Cerebral vascular accident (CVA)—known also as “stroke” or “brain attack”—is a disruption of normal blood supply to the brain. Its sudden onset requires immediate treatment to prevent or lessen neurological deficit and lifelong disability.
Countering "brain attacks"

Cerebral vascular accident (CVA)—known also as “stroke” or “brain attack”—is a disruption of normal blood supply to the brain. Its sudden onset requires immediate treatment to prevent or lessen neurological deficit and lifelong disability.

By Joan Harvey, RN, MSN, CCRN

Abstract: Proactive nursing markedly improves CVA patients’ outcomes. [Nurs Manage 2004:35(8):27-33]

Every year, approximately 700,000 Americans experience a new or recurrent stroke.1 And every three minutes, someone dies from the event.2 Because the brain’s metabolic demands are high compared to other organs, permanent damage can occur within minutes if blood supply is interrupted.

The basics: classification, causes
Strokes are classified according to type: ischemic and hemorrhagic. Ischemic is further delineated into thrombotic and embolic. Ischemic stroke is caused by an obstruction of cerebral blood flow, as in a vessel blockage. Ischemic stroke can also be caused by an embolus, then referred to as an embolic stroke.

Ischemic stroke causes include:
♦ intracranial arteriosclerosis
♦ carotid plaque with arteriogenic emboli
♦ flow-reducing carotid stenosis
♦ aortic arch plaque.

Cerebral vessel occlusion from thrombosis in both large and small arteries occurs most frequently in the presence of atherosclerotic cerebral vascular disease. Vascular changes in deep penetrating arteries associated with chronic hypertension can result in small vessel thrombosis.3

A stroke caused by a thrombus, referred to as a thrombotic stroke, is associated with the development of arteriosclerosis of the blood vessel wall. Plaque deposits build up on vessel walls and over time—usually years—cause decreased blood flow and eventual blockage. Inflammatory disease processes may also damage the vessel wall and cause narrowing. With any type of narrowing, the vessel wall is subject to clot formation from platelet adhesion, aggregation, and fibrin attachments.

Embolic stroke results when an embolus travels to the brain and cuts off blood supply. Major causes include:
♦ atrial fibrillation
♦ cardiogenic emboli
♦ valvular disease
♦ left ventricular thrombi.

Cerebral emboli are often a complication from cardiovascular disease. Cardiovascular disease, a risk factor in and of itself, further increases the risk of embolic formation when coupled with ventricular wall hypokinesis or atrial arrhythmia. Emboli are released from the cardiovascular system and carried to the cerebral circulation, thus obstructing blood flow and causing stoke.

Hemorrhagic stroke results from a vessel wall rupture and bleeding into the brain or into the spaces around the brain. Hemorrhagic stroke can be the result of an aneurysm rupture, uncontrolled hypertension, a subarachnoid hemorrhage, infections, clotting disorders, head/neck trauma, radiation to the head/neck, and cerebral angioplasty.

A closer look at circulation and neurofunction
Because stroke manifestations depend on the affected vessel or area, clinicians should clearly understand the circulation of the brain. The carotid arteries supply 80% of cerebral blood flow, with the vertebral arteries supplying the remaining 20%. The internal carotid arteries supply the anterior and middle parts of the cerebral hemispheres. The vertebral arteries join to form the basilar artery.

The vertebral-basilar arteries supply the posterior parts of the cerebral
Brain attacks

hemispheres, the brainstem, and the cerebellum. Blood flows from the internal carotid and vertebral-basilar arteries into a ring of vessels joined together known as the Circle of Willis, which sits at the base of the brain. It consists of the anterior communicating artery and the paired anterior cerebral, internal carotid, posterior communicating, and posterior cerebral arteries. Each of the arteries supplies blood flow to a different region of the brain. Specifically, the anterior cerebral artery supplies the medial surface of the frontal and parietal lobes. The middle cerebral arteries supply the lateral surfaces of the cerebral hemispheres and smaller branch penetrating arteries, bringing blood flow deeper into the brain’s structure. The posterior cerebral arteries supply the medial and inferior surfaces of the occipital and temporal lobes.

