44,66,68,69} Although the symptoms listed in the formal prehospital stroke assessment tools are classic, other stroke symptoms should also be described to EMS personnel. Table 4 lists neurological symptoms associated with strokes that occur in the different cerebral territories.

Table 3. Sample Face Arm Speech Test (FAST)⁶¹

Yes RT LT	No	Unknown
Yes RT LT	No	Unknown
Yes	No	Unknown
	Yes RT LT	Yes RT LT No

RT indicates right side; LT, left side.

Recommendations

Class I

- 1. To increase the number of stroke patients who receive timely treatment, educational programs for physicians, hospital personnel, and EMS personnel are recommended (*Class I, Level of Evidence B*).
- 2. Stroke education of EMS personnel should be provided on a regular basis, perhaps as often as twice per year, to ensure proper recognition, field treatment, and delivery of patients to appropriate facilities (*Class I, Level of Evidence C*).

Table 4.The 5 Key Stroke Syndromes: Classic SignsReferable to Different Cerebral Areas

Left (dominant hemisphere)	
Left gaze preference	
Right visual field deficit	
Right hemiparesis	
Right hemisensory loss	
Right (nondominant hemisphere)	
Right gaze preference	
Left visual field deficit	
Left hemiparesis	
Left hemisensory loss neglect (left hemi-inattention))
Brainstem	
Nausea and/or vomiting	
Diplopia, dysconjugate gaze, gaze palsy	
Dysarthria, dysphagia	
Vertigo, tinnitus	
Hemiparesis or quadriplegia	
Sensory loss in hemibody or all 4 limbs	
Decreased consciousness	
Hiccups, abnormal respirations	
Cerebellum	
Truncal/gait ataxia	
Limb ataxia, neck stiffness	
Hemorrhage	
Focal neurological deficits as in AIS	
Headache (especially in SAH)	
Neck pain	
Light intolerance	
Nausea, vomiting	
Decreased level of consciousness	

Education Priorities for Assessment and Treatment in the Field

Neurological assessment of the AIS patient should always include the ABCs, vital signs, cardiac monitoring during transport, and baseline neurological assessment. Because the field neurological examination will serve as a baseline for assessment of neurological improvement or worsening, the use of a prehospital stroke scale is recommended.

EMS personnel on the scene should ask the patient's family or bystanders when the patient was last known to be normal or without neurological deficits, ie, the last known well time. Documentation of this report of onset can be helpful in establishing an accurate time of stroke symptom onset.44,46,47,69 Ideally, standardized definitions should be developed in EMS systems to define the specific onset date and time. The date and time should be defined as the time when the stroke symptoms that brought the patient to the hospital first occurred. A specific time can be identified within a reasonable amount of certainty within ± 15 minutes. When possible, the information should be obtained directly from the patient. If the patient is unable to give this information, EMS personnel should look to another reliable source for this information.⁷⁰ If the time of onset of stroke symptoms is not identifiable, a standard method of time parameters should be used, such as morning (6:00 AM to 11:59 AM), afternoon (noon to 5:59 PM), evening (6:00 PM to 11:59 PM), and overnight (midnight to 5:59 AM).70 EMS providers must emphasize to families the importance of traveling to the hospital with the patient, particularly if symptom onset is within the time frame for rtPA administration and the patient's language or decision-making capability is compromised. When family members cannot accompany the patient, EMS personnel should document the family's contact information and provide it to the emergency physician.13

The current guidelines recommend the use of continuous cardiac monitoring during transport of a suspected stroke patient to determine the presence of cardiac arrhythmias.13 If there is no standing field protocol for management of cardiac conditions, EMS personnel should contact the base station or receiving institution if the electrocardiogram demonstrates possible acute myocardial ischemia or atrial fibrillation. Blood pressure should be monitored every 15 minutes, or more often if severe hypertension (systolic blood pressure >200 mm Hg) or relative hypotension (systolic blood pressure <110 mm Hg) is observed during transport. Administration of antihypertensive drugs in the field is not recommended, because induced hypotension carries a possible risk of extending the area of cerebral infarct.47,58,68,71 Supplemental oxygen should be given to hypoxic patients; in ambulances without oximetry capabilities, oxygen can be administered at low levels, eg, 2 to 3 L/min. If pulse oximetry is available and the patient's oxygen saturation is >92%, additional oxygen is not needed.13 Transport with the head of the bed elevated $\approx 30^{\circ}$ may help with oxygenation and may minimize the possibility of aspiration.44,71,72 To decrease the risk of aspiration, the patient should receive nothing by mouth (NPO).

Hypoglycemia, a common stroke mimic, can be identified quickly by measuring blood glucose during transport. Fingerstick tests can be performed if the emergency vehicle is appropriately equipped and personnel are trained. Treatment of severe hypoglycemia should be instituted promptly by EMS personnel. Intravenous access can be established in the field, and non–glucose-containing intravenous fluids can be started if the patient is hypotensive. Establishment of intravenous access should not delay transport.^{44,46,47,57,69,71}

Finally, the nurse educator should emphasize the value of early notification of the receiving ED of the arrival of a potential acute stroke patient. Historic cardiac trials have shown that prearrival notification of the ED enhances rapid diagnostic workup, reducing time between symptom onset and treatment.^{23,25,73}

Recommendations

Class I

- 1. EMS personnel should be trained to administer a validated prehospital stroke assessment, such as the Cincinnati Prehospital Stroke Scale or the Los Angeles Prehospital Stroke Screen (*Class I, Level of Evidence B*).
- 2. EMS personnel should be trained to determine the last known well time using standardized definitions to collect the most accurate information (*Class I, Level of Evidence B*).
- 3. EMS personnel should use the neurological/stroke assessment approach to gather basic physiological information about the patient and communicate the patient's condition to the receiving hospital (*Class I, Level of Evidence B*).

From the Field to the ED: Stroke Patient Triage and Care

Emergency personnel initiate basic triage and care modalities in the field. Once the stroke patient arrives in the ED, patient triage is usually a function of nursing staff. The Emergency Nurses Association and the American College of Emergency Physicians recommend a 5-level Emergency Severity Index as a preferred system for triage in a busy ED.⁵² This index puts all stroke patients in the level 2 or "needs immediate assessment" category, the same as for an unstable trauma patient or a critical care cardiac patient.^{46,52,69} The emergency nurse must be able to recognize neurological symptoms that suggest stroke and rapidly assess the initial time of symptom onset or the last known well time.^{44,46,57}

The triage nurse should use specialized checklists, protocols, and other tools to identify stroke patients.10,44,47,57,74 Once stroke is confirmed, the nurse uses these procedures and protocols that define who contacts the acute stroke team or appropriate neurological consultant. Emergency nurses understand that time is critical and are trained in rapid assessment and treatment of stroke patients. Studies have shown that the sooner thrombolytic therapy is started, the greater the benefit.23,44,46,75 It is critical that all emergency nurses and other emergency professional staff know that the NIH-National Institute of Neurological Disorders and Stroke (NINDS) benchmark treatment time for AIS with intravenous rtPA is within 60 minutes of arrival in the ED⁴⁶ (Table 5). In some cases, this time will need to be shortened to successfully initiate thrombolytic therapy within 3 hours of stroke onset, although there is growing evidence of safety and effective-

Table 5.NINDS Time Targets for Organized Triage of AcuteStroke Patients⁴⁴: Key Evaluation Time Targets for the PotentialrtPA Candidate

Maximum intervals recommended by NINDS	
Door-to-doctor first sees patient	10 min
Door-to-CT completed	25 min
Door-to-CT read	45 min
Door-to-thrombolytic therapy starts	60 min
Physician examination	15 min
Neurosurgical expertise available*	2 h
Admitted to monitored bed	3 h

CT indicates computed tomography.

*On-site or by transfer to another facility.

ness beyond the 3-hour window from stroke onset.^{76,77} The AHA AIS Writing Committee has issued a Science Advisory stating that some eligible patients may be treated between the 3- and 4.5-hour window after stroke. The recommendation comes with several caveats and follows the inclusion criteria described in the ECASS III results. The exceptions include persons >80 years of age, those taking oral anticoagulants with an international normalized ratio of <1.7, persons with an NIHSS score >25, and those with a history of stroke and diabetes.^{77a}

Recommendations

Class I

- 1. EDs should establish standard operating procedures and protocols to triage stroke patients expeditiously (*Class I, Level of Evidence B*).
- 2. Standard procedures and protocols should be established for benchmarking time to evaluate and treat eligible stroke patients with rtPA expeditiously (*Class I*, *Level of Evidence B*).
- 3. Target treatment with rtPA should be within 1 hour of the patient's arrival in the ED (*Class I, Level of Evidence A*).
- 4. Eligible patients can be treated between the 3- to 4.5-hour window when evaluated carefully for exclusions to treatment (*Class I, Level of Evidence B*).

Emergency Nursing Interventions in the Emergency/Hyperacute Phase of Stroke

The First 24 Hours

Stroke symptoms typically begin suddenly (but can evolve over minutes to hours) and are referable to the affected region of the brain. Ischemic stroke symptoms are generally divided into those that affect the anterior and posterior cerebral circulation (Table 4). To properly triage patients for AIS therapies such as rtPA, emergency nurses should be familiar with both typical and unusual stroke presentations.

As in the prehospital phase, initial patient assessments made by the emergency nurse are based on the principle of assessing the ABCs, vital signs, and neurological assessment. The majority of AIS patients will present to the ED in a hemodynamically stable condition; however, ischemic strokes involving the posterior circulation can require aggressive airway management, especially if the patient has an altered level of consciousness.^{26,78}

Circulatory collapse or cardiac arrest, although possible, is uncommon in isolated ischemic stroke.⁷⁹ The occurrence of either may indicate other medical conditions such as acute myocardial infarction, atrial fibrillation, or congestive heart failure. Cardiac monitoring of all suspected stroke patients in the ED helps identify these conditions.⁶⁹

Initial ED documentation of the stroke patient begins with the recording of all information included in the neurological/ stroke assessment. Vital signs, including temperature, may be measured frequently as clinically indicated but not less than every 30 minutes while the patient is in the ED. Hyperthermia is associated with poor outcome in stroke patients^{13,80–82}; therefore, it is important to consider treating any fever >99.6°F. During the 60-minute infusion of thrombolytic therapy, pulse and blood pressure should be checked at least every 15 minutes. Table 6 summarizes nursing care associated with thrombolysis and nonthrombolysis treatment of patients with acute cerebral ischemia on the basis of the original NIH-NINDS study protocol and AHA/ASA guidelines.^{2,29,83–86}

Brain Imaging

The overriding objective of emergency evaluation of the stroke patient is to determine whether the stroke is an ischemic infarction or ICH and to exclude a nonvascular lesion as the cause of symptoms. Additional objectives are to localize the lesion, determine its age and extent, and document its mechanism. Both computed tomography (CT) and magnetic resonance imaging (MRI) are acceptable initial imaging modalities for acute evaluation. The most commonly obtained study remains an immediate unenhanced (noncontrast) head CT scan.¹³ The emergency nurse prepares the patient for CT or MRI by explaining the test and may help transport the patient to the scanner. The nurse should prenotify the CT department that a patient with suspected acute stroke is in transport. This will allow technicians to reserve the scanner so that the patient can be imaged immediately on arrival. The CT scan should be completed in ≤ 25 minutes in patients who are eligible for treatment with rtPA. The initial scan is one of the most important diagnostic tests in the emergency phase after stroke. Rapid acquisition and results of imaging will define treatment.

Oxygenation, Positioning, and Oral Intake

Patients with AIS are at risk of hypoxemia and oxygen desaturation. Maximization of oxygenation of all acute stroke patients has been examined in 1 quasi-randomized trial and did not show clear findings of benefit from supplemental oxygen.⁸⁷ There is general agreement, however, that hypoxic patients will benefit from supplemental oxygen.¹³

Positioning of the head of the bed must be individualized for each patient. The traditional positioning at 25° to 30° is often used for potentially increased intracranial pressure (ICP), at least until large lobar, ICH, space-occupying lesions or other causes of increased ICP can be ruled out by imaging.⁸⁸ Stroke patients with increased ICP and chronic respiratory conditions may need head elevation for maximum

Table 6. Schedule of Neurological Assessment and Vital Signs and Other Acute Care Assessments in Thrombolysis-Treated and Nonthrombolysis-Treated Patients^{2,29,83-86}

Thrombolysis-Treated Patients	Nonthrombolysis–Treated Patients
Neurological assessment and vital signs (except temperature) every 15 min during rtPA infusion, then every 30 min for 6 h, then every 60 min for 16 h (total of 24 h) Note: Frequency of BP assessments may need to be increased if systolic BP stays \geq 180 mm Hg or diastolic BP stays \geq 105 mm Hg. Temperature every 4 h or as required. Treat temperatures $>$ 99.6°F with acetaminophen as ordered	In ICU, every hour with neurological checks or more frequently if necessary. In non-ICU setting, depending on patient's condition and neurological assessments, at a minimum check neurological assessment and vital signs every 4 h
Call physician if systolic BP >185 or <110 mm Hg; diastolic BP >105 or <60 mm Hg; pulse <50 or >110 per min; respirations >24 per min; temperature >99.6°F; or for worsening of stroke symptoms or other decline in neurological status	Call physician for further treatment based on physician and institutional preferences/guidelines: Systolic BP >220 or <110 mm Hg; diastolic BP >120 or <60 mm Hg; pulse <50 or >110 per min; temperature >99.6°F; respirations >24 per min; or for worsening of stroke symptoms or other decline in neurological status
For 0_2 saturation <92%, give 0_2 by cannula at 2 to 3 L/min	For 0_2 saturation <92%, give 0_2 by cannula at 2 to 3 L/min
Monitor for major and minor bleeding complications	N/A
Continuous cardiac monitoring up to 72 h or more	Continuous cardiac monitoring for 24 to 48 h
Measure intake and output	Measure intake and output
Bed rest	Bed rest
IV fluids NS at 75–100 mL/h	IV fluids NS at 75–100 mL/h
No heparin, warfarin, aspirin, clopidogrel, or dipyridamole for 24 h, then start antithrombotic as ordered	Antithrombotics should be ordered within first 24 h of hospital admission
Brain CT or MRI after rtPA therapy	Repeat brain CT scan or MRI may be ordered 24 to 48 h after stroke or as needed

BP indicates blood pressure; ICU, intensive care unit; N/A, not applicable; IV, intravenous; NS, normal saline; and MRI, magnetic resonance imaging.

oxygenation.⁸⁹ The bed should be elevated at least 30° if the patient is at risk of aspiration or airway obstruction due to dysphagia.²⁷ The optimal position for the head of the bed has not been identified.^{89–91} Recent studies have suggested that positioning of the head of the bed can facilitate an increase in cerebral blood flow and maximize oxygenation to cerebral tissue.^{92–94} A study using transcranial Doppler technology found that the head-flat position maximized blood flow to the brain.⁹⁴ Further studies on head positioning of the AIS patient need to be completed; if the patient has a lower risk of increased ICP and is not at risk for aspiration, the head-down position has been shown to be beneficial.⁹⁴

When significant hemiparesis is present, positioning on the paretic side may be more desirable to allow the patient to communicate and to prevent aspiration.⁷² AIS patients are more prone to atelectasis as a result of immobility regardless of position. The patient's neck should be kept straight, airway patency maintained, and slumped sitting avoided to prevent hypoxia.⁸⁹

Patients are kept NPO, including no oral medications, until ability to swallow can be assessed. Emergency nurses may be trained to perform a bedside swallowing assessment to establish whether the patient can safely receive oral intake and swallow ED medications such as aspirin.^{13,27,89} If swallowing is impaired, medications can be administered rectally or by nasogastric tube.

Laboratory Assessments

An electrolyte imbalance can sometimes produce strokelike symptoms. A comprehensive metabolic panel indicates fluid and electrolyte status. The blood and hemostatic system can be assessed by a complete blood count with platelet and coagulation studies such as prothrombin time, international normalized ratio, activated partial thromboplastin time, and fibrinogen. Urinalysis assesses renal function and coexisting urinary tract infection (UTI). Emergent laboratory specimens should be labeled STAT to expedite processing, if the patient is in the time window to receive thrombolytics. Table 7 lists the most commonly ordered stroke laboratory tests to measure eligibility to receive rtPA.

Intravenous Access

Ideally, 2 to 3 intravenous sites should be established if the acute stroke patient will receive thrombolytic therapy. One site is used for administration of intravenous fluids, another for administration of thrombolytic therapy, and the third for administration of intravenous medications.¹³ Diagnostic laboratory blood specimens should be drawn before intravenous fluids are started. Collection of specimens before the patient undergoes imaging allows simultaneous processing of both laboratory and imaging data and facilitates rapid turnaround.

Intravenous Fluids

Glucose can have detrimental effects in acute brain injury of all types. Therefore, intravenous solutions with glucose (such as D_5W [dextrose 5% in water]) should be avoided in AIS patients in the ED.^{13,27} An infusion rate that maintains normovolemia (75 to 100 mL/h) can help facilitate normal circulating blood volume. Stroke patients often present in a hypovolemic state, which may produce hypotension and cerebral hypoperfusion. In these instances, intravenous fluid

Table 7. Emergency Stroke Workup

All patients	Time patient last known well (will be used as presumed time of onset)		
Noncontrast brain CT or brain MRI	Time symptoms were first observed (if different from time last known well)		
Blood glucose	Was anyone with patient when symptoms began? If so, who?		
Serum electrolytes/renal function tests	History of diabetes?		
ECG	History of hypertension?		
Markers of cardiac ischemia	History of seizures?		
Complete blood count, including platelet count*	History of trauma related to current event?		
Prothrombin time/INR*	History of myocardial infarction or angina?		
aPTT*	History of cardiac arrhythmias? Atrial fibrillation?		
Oxygen saturation	History of prior stroke or TIA?		
Selected patients	What medications is patient currently taking? Is patient receiving		
Hepatic function tests	anticoagulation therapy with warfarin?		
Toxicology screen	TIA indicates transient ischemic attack.		
Blood alcohol level			
Pregnancy test	present with unilateral weakness (Todd's paralysis), and		
Arterial blood gas tests (if hypoxia is suspected)	strokelike deterioration can occur in a patient with a brain		
Object we discuss the difference is successfully	neoplasm. Migrainous aura may be confused with a transient		

Table 8.

Chest radiography (if lung disease is suspected)

Lumbar puncture (if SAH is suspected and CT scan is negative for blood) EEG (if seizures are suspected)

ECG indicates electrocardiogram; INR, international normalized ratio; aPTT, activated partial thromboplastin time; and EEG, electroencephalogram.

*Although it is desirable to know the results of these test before rtPA is given, thrombolytic therapy should not be delayed while waiting for the results unless (1) there is clinical suspicion of a bleeding abnormality or thrombocy-topenia, (2) the patient has received heparin or warfarin, or (3) use of anticoagulants is not known.

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boluses may be administered, with concomitant close evaluation of the patient's cardiovascular response.

Patient Medical History

It can be challenging and time-consuming to obtain a stroke history in the emergency phase. As part of the stroke team, emergency nurses can play a key role in helping obtain a pertinent clinical history from the AIS patient and family members (Table 8).

Neurological Assessment

The NIHSS (Table 9) is a valid, efficient, and reliable measure of the patient's status after a stroke and in assessing outcome after treatment. Among stroke neurologists and neuroscience nurses, it is the most widely used stroke deficit scale. Developed by stroke neurologists from the University of Cincinnati, the University of Iowa, and the NIH-NINDS,²⁶ the NIHSS contains parameters for observing changes in the patient's neurological status and measuring stroke severity. To ensure reliability, formal training and certification in use of the scale are recommended (www.strokeassociation.org or www.ninds.nih.gov). The NIHSS is a valid functional outcomes measure and should become the standard to measure neurological functional improvement over time, both in the hospital and during the poststroke period of follow-up.

