Guidelines for the management of patients with acute ischemic stroke. A statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association

HP Adams, Jr, TG Brott, RM Crowell, AJ Furlan, CR Gomez, J Grotta, CM Helgason, JR Marler, RF Woolson and JA Zivin

Circulation 1994;90;1588-1601

Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 1994 American Heart Association. All rights reserved. Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://circ.ahajournals.org
Guidelines for the Management of Patients With Acute Ischemic Stroke

A Statement for Healthcare Professionals
From a Special Writing Group of the Stroke Council,
American Heart Association

Harold P. Adams, Jr, MD, Chair; Thomas G. Brott, MD; Robert M. Crowell, MD;
Anthony J. Furlan, MD; Camilo R. Gomez, MD; James Grotta, MD; Cathy M. Helgason, MD;
John R. Marler, MD; Robert F. Woolson, PhD; Justin A. Zivin, MD, Members;
William Feinberg, MD, Marc Mayberg, MD, Ex Officio Members

In 1991, about 500,000 Americans had a stroke (400,000 had an ischemic stroke) and more than
143,000 died.1 More than 3,000,000 people in the
United States have survived a stroke. In 1994 the annual
economic costs of stroke due to health care expenses
and lost productivity are estimated to be nearly $20
billion.1 In spite of these human and financial costs,
stroke unfortunately has not received a great deal of
attention, and its management has been marred by an
element of nihilism.2 Caplan3 concludes that past fail-
ures to establish effective therapies for stroke are due to
problems in clinical trial design, lack of interest in care
of stroke, and lack of available technologies to evaluate
patients. However, with advances in diagnosis and treat-
ment, stroke can now be managed as the life-threatening
emergency that it is. In 1993 the American Heart
Association included emergent stroke care as part of its
special resuscitation situations for basic and advanced
life support.4 This report builds on that statement.

The goal of this special report is to provide informa-
tion about the current management of acute ischemic
stroke. It also provides recommendations for initial care
(within 24 hours of stroke) based on currently available
data from clinical trials. In the future, many therapies
for stroke will be linked to very early (within 6 hours)
intervention. No recommendations about rehabilitation
or chronic medical or surgical measures to prevent
recurrent stroke are made.

To prepare this report, the members of the Stroke
Council used the rules of evidence for specific treatments
that have been used by other panels (Table 1).5 These
rules give greater credence to the results of well-designed
clinical trials than to anecdotal case reports or case series.
The current recommendations will eventually be altered
by the results of several ongoing clinical trials. Although
the rules of evidence cannot be applied to the indications
for diagnostic procedures, the Stroke Council recom-
mends tests that can be ordered on an emergency basis.
For such instances, the council has prepared a consensus
statement that begins with the phrase “there is general
agreement.” The findings of new diagnostic studies may
further expedite the early diagnosis and management of
ischemic stroke. The Stroke Council has also prepared
guidelines for the management of transient ischemic at-
tack6 and subarachnoid hemorrhage7 using the same rules
of evidence.

The target audiences for this statement are primary
care physicians, emergency room physicians, and neu-
rologists who care for patients during the first few hours
after stroke. This report also includes recommendations
for the education of the general public and for inter-
ventions that may be started by emergency medical
services (EMS) personnel.

Early Recognition of Acute Ischemic Stroke

A rapid response to stroke requires the involvement
of the entire community. Recognition of stroke occurs
at three levels: (1) the public, (2) nonphysician EMS
personnel, and (3) physicians.

Public Response

Successful care begins when the signs of stroke are
first recognized by the patient or bystanders. This
requires wide public awareness of the clinical features
of stroke. Public education should include information
directed at the entire community and more detailed
instruction for those who are most interested or at
increased risk. The basic message should be simple:
 immediatelcall the EMS system (911 or other appli-
cable number) if a stroke is suspected. Do not pause to
decline if symptoms will spontaneously resolve or to
contact relatives or a personal physician for advice.
Even if symptoms do resolve, effective medical or
surgical therapies may prevent future stroke in such
cases.8-12

“Guidelines for the Management of Patients With Acute Isch-
emic Stroke” was approved by the Science Advisory Commi-
tee of the American Heart Association on February 16, 1994.

Requests for reprints should be sent to the Office of Scientific
Affairs, American Heart Association, 7272 Greenville Ave, Dallas,
TX 75231-4596.

© 1994 American Heart Association, Inc.
TABLE 1. Levels of Evidence and Grading of Recommendations for Treatment of Patients With Acute Ischemic Stroke

<table>
<thead>
<tr>
<th>Level of Evidence</th>
<th>Data from randomized trials with low false-positive (alpha) and low false-negative (beta) errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level I</td>
<td>Data from randomized trials with high false-positive (alpha) or high false-negative (beta) errors</td>
</tr>
<tr>
<td>Level II</td>
<td>Data from nonrandomized concurrent cohort studies</td>
</tr>
<tr>
<td>Level III</td>
<td>Data from nonrandomized cohort studies using historical controls</td>
</tr>
<tr>
<td>Level IV</td>
<td>Data from anecdotal case series</td>
</tr>
</tbody>
</table>

Strength of Recommendation

- Grade A: Supported by Level I evidence
- Grade B: Supported by Level II evidence
- Grade C: Supported by Levels III, IV, or V evidence

Descriptions of evidence and recommendations are adapted from Cook et al.6

Nonphysician EMS Personnel

EMS personnel should be instructed in the rapid recognition, evaluation, treatment, and transport of patients with stroke (Table 2). Most strokes can be readily recognized. The immediate diagnostic goal is not to differentiate subtle, unusual, or isolated neurological signs, and every minute may be important; thus, EMS personnel should be able to perform a baseline assessment within a few minutes.4 Some portions of the evaluation and initial management can be completed while the patient is being transported to the hospital. Notification of the hospital can save valuable time because the physicians, nurses, and technicians who will initiate emergent care and obtain the required studies can be assembled to meet the patient on arrival. In the future, specific therapies to limit the effects of stroke may be administered before a patient arrives in the hospital.

Physicians

Stroke should be suspected whenever a patient has the characteristic sudden onset of focal neurological signs, such as hemiparesis, aphasia, or hemianopia, or altered consciousness (Tables 3 and 4). Symptoms of stroke can develop in isolation, but they usually occur in combination. The early course can be one of worsening (stepwise or stuttering), gradual deterioration or the immediate appearance of severe deficits, or fluctuations.13-16 In the differential diagnosis of ischemic stroke, the most important alternative is intracranial hemorrhage. If a patient is comatose and no history is available, other diagnoses include hypoglycemia, drug overdose, seizures, or craniocerebral trauma.

