# Veterans Affairs/Department of Defense Clinical Practice Guideline for the Management of Adult Stroke Rehabilitation Care

## Executive Summary

Barbara Bates, MD; John Y. Choi, MD; Pamela W. Duncan, PhD, FAPTA; Jonathan J. Glasberg, MA, PT; Glenn D. Graham, MD, PhD; Richard C. Katz, PhD; Kerri Lamberty, PhD; Dean Reker, PhD; Richard Zorowitz, MD

***Background***—A panel of experts developed stroke rehabilitation guidelines for the Veterans Health Administration and Department of Defense Medical Systems.

***Methods***—Starting from previously established guidelines, the panel evaluated published literature through 2002, using criteria developed by the US Preventive Services Task Force. Recommendations were based on evidence from randomized clinical trials, uncontrolled studies, or consensus expert opinion if definitive data were lacking.

***Results***—Recommendations with Level I evidence include the delivery of poststroke care in a multidisciplinary rehabilitation setting or stroke unit, early patient assessment via the NIH Stroke Scale, early initiation of rehabilitation therapies, swallow screening testing for dysphagia, an active secondary stroke prevention program, and proactive prevention of venous thrombi. Standardized assessment tools should be used to develop a comprehensive treatment plan appropriate to each patient's deficits and needs. Medical therapy for depression or emotional lability is strongly recommended. A speech and language pathologist should evaluate communication and related cognitive disorders and provide treatment when indicated. The patient, caregiver, and family are essential members of the rehabilitation team and should be involved in all phases of the rehabilitation process. These recommendations are available in their entirety at http://stroke.ahajournals.org/cgi/content/full/36/9/e100. Evidence tables for each of the recommendations are also in the full document.

***Conclusions***—These recommendations should be equally applicable to stroke patients receiving rehabilitation in all medical system settings and are not based on clinical problems or resources unique to the Federal Medical System. **(*Stroke.* 2005;36:2049-2056.)**

**Key Words:** AHA/ASA-Endorsed Practice Guidelines ■ stroke ■ rehabilitation ■ quality of life

The Management of Stroke Rehabilitation Guideline was developed by clinical experts from the Department of Defense, Veterans Health Administration, and academia. The effort drew heavily from the Agency for Health Care Policy and Research Guidelines for Post-Stroke Rehabilitation (1995),[1] the Royal College of Physicians National Clinical Guidelines for Stroke (2000),[2] and the Scottish Intercollegiate Guidelines Network Management of Patients with Stroke (1998).[3]

The panel evaluated the medical evidence for each question according to criteria proposed by the US Preventive Services Task Force.[4] Where evidence was ambiguous or scientific data were lacking, the multidisciplinary group panel developed consensus-based recommendations. The strength of the recommendations made was based on the quality of the evidence, the overall quality of the study, and the net effect of the intervention. The quality of the evidence was determined by the type of study, with the highest rating given to randomized controlled trials and the lowest rating to consensus statements. Overall quality was rated as "good," "fair," or "poor" according to both the quality of the evidence and whether it was directly linked to health outcomes. Final recommendations were graded as follows:

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

The full-text version of this guideline is available online at http://stroke.ahajournals.org/cgi/content/full/36/9/e100 (*Stroke*. 2005;36:e100–e143).

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on April 8, 2005. A single reprint is available by calling 800-242-8721 (US only) or writing the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596. Ask for reprint No. 71-0330. To purchase additional reprints: up to 999 copies, call 800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 410-528-4121, fax 410-528-4264, or e-mail kgray@lww.com. To make photocopies for personal or educational use, call the Copyright Clearance Center, 978-750-8400.



***Stroke* is available at http://www.strokeaha.org** **DOI: 10.1161/01.STR.0000180432.73724.AD**

**A:** A strong recommendation that the intervention is always indicated and acceptable
**B:** A recommendation that the intervention may be useful/effective
**C:** A recommendation that the intervention may be considered
**D:** A recommendation that a procedure may be considered not useful/effective or may be harmful
**I:** Insufficient evidence to recommend for or against—the clinician will use clinical judgment

The full guideline can be found at http://stroke.ahajournals.org/cgi/content/full/36/9/e100.

New information and additional references were added during the development of this executive summary and are noted in *[bracketed italicized text].*

## Background

Stroke is a leading cause of disability in the United States.[5] Forty percent of stroke patients are left with moderate functional impairment and 15% to 30% with severe disability. Effective rehabilitation interventions initiated early after stroke can enhance the recovery process and minimize functional disability. Improved functional outcomes for patients also contribute to patient satisfaction and can reduce potential costly long-term care expenditures. Substantial evidence indicates that patients do better with a well-organized, multidisciplinary approach to post–acute stroke care[6–8] (Evidence Level=A). Duncan and colleagues[9] found that greater adherence to post–acute stroke rehabilitation guidelines was associated with improved patient outcomes and concluded, "compliance with guidelines may be viewed as a quality of care indicator with which to evaluate new organizational and funding changes involving post–acute stroke rehabilitation."

Stroke rehabilitation begins during the acute hospitalization, as soon as the diagnosis of stroke is established and life-threatening problems are controlled. The highest priorities of early stroke rehabilitation are to prevent recurrence of stroke, manage comorbidities, and prevent complications (Evidence Level=C). Comprehensive assessment of patients is necessary for clinical management and evaluation of outcomes for quality control and research.[10] The use of validated, standardized instruments ensures reliable documentation of neurological conditions, levels of disability, functional independence, family support, quality of life, and progress over time[1] (Evidence Level=A).

## Initiation of Secondary Stroke Prevention

After a stroke, patients are at increased risk for additional cerebrovascular events. Risk factor reduction must be an integral part of stroke rehabilitation and recovery. The need for secondary prevention of stroke is lifelong and is a critical component of rehabilitation, with clear data available on hypertension treatment, warfarin use in atrial fibrillation, and antiplatelet therapy use in cerebral ischemia[11–28] (Evidence Level=A).

Evidence-based recommendations with regard to selection and implementation of secondary prevention therapies have been issued previously by the American Heart Association/American Stroke Association (AHA/ASA).[29] These guidelines are currently being updated by the recently constituted AHA/ASA Stroke Council's Standing Committee on Secondary Stroke Prevention.

Table 1 highlights the key points of the guideline.

