The Neuroscience of Learning

John W. Collins

Abstract: Significant advances have been made in understanding the neurophysiological basis of learning, including the discovery of mirror neurons and the role of cyclic adenosine monophosphate (cAMP) responsive element binding (CREB) protein in learning. Mirror neurons help us visually compare an observed activity with a remembered action in our memory, an ability that helps us imitate and learn through watching. Long-term potentiation, the Hebb rule, and CREB protein are associated with the formation of long-term memories. Conversely, protein phosphatase 1 and glucocorticoids are neurophysiological phenomena that limit what can be learned and cause forgetfulness. Gardner's theory of multiple intelligences contends that different areas of the brain are responsible for different competencies that we all possess to varying degrees. These multiple intelligences can be used as strategies for improved learning. Repeating material, using mnemonics, and avoiding overwhelming stress are other strategies for improving learning. Imaging studies have shown that practice with resultant learning results in significantly less use of brain areas, indicating that the brain becomes more efficient. Experts have advantages over novices, including increased cognitive processing efficiency. Nurses are in a unique position to use their understanding of neurophysiological principles to implement better educational strategies to provide quality education to patients and others.

In 1986, Boss wrote about the neuroanatomical and neurophysiological basis of learning. Since then, significant advances have been made in our understanding of these phenomena. Long-term potentiation (LTP) and cyclic adenosine monophosphate (cAMP) responsive element binding (CREB) protein activation are two aspects of long-term memory formation. In addition, mirror neurons have been identified; they are implicated in learning new tasks. This article describes these and other advances in the neuroscience of learning—including Gardner's theory of multiple intelligences—that are applicable to patient and nurse education. Nursing implications are also presented.

Classification of Memory

There are several ways to classify memory (Table 1). Short-term (working) memory and long-term memory constitute the most basic classification (Miyashita, 2004). Long-term memory is divided into declarative and nondeclarative forms. Declarative memory, also called explicit long-term memory, is subdivided into semantic memory and episodic memory (Miyashita; Squire, 2004). *Declarative memory* is the conscious, intentional recognition of previous experiences, including facts (Kolb & Whishaw, 2003). *Semantic memory* involves remembering factual knowledge, including learning text, phone numbers, or trivia. *Episodic memory* is memory associated with events, locations, or circumstances.

Memory processing is categorized into three stages: acquisition, consolidation, and retrieval (Lupien & McEwen, 1997). *Acquisition* is the time when the individual acquires the information to be remembered. *Consolidation* is the conversion of declarative memory into long-term memory. *Retrieval* is the process of recalling a memory.

Both semantic and episodic memories are consolidated in the hippocampus, which is located in the medial temporal lobe (Miyashita, 2004; Squire, 2004). Semantic memories formed over time do not require the hippocampus for retrieval because these memories are retrieved from the cortex (Miyashita). It is unclear whether the hippocampus is required for the retrieval of episodic memory.

Nondeclarative memory, also known as implicit long-term memory, involves procedural memory, perceptual representation (e.g., recalling remembered sensory phenomena), and simple classical conditioning. *Nondeclarative memory* is an unconscious, nonintentional form of memory that is expressed through performance rather than recollection (Kolb & Whishaw, 2003; Squire, 2004). *Procedural memory*

Questions or comments about this article may be directed to John W. Collins at jc390@umkc.edu. He is a PhD student in the School of Nursing at the University of Missouri–Kansas City, Kansas City, MO. His area of study is learning with a neuropsychological foundation.

Table 1. Classification of Long-Term Memory

Memory Type	Item Remembered
Declarative or explicit	
Semantic	Facts
Episodic	Events
Nondeclarative or implicit	
Procedural	Skills
Perceptual representation	Sensory
Simple classical conditioning	Emotional or skeletal responses

Note. Information from "Cognitive Memory: Cellular and Network Machineries and Their Top-Down Control," by Y. Miyashita, 2004, *Science, 306*, 435–440 and "Memory Systems of the Brain: A Brief History and Current Perspective," by L. R. Squire, 2004, *Neurobiology of Learning and Memory, 82*, 171–177.