The initial evaluation should assess the patient’s airway, ventilation, and circulation. The neurological patterns of neurological abnormalities in patients with acute ischemic stroke are detailed in Table 4.

TABLE 3. Presentations of Acute Ischemic Stroke

| Alteration in consciousness |
| Stupor or coma |
| Confusion or agitation |
| Seizures |
| Aphasia or other higher cognitive disturbances |
| Dysarthria |
| Facial weakness or asymmetry (ipsilateral or contralateral to limb weakness) |
| Incoordination, weakness, paralysis, or sensory loss of one or more limbs (usually one half of the body) |
| Ataxia, poor balance, or clumsiness or difficulty walking |
| Visual loss |
| Monocular or binocular |
| May be partial loss of the field |
| Vertigo, double vision, unilateral hearing loss, nausea, vomiting, headache, photophobia, or phonophobia |

Adapted from Cook et al.5

TABLE 4. Common Patterns of Neurological Abnormalities in Patients With Acute Ischemic Stroke

<table>
<thead>
<tr>
<th>Left (Dominant) Hemisphere</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia, right hemiparesis, right-sided sensory loss, right visual field defect, poor right conjugate gaze, dysarthria, difficulty in reading, writing, or calculating</td>
</tr>
<tr>
<td>Right (Non-dominant) Hemisphere</td>
</tr>
<tr>
<td>Neglect of the left visual space, left visual field defect, left hemiparesis, left-sided sensory loss, poor left conjugate gaze, extinction of left-sided stimuli, dysarthria, spatial disorientation</td>
</tr>
</tbody>
</table>

Brain Stem/Cerebellum/Posterior Hemisphere

Motor or sensory loss in all four limbs, crossed signs, limb or gait ataxia, dysarthria, dysconjugate gaze, nystagmus, amnesia, bilateral visual field defects

Small Subcortical Hemisphere or Brain Stem (Pure Motor Stroke)

Weakness of face and limbs on one side of the body without abnormalities of higher brain function, sensation, or vision

Small Subcortical Hemisphere or Brain Stem (Pure Sensory Stroke)

Decreases sensation of face and limbs on one side of the body without abnormalities of higher brain function, motor function, or vision

EMS indicates emergency medical services.

Assure adequate airway
Monitor vital signs
Conduct general assessment
Evidence of trauma to head or neck
Cardiovascular abnormalities
Conduct neurological examination
Level of consciousness
Presence of seizure activity
Glasgow Coma Scale
Pupils: size, equality, reactivity
Individual limb movements

EMS indicates emergency medical services.
examination need not be exhaustive and can be completed in 5 to 10 minutes.\textsuperscript{17,18} Evidence of a head or neck injury, cardiac murmur, arterial bruit, or absent cranial artery pulses should be sought. Detection of ocular hemorrhages or nuchal rigidity predicts intracranial bleeding.\textsuperscript{19} Passive flexion of the neck should be performed only if there is no suggestion of coincidental cervical spine trauma. Coma, papilledema, or unexplained fever should alert the physician to other diagnoses such as subarachnoid hemorrhage, tumor, infection, or metabolic abnormalities.

Most patients do not have depression in consciousness within 24 hours of onset of ischemic stroke in the cerebral hemispheres; if consciousness is impaired, a stroke-related seizure, hemorrhage, hypoxia, increased intracranial pressure (ICP), or brain stem involvement should be suspected. Because aphasia, memory disturbances, visual field defects, or neglect of the left side of the environment are important localizing signs, they should be checked in alert patients. The pattern of motor and sensory deficits can also provide clues to the site of the stroke. While testing gait is often not possible, gait abnormalities are often prominent with cerebellar infarctions. Abnormalities in ocular rotation or involuntary ocular movements (nystagmus) are frequently seen with brain stem or cerebellar stroke. A conjugate lateral deviation of the eyes is found with either large hemispheric or brain stem strokes. The patterns of neurological signs in common stroke syndromes are summarized in Table 4.\textsuperscript{3}

**Admission to the Hospital**

Because of the nature of the neurological problems and the propensity for complications, most patients with acute ischemic stroke should be admitted to a hospital. A recent meta-analysis demonstrates that outcome can be improved if a patient is admitted to a facility that specializes in the care of stroke\textsuperscript{20} (Level of Evidence 1). Because most community hospitals do not have specialized stroke care units, physicians should consider transferring patients to institutions that have such facilities.

**Recommendations**

1. There is general agreement that separate educational programs tailored to the public, EMS personnel, and physicians should be developed. There is general agreement that an observer should dial 911 if a person has symptoms of acute stroke.

2. There is general agreement that EMS personnel should be trained in the early recognition and emergent care of stroke. There is general agreement that EMS transport of patients with acute stroke to a medical center should be expedited and should be the highest priority.

3. Early admission of most patients to a unit that has a specialized interest in the treatment of stroke is strongly recommended (Level of Evidence I, Grade A Recommendation). A team of physicians, nurses, and technicians that is devoted to the early care of patients with stroke should be assembled. Rapid transfer of a patient to a hospital that has a specialized stroke care unit is strongly recommended.

| Table 5. Tests for the Emergent Evaluation of the Patient With Acute Ischemic Stroke* |
|---------------------------------|---------------------------------|
| CT of the brain without contrast | Electrocardiogram               |
| Chest x-ray                     | Partial thromboplastin time     |
| Lateral cervical spine x-ray    | Lumbar puncture (if subarachnoid hemorrhage is suspected and CT is negative) |
| Arterial blood gas levels       | Electroencephalogram (if seizures are suspected) |
| Renal and hepatic chemical analyses | CT indicates computed tomography. |
| Electroencephalogram (if seizures are suspected) |                               |

*Studies that are available in most emergency rooms on a 24-hour per day basis. The results of these tests, which help both to confirm the stroke and to detect early, potentially life-threatening complications, may influence emergent care.

**Emergent Evaluation**

The aims of emergent evaluation are to (1) confirm the cause is stroke and not another brain lesion, (2) provide information about the possible reversibility of the pathology, (3) give clues about the most likely etiology, (4) predict the likelihood of immediate complications, and (5) begin appropriate treatment.\textsuperscript{4,21} Table 5 lists the diagnostic studies available at most medical centers in the United States that should be ordered on an emergent basis. Because an extensive evaluation to determine the most likely cause of stroke is time-consuming, these tests can be initiated after treatment is started.