**TABLE 1. Key Points**

- The primary goal of rehabilitation is to **prevent complications, minimize impairments, and maximize function.**
- Secondary prevention is fundamental to **preventing stroke recurrence.**
- **Early** assessment and intervention are critical to optimize rehabilitation.
- **Standardized** evaluations and valid assessment tools are essential to the development of a comprehensive treatment plan.
- Evidence-based interventions should be based on **functional goals.**
- Every candidate for rehabilitation should have access to an **experienced and coordinated** rehabilitation team to ensure optimal outcome.
- **The patients and family and/or caregiver** are essential members of the rehabilitation team.
- **Patient and family education** improves the likelihood of informed decision making, social adjustment, and maintenance of rehabilitation gains.
- The **rehabilitation** team should utilize **community resources for community reintegration.**
- **Ongoing** medical management of risk factors and comorbidities is essential to ensure survival.

## Prevention of Complications

### Deep Venous Thrombosis

See Table 2.[30–36]

### Pressure Ulcers

Risk factors for skin breakdown include dependence in mobility, diabetes, peripheral vascular disease, urinary incontinence, and lower body mass index.[37,38] A thorough assessment of skin integrity should be done on admission and daily checks performed thereafter (Evidence Level=C). Positioning, turning, and transferring techniques, and barrier sprays, lubricants, special mattresses, and protective dressings and

**TABLE 2. Prevention of Deep Venous Thrombosis**

| |
|---|
| **In ischemic stroke** |
| Early mobilization, walking at least 50 feet daily, should always be considered.[30] |
| Subcutaneous low-dose unfractionated heparin (LDUH) (5000 units BID) is an option.[31] |
| Low-molecular-weight heparin and heparinoid products may be better than LDUH, according to limited randomized trials.[32–34] These drugs are alternatives to LDUH. |
| Intermittent pneumatic compression devices and compression stockings should be used in conjunction with above interventions.[35] |
| [*Alternative anticoagulation such as argatroban or lepirudin should be used in acute heparin-induced thrombocytopenia.*[36]] |
| **In hemorrhagic stroke** |
| No studies are available. Acutely, anticoagulation is avoided and nonpharmacological measures are emphasized. |
| Mechanical filter devices can be considered when a deep venous thrombosis is evident. |

padding should be used to avoid skin injury[39] (Evidence Level=C).

### Bowel and Bladder Function

Fifty percent of stroke patients have incontinence during their acute admission, decreasing to 20% by 6 months after stroke.[40] Incontinence is a major burden on caregivers, and management of bladder and bowel problems is essential. Acute use of an indwelling catheter may facilitate fluid management, prevent urinary retention, and reduce skin breakdown in patients with stroke. However, use of a Foley catheter longer than 48 hours after stroke increases the risk of urinary tract infection and should be limited to situations that cannot be managed any other way (Evidence Level=B).

Constipation and fecal impaction are more common after stroke than bowel incontinence. Goals of management are to ensure adequate intake of fluid, bulk, and fiber and to help the patient establish a regular toileting schedule. Bowel training is more effective if the schedule is consistent with the patient's previous bowel habits.[41] Stool softeners and judicious use of laxatives may be helpful (Evidence Level=I).

### Dysphagia

Dysphagia occurs in 45% of all hospitalized stroke patients and can lead to worse outcomes, including aspiration pneumonia and death. Malnutrition is present in 15% of patients admitted to the hospital, and this percentage doubles during the first week after stroke.[2] A bedside swallow screening should be completed before oral intake (Evidence Level=B). If the patient's swallow screening is abnormal, a complete bedside swallow examination is recommended (Evidence Level=I).

### Detailed Rehabilitation Assessments

Early and thorough assessments of a patient's cognition, communication skills, physical functioning, and psychosocial history and resources are needed to identify current abilities, set realistic goals, and guide treatment planning and interventions (Evidence Level=B).

## Provision of Rehabilitation Services

Although the literature is clear that organized services are a dominant component of stroke rehabilitation, it is not possible to specify precise standards and protocols for types of specialized services needed. In general, evidence suggests that better clinical outcomes are achieved when post–acute stroke patients receive coordinated, multidisciplinary evaluation and intervention[6–8,42–49] (Evidence Level=A). The multidisciplinary care team may consist of a physician, nurse, physical therapist, occupational therapist, kinesiotherapist, speech and language pathologist, psychologist, recreational therapist, patient, and family members/caregivers. If an organized rehabilitation team is not available in the facility, patients with moderate or severe symptoms should be offered a referral to a facility with such a service (Evidence Level=I).

The most common care settings for rehabilitation services are inpatient rehabilitation facilities, nursing homes, outpatient therapy clinics, and home care. No study has demonstrated the superiority of one type of rehabilitation setting over another. The decision to provide rehabilitation services in an inpatient setting, either in the general inpatient ward, rehabilitation unit, or long-term care unit, is based on the patient's needs and availability of resources. The rehabilitation program should be guided by specific goals developed in consensus with the patient, family, and rehabilitation team. The patient's family/caregiver should participate in the decision-making process as well as the rehabilitation sessions and should be trained to assist the patient with functional activities when needed (Evidence Level=I).

## Intensity/Duration of Therapy

Studies support early mobilization of the patient with an acute stroke to prevent complications. Progressive activity should be provided as soon as medically tolerated (Evidence Level=A).

Two meta-analyses found that greater intensity of services produces better outcomes. Langhorne et al[50] concluded, "More intensive physiotherapy input was associated with a reduction in the combined poor outcome of death or deterioration and may enhance the rate of recovery." Kwakkel et al[51] reported a small but statistically significant intensity–effect relationship in the rehabilitation of stroke patients. Cifu and Stewart[6] concluded that greater intensity of therapy services has "a weak relationship with improved functional outcome." Because of the heterogeneity of the studies, however, no specific recommendations about intensity or duration of treatment can be made (Evidence Level=B).

## Patient and Family/Caregiver Education

Education should be provided to patients and families/caregivers in an interactive and written format (Evidence Level=B). Clinical teams should consider identifying a specific team member to be responsible for providing information to the patient and family/caregiver about the nature of the stroke, stroke management rehabilitation and outcome expectations, and their roles in the rehabilitation process (Evidence Level=C). Issues such as need for 24-hour supervision, home environment safety or equipment needs, and driving safety concerns need to be discussed. Younger stroke patients may require consideration of vocational needs as part of their rehabilitation programs. Addressing sexual issues is important given that many poststroke medications may impair this quality-of-life issue, but little research is available to make firm recommendations at this time. Some clinicians use general recommendations similar to those given to post–myocardial ischemic patients with regard to resuming sexual activities.