Ruling Out Stroke Mimics

Various conditions that mimic stroke should be ruled out quickly when stroke is suspected. Postseizure patients may

Blood Pressure Evaluation and Management

ischemic attack or stroke.44

Important Stroke/Medical History Questions

Blood pressure measurement is critical in the hyperacute phase of stroke. A history of hypertension is common in AIS patients. A transient rise in blood pressure can also be found in previously normotensive patients; these elevations are expected and may represent the body's compensatory response to the acute cerebrovascular occlusion, enhancing collateral flow.^{95–98} Unless blood pressure elevations are in the range to cause hypertensive encephalopathy, they are generally not treated, to facilitate adequate cerebral perfusion pressure. The AHA Stroke Council recommends that emergency administration of antihypertensive agents be withheld unless diastolic blood pressure is >120 mm Hg or systolic blood pressure is >220 mm Hg in AIS patients who are not treated with thrombolytics.¹³

For patients treated with intravenous thrombolysis, key data about treatment of hypertension in the emergent phase of stroke come from the 2 NIH-NINDS rtPA trials.⁶⁷ In these patients, systolic blood pressure was maintained at <185 mm Hg and diastolic blood pressure at <110 mm Hg. If 2 consecutive readings showed systolic blood pressure >185 mm Hg or diastolic blood pressure >110 mm Hg, fast-acting intravenous agents were used cautiously to lower blood pressure.¹³

Normally, an intrinsic process in cerebral blood vessels maintains constant brain blood flow regardless of systemic blood pressure, a concept known as autoregulation. In stroke, however, autoregulation may be impaired, particularly in the penumbra, where the cerebral blood flow is determined by systemic blood pressure. In such cases, it may be necessary to regulate systemic blood pressure. The attending physician will choose short-acting continuous infusion agents with a reliable dose-response relation and safety profile.⁹⁹ When reduced blood pressure is desired, labetalol is preferred if the patient has tachycardia at baseline, whereas nicardipine, a pure peripheral vasodilator, is preferable if the patient has bradycardia, congestive heart failure, a history of bronchos-

Table 9. National Institutes of Health Stroke Scale

Item Tested	Title/Domain	Response/Score
1A	LOC	0-Alert
		1–Drowsy
		2–Obtunded
		3-Coma/unresponsive
1B	Orientation	0-Answers both correctly
		1-Answers 1 correctly
		2-Answers none correctly
1C	Response/commands (2)	0–Performs both correctly
		1–Performs 1 correctly
		2–Performs none correctly
2	Gaze	0–Normal horizontal movements
		1–Partial palsy
		2-Complete gaze palsy
3	Visual fields	0-No visual field defect
		1–Partial hemianopia
		2-Complete hemianopia
		3–Bilateral hemianopia
4	Facial movement	0–Normal
		1–Minor facial weakness
		2-Partial facial weakness
		3-Complete unilateral palsy
5	Motor function (arm)	0–No drift
	a. Left	1–Drift before 5 seconds
	b. Right	2–Falls before 10 seconds
		3-No effort against gravity
		4–No movement
6	Motor function (leg)	0–No drift
-	a. Left	1–Drift before 5 seconds
	b. Right	2–Falls before 10 seconds
	5. rught	3-No effort against gravity
		4–No movement
7	Limb ataxia	0–No ataxia
,		1–Ataxia in 1 limb
		2-Ataxia in 2 limbs
8	Sensory	0-No sensory loss
0	ochoory	1-Mild sensory loss
		2-Severe sensory loss
9	Best language	0-Normal
5	Dest language	1–Mild aphasia
		2–Severe aphasia
		·
10	Articulation	3–Mute or global aphasia 0–Normal
10		
	Dysarthria	1–Mild dysarthria
11	Entirotion or instantion	2-Severe dysarthria
11	Extinction or inattention	0-Absent
		1-Mild (loss of 1 sensory modality)
	(0.40)	2–Severe (loss of 2 modalities)
Total NIHSS	score:(0-42)	

LOC indicates level of consciousness.

pasm, or chronic obstructive pulmonary disease. Neither of these agents appears to adversely affect ICP. Sodium nitroprusside has theoretical drawbacks related to cerebral venodilation, with exacerbation of any elevations of ICP and impairment of normal autoregulation.^{100,101}

The AHA guidelines recommend that blood pressure should not be treated in the hyperacute period unless 1 of the following exists: Systolic blood pressure >220 mm Hg or diastolic blood pressure >120 mm Hg after repeated measurements; cardiac ischemia, heart failure, or aortic dissection is present; thrombolytic therapy is planned; or ICH is identified.¹³ Table 10 lists approaches to treatment of elevated blood pressure in AIS.

If blood pressure does not decrease and cannot be maintained below the target levels of 185/110 mm Hg, rtPA should not be administered. Blood pressure management during and after treatment with rtPA or other acute recanalization therapy includes monitoring every 15 minutes during treatment and then for another 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours.

Administration of Thrombolytic Therapy

The nurse is responsible for administration of rtPA to eligible patients. The rt-PA dose for AIS is less than the recommended dose for myocardial infarction or pulmonary embolism treatment. rtPA is packaged as a crystalline powder and reconstituted with sterile water. After reconstitution, the preparation is 100 mg total. The total dose for an individual patient is calculated by multiplying the patient's weight per kilogram (up to 100 kg) by 0.9 mg. The total maximum dose for a patient with AIS is 90 mg. (The remaining portion of the preparation that will not be infused should be discarded to prevent accidental overdose.) To prevent accidental overdose, it is important to draw the waste dose from the bottle and verify the waste amount with another nurse before the rtPA bottle is connected to the intravenous pump tubing and administered to the patient. rtPA is administered in divided doses: 10% is given as a bolus over 1 minute, and the remaining 90% is administered as a continuous infusion over the next 60 minutes.

Before administering rtPA, the nurse should make sure that all intravenous lines are inserted. If needed, a Foley catheter and any other indwelling lines or tubes, including endotracheal tubes, should be inserted as well. There has been 1 report of fatal hemorrhage due to traumatic intubation.¹⁰² However, placement of lines and tubes should be rapid and should not delay administration of rtPA by more than a few minutes.

Intra-Arterial Thrombolysis

The use of intra-arterial thrombolysis as an alternative to intravenous rtPA is becoming more widespread. Intra-arterial therapy is based on the principle of delivering thrombolytic therapy at higher concentrations directly into the thrombus. The intra-arterial approach gives AIS patients more treatment options and should be initiated by specially trained interventional radiologists. In selected cases, intra-arterial thrombolysis extends the window of intervention to 6 hours after the onset of ischemic stroke symptoms.¹³ The intra-

Blood Pressure Level	Treatment
Not eligible for thrombolytic therapy	
Systolic <220 mm Hg <i>or</i> diastolic <120 mm Hg	Observe unless there is other end-organ involvement, eg, aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy. Treat other symptoms of stroke such as headache, pain, agitation, nausea, and vomiting. Treat other acute complications of stroke, including hypoxia, increased ICP, seizures, or hypoglycemia.
Systolic $>$ 220 mm Hg <i>or</i> diastolic $<$ 121–140 mm Hg	Labetalol 10–20 mg V over 1–2 min. May repeat or double every 10 min (maximum dose 300 mg) or
	Nicardipine 5 mg/h IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/h every 5 min to maximum of 15 mg/h. Aim for a 10% to 15% reduction of blood pressure
Diastolic >140 mm Hg	Nitroprusside 0.5 μ g/kg per min IV infusion as initial dose with continuous blood pressure monitoring. Aim for a 10% to 15% reduction of blood pressure.
Eligible for thrombolytic therapy	
Pretreatment	
Systolic $>$ 185 mm Hg <i>or</i> diastolic $>$ 110 mm Hg	Labetalol 10–20 mg IV over 1–2 min; may repeat $ imes$ 1 Or
	Nitropaste 1–2 in Or
	Nicardipine drip, 5 mg/h, titrate up by 0.25 mg/h at 5- to 15-min intervals (maximum dose 15 mg/h).
	If blood pressure is not reduced and maintained at desired levels (systolic \leq 185 mm Hg and diastolic \leq 110 mm Hg), do not administer rtPA.
During and after treatment	
1. Monitor blood pressure	Check blood pressure every 15 min for 2 h, then every 30 min for 6 h, and then every hour for 16 h.
2. Diastolic >140 mm Hg	Sodium nitroprusside 0.5 $\mu {\rm g/kg}$ per min IV infusion as initial dose and titrate to desired blood pressure level.
3. Systolic >230 mm Hg <i>or</i> diastolic 121–140 mm Hg	Labetalol 10 mg IV over 1–2 min, may repeat every 10–20 min (maximum dose 300 mg) <i>or</i> labetalol 10 mg IV followed by infusion at 2–8 mg/min. <i>Or</i>
	Nicardipine 5 mg/h IV drip as initial dose, titrate up to desired effect by increasing 2.5 mg/h every 5 min to maximum dose of 15 mg/h. If blood pressure is not controlled by labetalol, consider sodium nitroprusside but avoid if possible.
 Systolic 180–230 mm Hg or diastolic 105–120 mm Hg 	Labetalol 10 mg IV over 1–2 min. May repeat or double labetalol every 10–20 min to a maximum dose of 300 mg <i>or</i> give initial labetalol 10 mg IV followed by infusion at 2–8 mg/min.

Table 10. Recommendations for Treatment of Elevated Blood Pressure in Acute Ischemic Stroke

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arterial approach is commonly used for treating stroke due to a large thrombus in the middle cerebral artery, a lifethreatening vertebrobasilar stroke in the posterior circulation, and when intravenous rtPA is contraindicated.^{12,103} An emergent cerebral angiogram is required to place the delivery catheter at the site of the thrombus. The nurse should educate patients and their families about this treatment and should also watch for treatment-related complications.

In 1998, the PROlyse in Acute Cerebral Thromboembolism II (PROACT II) trial tested the effectiveness of the intra-arterial approach using prourokinase in patients with onset of stroke symptoms of <6 hours as a result of occlusion of the middle cerebral artery.¹⁰⁴ The primary analysis, intention to treat, was based on a modified Rankin Scale score of 0 to 2 at 3 months. The results were significant at P=0.04. Sixty-six percent of middle cerebral arteries were recanalized in patients treated with recombinant prourokinase compared with 18% in the control group (P<0.001). The ICH rate was 10% in the prourokinase group compared with 2% in the control group. There was no difference in death rate. On the basis of this single phase III trial, the Food and Drug Administration did not approve prourokinase for intra-arterial treatment of stroke, and it has not been marketed. Other thrombolytics, including rtPA and urokinase, have been extrapolated as intra-arterial therapy within 6 hours of symptom onset in selected cases of AIS.

The results of the cerebral angiogram and other radiological findings such as a hyperdense artery sign can help determine treatment options. Some authors have suggested that patients with a dense middle cerebral artery sign did better with intra-arterial thrombolysis than with intravenous thrombolysis, but there are no direct comparisons in clinical trials to prove this.^{103,105,106}

Combined therapy with intravenous thrombolysis and then intra-arterial thrombolysis is used occasionally. The Interventional Management of Stroke study is evaluating the combination of intravenous/intra-arterial administration of low-dose intravenous (0.6 mg/kg) rtPA followed by 22 mg of intra-arterial rtPA therapy. Currently, phase III of the study is comparing conventional intravenous rtPA alone with rtPA plus endovascular interventions.^{103,107} Further studies will provide more data on the efficacy of intra-arterial thrombolysis.

Mechanical Devices

The Merci Retriever (Concentric Medical, Mountain View, Calif) was the first retrieval device approved for clot retraction in AIS patients who were not candidates for rtPA or who had failed intravenous therapy.^{108–110} The Penumbra System (Penumbra Inc, Alameda, Calif) was the second retrieval device approved to remove blood clots in patients with AIS.¹¹¹ These devices have been used in combination with intravenous or intra-arterial therapy. Noser et al¹⁰⁹ have suggested that aggressive mechanical clot disruption may help increase recanalization rates compared with intra-arterial thrombolysis. This information is not yet supported by clinical trials.

Several other approaches to recanalization with device catheters are available. The EKOS catheter (EKOS Corp, Bothell, Wash) used in the Interventional Management of Stroke phase I and II trials delivered intra-arterial rtPA with concurrent intra-arterial low-energy ultrasound. Use of transcranial Doppler ultrasound to enhance the thrombolytic activity of intravenous rtPA is being evaluated in phase II clinical trials.¹¹² These device catheters have been evaluated in safety and technical efficacy trials. Randomized, controlled clinical trials of both the Merci and EKOS devices must be completed to evaluate their clinical efficacy.^{107,113}

Recommendations Class I

- 1. Emergency personnel should be highly trained in stroke care (*Class I, Level of Evidence B*).
- 2. Frequent neurological/stroke assessments should be done (*Class I, Level of Evidence C*); these should be done more frequently for patients receiving rtPA.
- 3. Supplemental oxygen should be given to patients with an oxygen saturation of <92% and a decreased level of consciousness (*Class I, Level of Evidence C*). There is little evidence that supplemental oxygen should be provided routinely.
- 4. The stroke patient's head should be positioned in neutral alignment with the body, and the head of the bed should be elevated 25° to 30° to help the patient handle oral secretions, especially if dysphagia is present (*Class I*, *Level of Evidence C*).
- 5. Stroke patients in the ED should be kept NPO (not given anything orally) until ability to swallow is assessed (*Class I, Level of Evidence B*).
- 6. Intravenous access should be obtained in at least 2 sites, with 1 site for administration of rtPA and 1 site for delivery of intravenous fluids or other medications if the patient is a candidate for rtPA (*Class I, Level of Evidence C*).
- 7. Only nondextrose, normotonic intravenous fluids such as normal saline should be used in the AIS patient (*Class I, Level of Evidence C*).
- 8. Intravenous rtPA should be administered without delay and should not be excluded in an eligible patient (*Class I, Level of Evidence A*).
- 9. See Table 11 for additional medical recommendations.

Table 11. Medical Recommendations

Recommendation	Class and Level of Evidence
Class I:	
CT or MRI of the head should be performed emergently in patients who present to the ED within the 3-h window.	Class I, Level of Evidence A
In the ED setting, laboratory tests should be obtained and processed rapidly to facilitate rapid assessment of the stroke patient, especially one who is a candidate for rtPA. At a minimum, the following tests should be performed: CBC, including platelets, blood chemistries, and coagulation studies (PT, aPTT, and INR).	Class I, Level of Evidence A
The use of intra-arterial thrombolysis is reasonable in select patients with a large MCA clot presenting within 6 h of stroke onset or who have contraindications to intravenous thrombolysis.	Class I, Level of Evidence B
Interventional treatment should be provided by a qualified interventionalist in centers that meet the guidelines for comprehensive stroke centers.	Class I, Level of Evidence C
When intra-arterial rtPA is considered, intravenous rtPA should always be given if the patient is eligible.	Class I, Level of Evidence C
Class IIa:	
The Merci Retriever and Penumbra System is a reasonable intervention for extraction of thrombi, but both require further evaluation of clinical efficacy.	Class IIa, Level of Evidence B
Intra-arterial thrombolysis is reasonable in patients with contraindications to use of intravenous thrombolysis, such as recent surgery.	Class IIa, Level of Evidence B

CBC indicates complete blood count; PT, prothrombin time; and MCA, middle cerebral artery.

Phase 2: Acute Care

During the acute care phase, nursing care should focus on continued stabilization of the stroke patient through frequent evaluation of neurological status, blood pressure management, and prevention of complications. Medical management focuses on establishing the cause or etiology of AIS, prevention of treatment-related complications, and evaluation of secondary prevention strategies. There is considerable evidence that dedicated stroke teams, units, and coordinated care improve clinical outcomes in the acute care phase.^{5,7,57,114–121}

Clinical Pathways and Stroke Order Sets

Specific order sets (standing orders) that address issues such as control of blood glucose, parameters to treat fever, and consultations with other multidisciplinary team members should be developed. The Brain Attack Coalition recommends succinct, organized stroke care in its recommendations for the development of primary stroke centers.¹⁰

Specific Care in the Acute Phase: Immediate Medical and Nursing Management

A key element of the management of patients with acute stroke is to prevent deterioration and medical complications,

such as respiratory problems associated with smoking or pneumonia, hypertension, hyperglycemia, dehydration, malnourishment, fever, coronary artery disease, cerebral edema, infection, and thromboembolism (deep vein thrombosis [DVT] or pulmonary embolism). These all worsen overall patient outcome.^{13,82,122–131}

To provide high-quality care, nurses must coordinate the activities of the multidisciplinary team. Clinical pathways or physician standing orders can guide the team in managing stroke patients and are useful for coordinating diagnostic tests and appropriate therapies and care issues. Clinical pathways improve coordination of acute stroke care and discharge planning, decrease hospital costs, decrease readmission rates, reduce length of hospital stay, and enhance usefulness of outcome measurement and quality improvement.^{114,132,133} Examples of professional resources including clinical pathways, guidelines, and standing orders can be found on the AHA Web site (http://www.americanheart.org/presenter.jhtml?identifier=3047992) and the Brain Attack Coalition Web site (http://www.stroke-site.org).

Intensive Management

Experts estimate up to 30% of all stroke patients will deteriorate in the first 24 hours.^{13,129} This statistic supports the need for intensive monitoring by nurses specifically trained in acute stroke care. Patients who receive thrombolytic therapy should also be monitored closely for at least 24 hours after treatment. Care may be provided in a designated intensive care unit or a stroke unit with continuous cardiac telemetry. In either area, nurses are trained in the care of patients after thrombolysis, are aware of bleeding complications, are trained in the use of appropriate neurological assessment tools, and are adept at recognizing the signs of increasing ICP often related to large stroke lesions.⁸⁶ The nurse-patient ratio is 1:2 for the first 24 hours; then, if the patient's condition is stable, the ratio is 1:4 as appropriate.^{53,134}

Bleeding assessment after administration of rtPA is the responsibility of the clinical nurse, who monitors the patient for major and minor bleeding complications in the first 24 to 36 hours after administration of rtPA.84,135 ICH is the major bleeding complication associated with thrombolytic therapy.83,135 In the NINDS trials, 6.4% of treated patients had symptomatic ICH, which is defined as "any CT-documented hemorrhage that was temporally related to deterioration in the patient's clinical condition in the judgment of the clinical investigator" within 36 hours of treatment.¹³⁵ Studies have shown that there is a natural rate of hemorrhagic transformation in ischemic stroke, and some studies suggest that petechial hemorrhages are frequently found in almost all cerebral infarcts.^{135–138} The use of thrombolytics increases the risk of serious hemorrhagic transformation. The nurse must identify which patients are at higher risk of ICH. One study has shown that only 3% of patients with an NIHSS score of <10 who were treated with rtPA had symptomatic ICH compared with 17% of those with an NIHSS score >20.135Other studies have shown that hemorrhagic transformations are more frequent when there has been a deviation from the national guidelines treatment protocol.^{139–142} Age >80 years

was determined to be an independent factor in development of hemorrhage after administration of rtPA.75,143 Hemorrhagic transformation should be suspected if there is a change in level of consciousness, elevation of blood pressure, deterioration in motor examination, onset of new headache, or nausea and vomiting. If hemorrhage is suspected, the rtPA infusion should be discontinued immediately.84 Management of ICH includes immediate physician notification and attainment of rapid brain imaging and laboratory work, including prothrombin time/international normalized ratio, activated partial thromboplastin time, fibrinogen level, complete blood count with platelets, and, if not already done, type and cross-match. The nursing staff must be prepared to administer 6 to 8 U of cryoprecipitate containing factor VIII and 6 to 8 U of platelets.83,84 The physician will decide on further action in collaboration with other team members, such as the consulting neurosurgeon. Facilities that treat patients with thrombolytics should have a hemorrhage algorithm (Table 12) and clinical guidelines to expedite assessment and management of a new ICH. Other major bleeding complications observed after thrombolytic therapy are retroperitoneal, genitourinary, and gastrointestinal hemorrhages. Minor bleeding complications are common, such as oozing from gums and venipuncture sites, as well as hematuria and hemoptysis.84 Assessment of the patient's skin may identify hematomas or areas of ecchymosis or purpura. If the patient has antecubital venous access, automatic blood pressure cuffs should be used with caution to prevent formation of a hematoma in the patient's arm. The cuff site should be checked frequently, rotated, and repositioned every 2 hours. If petechiae are noticed under the automatic blood pressure cuff, use of the cuff should be discontinued. To prevent trauma during oral care, soft sponges should be used instead of toothbrushes in the first 24 hours. Invasive procedures such as arterial punctures or insertion of catheters or nasogastric tubes should also be avoided in the first 24 hours after treatment.84

Neurological Stroke Assessment, Including the NIHSS

Intensive monitoring of stroke patients includes frequent monitoring of neurological assessment, including blood pressure, heart rate, and respirations. In patients treated with thrombolysis, blood pressure should be assessed at 15-minute intervals for 2 hours, every 30 minutes for the next 6 hours, and then once per hour until 24 hours after initiation of thrombolytic therapy. A complete bedside NIHSS assessment may be performed on admission to the intensive care unit, and an abbreviated version can be performed with more frequent assessments (Table 9).⁸⁶ The complete scale should be used if there is a decline in the abbreviated scale score.^{65,84}

The NIHSS provides valuable prognostic information and has been correlated with infarct volume.^{13,86,144} Patients with an NIHSS score of <10 have a much more favorable outcome at 1 year than patients with an NIHSS score of $>20.^{144}$ The nurse can use the NIHSS to identify patients who are at higher risk for ICH after thrombolytic treatment. In the NINDS rtPA trial, patients with an NIHSS score of >22 had a 17% risk of ICH, whereas those with an NIHSS score of <10 had only a

Care Element	Suspect ICH or Systemic Bleed	2–24 h After ICH	24–36 h After ICH
Consultations	Neurosurgery if ICH suspected Hematology if ICH suspected General surgery if systemic bleed suspected	Same	Same
Nursing assessments	Vital signs every 15 min Neurological examination, signs of ICP every 15 min Continuous ECG monitoring Look for other bleeding sites	Vital signs every 1 h and as necessary Signs of ICP, neurological examination GCS/pupil check every 1 h and as necessary Monitor ECG Monitor SV0 ₂ , ICP	Advance vital signs as necessary Advance neurological examination Consider discontinuing ECG
STAT diagnostics	CT head, noncontrast or MRI with GRE sequence Labs: PT/aPTT/INR, fibrinogen, CBC with platelets, type and cross-match Pulse oximetry, consider SVo ₂ , brain oximeter Consider ICP monitor Consider hemodynamic monitoring Check stool for occult blood	Labs: Na ²⁺ , osmolality (if on mannitol) Glucose every 6 h and as necessary (in patients with history of DM) ABGs Co ₂ 30–35 (hyperventilation if ordered) Consider ICP monitor	Consider discontinuing O ₂ monitoring
Treatments	If receiving thrombolytics, STOP INFUSION Consider hyperventilation Consider mannitol Consider blood products (cryoprecipitate, FFP, PLTs, PRBCs, other meds such as factor VIIa) Consider surgery Apply pressure to compressible sites for major or minor systemic bleeds	Keep Po ₂ >90 mm Hg Consider hyperventilation Consider mannitol 25 g every 4–6 h Consider surgery; treat DKA/HOC with insulin drip as necessary	Keep Po ₂ >90 mm Hg Wean hyperventilation Wean mannitol Wean blood pressure drips, add oral agent as tolerated
Activity	Bed rest Change position every 1–2 h as tolerated	Same	Advance as tolerated
Nutrition	Feed as soon as possible NPO Consider enteral feedings with NGT or DHT	Same	Consider feeding as swallowing screen defines, consider TPN or other enteral feeding

Table 12.	Nursing Alert! Critical Actions for Suspected ICH or Systemic Bleeding After rtPA Administration ⁸⁴
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NINDS rtPA Stroke Study Group hemorrhage algorithm.^{83,84}

GCS indicates Glasgow Coma Scale; GRE, gradient refocused echo; DM, diabetes mellitus; ABGs, arterial blood gases; DHT, Dobhoff tube; FFP, fresh frozen plasma; PLTs, platelets; PRBCs, packed red blood cells; DKA, diabetic ketoacidosis; HOC, hyperosmolar coma; NGT, nasogastric tube; and TPN, total parenteral nutrition.