Although the clinical features of intracranial hemorrhage and ischemic stroke overlap, their management is markedly different; thus, differentiation between these types of strokes is paramount.\textsuperscript{22} The clinical examination and analysis of the cerebrospinal fluid (CSF) do not reliably distinguish between hemorrhagic and ischemic stroke. Computed tomography (CT) of the brain, however, effectively discriminates between hemorrhagic and ischemic stroke and is the most important initial diagnostic study.\textsuperscript{18,19} CT also helps discriminate nonvascular lesions, such as brain tumors, that can produce focal neurological signs. This noninvasive test is readily available in most centers and can be quickly performed. Infusion of intravenous contrast is not necessary. While it can miss subtle subarachnoid hemorrhage, the yield of early CT in detecting intracerebral hemorrhage approaches 100%. Absence of blood on CT is taken as important information supporting the diagnosis of ischemic stroke. Early detection of edema, hydrocephalus, or hypodensity is possible in approximately 50% to 60% of cases; when present, these findings may indicate a
more serious ischemic injury. They are more often associated with poor outcome, may predict hemorrhagic transformation, or may dictate a change in acute treatment. The pattern of stroke or a dense artery sign (a clot in an intracranial artery) may provide clues to the etiology of stroke. CT can miss small subcortical or cortical infarctions or lesions in the posterior fossa. A study performed within the first few hours after stroke may not visualize an ischemic lesion, including potentially very large infarctions. Magnetic resonance imaging (MRI) is more sensitive than CT, but the MRI features of acute hemorrhage can be nonspecific. The use of MRI in the setting of acute stroke may be limited by the patient's refusal or claustrophobia or the presence of objects such as a cardiac pacemaker. Nonetheless, MRI may be particularly helpful in cases with suspected brain stem or cerebellar infarction, which are difficult to detect with CT. Magnetic resonance angiography may provide important clues about the presence of an occluded intracranial artery.

A strong correlation exists between acute ischemic stroke and the presence of heart disease. Myocardial infarction, arrhythmias, congestive heart failure, and sudden death are important possibilities to consider in caring for patients with acute ischemic stroke. Acute ischemic stroke can occur simultaneously with an acute myocardial infarction. Cardiac arrhythmias, including atrial fibrillation, can accompany stroke. For these reasons, a 12-lead electrocardiogram, chest x-ray, and clinical cardiac examination should be performed. Cardiac monitoring can be continued during the first 24 hours to detect potentially life-threatening arrhythmias. If baseline studies detect serious cardiac abnormalities, the use of more specific cardiac diagnostic studies or prolonged monitoring may be needed.

A series of blood tests should be completed to identify conditions that may have contributed to the acute stroke or that may influence emergent management (Table 5). Emergency CSF examination should be performed only in those patients whose symptoms suggest an acute infectious process or subarachnoid hemorrhage but in whom hemorrhage is not seen on CT.

If seizures are suspected or if nonconvulsive status epilepticus is the likely cause of the patient's neurological condition, an electroencephalogram can be an important early diagnostic test. Tests to detect risk factors or causes for stroke may also be ordered. Unfortunately, some of these tests are time-consuming or may not be available on an emergency basis. Cerebral arteriography, transcranial Doppler, and duplex examinations of the cervical arteries can detect severe arterial diseases, including atherosclerosis and dissection. Early ultrasound studies are used in some centers to screen for intracranial and extracranial disease. At present, the effects of the results of these studies on early management are uncertain, but in the future indications for therapies such as thrombolytic drugs might be linked to these studies. Transthoracic and transesophageal echocardiography are important ancillary tests to screen for cardiogenic causes of embolism. Special hematologic and serological studies for unusual causes of stroke such as vasculitis and the antiphospholipid antibody syndrome may also be indicated. Several advanced research technologies (diffusion or perfusion MRI, MRI spectroscopy, positron emission tomography, and single-photon emission tomography) exist. These technologies may help distinguish between dead brain tissue and dysfunctional but salvageable brain tissue. The usefulness of these techniques in helping management of acute stroke has not been established.

**Recommendations**

1. There is general agreement to strongly recommend that emergent CT be the initial brain imaging study.
2. There is general agreement to strongly recommend that electrocardiography, chest x-ray, and cardiac monitoring be components of the emergent evaluation of patients with suspected ischemic stroke.
3. There is general agreement to recommend the other diagnostic studies listed in Table 5 on an emergency basis.
4. Diagnostic studies aimed at establishing a likely etiology of acute ischemic stroke, including ultrasound or other imaging of intracranial or extracranial vessels, can, in some circumstances, be helpful in making decisions about treatment. However, there is general agreement that these tests should not delay treatment, and their use should be tailored to specific clinical situations.

**Emergent Supportive Care and Treatment of Acute Complications**

Maintaining adequate tissue oxygenation is an important component of emergent management (Table 6). Hypoxia results in anaerobic metabolism and depletion of energy stores that can increase the extent of brain injury and worsen outcome. The most common causes of hypoxia are partial airway obstruction, hyperventilation, aspiration pneumonia, and atelectasis. Protecting the airway and ventilatory assistance are critical components of resuscitation of seriously ill patients who

---

**TABLE 6. Most Common Complications of Acute Ischemic Stroke**

<table>
<thead>
<tr>
<th>Neurological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral edema</td>
</tr>
<tr>
<td>Hydrocephalus</td>
</tr>
<tr>
<td>Increased intracranial pressure</td>
</tr>
<tr>
<td>Hemorrhagic transformation</td>
</tr>
<tr>
<td>Seizures</td>
</tr>
<tr>
<td>Medical</td>
</tr>
<tr>
<td>Aspiration</td>
</tr>
<tr>
<td>Hypoventilation</td>
</tr>
<tr>
<td>Pneumonia</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Cardiac arrhythmias</td>
</tr>
<tr>
<td>Deep vein thrombosis</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Urinary tract infections</td>
</tr>
<tr>
<td>Decubitus ulcers</td>
</tr>
<tr>
<td>Malnutrition</td>
</tr>
<tr>
<td>Contractures</td>
</tr>
<tr>
<td>Stiff joints</td>
</tr>
</tbody>
</table>
have impaired consciousness. In such instances, an endotracheal tube should be placed if the airway is threatened. No data establish the benefit of supplemental oxygen, and there is no reason to routinely administer this therapy. However, if there is evidence of hypoxia by blood gas determination, desaturation as detected by pulse oximetry, or if there are other reasons for supplemental oxygen, it should be administered. Hyperbaric oxygen therapy may be useful for the occasional patient with stroke secondary to air embolism or Caisson's disease; its role in the general treatment of acute ischemic stroke has not been adequately tested.