## Specific Rehabilitation Interventions

### Dysphagia

Dysphagia treatment is effective[33,52] and may involve compensatory strategies such as posture changes, heightening sensory input, swallow maneuvers, active exercise programs, or diet modifications. Dysphagia management may include nonoral feeding and psychological support. At this time, it is unclear how dysphagic patients should be fed after acute stroke.[53] The literature supports the use of tube feeding for patients who cannot sustain sufficient oral caloric and/or fluid

intake to meet nutritional needs. Limited evidence suggests that percutaneous endoscopic gastrostomy feeding compares favorably with nasogastric tube feeding[54] (Evidence Level=B).

## Communication

Disorders of communication and related cognitive impairments occur in as many as 40% of poststroke patients. The most common communication disorders occurring after stroke are aphasia and dysarthria. Aphasia treatment can result in maximizing gains during the period of spontaneous recovery and developing compensatory strategies (including modification of the environment) during the chronic phase. Depending on the pathology and site of lesion, dysarthria for some patients may decrease dramatically, whereas for others, dysarthria persists and requires direct intervention of the affected subsystem (articulation, resonance, phonation, respiration, or prosody), development of compensatory behaviors, or training in the use of augmentative/alternative communication devices. If intervention is indicated, treatment can help maximize recovery of communication abilities and prevent learning of ineffective or inappropriate compensatory behaviors[55–61] (Evidence Level=A, B).

## Motor Functioning

Muscle weakness is common after stroke. Lower-extremity strength has been correlated with gait speed in stroke patients.[62] Additionally, lower-extremity muscle strength on admission to rehabilitation is a predictor of function at discharge[63] and also has been inversely correlated with risk of falling in elderly individuals. Strengthening should be included in the acute rehabilitation of patients with weakness after stroke (Evidence Level=I).

More than one half of stroke patients who survive the acute phase of stroke are not able to walk and will require a period of rehabilitation to achieve a functional level of ambulation.[64] A recently proposed gait-training strategy involves unloading the lower extremities by supporting a percentage of body weight to facilitate walking. One subsequent trial found equally beneficial results from a program that included aggressive bracing and assisted walking.[65] It is recommended that treadmill training with partial body weight support may be used as an adjunct to conventional therapy in patients with mild-to-moderate dysfunction resulting in impaired gait (Evidence Level=B).

Persistent loss of upper-extremity function is common among patients with substantial motor function loss after a stroke. One approach aimed at resolving upper-extremity dysfunction has been termed constraint-induced movement therapy, which involves forced use of the involved upper extremity and discourages the use of the unaffected extremity. One study showed a trend toward improved function with the use of constraint-induced movement therapy; however, conclusions are difficult to draw because of the small sample size and significant demographic differences between the study groups.[66] Use of constraint-induced therapy should be considered for a select group of patients—that is, patients with 20 degrees of wrist extension and 10 degrees of finger extension, who have no sensory and cognitive deficits (Evidence Level=C).

Functional electrical stimulation (FES) has been used as a therapeutic modality for poststroke patients but has not been a routine standard of care. There is evidence of short-term increases in motor strength and motor control and a reduction in impairment severity, but there is no evidence of an increase in the patient's level of function.[67–69] It is recommended that FES be used for patients who have demonstrated ankle/knee/wrist motor impairment, for patients who have shoulder subluxation, and for gait training after stroke (Evidence Level=B).

## Spasticity

Patients with paretic limbs and muscle spasticity are at high risk of developing contractures that restrict movement, cause pain, and adversely affect skin hygiene. Early treatment is key to preventing disabling complications. Spasticity is typically treated in a stepwise approach, beginning with noninvasive and progressing to more invasive modalities. Positioning, passive stretching, and range-of-motion exercise may provide relief and should be done several times daily in persons with spasticity.[1,2] Corrective measures for contractures that interfere with function include splinting, serial casting, and surgical correction (Evidence Level=C).

Oral drug agents such as tizanidine,[70] dantrolene,[71] and oral baclofen[72] can be used to treat spasticity that results in pain, poor skin hygiene, or decreased function. Tizanidine should be used specifically for chronic stroke patients (Evidence Level=B).

Evidence of functional gains with these agents is scant, and they may produce significant cognitive or other side effects.[73,74] Diazepam or other benzodiazepines are not recommended during the stroke recovery period because of possible deleterious effects on recovery[75–77] (Evidence Level=D).

Use of botulinum toxin,[78–80] phenol/alcohol neurolysis[81–83] (Evidence Level=B), or intrathecal baclofen[84] (Evidence Level=C) should be considered for selected patients with disabling or painful spasticity. Neurosurgical procedures, such as selective dorsal rhizotomy or dorsal root entry zone lesion, may be considered for spasticity, but they lack clinical trial evidence and carry significant risks such as unintended spinal cord damage (Evidence Level=I).

## Shoulder Pain

Shoulder pain is a common problem after stroke. As many as 72% of stroke patients will experience at least one episode of shoulder pain during the first year.[85] There are several causes of poststroke shoulder pain, including adhesive capsulitis, traction/compression neuropathy, complex regional pain syndrome, shoulder trauma, bursitis/tendonitis, rotator cuff tear, and heterotrophic ossification. Shoulder pain can delay rehabilitation and functional recuperation because the painful joint may mask improvement of motor function or may inhibit rehabilitation. The incidence of shoulder-hand pain syndrome has been reported to be as high as 67% in patients with a combination of motor, sensory, and visual-perceptual deficits.[30] A variety of treatment interventions could be considered for the treatment of poststroke shoulder pain,

including avoiding the use of overhead pulleys (which encourage uncontrolled abduction), staff education to prevent trauma to the hemiplegic shoulder, intra-articular steroid injections, shoulder strapping, stretching and mobilization techniques, ice, heat, soft tissue massage, FES, and shoulder girdle strengthening (Evidence Levels=B, C).