3% risk of ICH.¹³ The NIHSS may be useful for working with families on discharge planning needs.^{145,146} In 1 study of ischemic and hemorrhagic stroke patients, a 24-hour NIHSS score of <5 increased by nearly 5-fold the likelihood of discharge to home rather than inpatient rehabilitation or a skilled nursing facility.^{145,146}

Ongoing Blood Pressure Management

Blood pressure is a critical vital sign in the AIS patient. It is not uncommon to see variations in blood pressure after AIS.⁹⁶ Blood pressure is elevated in $\approx 40\%$ to 80% of all AIS patients,⁹⁶ especially in the first 24 to 48 hours after stroke, and will fall 10 to 14 days after the acute phase.¹⁴⁷ Elevated blood pressure may increase cerebral perfusion in the ischemic zone, where autoregulation is lost and perfusion is pressure dependent.¹⁴⁸ The current guidelines recommend maintaining blood pressure at <180/105 mm Hg for 24 hours in patients who have received thrombolytic therapy.¹³ It is recommended that antihypertensive treatment be initiated for nonthrombolytic candidates only if systolic blood pressure is >220 mm Hg or diastolic pressure is >110 mm Hg.¹³

Blood pressure should be monitored and assessed continually for causative factors of rises. Elevated blood pressure may be due to a physiological response to hypoxia, increasing ICP, hemorrhagic transformation, full bladder, pain, nausea, a loud environment, or preexisting hypertension.^{13,149,150} The ASA blood pressure guidelines may be included in standing physician orders to expedite treatment of elevated blood pressure in the phase that immediately follows thrombolytic therapy.¹³ At present, the optimum blood pressure in the immediate poststroke period is unclear and controversial, and further scientific evidence is needed.^{151,152} There is evidence, however, that rapid lowering of blood pressure may induce worsening of neurological symptoms by inducing lowered perfusion pressures to the area of ischemia.^{149,153,154}

Arterial hypotension is rare in the AIS patient but may be associated with volume depletion or decreased cardiac output related to arrhythmias or myocardial ischemia. Patients with hypotension require evaluation with advanced neurological nursing assessment and telemetry monitoring. Treatment consists of volume replacement with normal saline and correction of arrhythmias.

Temperature Management

Fever appears to exacerbate the ischemic injury to neurons and is associated with increased morbidity and mortality, particularly in acute stroke.¹⁵⁵ Data from additional metaanalyses found a correlation between temperature elevation and cerebral infarct volume.⁸⁰ Even an increase of 1°F is a predictor of poorer patient outcome and is an independent factor in short- and long-term mortality rates.^{82,129,156,157} The rationale for this additional injury may be related to increased metabolic demands and free radical production. Immediate treatment of the source of the fever will reduce its duration.^{82,157} One approach to maintaining normothermia is to immediately begin acetaminophen at 99.6°F.^{158,159} Even more rapid induction of cooling can be achieved by additionally treating temperature elevation with indwelling catheter temperature control systems or surface cooling systems.^{158,159}

Continuous Cardiac Monitoring

Cardiac monitoring is recommended for all ischemic stroke patients.^{13,160,161} Studies suggest that insular lesions can lead to cardiac arrhythmias and sudden cardiac death.13 Arrhythmias such as ventricular ectopy, tachycardia, and heart blocks have been associated with AIS.162 Right hemispheric infarcts have been associated with a higher incidence of arrhythmias, possibly due to sympathetic and parasympathetic nervous system dysfunction.¹⁶³ Atrial fibrillation, often paroxysmal, is commonly first detected only after it has caused cardioembolic stroke. If cardiac output is compromised, arrhythmias may further aggravate an already compromised cerebral blood flow. If not completed as part of the initial ED workup, a 12-lead ECG can be completed on admission. Many stroke patients have underlying cardiac problems and are at risk for an acute myocardial infarction during the acute stages of stroke. Patients may also need a cardiac evaluation by a cardiologist during the acute stages of stroke. If telemetry is unavailable, a Holter monitor can be used to check for arrhythmias.13

Assessment of Oxygenation

Monitoring of oxygen saturation will reduce the risk of neurological deterioration related to hypoxemia. Supplemental oxygen at 2 to 4 L/min is recommended for an oxygen saturation of <92%.^{13,164} Many factors compromise adequate oxygenation, for example, decreased level of consciousness, aspiration, and atelectasis. Vigilant assessment of the patient's lung sounds and ability to swallow will keep the nurse aware of pending threats to adequate oxygenation.^{13,135,165} If an oxygen saturation of 92% cannot be maintained, arterial blood gases and a chest radiograph are recommended. In the absence of hypoxemia, supplemental oxygen is not recommended.⁸⁷

Angioedema

Large prospective cohort studies have shown that orolingual angioedema occurs in a small proportion (1% to 2%) of patients with AIS or acute myocardial ischemia treated with rtPA. This was more commonly seen in patients with frontal cortex and insular ischemia, in patients who had received alteplase and were concurrently taking angiotensin-converting enzyme (ACE) inhibitors.¹⁰² In most cases, symptoms were mild and transient. The pharmacological insert that

accompanies rtPA addresses this and recommends that patients be monitored during infusion and for several hours afterward for signs of allergic reaction that exhibits as orolingual angioedema (http://www.gene.com/gene/products/ information/cardiovascular/activase/insert.jsp). Treatment includes immediate discontinuation of rtPA and administration of antihistamines, intravenous corticosteroids, or epinephrine. It is important for emergency and intensive care unit nurses to evaluate patients closely for throat or mouth edema and look for any difficulty in breathing due to angioedema.

Blood Glucose Monitoring: Hyperglycemia

Hyperglycemia in critically ill patients has long been associated with complications. Infarct expansion, hemorrhagic conversion, and poor clinical outcomes have been reported in the AIS population.¹²⁴ Even the benefit of recanalization after use of thrombolytics may be reduced.166-168 Increased blood glucose provides additional substrate for anaerobic metabolism, which promotes lactic acidosis and free radical production. Elevated serum glucose is common in the acute phase of stroke and may be related to uncontrolled or undetected diabetes mellitus or stress-induced hyperglycemia associated with cortisol and norepinephrine release at the time of insult.^{167,169–174} In 1 study, elevated glucose was present in two thirds of AIS patients.¹⁷⁵ Treatment with insulin confers a protective effect in critically ill patients.^{164,165} The ASA 2007 "Guidelines for the Early Management of Adults With Ischemic Stroke" recommend the use of rapid-acting insulin for a blood glucose level >140 mg/dL.13

Several studies have shown that elevated blood glucose is an independent factor in poor functional outcomes, increased infarct size, increased length of stay (7 versus 6 days), increased mortality at 30 days, and increased cost (\$6611 versus \$5262).^{176–178} Not only did patients have poorer outcomes associated with hyperglycemia, but in an analysis of the NINDS rtPA trial, it was found that the risk of hemorrhagic transformation increased by 75% per 100 mg/dL of blood glucose.^{167,177–180} Poorer outcomes were also seen in patients with a glucose level >140 mg/dL after administration of rtPA. There is speculation that elevated glucose levels may prevent early reperfusion and eliminate the benefit of rtPA.^{166–168}

Several nonrandomized studies have suggested improved outcome if there is an acute reversal of hyperglycemia.167,181-184 The Glucose Insulin in Stroke Trial (GIST), a multicenter, randomized trial, recruited 933 patients and evaluated the use of variable-dose glucose and potassium insulin (GKI) versus saline infusions in stroke patients with glucose levels of 6.0 to 17.0 mmol/L (\approx 300 mg/dL and defined as mild to moderate hyperglycemia).¹⁸⁵ The purpose of the GKI infusion was to maintain glucose at 4 to 7 mmol/L (euglycemic); there was no glucose intervention in the control group after stroke. Most patients were entered in the study within 24 hours after onset of stroke symptoms. The primary outcome was death at 90 days. The study was stopped early owing to low recruitment. Based on the intention-to-treat data, there was no significant reduction in morbidity or mortality at 90 days in the intervention group (GKI versus control: odds ratio 1.14, 95% confidence interval 0.86 to 1.52, P=0.37). Plasma glucose concentrations and blood pressure were significantly reduced in the GKI group. The investigators' interpretation of this study was that although the intervention group had lower glucose and blood pressure levels, there was not a significant clinical benefit. The study was underpowered, and an alternative result could not be excluded.¹⁸⁶ More randomized clinical trials are needed to understand the optimal level of blood glucose and best treatment modalities to produce the best clinical outcomes in stroke patients.

The nurse should monitor the blood glucose level based on the patient's glucose level at admission.¹⁷⁶ If blood glucose is >140 mg/dL and the patient has received thrombolytic therapy, it may be prudent to monitor glucose every 1 to 2 hours, because there is evidence that these patients are more prone to ICH. Treatment for hyperglycemia may be instituted, depending on individual hospital insulin or oral hypoglycemic treatment protocols. In patients who have not received thrombolysis, glucose may be monitored every 6 hours in the first 24 to 48 hours and continued if the patient is known to have diabetes. It is also important to evaluate the need for diabetic education whether or not the patient is a known or newly diagnosed diabetic.

Hypoglycemia

Hypoglycemia can uncommonly cause focal neurological deficit and mimic stroke. If present, it should be corrected promptly with administration of 1 ampule of 50% dextrose.

Cerebral Edema After Stroke

Cerebral edema is a common complication of large multilobar infarctions. It usually peaks 3 to 5 days after AIS and is not a significant problem in the first 24 hours except in patients with large cerebellar infarcts or in younger stroke patients. Young people usually do not have significant cerebral atrophy, thereby allowing no room for swelling. ICP increases as a result of cerebral edema, and monitoring for increased ICP should be part of the ongoing assessment of AIS patients. When invasive ICP monitoring is not available, the nurse must rely on the less accurate clinical signs of increasing pressure, which can include change in level of consciousness, worsening neurological deficits, new pupillary changes, or changes in respiratory patterns.¹³ Changes in level of consciousness are an early sign of increasing ICP, whereas pupillary changes are a late sign. Hydrocephalus may also develop as a result of obstruction of the cerebral spinal fluid pathways. Imaging studies will identify mass effect, which includes frontal horn compression, or shift of the septum pellucidum or the pineal gland. These signs demonstrate that the patient is at risk of clinically worsening and increasing pressure that can lead to brain herniation. The treatment plan goals should be to (1) reduce ICP, (2) maintain cerebral perfusion pressure to prevent worsening cerebral ischemia, and (3) prevent secondary brain injury¹³ (Table 13).

Hypotonic fluids containing excess free water should be avoided in patients who have or are at risk for cerebral edema.¹³ The nurse must evaluate the patient for hypoxia,

Table 13. Nursing Alert: Recognizing Increased ICP

igns and symptoms of increasing ICP-a medical emergency	
Early signs: decreased level of consciousness, deterioration in motor function, headache, visual disturbances, changes in blood pressure or heart rate, changes in respiratory pattern	
Late signs: pupillary abnormalities, more persistent changes in vital sig changes in respiratory pattern with changes in arterial blood gases	ns,
Intervention: thorough neurological assessment, notify physician immediately, emergency brain imaging, maintain ABCs	
eneral measures to prevent elevation of ICP	
HOB up 30° or as physician specifies; reverse Trendelenburg position may be used if blood pressure is stable. Head position may be one of single most important nursing modalities for controlling increased ICP	the
Good head and body alignment: prevents increased intrathoracic press and allows venous drainage.	ıre
Pain management: provide good pain control on a consistent basis	
Keep patient normothermic	

HOB indicates head of bed.

hypercarbia, or hypothermia that may lead to elevated ICP. The head of the bed should be elevated 20° to 30° , the neck should be in a neutral position to facilitate venous drainage, and the airway should be assessed for patency. As ICP increases, the patient's blood pressure may rise to maintain adequate cerebral perfusion pressure. The use of an aggressive antihypertensive agent with venodilating effects, such as nitroprusside, should be avoided because it can cause cerebral venodilation and can lead to a more elevated ICP. For cerebellar infarcts and hemorrhages in which hydrocephalus and a generalized increase in ICP are an issue, an ICP catheter is usually not inserted. For large hemispheric infarcts and hemorrhages, herniation rather than generalized increased ICP is the main concern, and ICP monitoring is generally not helpful. ICP treatment may include modest hyperventilation to decrease Pco2 by 5 to 10 mm Hg to produce enough vasoconstriction to temporarily lower ICP.13 However, hyperventilation is only a temporary measure, and brain perfusion may be compromised as vasoconstriction occurs. Frequent neurological assessments must be done to look for potential changes in brain perfusion.13 On the basis of studies conducted primarily among the head injury patient population, it has become clear that some nursing care activities increase ICP transiently in some patients; however, it is not possible to identify any given activity that is uniformly detrimental to patients with increased ICP. Therefore, nurses need to evaluate each patient's physiological response to routine care.187,188

Osmotic diuretics such as furosemide or mannitol can be used to treat cerebral edema. Intravenous mannitol (0.25 to 0.50 g/kg) administered over 20 minutes can be given every 6 hours. Serum and urine osmolality should be monitored if mannitol is used; however, a Cochrane systematic review found no evidence that routine use of mannitol reduced cerebral edema or improved stroke outcome in AIS.¹⁸⁹ Lasix 40 mg can be used as adjunctive therapy but should not be used long-term. Barbiturates can be used for severe cerebral edema. Continuous electroencephalographic monitoring

should be performed if barbiturates are administered. Hypothermia can also be used to treat elevated ICP.¹³ Unfortunately, all of these modalities are short-lived and palliative at best. The recommendations for reducing cerebral edema need further study with regard to AIS. Currently, there is no clinical evidence that the measures discussed reduce cerebral edema or improve outcome in patients with ischemic brain swelling.^{13,189}

If hydrocephalus is present, fluid drainage through an intraventricular catheter can rapidly reduce ICP. Surgical decompression (hemicraniectomy) is the most definitive and invasive treatment of massive cerebral edema. Large cerebellar infarctions and hemorrhages that cause direct cerebellar compression of the brain stem are best treated with surgical decompression.¹⁹⁰⁻¹⁹² Surgical evacuation may be done in patients with large hemispheric infarcts, but survivors have severe residual neurological deficits.¹⁹³⁻²⁰⁰ Subsequent to a 2002 Cochrane review that found some evidence for benefit from surgical decompression,201 larger nonrandomized prospective trials have shown a reduction in the mortality rate of patients who have hemicraniectomy of large lesions involving the middle cerebral artery territory.193-200,202 A recent pooled analysis of 3 randomized, controlled clinical trials (DEcompressive Craniectomy In MALignant middle cerebral artery infarcts [DECIMAL], DEcompressive Surgery for the Treatment of malignant INfarction of the middle cerebral arterY [DESTINY], and Hemicraniectomy After Middle cerebral artery infarction with Life-threatening Edema Trial [HAMLET]) in patients with malignant middle cerebral infarct showed that decompressive surgery performed within 48 hours of stroke onset reduced mortality and yielded more favorable outcomes.123,203

Seizures

Seizures are a possible complication of large cortical strokes and can be potentially life-threatening if not controlled. They can occur at the time of AIS, during the first few days after the event, or several months later. No study has specifically tested the usefulness of anticonvulsant medications in preventing or controlling seizures after stroke. Drugs proven to be of value in preventing seizures from other causes, however, are recommended for patients who have had more than 1 seizure after stroke.²⁰⁴ Routine prophylactic administration of anticonvulsant drugs to stroke survivors who have not had seizures should be avoided.13 A study of 3552 SAH patients in 21 countries found that anticonvulsant drugs were commonly used but were associated with more in-hospital complications and poorer outcomes.205 No similar studies were found regarding AIS.

If an anticonvulsant drug is required, the nurse must educate the patient and family about the seizure condition, pharmacological management, the medication regimen for treatment of seizures, and the side effects and precautions. The patient must be told to never adjust or take additional medications without consulting the physician. Patients may not have another seizure, but they and their families should be educated that the risk is ongoing.

Recommendations Class I

- 1. Stroke neurological assessments should be performed every 4 hours after the hyperacute phase of stroke, and then frequency should be based on the patient's stability and other comorbid conditions (*Class I*, *Level of Evidence B*).
- 2. Temperatures >99.6°F should be managed aggressively (*Class I, Level of Evidence C*).
- 3. Continuous cardiac monitoring of the stroke patient should be provided for at least 24 to 48 hours after stroke to detect potential cardiac problems (*Class I, Level of Evidence B*).
- 4. Careful, frequent monitoring and assessment for worsening of neurological deficits or bleeding should be performed for up to 24 hours after thrombolytic therapy (*Class I, Level of Evidence B*).
- 5. Hyperglycemia should be treated in patients with a serum glucose concentration >140 mg/dL (*Class I, Level of Evidence C*).
- 6. Management of arterial hypertension in the acute phase should be approached with caution because of the lack of data available to guide management (*Class I, Level of Evidence C*).
- 7. Oxygenation should be evaluated with an oxygen saturation monitor (*Class I, Level of Evidence C*).
- 8. To prevent aspiration pneumonia, the patient's lungs should be auscultated, and the patient should be evaluated for signs of respiratory compromise and dysphagia (*Class I, Level of Evidence C*). Nurses should report seizure activity, and treatment should begin immediately (*Class I, Level of Evidence B*). Prophylactic treatment of seizures should not be given.

Class IIa

1. It is reasonable to use clinical pathways, protocols, or preprinted stroke order sets to organize care of the stroke patient (*Class IIa, Level of Evidence B*).

Diagnostic Testing During the Acute Phase

Overview of Neuroimaging in Stroke

Diagnostic imaging techniques include CT, MRI, magnetic resonance angiography, CT angiography, conventional angiography, and carotid and transcranial ultrasound of the cervical and cephalic arteries.¹³ Imaging of extracranial and intracranial cerebral blood vessels may be essential for understanding the mechanism of AIS. The presence of a high-grade stenosis, occlusion, dissection, or vascular anomaly indicates the events that led to the stroke. The nurse will need to educate the patient and family regarding each test and what to expect.

Computed Tomography

Imaging in the hyperacute phase was discussed above. A repeat CT or MRI brain scan should be done 24 hours after the initial stroke event in patients treated with thrombolysis or in any situation in which the patient is clinically deteriorating to assess hemorrhagic transformation and infarct progression. The CT scan is still considered the gold standard in AIS

treatment, although recent technology has led to more sophisticated multimodal approaches in stroke imaging with CT evaluation. CT perfusion and CT angiography provide a map of cerebral blood volume, cerebral blood flow, and mean transit time. These studies identify the ischemic core and pneumbra regions that can guide the decision for further interventional treatment.

Magnetic Resonance Imaging

MRI demonstrates evidence of ischemic injury to the brain earlier than CT for all ischemic stroke subtypes. Magnetic resonance angiography is a useful noninvasive procedure for evaluating extracranial and intracranial vessels. Newer multimodal techniques (eg, diffusion-weighted imaging and perfusion-weighted imaging) have further increased the sensitivity of MRI. The advantage of diffusion-weighted imaging is early identification of the ischemic area within minutes of onset of acute stroke. Perfusion-weighted imaging indicates the hemodynamic status of cerebral blood flow. A diffusion-perfusion mismatch shows an ischemic penumbra change in perfusion without a diffusion abnormality. This is an opportunity to identify irreversible versus reversible cerebral tissue. If there is no mismatch, the hope of salvageable tissue is less. Treatment modalities and time to treatment may change as more is learned about the ischemic penumbra.13,206-208

Ultrasonography

Carotid duplex scanning is the standard ultrasound test initially used to screen for cervical internal carotid stenosis. Demonstration of stenosis >60% is highly accurate; however, differentiation between severe (95% to 99%) stenosis and 100% occlusion is not completely reliable.

Cerebral Angiography

Cerebral angiography is the best tool to accurately evaluate the surface characteristics of a stenosed artery and is considered the "gold standard" for measuring the degree of stenosis of a cervical or cephalic artery. After the procedure, the nurse will perform frequent measurement of vital signs, neurological assessment, femoral and pedal pulse checks, and groin checks. The radiologist should be notified if a groin hematoma or change in velocity of the pedal pulse develops. Postprocedure activity is restricted according to the closure device used and the patient's status.

Transesophageal and Transthoracic Echocardiography

All patients with ischemic stroke or transient ischemic attack should undergo a comprehensive assessment of cardiovascular risk to identify those with the highest likelihood of morbidity and mortality due to unrecognized coronary heart disease or the presence of a cardioembolic source of stroke.^{13,209,210} Transthoracic echocardiography is excellent for identifying ventricular sources, such as a dyskinetic ventricular wall segment, whereas transesophageal echocardiography excels at identifying atrial and aortic sources, such as patent foramen ovale or aortic arch atherosclerosis. A transesophageal echocardiogram is sensitive for detecting apical thrombi and atrial septal defects or patent foramen ovale. Transthoracic echocardiography is the less invasive of the 2 procedures and is the most commonly ordered initial test to evaluate for a cardioembolic source of stroke. During a transesophageal echocardiogram, the nurse must monitor the patient closely and position the patient to decrease risk of aspiration if nausea or vomiting occurs.^{210,211}

Chest Radiograph

A chest radiograph is no longer routinely recommended during the ED evaluation of stroke unless an underlying pulmonary or cardiac problem is suspected. Obtaining a chest radiograph may take valuable time that is needed to administer rtPA within the 3-hour window. Therefore, a chest radiograph is not a high priority in the ED but is done during the acute phase of hospitalization.

Recommendation Class I

1. All nurses should be familiar with the basic neuroimaging testing for stroke patients so that they can educate and prepare patients and families (*Class I, Level of Evidence C*).

General Supportive Care of Stroke: Remembering the Basics

Medical and nursing management both focus on the prevention of subacute complications of stroke, including malnutrition, aspiration, pneumonia, UTI, bowel or bladder dysfunction, DVT, pulmonary embolism, contractures, joint abnormalities, and skin breakdown.130 Depression is also common after stroke.²¹² Even in specialized stroke units, up to 63% of patients experience 1 or more complications after acute stroke. The most common complications during the first week in a Norwegian stroke unit were pain, fever, progressing stroke, and UTI; immobilization-related complications, such as pressure ulcers and clinical signs of DVT and pulmonary embolism, had a very low incidence in this modern stroke unit.213 However, many stroke patients are not cared for in specialized units, so it behooves nurses to continue to be vigilant for the following areas of care related to prevention or rapid detection of complications. 5,7,57,114-121,132,214-223

Infection

Pneumonia and UTI are frequently seen in the acute phase after stroke. Fever or a change in level of consciousness should give the nurse a high index of suspicion for infection. Stroke patients frequently present to the hospital with a compromised chest radiograph or a UTI.