While arterial hypertension is commonly detected after stroke, its optimal management has not been established. An elevated blood pressure can result from the stress of the stroke, a full bladder, pain, underlying hypertension, a physiological response to brain hypoxia, or increased intracranial pressure. Blood pressure can fall when the patient is moved to a quiet room, the bladder is emptied, pain is controlled, or the patient is allowed to rest. Blood pressure can also decrease if intracranial pressure is controlled. In most patients, blood pressure should not be lowered. In cases of severe hypertension, lowering should be done cautiously, because neurological status can be further impaired after administration of antihypertensive drugs, and the responses to medications can be exaggerated (Levels of Evidence III through V).

There are no data that define the levels of elevated blood pressure that mandate emergent treatment following stroke. Early use of parenteral drugs may be warranted if high blood pressure accompanies hemorrhagic transformation, myocardial infarction, renal failure secondary to accelerated hypertension, or dissection of the thoracic aorta. In general, antihypertensive drugs should be withheld unless the calculated mean blood pressure (the sum of the systolic pressure plus double the diastolic pressure, divided by three) is greater than 130 mm Hg or the systolic blood pressure is greater than 220 mm Hg (Levels of Evidence III through V). In such instances, the best parenteral drugs appear to be those that are easily titrated and that have a minimal effect on cerebral blood vessels, such as labetalol or enalapril. Most patients can be treated with oral agents such as captopril or nicardipine. Sublingual use of a calcium antagonist should be avoided because of rapid absorption and secondary precipitous decline in blood pressure.

Low blood pressure is rarely detected in patients with acute ischemic stroke. If hypotension is noted, the cause should be sought; volume depletion is the most common cause. Correction of hypovolemia and optimization of cardiac output are important priorities during the first few hours after stroke. Elevating blood pressure by the use of a vasopressor or intravenous fluids may be helpful. Hypervolemic hemodilution and drug-induced hypertension have been successfully used in patients with ischemia secondary to vasospasm after subarachnoid hemorrhage (Levels of Evidence III through V). This treatment requires the use of colloid solutions (eg, 5% albumin), and in many cases a pulmonary artery catheter or central venous pressure monitor is needed to assess cardiovascular responses.

Although there are no clinical data about the usefulness of hypothermia or antipyretics in treatment of stroke, experimental studies report that lowering body temperature reduces infarction size. The source of any fever following stroke should be ascertained and the fever treated with antipyretic agents. Whether hyperglycemia makes stroke worse is uncertain. Some studies have correlated poor outcome after stroke with an elevated blood glucose, but no data exist about the efficacy of treating an elevated blood glucose in improving the outcome of a stroke patient. Hyperglycemia can be a response to a serious brain injury. Until there are more data to guide treatment, management of an elevated blood glucose in stroke patients should be similar to that of other persons with an elevated blood glucose. Conversely, hypoglycemia can produce focal symptoms that mimic stroke; in such a situation, early administration of glucose may reverse these symptoms.

Recommendations

1. There is general agreement to recommend airway support and ventilatory assistance in the care of patients with acute stroke who have depressed levels of consciousness (Levels of Evidence III through V, Grade C).
2. There is general agreement to recommend supplemental oxygen to hypoxic patients (Levels of Evidence III through V, Grade C). There are insufficient data about the efficacy of hyperbaric oxygen to recommend this therapy for most patients with stroke.
3. There is general agreement that most patients with acute ischemic stroke do not need treatment with parenteral antihypertensive drugs (Level of Evidence II, Grade B). Oral antihypertensive drugs are preferred. Caution use of antihypertensive agents is recommended for patients with markedly elevated blood pressure (>130 mm Hg mean blood pressure or >220 mm Hg systolic blood pressure) (Levels of Evidence III through V, Grade C).
4. There is general agreement to recommend treatment of the sources of fever and use of antipyretics to control an elevated body temperature (Levels of Evidence III through V, Grade C). There are insufficient clinical data about the use of hypothermia to recommend this therapy.
5. There is general agreement to recommend control of hypoglycemia or hyperglycemia after stroke (Levels of Evidence III through V, Grade C).

Treatment of Acute Neurological Complications

The most important acute neurological complications of stroke are (1) cerebral edema and increased intracranial pressure, which can lead to herniation or brain stem compression, (2) seizures, and (3) hemorrhagic conversion with or without formation of a hematoma (Table 6).

Death during the first week after stroke is commonly caused by brain edema and an elevated intracranial pressure, which are largely complications of occlusions of major intracranial arteries and large multilobar infarctions. Only 10% to 20% of patients develop edema that produces clinical deterioration and warrants medical intervention. Brain edema peaks at 3 to 5 days after stroke and is usually not a problem within 24 hours of the ictus except among persons with large cerebellar infarctions. Increased intracranial pressure can also result from acute hydrocephalus secondary to obstruction of CSF pathways by large cerebellar infarc-
tions. The goals of management of brain edema are to (1) reduce intracranial pressure, (2) maintain adequate cerebral perfusion to avoid worsening of brain ischemia, and (3) prevent brain herniation. Initial care includes mild restriction of fluids48 (Levels of Evidence III through V). Hypo-osmolar fluids, such as 5% dextrose in water, may worsen edema. Factors that might exacerbate intracranial pressure (eg, hypoxia, hypercarbia, and hyperthermia) are treated, and the head of the bed can be elevated by 20 to 30 degrees. An elevation of blood pressure may be a compensatory response to maintain cerebral perfusion pressure in a patient with a markedly elevated intracranial pressure. In such instances antihypertensive agents, particularly those that induce cerebral vasodilation, are avoided46-50 (Levels of Evidence III through V).

Patients whose condition is deteriorating can be treated with hyperventilation, osmotic diuretics, drainage of CSF, or surgery, although there are no trials that address the efficacy of such aggressive management after stroke. Furthermore, the value of intracranial pressure monitoring in this population has not been established, even though it can guide the choice of therapies and may help predict the patient’s outcome.48 Hyperventilation is an emergency measure that acts almost immediately; a reduction of the pCO2 by 5 to 10 mm Hg lowers intracranial pressure by 25% to 30%51,52 (Levels of Evidence III through V). Hyperventilation should be followed by another intervention to control brain edema and intracranial pressure. Maintaining adequate brain perfusion is also necessary since hyperventilation can cause vasoconstriction that might aggravate ischemia. Conventional and large doses of corticosteroids have been tested in clinical trials, but no improvement of outcome after stroke was noted53-56 (Level of Evidence I), and infections were more common among patients treated with steroids. These drugs are not indicated in the emergent management of cerebral edema complicating ischemic stroke.

