

Guidelines for Adult Stroke Rehabilitation and Recovery A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

Endorsed by the American Academy of Physical Medicine and Rehabilitation and the American Society of Neurorehabilitation

The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists and the American Congress of Rehabilitation Medicine also affirms the educational value of these guidelines for its members

Accepted by the American Speech-Language-Hearing Association

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Purpose—The aim of this guideline is to provide a synopsis of best clinical practices in the rehabilitative care of adults recovering from stroke.

Methods—Writing group members were nominated by the committee chair on the basis of their previous work in relevant topic areas and were approved by the American Heart Association (AHA) Stroke Council's Scientific Statement Oversight Committee and the AHA's Manuscript Oversight Committee. The panel reviewed relevant articles on adults using computerized searches of the medical literature through 2014. The evidence is organized within the context of the AHA framework and is classified according to the joint AHA/American College of Cardiology and supplementary AHA methods of classifying the level of certainty and the class and level of evidence. The document underwent extensive AHA internal and external peer review, Stroke Council Leadership review, and Scientific Statements Oversight Committee review before consideration and approval by the AHA Science Advisory and Coordinating Committee.

Results—Stroke rehabilitation requires a sustained and coordinated effort from a large team, including the patient and his or her goals, family and friends, other caregivers (eg, personal care attendants), physicians, nurses, physical and occupational therapists, speech-language pathologists, recreation therapists, psychologists, nutritionists, social workers, and others. Communication and coordination among these team members are paramount in maximizing the effectiveness

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and efficiency of rehabilitation and underlie this entire guideline. Without communication and coordination, isolated efforts to rehabilitate the stroke survivor are unlikely to achieve their full potential.

Conclusions—As systems of care evolve in response to healthcare reform efforts, postacute care and rehabilitation are often considered a costly area of care to be trimmed but without recognition of their clinical impact and ability to reduce the risk of downstream medical morbidity resulting from immobility, depression, loss of autonomy, and reduced functional independence. The provision of comprehensive rehabilitation programs with adequate resources, dose, and duration is an essential aspect of stroke care and should be a priority in these redesign efforts. (*Stroke*. 2016;47:e98-e169. DOI: 10.1161/STR.0000000000000098.)

Key Words: AHA Scientific Statements ■ exercise ■ paresis ■ recovery of function ■ rehabilitation ■ stroke

Between 2000 and 2010, the relative rate of stroke deaths dropped by 35.8% in the United States.¹ However, each year stroke affects nearly 800 000 individuals, with many survivors experiencing persistent difficulty with daily tasks as a direct consequence. More than two thirds of stroke survivors receive rehabilitation services after hospitalization.² Despite the development of stroke center designation and improved systems to recognize stroke symptoms and deliver care promptly, only a minority of patients with acute stroke receive thrombolytic therapy, and many of them remain with residual functional deficits. Thus, the need for effective stroke rehabilitation is likely to remain an essential part of the continuum of stroke care for the foreseeable future.

Despite the extensive resources devoted to stroke rehabilitation and aftercare, large-scale, rigorous, clinical trials in this field have been few and have been conducted only in the past decade or so. Thus, many gaps continue to be seen in the evidence base for stroke rehabilitation, for which smaller trials of less rigorous design provide the only available data, and in some cases, even these are not yet available. Certain aspects of stroke rehabilitation care are well established in clinical practice and constitute a standard of care that is unlikely to be directly tested in a randomized, clinical trial, for example, the provision of physical therapy (PT) to early stroke survivors with impaired walking ability. Thus, practice guidelines such as this one will likely rely on a mixture of evidence and consensus. It is hoped that the relative proportion of recommendations based on rigorous evidence will grow over time.

This guideline uses the framework established by the American Heart Association (AHA) concerning classes and levels of evidence for use in guidelines, as shown in Tables 1 and 2.

We have organized this guideline into 5 major sections: (1) The Rehabilitation Program, which includes system-level sections (eg, organization, levels of care); (2) Prevention and Medical Management of Comorbidities, in which reference is made to other published guidelines (eg, hypertension); (3) Assessment, focused on the body function/structure level of the *International Classification of Functioning, Disability, and Health (ICF)*³; (4) Sensorimotor Impairments and Activities (treatment/interventions), focused on the activity level of the *ICF*; and (5) Transitions in Care and Community Rehabilitation, focused primarily on the participation level of the *ICF*.

Published guidelines are, by their very nature, a reflection of clinical practice at a particular point in time and the evidence base available. As new information becomes available, best practice can change quickly, and it is incumbent on the users of these guidelines to keep the ever-changing nature of clinical knowledge in mind. Equally important, no guideline can substitute for the careful evaluation of the individual patient by an

experienced clinician, in which the art and science of medicine intersect. Guidelines that are correct in the aggregate may not represent the best care for any specific individual, and careful individualization is needed at the point of care.

We have benefited from the published Veterans Affairs/Department of Defense stroke rehabilitation guidelines⁴ and several of the prior AHA stroke-related guidelines.^{4a} Although the current guideline is a fundamentally new work, it certainly reflects the insights and judgments of these prior guidelines.

Because stroke is fundamentally a chronic condition, we have attempted to span the entire course of rehabilitation, from the early actions taken in the acute care hospital through reintegration into the community. The end of formal rehabilitation (commonly by 3–4 months after stroke) should not mean the end of the restorative process. In many respects, stroke has been managed medically as a temporary or transient condition instead of a chronic condition that warrants monitoring after the acute event. Currently, unmet needs persist in many domains, including social reintegration, health-related quality of life, maintenance of activity, and self-efficacy (ie, belief in one's capability to carry out a behavior). Apathy is manifested in >50% of survivors at 1 year after stroke⁵; fatigue is a common and debilitating symptom in chronic stroke⁶; daily physical activity of community-living stroke survivors is low⁷; and depressive symptomology is high.⁸ By 4 years after onset, >30% of stroke survivors report persistent participation restrictions (eg, difficulty with autonomy, engagement, or fulfilling societal roles).⁹

The Rehabilitation Program

Organization of Poststroke Rehabilitation Care (Levels of Care)

Rehabilitation services are the primary mechanism by which functional recovery and the achievement of independence are promoted in patients with acute stroke. The array of rehabilitation services delivered to stroke patients in the United States is broad and highly heterogeneous, varying in the type of care settings used; in the duration, intensity, and type of interventions delivered; and in the degree of involvement of specific medical, nursing, and other rehabilitation specialists. The nature and organization of rehabilitation stroke services in the United States have changed considerably over time in response to various forces, including the increasing integration of hospital and outpatient care delivery systems (at both local and regional levels), the organization of medical and other specialty rehabilitation groups, and most important, repeated changes to the federal reimbursement fee structure (specifically, Centers for Medicare & Medicaid Services), which is

Table 1. Applying Classification of Recommendations and Level of Evidence

		SIZE OF TREATMENT EFFECT													
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT		CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/ administered	CLASS IIa <i>Benefit >> Risk</i> Additional studies with <i>focused objectives needed</i> IT IS REASONABLE to per- form procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> Additional studies with <i>broad objectives needed; additional registry data would be helpful</i> Procedure/Treatment MAY BE CONSIDERED	CLASS III <i>No Benefit</i> or CLASS III <i>Harm</i>										
					<table><tr><th></th><th>Procedure/ Test</th><th>Treatment</th></tr><tr><td>COR III: No benefit</td><td>Not Helpful</td><td>No Proven Benefit</td></tr><tr><td>COR III: Harm</td><td>Excess Cost w/o Benefit or Harmful</td><td>Harmful to Patients</td></tr></table>		Procedure/ Test	Treatment	COR III: No benefit	Not Helpful	No Proven Benefit	COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients	
		Procedure/ Test	Treatment												
	COR III: No benefit	Not Helpful	No Proven Benefit												
	COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients												
LEVEL A Multiple populations evaluated*	<ul style="list-style-type: none">Recommendation that procedure or treatment is useful/effectiveSufficient evidence from multiple randomized trials or meta-analyses	<ul style="list-style-type: none">Recommendation in favor of treatment or procedure being useful/effectiveSome conflicting evidence from multiple randomized trials or meta-analyses	<ul style="list-style-type: none">Recommendation's usefulness/efficacy less well establishedGreater conflicting evidence from multiple randomized trials or meta-analyses	<ul style="list-style-type: none">Recommendation that procedure or treatment is not useful/effective and may be harmfulSufficient evidence from multiple randomized trials or meta-analyses											
LEVEL B Limited populations evaluated*	<ul style="list-style-type: none">Recommendation that procedure or treatment is useful/effectiveEvidence from single randomized trial or nonrandomized studies	<ul style="list-style-type: none">Recommendation in favor of treatment or procedure being useful/effectiveSome conflicting evidence from single randomized trial or nonrandomized studies	<ul style="list-style-type: none">Recommendation's usefulness/efficacy less well establishedGreater conflicting evidence from single randomized trial or nonrandomized studies	<ul style="list-style-type: none">Recommendation that procedure or treatment is not useful/effective and may be harmfulEvidence from single randomized trial or nonrandomized studies											
LEVEL C Very limited populations evaluated*	<ul style="list-style-type: none">Recommendation that procedure or treatment is useful/effectiveOnly expert opinion, case studies, or standard of care	<ul style="list-style-type: none">Recommendation in favor of treatment or procedure being useful/effectiveOnly diverging expert opinion, case studies, or standard of care	<ul style="list-style-type: none">Recommendation's usefulness/efficacy less well establishedOnly diverging expert opinion, case studies, or standard of care	<ul style="list-style-type: none">Recommendation that procedure or treatment is not useful/effective and may be harmfulOnly expert opinion, case studies, or standard of care											
Suggested phrases for writing recommendations		should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	<table><tr><th>COR III: No Benefit</th><th>COR III: Harm</th></tr><tr><td>is not recommended</td><td>potentially harmful</td></tr><tr><td>is not indicated</td><td>causes harm</td></tr><tr><td>should not be performed/ administered/ other</td><td>associated with excess morbidity/mortality</td></tr><tr><td>is not useful/ beneficial/ effective</td><td>should not be performed/ administered/ other</td></tr></table>	COR III: No Benefit	COR III: Harm	is not recommended	potentially harmful	is not indicated	causes harm	should not be performed/ administered/ other	associated with excess morbidity/mortality	is not useful/ beneficial/ effective	should not be performed/ administered/ other
COR III: No Benefit	COR III: Harm														
is not recommended	potentially harmful														
is not indicated	causes harm														
should not be performed/ administered/ other	associated with excess morbidity/mortality														
is not useful/ beneficial/ effective	should not be performed/ administered/ other														
Comparative effectiveness phrases†		treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B												

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

the central driver of much of the system's organization and structure. Further systems-level changes are inevitable, given the ongoing federal changes to the healthcare system and the recent focus on "episodes of care," which promises to result in wholesale changes to the organization of medical care delivery in the United States.¹⁰

The highly heterogeneous organizational structure of stroke rehabilitation care in the United States brings with it challenges in terms of determining the quality of care delivered by the system (ie, timeliness, effectiveness, efficiency, safety, fairness, and patient-centeredness). The unique and somewhat idiosyncratic nature of the stroke rehabilitation system in the United

States also presents challenges in terms of assessment of which research findings, among the expanding evidence base of stroke rehabilitation care, are applicable to the system. For example, much of the research documenting the benefits of stroke units and other aspects of organized integrated interprofessional models of stroke care was developed in Europe and elsewhere, and the degree to which these findings are directly applicable to the US system of stroke care is often debated.

Organization of Acute and Postacute Rehabilitation Care in the United States

An excellent review of the current organizational structure of stroke rehabilitation care in the United States can be found in

Table 2. Definition of Classes and Levels of Evidence Used in AHA/ASA Recommendations

Class I	Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
Class IIa	The weight of evidence or opinion is in favor of the procedure or treatment
Class IIb	Usefulness/efficacy is less well established by evidence or opinion
Class III	Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful
Therapeutic recommendations	
Level of Evidence A	Data derived from multiple randomized, clinical trials or meta-analyses
Level of Evidence B	Data derived from a single randomized trial or nonrandomized studies
Level of Evidence C	Consensus opinion of experts, case studies, or standard of care
Diagnostic recommendations	
Level of Evidence A	Data derived from multiple prospective cohort studies using a reference standard applied by a masked evaluator
Level of Evidence B	Data derived from a single grade A study, ≥1 case-control studies, or studies using a reference standard applied by an unmasked evaluator
Level of Evidence C	Consensus opinion of experts

AHA/ASA indicates American Heart Association/American Stroke Association.

the 2010 AHA scientific statement “Comprehensive Overview of Nursing and Interdisciplinary Rehabilitation Care of the Stroke Patient.”¹¹ We briefly review the different stroke neurology, rehabilitation care settings that are essential components of this system (Appendix 1).

Ideally, rehabilitation services are delivered by a multidisciplinary team of healthcare providers with training in neurology, rehabilitation nursing, occupational therapy (OT), PT, and speech and language therapy (SLT). Such teams are directed under the leadership of physicians trained in physical medicine and rehabilitation (physiatrist) or by neurologists who have specialized training or board certification in rehabilitation medicine. Other health professionals who play an essential role in the process include social workers, psychologists, psychiatrists, and counselors.¹¹

Health care provided during the acute hospital stay is focused primarily on the acute stabilization of the patient, the delivery of acute stroke treatments, and the initiation of prophylactic and preventive measures. Although the delivery of rehabilitation therapies (OT/PT/SLT) is generally not the first priority, data strongly suggest that there are benefits to starting rehabilitation as soon as the patient is ready and can tolerate it.¹¹

The cardinal feature of acute inpatient care for stroke patients in the United States is its brevity; the median length of stay for patients with ischemic stroke is only 4 days. Regardless of whether rehabilitation is started during the inpatient stay, all patients should undergo a formal assessment (often conducted by the OT/PT/SLT services) of the patient's rehabilitation needs before discharge.¹² The discharge process may also involve rehabilitation nursing case managers and social workers who can assess psychosocial issues that may influence the transition.

Healthcare services provided after hospital discharge are referred to as postacute care services and are designed to support patients in their transition from the hospital to home and in their pursuit of achieving the highest level of functioning possible. In addition to the rehabilitation care provided by OT/PT/SLT, care may include physiatrists or other physicians, rehabilitation nurses, and nursing aides. The intensity of rehabilitation care varies widely, depending on the setting, with the most intensive rehabilitation care provided in inpatient rehabilitation facilities (IRFs), followed by skilled nursing facilities (SNFs), which provide “subacute” rehabilitation.

IRFs provide hospital-level care to stroke survivors who need intensive, 24-hour-a-day, interdisciplinary rehabilitation care that is provided under the direct supervision of a physician. Medicare (Centers for Medicare & Medicaid Services) regulations specify that admission to IRFs should be limited to patients for whom significant improvement is expected within a reasonable length of time and who are likely to return to a community setting (rather than being transferred to another setting such as a SNF or long-term care facility). Medicare regulations also generally dictate that IRFs provide at least 3 hours of rehabilitation therapy (defined as PT, OT, and SLT) per day for at least 5 d/wk.¹¹ Physicians are expected to have training or experience in rehabilitation, and daily physician visits are typical. Registered nurses are present on a continuous basis and commonly have specialty certification in rehabilitation nursing. An IRF can be located as a geographically distinct unit within an acute care hospital or as a free-standing facility.

SNFs (also known as subacute rehabilitation) provide rehabilitation care to stroke survivors who need daily skilled nursing or rehabilitation services. Admission to SNFs may be requested for patients who the rehabilitation team determines may not reach full or partial recovery or if skilled nursing services are required to maintain or prevent deterioration of the patient. SNFs are required to have rehabilitation nursing on site for a minimum of 8 h/d, and care must still follow a physician's plan, although there is no requirement for direct daily supervision by a physician.¹³ SNFs can be stand-alone facilities, but when located within an existing nursing home or hospital, they must be physically distinguishable from the larger institution (eg, a separate designated wing, ward, or building).

Nursing homes provide long-term residential care for individuals who are unable to live in the community. Many individuals who reside in nursing homes initially enter the facility under their Medicare short-term SNF benefit and then transition to long-term care once the needs for skilled nursing are no longer present. Medicare will provide insurance coverage for up to 100 days in an SNF but does not cover long-term nursing home care, which is generally paid out of pocket, by long-term care insurance, or through the Medicaid program.

Long-term acute care hospitals are another inpatient setting that delivers postacute rehabilitation care. Long-term acute care hospitals provide extended medical and rehabilitative care to stroke patients with complex medical needs resulting from a combination of acute and chronic conditions (eg, ventilator-dependent care, pain management). As a consequence of this high-needs patient population, facilities must demonstrate an average length of stay of at least 25 days.^{14,15} Because of these requirements, long-term acute care hospitals provide care to a relatively small but growing minority of stroke patients.¹⁴

For stroke patients who go home after an acute hospitalization, rehabilitation care can be provided in the community either by a home healthcare agency (HHCA) or through outpatient offices and clinics. The intensity of rehabilitation care can vary tremendously across these 2 settings. For patients in the Medicare program to be eligible for HHCA services, they must be certified as being homebound by a physician (defined by the Centers for Medicare & Medicaid Services as unable to leave the home except to receive medical care or to have occasional nonmedical trips). HHCA focus on delivering skilled nursing care and rehabilitation therapy (eg, OT, PT, SLT), as well as some limited assistance with daily tasks provided by home health aides supervised by nurses. Care encompasses medical and social needs and services that are designed to assist the patient in living in his or her own home.¹³ Currently, home healthcare services are reimbursed under a prospective payment system that covers up to 60 days of services. These services may be extended if they can be clinically justified. Home healthcare services may also be performed in assisted living facilities or other group homes but are not reimbursed if the services are duplicative of the services of another facility or agency.

Appropriateness of Early Supported Discharge Rehabilitation Services

For selected stroke patients, early discharge to a community setting for ongoing rehabilitation may provide outcomes similar to those achieved in an inpatient rehabilitation unit. This early supported discharge (ESD) model of care links inpatient care with community services and allows certain patients to be discharged home sooner with support of the rehabilitation team.

The efficacy of ESD for patients with acute stroke was evaluated in the ESD Trialists' systematic review.¹⁶ This 2012 review concluded that "appropriately resourced ESD services provided for a selected group of stroke patients can reduce long-term dependency and admission to institutional care as well as reducing the length of hospital stay." No adverse impacts were identified on either mood or the subjective health status of patients or caregivers with ESD. ESD has been studied primarily in Europe and Australia/New Zealand, where systems of care are different than in the United States and where the average acute care hospitalization length of stay for stroke is longer than in the United States. Extrapolation of these results to the United States should take these distinctions into account.

A meta-analysis conducted by Langhorne et al¹⁷ and updated by Langhorne and Holmqvist¹⁸ found that ESD services reduce inpatient length of stay and adverse events (eg, readmission rates) while increasing the likelihood of independence and living at home. Several recent systematic reviews have also reported that ESD after stroke was associated with shorter hospital lengths of

stay, lower overall costs of care, lower risk of institutionalization, and no adverse effects on functional recovery.^{19–21}

To be effective, ESD should be considered for patients with mild to moderate stroke when adequate community services for both rehabilitation and caregiver support are available and can provide the level of intensity of rehabilitation service needed.²² Patients should remain in an inpatient setting for their rehabilitation care if they are in need of skilled nursing services, regular contact by a physician, and multiple therapeutic interventions.

Examples for need of skilled nursing services include (but are not limited to) the following:

- Bowel and bladder impairment
- Skin breakdown or high risk for skin breakdown
- Impaired bed mobility
- Dependence for activities of daily living (ADLs)
- Inability to manage medications
- High risk for nutritional deficits

Examples for need of regular contact by a physician include (but are not limited to) the following:

- Medical comorbidities not optimally managed (eg, diabetes mellitus and hypertension)
- Complex rehabilitation issues (eg, orthotics, spasticity, and bowel/bladder)
- Acute illness (but not severe enough to prevent rehabilitation care)
- Pain management issues

Examples for need of multiple therapeutic interventions include (but are not limited to) the following:

- Moderate to severe motor/sensory deficits, and/or
- Cognitive deficits, and/or
- Communication deficits

Outpatient therapies require patients to travel from their home to obtain care at hospital-based or free-standing facilities. All outpatient OT, PT, and SLT services must be certified by a physician who is responsible for establishing a planned set of therapy services. These therapies must be complex enough that they can be performed only by a qualified healthcare professional. Treatment plans need to be reviewed and recertified every 30 days.

Multiple transitions in care are typical for individuals recovering from stroke and pose particular challenges for healthcare providers, stroke survivors, and their caregivers in terms of maintaining continuity of care and avoiding undesirable lapses in the rehabilitation program of care. Moreover, stroke survivors need to navigate the transition from a medical model of treatment to a more community-based model that includes return to work (for some), leisure activities, and exercise for fitness. The Transitions in Care and Community Rehabilitation section addresses transitions to the community after discharge.

Trends in the Use of Acute and Postacute Stroke Rehabilitation in the United States

The organization of rehabilitation stroke services in the United States has changed considerably over time in response to the frequent changes to the federal reimbursement fee structure for both acute (inpatient) and postacute

care. Currently, $\approx 70\%$ of Medicare beneficiaries discharged for acute stroke use Medicare-covered postacute care,²³ with most receiving rehabilitation care from multiple providers in several different settings.^{24,25} Considering the first setting after the acute hospitalization, the largest proportion of stroke patients are referred for rehabilitation to an SNF (32%), followed by an IRF (22%) and then HHCA (15%).²⁶ Major changes in the Medicare postacute care reimbursement policies starting in the 1990s dramatically affected use patterns,²⁶ particularly for HHCA, after the introduction of an interim payment system in 1997 with extensive changes to its rules and regulations in 2000. The introduction of prospective payment systems for SNFs (1998), IRFs (2002), and long-term acute care hospitals (2002) also affected their use.^{13,27} Between 1996 and 2003, the proportion of Medicare stroke patients who received care from HHCA declined by $>25\%$ during this period (from 20% to 15%),²⁶ whereas the proportion who received SNF or IRF care remained relatively unchanged. However, the proportion of stroke patients not referred to any postacute care increased from 26% to 31% during this period,²⁶ and an analysis of 2006 Medicare data found that this proportion had increased to 42%.²⁸ Although legislated payment changes have had major influences on where rehabilitation services are provided, several other nonclinical factors affect the use of postacute care rehabilitation services. There is considerable geographic variability in the use of these services in the United States,²⁹ which is driven in part by local differences in the availability of postacute care settings and regulatory practices.^{29–33} Factors such as the daily census, case mix, teaching status, ownership, and urbanicity of the hospital and the percentage of patients served by Medicare have been shown to influence use patterns of postacute services.^{30,34,35} At the patient level, sociodemographic factors such as age, income, race, and living circumstances have also been shown to affect the use and type of rehabilitation services provided.^{30–33,36–38}

Of central interest to researchers and policy makers is the need for a better understanding of the impact of rehabilitation care at these different rehabilitation settings on patient outcomes, especially relative to resource use and costs. The studies that have compared outcomes in hospitalized stroke patients first discharged to an IRF, an SNF, or a nursing home have generally shown that IRF patients have higher rates of return to community living^{39,40} and greater functional recovery,^{39–42} whereas patients discharged to an SNF or a nursing home have higher rehospitalization rates⁴³ and substantially poorer survival.^{44,45} However, all of these studies have limitations resulting from their observational designs, which rely on administrative data^{39–41} or data from a limited number of facilities.⁴² Importantly, most of these studies demonstrate substantial baseline differences in patient case mix between settings, with IRF patients having a more favorable prognostic outlook because of their younger age, lower prestroke disability, fewer comorbidities, and greater caregiver/family support and because they have been selected for their potential to return to the community.^{39–41,45} These differences serve to illustrate that the decision to refer a stroke patient to a particular setting after discharge is dictated by a complex set of demographic, clinical, and nonclinical factors that are also inevitably related

to patient outcomes. This inherent confounding or channeling bias⁴⁶ has been addressed by these studies through the application of complex statistical methods.^{39–41} However, uncertainty remains about how much of the final difference in outcome is attributable to residual confounding resulting from unmeasured factors (particularly stroke severity and prestroke disability). Despite these concerns, the consistency of the findings in favor of IRF referral suggests that stroke survivors who qualify for IRF services should receive this care in preference to SNF-based care.