Pneumonia is a serious complication occurring in the first 48 to 72 hours after AIS and accounts for approximately 15% to 25% of deaths associated with stroke.^{13,224} Stroke-associated pneumonia increases length of stay, mortality, and hospital costs.²²⁴ The most common cause of pneumonia is aspiration due to dysphagia.²²⁵ Immobility and atelectasis can also lead to development of pneumonia. The patient's airway and oxygenation must be monitored closely; some patients may require endotracheal intubation and mechanical ventilation. Early mobility and good pulmonary care can help prevent pneumonia.²²⁴ Preventive measures in intubated pa-

tients include ventilation in a semirecumbent position, positioning of the airway, suctioning, early mobility, and shortened use of intubation, if feasible.²²⁶ In a randomized trial, early prophylaxis with levofloxacin was beneficial in preventing systemic infections.²²⁷ Nursing management includes prompt recognition and reporting of fever; the source of the fever should be sought and treatment begun immediately. Early management of nausea and vomiting can help prevent aspiration pneumonia; use of antiemetic medications is warranted in this situation. Suctioning of the airway should be done carefully if increased ICP is present.²²⁸

UTIs are common, occurring in approximately 15% to 60% of stroke patients, and independently predict poor outcome.^{122,130,229–231} The use of an indwelling catheter and changes in sphincter control increase the risk of UTI. Indwelling catheters should be avoided if possible but are often required in the acute phase of stroke. The catheter should be removed as soon as the patient is medically and neurologically stable. Intermittent catheterization may lessen the risk of infection. External catheters, incontinence pants, and intermittent catheterization are alternatives to an indwelling catheter. The patient should be assessed for UTI if there is a change in level of consciousness and no known reason for neurological deterioration. A urinalysis and urine culture should be obtained if UTI is suspected.^{13,229,232,233}

Bowel and Bladder Care

Constipation is the most common bowel problem. The nurse should assess the patient's premorbid bowel elimination pattern, bowel sounds, and abdominal distention, if present. The patient should also be evaluated for hydration and impaction. The nurse must request medications if needed or develop a bowel program, which can integrate the use of stool softeners, laxatives, and enemas to prevent constipation early after stroke.

The most common urinary complication is incontinence, which occurs 30% to 60% of the time in the early recovery period.²³⁴ An infarct in the frontal lobe or the pons can lead to incontinence. Voiding problems include neurogenic bladder; hyperreflexia with urge incontinence, urgency, and frequency; and urinary retention with or without overflow incontinence. After the indwelling catheter is removed, intermittent catheterization may be necessary to retrain the bladder. Intermittent catheterization should occur every 4 to 6 hours to prevent filling of the bladder beyond 500 mL and to stimulate normal physiological filling and emptying. Intermittent catheterization is recommended if postvoid residual urine volume is >100 mL.²³⁵

Urinary incontinence can increase the incidence of dermatitis, skin breakdown, UTIs, and perineal thrush. Urinary incontinence also interferes with rehabilitation and is the major factor in patients being discharged to nursing homes.²³⁶ Voiding strategies should be incorporated into the daily plan of care. The nurse must initiate a bladder-training program to decrease the number of incontinent episodes. The patient should be offered a commode, bedpan, or urinal every 2 hours during waking hours and every 4 hours at night. Neurological deficits may complicate the task of going to the bathroom. High fluid intake during the day and decreased fluid intake in the evening should be encouraged.

Mobility and the Musculoskeletal System

Stroke patients may be initially kept on bed rest but should be mobilized when they are hemodynamically stable. Early mobilization reduces risk of atelectasis, pneumonia, DVT, and pulmonary embolism. Complications from immobility account for up to 51% of deaths in the first 30 days after ischemic stroke.237,238 Immobility can also lead to contractures, orthopedic complications, atrophy, and nerve pressure palsies. The nurse should monitor the first transfer from bed to an upright position, because some patients may have neurological worsening during movement. Joints on the paralyzed side must be positioned higher than joints proximal to it. The nurse must assess for deformities that may be found on the affected side (eg, shoulder adduction). Subluxation of the affected shoulder is common, and special care should be taken to avoid pulling on the affected arm and shoulder when repositioning or moving the patient. Nursing interventions, including range-of-motion and positioning techniques, can prevent joint contractures and atrophy.237,239

Pulmonary Embolism and DVT

Pulmonary embolism occurs more commonly than is suspected clinically and accounts for a substantial number of deaths after AIS. Wijdicks²⁴⁰ found a sudden death rate of 50% in patients with pulmonary embolism that occurred between 3 and 120 days after the initial stroke event. In that study, prophylaxis for DVT was noted in only 4 of 30 patients. Pulmonary embolism was associated with DVT diagnosed clinically or at autopsy in 11 patients and in the paralyzed leg. More recently, Indredavik and colleagues²¹³ found pulmonary embolism in fewer than 2.5% of patients during the first week in a specialized stroke unit. DVT and pulmonary emboli were more likely to occur in the first 3 months after stroke, with an incidence of 2.5% and 1.2%, respectively.

Both ischemic and hemorrhagic stroke patients are at risk for developing DVT, especially as a result of paralysis or impaired mobility. Prevention of DVT is one of the core performance measures for primary stroke center certification, and initiation of prevention has become a quality indicator in several populations.^{241,242} Safe ambulation should be started as soon after stroke as possible. Pneumatic compression devices and compression stockings can be used to prevent pulmonary embolism.^{243,244}

Until recently, anticoagulants such as low-molecularweight heparinoids and unfractionated heparin were also used.^{245,246} The results of the Prevention of VTE after Acute Ischemic stroke with LMWH enoxaparin (PREVAIL) Trial showed that a 40-mg injection of enoxaparin once daily was more effective than 5000 IU of unfractionated heparin twice a day for prevention of DVT in AIS patients.²⁴⁷ Stroke patients taking anticoagulants should be assessed for bleeding daily.

Falls

Falls are a common cause of injury in stroke patients, with hip fractures the most prevalent injury.²⁴⁸ Hip fractures in the first

7 days after stroke are associated with a poor prognosis and have been recognized as a consequence of hemiplegia since the 1950s.²⁴⁹ Most fractures occur on the paretic side.²⁵⁰ Patients with right hemispheric infarcts that cause neglect or inattention have the highest fall risk.

Minimization of fall risk is a global responsibility. Nurses must implement fall-prevention programs and educate other staff and family members about risks and fall precautions. These may include identifying patients at risk, use of alarm systems, use of special equipment (eg, enclosure beds), and placing call buttons and the patient's belongings near the patient to prevent the patient from reaching for something and risking a fall. Voiding times should be scheduled to prevent falls that occur when a patient tries to go to the bathroom. In some cases, it may be necessary to have a sitter stay with the patient to ensure the patient's safety.²⁴⁹

Skin Care

Stroke patients are at risk for skin breakdown because of loss of sensation and impaired circulation, older age, decreased level of consciousness, and inability to move themselves because of paralysis.239 Related complications such as incontinence can accelerate skin breakdown. Major pressure areas are the heels, sacrum, and lateral malleoli. Patients should be examined for skin breakdown when repositioned and after sitting. Special care should be taken when moving patients to avoid excessive friction or pressure.239 Patients should not be left in 1 position for >2 hours. The skin must be kept clean and dry, and special mattresses should be used where indicated. The Braden Scale is commonly used to predict the risk of the development of pressure ulcers (decubitus). Nursing personnel can use this predictive model to design the care of stroke patients with immobility and those who are at risk for the development of skin breakdown.²⁵¹⁻²⁵³

Detection of Dysphagia and Prevention of Aspiration

Aspiration is frequently a result of dysphagia. In 2001, Galvan²⁵⁴ found that approximately half of all aspirations due to dysphagia are "silent" and go unrecognized until there is a pulmonary manifestation or complication. Order sets should include a swallow assessment before oral intake, performed by a nurse; evaluation by a speech language pathologist; and institution of NPO status with intravenous normal saline at 75 to 100 mL/h until evaluation by the speech language pathologist. Swallow assessment requires an evidence-based tool.²²⁵ The Massey Bedside Swallowing Screen has been successfully evaluated for interrater reliability and predictive validity.²⁵⁵

Optimally, swallow assessment is performed soon after the patient's arrival in the ED. Until then, the patient should remain on NPO status, which means no ice chips, no oral medications, no water, and no exceptions.²⁵⁶ The nurse should assess swallowing by direct observation, looking for the presence of choking, coughing, a wet voice, a delay in initiating swallow, uncoordinated chewing or swallowing, extended time eating or drinking, pocketing of food, and loss of food from the mouth. When oral intake is authorized, the nurse should follow the speech language pathologist's recommendations,²⁵⁷ which include improving the patient's ability to concentrate while eating with minimal distractions. These assessments and interventions may make the difference

between improved recovery and increased morbidity or even mortality. $^{\rm 256}$

Nutritional Compromise

Fifty percent of patients with severe strokes were reported to be malnourished at 2 to 3 weeks after the stroke.²⁵⁸ Malnutrition was associated with higher complications and poorer functional outcomes.^{259,260} To avoid nutritional compromise, nutritional intervention should occur no later than 3 to 4 days after diagnosis of dysphagia.⁵ The Ontario Heart and Stroke Association suggests that early gastrostomy should be considered⁵ if it is anticipated that dysphagia will continue beyond 6 weeks; however, dysphagia resolves in at least 87% of stroke patients.²⁵⁷

A nutritional assessment of a patient can be performed by taking a diet history, with simple assessments such as body mass index or more complex anthropometric measures such as midarm circumference or triceps skin-fold thickness. In the stroke patient, it can be difficult to obtain a diet history because of communication problems, and anthropometric measures may not be accurate owing to paralysis of the arm.

When compromise of the patient's nutritional health is suspected, serum albumin levels may be checked; this is often cited as an index for long-term maintenance but may not be reflective of nutritional status.^{261,262} Serum albumin falls in acute illness owing to increased catabolism. No specific tool has been developed or tested in acute stroke patients to evaluate nutritional assessments.²⁶³

A dietitian can provide a more accurate assessment of nutritional health and nutrient needs. Without adequate nutrition, there is a risk of weight loss, impaired immune system, increased weakness, increased length of stay, and mortality. Nutritional assessment should be performed on the stroke unit at the time of admission and throughout the hospital stay. The simplest but most valuable thing the nurse can do to monitor nutrition is to monitor the patient's weight and weight change over time and monitor the patient's dietary intake.^{263–265}

Stroke may compromise the patient's ability to self-feed, which can impact self-esteem. A pleasant environment that encompasses patience and encouragement fosters hope in the stroke patient. Use of seasonings, the serving of foods at appropriate temperatures, and augmentation of food presentation may enhance appeal and compensate for the patient's loss of taste or smell. An interdisciplinary approach to treating dysphagia will result in early detection and early intervention to minimize the impact on the patient's life, family, and healthcare costs.

Recommendations

Medical complications after stroke are common. The basics of care become critical care in the stroke patient. An overriding concept is patient and family education at all steps of the acute care process.

Class I

1. Infections, such as pneumonia and UTI, should be identified and treated immediately with antibiotics (*Class I, Level of Evidence B*).

- 2. Early bowel and bladder care should be instituted to prevent complications such as constipation and urinary retention or infection (*Class I, Level of Evidence A*). Use of indwelling catheters should be avoided if possible because of the risk of UTI (*Class I, Level of Evidence A*).
- 3. Early implementation of anticoagulant therapy or physical compression modalities should be considered for all stroke patients who cannot ambulate at 2 days and who are at risk for DVT or pulmonary embolus (*Class I, Level of Evidence A*). Early mobility should always be attempted if safe for the patient (*Class I, Level of Evidence B*).
- 4. Fall precautions should be initiated, and the stroke patient should be told not to ambulate without assistance (*Class I, Level of Evidence B*).
- 5. Frequent turning should be instituted in bedridden patients to prevent skin breakdown (Class I, Level of Evidence A). Use of the Braden Scale in nursing practice can assist in the prediction of stroke patients at high risk of developing pressure ulcers (Class I, Level of Evidence A). Range-of-motion exercises should start in the early phase of acute stroke care once risk has been assessed (Class I, Level of Evidence C).
- 6. A swallow screen should be performed in the first 24 hours after stroke, preferably by the speech language pathologist (*Class I, Level of Evidence B*). Nurses should be familiar with bedside swallow assessment if a formal evaluation cannot be done within the specified period. Stroke patients should be kept NPO until the screen has been performed (*Class I, Level of Evidence B*). Further studies of dysphagia in the setting of acute stroke should be performed.
- 7. Patients who cannot swallow should have a nasogastric tube placed, or if severity warrants, a percutaneous endoscopic gastrostomy tube should be placed (*Class I*, *Level of Evidence B*). Assessment of proper hydration is included in this recommendation.

Class IIa

- 1. If an indwelling catheter is required, excellent pericare and prevention of infection modalities should be instituted to prevent complications (*Class IIa, Level of Evidence C*).
- 2. The stroke patient can be fed either by intravenous infusion or through nasogastric or percutaneous endoscopic gastrostomy tubes (*Class IIa, Level of Evidence B*).

Class IIb

1. Nurses may provide passive range-of-motion exercises between physical therapy visits to help patients maintain joint mobility and prevent complications of immobility (*Class IIb, Level of Evidence C*).

The Nurse's Role in Secondary Prevention of Stroke

Nurses historically have played an important role in patient and family education, both at the bedside and in the outpatient setting. Stroke education includes explaining stroke, discussing risk factors (with emphasis placed on those risk factors that are modifiable²⁶⁶), and describing secondary prevention measures, including compliance with prescribed medications. The AHA guidelines for secondary prevention of stroke are being revised. Pertinent recommendations from the 2006 guidelines are provided in Table 14.267 Nurses play a significant role in education and lifestyle modification strategies, and therefore, they must stay current with changing standards and how they translate into practice. Suggestions for reducing blood pressure include adopting the DASH (Dietary Approaches to Stop Hypertension) diet, engaging in regular aerobic activity, and moderating alcohol intake.268-273 Nurses should be familiar with smoking cessation programs and materials and should include this information in the discharge plan of care.274 Guidelines for smoking cessation for both the clinician and the consumer are available from the Office of the Surgeon General (www.surgeongeneral.gov/tobacco/ default.htm) and the Agency for Health Care Policy and Research.

For the stroke patient with atrial fibrillation, the nurse should explain that use of warfarin (to maintain an international normalized ratio of 2 to 3) is recommended for prevention of stroke unless an absolute contraindication to anticoagulation exists. The nurse should also discuss the potential side effect of bleeding, interactions between warfarin and other drugs or certain foods, and the importance of compliance. The patient should be informed that weekly testing is required when warfarin is begun, then monthly testing when the international normalized ratio is stable. Specific educational aids are found in Table 15.

Nurses can educate stroke patients about secondary prevention modalities. Patient education is a standard performance measure in primary stroke center certification (see http://www.jointcommission.org/CertificationPrograms/ PrimaryStrokeCenters/stroke_measure_set.htm or http://www. jointcommission.org/CertificationPrograms/PrimaryStrokeCenters/ guide_table_contents.htm). Patients should be taught the rationale for administration of an antiplatelet drug and its potential side effects. They should also be taught that antiplatelet therapy is lifelong, not short term.

Discharge Planning

The discharge destination of stroke patients requires comprehensive planning.²⁷⁵ The NINDS suggests that 30% of stroke survivors will recover almost completely or with minor impairments.^{67,276} Another 40% will require subacute care, and 10% will require care in a skilled nursing facility. The remaining 15% will die soon after stroke onset. Of all survivors, $\approx 14\%$ will experience recurrent stroke within 1 year of the primary event. Eventually, 68% to 74% of stroke survivors will require the care of family members in the home.²⁷⁷ Detailed planning for postacute stroke care will optimize outcomes and reduce risk and may control financial burden.

The average length of hospital stay for a stroke patient in 2006 was 4.9 days, which may not be enough time to learn the true impact of a stroke.²⁷⁸ Order sets that include stroke team referrals will lead to initiation of early rehabilitation intervention. Such referrals include physiotherapy, occupational therapy, speech therapy, nutritional assessment, psychology,

Table 14. Recommendations for Secondary Stroke Prevention²⁶⁷

		Class/Leve of				
Risk Factor	Recommendation	Evidence				
Recommendations for treatable vascular risk factors						
Hypertension	Antihypertensive treatment is recommended for prevention of recurrent stroke and other vascular events in persons who have had an ischemic stroke and are beyond the hyperacute period.					
	Because this benefit extends to persons with and without a history of hypertension, this recommendation should be considered for all ischemic stroke and TIA patients.					
	An absolute target BP level and reduction are uncertain and should be individualized, but benefit has been associated with an average reduction of <10/5 mm Hg, and normal BP levels have been defined as <120/80 by JNC-7.	I/B				
	Several lifestyle modifications have been associated with BP reductions and should be included as part of a comprehensive approach antihypertensive therapy.	I/C				
	Optimal drug regimen remains uncertain; however, available data support the use of diuretics and the combination of diuretics and an ACEI. Choice of specific drugs and targets should be individualized on the basis of reviewed data and consideration, as well as specific patient characteristics (eg, extracranial cerebrovascular occlusive disease, renal impairment, cardiac disease, and DM).	I/A				
Diabetes	More rigorous control of blood pressure and lipids should be considered in patients with diabetes.	I/B				
	Although all major classes of antihypertensive medications are suitable for the control of BP, most patients will require >1 agent. ACEIs and ARBs are more effective in reducing the progression of renal disease and are recommended as first-choice medications for patients with DM.	I/A				
	Glucose control is recommended to near-normoglycemic levels among diabetics with ischemic stroke or TIA to reduce microvascular complications.	I/A				
	The goal for Hb A1c should be \leq 7%.	I/B				
Cholesterol	Ischemic stroke or TIA patients with elevated cholesterol, comorbid CAD, or evidence of an atherosclerotic origin should be managed according to NCEP III guidelines, which include lifestyle modification, dietary guidelines, and medication recommendations.	I/A				
	Statin agents are recommended, and the target goal for cholesterol lowering for those with CHD or symptomatic atherosclerotic disease is an LDL-C of <100 mg/dL and LDL-C <70 mg/dL for very-high-risk persons with multiple risk factors.	I/A				
	Patients with ischemic stroke or TIA presumed to be due to an atherosclerotic origin but with no preexisting indications for statins (normal cholesterol levels, no comorbid CAD, or no evidence of atherosclerosis) are reasonable to consider for treatment with a statin agent to reduce the risk of vascular events.	IIa/B				
	Ischemic stroke or TIA patients with low HDL-C may be considered for treatment with niacin or gemfibrozil.	llb/B				
Recommendations for nodifiable behavioral risk factors						
Smoking	All ischemic stroke or TIA patients who have smoked in the past year should be strongly encouraged not to smoke.	I/C				
	Avoid environmental smoke.	I/C				
	Counseling, nicotine products, and oral smoking cessation medications have been found to be effective for smokers.	lla/B				
Alcohol	Patients with prior ischemic stroke or TIA who are heavy drinkers should eliminate or reduce their consumption of alcohol.	I/A				
	Light to moderate levels of \leq 2 drinks per day for men and 1 drink per day for nonpregnant women may be considered.	llb/C				
Obesity	Weight reduction may be considered for all overweight ischemic stroke or TIA patients to maintain the goal of a BMI of 18.5 to 24.9 kg/m ² and a waist circumference of <35 in for women and <40 in for men. Clinicians should encourage weight management through an appropriate balance of caloric intake, physical activity, and behavioral counseling.	IIb/C				
Physical activity	For those with ischemic stroke or TIA who are capable of engaging in physical activity, at least 30 min of moderate-intensity physical exercise most days may be considered to reduce risk factors and comorbid conditions that increase the likelihood of recurrence of stroke.	llb/C				
		(Continued				