Although furosemide or mannitol is often prescribed after stroke, no trials of these drugs support their use in controlling cerebral edema4 (Levels of Evidence III through V). An intravenous bolus of 40 mg furosemide has been used as an adjunct in the care of patients whose condition is rapidly deteriorating, but it is not used in long-term care. Mannitol (0.25 to 0.5 g/kg IV) given over 20 minutes rapidly lowers intracranial pressure and can be given every 6 hours.57 The usual maximum daily dose is 2 g/kg.57 Glycerol has been examined in clinical trials and can lower mortality among patients with large strokes58-60 (Level of Evidence II). However, it has not been widely used because glycerol is very sweet and is not well tolerated by patients. Intravenous glycerol can induce hemolysis. Barbiturates can be used to reduce intracranial pressure, but their role in treatment after ischemic stroke has not yet been clearly determined61 (Level of Evidence II).

If hydrocephalus is present, drainage of CSF via an intraventricular catheter can rapidly lower intracranial pressure. Hemicraniectomy and temporal lobe decompression have been used to control intracranial pressure and prevent herniation in those with very large hemispheric infarctions62,63 (Levels of Evidence III through V). Further information about the quality of life among persons who survive these aggressive therapies for multiobar infarctions is needed. Ventriculostomy and suboccipital craniectomy, especially in concert with aggressive medical therapies, appear effective in relieving hydrocephalus and brain stem compression caused by large cerebellar infarctions64,65 (Levels of Evidence III through V). In an ongoing randomized trial, a combination of both therapies is being compared with each therapy alone.66

The frequency of seizures during the acute period after stroke is reported to be 4% to 43%; the wide variation reflects differences in study designs.67-81 Seizures are most likely to occur within 24 hours of stroke and are usually partial, with or without secondary generalization.81 Recurrent seizures occur in approximately 20% to 80% of cases. Intermittent seizures seem not to alter the overall prognosis of stroke.79 However, status epilepticus can be a life-threatening complication; fortunately, it is uncommon. There are no data about the value of prophylactic administration of anticonvulsants after stroke. Until such data become available, stroke patients who are seizure-free should not receive anticonvulsant drugs. There are few data concerning the efficacy of anticonvulsants in the treatment of stroke patients who have experienced seizures; thus, recommendations are based on the established management of seizures that may complicate any acute neurological illness (Level of Evidence I). Care depends on the type and frequency of seizures, the route available for administration of medications, and any contraindications for the use of specific drugs. Almost 90% of seizures can be controlled by a single medication. Partial seizures seem to respond best to carbamazepine or phenytoin.82 Either lorazepam (1 to 2 mg IV) or diazepam (5 to 10 mg IV) can be used to treat status epilepticus. Phenytoin (usual loading dose, 20 mg/kg IV) can induce cardiac arrhythmias or hypotension and should not be administered more rapidly than 50 mg/min. Both blood pressure and heart rate should be monitored.

There is relatively little information about the natural rate of early hemorrhagic transformation of ischemic stroke. Some studies suggest that almost all infarctions have some element of petechial bleeding.83,84 Using CT, one prospective study estimates that approximately 5% of infarctions will spontaneously develop symptomatic hemorrhagic transformation or frank hematomas.85 The location, size, and etiology of stroke may influence the development of this complication. Further information about the influence of hemorrhagic transformation on outcome after ischemic stroke is needed. Small asymptomatic petechiae are much less important than hematomas, which are associated with neurological decline. Anticoagulants and thrombolytic drugs may increase the likelihood of serious hemorrhagic transformation, although there are no solid data about the risks of these therapies. Management of patients with hemorrhagic infarction depends on the amount of bleeding and its symptoms.

Recommendations

1. Corticosteroids are not recommended for the management of cerebral edema and increased intracranial pressure after stroke (Level of Evidence I, Grade A).
2. Osmotherapy and hyperventilation are recommended for patients whose condition is deteriorating secondary to increased intracranial pressure, including those with herniation syndromes (Level of Evidence II, Grade B). There is general agreement that surgical interventions, including continuous drainage of CSF, can be used to treat increased intracranial pressure secondary to hydrocephalus (Levels of Evidence III through V, Grade C).

3. There is general agreement to recommend surgical decompression and evacuation of large cerebral infarctions that compress the brain stem (Levels of Evidence III through V, Grade C). Surgical decompression and evacuation of a large hemispheric infarction can be a life-saving measure, but survivors may have severe residual neurological deficits (Levels of Evidence III through V, Grade C).

4. Administration of anticonvulsants to prevent recurrent seizures is strongly recommended (Level of Evidence I, Grade A). Prophylactic administration of anticonvulsants to patients with recent stroke who have not had seizures is not recommended.

**General Early Supportive Care**

The goals of early supportive care after admission to the hospital are to (1) observe changes in the patient’s condition that might prompt different medical or surgical interventions, (2) facilitate medical and surgical measures aimed at improving outcome after stroke, (3) institute measures to prevent subacute complications, (4) begin planning for chronic therapies to prevent recurrent stroke, and (5) begin efforts to restore neurological function through rehabilitation or other techniques. Many complications of stroke can be prevented by medical interventions and good supportive care. Approximately 25% of patients worsen during the first 24 to 48 hours after admission to the hospital, and it is often difficult to predict deterioration. Therefore, all patients should be considered at risk for neurological worsening.

Admission to a unit that is dedicated to the care of stroke patients helps to reduce mortality and morbidity (Level of Evidence I). There are no strict definitions of what constitutes such a unit, but at a minimum it should be staffed to allow the close monitoring of vital and neurological signs. Nursing and other personnel working in the unit should be trained to carefully observe patients so that fluctuations in neurological status can be promptly recognized. Regular communication among physicians, nurses, and rehabilitation personnel will improve coordination of care and may result in better outcome.

The patient’s vital signs and neurological status should be frequently assessed during the first 24 hours. Most patients are first treated with bed rest. An occasional patient may have worsening of neurological signs upon standing, sitting, or elevating the head. Observation of the patient’s neurological condition and blood pressure should continue as mobilization begins. Early mobilization is favored because it lessens the likelihood of major complications such as pneumonia, deep vein thrombosis, pulmonary embolism, and decubitus ulcers. Immobility can also lead to contractures, orthopedic complications, and pressure palsy. Passive full-range-of-motion exercises for paralyzed limbs can be started during the first 24 hours. Frequent turning, the use of alternating pressure mattresses, and close surveillance of the skin help prevent decubitus ulcers.