The brain’s vascular system automatically changes the diameter of its vessels to maintain a constant cerebral blood flow during blood pressure fluctuations—a process known as autoregulation. The brain can effectively autoregulate when systolic blood pressure is 60 to 100 mm/Hg. But when autoregulation fails, cerebral blood flow diminishes, resulting in ischemia.4

To understand the clinical presentation of stroke, you should have a basic understanding of neuroanatomy and function. The brain is divided into three main components: the cerebrum, the cerebellum, and the brain stem. The cerebrum is the largest and most advanced part, responsible for several functions, including higher intellect, speech, emotion, integration of sensory stimuli of all types, initiation of the final common pathways for movement, and fine control of movement. The cerebellum, the second largest area, is responsible for maintaining balance and assists in the control of movement and coordination. The brain stem is responsible for a variety of automatic functions, such as control of respiration, heart rate, and blood pressure, and wakefulness, arousal, and attention.

The cerebrum is divided into a right and a left hemisphere, each of which has a frontal, parietal, temporal, and occipital lobe. The right side of the brain controls the function of the left side of the body and vice versa. This occurs because the nerves cross in the brain stem.

Clinical presentation

Clinical presentation of stroke varies according to the location of actual blood supply deficit. Symptoms are sudden in onset and can range from deep coma to local manifestations on one side of the body. If the right cerebral hemisphere is affected, subsequent weakness on the left side of the body will occur, including left arm or leg weakness, numbness, or even neglect. 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transient ischemic attack (TIA). Patients who have a TIA must be evaluated promptly for stroke risk. According to the National Institute of Neurological Disorders and Stroke, approximately one-third of Americans who experience TIAs will have a stroke.5

**Diagnostics**

To effectively treat stroke, you must act quickly, as time is essential to brain tissue and function preservation. Upon patient arrival to the emergency department, the health care team will ask him or her a series of questions, the most important of which is when symptoms first occurred and what they are, specifically. It’s crucial to know when the symptoms began, as this will help tailor patient treatment. A series of bedside neurological assessments and a specific exam developed to determine the severity of stroke will ensue. (To view the National Institutes of Health Stroke Scale, visit http://www.strokecenter.org/trials/scales/nihss.pdf.)

Also, the clinician will order common blood tests such as a CBC, platelet count, PT, PTT, a metabolic panel, and a urinalysis. Carefully review blood glucose results, as blood sugar disturbances often mimic stroke. Additionally, an electrocardiogram is done to rule out arrhythmia.

The single most effective tool to diagnose a stroke is a computerized axial tomography (CT) scan. To devise an effective treatment plan, a radiologist will determine if the stroke is ischemic or hemorrhagic. In addition to a CT scan, the clinician may order an MRI to help determine the exact location and size of injury. MRIs, which can find evidence of a stroke within minutes of its occurrence, prove especially beneficial for assessing smaller strokes located deep in brain tissue.

The clinician may also order an electroencephalogram, which reveals the brain’s electrical activity and helps screen for any seizure activity resulting from brain injury. In addition, carotid Doppler studies help evaluate the patency of the carotid arteries.

**Nursing’s response**

The focus of therapeutic management for the stroke patient is the preservation of life, prevention of additional brain damage, and reduction of disability. Care of the acute stroke patient proves challenging, as brain cells die within minutes. The longer the patient’s brain is deprived of oxygen and blood, the more likely that he or she will suffer permanent damage.

Lifestyle changes may help decrease stroke risk factors. Encourage patients to lead a healthy lifestyle—one that includes a balanced diet; aerobic exercise for at least 30 minutes most days of the week; monitor and control blood pressure, blood glucose, and lipid levels; and smoking cessation, if appropriate.

Although nothing will stop a stroke from occurring, one drug—tissue plasminogen activator (t-PA)—can minimize the size of the infarcted area by opening blocked vessels that are occluded with clot. The drug works by converting plasminogen to plasmin, which in turn dissolves the clot. Patients eligible to receive t-PA, according to National Institutes of Health criteria, include those whose symptom onset is less than 3 hours, those older than 18 years of age, and those whose CT scan shows no evidence of intracerebral bleeding.

Although expectations of positive outcomes are reduced and symptomatic intracranial hemorrhage rates are higher in severe stroke population, treatment with t-PA improves outcomes, but overall success rates remain low.6 T-PA isn’t recommended for patients with isolated mild neuro deficits, such as ataxia alone, sensory loss alone, dysarthria alone, or trace weakness.7 (See “Contraindications and other t-PA considerations.”)

If t-PA is used, the recommended administered dose is 0.9mg/kg; maximum dose is 90 mg given over 60 minutes, with 10% of the total dose given as a bolus over one minute. Nurses should acquire all baseline laboratory data and ensure several working intravenous lines.