is the memory of knowing how to do something and includes the memory of motor events and skills, such as riding a bike or giving an injection (Squire). The striatum, a subcortical motor area, is responsible for procedural memory. The striatum consists of the caudate, putamen, and ventral striatum (Gazzaniga, 2004). *Perceptual representation* is the recalling of sensory memories (e.g., the way a bird looks or sounds when singing). Perceptual learning occurs in the neocortex. *Simple classical conditioning* consists of an emotional component, which is stored in the amygdala, and skeletal responses, which are stored in the cerebellum (Squire). These memory classifications have different neuroanatomical locations in the brain yet operate in parallel with each other (Miyashita, 2004; Squire).

Learning and long-term memory are intricately related. Kandel and Hawkins (1995) noted that learning is the process by which one acquires new knowledge. Memory is the process by which knowledge is retained. For long-term memory to occur, changes in the neurons must occur (Kolb & Whishaw, 2003). Specific changes are addressed later in this article.

In one of the most publicized neuropsychological case studies, HM illustrates the difference between the declarative and nondeclarative memory systems (Parkin, 1996). Subsequent to head trauma from a bicycle accident when he was 7 years old, HM developed petit mal and grand mal epilepsy, which significantly interfered with his activities of daily living. In the 1950s, as a treatment for seizures, he underwent a medial temporal lobectomy, including removal of the hippocampus. The hippocampus, which is part of the medial temporal lobe, is essential for consolidating long-term memories (Miyashita, 2004). After surgery, the quantity and severity of the seizures were reduced, but HM was left with antegrade amnesia. In HM's case, antegrade amnesia meant that he had no declarative memory of any events after the operation. In other words, HM lost the ability to commit to long-term memory any facts and events that occurred after the operation. He retained implicit (nondeclarative) memory, such as procedural memory, so he had the ability to learn new and remember previously learned skills and procedures; however, he could not learn any new facts. This example illustrates that declarative memory and nondeclarative memory are two separate memory systems. Though separate, some activities, such as performing a neurological exam and recognizing an abnormality by name, engage both memory systems.

Theory of Multiple Intelligences

Gardner (1983) recognized that different areas of the brain are responsible for different competencies. He labeled these competencies as different intelligences that we all possess to varying degrees. Gardner (1999) updated and slightly expanded the original list of multiple intelligences. He derived his theory from multiple sources, including personal musical experience, observations of gifted individuals, and evidence from experimental psychology and neuroscience. These intelligences are linguistic, logical-mathematical, spatial, bodily-kinesthetic, musical, interpersonal, intrapersonal, and naturalist (Table 2). Gardner noted that traditional IQ tests measure only a limited amount of our human potential, primarily logical-mathematical intelligence, with some testing of spatial and linguistic intelligences. Musical, bodily-kinesthetic, and intrapersonal intelligences are not measured on traditional IQ tests.

Gardner's theory of multiple intelligences has been criticized because research has not documented specific evidence of intelligences located in specific areas of the brain. Klein (1997) felt that some of Gardner's intelligences overlap in the cognitive areas of the brain. In contrast, Kornhaber (2004) noted that the theory of multiple intelligences has been associated with improvement in both standardized test scores and the behaviors of elementary students, including students with disabilities. In addition, the theory of multiple intelligences has also been associated with increased parental participation, suggesting that it is being used selectively in schools.

Mirror Neurons and Their Role in Learning

It is natural when learning to do a new task to mimic and copy this new task. Specific brain neurons, called mirror neurons, help us learn action tasks (Rizzolatti & Craighero, 2004). These mirror neurons help us visually compare an observed

Table 2. Gardner's Intelligences

Intelligence Name	Description of Intelligence	Occupations Favored
Linguistic	The ability to learn, speak, and write language	Lawyers, speakers, writers, and poets
Logical-mathematical	The ability to logically problem solve, perform math, and research scientifically	Mathematicians and scientists
Musical	The ability to perform, compose, and appreciate music	Musicians, musical composers, and musical directors
Bodily-kinesthetic	The ability to use all or part of the body	Athletes, dancers, surgeons, and crafts-people
Spatial	The ability to visualize and manipulate objects in space	Pilots, air traffic controllers, architects, and graphic artists
Interpersonal	The ability to understand and work well with other people	Clergy, salespeople, teachers, and politicians
Intrapersonal	The ability to understand oneself and to use the information effectively in one's own life	
Naturalist	The ability to recognize and classify the living world	Naturalists, botanists, hunters, farmers, and marine biologists