Sustaining nutrition is important because the malnutrition that can develop after stroke may interfere with recovery. Many patients initially should not receive any food or fluids by mouth, and intravenous fluids are needed. Assessment of the ability to swallow is imperative because of the high risk of aspiration. Patients with infarctions in the brain stem, multiple strokes, large hemispheric lesions, or depressed consciousness are at greatest risk for aspiration. An abnormal gag reflex, impaired voluntary cough, dysphonia, or cranial nerve palsies should alert the physician to this risk.

A preserved gag reflex may not indicate safety from aspiration. A water swallow test performed at the bedside is a useful screening test, and a videofluoroscopic modified barium swallow examination can be performed later. When necessary, a nasogastric or nasoduodenal tube can be inserted to provide feedings and to expedite administration of medications. Intravenous hyperalimentation is rarely indicated.

Pneumonia is an important cause of death after stroke. It most often occurs in patients who are immobile or who are unable to cough. Development of fever after stroke should prompt a search for pneumonia, and appropriate antibiotic therapy should be administered early. Pulmonary embolism accounts for approximately 10% of deaths after stroke. Paralysis of the leg and immobility increase the risk of deep vein thrombosis. Proximal deep vein thrombosis can be detected by plethysmography in approximately one third of patients who have moderately severe strokes. Subcutaneous administration of heparin or low-molecular-weight heparins or heparinoids is effective in preventing deep vein thrombosis (Level of Evidence I). Aspirin may also be effective in patients who have contraindications for anticoagulants. Alternating pressure stockings are effective in preventing deep vein thrombosis in patients who have a contraindication to treatment with antithrombotic drugs (Level of Evidence II). Support stockings are of unproven value.

Urinary tract infections are common, and secondary sepsis can develop in approximately 5% of patients. An indwelling catheter is sometimes needed to treat incontinence or urinary retention, but, if possible, it should be avoided because of the risk of infection. Acidification of the urine or intermittent catheterization can lessen the risk of infection and avoid the need for prophylactic antibiotics. Anticholinergic drugs may help in recovery of bladder function.

After stabilization of the patient’s condition, rehabilitation, measures to prevent long-term complications, chronic therapies to lessen the likelihood of recurrent stroke, family support, and treatment of depression can be started when appropriate.

**Recommendations**

1. Early mobilization and measures to prevent the subacute complications of stroke (aspiration, malnutrition, pneumonia, deep vein thrombosis, pulmonary embolism, decubitus ulcers, contractures, and joint abnormalities) are strongly recommended (Levels of Evidence I and II, Grades A and B).
2. Prophylactic administration of heparin or low-molecular-weight heparins or heparinoids to prevent deep vein thrombosis is strongly recommended for immobilized patients (Level of Evidence I, Grade A). Intermittent external compression stockings are recommended for patients who cannot receive antithrombotic drugs (Level of Evidence II, Grade B).

3. Antibiotics to treat infectious complications of stroke are strongly recommended (Level of Evidence I, Grade A). Concurrent medical conditions should be treated.

Acute Treatment With Antithrombotic or Antiplatelet-Aggregating Drugs

In a 1989 survey of neurologists randomly selected from across the United States, 82% of responders stated that heparin might be indicated for prevention of recurrent embolism, and 70% thought it might be indicated for care of progressing stroke.105 However, only 6% of the neurologists believed that heparin was of proven usefulness, and approximately 16% thought heparin had been proven to be ineffective. Five studies of heparin were performed before the advent of CT.106-112 Two randomized trials did not recruit sufficient numbers of patients for conclusive results108,109 (Level of Evidence II). The other three compared treated patients with nonrandomized concurrent control subjects110-112 (Level of Evidence III). While these studies hinted at a benefit from heparin, all had methodological flaws. Still, their results led to the widespread use of heparin. More recently, the value of heparin in acute ischemic stroke has been challenged by at least six clinical studies and a meta-analysis.113-119 In a small randomized trial that began treatment of most patients more than 24 hours after stroke, Duke et al119 found no benefit from treatment with heparin for patients with partial, stable stroke (Level of Evidence II).

Heparin has been safely administered to selected patients with nonseptic, nonhemorrhagic acute cardioembolic stroke, and it was associated with a low rate of recurrent events120,121 (Level of Evidence V). The Cerebral Embolism Study Group, which performed a small randomized trial in 45 patients with presumed embolic stroke, noted no bleeding complications related to heparin and a trend toward reduction of early recurrent embolism122 (Level of Evidence II). Another study linked the risk of hemorrhage to the size of infarction and estimated a 12% risk of recurrent embolism within 2 weeks among untreated patients123 (Level of Evidence V). Other studies have concluded that the risk of early recurrent embolism after cardioembolic stroke is very low and that heparin may not be necessary124,125 (Level of Evidence V).

There are disagreements about the desired level of anticoagulation, duration of treatment, and the use of a loading (bolus) dose. There are also disagreements about the severity of neurological deficits and the size of infarction on CT that contraindicate the use of heparin. No studies have analyzed the effect of the vascular distribution of the ischemic symptoms (carotid versus vertebrobasilar) and the underlying location, nature, and extent of the vascular lesions (eg, severity of stenosis, arterial dissection) on the responses to treatment with heparin. While some authorities prefer a continuous intravenous infusion of heparin, there are no data to confirm the superiority of this regimen over a subcutaneous route. One small trial of subcutaneous heparin demonstrated no efficacy119 (Level of Evidence II). The International Stroke Trial is currently testing two doses of subcutaneous heparin.

Physicians have valid concerns about the safety of heparin in patients with acute ischemic stroke.105 Besides increasing the risks of symptomatic hemorrhagic transformation, heparin can also induce thrombocytopenia or vascular thrombosis.84,119,123,125-129 Pilot studies found that the low-molecular-weight heparinoid Org 10172 could be safely prescribed and that it might be effective in improving outcome130,131 (Level of Evidence V). A large multicenter trial is now testing the drug. Low-molecular-weight heparins are also being examined in clinical trials.

There are no data about the usefulness of aspirin, warfarin, or ticlopidine in the care of patients with acute ischemic stroke. Because aspirin’s effects are immediate, it might be effective in patients with acute stroke. It is being tested in clinical trials.