Recommendations: Organization of Poststroke Rehabilitation Care (Levels of Care)	Class	Level of Evidence
It is recommended that stroke patients who are candidates for postacute rehabilitation receive organized, coordinated, interprofessional care.	I	A
It is recommended that stroke survivors who qualify for and have access to IRF care receive treatment in an IRF in preference to a SNF.	I	B
Organized community-based and coordinated interprofessional rehabilitation care is recommended in the outpatient or home-based settings.	I	C
ESD services may be reasonable for people with mild to moderate disability.	IIB	B

Rehabilitation Interventions in the Inpatient Hospital Setting

There is strong evidence that organized, interprofessional stroke care not only reduces mortality rates and the likelihood of institutional care and long-term disability but also enhances recovery and increases independence in ADLs.^{47–50} Although many small, randomized, clinical trials have studied interventions in the acute rehabilitation phase, the only large, randomized, clinical trials in stroke recovery and rehabilitation have focused on the chronic recovery phase.^{51,52} This section updates the scientific statement on the comprehensive overview of nursing and interprofessional rehabilitation care of the stroke patient and previously summarized recommendations for care of the stroke survivor in the inpatient rehabilitation phase.¹¹

Although acute stroke units have higher levels of nurse staffing, earlier assessments of stroke type and treatment, and more intensive physiological monitoring, rehabilitation units (including comprehensive stroke units in Europe) emphasize recovery and rehabilitation, involving rehabilitation physicians and allied health professionals, increased interprofessional staff education and training, greater patient and caregiver participation in rehabilitation, and early mobilization protocols.⁵³ Age, cognition, functional level after stroke, and to a lesser extent continence have shown consistent associations with poststroke outcomes, and stroke severity is associated with acute discharge disposition, final discharge disposition, and functional level.⁵⁴ In recent years, lengths of stay in IRFs have decreased significantly, but in survivors with mild to moderate stroke, patient satisfaction does not appear to be diminished, and recovery actually may be faster.⁵⁵ In the United States, data after the initiation of prospective payment for rehabilitation in 2002 suggest that discharges from IRFs to institutional settings have increased.⁵⁶

Timing and intensity of acute rehabilitation also are important issues in poststroke functional outcomes but remain controversial. Overall, a 2009 meta-analysis demonstrated insufficient evidence to support or refute the efficacy of routine very early mobilization after stroke compared with conventional care.⁵⁷ In the recently completed randomized, controlled trial (RCT) of the efficacy and safety of very early mobilization within 24 hours of stroke onset (A Very Early Rehabilitation Trial [AVERT]), the high-dose, very early mobilization protocol was associated with a reduction in the odds of a favorable outcome at 3 months.⁵⁸ Early mobilization after stroke is recommended in many clinical practice guidelines worldwide. The AVERT findings should affect clinical practice by refining present guidelines, but clinical recommendations should be informed by future analyses of dose-response associations.

The only evidence assessing the intensity of stroke rehabilitation comes from literature comparing IRFs with subacute rehabilitation. In a study of 222 subjects, Chan et al⁵⁹ reported that subjects whose care included an IRF stay experienced functional scores at least 8 points higher (twice the minimally detectable change) on the Activity Measure for Post-Acute Care than those who went to SNFs or received home health/outpatient care. A retrospective cohort study of 360 subjects demonstrated that subjects who received >3.0 hours of therapy daily made significantly more functional gains than those receiving <3.0 hours daily, although hemorrhagic stroke, left-sided brain injury, earlier IRF admission, and longer IRF stay also were associated with total functional improvement.⁶⁰

Finally, the efficacy of complementary medicine techniques has been studied in the IRF environment. In a randomized, clinical trial of 274 subjects receiving acupuncture, PT, or both, no synergistic effect was found when acupuncture was added to PT, although all subjects exhibited functional gains.⁶¹ An RCT of 53 subjects receiving whole-body somatosensory stimulation or exercise therapy in addition to conventional rehabilitation demonstrated no significant increases in the recovery of balance and ADLs.⁶²

For evidence pertaining to dysphagia; interventions for upper limb rehabilitation, including upper extremity activities (ie, ADLs, instrumental ADLs [IADLs]), touch, and proprioception; lower extremity rehabilitation, including mobility (eg, locomotion) and balance/vestibular rehabilitation; and therapies for cognitive impairments and hemi-spatial neglect, the reader is directed to those subsections in The Rehabilitation Program section.

Recommendations: Rehabilitation Interventions in the Inpatient Hospital Setting	Class	Level of Evidence
It is recommended that early rehabilitation for hospitalized stroke patients be provided in environments with organized, interprofessional stroke care.	I	A
It is recommended that stroke survivors receive rehabilitation at an intensity commensurate with anticipated benefit and tolerance.	I	B
High-dose, very early mobilization within 24 hours of stroke onset can reduce the odds of a favorable outcome at 3 months and is not recommended.	III	A

Prevention and Medical Management of Comorbidities

Prevention of Skin Breakdown and Contractures

Hemiparesis, sensory changes, and altered levels of consciousness place the patient with stroke at risk for joint and muscle contractures and skin breakdown. Pressure ulcers are also associated with impaired circulation, older age, and incontinence. Regular assessment of skin and the use of objective scales of risk such as the Braden scale are valuable in the prevention of skin injury and should be followed by regular skin inspection with documentation.⁶³ Agency for Healthcare Research and Quality (AHRQ) guidelines recommend minimizing or eliminating friction, minimizing pressure, providing appropriate support surfaces, avoiding excessive moisture, and maintaining adequate nutrition and hydration.⁶³ Specific measures include regular turning (at least every 2 hours), good hygiene, and the use of special mattresses and proper wheelchair seating to prevent skin injury.¹¹

After stroke with hemiparesis, 60% of patients will develop joint contracture on the affected side within the first year, with wrist contractures occurring most commonly in patients who do not recover functional hand use.^{65,66} The occurrence of elbow contractures within the first year after stroke is associated with the presence of spasticity within the first 4 months.⁶⁷ These contractures can cause pain and make self-care, including dressing and hygiene, difficult. Many clinicians recommend daily stretching of the hemiplegic limbs to avoid contractures, and patients and families should be taught proper stretching techniques to avoid injury and to maximize effectiveness. Resting hand splints are often applied to prevent contractures in hemiplegic wrist and fingers, but their effectiveness is not well established.^{68,69} There is controversy over the benefit of resting hand splints such that the Royal College of Physicians National Institute for Clinical Excellence guidelines recommend against the use of resting hand splints but the Veterans Affairs/Department of Defense clinical practice guidelines recommend their use.^{4,70,71} Application of resting hand splints combined with other treatments, including early botulinum toxin injection to wrist and finger flexors, may be beneficial.⁷² Early after stroke, positioning of the hemiplegic shoulder in maximum external rotation for 30 minutes each day either in bed or in a chair can be useful for preventing shoulder contracture.^{73,74} Applying serial casting or static adjustable splints may be beneficial in preventing elbow or wrist contractures, although data are conflicting.^{4,72,75,76} Surgical release of the brachialis, brachioradialis, and biceps muscles is a reasonable option to treat pain and range-of-motion limitations in patients with substantial established elbow flexor contractures.⁷⁷

Ankle plantarflexion contractures after stroke can affect gait quality and safety. The use of an ankle-foot orthosis (AFO) can improve gait in patients with active plantarflexion during the swing phase of gait but also may be beneficial in preventing ankle contracture.⁷⁸ For nonambulatory patients, the use of a resting ankle splint at night, set in the plantigrade position (ankle at 90° and subtalar neutral), or

standing on a tilt table for 30 min/d is probably useful in preventing contracture.⁷⁸

Recommendations: Prevention of Skin Breakdown and Contractures	Class	Level of Evidence
During hospitalization and inpatient rehabilitation, regular skin assessments are recommended with objective scales of risk such as the Braden scale.	I	C
It is recommended to minimize or eliminate skin friction, to minimize skin pressure, to provide appropriate support surfaces, to avoid excessive moisture, and to maintain adequate nutrition and hydration to prevent skin breakdown. Regular turning, good skin hygiene, and use of specialized mattresses, wheelchair cushions, and seating are recommended until mobility returns.	I	C
Patients, staff, and caregivers should be educated about the prevention of skin breakdown.	I	C
Positioning of hemiplegic shoulder in maximum external rotation while the patient is either sitting or in bed for 30 minutes daily is probably indicated.	Ila	B
Resting hand/wrist splints, along with regular stretching and spasticity management in patients lacking active hand movement, may be considered.	Ilb	C
Use of serial casting or static adjustable splints may be considered to reduce mild to moderate elbow and wrist contractures.	Ilb	C
Surgical release of brachialis, brachioradialis, and biceps muscles may be considered for substantial elbow contractures and associated pain.	Ilb	B
Resting ankle splints used at night and during assisted standing may be considered for prevention of ankle contracture in the hemiplegic limb.	Ilb	B

Prevention of Deep Venous Thrombosis

Survivors of acute stroke are at high risk of deep venous thrombosis (DVT) and pulmonary embolism (PE) as a result of a combination of limb immobility and reduced activity level.⁷⁹ Prevention of DVT and PE can be divided into pharmacological and mechanical methods in both ischemic and hemorrhage strokes. Prophylactic treatment is initiated depending on the type of stroke and use of thrombolytic therapy. Therapy usually is continued throughout the rehabilitation stay or until the stroke survivor regains mobility, with few studies examining the optimal duration of prophylaxis. For patients with mild motor impairments who are discharged directly home from the hospital, DVT prophylaxis may not be needed. For patients discharged to an SNF with a stay that extends beyond the active rehabilitation program, the duration of prophylactic treatment remains at the discretion of the treating physician.

Recommendations for the prevention of DVT and PE in ischemic stroke are delineated in great detail in the American College of Chest Physicians' "Antithrombotic Therapy and Prevention of Thrombosis, 9th edition."⁸⁰ One meta-analysis

of 16 trials involving 23043 patients with acute ischemic stroke compared stroke survivors receiving varying amounts of unfractionated heparin (UFH) with control subjects.⁸¹ The use of high-dose UFH (>15000 U/d) was associated with a reduction in PE (odds ratio [OR], 0.49; 95% confidence interval [CI], 0.29–0.83) but also with an increased risk of intracerebral hemorrhage (ICH; OR, 3.86; 95% CI, 2.41–6.19) and extracerebral hemorrhage (ECH; OR, 4.74; 95% CI, 2.88–7.78). Low-dose UFH (<15000 U/D) decreased the thrombosis risk (OR, 0.17; 95% CI, 0.11–0.26) but had no influence on the risk of PE (OR, 0.83; 95% CI, 0.53–1.31). The risk of ICH or ECH was not significantly increased (OR, 1.67; 95% CI, 0.97–2.87 for ICH; OR, 1.58; 95% CI, 0.89–2.81 for ECH) with prophylactic-dose UFH.

Adjusted-dose low-molecular-weight heparin (LMWH) decreased the risk of both DVT (OR, 0.07; 95% CI, 0.02–0.29) and PE (OR, 0.44; 95% CI, 0.18–1.11), but this benefit was offset by an increased risk of ICH (OR, 2.01; 95% CI, 1.02–3.96) and ECH (OR, 1.78; 95% CI, 0.99–3.17). Prophylactic-dose LMWH (defined as 3000–6000 IU/d) reduced the incidence of both DVT (OR, 0.34; 95% CI, 0.19–0.59) and PE (OR, 0.36; 95% CI, 0.15–0.87) without an increased risk of ICH (OR, 1.39; 95% CI, 0.53–3.67) or ECH (OR, 1.44; 95% CI, 0.13–16). For prophylactic-dose LMWH, the number needed to treat to avoid 1 event was 7 for DVT and 38 for PE.

Overall, the guidelines of the American College of Chest Physicians (9th edition) found an estimated reduction in overall mortality of 12 deaths per 1000 individuals receiving either UFH or LMWH compared with no anticoagulation⁸⁰; no form of prophylaxis is 100% effective in preventing venous thromboembolism in this population, however.

A meta-analysis⁸² and a Cochrane systematic review of 9 trials involving 3137 subjects confirmed the superiority of LMWH over UFH.⁸³ Only 1 high-quality cost-effectiveness analysis comparing LMWH with UFH in acutely ill medical subjects (not stroke) demonstrated fewer complications with LMWH at a lower overall cost.⁸⁴

Intermittent pneumatic compression or sequential compression devices are designed to spur blood flow by intermittently applying pressure on the calf muscles and vasculature. One Cochrane systematic review of 2 small studies including 177 subjects demonstrated a nonsignificant trend toward a lower risk of DVT (OR, 0.45; 95% CI, 0.19–1.10) with no significant effect on mortality (OR, 1.04; 95% CI, 0.37–2.89).⁸⁵

Elastic compression stockings, also referred to as graduated compression stockings, are designed to promote venous blood flow by applying a pressure gradient from the ankle more proximally. One large, randomized, clinical trial involving 2518 subjects failed to demonstrate a positive or negative effect on the occurrence of symptomatic proximal DVT or PE.⁸⁶ However, subjects using elastic compression stockings had an increase in skin complications (relative risk [RR], 4.18; 95% CI, 2.4–7.3). One Cochrane systematic review of 2 trials including 2615 subjects demonstrated no significant reduction in DVT (OR, 0.88; 95% CI, 0.72–1.08) or death (OR, 1.13; 95% CI, 0.87–1.47).⁸⁵

The addition of elastic compression stockings to intermittent pneumatic compression has been studied in a few small studies but has failed to demonstrate a positive or negative effect.⁸⁷ Studies in other patient populations have demonstrated

that the combination of elastic compression stockings and pharmacological prophylaxis significantly reduced the incidence of symptomatic or asymptomatic DVT (OR, 0.40; 95% CI, 0.25–0.65). However, the benefit of treatment should be weighed against the increase in skin complications observed with the use of elastic compression stockings.⁸⁸

With respect to hemorrhagic stroke, prophylactic-dose heparin does not increase the risk of recurrent intracranial bleeding significantly, although the overall quality of the evidence is low.⁸⁰ In 1 small study comparing the initiation of prophylactic heparin on the second and fourth hospital days, there were no harmful or beneficial effects on any outcomes.⁸⁹ This study provides very low-quality evidence that early use of prophylactic-dose heparin is safe in stroke survivors with primary ICH.

Comparisons of the effects between UFH and LMWH and the effects of intermittent pneumatic compression and elastic compression stockings have not been done in stroke survivors with primary ICH. Therefore, recommendations are consistent with those of ischemic stroke.⁸⁰

Recommendations: Prevention of DVT	Class	Level of Evidence
In ischemic stroke, prophylactic-dose subcutaneous heparin (UFH or LMWH) should be used for the duration of the acute and rehabilitation hospital stay or until the stroke survivor regains mobility.	I	A
In ischemic stroke, it is reasonable to use prophylactic-dose LMWH over prophylactic-dose UFH for prevention of DVT.	IIa	A
In ischemic stroke, it may be reasonable to use intermittent pneumatic compression over no prophylaxis during the acute hospitalization.	IIb	B
In ICH, it may be reasonable to use prophylactic-dose subcutaneous heparin (UFH or LMWH) started between days 2 and 4 over no prophylaxis.	IIb	C
In ICH, it may be reasonable to use prophylactic-dose LMWH over prophylactic-dose UFH.	IIb	C
In ICH, it may be reasonable to use intermittent pneumatic compression devices over no prophylaxis.	IIb	C
In ischemic stroke, it is not useful to use elastic compression stockings.	III	B
In ICH, it is not useful to use elastic compression stockings.	III	C

Treatment of Bowel and Bladder Incontinence

Urinary incontinence and fecal incontinence are common problems after stroke. Approximately 40% to 60% of stroke patients have urinary incontinence during their acute admission for stroke, falling to 25% by hospital discharge. At 1 year, 15% will remain incontinent of urine.⁹⁰ Age, cognition, and motor impairments are risk factors for bladder incontinence. Fecal incontinence prevalence is ≈40% acutely but diminishes to 20% by discharge from rehabilitation. Age and functional impairment are risk factors for fecal incontinence on admission for stroke.⁹¹ Impaired awareness of

urinary incontinence is correlated with mortality⁹² and the need for nursing home care 3 months after stroke.⁹³ On a positive note, many patients recover continence after stroke. Because of the risk of skin breakdown, the social stigma, and the burden of care associated with incontinence, management of bowel and bladder continence is an essential part of the rehabilitation process.

Although considerable data on the rate of urinary incontinence exist, there is a paucity of published studies on therapeutic interventions to improve rates of continence. The recommendation to remove indwelling urinary catheters within 24 hours is based on the Centers for Disease Control and Prevention recommendations for all hospitalized patients to prevent catheter-associated urinary tract infections and is not specific to stroke.⁹⁴

The studies reported by Pettersen et al⁹² and Myint et al⁹⁵ combined multiple recommendations representing “best practice” for bladder management and applied them to a modest-sized population of stroke patients. Their studies showed success but limited generalizability because of study design. It is impossible to ascertain which of the multiple interventions were responsible for the improvements seen.

Cognitive awareness plays a role in continence and ultimately in overall stroke outcome. There are many types and causes of incontinence, ranging from impaired awareness of the need to void to difficulty with mobility in reaching the bathroom to communication difficulties resulting from aphasia.

We were unable to identify any high-quality studies of treatment for fecal incontinence after stroke, and recommendations are based on the general population of adults.⁹⁶

Recommendations: Treatment of Bowel and Bladder Incontinence	Class	Level of Evidence
Assessment of bladder function in acutely hospitalized stroke patients is recommended.		
A history of urological issues before stroke should be obtained.	I	B
Assessment of urinary retention through bladder scanning or intermittent catheterizations after voiding while recording volumes is recommended for patients with urinary incontinence or retention.	I	B
Assessment of cognitive awareness of need to void or having voided is reasonable.	IIa	B
Removal of the Foley catheter (if any) within 24 hours after admission for acute stroke is recommended.	I	B
It is reasonable to use the following treatment interventions to improve bladder incontinence in stroke patients:	IIa	B
Prompted voiding		
Pelvic floor muscle training (after discharge home)		
It may be reasonable to assess prior bowel function in acutely hospitalized stroke patients and include the following:	IIb	C
Stool consistency, frequency, and timing (before stroke)		
Bowel care practices before stroke		

Assessment, Prevention, and Treatment of Hemiplegic Shoulder Pain

Shoulder pain is common after stroke, with an incidence during the first year of 1% to 22%.^{97,98} The reported prevalence of shoulder pain varies between 5% and 84%, depending on the acuity and definition of shoulder pain used.⁹⁹ The development of shoulder pain after stroke is associated with shoulder subluxation and motor weakness. Importantly, these 2 factors have strong covariance, suggesting that motor impairment may be the more important predictive factor.¹⁰⁰ However, motor weakness is not predictive of pain severity in the hemiplegic shoulder. Spasticity is believed to contribute to the genesis of shoulder pain in some patients, although a causal relationship has not been confirmed. Other predictors of shoulder pain include older age, left hemiplegia, the presence of tactile extinction and reduced proprioception in the painful limb, early complaints of pain, reduced passive shoulder abduction and external rotation of glenohumeral joint, a positive Neer impingement sign (shoulder pain with passive abduction of the internally rotated arm), and tenderness to palpation over the biceps tendon and supraspinatus.^{101–105}

Hemiplegic shoulder pain is multifactorial. Pain is associated with shoulder tissue injury, abnormal joint mechanics, and central nociceptive hypersensitivity. About one third of patients with acute stroke have abnormal ultrasound findings in the hemiplegic shoulder when studied at the time of admission to acute inpatient rehabilitation, including effusion in biceps tendon or subacromial bursa; tendinopathy of biceps, supraspinatus, or subscapularis; and rotator cuff tear.^{106,107} Such findings are more prevalent in the hemiplegic shoulder than in the non-hemiplegic shoulder and in those with more severe hemiplegia, subluxation, spasticity, limited joint range, and shoulder pain.¹⁰⁶ The frequency of abnormal ultrasound findings in the hemiplegic shoulder increases over the course of rehabilitation in patients with more severe motor impairment.^{106,107} Although there is an association between abnormal findings on shoulder ultrasound and hemiplegic shoulder pain in patients with acute stroke, a causal association has not been established. Among patients with acute and chronic stroke with hemiplegic shoulder pain, the presence of shoulder tissue injury on imaging is not associated with the severity of pain.^{108,109}

Patients with stroke-related hemiplegia demonstrate altered movement patterns at certain stages of recovery. In the acute phase of stroke, shoulder subluxation is associated with pain. In those with chronic stroke and hemiplegic shoulder pain, there is capsular stiffness and altered resting position of the scapula in lateral rotation.^{103,110} Compared with those without voluntary movement, patients with some movement in the painful hemiparetic shoulder have a higher rate of shoulder joint tissue injury on magnetic resonance imaging, suggesting that more physical activity promotes injury.¹⁰⁹ However, the relationship between altered kinematics and pain in the hemiparetic shoulder has not been established. For example, shoulder joint kinematics are altered with spasticity, yet there are no clear correlations between reductions in Ashworth and pain scores or reductions in subluxation and pain.¹¹¹ Thus, the exclusive role of peripheral nociceptive pain in the mechanically altered hemiplegic shoulder has been questioned.¹¹²

There is recent evidence supporting both a peripheral and a central neuropathic role for shoulder pain.^{112–114} Patients with

hemiplegic shoulder pain have a higher prevalence of altered somatosensory function with reduced sensory thresholds and decreased kinesthesia than patients without pain and normal control subjects.^{105,115–117} In addition, patients with shoulder pain have higher rates of allodynia and hyperpathia on both the affected and less affected sides than stroke patients without pain.^{116,117} Patients with painful shoulders also have higher heat pain thresholds and lower pain pressure thresholds.^{117,118} Soo Hoo and colleagues¹¹⁸ found lower pain pressure thresholds on the affected and less affected sides in patients with shoulder pain. Somatosensory evoked responses from the affected upper limb differ between stroke patients with and those without shoulder pain.¹¹⁹ Although diagnostically distinct from hemiplegic shoulder pain, complex regional pain syndrome (also called shoulder-hand syndrome) is characterized by allodynia and hyperpathia and includes shoulder pain as a key component. Thus, there is growing recognition that hemiplegic shoulder pain is a syndrome with biomechanical and central nervous system components and overlaps with complex regional pain syndrome.

Interventions to prevent the onset of and to treat shoulder pain in patients with stroke-related hemiplegia include proper positioning, maintenance of shoulder range of motion, and motor retraining. For people in wheelchairs, lap trays and arm troughs might be useful positioning devices to reduce shoulder pain and subluxation. Some suggest that consistent performance of aggressive passive range-of-motion exercises may reduce or prevent later shoulder problems, but the evidence in support of or against this suggestion is missing. Aggressive range of motion of the complex shoulder joint, if done improperly, could do more harm than good. The use of slings, especially during ambulation training to protect the shoulder from traction injury, may be considered, and the use of overhead pulley exercises should be avoided.^{70,120} Research has focused on several adjuvant treatments, including strapping, acupuncture, and neuromuscular electrical stimulation (NMES). There are a few RCTs with mixed results on shoulder strapping for the prevention of shoulder pain after acute stroke.^{121–123} Each study used different strapping (or taping) techniques and measured different pain outcomes. In the largest of these, Pandian and others¹²³ randomized 162 patients with acute stroke to either shoulder taping or sham taping. There was a trend toward a difference in visual analog pain scale and pain-related disability scores over 30 days, but these differences were not statistically or clinically significant. Currently, there is insufficient evidence to support or refute the efficacy of shoulder strapping (taping) for the prevention of hemiplegic shoulder pain.

Acupuncture in combination with standard therapeutic exercise may be a safe and effective adjuvant for the treatment of hemiplegic shoulder pain. This was suggested by Lee and colleagues¹²⁴ in a recent systematic review of this topic. They found 7 RCTs, all showing positive effects. However, they could not recommend concrete conclusions because of the limited number of available trials.