Note. Information from *Intelligence Reframed: Multiple Intelligences for the 21st Century,* by H. Gardner, 1999, New York: Basic Books.

activity with a remembered action in our motor memory repertoire (procedural memory). The closer the match, the easier it is to repeat this new activity.

Mirror neurons were discovered in the monkey premotor cortex in the early 1990s (Rizzolatti & Craighero, 2004). They were named mirror neurons because they fire both when an individual performs an activity and when the individual observes that same activity being performed by another individual. Mirror neurons have further been implicated in the understanding of the actual intentions behind others' actions (Iacoboni et al., 2005).

Neurophysiology of Learning

Several neurophysiological phenomena relate to learning. The Hebb rule, LTP, and CREB protein are associated with the formation of long-term memories. Neuroscientists believe that for short-term memories to be converted into long-term memories, a structural change must occur within the neurons (Miyashita, 2004; Silva, Kogan, Frankland, & Kida, 1998). Increased protein synthesis leads to the structural reorganization of the neurons. CREB protein activation has been implicated in the formation of long-term memories (Silva et al.). Without CREB protein activation, short-term memories are not converted into long-term memories. In experiments with mice, a mutated form of CREB protein was used, and long-term memories were not formed. Deficits in CREB protein activation can be compensated for through extended, spaced training sessions; when more CREB protein is activated, learning occurs with less training.

Hebb (1949) hypothesized that with repeated firing of a particular neuron's synapse, the cell's efficiency is improved at that synapse (Klein, 1999). This phenomenon has been named Hebb's rule, and it was first shown to occur with LTP.

LTP refers to a group of relatively longer-lasting enhancements of improved efficiency at the synapse (Hodgson, Standish, Boyd-Hodgson, Henderson, & Racine, 2004; Silva et al., 1998). After a burst of high-frequency stimulation to a monosynaptic pathway, the postsynaptic response magnitude is significantly increased. This enhanced response persists indefinitely and is associated with changes in the density and the length of the dendrite of the postsynaptic neuron (Kolb & Whishaw, 2003). LTP is a prime candidate for long-term memory formation because of its longevity and its associativity and reversibility properties (Silva et al.). Repeated stimulation of neurons in the hippocampus will cause LTP (Frey & Morris, 1997). Though LTP has been induced purposefully under experimental conditions, these same long-term changes are also believed to occur in the neuron with repeated stimulation—structural changes that are fundamental for long-term memory formation. It is hypothesized that a cAMP second messenger system with protein phosphatases determines when LTP can occur at synapses. Experiments have been performed with unilateral training in rats, with the untrained side serving as a control. Hodgson et al. concluded that the trained hemisphere relies on mechanisms in common with LTP.

Fields (2005) reviewed some molecular factors that cause the conversion of short-term to long-term memories. Genes in the nucleus are activated and synthesize proteins that strengthen synapses. The strength of signals, the frequency with which a synapse is used, and the passage of time allow this process to occur. Practice and repetition are major factors in whether a short-term memory becomes a long-term memory (Genoux et al., 2002).

Conversely, protein phosphatase 1 (PP1) and glucocorticoids are neurophysiological phenomena that limit what can be learned and cause forgetfulness. PP1 acts as a constraint on learning and memory (Genoux et al., 2002). It is believed that PP1 inactivates CREB protein (Silva et al., 1998). PP1 is activated by modest but enduring changes in intracellular calcium (Gazzaniga, 2004); conversely, PP1 is inhibited, and LTP is induced, by higher, yet brief, intracellular levels of calcium. Repetition of an activity to be learned, when interspersed with rest periods, has been shown to inhibit PP1 and induce LTP (Genoux et al.). One might ask why there is a protein that causes forgetfulness. It is not beneficial for people to remember everything. There is a survival advantage to remember, recall quickly, and learn and recognize important or threatening phenomena. PP1 provides a system of checks and balances.