Nurses also remain responsible for frequent vital sign monitoring, monitoring of neurological status, monitoring for bleeding complications, and limiting invasive procedures for at least 24 hours. In addition, no anticoagulants or antiplatelets may be used for 24 hours post t-PA. A CT scan is usually done within 24 hours. A follow-up CT rules out any type of bleeding post t-PA.

If t-PA isn’t used, the focus of treatment lies with the symptoms the patient exhibits and the potential cause of the stroke event. If the patient has a cardiac history and clinicians discover atrial fibrillation, he or she should receive anticoagulants, once a hemorrhagic stroke is ruled out by CT scan.

If the patient is hypertensive and the stroke is caused by hemorrhage, surgical intervention may be required to evacuate the bleed. The patient may receive an antiseizure medication as a prophylactic measure. If the patient is hypertensive, blood pressure is lowered slowly with medications to ensure adequate cerebral perfusion. If the patient has hyperlipidemia, anti-cholesterol medications like statins will be instituted, once liver function is determined to be normal. To treat cerebral vasospasm, the calcium channel blocker nimodipine (Nimotop) may be given, which relaxes smooth muscles of the vessel wall and reduces the incidence and severity of the spasm, thus improving cerebral blood flow and neurologic function.

Proactive nursing will help stroke patients avoid or successfully recover from complications such as pneumonia, aspiration, deep vein thrombosis, pulmonary embolism, pressure ulcers, malnutrition, and contractures. Target education to the patient, significant others, and additional caregivers.

Keep the patient’s airway patent and encourage deep breathing and coughing exercises, incentive spirome-
Brain attacks

try, early mobility, and frequent position changes to help prevent atelectasis. Always monitor vital signs and observe closely for symptoms of developing pneumonia, which include fever, chills, elevated white blood cell count, and changes in breath sounds. Alert the clinician to any of these changes and anticipate orders for blood cultures, sputum culture, chest x-ray, and antibiotic therapy.

Evaluate swallowing early in the patient’s admission to avoid aspiration. If the patient has trouble swallowing, keep him or her NPO until you receive a speech therapy evaluation, which should occur within 24 hours. Alert involved patient care staff and family members of the risk of aspiration. Conduct nutritional consultations to ensure that the patient meets his or her caloric needs either orally or via an alternate route.

Evaluate deep-vein-thrombosis risk and take measures to prevent its occurrence, including the use of sequential compression devices, antiembolic hose, or low-molecular-weight heparin, when appropriate. Movement of extremities will help prevent blood stasis and pressure ulcers.

Other considerations

Stroke patients’ lengths of stay greatly impact hospital finances. Quite frankly, stroke is expensive. The estimated direct and indirect cost of stroke in 2004 is $53.6 billion. Nurses and case managers who follow patients closely and consult with clinicians daily can help decrease costs. To that end, consider using care maps as a guide to best practice regarding diagnostic tests, speech evaluations, physical therapy, occupational therapy, and dietary instructions. These maps will encourage staff members to ask the right questions and prompt clinicians to order appropriate treatment.

Thrombolytic therapy proves costly to hospitals, but decreases global health care costs, in general. For 1,000 patients receiving t-PA, hospitalization costs total $1.7 million more than non-t-PA patients, rehabilitation costs $1.4 million less, and nursing home costs $4.8 million less—resulting in 564 quality-adjusted life-years saved.9

Insurance companies also play a pivotal role in the care of stroke patients. Despite the presence of clinical symptoms, some companies mandate certain studies to make a diagnosis. Delays in obtaining these studies will increase patient length of stay. Hospital administrators need to examine current reimbursement schedules and review delayed costs, weighing patient need and financial impact.

References


About the author

Joan Harvey is a clinical educator at Ocean Medical Center, Brick, N.J.

CE Test

Countering “brain attacks”

Instructions:
• Read the article beginning on page 27.
• Take the test, recording your answers in the test answers section (Section B) of the CE enrollment form. Each question has only one correct answer.
• Complete registration information (Section A) and course evaluation (Section C).
• Mail completed test with registration fee to: Lippincott Williams & Wilkins, CE Group, 333 7th Ave., 19th Floor, New York, N.Y. 10001.
• Within 3 to 4 weeks after your CE enrollment form is received, you will be notified of your test results.
• If you pass, you will receive a certificate of earned contact hours and an answer key. If you fail, you have the option of taking the test again at no additional cost.
• A passing score for this test is 11 correct answers.
• Need CE STAT? Visit http://www.nursingcenter.com for immediate results, other CE activities, and your personalized CE planner tool.
• No Internet access? Call 1-800-933-6525 for other rush service options.
• Questions? Contact Lippincott Williams & Wilkins: 646-674-6617 or 646-674-6621.