### Psychological

Cognitive deficits after stroke are very common. Although there are anecdotal and large case studies supporting the benefits of cognitive remediation, evidence-based research is lacking and the most research has been in the traumatic brain injury population. The data support a thorough assessment of cognitive functioning as well as treatment of patients with several areas of cognitive impairment via multiple disciplines.[86–91] Teaching compensatory strategies for memory deficits, in particular, may be beneficial[87,92] (Evidence Level=B).

Visual and spatial neglect may occur in patients with nondominant cortical stroke and is a significant contributor to poor prognosis after stroke because it impacts the patient's ability to function safely within the environment. The literature does not suggest any single intervention for addressing neglect, although a multifaceted approach with a strong educational component can help patients adapt to these deficits[93–98] (Evidence Level=B).

A variety of neuropsychiatric sequelae are seen after stroke, with depression in particular being common and underdiagnosed.[99] Anxiety and pathological affect are also seen. The assessment of emotional disorders can be difficult because of aphasia, flat affect, aprosodic speech, and the lack of standardized instruments. Therefore, observation of behavior and utilization of input from family and staff can aid in diagnosing neuropsychiatric disorders.[100] In many instances, it is necessary to call on the expertise of a psychologist or psychiatrist with background in assessment of patients with cognitive and communication disorders.

Once diagnosed, treatment of depression and other emotional disorders can greatly improve rehabilitation outcomes[101,102] (Evidence Level=A). Treatment with psychotherapy (Evidence Level=C) and/or pharmacotherapy (Evidence Level=A) can stabilize mood and improve ability to participate in therapies. Selective serotonin reuptake inhibitors and tricyclic antidepressant medications have been shown to be beneficial for treatment of depression and pathological affect. Given the high incidence of anticholinergic effects with tricyclic medications in older patients, selective serotonin reuptake inhibitors are the preferred agents.[2,103–111]

### Use of Pharmacological Agents

While undergoing rehabilitation, stroke patients frequently receive a variety of medications to treat complications of stroke or other unrelated chronic medical conditions. We do not recommend amphetamine use for motor recovery, on the basis of negative large amphetamine clinical stroke recovery trials[112–114] and the lack of documented long-term benefits. Limited data support the use of other neurotransmitter-releasing agents to promote stroke recovery, including methylphenidate,[115] levodopa,[116] and L-threo-3,4-dihydroxyphenyl serine (L-DOPS).[117] Fluoxetine in nondepressed patients appeared to have a small benefit in motor recovery independent of the treatment of depression.[118] Modafinil, a novel stimulant, is selectively used in stroke patients but without proven safety or efficacy. Current data do not permit discrimination among these agents, identification of optimal dosing, or selection of the preferred time of initiation of pharmacotherapy after stroke or the duration of treatment (Evidence Level=B).

Limited data suggest that central nervous system depressants such as neuroleptics, barbiturates, benzodiazepines, and anticonvulsants are associated with poorer outcomes.[78–80] Centrally acting $\alpha_2$-adrenergic receptor agonists and $\alpha_1$-receptor antagonists such as clonidine and prazosin have been associated with poorer recovery in studies in animals.[79] *[Notably, benzodiazepines have been demonstrated to cause reoccurrence of stroke symptoms in transient ischemic attack patients.*[119]*]* Clinicians should limit the use of these medications in patients recovering from stroke as much as is practical (Evidence Level=D). Atypical neuroleptics may be safer to use when necessary for behavioral control in stroke patients but are only available in oral form (Evidence Level=C).

**TABLE 3. Late-Phase Needs Assessments**

| | |
|---|---|
| 1. | Ongoing management of stroke risk factors and comorbid disease |
| 2. | Participation in a regular exercise program |
| 3. | Adaptive devices for activities of daily living |
| 4. | Lower-extremity orthoses and walking aids |
| 5. | Wheelchair |
| 6. | Vocation assessment and return to work |
| 7. | Driving |
| 8. | Sexuality |

### Late Phase

The majority of patients who have had a stroke will be managed initially in a hospital. Discharge from inpatient care to home (or to residential living or a nursing facility) constitutes an important watershed. Living with disabilities after a stroke is a lifelong challenge during which people continue to seek and find ways to compensate for or adapt to persisting neurological deficits. For many, the real work of recovery begins after formal rehabilitation when the patient attempts to use newly learned skills without the support of the rehabilitation environment or team. Adequate support from family and caregivers is critical to a successful outcome. It is also important to assure that all necessary equipment and support services are in place (Evidence Level=I). See Table 3 for late-phase needs assessments.

## Summary

The clinical practice guidelines described above represent the best efforts of a joint task force from the Department of Veterans Affairs and the US Department of Defense to provide evidence-based medical care for patients in need of stroke rehabilitation. In addition to providing state-of-the-art direction for clinicians, the guidelines can also help research-

ers to identify areas for further investigation. In turn, this research can result in more effective procedures and more efficient technology.