Low to moderate stress, however, actually facilitates memory. Thus, some endogenous corticosteroids are necessary for optimal mental performance.

Another factor that is detrimental to learning and memory formation and retrieval occurs with excessive glucocorticoid release secondary to stress (de Quervain, Roozendaal, & McGaugh, 1998). Corticosteroids have a U-shaped effect on memory (Lupien & McEwen, 1997). Low or high corticosteroid levels impair memory acquisition, consolidation, and retrieval. Both Addison disease (low corticosteroid levels) and Cushing syndrome (high corticosteroid levels) are known to cause fatigue and irritability (Kasper et al., 2005). Fatigue and irritability impede one's ability to concentrate, and these states are stressful. Prolonged or severe stress with excessive corticosteroids disrupts LTP and causes dendritic atrophy in the hippocampus (Gazzaniga, 2004). In addition, brain imaging in patients with Cushing syndrome has shown decreases in hippocampal volume that are normalized after several months of treatment. Low to moderate stress, however, actually facilitates memory. Thus, some endogenous corticosteroids are necessary for optimal mental performance.

Expert Learning

Studying people who are considered experts is advantageous for two reasons: categorizing what differentiates experts from novices is fundamentally enlightening, and the strategies that experts use may be beneficial for effective learning in general. Memory experts, or mnemonists, are able to remember many details, such as names, numbers, and items. They are able to memorize the order of each card in a shuffled deck of 52 standard playing cards in less than 3 minutes and then accurately recall the exact sequence of all 52 cards (Butcher, 2000).

Memory experts, in general, use different memory strategies than most other experts, including experts in diverse fields such as chess, bridge, music, nursing, medicine, computer programming, and teaching. Memory experts use mnemonics, especially visual mnemonics (Butcher, 2000; Maguire, Valentine, Wilding, & Kapur, 2003). In a study comparing 10 memory experts with 10 control participants using standard neuropsychological measures and functional magnetic resonance imaging (MRI; Maguire et al., 2003), those with superior memories used a spatial learning strategy to improve encoding and the retention of numbers. The memory experts were no better than the control participants when attempting to remember random patterns. This spatial strategy engages six specific brain regions: the medial parietal cortex, retrosplenial cortex, right posterior hippocampus, right cingulate cortex, left fusiform cortex, and left posterior inferior frontal sulcus. These brain regions are important in mental imagery and spatial working memory tasks (Gazzaniga, 2004).

Hill and Schneider (2006) discussed many aspects regarding the brain changes that occur with the gaining of expertise. There is a myth that we use only 10% of our brains, with the concomitant inference that more brain usage is better. The brain is very domain specific. For example, visual images are processed mostly in the occipital lobes, while sounds are processed in the parietal lobes. Furthermore, imaging studies have shown that practice with resultant learning from the repetitive practice results in significantly less utilization of brain areas. In other words, the brain becomes more efficient. Experts have greater cognitive processing efficiency compared to novices. In addition, experts have enriched representations of the knowledge of their specific fields. Experts have the ability to flexibly use different strategies by recruiting various brain regions to solve a range of problems, which gives them an advantage over novices. Experts also recognize and have in their repertoire a large number of patterns, which they can access automatically to solve problems (Hill & Schneider).

Nursing Implications for Learning

The nursing implications for learning include patient education and nurse education. The learning strategies presented here are not mutually exclusive to patients or nursing students. Presenting material in different sensory modalities is a useful strategy both in the classroom and in patient education. The material presented can be used to design teaching strategies or personal learning strategies.

Patient Education

Because different activities are processed in different parts of the brain, if one part of a patient's brain is injured or impaired, the nurse can search for other areas that are not impaired. For example, if Wernicke's language area is damaged, receptive aphasia will occur. This means the patient will have trouble reading and understanding written materials. Therefore, to attempt to communicate and gain connection with the patient, the nurse can explore alternate areas of the brain, such as musical, interpersonal, and bodily-kinesthetic areas. Simple gesturing, showing pictures, mirroring but not mimicking body mannerisms, and even singing can be used to communicate and connect with the patient.