Various types of skin surface electrical stimulation have been evaluated for the treatment of hemiplegic shoulder pain, including transcutaneous electrical nerve stimulation (TENS) and NMES. These modalities have not been evaluated sufficiently, and their efficacy for pain prevention and treatment

remains inconclusive.¹²⁵ The largest RCT to date testing surface NMES to a hemiplegic shoulder showed no effect on pain prevention in patients with acute stroke; however, pain was not a primary outcome measure in this study.¹²⁶ Compliance with the use of surface NMES has been variable in these studies, and surface NMES has been shown to be less well tolerated than intramuscular NMES.^{126–128} Intramuscular NMES for 6 h/d over 6 weeks with 4 implanted electrodes showed efficacy in 2 open-label trials.^{129,130} Pain differences between treatment and control groups remained significant 12 months after treatment, and NMES was more effective in patients with less chronic stroke (defined as <77 months after stroke in this study).^{131,132} Although fully implanted intramuscular stimulators for hemiplegic shoulder have been developed, there are insufficient data to support efficacy to date.¹³³

Corticosteroid injection into glenohumeral joint or subacromial space is commonly used to treat shoulder pain. There are limited studies on the use of steroid injection in the painful hemiplegic shoulder. Observational studies have shown a significant reduction in hemiplegic shoulder pain after either glenohumeral or subacromial injection, but the long-term pain reduction has not been verified.^{134,135} These injections result in superior short-term pain reduction compared with standard care.¹³⁶ There are only 2 randomized trials of shoulder joint injections for pain. Snels and colleagues¹³⁷ showed no significant effect on pain reduction after glenohumeral injection. In contrast, Rah and others¹³⁸ showed a significant reduction in pain after corticosteroid injection compared with placebo. In the latter study, Rah et al selected only patients with shoulder joint pathology that was verified by ultrasonography.

Botulinum toxin injections into the shoulder musculature have shown mixed results in the management of shoulder pain. de Boer and colleagues¹³⁹ showed no impact of botulinum toxin injection into the subscapularis of painful hemiplegic shoulders, whereas Yelnick and colleagues¹⁴⁰ showed significant reductions in pain scores in patients treated for shoulder spasticity. Some investigators have noted reduced pain with shoulder movement after botulinum toxin injections to the pectoralis major and biceps brachii, but others found no change in reported pain scores after pectoralis major injection.^{141–143} Lim et al¹⁴⁴ found botulinum toxin injections to the pectoralis major, infraspinatus, and subscapularis muscles superior to glenohumeral steroid injection. Botulinum toxin injections may decrease shoulder spasticity and pain associated with spasticity-related joint mobility restrictions but are not sufficient to reduce shoulder pain in general.

Suprascapular nerve blocks may be effective in reducing shoulder pain through a reduction of both nociceptive and neuropathic pain mechanisms. A recent randomized, clinical trial showed that suprascapular nerve blocks were superior to placebo injections in reducing hemiplegic shoulder pain for up to 12 weeks after treatment.^{145,146} In another small, comparison study of patients with nonneuropathic hemiplegic shoulder pain, suprascapular nerve blocks were as effective as glenohumeral triamcinolone injections.¹⁴⁷

Surgical tenotomy of the pectoralis major, latissimus dorsi, teres major, and subscapularis muscles may reduce pain in patients with severe hemiplegia and restrictions in shoulder range of motion.¹⁴⁸ In patients with clinical evidence of a central pain component associated with sensory changes,

allodynia, and hyperpathia, medication management with neuromodulating medications may be considered.^{70,120,149}

Recommendations: Assessment, Prevention, and Treatment of Hemiplegic Shoulder Pain	Class	Level of Evidence
Patient and family education (ie, range of motion, positioning) is recommended for shoulder pain and shoulder care after stroke, particularly before discharge or transitions in care.	I	C
Botulinum toxin injection can be useful to reduce severe hypertonicity in hemiplegic shoulder muscles.	IIa	A
A trial of neuromodulating pain medications is reasonable for patients with hemiplegic shoulder pain who have clinical signs and symptoms of neuropathic pain manifested as sensory change in the shoulder region, allodynia, or hyperpathia.	IIa	A
It is reasonable to consider positioning and use of supportive devices and slings for shoulder subluxation.	IIa	C
A clinical assessment can be useful, including:		
Musculoskeletal evaluation	IIa	C
Evaluation of spasticity	IIa	C
Identification of any subluxation	IIa	C
Testing for regional sensory changes	IIa	C
NMES may be considered (surface or intramuscular) for shoulder pain.	IIb	A
Ultrasound may be considered as a diagnostic tool for shoulder soft tissue injury.	IIb	B
Usefulness of acupuncture as an adjuvant treatment for hemiplegic shoulder pain is of uncertain value.	IIb	B
Usefulness of subacromial or glenohumeral corticosteroid injection for patients with inflammation in these locations is not well established.	IIb	B
Suprascapular nerve block may be considered as an adjunctive treatment for hemiplegic shoulder pain.	IIb	B
Surgical tenotomy of pectoralis major, latissimus dorsi, teres major, or subscapularis may be considered for patients with severe hemiplegia and restrictions in shoulder range of motion.	IIb	C
The use of overhead pulley exercises is not recommended.	III	C

Central Pain After Stroke

Central poststroke pain is pain that results from a lesion in the somatosensory system rather than from a peripheral nociceptive or psychogenic cause.^{150,151} Diagnostic criteria include requirements that the pain occur after stroke, be located in an area of the body that corresponds to the lesion in the central nervous system, and not be accounted for by nociceptive or peripheral neuropathic pain.¹⁰⁰ Central pain is classically associated with thalamic stroke (Dejerine-Roussy syndrome) but can result from a lesion anywhere along the spinothalamic and thalamocortical tracts within the central nervous system.¹⁵⁰ Central pain symptoms are usually described as burning or aching and often include

allodynia associated with touch, cold, or movement.^{152–155} Use of diagnostic criteria for central poststroke pain such as those proposed by Klit et al¹⁵¹ can be helpful. The incidence of central poststroke pain is estimated at 7% to 8%, and it typically begins within a few days after stroke, with the majority of patients becoming symptomatic within the first month.^{152,154}

There is limited evidence on the efficacy of proposed treatments for central poststroke pain. Pharmacotherapy combined with therapeutic exercise and psychosocial support is a reasonable approach.¹⁵⁶ Response to treatment is best assessed with standardized serial measurements such as pain diaries, visual analog scales, or pain questionnaires.¹⁵⁷ Pharmacotherapy has relied primarily on antidepressant medications and anticonvulsants. Amitriptyline 75 mg at bedtime has been shown to lower daily pain ratings and to improve global functioning.¹⁵⁸ Lamotrigine can reduce daily pain ratings and cold-induced pain, but only 44% of patients given this medication have a good clinical response.¹⁵⁹ Results for pregabalin have been mixed, with 2 clinical trials finding that daily pain reporting with pregabalin was not significantly better than with placebo.^{160,161} Sleep and anxiety were improved with pregabalin, however. Gabapentin has not been well studied for poststroke central pain but has been effective in other forms of neuropathic pain.^{162,163} Other options for central pain management include carbamazepine and phenytoin, but their usefulness is not well established.^{158,164}

There are few nonpharmacological options for the management of central poststroke pain. TENS was shown to be ineffective in a small trial.¹⁶⁵ Motor cortex stimulation can be given with a surgically implanted dural electrode overlying the motor cortex that is connected to a subcutaneous pulse generator. In several case series, pain reductions of >50% on the visual analog scale were achieved in 50% to 83% of patients, with effectiveness for up to 2 years after implantation.^{166–169} However, cortical stimulator implantation is associated with several complications, including infection, hardware failure, postoperative seizures, and long-term epilepsy. Motor cortex stimulation may be an option for intractable central poststroke pain. Deep brain stimulation has conflicting evidence for the management of central pain and currently cannot be recommended.^{170,171}

Recommendations: Central Pain After Stroke	Class	Level of Evidence
The diagnosis of central poststroke pain should be based on established diagnostic criteria after other causes of pain have been excluded.	I	C
The choice of pharmacological agent for the treatment of central poststroke pain should be individualized to the patient's needs and response to therapy and any side effects.	I	C
Amitriptyline and lamotrigine are reasonable first-line pharmacological treatments.	Ila	B
Interprofessional pain management is probably useful in conjunction with pharmacotherapy.	Ila	C
Standardized measures may be useful to monitor response to treatment.	IIb	C
Pregabalin, gabapentin, carbamazepine, or phenytoin may be considered as second-line treatments.	IIb	B

Recommendations: Central Pain After Stroke (Continued)	Class	Level of Evidence
TENS has not been established as an effective treatment.	III	B
Motor cortex stimulation might be reasonable for the treatment of intractable central poststroke pain that is not responsive to other treatments in carefully selected patients.	IIb	B
Deep brain stimulation has not been established as an effective treatment.	III	B

Prevention of Falls

A great deal of research literature exists on the epidemiology, risk factors, and development of prevention programs for falls in the general population of older adults.¹⁷² Less information is available for individuals with stroke. Falls and their prevention in individuals with stroke require special considerations.¹⁷³ Risk factors, interventions, and prevention programs developed for the community-living older population will not necessarily translate to the population of individuals with stroke. The Balance and Ataxia section provides more discussion.

Up to 70% of individuals with a stroke fall during the first 6 months after discharge from the hospital or rehabilitation facility.¹⁷⁴ Individuals with stroke are also at risk to be repeat fallers and to experience an injury associated with a fall.¹⁷⁵ A larger portion of fractures occurring in individuals with stroke (27%) involve the hip or pelvis compared with <10% of the general population of older adults who fall.¹⁷⁶ The loss of bone mineral density (BMD) associated with stroke may contribute to the higher hip fracture rate for individuals with stroke.¹⁷⁷

In addition to the physical consequences associated with fractures and related injuries, falls have psychological and social consequences. The impairments in balance, gait, motor control, perception, and vision contribute to a heightened fear of falling in individuals with stroke. Studies indicate that 30% to 80% of individuals with stroke report various levels of fear associated with falling and mobility.¹⁷⁸ Fear of falling can lead to reduced levels of physical activity and deconditioning, creating a cascade that may result in greater declines in physical activity, a decrease in ADLs, a loss of independence, fewer community interactions, social isolation, and depression. Ironically, the reduction in physical activity resulting from fear of falling can itself contribute to an increased risk of falls.¹⁷⁹

Risk Factors and Assessment

Evaluation of risk factors is widely recognized as the first step in preventing falls. A systematic review¹⁸⁰ of factors contributing independently to falls in the general older population identified previous falls, low muscle strength, impaired gait, poor balance, and use of specific and multiple medications as the strongest risk factors for falls. Research suggests that risk factors in the stroke population are similar overall but with some differences.¹⁷³ For example, a history of falls before a stroke does not appear to be as strong a risk factor as it is in the general older population.¹⁷³

The probability of falling also increases with the number of risk factors. Tinetti and others¹⁸¹ reported that the 1-year risk of falling among the general elderly population increased from a range of 8% to 19% for individuals with no risk factors to >70% for individuals with ≥4 risk factors.

The assessment of risk factors varies across settings and circumstances. For example, a majority of falls for individuals with stroke that occur during hospitalization are associated with transfers and attempting activities without supervision, whereas the majority of falls for individuals with stroke living in the community are associated with walking.¹⁸²

Numerous fall risk assessment tools are available. A recent systematic review¹⁸³ identified 8 commonly used fall risk assessment tools with existing reliability and validity. The most commonly used assessment instrument in the 43 prevention studies reviewed was the Morse Fall Scale.¹⁸⁴ The Berg Balance Scale has demonstrated good sensitivity and specificity in predicting falls in individuals with stroke.¹⁸⁵ Several federal and professional associations have developed fall prevention toolkits that include risk assessment instruments and protocols (eg, the National Center of Patient Safety Falls Toolkit, the Centers for Disease Control and Prevention Stopping Elderly Accidents, Deaths and Injuries Toolkit, the AHRQ Preventing Falls in Hospitals—A Toolkit for Improving Quality Care, and the AHRQ Step-Up to Stop Falls Toolkit).

Prevention Programs

The most comprehensive assessment of preventing falls in the general population of older adults is the recent Cochrane database review.¹⁷² The evidence specific for fall prevention in individuals with stroke is limited. A recent randomized trial of a multifactorial falls prevention program for individuals with stroke¹⁸⁶ reported no benefit for this intervention compared with usual care among 156 participants. Tai Chi has been found to be more effective than strength and range-of-movement exercises in a clinical trial.¹⁸⁷ A nonrandomized, small-scale, controlled study found a community-based progressive group exercise program that included walking and strength and balance training for 1 hour 3 times a week for participants with mild to moderate hemiparesis to be safe, feasible, and efficacious in a community setting.¹⁸⁸

Recommendations: Prevention of Falls	Class	Level of Evidence
It is recommended that individuals with stroke discharged to the community participate in exercise programs with balance training to reduce falls.	I	B
It is recommended that individuals with stroke be provided a formal fall prevention program during hospitalization.	I	A
It is reasonable that individuals with stroke be evaluated for fall risk annually with an established instrument appropriate to the setting.	Ila	B
It is reasonable that individuals with stroke and their caregivers receive information targeted to home and environmental modifications designed to reduce falls.	Ila	B
Tai Chi training may be reasonable for fall prevention.	Ilb	B

Seizure Prophylaxis

A new seizure diagnosis after stroke can be classified as early (beginning within the first few days of stroke) or late.

A seizure is most likely to arise during the first 24 hours after stroke onset, is usually partial at onset, and has a variable tendency to secondarily generalize. A poststroke seizure is more common with ICH¹⁸⁹ or when the stroke involves cerebral cortex¹⁹⁰; seizures in patients with lacunar stroke are rare.¹⁹¹ Estimates of the percentage of patients having a seizure during the first few days after a stroke range from 2% to 23% in various studies, with the true risk toward the lower end of this range.^{191,192} A minority of such patients will have a recurrent seizure, and status epilepticus is uncommon.¹⁹³

Estimates for the incidence of a seizure developing late after stroke are even more variable, ranging from 3% to 67%.¹⁹² One study found a 1.5% rate of seizures specifically during inpatient admission for stroke rehabilitation.¹⁹⁴ The probability of a late seizure is higher in patients with preexisting dementia.¹⁹⁵ Seizures with onset within 2 weeks of stroke are usually easy to control medically.¹⁹⁶

No data are available to guide the utility of prophylactic administration of antiepileptic drugs after stroke, and limited data are available on the efficacy of antiepileptic drugs in the treatment of stroke patients who have experienced a seizure. Any patient who develops a seizure should be treated with standard management approaches, including a search for reversible causes of seizure and any potential antiepileptic drugs. Subclinical seizures can be difficult to detect unless suspected, so the treating physician might consider pursuing this diagnosis in a patient with otherwise unexplained rapidly shifting sensorium or other deficits or transient fluctuations in vital signs.

Prophylactic administration of antiepileptic drugs to prevent a seizure is not recommended for patients with stroke,¹⁹² including patients with ICH.¹⁹⁷ RCTs are also lacking for the prevention or treatment of seizures in patients with subarachnoid hemorrhage.¹⁹⁸ However, prophylactic therapy with antiepileptic drugs is advocated by some on the basis of theoretical concerns such as an association of increased rate of seizures among subgroups of patients with subarachnoid hemorrhage with selected features such as thicker clot or rebleeding.¹⁹⁸

In all cases, it must be understood that prescribing a new antiepileptic drug carries a significant risk of side effects.^{199,200} Furthermore, some data suggest that prophylactic use of antiepileptic drug therapy may be associated with poorer outcome.^{199–202} The risk-benefit analysis of antiepileptic drug use after a recent stroke includes an important concern that does not pertain to many neurological settings. Evidence suggests that many of the medicines used to treat seizures, including phenytoin and benzodiazepines, dampen some mechanisms of neural plasticity that contribute to behavioral recovery after stroke.^{203–205}

Recommendations: Seizures	Class	Level of Evidence
Any patient who develops a seizure should be treated with standard management approaches, including a search for reversible causes of seizure in addition to potential use of antiepileptic drugs.	I	C
Routine seizure prophylaxis for patients with ischemic or hemorrhagic stroke is not recommended.	III	C

Secondary Stroke Prevention

Stroke shares many risk factors with other forms of cardiovascular disease such as hypertension, smoking, hyperlipidemia, and inactivity.²⁰⁶ With hospitalization for acute stroke brief, it is particularly important to address the secondary prevention of stroke and other cardiovascular diseases during the postacute rehabilitation phase of care. Readers are directed to the most recent AHA/American Stroke Association (ASA) secondary stroke prevention guideline for further information.²⁰⁶

Poststroke Depression, Including Emotional and Behavioral State

In the United States and globally, depression and anxiety are common after stroke and are associated with increased mortality and poor functional outcomes.^{207–214} There is evidence that the likelihood of depression increases with stroke severity,²¹⁵ but the mechanisms of poststroke depression are incompletely understood. Depression has been reported in up to 33% of stroke survivors compared with 13% of age- and sex-matched control subjects,²¹⁶ but reliable estimates of the incidence and prevalence of depression in a stroke cohort are limited.²¹⁷ Predictors of poststroke depression include a history of depression, severe disability, cognitive impairment, previous stroke, a positive family history of psychiatric disorder, and female sex.^{216–220} As poststroke psychosocial issues are studied, greater understanding of the complexity of the problem is obtained. For example, Vickery et al²¹⁴ analyzed how the stability of self-esteem plays a role in the rate of depressive symptoms. The depression and emotionalism section of the 2005 stroke rehabilitation clinical practice guidelines does an excellent job of describing the incidence of poststroke depression and pseudo-bulbar affect.¹⁴⁹ What is clear from the literature is that these issues are real and warrant assessment and treatment as early as possible and on an ongoing basis. The section on poststroke depression in the AHA/ASA “Palliative and End-of-Life Care in Stroke”²²¹ scientific statement gives highlights of prevention, assessment, and treatment. Here, we highlight how poststroke depression affects stroke rehabilitation and recovery and, vice versa, how rehabilitation and exercise affect depression.

Although data are inconclusive as to whether improvement of poststroke depression is independently associated with functional improvement,²²² depression can negatively affect a patient's ability to actively participate in rehabilitation therapies.²²³ It is important to address symptoms early in the rehabilitation process, especially given the recent trend for less time in rehabilitation. Depression frequently coexists with other psychiatric symptoms. Anxiety in particular is found to coexist with depression in the poststroke patient population but frequently goes undiagnosed.²²⁴ Anxiety can create uncomfortable or disabling feelings of worry/fear accompanied by physical symptoms that make participation in therapy more difficult. Shimoda and Robinson²²⁵ reported that generalized anxiety disorder accompanied by poststroke depression delayed recovery from depression, delayed ADL recovery, and reduced

overall social functioning. Unfortunately, few studies have been conducted to address the treatment of and recovery from poststroke generalized anxiety disorder.²²⁶ Anxiety symptoms in poststroke patients should be assessed and treated, particularly in those patients with a diagnosed depressive disorder. Any patient diagnosed with 1 form of mood disorder should be assessed for others.

A review of intervention trials for treatment of poststroke depression yielded no evidence of benefits of psychotherapy in treating depression after stroke.²²⁷ de Man-van Ginkel et al²²⁸ identified additional nursing practices that had a positive impact on reducing depression symptoms, including life review therapy, motivational interviewing, nursing support programs, and physical exercise.

Rehabilitation, Exercise, and Recovery

A study with 49 depressed patients (24 treated for depression and 25 not treated as determined by physician preference) was conducted to evaluate the effects of poststroke depression and antidepressant therapy on the improvement of motor scores and disability.²²⁹ Poststroke depression was found to have negative effects on functional recovery, and the pharmacological treatment of depression was found to counterbalance this effect. Similarly, a study with 55 patients with poststroke major or minor depression found that remission of poststroke depression over the first few months after stroke is associated with greater recovery of ADL function than continued depression.²³⁰ Early effective treatment of depression may have a positive effect on the rehabilitation outcome. No larger-scale studies following up on this line of research were found.

Physical exercise may provide a complementary treatment for depression. Exercise may affect depressive symptoms through a number of mechanisms. For example, the hypothalamic-pituitary-adrenal axis may be dysregulated in depression, resulting in elevated cortisol levels. Exercise can improve regulation of hypothalamic-pituitary-adrenal responses.²³¹ Depression also has direct and indirect consequences on immune function,²³² and regular exercise may serve as a nonpharmacological stimulus for enhancing immune function.²³³ Furthermore, social contact through group exercise may be beneficial for individuals with depression.

Meta-analyses in adults with depression (but without stroke) have shown positive effects of exercise on depressive symptoms. A Cochrane review reported a large clinical effect with a standardized mean difference of -0.82 of physical exercise on depressive symptoms.²³⁴ A systematic review suggested that physical exercise was effective in treating depression, especially in individuals with high baseline levels of depression.²³⁵

In a meta-analysis of 13 studies ($n=1022$ patients), Eng and Reime²³⁶ found that depressive symptoms after stroke were lower immediately after ≥ 4 weeks of exercise (standardized mean difference $= -0.13$ [95% CI, -0.26 to -0.01]). Exercise appeared to have a small beneficial effect on depressive symptoms across both the subacute and chronic stages of stroke recovery, but these effects were not retained after the exercise was terminated. Saunders et al²³⁷ reviewed

8 exercise studies that included a depression outcome in a stroke population and meta-analyzed 3 of these studies. They concluded that the results were inconsistent among the trials. A major criticism is that the majority of the stroke studies used depressive symptoms as a secondary outcome, and as a result, the levels of depressive symptoms varied widely in these studies. Given the strong evidence in nonstroke populations with depression, coupled with the preliminary evidence in stroke populations, exercise may be useful as a potential treatment to reduce depressive symptoms in individuals with stroke.

Depression and other psychological disorders, specifically anxiety, can occur at any time after stroke. Healthcare providers should evaluate these issues during poststroke follow-up visits. One study compared different diagnostic tools to determine whether one was superior over another. Bergersen et al²³⁸ reported that patients and their caregivers fail to discuss psychosocial issues or symptomology with their providers. There are cultural differences in reporting psychosocial issues, resulting in part from perceived cultural morays discouraging personal feelings.²⁰⁹ Varying poststroke assessments on the basis of cultural background is an important consideration specifically in poststroke depression. Nonpharmacological treatment options can provide some successful outcomes. Unfortunately, there are no well-designed RCTs in which various treatment interventions are compared to determine superiority. Because of the complexity of the psychosocial diseases and limited understanding, a number of treatment options should be tried to determine patient-specific effectiveness. This supports the need for ongoing monitoring after treatment.

Medication

Poststroke depression is treatable with a variety of antidepressant medications, with selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants being the most widely studied.^{223,239} Treatment with heterocyclic antidepressant medications and SSRIs appears to be a viable option for poststroke depression, but their absolute or relative efficacy has yet to be fully established.²⁴⁰ In 1 study of 870 veterans with poststroke depression, poststroke SSRI treatment was associated with longer survival. The authors concluded that after a stroke, SSRI initiation or resumption of treatment should be considered as part of a medication therapy management service, especially if the patient has a history of depression or was taking an SSRI before the stroke.²⁴¹ A 2008 Cochrane review analyzing data for 13 pharmaceutical agents, including tricyclic antidepressants, SSRIs, and monoamine oxidase inhibitors, found some benefit of pharmacotherapy in terms of a complete remission of depression and improvement in scores on depression rating scales, but there was also an associated increase in adverse events.²²⁷ The analyses were complicated by a lack of standardized diagnostic and outcome criteria and differing analytic methods. To the best of our knowledge, there have been no studies on the effectiveness of a combined drug intervention (eg, SSRIs) and rehabilitation intervention on recovery outcomes after stroke.