## References


1. Gresham GE, Duncan PW, Stason WB, et al. *Post-Stroke Rehabilitation*. Clinical Practice Guideline, No. 16. Rockville, Md: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; May 1995. AHCPR Publication No. 95-0662.
2. Royal College of Physicians. *National Clinical Guidelines for Stroke*. 2nd ed. Prepared by the Intercollegiate Stroke Working Party. London: Royal College of Physicians; 2004. Available at: http://www.rcplondon.ac.uk/pubs/books/stroke/index.htm. Accessed July 20, 2005.
3. Scottish Intercollegiate Guidelines Network (SIGN). *Management of Patients with Stroke Part III: Identification and Management of Dysphagia, No. 20*. Edinburgh, Scotland: Scottish Intercollegiate Guidelines Network; 1997. [Superseded by *Management of Patients with Stroke: Identification and Management of Dysphagia, No. 78*; 2004.]
4. United States Preventive Service Task Force (USPSTF). *Guide to Clinical Preventive Services.* 2nd ed. Baltimore, Md: Williams and Wilkins; 1996.
5. American Heart Association. *Heart and Stroke Statistical Update—2000*. Dallas, Tex: American Heart Association; 1999.
6. Cifu DX, Stewart DG. Factors affecting functional outcome after stroke: a critical review of rehabilitation interventions. *Arch Phys Med Rehabil*. 1999;80(5 suppl 1):S35–S39.
7. Stroke Unit Trialists' Collaboration. Organised inpatient (stroke unit) care for stroke. *Cochrane Database Syst Rev.* 2002(1):CD000197.
8. Evans RL, Connis RT, Hendricks RD, Haselkorn JK. Multidisciplinary rehabilitation versus medical care: a meta-analysis. *Soc Sci Med*. 1995; 40:1699–1706.
9. Duncan PW, Horner RD, Reker DM, Samsa GP, Hoenig H, Hamilton B, LaClair BJ, Dudley TK. Adherence to postacute rehabilitation guidelines is associated with functional recovery in stroke. *Stroke*. 2002; 33:167–177.
10. Duncan PW, Lai SM, van Culin V, Huang L, Clausen D, Wallace D. Development of a comprehensive assessment toolbox for stroke. *Clin Geriatr Med*. 1999;15:885–915.
11. Barnett HJ, Taylor DW, Eliasziw M, Fox AJ, Ferguson GG, Haynes RB, Rankin RN, Clagett GP, Hachinski VC, Sackett DL, Thorpe KE, Meldrum HE, Spence JD. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. *N Engl J Med*. 1998;339:1415–1425.
12. Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P. Antithrombotic and thrombolytic therapy for ischemic stroke. *Chest*. 2001;119(1 suppl): 300S–320S.
13. Mohr JP, Thompson JL, Lazar RM, Levin B, Sacco RL, Furie KL, Kistler JP, Albers GW, Pettigrew LC, Adams HP Jr, Jackson CM, Pullicino P; Warfarin-Aspirin Recurrent Stroke Study Group. A comparison of warfarin and aspirin for the prevention of recurrent ischemic stroke. *N Engl J Med*. 2001;345:1444–1451.
14. Albers GW. Choice of endpoints in antiplatelet trials: which outcomes are most relevant to stroke patients? *Neurology*. 2000;54:1022–1028.
15. Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P. Antithrombotic and thrombolytic therapy for ischemic stroke: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest*. 2004; 126(suppl 3):483S–512S.
16. Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, Ray JG. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest*. 2004; 126(suppl 3):338S–400S.
17. Prognosis of patients with symptomatic vertebral or basilar artery stenosis. The Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) Study Group. *Stroke*. 1998;29:1389–1392.
18. European Stroke Prevention Study. ESPS Group. *Stroke*. 1990;21: 1122–1130.
19. Bellavance A. Efficacy of ticlopidine and aspirin for prevention of reversible cerebrovascular ischemic events. The Ticlopidine Aspirin Stroke Study. *Stroke*. 1993;24:1452–1457.
20. Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, Dagenais G. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. *N Engl J Med*. 2000;342:145–153. [Errata in *N Engl J Med*. 2000;342:1376; *N Engl J Med*. 2000;342:748.]
21. Yusuf S, Zhao F, Mehta SR, Chrolavicius S, Tognoni G, Fox KK; Clopidogrel in Unstable Angina to Prevent Recurrent Events Trial Investigators. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. *N Engl J Med*. 2001;345:494–502. [Errata in *N Engl J Med*. 2001;345:1716; *N Engl J Med*. 2001;345:1506.]
22. Dahlof B, Devereux RB, Kjeldsen SE, Julius S, Beevers G, de Faire U, Fyhrquist F, Ibsen H, Kristiansson K, Lederballe-Pedersen O, Lindholm LH, Nieminen MS, Omvik P, Oparil S, Wedel H; LIFE Study Group. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. *Lancet*. 2002;359:995–1003.
23. PROGRESS Collaborative Group. Randomised trial of a perindopril-based blood-pressure–lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. *Lancet*. 2001; 358:1033–1041. [Errata in: *Lancet*. 2001;358:1556; *Lancet*. 2002;359: 2120.]
24. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet*. 1994;344:1383–1389.
25. Sacks FM, Pfeffer MA, Moye LA, Rouleau JL, Rutherford JD, Cole TG, Brown L, Warnica JW, Arnold JM, Wun CC, Davis BR, Braunwald E. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial investigators. *N Engl J Med*. 1996;335:1001–1009.
26. Hunt D, Young P, Simes J, Hague W, Mann S, Owensby D, Lane G, Tonkin A. Benefits of pravastatin on cardiovascular events and mortality in older patients with coronary heart disease are equal to or exceed those seen in younger patients: results from the LIPID trial. *Ann Intern Med*. 2001;134:931–940.
27. Schwartz GG, Olsson AG, Ezekowitz MD, Ganz P, Oliver MF, Waters D, Zeiher A, Chaitman BR, Leslie S, Stern T; Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) Study Investigators. Effects of atorvastatin on early recurrent ischemic events in acute coronary syndromes: the MIRACL study: a randomized controlled trial. *JAMA*. 2001;285:1711–1718.
28. Goldstein LB, Adams R, Becker K, Furberg CD, Gorelick PB, Hademenos G, Hill M, Howard G, Howard VJ, Jacobs B, Levine SR, Mosca L, Sacco RL, Sherman DG, Wolf PA, del Zoppo GJ. Primary prevention of ischemic stroke: a statement for healthcare professionals from the Stroke Council of the American Heart Association. *Circulation*. 2001;103:163–182.
29. Wolf PA, Clagett GP, Easton JD, Goldstein LB, Gorelick PB, Kelly-Hayes M, Sacco RL, Whisnant JP. Preventing ischemic stroke in patients with prior stroke and transient ischemic attack: a statement for healthcare professionals from the Stroke Council of the American Heart Association. *Stroke*. 1999;30:1991–1994.
30. Reding MJ, Potes E. Rehabilitation outcome following initial unilateral hemispheric stroke: life table analysis approach. *Stroke*. 1988;19: 1354–1358.
31. The International Stroke Trial (IST): a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. International Stroke Trial Collaborative Group. *Lancet*. 1997;349:1569–1581.
32. Bath PM, Iddenden R, Bath FJ. Low-molecular-weight heparins and heparinoids in acute ischemic stroke: a meta-analysis of randomized controlled trials. *Stroke*. 2000;31:1770–1778.
33. Bath PM, Bath FJ, Smithard DG. Interventions for dysphagia in acute stroke. *Cochrane Database Syst Rev*. 2000;(2):CD000323.
34. Bijsterveld NR, Hettiarachchi R, Peters R, Prins MH, Levi M, Buller HR. Low-molecular weight heparins in venous and arterial thrombotic disease. *Thromb Haemost*. 1999;82 suppl 1:139–147.
35. Kamran SI, Downey D, Ruff RL. Pneumatic sequential compression reduces the risk of deep vein thrombosis in stroke patients. *Neurology*. 1998;50:1683–1688.
36. Warkentin TE. Heparin-induced thrombocytopenia: diagnosis and management. *Circulation*. 2004;110:e454–e458.
37. Berlowitz DR, Brandeis GH, Anderson JJ, Ash AS, Kader B, Morris JN, Moskowitz MA. Evaluation of a risk-adjustment model for pressure ulcer development using the Minimum Data Set. *J Am Geriatr Soc*. 2001;49:872–876.
38. Berlowitz DR, Brandeis GH, Morris JN, Ash AS, Anderson JJ, Kader B, Moskowitz MA. Deriving a risk-adjustment model for pressure ulcer