Table 14. Continued

Diale Factor		Class/Level of Evidence			
Risk Factor Physical activity	Recommendation For those with disability after ischemic stroke, a supervised therapeutic exercise regimen is				
	recommended.				
Recommendations for interventional approaches to patients with stroke caused by large-artery atherosclerotic disease					
Extracranial carotid disease	For patients with recent TIA or ischemic stroke within the past 6 months and ipsilateral severe (70% to 99%) carotid artery stenosis, CEA is recommended by a surgeon with a perioperative morbidity and mortality of $< 6\%$.	I/A			
	For patients with recent TIA or ischemic stroke and ipsilateral moderate (50% to 69%) carotid stenosis, CEA is recommended, depending on patient-specific factors such as age, gender, comorbidities, and severity of initial symptoms.	I/A			
	When degree of stenosis is $<$ 50%, there is no indication for CEA.	III/A			
	When CEA is indicated, surgery within 2 weeks rather than delayed surgery is suggested.	I/B			
	Among patients with symptomatic severe stenosis (>70%) in whom the stenosis is difficult to access surgically, medical conditions are present that greatly increase the risk for surgery, or when other specific circumstances exist such as radiation-induced stenosis or restenosis after CEA, CAS is not inferior to endarterectomy and may be considered.	llb/B			
	CAS is reasonable when performed by operators with established periprocedural morbidity and mortality rates of 4% to 6%, similar to that observed in trials of CEA and CAS.	lla/B			
	Among patients with symptomatic carotid occlusion, EC/IC bypass surgery is not recommended routinely.	III/A			
Extracranial vertebrobasilar disease	Endovascular treatment of patients with symptomatic extracranial vertebral stenosis may be considered when patients are having symptoms despite medical therapies (antithrombotics, statins, and other treatments for risk factors).	llb/C			
Intracranial arterial disease	The usefulness of endovascular therapy (angioplasty and/or stent placement) is uncertain for patients with hemodynamically significant intracranial stenoses who have symptoms despite medical therapies (antithrombotics, statins, and other treatments for risk factors) and is considered investigational	IIb/C			
Recommendations for patients with cardioembolic stroke types					
Atrial fibrillation (AF)	For patients with ischemic stroke or TIA with persistent or paroxysmal (intermittent) AF, anticoagulation with adjusted-dose warfarin (target INR, 2.5; range, 2.0-3.0) is recommended.	I/A			
	In patients unable to take oral anticoagulants, aspirin 325 mg/d is recommended.	I/A			
	Acute MI and LV thrombus For patients with an ischemic stroke caused by an acute MI in whom LV mural thrombus is identified by echocardiography or another form of cardiac imaging, oral anticoagulation is reasonable, aiming for an INR of 2.0 to 3.0 for at least 3 months and up to 1 y.	lla/B			
	Aspirin should be used concurrently for the ischemic CAD patient during oral anticoagulant therapy in doses up to 162 mg/d, preferably in the enteric-coated form.	I/A			
Cardiomyopathy	For patients with ischemic stroke or TIA who have dilated cardiomyopathy, either warfarin (INR, 2.0 to 3.0) or antiplatelet therapy may be considered for prevention of recurrent events.	llb/C			
Valvular heart disease, Rheumatic mitral valve disease	For patients with ischemic stroke or TIA who have rheumatic mitral valve disease, whether or not AF is present, long-term warfarin therapy is reasonable, with a target INR of 2.5 (range, 2.0–3.0).	lla/C			
	Antiplatelet agents should not be routinely added to warfarin in the interest of avoiding additional bleeding risk.	III/C			
	For ischemic stroke or TIA patients with rheumatic mitral valve disease, whether or not AF is present, who have a recurrent embolism while receiving warfarin, adding aspirin (81 mg/d) may be indicated.	lla/C			
Mitral valve prolapse (MVP)	For patients with MVP who have ischemic stroke or TIAs, long-term antiplatelet therapy is reasonable.	IIa/C			
MAC	For patients with ischemic stroke or TIA and MAC not documented to be calcific, antiplatelet therapy may be considered.	IIb/C			
	Among patients with mitral regurgitation resulting from MAC without AF, antiplatelet or warfarin therapy may be considered.	IIb/C			
		(Continued			

Table 14. Continued

Risk Factor	Recommendation	Class/Level of Evidence
Aortic valve disease	For patients with ischemic stroke or TIA and aortic valve disease who do not have AF, antiplatelet therapy may be considered.	llb/C
Prosthetic heart valves	For patients with ischemic stroke or TIA who have modern mechanical prosthetic heart valves, oral anticoagulants are recommended, with an INR target of 3.0 (range, 2.5–3.5).	I/B
	For patients with mechanical prosthetic heart valves who have an ischemic stroke or systemic embolism despite adequate therapy with oral anticoagulants, aspirin 75 to 100 mg/d, in addition to oral anticoagulants, and maintenance of the INR at a target of 3.0 (range, 2.5–3.5) is reasonable.	lla/B
	For patients with ischemic stroke or TIA who have bioprosthetic heart valves with no other source of thromboembolism, anticoagulation with warfarin (INR, 2.0–3.0) may be considered.	llb/C

JNC-7 indicates Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; ACEI, angiotensin-converting enzyme inhibitor; ARBs, angiotensin receptor blockers; Hb, hemoglobin; CAD, coronary artery disease; NCEP, National Cholesterol Education Panel; CHD, coronary heart disease; LDL-C, low-density lipoprotein cholesterol; BMI, body mass index; CEA, carotid endarterectomy; CAS, carotid angioplasty and stenting; EC/IC, extracranial/intracranial; AF, atrial fibrillation; MI, myocardial infarction; LV, left ventricular; MVP, mitral valve prolapse; and MAC, mitral annular calcification.

and social services. Once assessed by each discipline, the patient's discharge planning process may begin.

Neurological findings identified during the acute phase and throughout recovery will direct medical management, rehabilitation, and the nursing plan of care. Deficits include altered level of consciousness, confusion, behavioral disturbances, cognitive deficits in higher functions such as memory and ability to learn, motor deficits, disturbance in balance and coordination, somatosensory deficits, disorders of vision, unilateral neglect, speech and language deficits, swallowing disorder (dysphagia), and affective disorder. The rehabilitation medicine team (physiatrist, and speech, physical, and occupational therapists) will evaluate the patient during the acute hospitalization and develop a rehabilitation plan for discharge that will address the neurological deficits and appropriate rehabilitation needs. In addition, the rehabilitation team will give recommendations on the next level of care placement. The nurse caring for the patient must communicate the discharge plan to the patient and family and be the liaison between the patient/family and rehabilitation team and discharge planners. Table 16 identifies key strategies or components that will help nurses to facilitate successful discharge planning and transition to the next level of care.266,279,280

The goal of discharge planning is to ensure a safe transition between the acute care facility, rehabilitation, outpatient settings, primary care physician, and community while maintaining a continuity of care that will optimize rehabilitation potential and ensure proper secondary prevention as appropriate. Discharge planners (eg, social workers, case managers) will integrate the concerns and expectations of the interdisciplinary team with those of the patient, family, and associated support system(s). Meeting the discharge needs for less obvious deficits (eg, memory problems) may pose as great a challenge as addressing needs related to overt hemiparesis or aphasia. Teaching patients, families, and support systems how to identify and deal with the nuances of stroke will facilitate community reintegration and optimize outcomes. A study from a Cochrane review²⁸¹ found that recovering stroke patients placed on a structured care pathway demonstrated significant improvement in functional ability and quality of life. Another study found that improved patient outcomes were highly dependent on support systems, including family support and an organized systems approach to meeting the discharge needs of the patient and family.²⁸²

What if the stroke victim is too disabled to participate in or benefit from rehabilitation? Hospital staff plays a crucial role in discharge decisions.²⁸³ Continued care options are skilled nursing care to assess for future rehabilitation potential, skilled nursing care alone, palliative care, or hospice care.

In a 2003 report on public health and aging, the Centers for Disease Control and Prevention noted that among the Medicare population, postacute care was one of the fastestgrowing categories, and stroke had the highest number of post–acute care beneficiaries.²⁸⁴ Additional findings suggest that after adjustment for stroke severity, organized home health care for this population resulted in overall improved functional outcomes and increased cost-effectiveness compared with other discharge facility options.²⁸⁴ Reported costs of nursing home care approached \$15.2 billion compared with a home healthcare cost of \$3.8 billion.²⁸⁵ These 2 resources should alert discharge planners to the potential for and challenges of an increasing trend to home discharge.

Although outcomes for home care of stroke survivors are positive, home care may be associated with negative health outcomes for caregivers.²⁷⁷ Therefore, appropriate referrals to community resources, physician follow-up, support groups, governmental agencies, free services, faith-based communities, and research opportunities must be incorporated into discharge planning. However detailed it may be, discharge planning may not address all family questions or prepare family members for future issues. It is, however, the backdrop against which healing continues.

The Advanced Practice Nurse's Role in Acute Stroke Care

The role of the nurse-practitioner or clinical nurse specialist is vital to the care of AIS patients throughout the continuum.

Table 15. Stroke Educational Programs

ASA/AHA (www.strokeassociation.org and www.americanheart.org)
Stroke: Patient Education Tool Kit
Power to End Stroke
African American Power to End Stroke
Power to End Stroke—Family Reunion Toolkit
Stroke Connection magazine
How Stroke Affects Behavior: Our Guide to Physical and Emotional Changes
Living With Atrial Fibrillation: Our Guide to Managing a Key Stroke Risk Factor
Living With Disability After Stroke
Sex After Stroke: Our Guide to Intimacy After Stroke
Stroke: Are You at Risk? Our Guide to Stroke Risk Factors
Understanding Stroke: Our Guide to Explaining Stroke and How to Redu Your Risk
Caring for Someone With Aphasia
High Blood Pressure and Stroke
Warning Signs of Stroke: Our Easy-Reading Guide to Emergency Action
Being a Stroke Family Caregiver
Smoking and Your Risk of Stroke
Just Move: Our Guide to Physical Activity
Diabetes, Heart Disease, and Stroke
NINDS (www.ninds.nih.gov)
What You Need to Know About Stroke
Stroke Risk Factors and Symptoms
Brain Basics: Preventing Stroke
Neurological Diagnostic Tests and Procedures
Questions and Answers About Stroke
Questions and Answers About Carotid Endarterectomy
National Stroke Association (www.stroke.org)
Stroke Smart magazine
Stroke Fact Sheet
African Americans and Stroke Brochure
Cholesterol Brochure
Explaining Stroke Brochure
Intracranial Atherosclerosis Brochure
Recurrent Stroke Prevention Brochure
Reducing Risk and Recognizing Symptoms Brochure
Transient Ischemic Attack Brochure
Stroke Rapid Response EMS/Prehospital Education
Hip Hop Stroke–Brainiac Kids Stroke Education
Brain Attack Coalition (www.stroke-site.org)

These roles will be referred to as advanced practice nurses (APNs) for the purpose of the present report.

The APN plays an essential role in planning and leading a team to develop clinical tools, organizing team members and departments to expedite activities in the care of the AIS patient, and monitoring outcomes and initiating quality initiatives to improve care. The Brain Attack Coalition recommends that a primary stroke center have at least 1 APN to implement and coordinate program activities.53

Effective Nursing Strategies for Successful Table 16. Discharge Planning^{266,279,280}

- · Involve patient's family/caregiver in assessment of postdischarge needs and decision making and treatment planning.
- · Family and team meeting to discuss patient progress, rehabilitation goals, discharge needs or discharge issues, explanation of next level of care, providing care and support associated with these deficits and discuss ways to cope with stress associated with these impairments (eg, cognitive loss, urinary incontinence).
- Encourage the patient's family/caregiver to participate in the rehabilitation sessions and to be trained to assist the patient with functional activities.
- · Predischarge needs assessment of home before discharge performed by an occupational therapist to evaluate home environment and determine safety, need for home modification(s), and equipment needs.
- · Consider availability of support services and the desires of the patient's family/caregiver.
- Caregiver training (multidisciplinary education about communication strategies if the patient is aphasic, positioning and handling, transfers, shoulder care, how to promote independence, according to individual patient's strengths and limitations).
- Postdischarge follow-up (ensure plans are made for medical care, secondary prevention, rehabilitation, social support, home care, and nursing if needed, caregiver support and education).
- Provide education for patient's family/caregivers on stroke pathology, stroke prevention, stroke signs and symptoms, actions to take, follow-up appointments/therapy, treatment plan, and community resources and how to access. Education should be provided in interactive and written format and documented in the medical record to facilitate communication of education completed to other healthcare providers.
- · Liaison with community providers, linkage with appropriate resources.
- · Regular review of individual patient and caregiver psychosocial and support needs.
- Provision of information on discharge plans and postdischarge management to primary care physicians and community services.
- If the patient is aphasic, the nurse and rehabilitation team should assist in establishing a communication pattern with the patient's family/caregiver before discharge.

One study showed that financial outcomes were significantly improved when APNs functioned as outcome managers and collaborated with the multidisciplinary team to care for AIS patients.²⁸⁶ Patients had shorter lengths of stay, lower rates of UTI and skin breakdown, and less time until mobilization. The hospital had 2306 fewer patient-days and a total cost savings of \$2 467 328.

Stroke programs have shown that APNs have been key in improving recognition and management of AIS patients.²⁸⁷⁻²⁹⁰ The use of APNs in the Calgary Stroke Program enhanced patient care and patient satisfaction with continuity of care throughout hospitalization. Improvements in the care process were also noted: CT image time was reduced from 60 to 30 minutes, door-to-needle time in administration of rtPA decreased from 90 to 60 minutes, and consultation was faster.

The APN can integrate education, research, management, leadership, and consultation into the clinical role in making decisions about clinical management, in diagnostic reasoning, and in developing therapeutic interventions for the care of AIS patients. APNs are assuming leadership roles in developing programs such as dysphagia teams,

Reduce

incontinence teams, and anticoagulation clinics and are actively involved in discharge, palliative care, and secondary prevention clinics.^{291–299}

The Nurse's Role in Stroke Education in the Community

A landmark study evaluating the knowledge of patients presenting to the ED was first conducted in 1997 and repeated in 1998 and 2003.300 The first study showed that 40% of patients admitted did not know the signs, symptoms, or risk factors of stroke. A need for further public education to increase awareness of stroke warning signs was identified, and all healthcare professionals were encouraged to become involved in community education.³⁰⁰ In 1998, a populationbased interview was conducted in the greater Cincinnati, Ohio, area to establish the public's knowledge of risk factors and warning signs of acute stroke.301 Fifty-seven percent correctly named only 1 of the 5 warning signs of stroke defined by the NINDS, and 68% could identify only 1 stroke risk factor. Only 57% of patients with hypertension said that they were at risk for stroke. Sixty percent of patients <75 years of age correctly identified 1 warning sign of stroke compared with only 47% of patients >75 years of age.³⁰¹ As a result of organized community education in the greater Cincinnati region, community awareness of stroke warning signs significantly improved over time, but knowledge of risk of stroke based on individual risk factors did not improve.302

Studies have shown that some groups have less knowledge about stroke, delay seeking medical attention, and do not phone 9-1-1. These groups include the elderly, women, those with lower levels of education, and racial/ethnic populations such as blacks and Latinos. Healthcare professionals must educate the public about stroke warning signs and risk factors and actions to take when symptoms occur.^{303,304}

The effect of stroke screening on knowledge and behavioral changes has been studied.³⁰⁵ One screening used the National Stroke Association guidelines. Participants were evaluated before and after screening and 3 months after the event. Knowledge increased from 59% to 94% immediately after the event but at 3 months was only 77%. Only 27% of patients attending the screening had implemented an intervention to decrease stroke risk.³⁰⁵ Similar studies showed that \approx 50% of those attending a stroke screening made at least 1 behavioral change to reduce stroke risk and that community education improved the participant's knowledge of stroke warning signs and symptoms.^{306–308}

The National Stroke Association states that "80% of strokes can be prevented" (http://www.stroke.org/site/PageServer?pagename=PREVENT). This should compel nurses to educate the public about primary and secondary stroke prevention, including stroke awareness and stroke risk factors, and to call 9-1-1 if warning signs of stroke occur. The acronym FAST (Face, Arm, Speech, Time) is being used as a way to educate the public. "The Beauty Shop Project" educated beauticians on the FAST acronym and asked them to educate their clients about acute stroke signs by emphasizing the FAST acronym. After the project, 51% of participants could identify at least 3 stroke warning signs compared

Table 17. Specific Roles of Nursing in the Joint Commission Primary Stroke Center Certification

Bring team together toward common goal.

Organize team to accomplish stroke center certification.

Educate in-house nursing personnel, medical personnel, and ancillary personnel on stroke-related issues and philosophy of the Joint Commission stroke center certification.

Understand the Joint Commission requirements and understand deficits hospital may have in achieving goal.

Develop preprinted or standing order sets and critical pathways as required by the Joint Commission.

Develop stroke care procedures for strategic in-hospital units that care for stroke patients.

Appoint quality-assurance nurse to collect data on execution of performance measures.

Understand whether hospital is capable of responding to recommendations of Brain Attack Coalition.

Understand hospital's medical record coding to ensure it is appropriately capturing ICD-10 and DRGs for proper billing for optimum reimbursement potential.

Recognize that nursing personnel play an important role in the application process for Joint Commission certification in pulling together required documents for the site visit.

Ensure that hospital personnel are prepared for the Joint Commission reviewer.

Monitor performance measures established by the Joint Commission.

ICD indicates International Classification of Diseases; DRGs, Diagnosis-Related Groups.

with 41% at the beginning of the project. Ninety-three percent recognized the need to call 9-1-1 compared with 85% before the project began (http://www.webmd.com/stroke/ news/20070207/hip-hop-to-stroke-awareness).

The Massachusetts Health Promotion Clearinghouse Catalog has a 3-minute video on stroke awareness titled *Stroke Heroes Act FAST*. Three scenarios educate viewers about recognizing and responding to the signs of stroke using the FAST model (http://www.maclearinghouse.com/CatalogHDSP.htm).

In 2004, the National Stroke Association created "Hip Hop Stroke," a stroke education program for school-aged children. The interactive program is presented for 1 hour a day for 3 days. Hip-hop music and the character "Brainiac" teach children about lifestyle changes, warning signs of stroke, and the importance of calling 9-1-1 (http://www.stroke.org/site/PageNavigator/HipHopStroke).

Nurses have many avenues through which to educate the public: Churches, schools, and community and professional organizations. The AHA/ASA and National Stroke Association have educational information on their Web sites, as well as materials for stroke education (Table 15). Stroke education can be done through blood pressure, lipid, and glucose screenings; health education forums; and smoking cessation, exercise, and nutritional programs.

The Nurse's Role in Certification of Primary Stroke Centers

Certification of stroke centers has gained national recognition since publication of the Brain Attack Coalition recommendations in June 2000¹⁰ amid concern that stroke centers should require certification by a rigorous examining body. The Joint Commission, with the support of the AHA and ASA, developed a certification program using the ideas proposed in the Brain Attack Coalition paper as the template for the model (http://www.jointcommission.org/CertificationPrograms/PrimaryS-trokeCenters/). In 2004, the certification program was rolled out. More than 200 hospitals were certified by the end of 2006.⁵

Nurses will play an important role in obtaining and maintaining stroke center certification (Table 17), just as they do in the Joint Commission's general hospital accreditation. Nurses will ensure that hospital personnel are properly trained and knowledgeable about all aspects of stroke care and will interact with the multidisciplinary team throughout the care continuum. Nurses ensure that a patient's needs are met by all the disciplines involved in care and in many cases help integrate this care so that patients are discharged having received optimum care through best practices. Nurses will work to ensure that all aspects of this complicated disease are managed in a way that will produce positive outcomes (Table 18).

The AHA/ASA patient education toolkit is available to assist nurses and other healthcare professionals in providing patient and family education about stroke. The toolkit can be ordered online through the AHA/ASA Web site (www.strokeassociation.org) or by calling the AHA/ASA directly (1–888-4-STROKE). Information about Joint Commission primary stroke center certification can be found at the Joint Commission Web site (www.jointcommission. org/CertificationPrograms/ PrimaryStrokeCenters/).

The Nurse's Role in Quality Improvement Initiatives

Writing Group Disclosures

Nurses historically have been at the forefront of inpatient hospital quality improvement programs. This role will con-

Table 18.	Performance Measures From the Joint Commission
Primary Stro	oke Center Certification Program

Stroke-1	Deep vein thrombosis prophylaxis					
Stroke-2	Discharged on antithrombotic therapy					
Stroke-3	Patients with atrial fibrillation receiving anticoagulation therapy					
Stroke-4	Thrombolytic therapy administered					
Stroke-5	Antithrombotic therapy by end of hospital day two					
Stroke-6	Discharged on statin medication					
Stroke-7	Dysphagia screening					
Stroke-8	Stroke education					
Stroke-9	Smoking cessation/advice/counseling					
Stroke-10	Assessed for rehabilitation					

tinue to expand as nurses institute quality improvement programs for secondary prevention such as Get With the Guidelines Stroke, a patient management tool, or the Coverdale registry, both of which are used to collect data for primary stroke center certification.^{309,310} Get With the Guidelines Stroke offers evidence-based, best-practice information that can be compared with other hospitals using this quality improvement tool. Information derived from this data collection program can also assist in patient and family education.

In summary, the present scientific statement describes nursing's vital role in the first 2 phases of stroke, the emergency or hyperacute phase and the acute phase. Using strength of evidence, the recommendations on the organization and integration of care are designed to improve outcomes, decrease lengths of stay, decrease costs, and decrease event recurrence.¹⁰

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Pamela H. Mitchell	University of Washington	Principal Investigator of NIH research grant: Psychosocial/ Behavioral Intervention in PSD, R01 NR07755, 4/1/02–3/31/08†	None	None	None	None	None

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives \$10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns \$10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition. *Modest.

+Significant.

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Harold Adams	University of Iowa	None	None	None	None	None	None	None
Linda Baas	University of Cincinnati	None	None	None	None	None	None	None
Rosemarie B. King	Northwestern University	NIH†	None	None	None	None	None	3 Rehabilitation research sites, Stroke Caregiver Study—admit stroke survivors†

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+Significant.

References

- Rosamond W, Flegal K, Furie K, Go A, Greenlund K, Haase N, Hailpern SM, Ho M, Howard V, Kissela B, Kittner S, Lloyd-Jones D, McDermott M, Meigs J, Moy C, Nichol G, O'Donnell C, Roger V, Sorlie P, Steinberger J, Thom T, Wilson M, Hong Y. Heart disease and stroke statistics: 2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2008;117:e25–e146.
- Donnarumma R, Kongable G, Barch C, Braimah J, Bratina P, Daley S, Rapp K, Sailor S, Spilker J. Overview: hyperacute rt-PA stroke treatment: the NINDS rt-PA Stroke Study Group. J Neurosci Nurs. 1997;29:351–355.
- Bader MK, Palmer S. What's the "hyper" in hyperacute stroke? Strategies to improve outcomes in ischemic stroke patients presenting within 6 hours. AACN Adv Crit Care. 2006;17:194–214.
- Birbeck GL, Zingmond DS, Cui X, Vickrey BG. Multispecialty stroke services in California hospitals are associated with reduced mortality. *Neurology*. 2006;66:1527–1532.