Recommendations: Poststroke Depression, Including Emotional and Behavioral State	Class	Level of Evidence
Administration of a structured depression inventory such as the Patient Health Questionnaire-2 is recommended to routinely screen for poststroke depression.	I	B
Patient education about stroke is recommended. Patients should be provided with information, advice, and the opportunity to talk about the impact of the illness on their lives.	I	B
Patients diagnosed with poststroke depression should be treated with antidepressants in the absence of contraindications and closely monitored to verify effectiveness.	I	B
A therapeutic trial of an SSRI or dextromethorphan/quinidine is reasonable for patients with emotional lability or pseudobulbar affect causing emotional distress.	IIa	A
Periodic reassessment of depression, anxiety, and other psychiatric symptoms may be useful in the care of stroke survivors.	IIa	B
Consultation by a qualified psychiatrist or psychologist for stroke survivors with mood disorders causing persistent distress or worsening disability can be useful.	IIa	C
The usefulness of routine use of prophylactic antidepressant medications is unclear.	IIb	A
Combining pharmacological and nonpharmacological treatments of poststroke depression may be considered.	IIb	A
The efficacy of individual psychotherapy alone in the treatment of poststroke depression is unclear.	IIb	B
Patient education, counseling, and social support may be considered as components of treatment for poststroke depression.	IIb	B
An exercise program of at least 4 weeks duration may be considered as a complementary treatment for poststroke depression.	IIb	B
Early effective treatment of depression may have a positive effect on the rehabilitation outcome.	IIb	B
No recommendation for the use of any particular class of antidepressants is made. SSRIs are commonly used and generally well tolerated in this patient population.	III	A

Poststroke Osteoporosis

BMD and lean tissue mass commonly decline after stroke.^{242–244} Although declines in BMD and lean tissue mass can occur in both limbs, changes on the paretic side are more profound. BMD can decrease by >10% in <1 year in the paretic lower limb.²⁴² Moreover, the decline in BMD, coupled with balance deficits resulting from stroke, increases fracture risk.²⁴⁵ Changes in BMD after stroke are correlated with functional deficits in the paretic limb(s). Jørgensen et al²⁴⁶ assessed 40 patients at 6 days, 7 months, and 1 year after stroke. Seventeen patients were

initially nonambulatory, and 23 were ambulatory. Ambulatory status was predictive of changes in BMD 1 year after stroke. The nonambulatory patients had a 10% reduction in BMD in the paretic lower limb compared with a 3% reduction in BMD in ambulatory patients. Moreover, among the 17 patients who were initially nonambulatory, 12 regained walking ability with assistance 2 months after stroke. Those patients who regained ambulation ability had an 8% reduction in BMD in the paretic lower limb compared with a 13% reduction in those who remained nonambulatory. Pang et al²⁴⁷ found that femur BMD and lean mass were significantly lower and fat mass was significantly higher on the paretic side compared with the nonparetic side in ambulatory men and women who suffered a stroke >1 year earlier. However, the degree to which BMD was preserved in the paretic lower extremity was significantly correlated with 6-minute walk test distance, peak oxygen consumption ($\dot{V}O_2$), and handheld dynamometry. Multiple regression analysis revealed that peak $\dot{V}O_2$ was a significant predictor of paretic limb BMD and lean tissue mass. Paretic upper limbs also demonstrate significant declines in BMD and lean mass after stroke. The decline in BMD and lean mass is associated with paretic upper limb strength assessed by handheld dynamometry.²⁴⁸

The US Preventive Services Task Force²⁴⁹ recommends osteoporosis screening in all women ≥ 65 years of age; women <65 years of age whose fracture risk is greater than or equal to that of older white women with no additional risk factors should also undergo osteoporosis screening. The US Preventive Services Task Force concludes that there is inconclusive evidence to make any osteoporosis screening recommendations for men. Individuals with stroke have an increased risk for osteoporosis, particularly on the paretic side.²⁵⁰ The risk of fracture is also increased in patients with stroke.²⁵¹ In men with stroke, although osteoporosis and fracture risks are higher, no clear guidance on screening can be provided at this time.²⁵² The current US Preventive Services Task Force recommendations are appropriate in the stroke population.

Limited research indicates that increased levels of physical activity such as ambulation and resistance training attenuate the decline in, maintain, or increase BMD and lean tissue mass after stroke.^{245,246,253–257}

field deficit, cognitive changes such as executive dysfunction or memory loss, major depression, sensory deficits, dysarthria, and problems with coordination.^{11,258,259}

Measures of body function tend to be more objective, easier to define, and easier to measure compared with other levels of the World Health Organization's *ICF* but may have less relevance to a patient's function and independence. Limited correlation exists across *ICF* dimensions.^{11,260} The reason is that numerous factors have a greater influence on outcome as one moves from body function/structure to activity limitations, participation restrictions, and quality of life.²⁶¹ During acute stroke management, the focus tends to be more on measures of body function, whereas toward the more chronic phases, the emphasis shifts to activities and participation.¹¹ Regardless of *ICF* dimension, formal standardized and validated measures should be used to the extent possible.

Many methods are available to measure loss of body function/structure. Chief among these is the physical examination. Many scales have been devised.²⁶² Some are global scales that aim to capture all major deficits and to combine the assessment into a single score, whereas others are modality specific. In the United States, the most widely used global assessment of impairment is the National Institutes of Health Stroke Scale, which ranges from 0 to 42, with higher scores indicating more severe loss of body function/structure. Training and formal certification on National Institutes of Health Stroke Scale scoring are widely available, increasing the precision of this measure and permitting the use of this tool by a variety of disciplines. The National Institutes of Health Stroke Scale is a good predictor of short-term and long-term morbidity and mortality²⁶³ and has been found to be sensitive to change in numerous studies. Limitations of the National Institutes of Health Stroke Scale include low granularity for defining differences in level of impairment and insensitivity to many common poststroke deficits such as depression, hand-motor deficits, swallowing, or memory loss.

Many modality-specific measures have been constructed for measuring loss of body function/structure across the many brain neural systems. Common examples include the upper limb motor section of the Fugl-Meyer scale or the Box and Block Test for measuring arm motor deficits; the leg motor section of the Fugl-Meyer scale or gait velocity for measuring leg motor deficits; the Western Aphasia Battery or the Boston Naming Test for language deficits; the Behavioral Inattention Test or The Line Cancellation test for measuring neglect; the Nottingham Sensory Assessment or the sensory section of the Fugl-Meyer scale for measuring somatosensory deficits; the Hamilton Depression Scale or the Beck Depression Inventory II for measuring severity of depression symptoms; and the Mini-Mental Status Exam or Trail Making Tests (A and B) for cognitive deficits. More complete lists of such tests have been compiled.^{11,258} In addition, the National Institute of Neurological Disorders and Stroke has compiled a set of common data elements for each dimension of the *ICF*,³ including the 3 major dimensions of body structures/body functions (impairments), activities (activity limitations), and participation (participation restrictions).

Some scales focus on measures that require specific equipment such as a dynamometer for measuring hand grip strength, various perimeter devices (eg, Humphrey or octopus) for measuring visual field loss, an electric goniometer for measuring

Recommendations: Poststroke Osteoporosis	Class	Level of Evidence
It is recommended that individuals with stroke residing in long-term care facilities be evaluated for calcium and vitamin D supplementation.	I	A
It is recommended that US Preventive Services Task Force osteoporosis screening recommendations be followed in women with stroke.	I	B
Increased levels of physical activity are probably indicated to reduce the risk and severity of poststroke osteoporosis.	Ila	B

Assessment

Level of Disability

Stroke can affect numerous aspects of neural function and structure. Clinically, this most often manifests as weakness, with other common impairments being aphasia, neglect, visual

range of motion, or von Frey filaments for measuring tactile sensory deficits. Robotic devices are receiving increasing attention for their ability to quantify loss of body function/structure,²⁶⁴ in some cases generating data that cannot be obtained by a human examiner.²⁶⁵ Telemedicine may be used by examiners in remote locations to measure level of disability.²⁶⁶

The assessment of body function/structure in a patient recovering from stroke may be performed to predict outcome, to monitor recovery, to monitor response to a new therapy, to guide new treatment decisions, to document clinical status as part of reimbursement, to inform patient stratification such as in selecting postdischarge setting, in the context of a clinical trial, as part of stroke center or rehabilitation ward certification requirements, or in compliance with a stroke care plan protocol. Valid reliable measures have been defined for each of these purposes. Similar considerations apply to choosing the frequency with which impairments are measured.

Assessing Overall Rehabilitation Needs

After acute hospital admission for stroke, patients should have comprehensive assessments of body structures and function, activity limitations, and participation restrictions according to the *ICF*.^{11,267,268} These assessments can be performed concurrently with diagnostic testing as soon as 24 hours after admission, as the patient's medical stability allows. Evaluation of a stroke survivor's rehabilitation needs is best performed by an interprofessional team that can include a physician with expertise in rehabilitation, nurses, physical therapists, occupational therapists, speech/language therapists, psychologists, and orthotists.^{4,149,258} Prvu Bettger and colleagues¹² noted that among acute hospitals participating in the AHA's Get With The Guidelines program, 90% of patients have an assessment for postacute rehabilitation services documented, but little information is available about the nature or reliability of these assessments. If clinically indicated, appropriate postacute rehabilitation settings include outpatient rehabilitation or day rehabilitation programs, skilled nursing-level rehabilitation, long-term acute care hospitals, and acute rehabilitation hospitals.

Selection of the most appropriate level of care requires consideration of many factors, including the severity of residual neurological deficits, resulting activity limitations, cognitive and communicative ability, psychological status, swallowing ability, premorbid functional ability, medical comorbidities, level of family/caregiver support, likelihood of returning to community living, and ability to participate in a rehabilitation program.^{70,269,270} Certain factors such as older age, impaired cognition, lower functional level after stroke, and urinary incontinence are predictors of the need for inpatient rehabilitation care.^{54,271} The presence of neglect syndrome can predict a longer rehabilitation stay and lower functional status at discharge.²⁷² Among patients with less neurological impairment, assessment of balance ability with standardized measures such as the Berg Balance Scale or the Postural Assessment Scale for Stroke can help determine the risk of fall and need for inpatient rehabilitation rather than discharge home with outpatient services.^{273–275} (The Prevention of Falls section provides more information). For patients who can walk, assessment of gait speed with the 10-m walk test can help determine functional ambulatory ability.^{276,277} Risk of fall with ambulation is important for counseling patient and family on safety.

A comprehensive determination of functional abilities appears to be useful before acute hospital discharge with standardized assessments such as the Barthel Index or the Functional Independence Measure (FIM). Both the Barthel Index and the FIM are strong predictors of discharge functional status, discharge destination after inpatient rehabilitation, and length of rehabilitation stay.^{278–281} The FIM is the most commonly used functional measure in the United States because it is tied to the prospective payment system of the Centers for Medicare & Medicaid Services.

There currently is no single functional assessment with measurement properties that is used throughout the entire clinical course of stroke care (acute hospital, inpatient rehabilitation, and outpatient care) for tracking stroke rehabilitation outcome. A computerized questionnaire called the Activity Measure for Post-Acute Care is not specific to stroke but has demonstrated feasibility as such a tool in stroke populations.²⁸² Although it requires cognitive and language ability to complete, proxy responses to the Activity Measure for Post-Acute Care are well correlated with patient responses.²⁸³ Thus, the Activity Measure for Post-Acute Care may prove to be a suitable longitudinal outcome measure for stroke patients, including those with cognitive deficits and aphasia.

ADLs, IADLs, and Disability Measurement

The term ADLs typically refers to routine self-care tasks that people perform as part of their everyday life.²⁸⁴ ADLs are generally subdivided into those associated with personal self-care and fundamental mobility, often referred to as basic ADLs, and tasks involving more complex domestic, community, and leisure activities, referred to as IADLs.²⁸⁵

An evidence-based consensus conference on improving measurement of disability sponsored by the AHRQ concluded that a single consensus definition of disability is not feasible or desirable.²⁸⁶ The AHRQ report contends that the meaning of disability is dependent on context and the purpose for which the definition will be used. The *ICF* uses disability as a generic term that includes aspects of body functions and structure, activity, and participation within the context of the environment and personal/social factors.^{3,287} The recommendations below for ADLs, IADLs, and disability are based on the conceptual approach to disability endorsed by the World Health Organization.³

In the 2005 stroke rehabilitation clinical practice guidelines, there were 2 recommendations on the assessment of function. The first was that a standardized assessment tool be used to evaluate functional status in individuals with stroke. The second recommendation was to consider using the FIM as the standardized assessment for function in individuals with stroke.¹⁴⁹

Over the past decade, there has been substantial progress in 2 general areas pertaining to measurement of function and disability, including ADLs and IADLs. The first is more sophisticated methodological approaches to assessment, specifically the development of methods based on item response theory and computer-adapted testing.²⁸⁸ The second is the recent attention to patient-centered and patient-reported outcome measures. The emphasis on patient-centered and patient-reported measures is related to healthcare reform and the implementation of the Patient Protection and Affordable Care Act.²⁸⁹

New tools for assessment include the Patient-Reported Outcomes Measurement Information System²⁹⁰ and the NIH Toolbox.²⁹¹ Both the Patient-Reported Outcomes Measurement Information System and the NIH Toolbox are designed to help clinicians and healthcare consumers by providing a common platform based on procedures and metrics that will generate outcomes comparable across large populations, including individuals with stroke.

The largest and most comprehensive source of evidence-based reviews and reports focused on stroke rehabilitation is available from the Evidence-Based Review of Stroke Rehabilitation (EBRSR) program supported by the Canadian Stroke Network.^{270,292} Information and the evidence-based reports from EBRSR are available online.^{292a}

Specific to the assessment of ADLs and IADLs (disability), the EBRSR has produced an evidence-based report titled "Outcome Measures in Stroke Rehabilitation."^{292b} All reviewed assessments are classified according to the World Health Organization's *ICF* conceptual framework. The frequently used modified Rankin Scale is included within the Activity/Disability Outcome Measures section. With the use of the *ICF*, each assessment is categorized as providing information at the level of body functions and structure, activities, or participation. All assessment instruments in the EBRSR report are evaluated with 8 criteria. The criteria were derived from a comprehensive review of 413 articles on measurement methodology by the Health Technology Assessment Program.²⁹³ The criteria include operationally defined ratings for appropriateness, reliability, validity, responsiveness, precision, interpretability, acceptability, and feasibility. Appendix 2 includes measures reviewed in the EBRSR report as of November 2012.

Assessment Challenges

The instruments included in Appendix 2 and the evidence-based reviews in the EBRSR are based on traditional measurement models. As noted above, new assessments are being developed with the use of item response theory and computer-adapted testing. These assessments are difficult to evaluate with the traditional criteria such as validity and reliability normally used in evidence-based reviews. For example, Hsueh and colleagues³²⁹ reported the development of a computer-adapted test for evaluating ADLs in individuals with stroke referred to as the ADL-CAT (computer-adapted test). The authors report the ADL-CAT produced scores that were highly correlated with traditional ADL measures such as the Barthel Index but could be completed in one-fifth the time required to administer the Barthel Index.³²⁹ New or refined criteria consistent with advances in measurement approaches need to be developed and incorporated into existing levels of evidence hierarchies to accommodate the evaluation and evidence-based reviews of assessments.

Another challenge in establishing functional assessment guidelines is how to incorporate the growing emphasis on patient reported and patient-centered measures within the assessment of ADLs, IADLs, and other disability measures. The solution to this challenge extends beyond simply asking patients or consumers to respond to traditional ADL questions such as "Can you put on an article of clothing?" Rather, it requires patients and other stakeholders to be active partners in the assessment process and to help identify the items and outcomes that should be measured. Until computer-adapted tests (eg, ADL-CAT) for ADLs and

IADLs become routine in practice, a combination of assessments such as a basic ADL measure (eg, the 10-item Barthel Index)³³⁰ or the FIM and an IADL measure (eg, the 15-item Frenchay Activity Index)³³¹ is recommended to capture the broad spectrum of ADL function. Recently, a Rasch analysis was used to validate a combined measure of basic and extended daily life functioning after stroke.³³² Even those recovering from mild stroke or transient ischemic attack (eg, those scoring 100 on the Barthel Index) continue to demonstrate deficits in health status. Although basic ADL measures may not be sufficiently sensitive to change among the least impaired stroke survivors, the IADL assessment tool will likely be more sensitive to these more subtle deficits at discharge and provide useful information for discharge planning.

Recommendations: Assessment of Disability and Rehabilitation Needs	Class	Level of Evidence
It is recommended that all individuals with stroke be provided a formal assessment of their ADLs and IADLs, communication abilities, and functional mobility before discharge from acute care hospitalization and the findings be incorporated into the care transition and the discharge planning process.	I	B
It is recommended that all individuals with stroke discharged to independent community living from postacute rehabilitation or SNFs receive ADL and IADL assessment directly related to their discharge living setting.	I	B
A functional assessment by a clinician with expertise in rehabilitation is recommended for patients with an acute stroke with residual functional deficits.	I	C
Determination of postacute rehabilitation needs should be based on assessments of residual neurological deficits; activity limitations; cognitive, communicative, and psychological status; swallowing ability; determination of previous functional ability and medical comorbidities; level of family/caregiver support; capacity of family/caregiver to meet the care needs of the stroke survivor; likelihood of returning to community living; and ability to participate in rehabilitation.	I	C
It is reasonable that individuals with stroke discharged from acute and postacute hospitals/centers receive formal follow-up on their ADL and IADL status, communication abilities, and functional mobility within 30 days of discharge.	IIa	B
The routine administration of standardized measures can be useful to document the severity of stroke and resulting disability, starting in the acute phase and progressing over the course of recovery and rehabilitation.	IIa	C
A standardized measure of balance and gait speed (for those who can walk) may be considered for planning postacute rehabilitation care and for safety counseling with the patient and family.	IIb	B

Assessment of Motor Impairment, Activity, and Mobility

Motor impairments are common after stroke and occur when the stroke lesion includes the corticospinal system, that is, the motor cortical areas and the corticospinal tract.³³³ Indeed, the

extent of damage to the corticospinal system is predictive of motor outcomes and response to treatment.^{334–336} Assessment of motor impairments enables the clinician to understand which aspects of movement and motor control are disrupted after stroke. Assessment of activity such as upper extremity function, balance, and mobility is used to quantify the functional consequences of the motor impairments. Accurate assessment provides prognostic information^{337–341} and guides the selection of motor interventions and the tailoring of these interventions to each individual.²⁹⁴

Assessment of motor impairments and activity is critical for delivering efficient, high-quality rehabilitation services to individuals with stroke. Assessment results are used to determine who needs further services, what types of services are required, what is the most appropriate setting for those services, which interventions to select, how to tailor the interventions to individual patients, and whether the rehabilitation services are achieving the desired outcomes.^{342–344} When standardized assessments are implemented within and across facilities, measures that are familiar and clinician friendly and meet the clinical needs of the service are generally implemented most easily.^{345–347}

Technology to objectively measure real-world activity has been emerging over the past decades. Alternatively, clinicians have relied on self-report measures to gain insight into what a person is doing in daily life. The assumption that clinic performance is equivalent to outside-of-clinic performance may not be true.³²¹ Whereas patient-reported outcomes allow a more patient-centered approach, some self-report measures are prone to reporting biases.^{348,349} Commercially available devices to measure movement when people are outside the rehabilitation clinic are now readily available and becoming more user friendly. These devices include wrist-worn accelerometers,^{294,326} ankle-worn accelerometers,³²⁵ step-activity monitors,^{328,350} and the more economical alternative, pedometers.³²⁷ Recording movements allow the clinician to measure the quantity and sometimes the types of movements occurring in everyday life.

Recommendations: Assessment of Motor Impairment, Activity, and Mobility	Class	Level of Evidence
Motor impairment assessments (paresis/muscle strength, tone, individuated finger movements, coordination) with standardized tools may be useful.	IIb	C
Upper extremity activity/function assessment with a standardized tool may be useful.	IIb	C
Balance assessment with a standardized tool may be useful.	IIb	C
Mobility assessment with a standardized tool may be useful.	IIb	C
The use of standardized questionnaires to assess stroke survivor perception of motor impairments, activity limitations, and participation may be considered.	IIb	C
The use of technology (accelerometers, step-activity monitors, pedometers) as an objective means of assessing real-world activity and participation may be considered.	IIb	C
Periodic assessments with the same standardized tools to document progress in rehabilitation may be useful.	IIb	C

Assessment of Communication Impairment

Communication is a vital aspect of daily functioning, and stroke frequently results in communication impairment. One million people in the United States are estimated to have aphasia, commonly as a result of stroke.³⁵¹ Communication impairment can negatively affect participation in life activities immediately after the stroke and can result in long-term deficits. It is important to identify problems early with a thorough and holistic assessment. It is equally important to identify strengths and compensatory strategies that can enable the patient to maximize independence and to reenter life activities with as much competency and confidence as possible.

In recent years, more attention has been given to incorporating the *ICF* framework and principles into the assessment of communication. Communication is required for most daily activities, so everyday life can be significantly affected by impairment. In previous years, assessment focused on disability; now attention is focused on maximizing quality of life and participating in daily activities. Additionally, caregivers are increasingly included in the evaluation process because their skill and attitude have a significant impact on creating successful communication exchanges.

Telerehabilitation is becoming an accepted alternative to face-to-face communication assessment for people with communication impairment; however, telerehabilitation requires adequate technology. Multiple studies have demonstrated that telepractice for communication assessment is feasible and effective.^{352–354}

Recommendations: Assessment of Communication Impairment	Class	Level of Evidence
Communication assessment should consist of interview, conversation, observation, standardized tests, or nonstandardized items; assess speech, language, cognitive-communication, pragmatics, reading, and writing; identify communicative strengths and weaknesses; and identify helpful compensatory strategies.	I	B
Telerehabilitation is reasonable when face-to-face assessment is impossible or impractical.	IIa	A
Communication assessment may consider the individual's unique priorities using the <i>ICF</i> framework, including quality of life.	IIb	C

Assessment of Cognition and Memory

Cognitive impairment is found in a substantial portion of stroke survivors, affecting more than one third of stroke survivors at 3 and 12 months after stroke.³⁵⁵ These impairments persist in many individuals for years^{356,357} and are associated with poor long-term survival, higher disability, and greater institutionalization rates. Tatemichi et al³⁵⁸ found that the RR for dependent living associated with cognitive impairment was 2.4 at 3 months after stroke after adjustment for age and physical impairment. Another study found the RR of death associated with dementia 5 years after stroke was 3.11 (95% CI, 1.79–5.41) after adjustment for the effects of demographic factors, cardiac disease, severity of stroke, stroke type, and recurrent stroke.³⁵⁹ The cognitive domains most likely to be defective in patients with stroke compared with

control subjects were memory, orientation, language, and attention. Because physical and cognitive impairments after stroke have independent prognostic implications, evaluation of both domains should be routine in the clinical care of stroke patients. Prospective studies have shown that cognitive status is an important determinant of poststroke success. The Neurobehavioral Cognitive Status Examination is a brief screening tool that assesses cognition in the ability areas of language, constructions, memory, calculation, and reasoning. A small prospective study found that the Neurobehavioral Cognitive Status Examination both provides a rapid and sensitive measure of cognitive function and appears to predict functional status change as a result of inpatient stroke rehabilitation.³⁶⁰ A formal neuropsychological examination (including assessment of language, neglect, praxis, memory, emotional responses, and specific cognitive syndromes) may be helpful after the detection of cognitive impairment with a screening instrument. Neuropsychological protocols must be sensitive to a wide range of abilities, especially the assessment of executive and attentional functions. Brief mental status scales inadequately assess executive skills and other higher-level cognitive functions. Specific areas that should be included in this type of assessment include the following:

- Processing speed
- Simple attention and complex attention (“working memory”)
- Receptive, expressive, and repetition language abilities
- Praxis (performing skilled actions such as using a tool)
- Perceptual and constructional visual-spatial abilities, including issues related to visual fields and neglect
- Memory, including language-based memory and visual-spatial memory, and differentiating learning, recall, recognition, and forced-choice memory
- Executive functioning, including awareness of strengths and weaknesses, organization and prioritization of tasks, task maintenance and switching, reasoning and problem solving, error awareness and safety judgment, and emotional regulation

Recommendations: Assessment of Cognition and Memory	Class	Level of Evidence
Screening for cognitive deficits is recommended for all stroke patients before discharge home.	I	B
When screening reveals cognitive deficits, a more detailed neuropsychological evaluation to identify areas of cognitive strength and weakness may be beneficial.	Ila	C

Sensory Impairments, Including Touch, Vision and Hearing

Stroke may result in a variety of different types of sensory impairment such as loss of vision, touch, proprioception, hearing, and others. Sensory impairments are often assessed through physical examination, although methods exist for more precise measurement of certain sensory deficits such as automated perimetry for visual field loss or audiometry for hearing loss. Although these are not routinely used, such testing may be useful when a detailed understanding of sensory impairment is needed.