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**Countering “brain attacks”**

**GENERAL PURPOSE:** To provide professional nurses with an understanding of the care of a patient who has had a cerebral vascular accident.

**LEARNING OBJECTIVES:** After reading the preceding article and taking the following test, you’ll be able to: 1. Identify risk factors associated with cerebral vascular accidents. 2. Discuss specific cerebral anatomy and physiology as it relates to cerebral vascular accidents. 3. Describe clinical manifestations of cerebral vascular accidents. 4. Describe therapeutic interventions for patients who have had a cerebral vascular accident.

1. Thrombotic strokes may develop as a result of:
   a. chronic hypertension.
   b. long-term use of aspirin.
   c. head trauma.
   d. acute renal failure.

2. Patients at risk for embolic strokes include those who have:
   a. deep vein thrombosis.
   b. uncontrolled hypertension.
   c. diabetes mellitus.
   d. atrial fibrillation.

3. A hemorrhagic stroke may develop as a result of:
   a. a cerebral angioplasty.
   b. diabetes mellitus.
   c. elevated cholesterol.
   d. cardiac dysrhythmias.

4. The carotid arteries provide what percentage of the blood supply to the brain?
   a. 40%
   b. 60%
   c. 80%
   d. 100%

5. The middle cerebral arteries provide blood flow to which area of the brain?
   a. medial surface of the parietal lobes
   b. lateral surface of the cerebral hemispheres
   c. inferior surface of the occipital lobes
   d. posterior surface of the temporal lobe

6. Which statement is true about the blood supply in the brain?
   a. The internal carotid artery provides blood to the posterior regions of the brain.
   b. The purpose of autoregulation is to maintain adequate oxygenation to the brain.
   c. The vertebral-basilar arteries supply 60% of the blood that reaches the Circle of Willis.
   d. Ischemia may develop if the systolic blood pressure is less than 70 mm Hg.

7. One function of the cerebrum is to:
   a. integrate sensory input.
   b. maintain balance.
   c. control wakefulness.
   d. regulate blood pressure.

8. A patient who has had a right cerebral vascular accident may have:
   a. a gaze that’s prominent to the left.
   b. expressive aphasia.
   c. a loss of the visual field in both eyes.
   d. neglect.

9. A patient who has had a left cerebral vascular accident may have:
   a. a loss of the left visual field.
   b. asymmetry of the facial muscles on the left side.
   c. mixed aphasia.
   d. a gaze that’s prominent to the right.

10. Cerebral vascular accidents in the thalamic area produce:
    a.nausea.
    b. abnormal eye movements.
    c. tinnitus.
    d. sensory alterations.

11. Transient ischemic attacks:
    a. usually last 48 hours.
    b. may result in residual disabilities.

**ENROLLMENT FORM: Nursing Management, August 2004, Countering “brain attacks”**

**A. Registration Information:**

Last name ___________________________ First name ________________________ MI _____
Job title ___________________________ Specialty ___________________________
Type of facility ___________________________ Are you certified? a. Yes b. No
Certified by __________________________________________________________________________
State of license (1) ___________________________ License # ___________________________
State of license (2) ___________________________ License # ___________________________
Social Security # _____________________________________________________________________

**B. Test Answers:** Darken one circle for your answer to each question.

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**C. Course Evaluation*”**

1. Did this CE activity’s learning objectives relate to its general purpose? a. Yes b. No
2. Was the journal home study format an effective way to present the material? a. Yes b. No
3. Was the content relevant to your nursing practice? a. Yes b. No
4. How long did it take you to complete this CE activity? ________ hours ________ minutes
5. Suggestion for future topics ___________________________________________________________

**D. Two Easy Ways to Pay:**

a. Check or money order enclosed (Payable to Lippincott Williams & Wilkins)
b. Charge my: Mastercard Visa American Express
Card # _______________ Exp. date _______________

Signature __________________________________________________________

*In accordance with the Iowa Board of Nursing administrative rules governing grievances, a copy of your evaluation of the CE offering may be submitted directly to the Iowa Board of Nursing.