- Stroke Unit Trialists' Collaboration. Organised inpatient (stroke unit) care for stroke. *Cochrane Database Syst Rev.* 2007;No. 4:CD000197.
- Long AF, Kneafsey R, Ryan J, Berry J. The role of the nurse within the multi-professional rehabilitation team. J Adv Nurs. 2002;37: 70–78.
- Jahnke HK, Zadrozny D, Garrity T, Hopkins S, Frey JL, Christopher M. Stroke teams and acute stroke pathways: one emergency department's two-year experience. *J Emerg Nurs*. 2003;29:133–139.
- Gillum RF. New considerations in analyzing stroke and heart disease mortality trends: the Year 2000 Age Standard and the International Statistical Classification of Diseases and Related Health Problems, 10th Revision. *Stroke*. 2002;33:1717–1721.
- Seshadri S, Beiser A, Kelly-Hayes M, Kase CS, Au R, Kannel WB, Wolf PA. The lifetime risk of stroke: estimates from the Framingham Study. *Stroke*. 2006;37:345–350.
- Alberts MJ, Hademenos G, Latchaw RE, Jagoda A, Marler JR, Mayberg MR, Starke RD, Todd HW, Viste KM, Girgus M, Shephard T, Emr M, Shwayder P, Walker MD. Recommendations for the establishment of

primary stroke centers: Brain Attack Coalition. JAMA. 2000;283: 3102–3109.

- Sackett DL. Rules of evidence and clinical recommendations on the use of antithrombotic agents. *Chest.* 1989;95(suppl):2S-4S.
- 12. Adams HP Jr, Adams RJ, Brott T, del Zoppo GJ, Furlan A, Goldstein LB, Grubb RL, Higashida R, Kidwell C, Kwiatkowski TG, Marler JR, Hademenos GJ; Stroke Council of the American Stroke Association. Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association. *Stroke*. 2003;34:1056–1083.
- 13. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, Grubb RL, Higashida RT, Jauch EC, Kidwell C, Lyden PD, Morgenstern LB, Qureshi AI, Rosenwasser RH, Scott PA, Wijdicks EF. 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 [published corrections appear in *Stroke*. 2007;38:e38 and 2007;38:e96]. *Stroke*. 2007;38:1655–1711.
- 14. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, Krieger D, Mayberg M, Morgenstern L, Ogilvy CS, Vespa P, Zuccarello M. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group [republished in *Circulation*. 2007;116:e391-e413]. *Stroke*. 2007;38:2001-2023.
- 15. Mayberg MR, Batjer HH, Dacey R, Diringer M, Haley EC, Heros RC, Sternau LL, Torner J, Adams HP Jr, Feinberg W. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke*. 1994;25:2315–2328.
- Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P. Antithrombotic and thrombolytic therapy for ischemic stroke: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest.* 2004; 126(suppl):483S–512S.
- Sturgeon JD, Folsom AR, Longstreth WT Jr, Shahar E, Rosamond WD, Cushman M. Hemostatic and inflammatory risk factors for intracerebral hemorrhage in a pooled cohort. *Stroke*. 2008;39:2268–2273.
- Sturgeon JD, Folsom AR, Longstreth WT Jr, Shahar E, Rosamond WD, Cushman M. Risk factors for intracerebral hemorrhage in a pooled prospective study. *Stroke*. 2007;38:2718–2725.
- Okada Y, Yamaguchi T, Minematsu K, Miyashita T, Sawada T, Sadoshima S, Fujishima M, Omae T. Hemorrhagic transformation in cerebral embolism. *Stroke*. 1989;20:598–603.
- Sahni R, Weinberger J. Management of intracerebral hemorrhage. Vasc Health Risk Manag. 2007;3:701–709.
- 21. Acker JE III, Pancioli AM, Crocco TJ, Eckstein MK, Jauch EC, Larrabee H, Meltzer NM, Mergendahl WC, Munn JW, Prentiss SM, Sand C, Saver JL, Eigel B, Gilpin BR, Schoeberl M, Solis P, Bailey JR, Horton KB, Stranne SK. Implementation strategies for emergency medical services within stroke systems of care: a policy statement from the American Heart Association/American Stroke Association Expert Panel on Emergency Medical Services Systems and the Stroke Council. *Stroke.* 2007;38:3097–3115.
- Kothari RU, Pancioli A, Liu T, Brott T, Broderick J. Cincinnati Prehospital Stroke Scale: reproducibility and validity. *Ann Emerg Med.* 1999;33:373–378.
- Barsan WG, Brott TG, Broderick JP, Haley EC, Levy DE, Marler JR. Time of hospital presentation in patients with acute stroke. *Arch Intern Med.* 1993;153:2558–2561.
- Marler JR. Early stroke diagnosis saves time. Ann Emerg Med. 1999; 33:450–451.
- Bratina P, Greenberg L, Pasteur W, Grotta JC. Current emergency department management of stroke in Houston, Texas. *Stroke*. 1995;26: 409–414.
- Brott T, Reed RL. Intensive care for acute stroke in the community hospital setting: the first 24 hours. *Stroke*. 1989;20:694–697.
- Carrozzella J, Jauch EC. Emergency stroke management: a new era. Nurs Clin North Am. 2002;37:35–57, vi.

- Crocco TJ, Moreno R, Jauch EC, Racine AN, Pio BJ, Liu T, Kothari RU. Teaching ACLS stroke objectives to prehospital providers: a case-based approach. *Prehosp Emerg Care*. 2003;7:229–234.
- Daley S, Braimah J, Sailor S, Kongable GL, Barch C, Rapp K, Bratina P, Spilker J, Donnarumma R. Education to improve stroke awareness and emergent response: the NINDS rt-PA Stroke Study Group. *J Neurosci Nurs.* 1997;29:393–396.
- Kidwell CS, Starkman S, Eckstein M, Weems K, Saver JL. Identifying stroke in the field: prospective validation of the Los Angeles prehospital stroke screen (LAPSS). *Stroke*. 2000;31:71–76.
- Kothari R, Barsan W, Brott T, Broderick J, Ashbrock S. Frequency and accuracy of prehospital diagnosis of acute stroke. *Stroke*. 1995;26: 937–941.
- Kothari R, Hall K, Brott T, Broderick J. Early stroke recognition: developing an out-of-hospital NIH Stroke Scale. *Acad Emerg Med.* 1997;4:986–990.
- Lacy CR, Suh DC, Bueno M, Kostis JB. Delay in presentation and evaluation for acute stroke: Stroke Time Registry for Outcomes Knowledge and Epidemiology (S.T.R.O.K.E.). Stroke. 2001;32:63–69.
- Llanes JN, Kidwell CS, Starkman S, Leary MC, Eckstein M, Saver JL. The Los Angeles Motor Scale (LAMS): a new measure to characterize stroke severity in the field. *Prehosp Emerg Care*. 2004;8:46–50.
- Morris DL, Rosamond W, Madden K, Schultz C, Hamilton S. Prehospital and emergency department delays after acute stroke: the Genentech Stroke Presentation Survey. *Stroke*. 2000;31:2585–2590.
- 36. Deleted in proof.
- Morris DL, Rosamond WD, Hinn AR, Gorton RA. Time delays in accessing stroke care in the emergency department. *Acad Emerg Med.* 1999;6:218–223.
- Porteous GH, Corry MD, Smith WS. Emergency medical services dispatcher identification of stroke and transient ischemic attack. *Prehosp Emerg Care*. 1999;3:211–216.
- Smith WS, Corry MD, Fazackerley J, Isaacs SM. Improved parametic sensitivity in identifying stroke victims in the prehospital setting. *Prehosp Emerg Care*. 1999;3:207–210.
- Emergency Nurses Association. Position statements: ENA board approves statement on joint ENA/ACEP five-level triage task force. Available at: http://www.ahrq.gov/research/esi/esi2.htm. Accessed July 8, 2007.
- Williams JE, Rosamond WD, Morris DL. Stroke symptom attribution and time to emergency department arrival: the delay in accessing stroke healthcare study. *Acad Emerg Med.* 2000;7:93–96.
- Williams LS, Bruno A, Rouch D, Marriott DJ. Stroke patients' knowledge of stroke: influence on time to presentation. *Stroke*. 1997; 28:912–915.
- Zweifler RM, York D, U TT, Mendizabal JE, Rothrock JF. Accuracy of paramedic diagnosis of stroke. J Stroke Cerebrovasc Dis. 1998;7: 446–448.
- Field JM, ed. Advanced Cardiovascular Life Support. Dallas, Tex: American Heart Association; 2006:103–117.
- Sahni R. Acute stroke: implications for prehospital care: National Association of EMS Physicians Standards and Clinical Practice Committee. *Prehosp Emerg Care*. 2000;4:270–272.
- Marler JR, Jones PW, Emr M, eds. Proceedings of a National Symposium on Rapid Identification and Treatment of Acute Stroke. Bethesda, Md: National Institute of Neurological Disorders and Stroke; 1997. NIH publication No. 97–4239.
- National Institutes of Health. Improving the Chain of Recovery for Acute Stroke in Your Community. Bethesda, Md: National Institute of Neurological Disorders and Stroke; 2003. NIH publication No. 03–5348.
- Pepe PE, Zachariah BS, Sayre MR, Floccare D. Ensuring the chain of recovery for stroke in your community. *Acad Emerg Med.* 1998;5: 352–358.
- Wojner AW, Morgenstern L, Alexandrov AV, Rodriguez D, Persse D, Grotta JC. Paramedic and emergency department care of stroke: baseline data from a citywide performance improvement study. *Am J Crit Care*. 2003;12:411–417.
- Wojner-Alexandrov AW, Alexandrov AV, Rodriguez D, Persse D, Grotta JC. Houston paramedic and emergency stroke treatment and outcomes study (HoPSTO). *Stroke*. 2005;36:1512–1518.
- American Heart Association. Acute stroke case. In: Advanced Cardiovascular Life Support Provider Manual. Dallas, Tex: American Heart Association; 2006:103–117.
- Gilboy N, Tanabe P, Travers D, Rosenau A, Eitel D. Emergency Severity Index, Version 4: Implementation Handbook. Rockville, Md: Agency for

Healthcare Research and Quality; 2005. AHRQ publication No. 05–0046-2.

- 53. Alberts MJ, Latchaw RE, Selman WR, Shephard T, Hadley MN, Brass LM, Koroshetz W, Marler JR, Booss J, Zorowitz RD, Croft JB, Magnis E, Mulligan D, Jagoda A, O'Connor R, Cawley CM, Connors JJ, Rose-DeRenzy JA, Emr M, Warren M, Walker MD; Brain Attack Coalition. Recommendations for comprehensive stroke centers: a consensus statement from the Brain Attack Coalition. *Stroke*. 2005;36: 1597–1616.
- 54. Florida Stroke Act. 2004-325.
- Division for Heart Disease and Stroke Prevention. State program: Massachusetts basic implementation. Available at: http://www.cdc.gov/ dhdsp/state_program/ma.htm. Accessed May 4, 2009.
- 56. Gropen TI, Gagliano PJ, Blake CA, Sacco RL, Kwiatkowski T, Richmond NJ, Leifer D, Libman R, Azhar S, Daley MB; NYSDOH Stroke Center Designation Project Workgroup. Quality improvement in acute stroke: the New York State Stroke Center Designation Project. *Neurology*. 2006;67:88–93.
- 57. Schwamm LH, Pancioli A, Acker JE III, Goldstein LB, Zorowitz RD, Shephard TJ, Moyer P, Gorman M, Johnston SC, Duncan PW, Gorelick P, Frank J, Stranne SK, Smith R, Federspiel W, Horton KB, Magnis E, Adams RJ. Recommendations for the establishment of stroke systems of care: recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems. *Circulation*. 2005;111: 1078–1091.
- 58. Suyama J, Crocco T. Prehospital care of the stroke patient. *Emerg Med Clin North Am.* 2002;20:537–552.
- Crocco TJ, Kothari RU, Sayre MR, Liu T. A nationwide prehospital stroke survey. *Prehosp Emerg Care*. 1999;3:201–206.
- Kidwell CS, Saver JL, Schubert GB, Eckstein M, Starkman S. Design and retrospective analysis of the Los Angeles Prehospital Stroke Screen (LAPSS). *Prehosp Emerg Care*. 1998;2:267–273.
- Harbison J, Hossain O, Jenkinson D, Davis J, Louw SJ, Ford GA. Diagnostic accuracy of stroke referrals from primary care, emergency room physicians, and ambulance staff using the face arm speech test. *Stroke*. 2003;34:71–76.
- Bray JE, Martin J, Cooper G, Barger B, Bernard S, Bladin C. An interventional study to improve paramedic diagnosis of stroke. *Prehosp Emerg Care*. 2005;9:297–302.
- Bray JE, Martin J, Cooper G, Barger B, Bernard S, Bladin C. Paramedic identification of stroke: community validation of the Melbourne ambulance stroke screen. *Cerebrovasc Dis.* 2005;20:28–33.
- Mosley I, Nicol M, Donnan G, Patrick I, Dewey H. Stroke symptoms and the decision to call for an ambulance. *Stroke*. 2007;38:361–366.
- Tirschwell DL, Longstreth WT Jr, Becker KJ, Gammans RE Sr, Sabounjian LA, Hamilton S, Morgenstern LB. Shortening the NIH Stroke Scale for use in the prehospital setting. *Stroke*. 2002;33:2801–2806.
- 66. Marler JR. Introduction. In: Proceedings of a National Symposium on Rapid Identification and Treatment of Acute Stroke. Bethesda, Md: National Institute of Neurological Disorders and Stroke; 1997. NIH publication No. 97–4239.
- National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med.* 1995;333:1581–1587.
- Sayre MR. Damage control: past, present, and future of prehospital stroke management. *Emerg Med Clin North Am.* 2002;20:877–886.
- Thurman RJ, Jauch EC. Acute ischemic stroke: emergent evaluation and management. *Emerg Med Clin North Am.* 2002;20:609–630, vi.
- Rosamond WD, Reeves MJ, Johnson A, Evenson KR; Paul Coverdell National Acute Stroke Registry Prototype Investigators. Documentation of stroke onset time: challenges and recommendations. *Am J Prev Med.* 2006;31(suppl 2):S230–234.
- Boatright JR. New urgency for rapid transport of patients with stroke to appropriate hospitals. J Emerg Nurs. 2003;29:344–346.
- Brainin M, Funk G, Dachenhausen A, Huber G, Matz K, Eckhardt R. Stroke emergency: evidence favours laying the patient on the paretic side. *Wien Med Wochenschr*. 2004;154:568–570.
- 73. Kereiakes DJ, Weaver WD, Anderson JL, Feldman T, Gibler B, Aufderheide T, Williams DO, Martin LH, Anderson LC, Martin JS, McKendall G, Sherrid M, Greenberg H, Teichman SL. Time delays in the diagnosis and treatment of acute myocardial infarction: a tale of eight cities: report from the Pre-hospital Study Group and the Cincinnati Heart Project. Am Heart J. 1990;120:773–780.

- Hacke W, Kaste M, Skyhoj Olsen T, Bogousslavsky J, Orgogozo JM. Acute treatment of ischemic stroke: European Stroke Initiative (EUSI). *Cerebrovasc Dis.* 2000;10(suppl 3):22–33.
- 75. Hacke W, Donnan G, Fieschi C, Kaste M, von Kummer R, Broderick JP, Brott T, Frankel M, Grotta JC, Haley EC Jr, Kwiatkowski T, Levine SR, Lewandowski C, Lu M, Lyden P, Marler JR, Patel S, Tilley BC, Albers G, Bluhmki E, Wilhelm M, Hamilton S; ATLANTIS Trials Investigators; ECASS Trials Investigators; NINDS rt-PA Study Group Investigators. Association of outcome with early stroke treatment: pooled analysis of ATLANTIS, ECASS, and NINDS rt-PA Stroke Trials. *Lancet.* 2004;368:768–774.
- 76. Hacke W, Kaste M, Bluhmki E, Brozman M, Dávalos A, Guidetti D, Larrue V, Lees KR, Medeghri Z, Machnig T, Schneider D, von Kummer R, Wahlgren N, Toni D; ECASS Investigators. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. *N Engl J Med.* 2008;359:1317–1329.
- Wahlgren N, Ahmed N, Dávalos A, Hacke W, Millán M, Muir K, Roine RO, Toni D, Lees KR; SITS Investigators. Thrombolysis with alteplase 3–4.5 h after acute ischaemic stroke (SITS-ISTR): an observational study. *Lancet*. 2008;372:1303–1309.
- 77a.del Zoppo GJ, Saver JL, Jauch EC, Adams HP Jr; on behalf of the American Heart Association Stroke Council. 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.* May 28, 2009. doi:10.1161/ STROKEAHA.109.192535. Available at: http://stroke.ahajournals.org/cgi/ reprint/STROKEAHA.109.192535.
- Brott T, Bogousslavsky J. Treatment of acute ischemic stroke. N Engl J Med. 2000;343:710–722.
- Jauch E, Kissella B, Stettler B. Acute stroke management. Updated April 9, 2007. Available at: http://emedicine.medscape.com/article/ 1159752-overview. Accessed November 26, 2008.
- Reith J, Jørgensen HS, Pedersen PM, Nakayama H, Raaschou HO, Jeppesen LL, Olsen TS. Body temperature in acute stroke: relation to stroke severity, infarct size, mortality, and outcome. *Lancet*. 1996;347: 422–425.
- Ginsberg MD, Busto R. Combating hyperthermia in acute stroke: a significant clinical concern. *Stroke*. 1998;29:529–534.
- Wang Y, Lim LL, Levi C, Heller RF, Fisher J. Influence of admission body temperature on stroke mortality. *Stroke*. 2000;31:404–409.
- Barch C, Spilker J, Bratina P, Rapp K, Daley S, Donnarumma R, Sailor S, Braimah J, Kongable G; NINDS rt-PA Stroke Study Group. Nursing management of acute complications following rt-PA in acute ischemic stroke. *J Neurosci Nurs.* 1997;29:367–372.
- Braimah J, Kongable G, Rapp K, Daley S, Bratina P, Sailor S, Barch C, Donnarumma R, Spilker J; NINDS rt-PA Stroke Study Group. Nursing care of acute stroke patients after receiving rt-PA therapy. *J Neurosci Nurs.* 1997;29:373–383.
- Bratina P, Rapp K, Barch C, Kongable G, Donnarumma R, Spilker J, Daley S, Braimah J, Sailor S; NINDS rt-PA Stroke Study Group. Pathophysiology and mechanisms of acute ischemic stroke. *J Neurosci Nurs.* 1997;29:356–360.
- Spilker J, Kongable G, Barch C, Braimah J, Brattina P, Daley S, Donnarumma R, Rapp K, Sailor S; NINDS rt-PA Stroke Study Group. Using the NIH Stroke Scale to assess stroke patients. *J Neurosci Nurs*. 1997;29:384–392.
- Ronning OM, Guldvog B. Should stroke victims routinely receive supplemental oxygen? A quasi-randomized controlled trial. *Stroke*. 1999; 30:2033–2037.
- Mitchell PH. Intracranial hypertension: influence of nursing care activities. Nurs Clin North Am. 1986;21:563–576.
- Rowat AM, Wardlaw JM, Dennis MS, Warlow CP. Patient positioning influences oxygen saturation in the acute phase of stroke. *Cerebrovasc Dis.* 2001;12:66–72.
- Pang JA, Yeung VT, Zhang YG. Do postural changes affect gas exchange in acute hemiplegia? Br J Clin Pract. 1988;42:501–502.
- Tyson SF, Nightingale P. The effects of position on oxygen saturation in acute stroke: a systematic review. *Clin Rehabil.* 2004;18:863–871.
- Wojner AW, El-Mitwalli A, Alexandrov AV. Effect of head positioning on intracranial blood flow velocities in acute ischemic stroke: a pilot study. *Crit Care Nurs Q.* 2002;24:57–66.
- Schwarz S, Georgiadis D, Aschoff A, Schwab S. Effects of body position on intracranial pressure and cerebral perfusion in patients with large hemispheric stroke. *Stroke*. 2002;33:497–501.