Various forms of sensory deficit are commonly seen after stroke. For example, somatosensory deficits are present

in 45%²⁵⁹ to 80%³⁶² of patients, and visual field loss occurs in roughly 30%³⁶³ (estimates range from 15%²⁵⁹–52%³⁶⁴) of patients. The high degree of connectivity³⁶⁵ in the human brain not only results in loss of function directly in the affected sensory modality but also affects complex behaviors that require distributed multimodal processing such as fine motor control.^{362,366} As a result, sensory impairments are directly linked to activity limitations and participation restrictions after stroke³⁶⁷ and can improve with therapeutic intervention,³⁶⁸ particularly those based on multimodal interventions such as virtual reality³⁶⁹ and augmented reality.³⁷⁰

Somatosensory Impairments

Somatosensory impairments include tactile, pain, temperature, pressure, vibration, proprioception, stereognosis, and graphesthesia. Tactile deficits may be the most common form of sensory deficit after stroke.³⁶⁷ In the months after a stroke, patients show substantial but variable somatosensory recovery, especially for proprioception.³⁷¹ Studies of experimental stroke in primates^{372,373} and rats³⁷⁴ describe the neurobiological basis of sensory recovery after stroke, with overall similar findings in human subjects scanned with functional magnetic resonance imaging.^{375,376} Assessment of sensory deficits remains largely a matter of bedside examination³⁷⁷; however, sensory scales are under study,^{378,379} and new devices can quantify deficits.^{380,381}

Visual Impairments

The most common visual impairment after stroke is visual field loss, affecting ≈30% of stroke survivors.³⁶³ Vision plays a central role in many human functions, so a reduction in vision can affect many roles, quality of life, motivation, and social behaviors.³⁸² Although assessment of visual field loss is most often obtained with confrontation methods at the bedside, automated perimetry methods are more sensitive and precise and thus may be preferred in settings where such clarity is deemed important such as evaluation for driving.³⁶⁴ Some degree of spontaneous restoration of visual fields generally occurs after stroke. However, the percentage of patients who achieve significant recovery is uncertain, with estimates ranging from 7% to 85%,³⁸³ and the degree of recovery is variable.³⁶⁴ As with many features of spontaneous behavioral recovery after stroke, gains are highest early after the injury, with the maximum period of spontaneous recovery of visual fields being reported to be in the first 2 to 10 days,³⁸⁴ the first month,³⁸⁵ or the first 3 months.³⁶³ Numerous other forms of visual impairment may be seen after stroke such as abnormal eye movements, reduced visual acuity, diplopia, impaired color vision, difficulty with reading, and deficits in higher-order visual processing.

Hearing Impairments

Stroke can also result in acute hearing loss. This may be present in as many as 21% of patients with posterior circulation ischemia,³⁸⁶ often resulting from ischemia in the distribution of the anterior inferior cerebellar artery, and in most cases is attributable to infarction in the inner ear. As a result, stroke-related hearing loss is usually accompanied by vertigo and often with additional deficits related to brainstem/cerebellar infarction.³⁸⁷ Audiometry

is more sensitive than bedside assessment of hearing loss. Neurotologic testing may provide insights by characterizing and measuring associated forms of vestibular dysfunction. Most patients show partial or complete recovery by 1 year after stroke.³⁸⁸

Recommendation: Sensory Impairments, Including Touch, Vision, and Hearing	Class	Level of Evidence
Evaluation of stroke patients for sensory impairments, including touch, vision, and hearing, is probably indicated.	Ila	B

Sensorimotor Impairments and Activities

Dysphagia Screening, Management, and Nutritional Support

Dysphagia is common after stroke, affecting 42% to 67% of patients within 3 days after stroke. Of these patients, about half aspirate, and one third of those patients develop pneumonia.³⁸⁹ Dysphagia or aspiration can lead to pneumonia, malnutrition, dehydration, weight loss, and overall decreased quality of life. Aspiration may be “silent” or “occult” and not clinically obvious. Early identification through screening can reduce the risk of developing these adverse health consequences.³⁸⁹ Additionally, observational studies suggest that dysphagia screening reduces the risk of pneumonia.³⁹⁰

A systematic review of 8 studies demonstrated that the odds of being malnourished were increased if dysphagia was present after stroke.³⁹¹ Despite the potential consequences of dysphagia, a review of nursing nutritional care concluded that a functional, supportive, and educational nursing nutritional role was essential, but little evidence was of sufficient quality to support policy and practice development or to inform education.³⁹²

In 2012, a group of dysphagia experts came to the consensus that early dysphagia screening should be conducted and that although no one screening tool can be recommended, a valid tool should be used.³⁹³ Additional systematic reviews and studies also support early screening for dysphagia. However, because dysphagia screening has not been well standardized and its utility has not been established rigorously in RCTs, it has been removed from The Joint Commission performance standards and from Get With The Guidelines–Stroke performance measures. Nonetheless, it remains an important component of clinical care. Therefore, we include the same recommendation that appears in the most recent “Guidelines for the Early Management of Patients With Acute Ischemic Stroke.”³⁹⁴

Once dysphagia or aspiration risk has been identified, a clinical bedside evaluation can provide valuable diagnostic information about the swallow mechanism and how to proceed with managing the patient. However, a bedside evaluation alone cannot predict the presence or absence of aspiration because patients can aspirate without overt clinical signs or symptoms.³⁹⁵

Instrumental evaluation (videofluoroscopy, fiberoptic endoscopic evaluation of swallowing, or fiberoptic endoscopic evaluation of swallowing with sensory testing)

allows the clinician to visualize swallow physiology, thus determining the presence or absence of aspiration, the quantity of aspiration, and the physiological or structural causes for dysphagia. This information is necessary for forming an appropriate and effective treatment plan, which can include swallow therapy and diet recommendations.^{396–398} There is no consensus in the literature on a preferred instrumental study. Both videofluoroscopy and fiberoptic endoscopic evaluation of swallowing can be used to evaluate the swallow mechanism.

Additionally, a large cohort study was completed, showing that fiberoptic endoscopic evaluation of swallowing with sensory testing is a relatively safe procedure for evaluating the sensory and motor aspects of dysphagia. Clinical judgment should be used to weigh the advantages and disadvantages of each study for each individual patient.³⁹⁹

Multiple systematic reviews showed that behavioral interventions, including “swallowing exercises, environmental modifications such as upright positioning for feeding, safe swallowing advice, and appropriate dietary modifications,”⁴⁰⁰ should be considered for the management and treatment of dysphagia.^{400,401} A group of dysphagia and swallow rehabilitation experts reviewed 10 principles of neural plasticity and discussed how they should be incorporated into dysphagia rehabilitation strategies and interventions to promote evidence-based practice.⁴⁰² Other therapies considered in systematic reviews, including drug therapy, NMES, pharyngeal electric stimulation, physical stimulation, transcranial direct current stimulation (tDCS), and transcranial magnetic stimulation, have no conclusive evidence supporting their use in dysphagia treatment.⁴⁰⁰ Additionally, acupuncture may be a beneficial alternative treatment of dysphagia.⁴⁰³ Cohort studies have shown that oral hygiene protocols may help reduce aspiration pneumonia after stroke.^{404,405}

Recently, there have been a series of clinical trials called the Feed or Ordinary Diet (FOOD) trials, which are large, well-designed RCTs that address when and how to feed patients after stroke.^{406–408} As a result of underrecruitment, definitive conclusions cannot be made; however, these studies and a Cochrane review⁴⁰⁰ offer much information.

Nutritional supplements are recommended only for patients with malnutrition or those at risk of malnutrition. Routine oral nutritional supplements are not associated with improved functional outcome at 6 months after stroke. This clinical trial has found that few participants (8%) were malnourished at baseline and that supplements may contribute to hyperglycemia if the patient is not malnourished.⁴⁰⁸

Early tube feeding (started within 7 days) may increase the survival of dysphagic patients who cannot safely eat by mouth; however, this may keep patients alive “in a severely disabled state when they otherwise would have died.”⁴⁰⁷ Therefore, to reduce case fatality, providers should initiate early tube feeds; however, they can wait up to 7 days after a stroke to initiate tube feeds, especially when conversations about the goals of care are needed. Tube feeds via nasogastric route are reasonable for the first 2 to 3 weeks after stroke unless there is a strong reason to opt for percutaneous endoscopic gastrostomy placement (eg, cannot pass a nasogastric tube).⁴⁰⁷

Early percutaneous endoscopic gastrostomy placement is not supported for stroke patients.⁴⁰⁶ After this time period, percutaneous endoscopic gastrostomy placement is recommended because it is associated with fewer treatment failures, higher feed delivery, and improved albumin concentration.⁴⁰⁰

Recommendations: Dysphagia Screening, Management, and Nutritional Support	Class	Level of Evidence
Early dysphagia screening is recommended for acute stroke patients to identify dysphagia or aspiration, which can lead to pneumonia, malnutrition, dehydration, and other complications.	I	B
Dysphagia screening is reasonable by a speech-language pathologist or other trained healthcare provider.	Ila	C
Assessment of swallowing before the patient begins eating, drinking, or receiving oral medications is recommended.	I	B
An instrumental evaluation is probably indicated for those patients suspected of aspiration to verify the presence/absence of aspiration and to determine the physiological reasons for the dysphagia to guide the treatment plan.	Ila	B
Selection of instrumental study (fiberoptic endoscopic evaluation of swallowing, videofluoroscopy, fiberoptic endoscopic evaluation of swallowing with sensory testing) may be based on availability or other considerations.	Ilb	C
Oral hygiene protocols should be implemented to reduce the risk of aspiration pneumonia after stroke.	I	B
Enteral feedings (tube feedings) should be initiated within 7 days after stroke for patients who cannot safely swallow.	I	A
Nasogastric tube feeding should be used for short term (2–3 weeks) nutritional support for patients who cannot swallow safely.	I	B
Percutaneous gastrostomy tubes should be placed in patients with chronic inability to swallow safely.	I	B
Nutritional supplements are reasonable to consider for patients who are malnourished or at risk of malnourishment.	Ila	B
Incorporating principles of neuroplasticity into dysphagia rehabilitation strategies/interventions is reasonable.	Ila	C
Behavioral interventions may be considered as a component of dysphagia treatment.	Ilb	A
Acupuncture may be considered as a adjunctive treatment for dysphagia.	Ilb	B
Drug therapy, NMES, pharyngeal electrical stimulation, physical stimulation, tDCS, and transcranial magnetic stimulation are of uncertain benefit and not currently recommended.	III	A

Nondrug Therapies for Cognitive Impairment, Including Memory

Impairments in multiple domains of cognition, including attention, processing speed, executive function, verbal and visual memory, language, and perception, occur frequently after stroke. Stroke doubles an individual's risk for dementia (including Alzheimer disease).⁴⁰⁹

Cognitive rehabilitation has been the traditional nonpharmacological method to treat cognitive impairment and has been defined as a “systematic, functionally-oriented service of therapeutic cognitive activities, based on an assessment and understanding of the person's brain-behavior deficits.”⁴¹⁰ These treatments are directed at the restoration or reestablishment of cognitive activity, the acquisition of strategies to compensate for impaired cognitive function, and the use of adaptive technique or equipment for increasing independence. Few studies have assessed interventions for cognitive deficits in the IRF environment. An RCT (n=83 at >4 months after stroke) compared a multicomponent cognitive therapy and graded activity training with cognitive therapy alone over 12 weeks and demonstrated that the multicomponent therapy exceeded the cognitive therapy in fatigue reduction and improved physical endurance.⁴¹¹ A systematic review⁴¹² published in 2011 of cognitive rehabilitation in stroke that searched guidelines in stroke management, other systematic reviews, and clinical RCTs concluded that compensatory strategies can be used to improve memory outcomes. However, use of an external memory aid is in itself a memory task, so those with the greatest need also have the greatest problems using them. One solution to this problem has been the development of a paging system whereby a paging service with a customized set of reminders and appropriate date and time sends out reminders to the individual pager that is carried by the person who needs to be reminded. Recently, this idea has been modernized by the use of text message reminders to one's mobile device. The use of a paging system can significantly reduce everyday failures of memory and planning in stroke survivors. However, there was not enough evidence from RCTs to determine whether cognitive rehabilitation for memory problems after stroke is helpful.

Recently, attention has focused on the application of physical activity and exercise to improve cognitive function after stroke. Meta-analysis suggests that physical activity has a protective effect against cognitive decline⁴¹³ and may improve cognitive function in older adults without cognitive impairment.⁴¹⁴ A number of mechanisms have been suggested to explain the effects of exercise on cognition after stroke, including the increase in cerebral blood volume, increased expression of growth factors such as brain-derived neurotrophic factor, and a positive effect on depressive symptoms, which may mediate an improvement in cognitive performance.⁴¹⁵

In animal models, a stimulating and enriched environment has been shown to improve neurobehavioral function and learning after stroke.⁴¹⁶ Although it is not yet known exactly what type of environment might provide optimal stimulation for a person who has had a stroke, it has been suggested that the setting should be conducive to participating in physical activity and cognitive and social activities.⁴¹⁷

Cognitive Rehabilitation

Systematic reviews that include people with both traumatic brain injury and stroke are generally more positive on the benefits of cognitive rehabilitation⁴¹⁸ than those involving people with stroke alone.^{419–421} This may be due in part to the smaller number of stroke-only studies and the confounding factors of age and vascular involvement with stroke. A Cochrane review of 6 RCTs found a benefit of cognitive rehabilitation after stroke on some aspects of attention deficits at the end of the treatment period.⁴²⁰ Not all aspects of attention are similarly affected; attention training had a positive effect on divided attention immediately after the intervention (4 studies) but no effect on selective attention (6 studies), alertness (4 studies), or sustained attention (4 studies).⁴²⁰ Two cognitive rehabilitation RCTs found improvements in subjective measures of attention⁴²² and mental slowness⁴²³ after stroke immediately after treatment and at follow-up.

The European Federation of Neurological Societies guidelines on cognitive rehabilitation⁴²⁴ summarized a number of publications related to memory rehabilitation interventions without external memory aids, rehabilitation interventions with nonelectronic external memory aids, and rehabilitation interventions with assistive electronic technologies (the specific number of studies identified and reviewed was not given).

They concluded the following:

- That memory strategies without electronic aids are possibly effective (Level C recommendation)
- That specific learning strategies such as errorless learning are probably effective (Level B recommendation)
- That nonelectronic external memory aids such as diary or notebook keeping are possibly effective (Level C recommendation)
- That electronic external memory devices such as computers, paging systems, and portable voice organizers are probably effective (Level B recommendation)
- That the use of virtual environments has shown positive effects on verbal, visual, and spatial learning and that memory training in virtual environments is rated as possibly effective (Level C recommendation)
- That a direct comparison of memory training in virtual environments versus nonvirtual environments is still lacking and no recommendation can be made as to the specificity of the technique

An updated review of the literature (2003–2008)⁴¹⁸ concluded that (1) for individuals with mild memory impairments, memory strategy training, including the use of internalized strategies (eg, visual imagery) and external memory compensations (eg, notebooks), is recommended as a practice standard; (2) for individuals with severe memory deficits, the use of external compensations, including assistive technology, with direct application to functional activities is recommended as a practice guideline; and (3) for individuals with severe memory impairments, errorless learning techniques may be effective for learning specific skills or knowledge, although with limited transfer to novel tasks or reduction in overall functional memory problems.

However, a recent Cochrane meta-analysis⁴²⁵ with 13 cognitive rehabilitation RCTs reported no benefit to executive

functioning after stroke, whereas other systematic reviews using a broader range of evidence have suggested some limited evidence.^{426,427} Current studies are small and have highly varied content, making comparisons difficult. Notably, an RCT delivered strategies focused on problem solving by 3 methods (face to face, online, and computer training) and found that although all improved problem-solving and IADL abilities, the face-to-face training group resulted in the most improvement in problem-solving self-efficacy.⁴²⁸ Another RCT⁴²⁹ found that using a pager was effective in increasing goal attainment (ie, medication and appointments) but that stroke participants' performance returned to baseline levels when the pager was discontinued. In contrast, specific aspects of memory (eg, visual-spatial recall, subjective memory experience, verbal and prospective memory, working memory, and attention) have been shown to improve after stroke in 6 different controlled trials that used very diverse cognitive training strategies.^{430–435}

A systematic review of the literature (1995–2011) focused specifically on information and communication technology tools for individuals with acquired brain injury, including stroke,⁴³⁶ reviewed 5 studies that addressed memory problems. The quality of the studies was so low that it was not possible to determine whether the tools were beneficial.

Only 2 studies have examined the effects of tDCS on attention in stroke patients.^{437,438} The first study⁴³⁸ found that anodal tDCS over the left dorsolateral prefrontal cortex was associated with enhanced complex attention (working memory) performance. The second study⁴³⁷ found that noninvasive anodal tDCS applied to the left dorsolateral prefrontal cortex improved attention compared with sham stimulation. Although improved attention may result in improved memory because people are better able to initially register information, neither addressed whether the performance benefits resulted in improved memory learning and retention.

In summary, most cognitive rehabilitation programs use a variety of activities, including practice requiring attention, planning or working memory with pencil and paper or computerized activities, and teaching of compensatory strategies. Although a growing number of RCTs have addressed immediate effects on standardized psychobehavioral tests, few studies have assessed the durability of treatment effects or relevance to everyday functioning.

Exercise

Cumming et al⁴¹⁵ performed a systematic review through 2011 and found 12 RCTs and controlled, clinical trials that studied the effects of a physical activity or exercise-based intervention on cognitive function in stroke. They concluded that there are reasonably consistent and relatively small positive effects of exercise on cognition, with some studies finding specific positive effects on memory. However, the pool of studies identified was small, and methodological shortcomings were widespread.

Because most studies measured cognition or memory as a secondary outcome, there was a wide range of baseline cognitive abilities, including those without cognitive impairment. The dose and content of the exercise protocols have

been highly diverse,^{415,440,441} preventing recommendations on the optimal intensity or timing. Although no longitudinal exercise or physical activity studies have been undertaken to prevent cognitive impairment or dementia after stroke, it would seem reasonable to extend the results of studies in older adults that suggest a protective effect of exercise on cognitive decline.⁴¹³

Enriched Environment

An RCT that modified the stroke rehabilitation environment with the provision of a computer with Internet, books, games, virtual reality gaming technology, and encouragement from staff to use the activities increased the engagement of patients with cognitive activities and reduced time spent inactive and alone.⁴¹⁷ Särkämö et al⁴⁴² performed a single-blind RCT to determine whether listening to music everyday can facilitate the recovery of cognitive functions after stroke. Two months of daily listening (95 minutes daily) to self-selected music after acute stroke improved verbal memory, focused attention, and depressive symptoms compared with listening to an audio book or not listening to music.⁴⁴²

Four weeks of playing virtual reality games for 30-minute sessions 3 times weekly improved visual attention and short-term visuospatial memory in a very small RCT of patients early after stroke.⁴⁴³ These games required primarily paretic arm movements (eg, raise a hand to stop soccer balls from entering the goal).

Recommendations: Nondrug Therapies for Cognitive Impairment, Including Memory (Continued)	Class	Level of Evidence
Exercise may be considered as adjunctive therapy to improve cognition and memory after stroke.	IIb	C
Virtual reality training may be considered for verbal, visual, and spatial learning, but its efficacy is not well established.	IIb	C
Anodal tDCS over the left dorsolateral prefrontal cortex to improve language-based complex attention (working memory) remains experimental.	III	B

Use of Drugs to Improve Cognitive Impairments, Including Attention

Several medications are used to treat general cognitive disorders, but little literature addresses their use for poststroke cognitive deficits. Dextroamphetamine has been studied for poststroke motor recovery,⁴⁴⁴ but no studies have substantiated its use for cognitive disorders. Although the effect of methylphenidate in 1 small trial might rely partly on an improvement in attention and effort through cingulum modulation,⁴⁴⁵ no studies have assessed its use in cognitive rehabilitation after stroke. Modafinil has been studied for the treatment of post-stroke depression⁴⁴⁶ and fatigue⁴⁴⁷ but not cognitive recovery. Atomoxetine also has been studied for the treatment of post-stroke depression but not cognitive deficits.

Donepezil has been studied in a small, randomized, clinical trial.⁴⁴⁸ Ten right-hemispheric stroke survivors were randomized to receive either 5 mg donepezil or placebo. The donepezil group demonstrated significant improvements on the Mini-Mental Status Examination 1 month after completion of treatment, and functional magnetic resonance imaging showed increased activation in both prefrontal areas, both inferior frontal lobes, and the left inferior parietal lobe.

A pilot study randomized 50 subjects to receive either rivastigmine or placebo.⁴⁴⁹ Subjects receiving rivastigmine demonstrated statistically significant improvement (1.70 versus 0.13; $P=0.02$) on the animal subtask of the verbal fluency measure compared with those on placebo, but a non-significant trend toward improvement was observed in the Color Trails II test, described as a culture-fair test of visual attention, graphomotor sequencing, and effortful executive processing abilities.

A study of 47 subjects at least 6 months after stroke were randomized to receive fluoxetine, nortriptyline, or placebo.⁴⁵⁰ Although no significant group effect was found at the end of treatment, the placebo group exhibited deterioration in executive functioning 21 months after treatment, whereas the groups who received fluoxetine or nortriptyline significantly improved, independently of depressive symptoms ($F=12.1$ $df=1, 45$; $P=0.001$). The improvement was attributed to possible reorganization of neuronal networks associated with prefrontal functions based on modulation of monoaminergic neurotransmission and the activity of neurotrophins.

Recommendations: Nondrug Therapies for Cognitive Impairment, Including Memory	Class	Level of Evidence
Enriched environments to increase engagement with cognitive activities are recommended.	I	A
Use of cognitive rehabilitation to improve attention, memory, visual neglect, and executive functioning is reasonable.	IIa	B
Use of cognitive training strategies that consider practice, compensation, and adaptive techniques for increasing independence is reasonable.	IIa	B
Compensatory strategies may be considered to improve memory functions, including the use of internalized strategies (eg, visual imagery, semantic organization, spaced practice) and external memory assistive technology (eg, notebooks, paging systems, computers, other prompting devices).	IIb	A
Some type of specific memory training is reasonable such as promoting global processing in visual-spatial memory and constructing a semantic framework for language-based memory.	IIb	B
Errorless learning techniques may be effective for individuals with severe memory impairments for learning specific skills or knowledge, although there is limited transfer to novel tasks or reduction in overall functional memory problems.	IIb	B
Music therapy may be reasonable for improving verbal memory.	IIb	B

Recommendations: Use of Drugs to Improve Cognitive Impairments, Including Attention	Class	Level of Evidence
The usefulness of donepezil in the treatment of poststroke cognitive deficits is not well established.	IIb	B
The usefulness of rivastigmine in the treatment of poststroke cognitive deficits is not well established.	IIb	B
The usefulness of antidepressants in the treatment of poststroke cognitive deficits is not well established.	IIb	B
The usefulness of dextroamphetamine, methylphenidate, modafinil, and atomoxetine in the treatment of poststroke cognitive deficits is unclear.	IIb	C

Limb Apraxia

Limb apraxia is “a decrease or difficulty in performing purposeful, skilled movements” that cannot be attributed to hemiplegia or lack of effort.⁴⁵¹ It is more common after left hemispheric than right hemispheric stroke.⁴⁵² Although not traditionally believed to affect daily life function,^{453,454} there is now evidence that apraxia is associated with reduced independence in daily life activities.^{455–457} Despite its incidence and its impact on independent functioning, there is a paucity of research on therapeutic interventions for limb apraxia. Several systematic reviews have been conducted since 2005,^{458–461} reviewing 5 small RCTs across the 4 reviews. Since these reviews, no additional RCTs and only 1 case study have been published.⁴⁶² Two reviews concluded that there was not enough information to determine whether interventions for apraxia were efficacious.^{458,459} Some studies have found immediate postintervention improvements on apraxia tests or in daily life activities, but few have found lasting advantages for the trained groups.⁴⁵⁹

Recommendations: Limb Apraxia	Class	Level of Evidence
Strategy training or gesture training for apraxia may be considered.	IIb	B
Task practice for apraxia with and without mental rehearsal may be considered.	IIb	C

Hemispatial Neglect or Hemi-Inattention

Hemispatial neglect, also called hemiagnosia, hemineglect, unilateral neglect, spatial neglect, contralateral neglect, unilateral visual inattention, hemi-inattention, neglect syndrome, or contralateral hemispatialagnosia, is a neuropsychological condition in which, after damage to a part of 1 hemisphere of the brain is sustained, a deficit in attention to and awareness of 1 side of space is observed. These symptoms are not attributable to a primary sensory (eg, visual) or motor deficit; they are typically contralateral to the lesion. Hemispatial neglect is common after stroke⁴⁶³ and significantly impairs the ability to participate effectively in rehabilitation.⁴⁶⁴ Although neglect improves over time, neglect symptoms continue to interfere with daily functioning long after stroke.^{465–467} The interventions developed for neglect fall into 2 general categories: bottom-up approaches, designed

to remediate attention processes for the left hemispace and internal representations of space, and top-down approaches, aimed at teaching the person strategies for compensating for neglect.⁴⁶⁸ Most studies of neglect have been plagued by low-quality methods and small sample sizes.