An important practice for optimal learning is that rest intervals be interspersed between learning sessions. When patients appear overwhelmed with the learning, allowing them to rest and then resuming when they are more alert will increase the amount learned. Physical and psychological stress is usually a component of illness. Learning in healthcare settings is often impaired by the effects of stress and the concomitant high compensatory glucocorticoid secretion, increased production of PP1, and decreased CREB protein activation and LTP. A patient education program may include the learning of many new details. If these details are lumped into a single lesson, some patients will be overwhelmed. Thus, these patients experience the activity but do not retain anything. The antidote is to include patient participation in presentations, including restating the teaching and demonstrating the information taught.

Nurse Education

Because practice and repetition are major factors in committing information to long-term memory, important material needs to be repeated and emphasized. Neuroscience teaches us several ways that material can be emphasized. A powerful technique is to build on existing learning networks. This approach incorporates new learning experiences into existing long-term memory. Presenting material in different sensory modalities is another technique used by educators. Linking visual pictures of the material with existing knowledge is an example of using sensory modalities in education.

Gardner's theory of multiple intelligences suggests another learning strategy. Using the different intelligences to emphasize an important lesson is not only useful but also common in nursing. For example, learning about intravenous fluid and medication administration is challenging for most nursing students. It is common to study the medications and the technique, which involves linguistic intelligence. Calculating flow rates and the quantities of medication is categorized as logical-mathematical learning. Finally, nursing students practice the technique in a nursing lab, which involves both spatial and bodily-kinesthetic intelligences.

Summary

Nurses are in a unique position to understand neurophysiological principles and implement educational strategies based on these principles to provide quality education to patients and others. This is an exciting time because cognitive neuroscience is finally able to explain how and why these techniques work from a neurophysiological perspective. Even more exciting is that there will be many more breakthroughs and refinements in our understanding of learning during this century.

Acknowledgment

I would like to express my gratitude to Chris Lovelace, PhD, of the University of Missouri–Kansas City for sharing his expertise of neuropsychology and for teaching me this fascinating subject.

References

Boss, B. J. (1986). The neuroanatomical and neurophysiological basis of learning. *Journal of Neuroscience Nursing, 18,* 256–264.

Butcher, J. (2000). Dominic O'Brien—Master mnemonist. *Lancet, 356,* 836.

de Quervain, D. J., Roozendaal, B., & McGaugh, J. L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature, 394,* 787–790.

Fields, R. D. (2005). Making memories stick. *Scientific American, 292*(2), 74–81.

Frey, U., & Morris, R. G. (1997). Synaptic tagging and long-term potentiation. *Nature, 385,* 533–536.

Gardner, H. (1983). *Frames of mind: The theory of multiple intelligences.* New York: Basic Books.

Gardner, H. (1999). *Intelligence reframed: Multiple intelligences for the 21st century.* New York: Basic Books.

Gazzaniga, M. S. (Ed.). (2004). *The cognitive neurosciences* (3rd ed.). Cambridge, MA: Massachusetts Institute of Technology.

Genoux, D., Haditsch, U., Knobloch, M., Michalon, A., Storm, D., & Mansuy, I. M. (2002). Protein phosphatase 1 is a molecular constraint on learning and memory. *Nature, 418,* 970–975.

Hebb, D. O. (1949). *The organization of behavior: A neuropsychological theory.* New York: Wiley.

Hill, N. M., & Schneider, W. (2006). Brain changes in the development of expertise: Neuroanatomical and neurophysiological evidence about skill-based adaptations. In K. A. Ericsson, N. Charness, P. J. Feltovich, & R. R. Hoffman (Eds.), *The Cambridge handbook of expertise and expert performance* (pp. 653–682). New York: Cambridge University Press.

Hodgson, R. A., Standish, Z. J., Boyd-Hodgson, T. E., Henderson, A. K., & Racine, R. J. (2004). Training-induced and electrically induced potentiation in the neocortex. *Neurobiology of Learning and Memory, 83,* 22–32.