- Wojner-Alexander AW, Garami Z, Chernyshev OY, Alexandrov AV. Heads down: flat positioning improves blood flow velocity in acute ischemic stroke. *Neurology*. 2005;64:1354–1357.
- Phillips AM, Jardine DL, Parkin PJ, Hughes T, Ikram H. Brain stem stroke causing baroreflex failure and paroxysmal hypertension. *Stroke*. 2000;31:1997–2001.
- Semplicini A, Maresca A, Boscolo G, Sartori M, Rocchi R, Giantin V, Forte PL, Pessina AC. Hypertension in acute ischemic stroke: a compensatory mechanism or an additional damaging factor? *Arch Intern Med.* 2003;163:211–216.
- Fischberg GM, Lozano E, Rajamani K, Ameriso S, Fisher MJ. Stroke precipitated by moderate blood pressure reduction. *J Emerg Med.* 2000; 19:339–346.
- Treib J, Haass A, Stoll M, Grauer M. Monitoring and management of antihypertensive therapy induced deterioration in acute ischemic stroke. *Am J Hypertens*. 1996;9:513–514.
- Rose JC, Mayer SA. Optimizing blood pressure in neurological emergencies [published correction appears in *Neurocrit Care*. 2006;4:98; dosage error in text]. *Neurocrit Care*. 2004;1:287–289.
- Cottrell JE, Patel K, Turndorf H, Ransohoff J. Intracranial pressure changes induced by sodium nitroprusside in patients with intracranial mass lesions. *J Neurosurg*. 1978;48:329–331.
- Weiss MH, Spence J, Apuzzo ML, Heiden JS, McComb JG, Kurze T. Influence of nitroprusside on cerebral pressure autoregulation. *Neurosurgery*. 1979;4:56–59.
- 102. Hill MD, Lye T, Moss H, Barber PA, Demchuk AM, Newcommon NJ, Green TL, Kenney C, Cole-Haskayne A, Buchan AM. Hemi-orolingual angioedema and ACE inhibition after alteplase treatment of stroke. *Neurology*. 2003;60:1525–1527.
- 103. Agarwal P, Kumar S, Hariharan S, Eshkar N, Verro P, Cohen B, Sen S. Hyperdense middle cerebral artery sign: can it be used to select intraarterial versus intravenous thrombolysis in acute ischemic stroke? *Cerebrovasc Dis.* 2004;17:182–190.
- 104. Furlan A, Higashida R, Wechsler L, Gent M, Rowley H, Kase C, Pessin M, Ahuja A, Callahan F, Clark WM, Silver F, Rivera F. Intra-arterial prourokinase for acute ischemic stroke: the PROACT II study: a randomized controlled trial: Prolyse in Acute Cerebral Thromboembolism. *JAMA*. 1999;282:2003–2011.
- Qureshi AI. Endovascular treatment of cerebrovascular diseases and intracranial neoplasms. *Lancet*. 2004;363:804–813.
- 106. Mohammad Y, Xavier AR, Christoforidis G, Bourekas E, Slivka A. Qureshi grading scheme for angiographic occlusions strongly correlates with the initial severity and in-hospital outcome of acute ischemic stroke. J Neuroimaging. 2004;14:235–241.
- IMS Study Investigators. Combined intravenous and intra-arterial recanalization for acute ischemic stroke: the Interventional Management of Stroke Study. *Stroke*. 2004;35:904–911.
- 108. Smith WS, Sung G, Starkman S, Saver JL, Kidwell CS, Gobin YP, Lutsep HL, Nesbit GM, Grobelny T, Rymer MM, Silverman IE, Higashida RT, Budzik RF, Marks MP; MERCI Trial Investigators. Safety and efficacy of mechanical embolectomy in acute ischemic stroke: results of the MERCI trial. *Stroke*. 2005;36:1432–1438.
- 109. Noser EA, Shaltoni HM, Hall CE, Alexandrov AV, Garami Z, Cacayorin ED, Song JK, Grotta JC, Campbell MS III. Aggressive mechanical clot disruption: a safe adjunct to thrombolytic therapy in acute stroke? *Stroke*. 2005;36:292–296.
- 110. Flint AC, Duckwiler GR, Budzik RF, Liebeskind DS, Smith WS; MERCI and Multi MERCI Writing Committee. Mechanical thrombectomy of intracranial internal carotid occlusion: pooled results of the MERCI and Multi MERCI Part I trials. *Stroke*. 2007;38:1274–1280.
- 111. Bose A, Henkes H, Alfke K, Reith W, Mayer TE, Berlis A, Branca V, Sit SP; Penumbra Phase 1 Stroke Trial Investigators. The Penumbra System: a mechanical device for the treatment of acute stroke due to thromboembolism. *AJNR Am J Neuroradiol.* 2008;29:1409–1413.
- 112. Alexandrov AV, Molina CA, Grotta JC, Garami Z, Ford SR, Alvarez-Sabin J, Montaner J, Saqqur M, Demchuk AM, Moyé LA, Hill MD, Wojner AW; CLOTBUST Investigators. Ultrasound-enhanced systemic thrombolysis for acute ischemic stroke. *N Engl J Med.* 2004; 351:2170–2178.
- 113. Ernst R, Pancioli A, Tomsick T, Kissela B, Woo D, Kanter D, Jauch E, Carrozzella J, Spilker J, Broderick J. Combined intravenous and intraarterial recombinant tissue plasminogen activator in acute ischemic stroke. *Stroke*. 2000;31:2552–2557.

- Rymer MM, Summers D, Soper P. Development of clinical pathways for stroke management: an example from Saint Luke's Hospital, Kansas City. *Clin Geriatr Med.* 1999;15:741–764.
- 115. Bonnono C, Criddle LM, Lutsep H, Stevens P, Kearns K, Norton R. Emergi-paths and stroke teams: an emergency department approach to acute ischemic stroke. *J Neurosci Nurs*. 2000;32:298–305.
- Furåker C, Hellström-Muhli U, Walldal E. Quality of care in relation to a critical pathway from the staff's perspective. *J Nurs Manag.* 2004;12: 309–316.
- 117. Gibbon B, Watkins C, Barer D, Waters K, Davies S, Lightbody L, Leathley M. Can staff attitudes to team working in stroke care be improved? J Adv Nurs. 2002;40:105–111.
- 118. Kwan J, Hand P, Dennis M, Sandercock P. Effects of introducing an integrated care pathway in an acute stroke unit. *Age Ageing*. 2004;33: 362–367.
- Panella M, Marchisio S, Di Stanislao F. Reducing clinical variations with clinical pathways: do pathways work? Int J Qual Health Care. 2003;15:509–521.
- Sulch D, Melbourn A, Perez I, Kalra L. Integrated care pathways and quality of life on a stroke rehabilitation unit. *Stroke*. 2002;33: 1600–1604.
- Wilkinson G, Parcell M, MacDonald A. Cerebrovascular accident clinical pathway. J Qual Clin Pract. 2000;20:109–112.
- Roth EJ, Lovell L, Harvey RL, Heinemann AW, Semik P, Diaz S. Incidence of and risk factors for medical complications during stroke rehabilitation. *Stroke*. 2001;32:523–529.
- Katzan IL, Cebul RD, Husak SH, Dawson NV, Baker DW. The effect of pneumonia on mortality among patients hospitalized for acute stroke. *Neurology*. 2003;60:620–625.
- Bae HJ, Yoon DS, Lee J, Kim BK, Koo JS, Kwon O, Park JM. In-hospital medical complications and long-term mortality after ischemic stroke. *Stroke*. 2005;36:2441–2445.
- 125. Heuschmann PU, Kolominsky-Rabas PL, Misselwitz B, Hermanek P, Leffmann C, Janzen RW, Rother J, Buecker-Nott HJ, Berger K. Predictors of in-hospital mortality and attributable risks of death after ischemic stroke: the German Stroke Registers Study Group. *Arch Intern Med.* 2004;164:1761–1768.
- 126. Baird TA, Parsons MW, Phanh T, Butcher KS, Desmond PM, Tress BM, Colman PG, Chambers BR, Davis SM. Persistent poststroke hyperglycemia is independently associated with infarct expansion and worse clinical outcome. *Stroke*. 2003;34:2208–2214.
- Castillo J, Dávalos A, Noya M. Aggravation of acute ischemic stroke by hyperthermia is related to an excitotoxic mechanism. *Cerebrovasc Dis.* 1999;9:22–27.
- Castillo J, Dávalos A, Marrugat J, Noya M. Timing for fever-related brain damage in acute ischemic stroke. *Stroke*. 1998;29:2455–2460.
- Castillo J. Deteriorating stroke: diagnostic criteria, predictors, mechanisms and treatment. *Cerebrovasc Dis.* 1999;9(suppl 3):1–8.
- Langhorne P, Stott DJ, Robertson L, MacDonald J, Jones L, McAlpine C, Dick F, Taylor GS, Murray G. Medical complications after stroke: a multicenter study. *Stroke*. 2000;31:1223–1229.
- 131. Rocco A, Pasquini M, Cecconi E, Sirimarco G, Ricciardi MC, Vicenzini E, Altieri M, Di Piero V, Lenzi GL. Monitoring after the acute stage of stroke: a prospective study. *Stroke*. 2007;38:1225–1228.
- Brown MJ. Stroke management: beginnings. Outcomes Manag Nurs Pract. 2000;4:34–38.
- Wolff AM, Taylor SA, McCabe JF. Using checklists and reminders in clinical pathways to improve hospital inpatient care. *Med J Aust.* 2004; 181:428–431.
- 134. Furlan AJ. Overview: hospital care of acute stroke. In: *Proceedings of a National Symposium on Rapid Identification and Treatment of Acute Stroke.* Bethesda, Md: National Institute of Neurological Disorders and Stroke; 1997. NIH publication No. 97–4239.
- The NINDS t-PA Stroke Study Group. Intracerebral hemorrhage after intravenous t-PA therapy for ischemic stroke. *Stroke*. 1997;28: 2109–2118.
- Alexandrov AV, Black SE, Ehrlich LE, Caldwell CB, Norris JW. Predictors of hemorrhagic transformation occurring spontaneously and on anticoagulants in patients with acute ischemic stroke. *Stroke*. 1997;28: 1198–1202.
- 137. Toni D, Fiorelli M, Bastianello S, Sacchetti ML, Sette G, Argentino C, Montinaro E, Bozzao L. Hemorrhagic transformation of brain infarct: predictability in the first 5 hours from stroke onset and influence on clinical outcome. *Neurology*. 1996;46:341–345.

- Motto C, Ciccone A, Aritzu E, Boccardi E, De Grandi C, Piana A, Candelise L. Hemorrhage after an acute ischemic stroke: MAST-I Collaborative Group. *Stroke*. 1999;30:761–764.
- Lopez-Yunez AM, Bruno A, Williams LS, Yilmaz E, Zurrú C, Biller J. Protocol violations in community-based rTPA stroke treatment are associated with symptomatic intracerebral hemorrhage. *Stroke*. 2001;32: 12–16.
- 140. Grotta JC, Burgin WS, El-Mitwalli A, Long M, Campbell M, Morgenstern LB, Malkoff M, Alexandrov AV. Intravenous tissue-type plasminogen activator therapy for ischemic stroke: Houston experience 1996 to 2000. Arch Neurol. 2001;58:2009–2013.
- 141. Katzan IL, Furlan AJ, Lloyd LE, Frank JI, Harper DL, Hinchey JA, Hammel JP, Qu A, Sila CA. Use of tissue-type plasminogen activator for acute ischemic stroke: the Cleveland area experience. *JAMA*. 2000;283: 1151–1158.
- 142. Albers GW, Bates VE, Clark WM, Bell R, Verro P, Hamilton SA. Intravenous tissue-type plasminogen activator for treatment of acute stroke: the Standard Treatment with Alteplase to Reverse Stroke (STARS) study. JAMA. 2000;283:1145–1150.
- 143. Simon JE, Sandler DL, Pexman JH, Hill MD, Buchan AM; Calgary Stroke Programme. Is intravenous recombinant tissue plasminogen activator (rt-PA) safe for use in patients over 80 years old with acute ischaemic stroke? - The Calgary experience. *Age Ageing*. 2004;33: 143–149.
- 144. Kwiatkowski TG, Libman RB, Frankel M, Tilley BC, Morgenstern LB, Lu M, Broderick JP, Lewandowski CA, Marler JR, Levine SR, Brott T. Effects of tissue plasminogen activator for acute ischemic stroke at one year: National Institute of Neurological Disorders and Stroke Recombinant Tissue Plasminogen Activator Stroke Study Group. N Engl J Med. 1999;340:1781–1787.
- 145. Schlegel D, Kolb SJ, Luciano JM, Tovar JM, Cucchiara BL, Liebeskind DS, Kasner SE. Utility of the NIH Stroke Scale as a predictor of hospital disposition. *Stroke*. 2003;34:134–137.
- 146. Saposnik G, Webster F, O'Callaghan C, Hachinski V. Optimizing discharge planning: clinical predictors of longer stay after recombinant tissue plasminogen activator for acute stroke. *Stroke*. 2005;36:147–150.
- Semplicini A, Calò L. Administering antihypertensive drugs after acute ischemic stroke: timing is everything. CMAJ. 2005;172:625–626.
- 148. Marchal G, Young AR, Baron JC. Early postischemic hyperperfusion: pathophysiologic insights from positron emission tomography. J Cereb Blood Flow Metab. 1999;19:467–482.
- Johnston KC, Mayer SA. Blood pressure reduction in ischemic stroke: a two-edged sword? *Neurology*. 2003;61:1030–1031.
- Phillips SJ. Pathophysiology and management of hypertension in acute ischemic stroke. *Hypertension*. 1994;23:131–136.
- 151. Potter J, Robinson T, Ford G, James M, Jenkins D, Mistri A, Bulpitt C, Drummond A, Jagger C, Knight J, Markus H, Beevers G, Dewey M, Lees K, Moore A, Paul S; CHIRPS Trial Group. CHHIPS (Controlling Hypertension and Hypotension Immediately Post-Stroke) Pilot Trial: rationale and design. J Hypertens. 2005;23:649–655.
- 152. Chalmers J. The management of blood pressure in acute stroke. *Lancet Neurol.* 2003;2:593.
- Goldstein LB. Blood pressure management in patients with acute ischemic stroke [Published correction appears in *Hypertension*. 2004;43:e35]. *Hypertension*. 2004;43:137–141.
- Vemmos KN, Tsivgoulis G, Spengos K, Synetos A, Manios E, Vassilopoulou S, Zis V, Zakopoulos N. Blood pressure course in acute ischaemic stroke in relation to stroke subtype. *Blood Press Monit*. 2004;9:107–114.
- 155. Hajat C, Hajat S, Sharma P. Effects of poststroke pyrexia on stroke outcome: a meta-analysis of studies in patients. *Stroke*. 2000;31: 410–414.
- Kilpatrick MM, Lowry DW, Firlik AD, Yonas H, Marion DW. Hyperthermia in the neurosurgical intensive care unit. *Neurosurgery*. 2000; 47:850–855.
- 157. Kammersgaard LP, Jørgensen HS, Rungby JA, Reith J, Nakayama H, Weber UJ, Houth J, Olsen TS. Admission body temperature predicts long-term mortality after acute stroke: the Copenhagen Stroke Study. *Stroke*. 2002;33:1759–1762.
- 158. Knoll T, Wimmer ML, Gumpinger F, Haberl RL. The low normothermia concept: maintaining a core body temperature between 36 and 37 degrees C in acute stroke unit patients. *J Neurosurg Anesthesiol*. 2002;14:304–308.
- Singh V. Critical care assessment and management of acute ischemic stroke. J Vasc Interv Radiol. 2004;15(pt 2):S21–S27.

- Cavallini A, Micieli G, Marcheselli S, Quaglini S. Role of monitoring in management of acute ischemic stroke patients. *Stroke*. 2003;34: 2599–2603.
- Sulter G, Elting JW, Langedijk M, Maurits NM, De Keyser J. Admitting acute ischemic stroke patients to a stroke care monitoring unit versus a conventional stroke unit: a randomized pilot study. *Stroke*. 2003;34: 101–104.
- 162. Christensen H, Boysen G, Christensen AF, Johannesen HH. Insular lesions, ECG abnormalities, and outcome in acute stroke. J Neurol Neurosurg Psychiatry. 2005;76:269–271.
- Korpelainen JT, Sotaniemi KA, Mäkikallio A, Huikuri HV, Myllylä VV. Dynamic behavior of heart rate in ischemic stroke. *Stroke*. 1999;30: 1008–1013.
- Díez-Tejedor E, Fuentes B. Acute care in stroke: the importance of early intervention to achieve better brain protection. *Cerebrovasc Dis.* 2004; 17(suppl 1):130–137.
- 165. Perry L. Screening swallowing function of patients with acute stroke: part two: detailed evaluation of the tool used by nurses. J Clin Nurs. 2001;10:474–481.
- 166. Yong M, Kaste M. Dynamic of hyperglycemia as a predictor of stroke outcome in the ECASS-II trial. *Stroke*. 2008;39:2749–2755.
- Lindsberg PJ, Roine RO. Hyperglycemia in acute stroke. *Stroke*. 2004; 35:363–364.
- Leigh R, Zaidat OO, Suri MF, Lynch G, Sundararajan S, Sunshine JL, Tarr R, Selman W, Landis DM, Suarez JI. Predictors of hyperacute clinical worsening in ischemic stroke patients receiving thrombolytic therapy. *Stroke*. 2004;35:1903–1907.
- Van den Berghe G, Schoonheydt K, Becx P, Bruyninckx F, Wouters PJ. Insulin therapy protects the central and peripheral nervous system of intensive care patients. *Neurology*. 2005;64:1348–1353.
- Miranda-Ruiz R, Castanon-Gonzalez JA. Hyperglycemia in critically ill patients: clinical implications for treatment [in Spanish]. *Cir Cir.* 2004; 72:517–524.
- 171. Nazzaro P, Ciancio L, Vulpis V, Triggiani R, Schirosi G, Pirrelli A. Stress-induced hemodynamic responses are associated with insulin resistance in mild hypertensives. *Am J Hypertens*. 2002;15(pt 1):865–871.
- 172. Alvarez-Sabín J, Molina CA, Montaner J, Arenillas JF, Huertas R, Ribo M, Codina A, Quintana M. Effects of admission hyperglycemia on stroke outcome in reperfused tissue plasminogen activator-treated patients. *Stroke*. 2003;34:1235–1241.
- 173. Aronson D, Rayfield EJ. How hyperglycemia promotes atherosclerosis: molecular mechanisms. *Cardiovasc Diabetol.* 2002;1:1.
- 174. Melamed E. Reactive hyperglycaemia in patients with acute stroke. J Neurol Sci. 1976;29:267–275.
- 175. Juvela S, Siironen J, Kuhmonen J. Hyperglycemia, excess weight, and history of hypertension as risk factors for poor outcome and cerebral infarction after aneurysmal subarachnoid hemorrhage. J Neurosurg. 2005;102:998–1003.
- 176. Bruno A, Levine SR, Frankel MR, Brott TG, Lin Y, Tilley BC, Lyden PD, Broderick JP, Kwiatkowski TG, Fineberg SE; NINDS rt-PA Stroke Study Group. Admission glucose level and clinical outcomes in the NINDS rt-PA Stroke Trial. *Neurology*. 2002;59:669–674.
- 177. Els T, Klisch J, Orszagh M, Hetzel A, Schulte-Mönting J, Schumacher M, Lücking CH. Hyperglycemia in patients with focal cerebral ischemia after intravenous thrombolysis: influence on clinical outcome and infarct size. *Cerebrovasc Dis.* 2002;13:89–94.
- Williams LS, Rotich J, Qi R, Fineberg N, Espay A, Bruno A, Fineberg SE, Tierney WR. Effects of admission hyperglycemia on mortality and costs in acute ischemic stroke. *Neurology*. 2002;59:67–71.
- 179. Lindsberg PJ, Soinne L, Roine RO, Salonen O, Tatlisumak T, Kallela M, Häppölä O, Tiainen M, Haapaniemi E, Kuisma M, Kaste M. Community-based thrombolytic therapy of acute ischemic stroke in Helsinki. *Stroke*. 2003;34:1443–1449.
- Bruno A, Biller J, Adams HP Jr, Clarke WR, Woolson RF, Williams LS, Hansen MD. Acute blood glucose level and outcome from ischemic stroke: Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. *Neurology*. 1999;52:280–284.
- 181. Klijn CJ, Hankey GJ. Management of acute ischaemic stroke: new guidelines from the American Stroke Association and European Stroke Initiative. *Lancet Neurol*. 2003;2:698–701.
- Gray CS, Hildreth AJ, Alberti GK, O'Connell JE; GIST Collaboration. Poststroke hyperglycemia: natural history and immediate management [published correction appears in *Stroke*. 2004;35:1229]. *Stroke*. 2004; 35:122–126.

- 183. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in the critically ill patients. *N Engl J Med.* 2001;345:1359–1367.
- 184. Malmberg K; DIGAMI (Diabetes Mellitus, Insulin Glucose Infusion in Acute Myocardial Infarction) Study Group. Prospective randomised study of intensive insulin treatment on long term survival after acute myocardial infarction in patients with diabetes mellitus. *BMJ*. 1997;314: 1512–1515.
- 185. Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS. Glucose potassium insulin infusions in the treatment of acute stroke patients with mild to moderate hyperglycemia: the Glucose Insulin in Stroke Trial (GIST). *Stroke*. 1999;30:793–799.
- 186. Gray CS, Hildreth AJ, Sandercock PA, O'Connell JE, Johnston DE, Cartlidge NE, Bamford JM, James OF, Alberti KG; GIST Trialists Collaboration. Glucose-potassium-insulin infusions in the management of post-stroke hyperglycaemia: the UK Glucose Insulin in Stroke Trial (GIST-UK). *Lancet Neurol.* 2007;6:397–406.
- Chudley S. The effect of nursing activities on intracranial pressure. Br J Nurs. 1994;3:454–459.
- Price AM, Collins TJ, Gallagher A. Nursing care of the acute head injury: a review of the evidence. *Nurs Crit Care*. 2003;8:126–133.
- Bereczki D, Liu M, do Prado GF, Fekete I. Mannitol for acute stroke. Cochrane Database Syst Rev. 2001;No. 1:CD001153.
- Chen HJ, Lee TC, Wei CP. Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke*. 1992;23:957–961.
- 191. Koh MS, Goh KY, Tung MY, Chan C. Is decompressive craniectomy for acute cerebral infarction of any benefit? *Surg Neurol.* 2000;53: 225–230.
- Tulyapronchote R, Malkoff MD, Selhorst JB, Gomez CR. Treatment of cerebellar infarction by decompression suboccipital craniectomy. *Stroke*. 1993;24:478–480.
- Lanzino DJ, Lanzino G. Decompressive craniectomy for spaceoccupying supratentorial infarction: rationale, indications, and outcome. *Neurosurg Focus*. 2000;8:e3.
- 194. Jaeger M, Soehle M, Meixensberger J. Improvement of brain tissue oxygen and intracranial pressure during and after surgical decompression for diffuse brain oedema and space occupying infarction. Acta Neurochir Suppl. 2005;95:117–118.
- 195. Malm J, Bergenheim AT, Enblad P, Hårdemark HG, Koskinen LO, Naredi S, Nordström CH, Norrving B, Uhlin J, Lindgren A. The Swedish Malignant Middle cerebral artery Infarction Study: long-term results from a prospective study of hemicraniectomy combined with standardized neurointensive care. Acta Neurol Scand. 2006;113:25–30.
- Schwab S, Hacke W. Surgical decompression of patients with large middle cerebral artery infarcts is effective. *Stroke*. 2003;34:2304–2305.
- 197. Fraser JF, Hartl R. Decompressive craniectomy as a therapeutic option in the treatment of hemispheric stroke. *Curr Atheroscler Rep.* 2005;7: 296–304.
- Curry WT Jr, Sethi MK, Ogilvy CS, Carter BS. Factors associated with outcome after hemicraniectomy for large middle cerebral artery territory infarction. *Neurosurgery*. 2005;56:681–692.
- Hanley DF, Hacke W. Critical care and emergency medicine neurology. Stroke. 2004;35:365–366.
- Gupta R, Connolly ES, Mayer S, Elkind MS. Hemicraniectomy for massive middle cerebral artery territory infarction: a systematic review. *Stroke*. 2004;35:539–543.
- 201. Morley NC, Berge E, Cruz-Flores S, Whittle IR. Surgical decompression for cerebral oedema in acute ischaemic stroke. *Cochrane Database Syst Rev.* 2002;No. 3:CD003435.
- 202. Foerch C, Lang JM, Krause J, Raabe A, Sitzer M, Seifert V, Steinmetz H, Kessler KR. Functional impairment, disability, and quality of life outcome after decompressive hemicraniectomy in malignant middle cerebral artery infarction. J Neurosurg. 2004;101:248–254.
- 203. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, Amelink GJ, Schmiedeck P, Schwab S, Rothwell PM, Bousser MG, van der Worp HB, Hacke W; DECIMAL, DESTINY, and HAMLET Investigators. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. *Lancet Neurol.* 2007;6:215–222.
- 204. De Reuck J, De Groote L, Van Maele G. Single seizure and epilepsy in patients with a cerebral territorial infarct. J Neurol Sci. 2008;271: 127–130.