development using the Minimum Data Set. *J Am Geriatr Soc*. 2001;49: 866–871.
39. Sussman C, Bates-Jensen BM, eds. *Wound Care: A Collaborative Practice Manual for Physical Therapists and Nurses.* Gaithersburg, Md: Aspen Publishers; 1998.
40. Nakayama H, Jorgensen HS, Pedersen PM, Raaschou HO, Olsen TS. Prevalence and risk factors of incontinence after stroke. The Copenhagen Stroke Study. *Stroke*. 1997;28:58–62.
41. Venn MR, Taft L, Carpentier B, Applebaugh G. The influence of timing and suppository use on efficiency and effectiveness of bowel training after a stroke. *Rehabil Nurs*. 1992;17:116–120.
42. Evans A, Perez I, Harraf F, Melbourn A, Steadman J, Donaldson N, Kalra L. Can differences in management processes explain different outcomes between stroke unit and stroke-team care? *Lancet*. 2001;358: 1586–1592.
43. Langhorne P, Duncan P. Does the organization of postacute stroke care really matter? *Stroke*. 2001;32:268–274.
44. Early Supported Discharge Trialists. Services for reducing duration of hospital care for acute stroek patients (Cochrane Review). *The Cochrane Library*. Issue 4. Oxford, UK: Update Software; 2001.
45. Rudd AG, Wolfe CD, Tilling K, Beech R. Randomised controlled trial to evaluate early discharge scheme for patients with stroke. *BMJ*. 1997; 315:1039–1044. [Erratum in: *BMJ*. 1998;316:435.]
46. Indredavik B, Slordahl SA, Bakke F, Rokseth R, Haheim LL. Stroke unit treatment. Long-term effects. *Stroke*. 1997;28:1861–1866.
47. Indredavik B, Bakke F, Slordahl SA, Rokseth R, Haheim LL. Stroke unit treatment improves long-term quality of life: a randomized controlled trial. *Stroke*. 1998;29:895–899.
48. Indredavik B, Bakke F, Slordahl SA, Rokseth R, Haheim LL. Stroke unit treatment: 10-year follow-up. *Stroke*. 1999;30:1524–1527.
49. Kalra L, Evans A, Perez I, Knapp M, Donaldson N, Swift CG. Alternative strategies for stroke care: a prospective randomised controlled trial. *Lancet*. 2000;356:894–899.
50. Langhorne P, Wagenaar R, Partridge C. Physiotherapy after stroke: more is better? *Physiother Res Int*. 1996;1:75–88.
51. Kwakkel G, Wagenaar RC, Twisk JW, Lankhorst GJ, Koetsier JC. Intensity of leg and arm training after primary middle-cerebral-artery stroke: a randomised trial. *Lancet*. 1999;354:191–196.
52. Finestone HM, Foley NC, Woodbury MG, Greene-Finestone L. Quantifying fluid intake in dysphagic stroke patients: a preliminary comparison of oral and nonoral strategies. *Arch Phys Med Rehabil*. 2001; 82:1744–1746.
53. Martin-Harris B, Logemann JA, McMahon S, Schleicher M, Sandidge J. Clinical utility of the modified barium swallow. *Dysphagia*. 2000;15: 136–141.
54. Rasley A, Logemann JA, Kahrilas PJ, Rademaker AW, Pauloski BR, Dodds WJ. Prevention of barium aspiration during videofluoroscopic swallowing studies: value of change in posture. *AJR Am J Roentgenol*. 1993;160:1005–1009.
55. Robey RR. The efficacy of treatment for aphasic persons: a meta-analysis. *Brain Lang*. 1994;47:582–608.
56. Robey RR. A meta-analysis of clinical outcomes in the treatment of aphasia. *J Speech Lang Hear Res*. 1998;41:172–187.
57. Whurr R, Lorch MP, Nye C. A meta-analysis of studies carried out between 1946 and 1988 concerned with the efficacy of speech and language therapy treatment for aphasic patients. *Eur J Disord Commun*. 1992;27:1–17.
58. Whurr R, Lorch MP, Nye C. Efficacy of speech and language therapy for aphasia: a meta-analytic review. *Neurol Rev Int*. 1997;1:7–11.
59. Elman RJ, Bernstein-Ellis E. The efficacy of group communication treatment in adults with chronic aphasia. *J Speech Lang Hear Res*. 1999;42:411–419.
60. Katz RC, Wertz RT. The efficacy of computer-provided reading treatment for chronic aphasic adults. *J Speech Lang Hear Res*. 1997;40: 493–507.
61. Wertz RT, Weiss DG, Aten JL, Brookshire RH, Garcia-Bunuel L, Holland AL, Kurtzke JF, LaPointe LL, Milianti FJ, Brannegan R, et al. Comparison of clinic, home, and deferred language treatment for aphasia: a Veterans Administration Cooperative Study. *Arch Neurol*. 1986;43:653–658.
62. Bohannon RW, Walsh S. Nature, reliability, and predictive value of muscle performance measures in patients with hemiparesis following stroke. *Arch Phys Med Rehabil*. 1992;73:721–725.