Recommendations

1. Because data about the safety and efficacy of heparin in patients with acute ischemic stroke are insufficient and conflicting, no recommendation can be offered (Levels of Evidence II through V). Data about the safety and efficacy of heparin in patients with recent cardioembolic stroke are also too sparse to support a recommendation (Levels of Evidence II through V). There are no data concerning any effects of the vascular distribution of the ischemic symptoms or the underlying vascular disease on the responses to heparin.

2. Until more data are available, the use of heparin remains a matter of preference of the treating physician. It should be understood that the use of heparin (or the lack of its administration) may not alter the outcome of a patient with acute ischemic stroke.

Thrombolytic Therapy

Measures that expedite clot lysis and restore normal circulation may limit brain injury and improve neurological outcome. Unfortunately, brain hemorrhage was a frequent complication among persons treated with thrombolytic drugs who were enrolled in studies performed in the late 1960s and early 1970s, and consequently the therapy was largely abandoned132-134 (Level of Evidence II). However, these studies were performed before CT, and patients with hemorrhages may have been treated. Patients were also treated many hours after stroke. More recently, interest in thrombolytic therapy has increased because of the development of new thrombolytic agents and the successful use of these drugs in the care of patients with acute myocardial ischemia. Experimental studies suggest that thrombolytic drugs may limit the extent of brain injury and may not be complicated by high rates of hemorrhage.135,136

Early clot lysis and arterial recanalization have been reported in approximately 50% of persons treated with intra-arterial streptokinase or urokinase137 (Level of Evidence V). Clot lysis and arterial recanalization can follow intravenous delivery of streptokinase, urokinase, or recombinant tissue plasminogen activator (TPA). The rate of recanalization is lower with intravenous
treatment even though drugs have usually been given soon after stroke onset. In an arteriography-based study of TPA, the Acute Stroke Study Group noted recanalization within 1 hour of starting treatment in 26% of patients with occlusion of the stem of the middle cerebral artery, in 38% of patients with distal branch occlusions, and in 8% of patients with occlusion of the extracranial internal carotid artery138 (Level of Evidence V). In other recent studies of intravenously given TPA, the rates of recanalization ranged from 21% to 53%139-141 (Level of Evidence V).

Thrombolytic therapy is potentially dangerous. The frequency of parenchymal hemorrhage ranges from 4% to 11%139-145 (Levels of Evidence II through V). The mortality among patients who have had hemorrhages is reported to be 18% to 50%139-146 (Levels of Evidence II through V). Hemorrhagic transformation of the ischemic lesion may be more frequent than frank hematoma formation, but clinical deterioration has not been frequent, and its relation to thrombolytic therapy is still uncertain.142

The efficacy of thrombolytic therapy has not been established. The only large completed trials investigated low doses of urokinase122 (Level of Evidence II). Only small numbers of patients have been recruited to the randomized trials evaluating the usefulness of thrombolytic drugs in clot lysis. In three recent randomized trials testing TPA, cohorts of 27, 31, and 98 patients139,141,145 were enrolled (Level of Evidence II). Only one of these studies reported outcomes among treated patients at 30 days after stroke. Clinical trials are now testing the usefulness of thrombolytic drugs. As yet the best route of administration (intravenous or intra-arterial), dose, agent, ancillary care, and duration of treatment have not been established. The potential efficacy of thrombolytic drugs may be greatly influenced by the length of the interval between the onset of stroke and the initiation of treatment.

**Recommendation**

Thrombolytic therapy is not currently recommended for the treatment of patients with acute ischemic stroke (Levels of Evidence II through V, Grade B). While the results of pilot studies are promising, caution should be exercised because intracranial bleeding is a potential complication. Thrombolytic drugs should be used only in an investigational setting, preferably within randomized clinical trials.

**Hemodilution**

Isovolemic hemodilution that lowers the hematocrit by 15% or more results in reductions in blood viscosity and improvements in cerebral blood flow.146 Two large clinical trials of isovolemic hemodilution were unable to demonstrate a decline in mortality or disability with treatment147,148 (Level of Evidence I). Hypervolemic hemodilution has been examined in two small randomized trials149,150 (Level of Evidence II). In a trial of 88 patients, mortality among patients treated with pentastarch was 20% compared with 7% among control patients.149 Among the actively treated patients, four of five deaths were due to cerebral edema. Koller et al150 treated 47 patients within 24 hours of stroke and noted improved neurological outcomes among treated patients. The clinical benefit of hemodilution therapy has not been established, and the possibility of excess brain edema has not been excluded. However, the potential usefulness of very early administration of therapy that rapidly achieves the desired levels of hemodilution during the first few hours after stroke has not been adequately tested. New agents that improve oxygen-carrying capacity are being examined. Further studies of hemodilution therapy in patients with polycythemia and stroke are needed.

**Recommendation**

Hemodilution therapy is not presently recommended for the management of patients with acute ischemic stroke (Level of Evidence I, Grade A).

**Cytoprotective Therapies**

Cytoprotective (neuroprotective) drugs protect brain tissue from ischemic damage. Three general strategies exist for the use of these drugs: (1) prophylaxis in patients at high risk, (2) administration shortly after the stroke begins, and (3) restoration of function. The use of several drugs that may have cytoprotective properties has been supported by anecdotal reports (Level of Evidence V).

The most commonly used drugs are steroids, barbiturates, and calcium channel blockers. Despite their general use, none of these drugs has been demonstrated as effective, possibly because they have been administered too late. Drugs that lessen neurological damage after stroke are likely to be more effective when given before ischemia begins; for example, when administered to a patient who is having frequent transient ischemic attacks or during a surgical procedure such as carotid endarterectomy. However, these drugs will be helpful only if sufficient levels can be achieved to maintain protection of the brain without side effects that limit patient acceptance.

An excessive influx of calcium occurs at the time of neuronal death.151 Several trials have tested blockers of the L-type calcium channels; although there was initial enthusiasm, the findings are mixed152-155 (Levels of Evidence I and II). One trial noted that nimodipine was associated with lower mortality152 (Level of Evidence II), but other clinical trials have not demonstrated any benefit153,154 (Levels of Evidence I and II). Intravenous nicardipine has been evaluated in pilot studies, but no conclusion about its potential efficacy was drawn156 (Level of Evidence V). The reasons for the lack of usefulness of L-channel blockers are unclear, but their effects in inducing hypotension may have counteracted any neuroprotective effects.