- 205. Rosengart AJ, Huo JD, Tolentino J, Novakovic RL, Frank JI, Goldenberg FD, Macdonald RL. Outcome in patients with subarachnoid hemorrhage treated with antiepileptic drugs. *J Neurosurg*. 2007;107:253–260.
- 206. Schramm P, Schellinger PD, Klotz E, Kallenberg K, Fiebach JB, Kulkens S, Heiland S, Knauth M, Sartor K. Comparison of perfusion computed tomography and computed tomography angiography source images with perfusion-weighted imaging and diffusion-weighted imaging in patients with acute stroke of less than 6 hours' duration. *Stroke.* 2004;35:1652–1658.
- Lee LJ, Kidwell CS, Alger J, Starkman S, Saver JL. Impact on stroke subtype diagnosis of early diffusion-weighted magnetic resonance imaging and magnetic resonance angiography. *Stroke*. 2000;31: 1081–1089.
- Kidwell CS, Alger JR, Saver JL. Beyond mismatch: evolving paradigms in imaging the ischemic penumbra with multimodal magnetic resonance imaging. *Stroke*. 2003;34:2729–2735.
- 209. Wong TY, Klein R, Sharrett AR, Couper DJ, Klein BE, Liao DP, Hubbard LD, Mosley TH; ARIC Investigators, Atherosclerosis Risk In Communities Study. Cerebral white matter lesions, retinopathy, and incident clinical stroke. JAMA. 2002;288:67–74.
- Society of Gastroenterology Nurses and Associates. SGNA guidelines for nursing care of the patient receiving sedation and analgesia in the gastrointestinal endoscopy setting. *Gastroenterol Nurs.* 2000;23: 125–129.
- Goodwin SA. Pharmacologic management of patients undergoing conscious sedation. *Clin Nurse Spec.* 2001;15:269–271.
- Williams LS, Jones WJ, Shen J, Robinson RL, Weinberger M, Kroenke K. Prevalence and impact of depression and pain in neurology outpatients. *J Neurol Neurosurg Psychiatry*. 2003;74:1587–1589.
- Indredavik B, Rohweder G, Naalsund E, Lydersen S. Medical complications in a comprehensive stroke unit and an early supported discharge service. *Stroke*. 2008;39:414–420.
- Pearson SD, Goulart-Fisher D, Lee TH. Critical pathways as a strategy for improving care: problems and potential. *Ann Intern Med.* 1995;123: 941–948.
- Summers D, Soper PA. Implementation and evaluation of stroke clinical pathways and the impact on cost of stroke care. J Cardiovasc Nurs. 1998;13:69–87.
- Wentworth DA, Atkinson RP. Implementation of an acute stroke program decreases hospitalization costs and length of stay. *Stroke*. 1996;27:1040–1043.
- 217. Langhorne P. Organisation of acute stroke care. *Br Med Bull*. 2000;56: 436–443.
- Rudd AG, Hoffman A, Irwin P, Pearson M, Lowe D; Intercollegiate Working Party for Stroke. Stroke units: research and reality: results from the National Sentinel Audit of Stroke. *Qual Saf Health Care*. 2005; 14:7–12.
- Rudd AG, Hoffman A, Irwin P, Lowe D, Pearson MG. Stroke unit care and outcome: results from the 2001 National Sentinel Audit of Stroke (England, Wales, and Northern Ireland). *Stroke*. 2005;36:103–106.
- Launois R, Giroud M, Mégnigbêto AC, Le Lay K, Presenté G, Mahagne MH, Durand I, Gaudin AF. Estimating the cost-effectiveness of stroke units in France compared with conventional care. *Stroke*. 2004;35: 770–775.
- 221. Krespi Y, Gurol ME, Coban O, Tuncay R, Bahar S. Stroke unit versus neurology ward: a before and after study. J Neurol. 2003;250: 1363–1369.
- 222. Steiner MM, Brainin M. The quality of acute stroke units on a nation-wide level: the Austrian Stroke Registry for acute stroke units. *Eur J Neurol.* 2003;10:353–360.
- Brown MM. Brain attack: a new approach to stroke. Clin Med. 2002; 2:60-65.
- 224. Hilker R, Poetter C, Findeisen N, Sobesky J, Jacobs A, Neveling M, Heiss WD. Nosocomial pneumonia after acute stroke: implications for neurological intensive care medicine. *Stroke*. 2003;34:975–981.
- 225. Hinchey JA, Shephard T, Furie K, Smith D, Wang D, Tonn S; Stroke Practice Improvement Network Investigators. Formal dysphagia screening protocols prevent pneumonia. *Stroke*. 2005;36:1972–1976.
- 226. Upadya A, Thorevska N, Sena KN, Manthous C, Amoateng-Adjepong Y. Predictors and consequences of pneumonia in critically ill patients with stroke. J Crit Care. 2004;19:16–22.
- 227. Chamorro A, Horcajada JP, Obach V, Vargas M, Revilla M, Torres F, Cervera A, Planas AM, Mensa J. The Early Systemic Prophylaxis of Infection After Stroke study: a randomized clinical trial. *Stroke*. 2005; 36:1495–1500.

- Celik SA, Kanan N. A current conflict: use of isotonic sodium chloride solution on endotracheal suctioning in critically ill patients. DCCN -Dimensions of Critical Care Nursing. 2006;25:11–14.
- 229. Aslanyan S, Weir CJ, Diener HC, Kaste M, Lees KR; GAIN International Steering Committee and Investigators. Pneumonia and urinary tract infection after acute ischaemic stroke: a tertiary analysis of the GAIN International trial. *Eur J Neurol.* 2004;11:49–53.
- Kong KH, Young S. Incidence and outcome of poststroke urinary retention: a prospective study. Arch Phys Med Rehabil. 2000;81: 1464–1467.
- McLean DE. Medical complications experienced by a cohort of stroke survivors during inpatient, tertiary-level stroke rehabilitation. *Arch Phys Med Rehabil.* 2004;85:466–469.
- Field TS, Green TL, Roy K, Pedersen J, Hill MD. Trends in hospital admission for stroke in Calgary. *Can J Neurol Sci.* 2004;31:387–393.
- Ween JE, Alexander MP, D'Esposito M, Roberts M. Incontinence after stroke in a rehabilitation setting: outcome associations and predictive factors. *Neurology*. 1996;47:659–663.
- 234. Gelber DA, Good DC, Laven LJ, Verhulst SJ. Causes of urinary incontinence after acute hemispheric stroke. *Stroke*. 1993;24:378–382.
- Chan H. Bladder management in acute care of stroke patients: a quality improvement project. J Neurosci Nurs. 1997;29:187–190.
- Patel M, Coshall C, Rudd AG, Wolfe CD. Natural history and effects on 2-year outcomes of urinary incontinence after stroke. *Stroke*. 2001;32: 122–127.
- 237. Bernhardt J, Dewey H, Thrift A, Donnan G. Inactive and alone: physical activity within the first 14 days of acute stroke unit care. *Stroke*. 2004;35:1005–1009.
- Bamford J, Dennis M, Sandercock P, Burn J, Warlow C. The frequency, causes and timing of death within 30 days of a first stroke: the Oxfordshire Community Stroke Project. *J Neurol Neurosurg Psychiatry*. 1990;53:824–829.
- Brandstater ME, Shutter LA. Rehabilitation interventions during acute care of stroke patients. *Top Stroke Rehabil*. 2002;9:48–56.
- Wijdicks EF, Scott JP. Pulmonary embolism associated with acute stroke. Mayo Clin Proc. 1997;72:297–300.
- Jacobs BS, Baker PL, Roychoudhury C, Mehta RH, Levine SR. Improved quality of stroke care for hospitalized Medicare beneficiaries in Michigan. *Stroke*. 2005;36:1227–1231.
- Roychoudhury C, Jacobs BS, Baker PL, Schultz D, Mehta RH, Levine SR. Acute ischemic stroke in hospitalized Medicare patients: evaluation and treatment. *Stroke*. 2004;35:e22–e23.
- Black PM, Crowell RM, Abbott WM. External pneumatic calf compression reduces deep venous thrombosis in patients with ruptured intracranial aneurysms. *Neurosurgery*. 1986;18:25–28.
- Kamran SI, Downey D, Ruff RL. Pneumatic sequential compression reduces the risk of deep vein thrombosis in stroke patients. *Neurology*. 1998;50:1683–1688.
- 245. Gould MK, Dembitzer AD, Doyle RL, Hastie TJ, Garber AM. Lowmolecular-weight heparins compared with unfractionated heparin for treatment of acute deep venous thrombosis: a meta-analysis of randomized, controlled trials. *Ann Intern Med.* 1999;130:800–809.
- 246. Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, Ray JG. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest.* 2004; 126(suppl):338S–400S.
- 247. Sherman DG, Albers GW, Bladin C, Fieschi C, Gabbai AA, Kase CS, O'Riordan W, Pineo GF; PREVAIL Investigators. 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.
- Jørgensen L, Engstad T, Jacobsen BK. Higher incidence of falls in long-term stroke survivors than in population controls: depressive symptoms predict falls after stroke. *Stroke*. 2002;33:542–547.
- Kelly-Hayes M. Stroke outcome measures. J Cardiovasc Nurs. 2004; 19:301–307.
- Poole KE, Reeve J, Warburton EA. Falls, fractures, and osteoporosis after stroke: time to think about protection? *Stroke*. 2002;33:1432–1436.
- Braden BJ, Bergstrom N. Clinical utility of the Braden scale for predicting pressure sore risk. *Decubitus*. 1989;2:44–46, 50–51.
- 252. Bergstrom N, Demuth PJ, Braden BJ. A clinical trial of the Braden Scale for predicting pressure sore risk. *Nurs Clin North Am.* 1987;22: 417–428.

- Comfort EH. Reducing pressure ulcer incidence through Braden Scale risk assessment and support surface use. *Adv Skin Wound Care*. 2008; 21:330–334.
- 254. Galvan TJ. Dysphagia: going down and staying down. Am J Nurs. 2001;101:37–42, quiz 43.
- 255. Massey R, Jedlicka D. The Massey Bedside Swallowing Screen. *J Neurosci Nurs.* 2002;34:252–253, 257–260.
- 256. Scottish Intercollegiate Guidelines Network (SIGN). Management of Patients with Stroke: Identification and Management of Dysphagia: A National Clinical Guideline. Edinburgh, United Kingdom: Scottish Intercollegiate Guidelines Network; 2004. No. 78.
- 257. Paik NJ, Han TR, Park JW, Lee EK, Park MS, Hwang IK. Categorization of dysphagia diets with the line spread test. Arch Phys Med Rehabil. 2004;85:857–861.
- Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW. Malnutrition in stroke patients on the rehabilitation service and at follow-up: prevalence and predictors. *Arch Phys Med Rehabil*. 1995;76:310–316.
- 259. Aptaker RL, Roth EJ, Reichhardt G, Duerden ME, Levy CE. Serum albumin level as a predictor of geriatric stroke rehabilitation outcome. *Arch Phys Med Rehabil.* 1994;75:80–84.
- Finestone HM, Greene-Finestone LS, Wilson ES, Teasell RW. Prolonged length of stay and reduced functional improvement rate in malnourished stroke rehabilitation patients. *Arch Phys Med Rehabil*. 1996; 77:340–345.
- 261. Hickey JV. *The Clinical Practice of Neurological and Neurosurgical Nursing*. Philadelphia, Pa: Lippincott, Williams & Wilkins; 2003.
- 262. Mead GE, Donaldson L, North P, Dennis MS. An informal assessment of nutritional status in acute stroke for use in an international multicentre trial of feeding regimens. *Int J Clin Pract.* 1998;52:316–318.
- Nightingale JM, Walsh N, Bullock ME, Wicks AC. Three simple methods of detecting malnutrition on medical wards. J R Soc Med. 1996;89:144–148.
- 264. Frisancho AR. New standards of weight and body composition by frame size and height for assessment of nutritional status of adults and the elderly. *Am J Clin Nutr.* 1984;40:808–819.
- Ha L, Iversen PO, Hauge T. Nutrition for elderly acute stroke patients [in Norwegian]. *Tidsskr Nor Laegeforen*. 2008;128:1946–1950.
- 266. Smith J, Forster A, House A, Knapp P, Wright J, Young J. Information provision for stroke patients and their caregivers. *Cochrane Database Syst Rev.* 2008; No. 2: CD001919.
- 267. Sacco RL, Adams R, Albers G, Alberts MJ, Benavente O, Furie K, Goldstein LB, Gorelick P, Halperin J, Harbaugh R, Johnston SC, Katzan I, Kelly-Hayes M, Kenton EJ, Marks M, Schwamm LH, Tomsick T. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: a statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke: co-sponsored by the Council on Cardiovascular Radiology and Intervention: the American Academy of Neurology affirms the value of this guideline. *Stroke*. 2006;37:577–617.
- 268. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER III, Simons-Morton DG, Karanja N, Lin PH; 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.
- 269. Vollmer WM, Sacks FM, Ard J, Appel LJ, Bray GA, Simons-Morton DG, Conlin PR, Svetkey LP, Erlinger TP, Moore TJ, Karanja N; DASH-Sodium Collaborative Research Group. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. Ann Intern Med. 2001;135:1019–1028.
- Chobanian AV, Hill M. National Heart, Lung, and Blood Institute Workshop on Sodium and Blood Pressure: a critical review of current scientific evidence. *Hypertension*. 2000;35:858–863.
- Kelley GA, Kelley KS. Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2000;35:838–843.
- Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med.* 2002;136:493–503.
- 273. Xin X, He J, Frontini MG, Ogden LG, Motsamai OI, Whelton PK. Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2001;38:1112–1117.
- Litt J, Ling MY, McAvoy B. How to help your patients quit: practice-based strategies for smoking cessation. Asia Pacific Family Medicine. 2003;2:175–179.

- Early Supported Discharge Trialists. Services for reducing duration of hospital care for acute stroke patients. *Cochrane Database Syst Rev.* 2005;No. 2:CD000443.
- 276. National Institute of Neurological Disorders Stroke rt-PA Stroke Study Group. Recombinant tissue plasminogen activator for minor strokes: the National Institute of Neurological Disorders and Stroke rt-PA Stroke Study experience. Ann Emerg Med. 2005;46:243–252.
- 277. Bakas T, Austin JK, Jessup SL, Williams LS, Oberst MT. Time and difficulty of tasks provided by family caregivers of stroke survivors. *J Neurosci Nurs.* 2004;36:95–106.
- DeFrances CJ, Lucas CA, Buie VC, Golosinskiy A. 2006 National Hospital Discharge Survey. National Health Statistics Reports, No. 5. Hyattsville, Md: National Center for Health Statistics; 2008:12. Table 4.
- 279. Lindsay P, Bayley M, McDonald A, Graham ID, Warner G, Phillips S. Toward a more effective approach to stroke: Canadian Best Practice Recommendations for Stroke Care [published correction appears in *CMAJ*. 2008;179:56]. *CMAJ*. 2008;178:1418–1425.
- 280. VA/DoD Clinical Practice Guideline Working Group. Management of Stroke Rehabilitation. Washington, DC: VHA, Department of Veterans Affairs and Health Affairs, Department of Defense; February 2003. Office of Quality and Performance publication No. 10Q CPG/STR-03.
- Shepperd S, Parkes J, McClaren J, Phillips C. Discharge planning from hospital to home. *Cochrane Database Syst Rev.* 2004;No. 1:CD000313.
- Clark PA, Drain M, Gesell SB, Mylod DM, Kaldenberg DO, Hamilton J. Patient perceptions of quality in discharge instruction. *Patient Educ Couns.* 2005;59:56–68.
- 283. Gresham GE, Duncan PW, Stason WB, et al. *Post-Stroke Rehabilitation*. Clinical Practice Guideline, No. 16. Rockville, Md: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1995. AHCPR publication No. 95–0062.
- Centers for Disease Control and Prevention (CDC). Public health and aging: hospitalizations for stroke among adults aged >/=65 years: United States, 2000. MMWR Morb Mortal Wkly Rep. 2003;52:586–589.
- 285. Rosamond W, Flegal K, Friday G, Furie K, Go A, Greenlund K, Haase N, Ho M, Howard V, Kissela B, Kittner S, Lloyd-Jones D, McDermott M, Meigs J, Moy C, Nichol G, O'Donnell CJ, Roger V, Rumsfeld J, Sorlie P, Steinberger J, Thom T, Wasserthiel-Smoller S, Hong Y; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics: 2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee [published correction appears in *Circulation*. 2007;115:e172]. *Circulation*. 2007;115:e69–e171.
- Russell D, VorderBruegge M, Burns SM. Effect of an outcomesmanaged approach to care of neuroscience patients by acute care nurse practitioners. *Am J Crit Care*. 2002;11:353–362.
- Green T, Newcommon N. Advancing nursing practice: the role of the nurse practitioner in an acute stroke program. *J Neurosci Nurs*. 2006; 38(suppl):328–330.
- Minchin A, Wensley M. The medical nurse practitioner's role in early stroke recognition. *Nurs Times*. 2003;99:33–35.
- Bahouth MN, LaMonte MP. Acute ischemic stroke: evaluation and management strategies. *Topics in Advanced Practice Nursing eJournal*. 2005;5:1–11.
- Fitzpatrick M, Birns J. Thrombolysis for acute ischaemic stroke and the role of the nurse. Br J Nurs. 2004;13:1170–1174.
- Lees L, Sharpe L, Edwards A. Nurse-led dysphagia screening in acute stroke patients. *Nurs Stand.* 2006;21:35–42.
- 292. Shaughnessy M, Michael KM, Normandt P. When stroke strikes: minimizing impact and maximizing recovery. *Adv Nurse Pract.* 2006;14: 26–33; quiz 34.

- Neal-Boylan L. The rehabilitation nurse specialist in home care. *Home Healthc Nurse*. 2006;24:457–458.
- Gordon C, Weller C. A continence pathway for acute stroke care. *Nurs Times*. 2006;102:57–58.
- 295. Werner H. The benefits of the dysphagia clinical nurse specialist role. *J Neurosci Nurs.* 2005;37:212–215.
- Allen K, Hazelett S, Jarjoura D, Wright K, Clough L, Weinhardt J. Improving stroke outcomes: implementation of a postdischarge care management model. J Clin Outcomes Manag. 2004;11:707–717.
- 297. Hamlin SK, Brown TR. Comparing weight reduction and medications in treating hypertension: a systematic literature review. *Internet J Adv Nurs Pract.* 1999;3(2). Available at: http://www.ispub.com/journal/ the_internet_journal_of_advanced_nursing_practice/volume_9_ number_2_8/article/comparing_weight_reduction_and_medications_ in_treating_mild_hypertension_a_systematic_literature_review.html. Accessed May 5, 2009.
- 298. Frank RR. Caring for terminally ill patients: one hospital's team approach. J Critical Illness. 1999;14:51–55.
- McBride KL, White CL, Sourial R, Mayo N. Postdischarge nursing interventions for stroke survivors and their families. J Adv Nurs. 2004; 47:192–200.
- Kothari R, Sauerbeck L, Jauch E, Broderick J, Brott T, Khoury J, Liu T. Patients' awareness of stroke signs, symptoms, and risk factors. *Stroke*. 1997;28:1871–1875.
- Pancioli AM, Broderick J, Kothari R, Brott T, Tuchfarber A, Miller R, Khoury J, Jauch E. Public perception of stroke warning signs and knowledge of potential risk factors. *JAMA*. 1998;279:1288–1292.
- 302. Schneider AT, Pancioli AM, Khoury JC, Rademacher E, Tuchfarber A, Miller R, Woo D, Kissela B, Broderick JP. Trends in community knowledge of the warning signs and risk factors for stroke. *JAMA*. 2003;289:343–346.
- Nicol MB, Thrift AG. Knowledge of risk factors and warning signs of stroke. Vasc Health Risk Manag. 2005;1:137–147.
- Zerwic J, Hwang SY, Tucco L. Interpretation of symptoms and delay in seeking treatment by patients who have had a stroke: exploratory study. *Heart Lung*. 2007;36:25–34.
- 305. DeLemos CD, Atkinson RP, Croopnick SL, Wentworth DA, Akins PT. How effective are "community" stroke screening programs at improving stroke knowledge and prevention practices? Results of a 3-month follow-up study. *Stroke*. 2003;34:e247–e249.
- Willoughby DF, Sanders L, Privette A. The impact of a stroke screening program. *Public Health Nurs*. 2001;18:418–423.
- Becker K, Fruin M, Gooding T, Tirschwell D, Love P, Mankowski T. Community-based education improves stroke knowledge. *Cerebrovasc Dis.* 2001;11:34–43.
- Ferris A, Robertson RM, Fabunmi R, Mosca L. American Heart Association and American Stroke Association national survey of stroke risk awareness among women. *Circulation*. 2005;111:1321–1326.
- LaBresh KA, Ellrodt AG, Gliklich R, Liljestrand J, Peto R. Get with the guidelines for cardiovascular secondary prevention: pilot results. *Arch Intern Med.* 2004;164:203–209.
- Schwamm L, Reeves MJ, Frankel M. Designing a sustainable national registry for stroke quality improvement. *Am J Prev Med.* 2006;31(suppl 2):S251–S257.

KEY WORDS: AHA Scientific Statements ■ stroke, ischemic ■ stroke management ■ emergency service, hospital ■ tissue plasminogen activator ■ nursing care