63. Andrews AW, Bohannon RW. Discharge function and length of stay for patients with stroke are predicted by lower extremity muscle force on admission to rehabilitation. *Neurorehabil Neural Repair*. 2001;15: 93–97.
64. Visintin M, Barbeau H, Korner-Bitensky N, Mayo NE. A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. *Stroke*. 1998;29:1122–1128.
65. Kosak MC, Reding MJ. Comparison of partial body weight-supported treadmill gait training versus aggressive bracing assisted walking post stroke. *Neurorehabil Neural Repair*. 2000;14:13–19.
66. Dromerick AW, Edwards DF, Hahn M. Does the application of constraint-induced movement therapy during acute rehabilitation reduce arm impairment after ischemic stroke? *Stroke*. 2000;31:2984–2988.
67. Glanz M, Klawansky S, Stason W, Berkey C, Chalmers TC. Functional electrostimulation in poststroke rehabilitation: a meta-analysis of the randomized controlled trials. *Arch Phys Med Rehabil*. 1996;77: 549–553.
68. Price CI, Pandyan AD. Electrical stimulation for preventing and treating post-stroke shoulder pain: a systematic Cochrane review. *Clin Rehabil*. 2001;15:5–19. Review.
69. Bogataj U, Gros N, Kljajic M, Acimovic R, Malezic M. The rehabilitation of gait in patients with hemiplegia: a comparison between conventional therapy and multichannel functional electrical stimulation therapy. *Phys Ther*. 1995;75:490–502.
70. Gelber DA, Good DC, Dromerick A, Sergay S, Richardson M. Open-label dose-titration safety and efficacy study of tizanidine hydrochloride in the treatment of spasticity associated with chronic stroke. *Stroke*. 2001;32:1841–1846.
71. Ketel WB, Kolb ME. Long-term treatment with dantrolene sodium of stroke patients with spasticity limiting the return of function. *Curr Med Res Opin*. 1984;9:161–169.
72. Milanov IG. Mechanisms of baclofen action on spasticity. *Acta Neurol Scand*. 1992;85:305–310.
73. Katrak PH, Cole AM, Poulos CJ, McCauley JC. Objective assessment of spasticity, strength, and function with early exhibition of dantrolene sodium after cerebrovascular accident: a randomized double-blind study. *Arch Phys Med Rehabil*. 1992;73:4–9.
74. Pedersen E, Arlien-Soborg P, Mai J. The mode of action of the gaba derivative baclofen in human spasticity. *Acta Neurol Scand*. 1974;50: 665–680.
75. Goldstein LB. Common drugs influence motor recovery after stroke. The Sygen In Acute Stroke Study Investigators. *Neurology*. 1995;45: 865–871.
76. Goldstein LB. Potential effects of common drugs on stroke recovery. *Arch Neurol*. 1998;55:454–456.
77. Troisi E, Paolucci S, Silvestrini M, Matteis M, Vernieri F, Grasso MG, Caltagirone C. Prognostic factors in stroke rehabilitation: the possible role of pharmacological treatment. *Acta Neurol Scand*. 2002;105: 100–106.
78. Simpson DM, Alexander DN, O'Brien CF, Tagliati M, Aswad AS, Leon JM, Gibson J, Mordaunt JM, Monaghan EP. Botulinum toxin type A in the treatment of upper extremity spasticity: a randomized, double-blind, placebo-controlled trial. *Neurology*. 1996;46:1306–1310.
79. Bakheit AM, Thilmann AF, Ward AB, Poewe W, Wissel J, Muller J, Benecke R, Collin C, Muller F, Ward CD, Neumann C. A randomized, double-blind, placebo-controlled, dose-ranging study to compare the efficacy and safety of three doses of botulinum toxin type A (Dysport) with placebo in upper limb spasticity after stroke. *Stroke*. 2000;31: 2402–2406.
80. Brashear A, Gordon MF, Elovic E, Kassicieh VD, Marciniak C, Do M, Lee CH, Jenkins S, Turkel C; Botox Post-Stroke Spasticity Study Group. Intramuscular injection of botulinum toxin for the treatment of wrist and finger spasticity after a stroke. *N Engl J Med*. 2002;347:395–400.
81. Kirazli Y, On AY, Kismali B, Aksit R. Comparison of phenol block and botulinus toxin type A in the treatment of spastic foot after stroke: a randomized, double-blind trial. *Am J Phys Med Rehabil*. 1998;77: 510–515.
82. Kong KH, Chua KS. Neurolysis of the musculocutaneous nerve with alcohol to treat poststroke elbow flexor spasticity. *Arch Phys Med Rehabil*. 1999;80:1234–1236.
83. On AY, Kirazli Y, Kismali B, Aksit R. Mechanisms of action of phenol block and botulinus toxin Type A in relieving spasticity: electrophysiologic investigation and follow-up. *Am J Phys Med Rehabil*. 1999;78: 344–349.
84. Meythaler JM, Guin-Renfroe S, Brunner RC, Hadley MN. Intrathecal baclofen for spastic hypertonia from stroke. *Stroke*. 2001;32: 2099–2109.