Three systematic reviews have been completed since 2005,^{468–470} reviewing 24 unique randomized, clinical trials and 14 additional studies with weaker designs. The interventions studied and outcome measures varied widely in these reviews. Fifteen additional RCTs investigating neglect were found that were not included in those reviews (prism adaptation, 2; virtual reality, 2; limb activation, 2; neck vibration with prism adaptation, 1; visual scanning with limb activation, 1; mental practice, 1; repetitive transcranial magnetic stimulation, 4; and optokinetic stimulation, 2).^{471–483} There is evidence for the efficacy of several top-down and bottom-up approaches in improving both immediate performance and long-term performance on standard neglect tests such as cancellation tests and line bisection tests.* These include half-field eye patching, visual scanning training, prism adaptation, limb activation, optokinetic stimulation, mental imagery (but see the work by Welfringer and colleagues⁴⁸²), and brain stimulation with repetitive transcranial magnetic stimulation, theta burst transcranial magnetic stimulation, or tDCS. Two randomized, clinical trials of eye patching for unilateral neglect in 35 subjects⁴⁸⁷ and 60 subjects⁴⁸⁸ did not demonstrate any significant functional improvement. None of these treatments resulted in improvement on all neglect tests.

Few studies have examined the efficacy of these interventions on daily life functioning. Several have used the behavioral tests from the Behavioral Inattention Test⁴⁸⁹ or the Baking Tray Test,⁴⁹⁰ which are simulated real-life activities. Some studies have examined functional outcomes with the Catherine Bergego Scale,⁴⁹¹ which measures neglect symptoms during everyday activities or paragraph reading tasks. Others have used the less sensitive, general tests of functioning in ADLs such as the Barthel Index³³⁰ and the FIM.⁴⁹² There is limited evidence to date that these interventions increase daily life functioning, even when performance on neglect tests has improved,^{468,470} although some individual RCTs have found positive results on daily function.^{469,471,475,481,484}

Cognitive rehabilitation may have immediate benefits on tests of neglect, as supported by a meta-analysis of 23 RCTs, but it is uncertain whether disability associated with neglect was altered.⁴¹⁹ Finally, a meta-analysis⁴⁹³ found that compensatory scanning training improved reading and visual scanning in people with visual field defects (and possibly coexisting visual neglect).

It is important to note that in many of the studies, the target intervention was provided in addition to regular therapy or scanning training. Therefore, there is not sufficient evidence to ascertain whether neglect interventions are effective when provided in isolation. In addition, several issues in understanding how to treat neglect exist. These include understanding the heterogeneous response to treatment across clients, the heterogeneous response to treatment across measured tasks, the parameters of treatment (dosing, type of practice activity during or after treatment), and the relative efficacy of the various interventions, either alone or in combination.

*References 469–471, 473, 475, 476, 478, 480, 481, 484–486

Recommendations: Hemispatial Neglect or Hemi-Inattention	Class	Level of Evidence
It is reasonable to provide repeated top-down and bottom-up interventions such as prism adaptation, visual scanning training, optokinetic stimulation, virtual reality, limb activation, mental imagery, and neck vibration combined with prism adaptation to improve neglect symptoms.	Ila	A
Right visual field testing may be considered.	IIb	B
Repetitive transcranial magnetic stimulation of various forms may be considered to ameliorate neglect symptoms.	IIb	B

Communication Disorders

Disorders of communication and related cognitive impairments are common after stroke and include aphasia, cognitive-communication disorders, dysarthria, and apraxia of speech. Communication disorders may affect speaking, listening, reading, writing, gestures, and pragmatics. The presence of a communication disorder may negatively affect social participation, psychosocial well-being, and quality of life.

A certified speech and language pathologist normally performs the evaluation and treatment of communication disorders. The overall goals of speech and language treatment are to facilitate the recovery of communication, to assist patients in developing strategies to compensate for communication disorders, and to counsel and educate people in the patient's environment on assistive communication supports to facilitate communication, to decrease isolation, and to meet the patient's wants and needs. Compensatory and assistive communication supports may range from low-tech strategies such as paper/pencil and communication boards/books to high-tech devices that include smart phones and speech-generating devices.

Cognitive-Communication Disorders

There is great diversity in the presentation of cognitive-communication problems after stroke.⁴⁹⁴ A systematic review of cognitive-communication disorders after right hemispheric stroke suggested that many individuals at both the chronic and acute phases of recovery benefit from sentence- or discourse-level communication treatments.⁴⁹⁵

Several reviews summarize research evidence for treatments of attention, visual neglect, memory training, and other cognitive treatments for individuals with acquired brain injuries, including right hemispheric stroke. Although RCTs are lacking,^{419,420,425} a systematic review concludes that there is now sufficient information to support evidence-based protocols to implement empirically supported treatments for cognitive and communication disability after stroke.⁴¹⁸ The Nondrug Therapies for Cognitive Impairment, Including Memory section above provides more information on nonpharmacological treatments for cognitive disorders after stroke.

Aphasia

An RCT indicated that daily aphasia therapy in very early stroke recovery (starting at 3 days) improved communication

outcomes in people with moderate to severe aphasia.⁴⁹⁶ One systematic review of treatment in patients at >6 months after stroke concluded that aphasia therapy continued to be efficacious in the chronic stages,⁴⁹⁷ whereas another concluded that there was no significant relationship between time after onset and response to treatment.⁴⁹⁸ Insufficient evidence exists to know when treatment should start or how long it should continue.

Several systematic reviews have indicated that intensive treatment is favored,^{499–501} but there is no consensus on the optimum amount, intensity, distribution, or duration of treatment.³⁵³ For subacute aphasia, 1 RCT has shown that a short duration (3 weeks) of intensive therapy is efficacious,⁵⁰² whereas another RCT indicated that intensive treatment over a longer duration (12 weeks) may not always be feasible.⁵⁰³ Therefore, intensive therapy should be provided as tolerated and feasible.

A variety of different treatment approaches for aphasia have been developed. Small-group and single-subject studies support their efficacy.⁴⁹⁷ A systematic review of RCTs of aphasia treatment stated that no conclusions can be made about the effectiveness of one treatment over another.⁴⁹⁹

Three RCTs evaluated computer-based therapy, with 1 RCT comparing it with no treatment, 1 comparing it with the same treatment provided by a speech and language therapist, and the third comparing it with the same amount of nonlinguistic computer training.^{504–506} These 3 trials concluded that computer-based therapy is feasible and efficacious. Therefore, computerized treatment is beneficial and can be used to supplement treatment provided by a speech-language pathologist.

A systematic review concluded that communication partner training is effective in improving communication activities or the participation of the communication partner. It is also probably effective in improving communication activities or the participation of individuals with chronic aphasia when they are interacting with trained communication partners.⁵⁰⁷ Communication partners may include family members and caregivers, healthcare professionals, and others in the community or organization. Further studies are needed to examine the impact of communication partner training with individuals with acute aphasia.⁵⁰⁷

Two systematic reviews have addressed group therapy.^{499,508} Group treatments for people with aphasia occur across the continuum of care.⁵⁰⁸ Overall, results indicate that group participation can improve specific linguistic processes with no significant difference in outcomes between individual one-on-one therapy and group therapy. There is also some evidence that outpatient and community-based group participation can benefit social networks and community access.⁵⁰⁸

Several small RCTs have shown that drug therapy appears to be beneficial in conjunction with SLT, whereas other studies have failed to show a benefit. Drugs showing promise include donepezil,⁵⁰⁹ memantine,⁵¹⁰ and galantamine.⁵¹¹ Bromocriptine⁵¹² and piracetam⁵¹³ do not appear beneficial. More extensive studies of pharmacotherapy for aphasia are needed before the routine use of any medication can be

recommended. Further research on the dose and timing of administration is needed.

Brain stimulation techniques, including epidural cortical stimulation, repetitive transcranial magnetic stimulation, and tDCS, have been used to modulate cortical excitability during poststroke language recovery. Small studies have shown therapeutic benefits when brain stimulation techniques are used, typically in combination with behavioral language therapy.^{504,514–516} Most studies are small-group or single-subject studies and have been conducted in patients with chronic aphasia. Two RCTs investigating repetitive transcranial magnetic stimulation in acute and subacute aphasia^{517,518} found mixed results. Brain stimulation combined with speech language therapy may benefit selected patients, but more information on the site of stimulation and stimulation parameters is needed before it can be used in routine clinical practice.^{437,438,516}

Recommendation: Cognitive Communication Disorders	Class	Level of Evidence
Interventions for cognitive-communication disorders are reasonable to consider if they are individually tailored and target:	Ila	B
The overt communication deficit affecting prosody, comprehension, expression of discourse, and pragmatics		
The cognitive deficits that accompany or underlie the communication deficit, including attention, memory, and executive functions		

Recommendations: Aphasia	Class	Level of Evidence
Speech and language therapy is recommended for individuals with aphasia.	I	A
Treatment for aphasia should include communication partner training.	I	B
Intensive treatment is probably indicated, but there is no definitive agreement on the optimum amount, timing, intensity, distribution, or duration of treatment.	Ila	A
Computerized treatment may be considered to supplement treatment provided by a speech-language pathologist.	Ilb	A
A variety of different treatment approaches for aphasia may be useful, but their relative effectiveness is not known.	Ilb	B
Group treatment may be useful across the continuum of care, including the use of community-based aphasia groups.	Ilb	B
Pharmacotherapy for aphasia may be considered on a case-by-case basis in conjunction with speech and language therapy, but no specific regimen is recommended for routine use at this time.	Ilb	B
Brain stimulation techniques as adjuncts to behavioral speech and language therapy are considered experimental and therefore are not currently recommended for routine use.	III	B

Motor Speech Disorders: Dysarthria and Apraxia of Speech

Dysarthria is a collective term for a group of speech disorders that result from paralysis, weakness, or incoordination of the speech musculature after neurological damage. Dysarthria can affect, singly or in combination, any of the subsystems underlying speech production: the respiratory, laryngeal, velopharyngeal, and oral-articulatory subsystems. It is estimated that 20% of stroke patients present with dysarthria,⁵¹⁹ although the type of dysarthria and its specific characteristics vary, depending on factors such as lesion site and severity.

Apraxia of speech is a disorder of motor planning or programming resulting in difficulty in volitionally producing the correct sounds of speech. In addition to articulatory disturbances, prosodic deficits such as slow rate of speech and restricted variations in pitch and loudness may be present. Apraxia of speech typically co-occurs with nonfluent aphasia, and the existence of a pure apraxia of speech without aphasia is debatable.

Motor speech disorders affect the intelligibility, naturalness, and efficiency of communication. The presence of a motor speech disorder may negatively affect social participation, psychosocial well-being, and quality of life.

Speech and language therapists use a range of behavioral treatments to address motor speech disorders in individuals after stroke.^{520–523} Behavioral treatments for motor speech disorders are diverse in their focus and theoretical underpinnings and should be tailored to the individual's unique strengths, deficits, goals, priorities, and circumstances. Behavioral treatments may focus on improving the physiological support for speech and target impairments in respiration, phonation, articulation, and resonance. Behavioral treatments may also include strategies to increase the precision of articulation, to modify the rate and loudness of speech, and to improve prosody. To date, no randomized, clinical trials have addressed the efficacy of these approaches,^{524,525} but small, nonrandomized group studies and carefully designed, single-subject, experimental studies have demonstrated positive results.^{521,526–528} Individuals with motor speech disorders may improve as a result of treatment, even when the condition is chronic.^{521,522,528,529} There is no consensus on the optimum amount, distribution, or variability of practice or the best type, frequency, and timing of treatment.

Patients with motor speech disorders may benefit from using augmentative and alternative communication devices to supplement their communication. Augmentative and alternative communication devices range from simple picture boards or spelling boards to portable amplification systems and high-tech electronic devices with eye-tracking capability.^{522,530} Supplemental strategies such as gesture or writing can be used to enhance communication attempts. Two systematic reviews have concluded that augmentative and alternative communication and speech supplementation techniques may be useful for individuals with motor speech disorders, when speech is insufficient to meet the individual's communication needs.^{527,531}

The effects of motor speech disorders after stroke extend beyond the physiological characteristics of the impairment. Studies have shown that the resulting communication difficulties affect social participation and quality of life^{532,533} and that the psychosocial impact of a motor speech disorder is disproportionate to the severity of the physiological impairment.^{532,533}

Behavioral management of motor speech disorders includes support and counseling. Interventions addressing the broad life implications of motor speech disorders are being developed, and pilot studies are underway.⁵³⁴

Addressing environmental factors during rehabilitation is consistent with the *ICF* and warrants consideration.^{535–537} For individuals with motor speech disorders, this may include providing education that addresses the knowledge and attitudes of communication partners or modifying the characteristics of the physical environment such as reducing noise levels.^{535–537}

Telerehabilitation may be used to overcome barriers of access to services.⁵³⁸ The quality of telerehabilitation services must be consistent with the quality of services delivered face to face.⁵³⁸ Studies demonstrating the feasibility of telerehabilitation in the management of dysarthria are emerging.³⁵³

Recommendations: Motor Speech Disorders: Dysarthria and Apraxia of Speech	Class	Level of Evidence
Interventions for motor speech disorders should be individually tailored and can include behavioral techniques and strategies that target:	I	B
Physiological support for speech, including respiration, phonation, articulation, and resonance		
Global aspects of speech production such as loudness, rate, and prosody		
Augmentative and alternative communication devices and modalities should be used to supplement speech.	I	C
Telerehabilitation may be useful when face-to-face treatment is impossible or impractical.	Ila	C
Environmental modifications, including listener education, may be considered to improve communication effectiveness.	Ilb	C
Activities to facilitate social participation and promote psychosocial well-being may be considered.	Ilb	C

Spasticity

Spasticity, classically defined as a velocity-dependent resistance to stretch of a muscle, is a component of the upper motor neuron syndrome. Poststroke spasticity may have dystonic features, including involuntary muscle activity and limb positioning. Spasticity is correlated with activity limitations associated with hygiene, dressing, and pain. These activity limitations increase caregiver burden and reduce quality of life as measured by the EuroQol-5.⁵³⁹

When spasticity is present, the cost of care is 4 times higher than when spasticity is absent; however, because spasticity is strongly associated with stroke severity, the independent impact of spasticity on costs is not known.⁵⁴⁰ Thus, the cost of treating spasticity may not reduce the overall cost of stroke-related care. For example, in 1 study, the use of botulinum toxin injections for upper limb spasticity combined with therapy was not found to be cost-effective compared with therapy alone.⁵⁴¹

The prevalence of poststroke spasticity in any limb is in the range of 25% to 43% over the first year after stroke.^{542–545}

For patients who require acute rehabilitation after stroke, the prevalence of spasticity in any limb is 42%.⁵⁴⁶ The incidence of upper limb spasticity over the first 3 months in patients admitted to rehabilitation is 33%.⁹ The strongest predictor of moderate to severe spasticity (Ashworth scale score ≥ 2) is severe proximal and distal limb weakness on acute hospital or rehabilitation admission.^{543,547}

The use of resting hand splints is not effective for reducing wrist and finger spasticity, and the use of such splints is controversial for the prevention of contracture in the setting of spasticity.⁷⁵ For ankle plantarflexor spasticity, a short course of ankle casting may facilitate spasticity reduction after injection of botulinum toxin. Taping, however, has no effect on spasticity after lower limb botulinum toxin injection and is not recommended.^{548,549}

NMES combined with therapy may improve spasticity, but there is insufficient evidence that the addition of NMES improves functional gait or hand use.⁵⁵⁰ Vibration applied to spastic muscle groups might be considered to reduce spasticity transiently, but it is not effective for long-term reduction of spastic hypertonia.^{551–553}

Injection of botulinum toxin is used commonly to treat upper limb spasticity in patients with stroke and is recommended in several recent review articles and previously published guidelines as an important tool in the comprehensive management of poststroke spastic hypertonia.^{149,554–557} Injections of botulinum toxin A can reduce spasticity significantly as measured by the Ashworth scale. In a meta-analysis, botulinum toxin was shown to have a small but statistically significant effect on activity as measured by the Disability Assessment Scale after injection into the upper limb.⁵⁵⁸ However, improvements were attributable to the lowered resistance to muscle stretch during passive repositioning of the upper limb rather than to the actual skilled functional use of the arm and hand. Thus, there is no evidence to suggest that botulinum toxin injections will improve functional upper limb use, but it may improve limb active or passive limb positioning for activities such as dressing and hygiene.^{559,560} Although botulinum toxins are clinically recommended for spasticity reduction, it is not clear that they are a cost-effective means to manage spastic hypertonia compared with physical or occupational therapies alone.⁵⁴¹ However, if a reduction in caregiver burden is taken into account, the use of botulinum toxins with therapy may be cost-effective.⁵⁶¹ The early injection of botulinum toxins as soon as hypertonia appears may be effective in preventing later spasticity, but this needs further study.^{562,563}

Botulinum toxins injected into the ankle plantarflexor and inverter muscles significantly reduce lower limb spasticity as measured by the Ashworth scale.^{564–566} Injections may also improve gait speed, although only slightly.⁵⁶⁷ Botulinum toxin injections into the rectus femoris muscle may improve tonic knee extension during the swing phase of gait in stroke, but further study is needed.⁵⁶⁸ Although botulinum toxins have been used to improve orthotic fit, no studies of this application have been reported.

Oral antispasticity agents, including baclofen, dantrolene sodium, and tizanidine, have a marginal effect on reducing generalized spasticity, but dose-limiting side effects such as tiredness and lethargy are common.^{569–577} Intrathecal baclofen therapy is effective in reducing generalized spastic

hypertonia in patients with stroke.^{570,578–582} A consensus panel in 2006 recommended that intrathecal baclofen therapy is appropriate in those patients with spasticity who do not respond well to other interventions or in patients who experience adverse effects from other treatments. They also concluded that intrathecal baclofen therapy can be considered as early as 3 to 6 months after stroke for patients refractory to other treatments.⁵⁸³

Recommendations: Spasticity	Class	Level of Evidence
Targeted injection of botulinum toxin into localized upper limb muscles is recommended to reduce spasticity, to improve passive or active range of motion, and to improve dressing, hygiene, and limb positioning.	I	A
Targeted injection of botulinum toxin into lower limb muscles is recommended to reduce spasticity that interferes with gait function.	I	A
Oral antispasticity agents can be useful for generalized spastic dystonia but may result in dose-limiting sedation or other side effects.	Ila	A
Physical modalities such as NMES or vibration applied to spastic muscles may be reasonable to improve spasticity temporarily as an adjunct to rehabilitation therapy.	Ilb	A
Intrathecal baclofen therapy may be useful for severe spastic hypertonia that does not respond to other interventions.	Ilb	A
Postural training and task-oriented therapy may be considered for rehabilitation of ataxia.	Ilb	C
The use of splints and taping are not recommended for prevention of wrist and finger spasticity after stroke.	III	B

Balance and Ataxia

Balance depends on sensory inputs from the visual, vestibular, and somatosensory systems. These sensory inputs are integrated and used to control anticipatory and reactive motor output to postural disturbances. Balance impairment (inclusive of postural control impairment) is common after stroke^{182,584,585} because stroke can affect 1 or more of the sensory and motor networks. Impaired balance makes it difficult to safely complete ADLs, to move about the home and community, and to live independently. A large percentage of people report falling at least once in the first 6 months after stroke.^{182,585} People with stroke who fall are twice as likely to sustain a hip fracture compared with those who fall but do not have a stroke.⁵⁸⁶ Balance impairments can result in low balance confidence, which in turn may further reduce activity.⁵⁸⁷ If left undetected or untreated, balance impairments can result in a cascade of serious, undesirable, and expensive events.^{175,245}

Evaluation of balance abilities is considered part of routine clinical practice in individuals with stroke.^{308,588,589} Standardized tests of balance challenge different aspects of postural control such as anticipatory postural reactions during a variety of functional behaviors. Specific balance limitations

identified during the evaluation will help determine the risk of falling and guide the selection and tailoring of balance-specific interventions.^{308,591}

Although balance training programs have been shown to be beneficial after stroke, no specific approach or program has been demonstrated to be superior, nor is the optimal timing clear. Balance training has been successfully implemented as group and one-on-one sessions, circuit training, and hospital-versus home- versus community-based programs. Content of the training typically includes balance-specific activities, (eg, practice responding to challenges in standing) and more general activities (eg, strengthening exercises, gait activities).⁵⁹² Shorter, more time-intensive programs appear comparable to longer, less time-intensive programs.⁵⁹² Progression to more challenging training activities over the course of training is important. The one type of training that has not been shown to be beneficial for balance is water-based programs.⁵⁹³

Studies of balance training have generally been small, typically 10 to 60 subjects. Subjects typically have been able to ambulate independently (with or without an assistive device) and be relatively cognitively intact. Four systematic reviews and meta-analyses have reviewed the effects of various interventions on balance after stroke, with the latest one published in 2013. Findings across these reviews show inconsistent effects on balance outcomes. Subsequent published RCTs have tested a variety of types of balance training devices (sliding board, trunk exercises on a physioball, shoe wedge) or programs (yoga, Tai Chi,¹⁸⁷ gait training, motor imagery). The later studies have similar methodological challenges (8–40 subjects per group) and lead to similar, inconsistent conclusions about the superiority of any 1 specific treatment.^{594–604} Likewise, a systematic review of fall prevention after stroke has shown that inconsistencies in outcome measures, intervention type, and implementation in previous research make it difficult to determine the effectiveness of fall prevention programs after stroke.¹⁷⁴ The Prevention of Falls section provides more discussion.

Use of devices and orthotics (eg, cane, AFO) also improves balance.⁶⁰⁵ Finally, it should be noted that improving balance alone may not be sufficient for preventing falls because falls may have multiple contributing causes.

Ataxia is a disorder of coordinated muscle activity during voluntary movement associated with injury to the cerebellum, cerebellar peduncles, and brainstem cerebellar tracts. Patients with ataxia have delayed movement initiation, timing errors, abnormal limb trajectories, and dysmetria.^{606,607} Ataxia is present in 68% to 86% of patients with brainstem stroke. Ataxia typically improves during acute rehabilitation.^{608,609} Ataxia without concurrent hemiparesis has a better prognosis for functional recovery in acute rehabilitation.⁶¹⁰ However, the presence of ataxia with or without weakness does not affect general functional recovery negatively.^{608,609} Ataxia can affect the quality of use of the functional hand negatively because patients with cerebellar lesions can have impaired motor learning (eg, reduced skill improvement on a pursuit rotor task or ability to learn a finger sequence).^{611,612} Despite this, case studies indicate that intensive task-oriented therapy may improve motor performance and actual use of ataxic limbs in patients with stroke-related ataxia.