Iacoboni, M., Molnar-Szakacs, I., Gallese, V., Buccino, G., Mazziotta, J. C., & Rizzolatti, G. (2005). Grasping the intentions of others with one's own mirror neuron system. *PLoS Biology, 3*(3), 529–535.

Kandel, E. R., & Hawkins, R. D. (1995). Neuronal plasticity and learning. In R. D. Broadwell (Ed.), *Decade of the brain: Vol. 1. Neuroscience, memory, and language* (pp. 45–58). Washington, DC: U.S. Government Printing Office.

Kasper, D. L., Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (2005). *Harrison's principles of internal medicine* (16th ed.). New York: McGraw-Hill.

Klein, P. D. (1997). Multiplying the problems of intelligence by eight: A critique of Gardner's theory. *Canadian Journal of Education, 22,* 377–394.
Klein, R. M. (1999). The Hebb legacy. *Canadian Journal of Experimental Psychology, 53,* 1–3.
Kolb, B., & Whishaw, I. Q. (2003). *Fundamentals of human neuropsychology* (5th ed.). New York: Worth.
Kornhaber, M. L. (2004). Multiple intelligences: From the ivory tower to the dusty classroom—But why? *Teachers College Record, 106,* 67–76.
Lupien, S. J., & McEwen, B. S. (1997). The acute effects of corticosteroids on cognition: Integration of animal and human model studies. *Brain Research Reviews, 24,* 1–27.
Maguire, E. A., Valentine, E. R., Wilding, J. M., & Kapur, N. (2003). Routes to remembering: The brains behind superior memory. *Nature Neuroscience, 6*(1), 90–95.
Miyashita, Y. (2004). Cognitive memory: Cellular and network machineries and their top-down control. *Science, 306,* 435–440.
Parkin, A. J. (1996). H.M.: The medial temporal lobes and memory. In C. Code, C. W. Wallesch, Y. Joanette, & A. R. Lecours (Eds.), *Classic cases in neuropsychology* (pp. 337–347). Hove, East Sussex, UK: Psychology Press.
Rizzolatti, R., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27,* 169–192.
Silva, A. J., Kogan, J. H., Frankland, P. W., & Kida, S. (1998). CREB and memory. *Annual Review of Neuroscience, 21,* 127–148.
Squire, L. R. (2004). Memory systems of the brain: A brief history and current perspective. *Neurobiology of Learning and Memory, 82,* 171–177.

Continuing Education Credit

CE

The *Journal of Neuroscience Nursing* is pleased to offer the opportunity to earn neuroscience nursing CE for this article online. Go to www.aann.org, and select "Continuing Education." There you can read the article again or go directly to the posttest assessment. The cost is $15 for each article. You will be asked for a credit card or online payment service number.

The posttest consists of 10 questions based on the article, plus several assessment questions (e.g., How long did it take you to read the article and complete the posttest?). A passing score of 80% (8 of 10 questions correct) on the posttest and completion of the assessment questions yields 1 hour of continuing education in neuroscience nursing for each article.

The American Association of Neuroscience Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