Ischemia induces a complex series of metabolic events that culminates in irreversible damage.157 Because the most critical links have not been identified, deciding which therapeutic techniques are most likely to reverse or ameliorate these events is difficult. Small studies of naloxone have not been encouraging, although the dose given to most patients was far lower than that given in experimental models158-160 (Levels of Evidence III through V). Since barbiturates suppress the metabolic activity of neurons, the analogy to reducing myocardial damage after myocardial ischemia by reducing cardiac workload is natural. Experimental studies suggest that barbiturates may be effective, but a clinical trial testing the usefulness of this therapy in
protecting the brain after cardiac arrest failed to demonstrate any benefit.\textsuperscript{161-162} (Level of Evidence I). A clinical trial of barbiturates in patients with focal brain ischemia has not been performed. The doses of barbiturates that may be needed to protect the brain may be sufficiently large to cause coma, which would complicate ancillary care.

Experimental studies demonstrate the efficacy of 21-aminosteroids in reducing brain damage after ischemic stroke.\textsuperscript{163} These drugs do not have glucocorticoid actions and may act by scavenging oxygen free radicals. The general concept of free radical damage is that highly reactive compounds are generated during ischemia, and that they, in turn, degrade structural molecules and liberate more free radicals in a chain reaction. Whether this process occurs in the brain during ischemia has not been proved. Pilot clinical trials of 21-aminosteroids have been completed, and trials testing their efficacy are under way. Other drugs that might inhibit free radicals, such as superoxide dismutase and GM1 gangliosides, are also being evaluated.

Excitatory amino acids, such as glutamate in high concentrations, can cause tissue necrosis.\textsuperscript{164} During ischemia, glutamate is released from presynaptic terminals, and normal inactivation mechanisms fail; as a result, postsynaptic neurons are excessively stimulated. The glutamate receptors are linked to calcium channels, which are kept open, and large amounts of unrestrained calcium enter the cells. Several classes of glutamate receptors have been identified, and experimental evidence suggests they will limit neurological damage.\textsuperscript{165} Yet, some of these glutamate antagonists have side effects such as changes in mental status. Preliminary clinical studies are testing the safety and potential efficacy of a number of receptor antagonists, including dextrophan and CGS 19755.

While experimental evidence suggests that hypothermia reduces brain injury, no data support the clinical usefulness of this therapy.\textsuperscript{166} Other biochemical pathways that may be involved in worsening ischemic damage include alterations in adenosine and arachidonic acid products.\textsuperscript{167,168} Several strategies for managing abnormalities in these metabolic pathways are being developed.

Experimental evidence suggests that resumption of flow in large arteries during reperfusion of ischemic brain may sometimes be ineffective in restoring microcirculatory flow. Reflow may actually exacerbate the initial ischemic injury by mechanisms that are not yet well defined.\textsuperscript{170} The offending agents may be white blood cells that irreversibly block the microcirculation even when occlusions are removed, and blood flow in large arteries recovers. The white blood cells may also release toxins that produce further damage. Antibodies to leukocytes are effective in reducing ischemic injury in animal models.\textsuperscript{171}

Therapies that might accelerate or increase neurological recovery are being evaluated; data from animal studies suggest that amphetamines administered soon after stroke can improve outcome.\textsuperscript{172} Drugs such as haloperidol may retard recovery. Several neurotrofic proteins have been shown to improve neuronal growth in tissue cultures and improve function in various types of acute injury models.\textsuperscript{173} Exploration of the use of these substances in recovery after stroke is just beginning.

**Recommendations**

1. Data about the efficacy of nimodipine in improving outcome after acute ischemic stroke are conflicting, and thus no recommendation is given (Levels of Evidence I and II).
2. Large doses of barbiturates do not protect the brain in patients with global brain ischemia; pending more definitive information, the use of this therapy in patients with ischemic stroke is not recommended (Level of Evidence I, Grade A).
3. Administration of naloxone in the management of acute ischemic stroke is not recommended (Level of Evidence II, Grade B).
4. Administration of glutamate antagonists to patients with ischemic stroke, outside the setting of clinical trials, is not recommended.
5. Administration of amphetamines for stimulating recovery after ischemic stroke, outside the setting of clinical studies, is not recommended.

**Surgical Interventions**

Clinical studies of surgical management in patients who have acute ischemic stroke provide limited information. Some surgeons report encouraging results from emergency carotid endarterectomy\textsuperscript{174,175} (Levels of Evidence III through V). Improvement can follow surgical correction of a severe stenosis or occlusion of the internal carotid artery in patients with mild or moderate neurological deficits has been recorded. The usefulness of surgery for patients with severe signs is unknown. Factors that may favor surgery are younger age, short interval from stroke to surgery, good collateral circulation as demonstrated by arteriography, and few severe concurrent medical diseases. Relative contraindications appear to be advanced age, depression of consciousness, ischemia of the dominant hemisphere, poor medical condition, poor collateral circulation, and a time interval beyond 24 hours from onset of symptoms.

Although a new neurological deficit following carotid endarterectomy may prompt immediate re-exploration of the artery to search for a thrombus or occlusion, neurological signs may also be due to intraoperative ischemia, embolism, edema secondary to hyperperfusion, or hemorrhage. Emergent evaluation (CT, duplex imaging, transcranial Doppler, or arteriography) should be performed before returning the patient to the operating room.

Occasionally, sudden neurological deficits from an embolus in the middle cerebral artery can be reversed by prompt microsurgical embolectomy\textsuperscript{176} (Level of Evidence V). Emergent extracranial-intracranial arterial bypass surgery after an acute stroke in the distribution of the middle cerebral artery has failed to improve outcome and may be associated with a high risk of brain hemorrhage\textsuperscript{177} (Level of Evidence V). Experience with bypass operations for treatment of acute stroke in the vertebrobasilar circulation is limited. Triple lumen catheters have been designed for angioplasty treatment of the arterial lesions in either the carotid or vertebrobasilar circulation. Preliminary data about the efficacy of angioplasty are encouraging but limited\textsuperscript{178,179} (Level of Evidence V).
Recommendations

1. Data about the safety and efficacy of emergent carotid endarterectomy in the care of acute ischemic stroke are insufficient to permit a recommendation (Levels of Evidence III through V). Data about the safety and efficacy of embolectomy or angioplasty in treatment of acute ischemic stroke are equally insufficient, and thus no recommendation is offered (Level of Evidence V).

2. Until more data are available, the use of surgical procedures in the care of acute ischemic stroke remains a matter of the treating physician’s preference. It should be understood that these procedures may have serious risks and may not favorably alter the outcome of the patient with acute ischemic stroke.

References