After participating in a task-oriented training program, patients improved reaching speed and had reduced trunk motion during reaching.⁶¹³ Stoykov and others⁶⁰⁶ noted that postural training and provision of trunk support could have a positive impact on upper limb motor control and dexterity in a patient with upper limb ataxia. There is a paucity of research on rehabilitation approaches to limb ataxia, but at present, postural training and task-oriented upper limb training are recommended.

Recommendations: Balance and Ataxia	Class	Level of Evidence
Individuals with stroke who have poor balance, low balance confidence, and fear of falls or are at risk for falls should be provided with a balance training program.	I	A
Individuals with stroke should be prescribed and fit with an assistive device or orthosis if appropriate to improve balance.	I	A
Individuals with stroke should be evaluated for balance, balance confidence, and fall risk.	I	C
Postural training and task-oriented therapy may be considered for rehabilitation of ataxia.	IIb	C

Mobility

The loss or difficulty with ambulation is one of the most devastating sequelae of stroke, and restoration of gait is often one of the primary goals of rehabilitation. Gait-related activities include such tasks as mobility during rising to stand, sitting down, stair climbing, turning, transferring (eg, wheelchair to bed or bed to chair), using a wheelchair after stroke, walking quickly, and walking for specified distances.⁶¹⁴ Limitations in gait and gait-related activities are associated with an increase in fall risk. A number of systematic reviews have demonstrated enhanced outcomes of gait, gait-related activities, and ADLs⁶¹⁵ after intensive, repetitive task training.^{616–618} The role of treadmill training and electromechanics-assisted gait training remains under study.⁶¹⁹

Key training parameters for improving mobility after stroke are activity-specific and functional task practice; practice that is progressively more difficult and challenging; practice that is of sufficient intensity, frequency, and duration; and practice that is at an appropriate time relative to stroke onset.^{616,620} These parameters pertain to treadmill training with or without body weight support, circuit training, mobility training, and electromechanics-assisted training.⁶¹⁶

Dickstein⁶²¹ reviewed a variety of mobility training techniques and found that gains were comparable across treatments but generally insufficient for patients to advance to a higher functional walking category on the basis of the categories defined by Perry et al.²⁷⁷ No benefit was seen for more complex methods such as treadmill and robotic-based interventions compared with more traditional approaches.

Circuit class therapy is a form of group treatment with exercises focused on repetitive practice of functional tasks.^{622–624} A 2009 meta-analysis and recent systematic review concluded that circuit class therapy was a safe and effective method for improving mobility after stroke.^{623,625}

Treadmill training in the context of task-specific training may be used with or without body weight support or therapists to assist the paretic lower extremity in stepping. A recent systematic review concluded that compared with no intervention or with an intervention with no walking component, treadmill training without body weight support improved walking speed and distance among ambulatory people after stroke. Although these benefits were maintained beyond the intervention period, it is not yet known whether treadmill training is superior to overground walking training.^{621,626} Recently, it was demonstrated that treadmill training with body weight support and traditional gait training were equally effective in improving walking and transfers in patients dependent on walking assistance after stroke.^{51,627} A recent systematic review, including those <3 months after stroke and unable to walk, reported that those individuals who are earlier after stroke and more severe are more likely to have a better gait recovery outcome with mechanically assisted training compared with overground training and by using a harness in conjunction with the mechanical device. Mechanically assisted walking (eg, treadmill, electromechanical gait trainer, robotic device, servo-motor) with body weight support was found to be more effective than overground walking at increasing independent walking in nonambulatory patients early after stroke.⁶²⁸

Lower Extremity Strengthening

A 2007 review concluded that graded strength training improves the ability to generate force but does not transfer to improvements in walking.⁶¹⁸ However, a more recent meta-analysis demonstrated that providing lower limb resistance training to community-dwelling individuals who are 6 months after stroke has the capacity to improve comfortable gait speed and total distance walked.⁶²⁹ Similarly, a 2008 review concluded that despite limited long-term follow-up data, there is evidence that resistance training produces increased strength, gait speed, and functional outcomes, as well as improved quality of life.⁶³⁰

NMES has been used to stimulate the ankle dorsiflexors during the swing phase of the gait cycle. A recent systematic review revealed a small but significant treatment effect of NMES on gait capacity in individuals in the chronic phase after stroke.⁶³¹ Similarly, a meta-analysis revealed the effectiveness of NMES at improving gait speed in subjects after stroke.⁶³² Several RCTs have observed improved recovery of gait function after stroke in the chronic^{550,633–635} and acute phases^{636,637} when NMES was applied in conjunction with a conventional rehabilitation program. Studies comparing the use of an AFO to NMES in controlling foot drop during walking have found similar results.^{638,639} Although subjects preferred the foot drop stimulator used in 2 multisite RCTs, both the stimulator and a conventional AFO produced equivalent functional gains.^{638,640,641} Similar results were obtained in a comparison of surface peroneal nerve stimulation and use of an AFO.^{642,643} Significant improvements in functional mobility were found with both peroneal nerve stimulation and AFO during the treatment period and were maintained at the 6-month follow-up.

Medications for Motor Recovery

Several medications have been studied as potential contributors to stroke recovery in general and to motor recovery in

particular, including dextroamphetamine, methylphenidate, levodopa, and SSRIs. Fluoxetine was found to be helpful for motor recovery in a double-blind, placebo-controlled trial,⁶⁴⁴ and several smaller studies of SSRIs were also suggestive of benefit.^{645–648} A systematic review and meta-analysis found evidence of benefit for SSRIs in overall disability after stroke.⁶⁴⁹ The overall quality of these studies was not sufficient, however, to make a definitive recommendation, and larger, well-controlled trials are in progress. A randomized, double-blind, placebo-controlled trial of dextroamphetamine in 71 subjects was negative,⁴⁴⁴ and a subsequent systematic review of the use of amphetamines for improving motor recovery after stroke found inconsistent findings,⁶⁵⁰ and these carry a risk of adverse cardiovascular effects. A randomized, double-blind, placebo-controlled trial of levodopa found short-term benefit of this therapy compared with placebo for motor function but was limited by relatively small size (47 subjects analyzed), baseline differences in stroke severity and patient age between the 2 treatment groups, and the short-term follow-up of only 3 weeks after the completion of therapy.⁶⁵¹

Acupuncture

The Ottawa Panel recommends that there is good scientific evidence to consider including acupuncture as an adjunct to standard stroke rehabilitation to improve walking mobility.⁶³⁹ Shifflett⁶⁵² reviewed a number of RCTs of acupuncture for stroke recovery and performed a reanalysis suggesting that acupuncture may be effective as an adjunctive treatment for improving walking speed.

Transcutaneous Electrical Nerve Stimulation

TENS provides electrically induced sensory input to the lower limb. A meta-analysis revealed that there was insufficient research to make conclusions about the effectiveness of TENS in improving gait and gait-related activities.⁶³² Three subsequent RCTs provided evidence of a potential benefit of TENS on physical function after stroke, particularly when combined with task-related activity.^{653–655}

Rhythmic Auditory Cueing

Rhythmic auditory cueing is a therapy approach in which overground walking is synchronized to a rhythmic auditory cue to improve temporal and spatial gait measures. An evidence synthesis found moderate evidence of improved velocity and stride length in people with stroke after gait training with rhythmic music. Synchronizing walking to rhythmic auditory cues can result in short-term improvement in gait measures of people with stroke. Further high-quality studies are needed before recommendations for clinical practice can be made.⁶⁵⁶

Use of AFOs

Use of AFOs is an effective method of compensating for motor impairments in the lower limb after stroke.^{657–660} The reader is referred to the section below on adaptive equipment for details.

Robotic and Electromechanics-Assisted Training Devices

Robots and electromechanics-assisted training devices have been used in an effort to promote gait recovery after stroke.

Most of these devices incorporate body weight support along with treadmills or foot platform pedals analogous to an elliptical trainer. Their main advantage over conventional gait training is that they reduce the need for intensive therapist support. These devices include the Lokomat, the Gait Trainer GT 1, and the AutoAmbulator. A Cochrane systematic review updated in 2013 concluded that patients with stroke who received electromechanics-assisted gait training in combination with PT were more likely to achieve independent walking than patients receiving gait training without these devices, but it did not find an increase in gait velocity.⁶⁶¹ The review concluded that the individuals most likely to benefit from this therapy appear to be those who are within the first 3 months after stroke and those who are unable to walk. In contrast, a study by Hornby et al⁶⁶² demonstrated greater improvement in gait velocity and single limb support time on the paretic limb after therapist-assisted locomotor training compared with robotic-assisted locomotor training.⁶⁶² A systematic review found improved balance for stroke survivors receiving robotic gait training, but there was insufficient evidence comparing robotic gait training with conventional gait training to determine whether these therapies are similar in this regard.⁶⁶³

Exoskeletal wearable lower limb robotic devices are also available for gait training after stroke and allow overground walking with the device. Most of these devices (eg, Ekso, Ekso Bionics, Richmond, CA; Indego, Parker-Hannifin; and ReWalk, Marlborough, MA) are bilateral in design, although unilateral exoskeletal wearable devices have also been developed (eg, Bionic Leg, AlterG, Fremont, CA). Although a pilot study of a unilateral device did not demonstrate benefit compared with conventional exercise therapy,⁶⁶⁴ most of the devices in this class have not yet been examined in controlled trials for stroke survivors. Overall, although robotic therapy remains a promising therapy as an adjunct to conventional gait training, further studies are needed to clarify the optimal device type, training protocols, and patient selection to maximize benefits.

Electromyographic Biofeedback

Electromyographic biofeedback is a technique that uses visual or audio signals to provide the patient with feedback on his/her muscle activity. The literature on the use of electromyographic biofeedback plus conventional rehabilitation includes some studies suggesting improved motor power, functional recovery, and gait quality compared with conventional rehabilitation alone. However, a 2007 Cochrane database systematic review did not find a treatment benefit. The results of the systematic review are limited because the trials were small, were generally poorly designed, and used varying outcome measures, making it difficult to compare across studies.⁶⁶⁵

Virtual Reality

Virtual reality is the use of computerized technology to allow patients to engage in specific task practice within a computer-generated visual environment in a naturalistic fashion. An environment that may be more interesting to a subject may enhance motivation to practice. In 2011, the Cochrane Stroke Group concluded that there was insufficient evidence to reach conclusions about the effect of virtual reality and interactive video gaming on gait speed.⁶⁶⁶ However, a recent systematic

review⁶⁶⁷ suggests that virtual reality promotes changes in gait parameters despite diversity of protocols, participant characteristics, and number of subjects included.

Traditional Physiotherapeutic Approaches (Neurodevelopmental Therapy/Bobath, Brunnstrum, Proprioceptive Neuromuscular Facilitation)

A recent systematic review conducted by Langhammer and Stanghelle⁶⁶⁸ assessed the efficacy of the traditional physiotherapeutic approaches. Although improvements in motor function were demonstrated, no trial showed that these approaches were superior to the respective comparison therapies.⁶⁶⁸ Similarly, it was concluded that neurodevelopmental approaches were equivalent or inferior to other approaches in improving walking ability in a 2007 systematic review.⁶¹⁸

Water-Based Exercises

The conclusions drawn in a 2012 Cochrane systematic review revealed that the evidence from RCTs to date does not confirm or refute that water-based exercises after stroke might help to improve gait and gait-related activities.⁵⁹³

Recommendations: Mobility	Class	Level of Evidence
Intensive, repetitive, mobility- task training is recommended for all individuals with gait limitations after stroke.	I	A
An AFO after stroke is recommended in individuals with remediable gait impairments (eg, foot drop) to compensate for foot drop and to improve mobility and paretic ankle and knee kinematics, kinetics, and energy cost of walking.	I	A
Group therapy with circuit training is a reasonable approach to improve walking.	Ila	A
Incorporating cardiovascular exercise and strengthening interventions is reasonable to consider for recovery of gait capacity and gait-related mobility tasks.	Ila	A
NMES is reasonable to consider as an alternative to an AFO for foot drop.	Ila	A
Practice walking with either a treadmill (with or without body-weight support) or overground walking exercise training combined with conventional rehabilitation may be reasonable for recovery of walking function.	Ilb	A
Robot-assisted movement training to improve motor function and mobility after stroke in combination with conventional therapy may be considered.	Ilb	A
Mechanically assisted walking (treadmill, electromechanical gait trainer, robotic device, servo-motor) with body weight support may be considered for patients who are nonambulatory or have low ambulatory ability early after stroke.	Ilb	A
There is insufficient evidence to recommend acupuncture for facilitating motor recovery and walking mobility.	Ilb	B

Recommendations: Mobility (Continued)	Class	Level of Evidence
The effectiveness of TENS in conjunction with everyday activities for improving mobility, lower extremity strength, and gait speed is uncertain.	Ilb	B
The effectiveness of rhythmic auditory cueing to improve walking speed and coordination is uncertain.	Ilb	B
The usefulness of electromyography biofeedback during gait training in patients after stroke is uncertain.	Ilb	B
Virtual reality may be beneficial for the improvement of gait.	Ilb	B
The effectiveness of neurophysiological approaches (ie, neurodevelopmental therapy, proprioceptive neuromuscular facilitation) compared with other treatment approaches for motor retraining after an acute stroke has not been established.	Ilb	B
The effectiveness of water-based exercise for motor recovery after an acute stroke is unclear.	Ilb	B
The effectiveness of fluoxetine or other SSRIs to enhance motor recovery is not well established.	Ilb	B
The effectiveness of levodopa to enhance motor recovery is not well established.	Ilb	B
The use of dextroamphetamine or methylphenidate to facilitate motor recovery is not recommended.	III	B

Upper Extremity Activity (Includes ADLs, IADLs, Touch, Proprioception)

The majority of individuals with stroke experience problems with the upper extremity, most commonly paresis,^{670,671} which is the key impairment in most cases.^{333,337,341,672,673} Only a small portion of people fully recover from upper limb paresis after a stroke, with the remainder left with lingering upper extremity impairments, activity limitations, and participation restrictions.^{338,674} An inability to use the upper extremity in daily life can lead to loss of independence with ADLs and of important occupations (eg, work, driving) and can even contribute to institutionalization.

Task-specific training, or functional task practice, is based on the premise that practice of an action results in improved performance of that action and is focused on learning or relearning a motor skill.^{675,676} Task-specific practice is an element of or used in combination with many upper extremity interventions such as constraint-induced movement therapy (CIMT) and NMES. Across a large number of studies, the key elements of task-specific training are repeated, challenging practice of functional, goal-oriented activities. Trunk restraint during task-specific training is beneficial in reducing compensatory trunk movements and promoting proximal movement control.^{677,678} Strengthening upper extremity muscles may be beneficial as an adjunct to task-specific training,^{679,680} when therapy time permits, or when the strengthening activities can be performed outside formal therapy sessions.

CIMT has been demonstrated to improve upper extremity activity, participation, and quality of life in individuals with baseline ability to control wrist and finger extension compared with usual care.^{52,678,681–685} It is less clear whether CIMT has

any advantage over dose-matched conventional upper limb therapy.^{686,687} CIMT can be delivered in its original form 3 to 6 h/d for 5 d/wk for 2 weeks or in a modified version 1 h/d for 3 d/wk for 10 weeks. The modified CIMT intervention appears to result in improvements that are comparable to the original version, although it has not been as extensively tested.^{688–694}

Bilateral upper limb training has not been as well studied as CIMT. Two meta-analyses and more recent trials suggest that there is a small but measurable benefit compared with no intervention, but no consistent evidence of superiority over other task-specific training interventions has been shown.^{695–699} Recent trials comparing bilateral training with CIMT or modified CIMT indicate that they may have similar efficacy for individuals with preserved isolated wrist and finger movement.^{700–702}

For individuals with more severe paresis, the potential for recovery of upper extremity function is greatly reduced, particularly later after stroke.⁶⁷⁴ Robotic therapy can deliver larger amounts of upper extremity movement practice for these individuals. There are a variety of types of upper extremity robots, consisting primarily of workstation devices used in a rehabilitation facility but also including some wearable exoskeletal devices that can be used in a home environment. A Cochrane review updated in 2012 found that upper limb robotic therapy provided benefit with regard to ADLs and arm function but not arm muscle strength.⁷⁰³ The variation within the trials with regard to duration and amount of training, the specific devices used, and patient populations studied limits the interpretation of these results. Moreover, many of the studies performed with robot-aided therapy have compared it with usual care rather than dose-matched conventional upper limb exercise therapy. Those studies incorporating dose-matched exercise as a comparison treatment show minimal or no differences in the efficacy between these 2 treatments.^{704,705} Overall, robotic therapy appears to provide some benefit for upper extremity motor abilities and participation but is of uncertain utility compared with dose-matched conventional upper limb exercise therapies.^{706–713}

NMES can be used for those with minimal ability for volitional muscle activation. It may be beneficial for improving upper extremity activity if used in combination with task-specific training, particularly when applied to the wrist and hand muscles.^{714–716} Alternatively, it is beneficial in preventing or correcting shoulder subluxation.^{125,132,717}

Mental practice, or mental imagery, may be useful as an adjunct to upper extremity exercise therapies.^{718–722} Initial training in mental practice occurs within a therapy session, but additional practice can happen outside formal therapy time. It is feasible to integrate mental practice with physical practice.⁷²³ Longer durations of mental practice appear to produce more benefit.⁷²⁴

Virtual reality and video gaming have the potential to increase participant engagement and the amount of upper extremity movement practice. Computer-based video games are widely available for recreational purposes for the general public, including those with handheld controllers (eg, Wii) and motion capture systems (Xbox Kinect, Microsoft, Inc). In addition, these systems can be used as remotely monitored telerehabilitation systems.⁷²⁵ To date, most studies of efficacy have been small and have used a variety of technologies and training programs, making generalization difficult. A Cochrane review⁶⁶⁶ found benefit in terms of upper limb function and

ADLs but no improvements in upper limb strength. The studies were of low quality in many cases, reducing confidence in this finding. Efficacy of Virtual Reality Exercises in STroke rehabilitation (EVREST),⁷²⁷ a multicenter, randomized, clinical trial, is under way that may provide more definitive evidence. At present, virtual reality and video gaming are reasonable alternative methods to engage individuals with stroke in the rehabilitation process and to increase the amount of movement practice.^{666,728,729,731–733}

A variety of interventions have been the focus of ≥1 studies but have not yet been shown to be consistently beneficial for upper limb motor rehabilitation. These include somatosensory stimulation^{734–738} and noninvasive brain stimulation (transcranial magnetic stimulation or tDCS) in combination with upper extremity exercise therapy,^{739–746} interventions targeting motor apraxia,⁴⁵⁸ and manual therapy approaches such as stretching, passive exercise, and mobilization,⁷⁴⁸ although these approaches are a routine part of practice for individuals with more severely affected upper extremities to prevent contractures and to manage spasticity.

Finally, upper extremity rehabilitation programs can be delivered in a variety of settings such as inpatient hospitals and outpatient clinics and within the home. A recent systematic review and subsequent RCT indicate that both outpatient and home service delivery models produce similar results on upper extremity activity, including the ability to perform ADLs.^{749,750}

Recommendations: Upper Extremity Activity, Including ADLs, IADLs, Touch, and Proprioception	Class	Level of Evidence
Functional tasks should be practiced; that is, task-specific training, in which the tasks are graded to challenge individual capabilities, practiced repeatedly, and progressed in difficulty on a frequent basis.	I	A
All individuals with stroke should receive ADL training tailored to individual needs and eventual discharge setting.	I	A
All individuals with stroke should receive IADL training tailored to individual needs and eventual discharge setting.	I	B
CIMT or its modified version is reasonable to consider for eligible stroke survivors.	Ila	A
Robotic therapy is reasonable to consider to deliver more intensive practice for individuals with moderate to severe upper limb paresis.	Ila	A
NMES is reasonable to consider for individuals with minimal volitional movement within the first few months after stroke or for individuals with shoulder subluxation.	Ila	A
Mental practice is reasonable to consider as an adjunct to upper extremity rehabilitation services.	Ila	A
Strengthening exercises are reasonable to consider as an adjunct to functional task practice.	Ila	B
Virtual reality is reasonable to consider as a method for delivering upper extremity movement practice.	Ila	B

Recommendations: Upper Extremity Activity, Including ADLs, IADLs, Touch, and Proprioception (Continued)	Class	Level of Evidence
Somatosensory retraining to improve sensory discrimination may be considered for stroke survivors with somatosensory loss.	IIb	B
Bilateral training paradigms may be useful for upper limb therapy.	IIb	A
Acupuncture is not recommended for the improvement of ADLs and upper extremity activity.	III	A

Adaptive Equipment, Durable Medical Devices, Orthotics, and Wheelchairs

Many patients require assistive devices, adaptive equipment, mobility aids, wheelchairs, and orthoses to maximize independent functioning after stroke. Many types of adaptive devices and equipment are available. Type and level of functional deficit, degree of achieved adaptation, and the structural characteristics of the living environment determine the need for a particular item.

A vast array of adaptive devices are available, including devices to make eating, bathing, grooming, and dressing easier for patients with functional limitations. The Convention on the Rights of Persons With Disabilities supports facilitating access by individuals with disabilities to quality mobility aids, devices, and assistive technologies by making them available at affordable cost.⁷⁵¹ Many patients may need to use adaptive devices early during rehabilitation but will not require long-term use. This should be taken into account when the provision of a device is considered. Examples of adaptive devices include (but are not limited to) eating utensils with built-up handles, rocker knives, plate guards, nonskid placemats, long-handled sponges for bathing, handheld showers, tub and shower chairs, grab bars for bathrooms, and elevated toilet seats. A meta-analysis found that OT increased independence in ADLs.⁷⁵² The protocols in these studies focused on improving personal ADLs, including the provision and training in the use of adaptive equipment.

Stroke can cause a number of gait impairments; consequently, stroke patients often have an unstable, inefficient walking pattern and a high risk for falls (see the sections Prevention of Falls and Mobility). More than half of stroke patients require an assistive device (cane, walker, wheelchair) to assist mobility, most frequently a cane.⁷⁵³ Studies that have assessed the immediate effects of different assistive devices provided in random order have shown that ambulatory function (speed, step length, functional ambulation category) was improved with a cane after stroke.^{754,755} Patients felt that their walking, walking confidence, and walking safety improved and said they would rather walk with an assistive device than delay walking to achieve a normal gait pattern.⁷⁵⁵ Walking devices increase the base of support around a patient's center of gravity and reduce the balance and effort needed to walk. Walking aids include (but are not limited to) the following:

- Single-point cane: a conventional cane that provides 1 point of contact and limited improvement in balance and stability.
- Tripod and quad cane: canes that have 3 or 4 points of contact and offer more stability than a single-point cane but are heavier, bulkier, and more awkward to use. A quad cane has been shown to reduce postural sway more than a single-point cane in patients with stroke.⁷⁵⁶
- Two-wheeled walkers, 4-wheeled walkers, or rollators (ie, 4-wheeled walker with a seat): devices that require the use of both arms and legs. They support more body weight than a cane and are more energy efficient but cannot be used on stairs. They should be lightweight and foldable for use outside the home. Four-wheeled walkers may require hand-motor coordination to manage hand-brakes on a downhill slope.

For individuals with stroke who cannot ambulate safely, a wheelchair can enhance mobility. Up to 40% of stroke patients have been reported to use a manual wheelchair at rehabilitation discharge.⁷⁵⁷ A wheelchair may be required when a patient is unable to ambulate or when there is concern about his/her ability to ambulate safely or functionally.⁷⁵⁸ The patient often propels the chair by using the less affected hand on 1 wheel and foot on the floor. Self-propulsion in a wheelchair early after a stroke has not been shown to be detrimental to muscle tone or functional outcomes.⁷⁵⁹ Many stroke survivors also use manual wheelchairs for longer-distance travel such as shopping or physician appointments although they are capable of short-distance ambulation within the home. In these situations, the wheelchair is typically propelled by a caregiver.