85. Van Ouwenaller C, Laplace PM, Chantraine A. Painful shoulder in hemiplegia. *Arch Phys Med Rehabil*. 1986;67:23–26.
86. Cicerone KD, Dahlberg C, Kalmar K, Langenbahn DM, Malec JF, Bergquist TF, Felicetti T, Giacino JT, Harley JP, Harrington DE, Herzog J, Kneipp S, Laatsch L, Morse PA. Evidence-based cognitive rehabilitation: recommendations for clinical practice. *Arch Phys Med Rehabil*. 2000;81:1596–1615. Review.
87. Gray JM, Robertson I, Pentland B, et al. Microcomputer-based attentional retraining after brain damage: a randomized group controlled trial. *Neuropsychol Rehabil*. 1992;2:97–115.
88. Niemann H, Ruff RM, Baser CA. Computer-assisted attention retraining in head-injured individuals: a controlled efficacy study of an outpatient program. *J Consult Clin Psychol*. 1990;58:811–817.
89. Sohlberg MM, Mateer CA. Effectiveness of an attention-training program. *J Clin Exp Neuropsychol*. 1987;9:117–130.
90. Sohlberg MM, Mateer CA. Training use of compensatory memory books: a three stage behavioral approach. *J Clin Exp Neuropsychol*. 1989;11:871–891.
91. Strache W. Effectiveness of two modes of training to overcome deficits of concentration. *Int J Rehabil Res*. 1987;10:141–145.
92. Ryan TV, Ruff RM. The efficacy of structured memory retraining in a group comparison of head trauma patients. *Arch Clin Neuropsychol*. 1988;3:165–179.
93. Antonucci G, Guariglia C, Judica A, Magnotti L, Paolucci S, Pizzamiglio L, Zoccolotti P. Effectiveness of neglect rehabilitation in a randomized group study. *J Clin Exp Neuropsychol*. 1995;17:383–389.
94. Beis JM, Andre JM, Baumgarten A, Challier B. Eye patching in unilateral spatial neglect: efficacy of two methods. *Arch Phys Med Rehabil*. 1999;80:71–76.
95. Fanthome Y, Lincoln NB, Drummond A, Walker MF. The treatment of visual neglect using feedback of eye movements: a pilot study. *Disabil Rehabil*. 1995;17:413–417.
96. Paolucci S, Antonucci G, Guariglia C, Magnotti L, Pizzamiglio L, Zoccolotti P. Facilitatory effect of neglect rehabilitation on the recovery of left hemiplegic stroke patients: a cross-over study. *J Neurol*. 1996; 243:308–314.
97. Rossetti Y, Rode G, Pisella L, Farne A, Li L, Boisson D, Perenin MT. Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. *Nature*. 1998;395:166–169.
98. Wiart L, Come AB, Debelleix X, Petit H, Joseph PA, Mazaux JM, Barat M. Unilateral neglect syndrome rehabilitation by trunk rotation and scanning training. *Arch Phys Med Rehabil*. 1997;78:424–429.
99. Robinson RG. *The Clinical Neuropsychiatry of Stroke.* New York: Cambridge University Press; 1998.
100. Tsouna-Hadjis E, Vemmos KN, Zakopoulos N, Stamatelopoulos S. First-stroke recovery process: the role of family social support. *Arch Phys Med Rehabil*. 2000;81:881–887.
101. Andersen G. Treatment of uncontrolled crying after stroke. *Drugs Aging*. 1995;6:105–111. Review.
102. Cole MG, Elie LM, McCusker J, Bellavance F, Mansour A. Feasibility and effectiveness of treatments for post-stroke depression in elderly inpatients: systematic review. *J Geriatr Psychiatry Neurol*. 2001;14: 37–41.
103. Gill D, Hatcher S. Antidepressants for depression in medical illness. *Cochrane Database Syst Rev*. 2000(4):CD001312.
104. Kimura M, Robinson RG, Kosier JT. Treatment of cognitive impairment after poststroke depression: a double-blind treatment trial. *Stroke*. 2000; 31:1482–1486.
105. Reding JJ, Orto LA, Winters SW, Fortuna IM, DiPonte P, McDowell FH. Antidepressant therapy after stroke: a double-blind trial. *Arch Neurol*. 1986;43:763–765.
106. Robinson RG, Schultz SK, Castillo C, Kopel T, Kosier JT, Newman RM, Curdue K, Petracca G, Starkstein SE. Nortriptyline versus fluoxetine in the treatment of depression and in short-term recovery after stroke: a placebo-controlled, double-blind study. *Am J Psychiatry*. 2000; 157:351–359.
107. Wiart L, Petit H, Joseph PA, Mazaux JM, Barat M. Fluoxetine in early poststroke depression: a double-blind placebo-controlled study. *Stroke*. 2000;31:1829–1832.
108. Brown KW, Sloan RL, Pentland B. Fluoxetine as a treatment for poststroke emotionalism. *Acta Psychiatr Scand*. 1998;98:455–458.
109. Burns A, Russell E, Stratton-Powell H, Tyrell P, O'Neill P, Baldwin R. Sertraline in stroke-associated lability of mood. *Int J Geriatr Psychiatry*. 1999;14:681–685.
110. Gordon MT. Developing clinical guidelines for the management of patients with stroke. Scottish Intercollegiate Guidelines Network (SIGN). *Int J Lang Commun Disord*. 1998;33 suppl:152–157.
111. Robinson RG, Parikh RM, Lipsey JR, Starkstein SE, Price TR. Pathological laughing and crying following stroke: validation of a measurement scale and a double-blind treatment study. *Am J Psychiatry*. 1993;150:286–293.
112. Reding MJ, Borucki S. Effect of dextroamphetamine on motor recovery after stroke. *Neurology*. 1995;45(suppl 4):A222. Abstract.
113. Sonde L, Nordstrom M, Nilsson CG, Lokk J, Viitanen M. A double-blind placebo-controlled study of the effects of amphetamine and physiotherapy after stroke. *Cerebrovasc Dis*. 2001;12:253–257.
114. Gladstone DJ, Danells CJ, Armesto A, McIlroy WE, Staines R, Graham SJ, Herrmann N, Szalai JP, Black SE; for the Subacute Therapy with Amphetamine and Rehabilitation for Stroke (STARS) Study Investigators. Physiotherapy coupled with dextroamphetamine for motor rehabilitation after hemiparetic stroke: a randomized controlled trial. In: Program Schedule and Abstracts of the 29th International Stroke Conference. *Stroke*. 2004;35:239. Abstract No. 23.
115. Grade C, Redford B, Chrostowski J, Toussaint L, Blackwell B. Methylphenidate in early poststroke recovery: a double-blind, placebo-controlled study. *Arch Phys Med Rehabil*. 1998;79:1047–1050.
116. Scheidtmann K, Fries W, Muller F, Koenig E. Effect of levodopa in combination with physiotherapy on functional motor recovery after stroke: a prospective, randomised, double-blind study. *Lancet*. 2001; 358:787–790.
117. Nishino K, Sasaki T, Takahashi K, Chiba M, Ito T. The norepinephrine precursor L-threo-3,4-dihydroxyphenylserine facilitates motor recovery in chronic stroke patients. *J Clin Neurosci*. 2001;8:547–550.
118. Dam M, Tonin P, De Boni A, Pizzolato G, Casson S, Ermani M, Freo U, Piron L, Battistin L. Effects of fluoxetine and maprotiline on functional recovery in poststroke hemiplegic patients undergoing rehabilitation therapy. *Stroke*. 1996;27:1211–1214.
119. Lazar RM, Fitzsimmons BF, Marshall RS, Mohr JP, Berman MF. Midazolam challenge reinduces neurological deficits after transient ischemic attack. *Stroke*. 2003;34:794–796.