Acute Ischemic Stroke Review

continued from page 293

Holloway, R. G., Vickrey, B. G., Benesch, C., Hinchey, J., & Bieber, J. (2001). Development of performance measures for acute ischemic stroke. *Stroke, 32*(9), 2058–2074.
Joshipura, K. J., Hung, H., Rimm, E., Willett, W., & Ascherio, A. (2003). Periodontal disease, tooth loss, and incidence of ischemic stroke. *Stroke, 34*(1), 47–52.
Kongable, G. (1997). Code Stroke: Using t-pa to prevent ischemic brain injury. *American Journal of Nursing, 97*(11), 16bb–16hh.
LaMonte, M., Bahouth, M. N., Hu, P., Pathan, M. Y., Yarbrough, K. L., Gunawardane, R., et al. (2003). Telemedicine for acute stroke triumphs and pitfalls. *Stroke, 34*(3), 725–728.
Lees, K. R., Zivin, J. A., Ashwood, T., Davalos, A., Davis, S. M., Diener, H. C., et al. (2006). NXY-059 for acute ischemic stroke. *New England Journal of Medicine, 354*(6), 588–600.
Miller, E. T., & Spilker, J. (2003). Readiness to change and brief educational interventions: Successful strategies to reduce stroke risk. *Journal of Neuroscience Nursing, 35*(4), 215–222.
Moore, K. (2001). Managing TIA: A nursing perspective. *Transient ischemic attack (TIA) disease management guide*. Montvale, NJ: Medical Economics Company.
Muir, K. W., Buchan, A. M., von Kummer, R., Rother, J., & Baron, J. C. (2006). Imaging of acute stroke. *Lancet Neurology, 5*, 744–768.
National Institutes of Health. (2003). *The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7)*. Department of Health and Human Services. Retrieved November 21, 2003, from www.nhlbi.nih.gov/guidelines/hypertension/.
Rapp, K., Bratina, P., Barch, C., Braimah, J., Daley, S., Donnarumma, R., et al. (1997) Code Stroke: Rapid transport, triage and treatment using rt-PA therapy. The NINDS rt-PA Stroke Study Group. *Journal of Neuroscience Nursing, 29*(6), 361–366.
Ribo, M., Molina, C., Montaner, J., Rubiera, M., Delgado-Mederos, R., Arenillas, J. F., et al. (2005). Acute hyperglycemia state is associated with lower tPA-induced recanalization rates in stroke patients. *Stroke, 36*(8), 1705–1709.
Rice, V. H. (2006). Nursing intervention and smoking cessation: Meta-analysis update. *Heart and Lung*, *35*(3) 147–163.
Richardson, J., Murray, D., House, K., & Lewenkopf, T. (2006). Successful implementation of the National Institutes of Health Stroke Scale on a stroke/neurovascular unit. *Journal of Neuroscience Nursing, 38*(Suppl. 4), 309–314.
Rordorf, G., Koroshetz, W. J., Ezzeddine, M. A., Segal, A. Z., & Buonanno, F. S. (2001). A pilot study of drug-induced hypertension for treatment of acute stroke. *Neurology, 56*(9), 1210–1213.
Sacco, R. L., Adams, R., Albers, G., Alberts, M. J., Benavente, O., Furie, K., et al. (2006). Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack. *Stroke, 37*(2), 577–617.
Sahni, R. (2000). Acute stroke: Implications for prehospital care. *Prehospital Emergency Care, 4*(3), 270–272.
Thom, T., Haase, N., Rosamond, W., Howard, V. J., Rumsfeld, J., Manolio, T., et al. (2006). Heart disease and stroke statistics—2006 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 113*(6), e85–151.
Tietgen, G. E. (2005). The risk of stroke in patients with migraine and implications for migraine management. *CNS Drugs, 19*(8), 683–692.
Tirschwell, D. L., Longstreth, W. T., Becker, K. J., Gammans, R. E., Saboujian, L. A., Hamilton, S., et al. (2002). Shortening the NIH stroke scale for use in the prehospital setting. *Stroke, 33*(12), 2801–2806.
Townend, B. S., Hanson, J. A., Sturm, J. W., & Whyte, S. (2005). Stroke or encephalitis? *Emergency Medicine Australasia, 17*(4), 401–404.
Warlow, C., Dennis, M., van Gign, J., Hankey, G. J., Sandercock, P., Bamford, J., et al. (2001). *Stroke: A practical guide to management* (2nd ed.). London: Blackwell Science.

Continuing Education Credit

CE

The *Journal of Neuroscience Nursing* is pleased to offer the opportunity to earn neuroscience nursing CE for this article online. Go to www.aann.org, and select "Continuing Education." There you can read the article again or go directly to the posttest assessment. The cost is $15 for each article. You will be asked for a credit card or online payment service number.

The posttest consists of 10 questions based on the article, plus several assessment questions (e.g., How long did it take you to read the article and complete the posttest?). A passing score of 80% (8 of 10 questions correct) on the posttest and completion of the assessment questions yields 1 hour of continuing education in neuroscience nursing for each article.

The American Association of Neuroscience Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.