Although powered wheelchairs are less commonly used after stroke, many stroke patients can learn to use powered wheelchairs safely with appropriate training.⁷⁶⁰ Wheelchair designs vary greatly, and a wheelchair prescription should be specific to the patient's needs and environment and patient and family/caregiver preferences. The prescription of a wheelchair (manual or powered) in the community can increase participation and improve quality of life.^{761,762}

A common approach to managing the lower limb motor impairments resulting from a stroke is to use an orthotic device (an orthosis), most commonly an AFO. Meta-analyses have shown a favorable impact of lower limb orthoses on walking disability (speed), walking impairment (step/stride length), and balance (weight distribution in standing).^{659,605} However, the included studies examined only the immediate effects while the orthosis was worn.⁶⁵⁹ A recent meta-analysis and systematic review suggested the potential mechanism(s) associated with the above effects by demonstrating a positive effect of an AFO on ankle kinematics, knee kinematics in stance phase, kinetics, and energy cost.⁶⁵⁸ Two RCTs^{763,764} showed that after 3 months of AFO use, AFO users had better mobility while wearing the AFO. One small RCT⁷⁶⁴ found that although a dynamic hinged AFO improved ambulatory function over a standard AFO, it induced some dependence; the standard AFO group performed better after 3 months of use when walking without any orthosis. With respect to the patient's perspective, it is important to determine whether an individual is willing to wear an AFO regularly. Considerations to improve compliance with using an AFO

include verification that it fits correctly and comfortably and is acceptable in appearance.

Recommendations: Adaptive Equipment, Durable Medical Devices, Orthotics, and Wheelchairs	Class	Level of Evidence
Ambulatory assistive devices (eg, cane, walker) should be used to help with gait and balance impairments, as well as mobility efficiency and safety, when needed.	I	B
AFOs should be used for ankle instability or dorsiflexor weakness.	I	B
Wheelchairs should be used for nonambulatory individuals or those with limited walking ability.	I	C
Adaptive and assistive devices should be used for safety and function if other methods of performing the task/activity are not available or cannot be learned or if the patient's safety is a concern.	I	C

Motor Impairment and Recovery: Deconditioning and Fitness After Stroke

People having sustained a stroke present with varying degrees of compromised cardiorespiratory fitness, as reflected in peak $\dot{V}O_2$ levels of 8 to 22 mL $O_2 \cdot kg^{-1} \cdot min^{-1}$ (an average of $\approx 53\%$ of age- and sex-matched normative values).⁷⁶⁵ Given that 15 to 18 mL $O_2 \cdot kg^{-1} \cdot min^{-1}$ is deemed necessary for independent living, the state of fitness after stroke is a significant health, functional, and quality-of-life issue.⁷⁶⁶ Multiple factors before stroke, at the time of stroke, and after stroke help explain this state. The result is often a profound and persistent deconditioned state that leads to further physical inactivity, reduced socialization, and heightened risk of further vascular events, including a second stroke.

The lifetime risk of stroke recurrence among people with stroke is $\approx 30\%$, and the risk of either nonstroke vascular death or myocardial infarction is $\approx 2\%/y$.⁷⁶⁷ Recurrence of stroke has been found to vary by sex: 24% of women and 42% of men experience a recurrence within 5 years of onset.^{768,769} The reported rates of vascular risks are high among people who have a recurrence: The prevalence of hypertension (75%), ischemic heart disease (37%), hyperlipidemia (56%), atrial fibrillation (29%), and diabetes mellitus (24%) is significant in individuals who sustain a second stroke.⁷⁷⁰ For a comprehensive and timely set of evidence-based recommendations for all clinicians who manage secondary prevention, the reader is directed to the AHA/ASA guidelines for the prevention of stroke in patients with stroke and transient ischemic attack.²⁰⁶

Activity level after stroke is an independent predictor of life satisfaction, after controlling for demographic variables and depression.⁷⁷¹ Low levels of physical activity have been documented across the continuum of stroke severity and care, even among people who have had what is considered a mild stroke.⁷⁷² A behavioral mapping study revealed that activity out of bed during acute stroke care (ie, <14 days after the onset of stroke) varied widely among the European countries studied, ranging between 2% and 56% of the total time of the observation periods.⁷⁷³ Stroke rehabilitation sessions have

been reported to be of inadequate intensity to induce a cardiovascular training effect,^{774,775} with an average of 17 minutes spent in standing and walking per session.⁷⁷⁶ Daily ambulatory activity of community-dwelling stroke survivors has been reported to be 50%⁷⁷⁷ to 61%⁷⁷⁸ of that of nondisabled control subjects, less than that of older adults with other chronic health conditions of the musculoskeletal or cardiovascular system.⁷⁷⁹ At the same time, self-reports of physical activity among people with chronic stroke tend to be highly inflated.⁷⁸⁰

Sedentary behavior is defined as a waking behavior such as sitting or lying that involves an energy expenditure of <1.5 metabolic equivalents (METs; 1 MET is the amount of oxygen consumed while sitting at rest and is ≈ 3.5 mL $O_2 \cdot kg^{-1} \cdot min^{-1}$). Less sedentary behavior has been found to be an independent predictor of successful aging among individuals ≥ 45 years of age.⁷⁸¹ Moreover, prolonged bouts of sedentary behavior and total amount of physical inactivity appear to be independently related to risk factors associated with metabolic syndrome (eg, increased waist circumference, body mass index, triglycerides, and plasma glucose).⁷⁸² To date, little research has been conducted on patterns of sedentary behavior after stroke. A cohort study reported that people after stroke ($n=25$) spent less time being physically active and had fewer breaks in sedentary behavior at 1 week, 3 months, and 6 months after stroke compared with nondisabled control subjects matched by age, sex, and body mass index.⁷⁸¹

Intervention strategies are needed to break the relentless poststroke cycle of reduced physical activity leading to further reductions in functional capacity and heightened risk of secondary complications. The central role that aerobic exercise plays in improving cardiorespiratory fitness is well known and strongly supported by evidence.⁷⁸³ It is now clear that people with mild or moderate stroke are capable of improving their exercise capacity through exercise or structured physical activity.^{784–786} Enhanced fitness enables individuals to engage in daily physical activities at a lower percentage of their maximal capacity and hence with a lower physiological burden.⁷⁸⁷ Exercise-induced gains in peak $\dot{V}O_2$ have been relatively modest, with the magnitude of improvement ranging from 0.3 METs⁷⁸⁸ to 1.2 METs⁷⁸⁹ in trials of individuals in the subacute poststroke period and averaging ≈ 0.5 METs in trials of individuals with chronic stroke. However, even modest improvements in exercise capacity are associated with reduced cardiac complications in people with coronary artery disease⁷⁹⁰ and increased survival (10%–25% reduction in mortality for every 1-MET increase in exercise capacity).⁷⁹¹

Emerging research suggests that aerobic exercise after stroke confers clinically meaningful health benefits in numerous physical and psychosocial domains that extend well beyond the cardiorespiratory system. At the impairment level, some evidence exists that exercise positively affect bone health⁷⁹² (but not risk of fracture²⁵³), fatigue,⁴¹¹ executive functioning and memory, depressive symptoms,^{794,795} and emotional well-being¹⁸⁸ (see the earlier section on the benefits of exercise for poststroke depression). At the activity level, improvements have been noted in walking ability⁷⁹⁶ (endurance more than speed⁷⁹⁷) and upper extremity muscle strength.⁶⁸⁰ At the participation level, preliminary evidence has reported an association between exercise training after

stroke and social participation,¹⁸⁸ as well as return to work.⁷⁹⁹ Finally, a meta-analysis reported that exercise interventions for community-based stroke survivors have significant effects on health-related quality of life, which is arguably the ultimate goal of stroke rehabilitation.⁸⁰⁰

The role of exercise in preventing further vascular events after stroke, including a second stroke, myocardial infarction, and vascular death, has not been firmly established.⁷⁸⁶ There is evidence that aerobic exercise as a stand-alone intervention after stroke improves certain vascular risk factors, including glucose intolerance,⁸⁰¹ vascular stiffness,⁸⁰² high resting blood pressure,^{803,804} and elevated total cholesterol.⁸⁰³ A multifaceted approach that combines nonpharmacological interventions (ie, exercise, dietary advice, lifestyle counseling, and patient education) and appropriate pharmacological therapy has been encouraged,⁸⁰⁵ but the effectiveness of specific nonpharmacological components remains to be investigated.⁸⁰⁶ Pilot studies of second stroke prevention using a cardiac rehabilitation approach have demonstrated a reduction in cardiac risk scores⁸⁰⁷ and improvements in total cholesterol, body composition, and resting blood pressure,⁸⁰⁸ but these results must be confirmed in larger, controlled trials. Despite a lack of robust evidence, exercise and physical activity are regarded as key components of comprehensive stroke risk-reduction efforts.²⁰⁶

Individually Tailored Exercise Program Prescription

Active participation in exercise should be initiated early after stroke for several reasons: to minimize the detrimental effects of bedrest and inactivity, to capitalize on heightened neuroplasticity present in the early poststroke period, and to begin the important process of fostering exercise self-efficacy and self-monitoring. Mobilization within 24 hours after stroke has been shown in a phase II trial to accelerate recovery of walking and functional ability⁸⁰⁹; however, a recent study reported possible detrimental effects with such early activity.⁸¹⁰ In the recently completed AVERT RCT, the high-dose, very early mobilization protocol was associated with a reduction in the odds of a favorable outcome at 3 months.⁵⁸ In contrast to very early mobilization, there is growing evidence that the initiation of aerobic exercise in the subacute period (ie, a mean of 11–78 days after stroke) is safe and effective in improving exercise capacity and walking endurance.^{784,789} Specific recommendations for graded exercise testing can be found in the AHA guideline on stable ischemic heart disease.^{811,812} The ASH/ASA scientific statement “Physical Activity and Exercise Recommendations for Stroke Survivors”⁸¹³ provides more details on the pre-exercise evaluation.

As with all aspects of stroke rehabilitation, the training regimen should emphasize repetition, gradually progressive task difficulty, and functional practice.⁸¹⁴ The standard parameters of exercise prescription, that is, mode, frequency, duration, and intensity, require careful consideration to ensure a safe intervention that accommodates the individual’s functional limitations, comorbidities, motivation, and goals. Because the optimal training parameters have not been determined specifically for the stroke population,⁸¹⁵ current recommendations are based on general exercise guidelines⁸¹⁶ and on protocols shown to be effective in training studies involving people after stroke.⁷⁹⁶ A wide range of exercise modes (eg,

treadmill, body weight–supported treadmill, recumbent bicycle, cycle ergometer, stepper, aqua aerobics) have been used effectively in training studies.⁷⁹⁶ Because overground walking at self-selected speeds after stroke elicits oxidative stress in the range of 2.6 METs⁸¹⁸ to 3.4 METs,⁸¹⁹ it may be an appropriate aerobic modality for people who are moderately unfit. Preliminary evidence also suggests that participants in the chronic poststroke period can achieve low to moderate exercise intensities when playing an active video game (Nintendo Wii Sports).⁸²⁰ Furthermore, a recent trial involving people with subacute stroke demonstrated greater gains in peak $\dot{V}O_2$ with a combination of robot-assisted gait training and conventional PT than conventional therapy alone.⁸²¹

There is some evidence that the combination of aerobic and strengthening exercises in nonstroke populations enhances health outcomes (eg, reducing resting blood pressure⁸²² and metabolic syndrome risk factors⁸²³). However, conclusions from a meta-analysis indicated the need for further investigation to determine whether combining aerobic and strengthening exercises bestows similar advantages in the stroke population.⁷⁸⁵ Since then, a small, single-cohort study involving individuals with chronic stroke reported improved muscle strength and walking endurance but no change in peak $\dot{V}O_2$ after an 8-week program of lower extremity strength training at 85% to 95% of 1-repetition maximum.⁸²⁵

Benefits derived from aerobic training are dose dependent. The appropriate total volume of exercise, achieved through various combinations of frequency, duration, and intensity, is key to attaining and maintaining cardiorespiratory fitness. Nevertheless, there appears to be a minimal threshold for each parameter to achieve the most favorable outcomes. The frequency of structured aerobic exercise should be at least 3 d/wk for a minimum of 8 weeks, with lighter forms of physical activity (eg, brisk walking, stair climbing) promoted on the other days of the week. The duration of each session should be a minimum of 20 minutes in the training zone in addition to 3- to 5-minute periods of low-intensity warm-up and cool-down. For very deconditioned individuals, including many people after stroke, exercise may be delivered in multiple bouts of ≤ 5 minutes in a single session or throughout the day.⁷⁸³

Exercise intensity is the most challenging parameter to determine but also the most critical to ensure that a dose that is safe, attainable, and adequate to elicit a training effect. Factors that affect intensity are baseline fitness level, neurological and cardiac status, comorbidities, motivation, and goals of the program. Heart rate is typically used to establish and monitor training intensity, with resting rate measured after a minimum of 5 minutes of quiet sitting and exercise heart rate measured with an electronic device. It is important to note that β -blocker medication depresses the heart rate response to exercise and that atrial fibrillation (common after stroke) yields a chronically irregular ventricular rate, thus posing challenges in the prescription of exercise intensity.⁸²⁶ Various recommendations have been made on the appropriate exercise intensity for patients after stroke, including “moderate training intensities,”²⁰⁶ 40% to 70% of heart rate reserve (maximal heart rate minus resting heart rate),⁸²⁷ and 50% to 80% of maximal heart rate.⁷⁸⁵ A meta-analysis concluded that for extremely unfit individuals, intensities as low as 30% of heart rate reserve can induce a cardiovascular training

effect.⁸²⁸ At the other end of the spectrum, 2 pilot exercise studies provided early evidence supporting the safe and effective use, at least in the chronic stroke population, of high-intensity exercise (ie, 60%–80% of heart rate reserve,⁸²⁹ 85%–95% of peak heart rate⁸³⁰). The recent AHA/ASA scientific statement “Physical Activity and Exercise Recommendations for Stroke Survivors”⁸¹³ gives more details on exercise/physical activity recommendations for stroke survivors.

Chronic Care Management: Home- and Community-Based Participation

Because exercise confers health benefits even years after stroke, participation in physical activity should be encouraged regardless of how much time has elapsed since stroke onset. The effectiveness of exercise training in the chronic stages of stroke is no longer in question; in fact, the vast majority of fitness trials have involved people at this stage of stroke chronicity.⁷⁹⁶ Moreover, it has long been recognized that benefits of training decline significantly without ongoing participation in physical activity.⁸³¹ Thus, physical activity designed to promote cardiovascular fitness should be an important aspect of community reintegration after stroke. However, adherence to regular physical activity is influenced by a host of individual factors (eg, stroke severity, preexisting/comorbid conditions, motivation, health beliefs, exercise history, fatigue, depression, adaptability, coping skills, cognition), social/cultural factors (eg, family support, social policies, professionals’ attitudes about exercise, social norms and stigmas), and environmental factors (eg, program costs, access to transportation, fitness facilities and equipment).^{832,833} These factors must be systematically addressed to achieve the goal of long-term commitment to healthy, active living behaviors among stroke survivors.

Strategies to instill long-term commitment to a physically active lifestyle should be initiated during formal stroke rehabilitation, but evidence to guide intervention is lacking.⁸³⁴ Considering the high likelihood of a prestroke history of sedentary behavior, fostering exercise self-efficacy is particularly important to ease the transition from structured, institution-based aerobic training to home- and community-based physical activity.⁸³⁴ Incorporating principles of adult learning (eg, observation, practice, repetition, relevance) and self-management (eg, problem solving, goal setting, making choices, taking action, using available resources) is essential.^{835,836} Early participation in fitness training and education on lifestyle choices, risk factor reduction, and secondary prevention may facilitate uptake of healthy behaviors. Myths about exercise (exercise is unsafe, causes second stroke, increases fatigability)^{833,837,838} need to be dispelled in the process of rehabilitation. Most important, patients’ preferences concerning exercise must be sought out and respected.⁸³⁹ Finally, stroke survivors who are unable to exercise will need alternative solutions to maintain an active and engaged lifestyle.

The fitness program should be customized on the basis of the participant’s functional limitations, long-term health-related goals, and social and environmental factors. Periodic monitoring of the intensity of the program and the participant’s fitness level and adherence may be reasonable. Investigations of the effectiveness of predischARGE counseling

in increasing long-term adherence to activity after stroke have yielded mixed results.^{840,841} In addition, a self-guided stroke workbook did not elicit demonstrable changes in physical activity.⁸⁴² It appears that passive approaches (professional advice, written material) alone are not adequate to increase physical activity after stroke.⁸⁴¹ Given that the most common motivator to physical activity after stroke is the opportunity to meet other stroke survivors,⁸³³ together with the findings that stroke survivors report greater preferences for exercising in groups and at fitness centers,⁸³⁹ it is prudent to direct resources to facilitating participation in physical activity in community settings. Developing partnerships between healthcare professionals and fitness centers or community exercise programs could help to address a concern expressed by patients after stroke that exercise instructors must be suitably trained and knowledgeable about stroke.⁸³⁷ Integrated care models that include periodic liaison between care providers and patients after stroke via telephone or electronic follow-up may be the solution to providing ongoing support for physical activity.⁸⁴³

Recommendations: Chronic Care Management: Home- and Community-Based Participation	Class	Level of Evidence
After successful screening, an individually tailored exercise program is indicated to enhance cardiorespiratory fitness and to reduce the risk of stroke recurrence.	I	A (for improved fitness); B (for reduction of stroke risk)
After completion of formal stroke rehabilitation, participation in a program of exercise or physical activity at home or in the community is recommended.	I	A

Treatments/Interventions for Visual Impairments
Treatments and interventions for visual impairments after stroke focus on 3 areas: deficits in eye movements, deficits in visual fields, and deficits in visual-spatial or perceptual deficits. There have been 7 systematic reviews of treatments for visual impairments after stroke.^{382,418,493,737,844,846,847} These systematic reviews covered reports up to 2011. The literature is generally limited in this area, and the methodological quality was poor in general or poorly reported, providing insufficient high-quality evidence on which to reach generalizable conclusions. However, limited evidence suggested that compensatory scanning training is effective at improving scanning and reading outcomes but not improving visual field deficits. There was insufficient evidence of the impact of compensatory scanning training on ADLs. There was also insufficient evidence about the benefits of vision restoration therapy (restitutive intervention) after stroke. Across these systematic reviews, 2 studies targeted eye movement deficits, 2 case studies and 1 nonrandomized prospective study assessed interventions for visual field cuts, and 3 studies dealt with perceptual deficits. In general, there was insufficient evidence to reach conclusions about the effectiveness of interventions for patients with any of these visual deficits after stroke. Barrett⁸⁴⁴ reviewed the behavioral optometry literature. Behavioral optometry proposes that eye and visual function can be improved through various vision therapy methods, including

eye exercises and the use of lenses, prisms, filters, occluders, specialized instruments, and computer programs to improve vision skills such as eye movement control, eye focusing, and coordination. Barrett concluded that there is a paucity of controlled trials in the literature to support behavioral optometry approaches and that a large majority of behavioral management approaches are not evidence based. However, there was evidence supporting the use of eye exercises for treatment of convergence insufficiency, the use of yoked prisms in stroke patients with visual field cuts, and the use of vision rehabilitation of visual field defects (selecting areas of residual vision that are then stimulated during computer-assisted training to achieve visual field enlargement).

A number of studies included as part of a broader review dealing with rehabilitation of cognitive deficits⁴¹⁸ focused on visual neglect, which is addressed elsewhere in this guideline. However, with regard to other forms of visual deficits, those studies concluded that systematic training of visual organization skills may be considered for individuals with visual perceptual deficits, without visual neglect, and after right hemispheric stroke as part of acute rehabilitation and that computer-based interventions intended to produce extension of damaged visual fields may be considered for people with traumatic brain injury or stroke.

In addition to those covered by the 7 systematic reviews, 3 studies dealt with treatments for visual impairments after stroke.^{848–850} Mödden et al⁸⁵⁰ concluded that computer-based compensatory therapy improved functional deficits after visual field loss compared with compensation strategies training (ie, standard OT). A 2010 study⁸⁴⁸ concluded that multimodal audiovisual exploration training is more effective than exploration training alone. Finally, a 2012 study⁸⁴⁹ reported that a virtual reality training group showed a significant difference in all Motor-Free Visual Perception Test raw scores and response times, with improvements in recognizing shapes, solving pictorial puzzles, and object perception.

Recommendations: Treatments/Interventions for Visual Impairments	Class	Level of Evidence
For deficits in eye movements:		
Eye exercises for treatment of convergence insufficiency are recommended.	I	A
Compensatory scanning training may be considered for improving functional ADLs.	IIb	B
Compensatory scanning training may be considered for improving scanning and reading outcomes.	IIb	C
For deficits in visual fields:		
Yoked prisms may be useful to help patients compensate for visual field cuts.	IIb	B
Compensatory scanning training may be considered for improving functional deficits after visual field loss but is not effective at reducing visual field deficits.	IIb	B
Computerized vision restoration training may be considered to expand visual fields, but evidence of its usefulness is lacking.	IIb	C

Recommendations: Treatments/Interventions for Visual Impairments (Continued)	Class	Level of Evidence
For visual-spatial/perceptual deficits:		
Multimodal audiovisual spatial exploration training appears to be more effective than visual spatial exploration training alone and is recommended to improve visual scanning	I	B
There is insufficient evidence to support or refute any specific intervention as effective at reducing the impact of impaired perceptual functioning.	IIb	B
The use of virtual reality environments to improve visual-spatial/perceptual functioning may be considered.	IIb	B
The use of behavioral optometry approaches involving eye exercises and the use of lenses and colored filters to improve eye movement control, eye focusing, and eye coordination is not recommended.	III	B

Hearing Loss

The healthcare provider's ability to effectively communicate with a patient who has had a stroke is essential to provide adequate patient care. Unfortunately, hearing impairment is common among stroke patients, and this may significantly affect communication. This impairment must be considered when communicating with patients to provide effective patient-centered care.

Hearing impairment is commonly associated with aging, and the associated communication difficulties are only further exacerbated after stroke. It has been reported that the most common type of communication impairment within an acute hospital stroke unit is a hearing impairment, with estimates that 67% to 90% of these patients have a mild or greater hearing impairment.⁸⁵¹ Although a sudden onset of hearing loss resulting from a stroke is uncommon, stroke patients often have a preexisting or an undiagnosed hearing loss. In some instances, difficulty hearing may simply be caused by cerumen impaction or may be attributable to age-related hearing loss.⁸⁵¹ Stroke patients with communication or cognitive impairments may be unable to relay information about their hearing history. Reports from family or significant others often give healthcare providers some indication of the patient's hearing abilities before the stroke. It is recommended that any noticeable hearing impairment be assessed and documented to improve patient care. Edwards et al⁸⁵² reported that 86% of stroke patients in acute care facilities had a hearing impairment that was not documented in their chart.

Amplification can often help patients who have had a stroke to overcome the barrier of a hearing impairment. One study reported that of 52 patients who had suffered a stroke and had a hearing impairment, 11 (21%) owned hearing aids.⁸⁵¹ By verifying that the hearing aids or amplification devices are working and reminding the patients to wear them, healthcare providers will be able to better communicate with these patients. Unfortunately, not all patients with a hearing impairment have hearing aids. In this case, it is important to incorporate communication strategies such as looking at the

patient when talking to him/her and minimizing the level of background noise.

Recommendations: Hearing Loss	Class	Level of Evidence
If a patient is suspected of a hearing impairment, it is reasonable to refer to an audiologist for audiometric testing.	Ila	C
It is reasonable to use some form of amplification (eg, hearing aids).	Ila	C
It is reasonable to use communication strategies such as looking at the patient when speaking.	Ila	C
It is reasonable to minimize the level of background noise in the patient's environment.	Ila	C

Transitions in Care and Community Rehabilitation

Ensuring Medical and Rehabilitation Continuity Through the Rehabilitation Process and Into the Community

The transition from inpatient care to home after a stroke can be difficult for patients and caregivers. Those patients who require ongoing rehabilitation after discharge should continue to be followed up by a care team with expertise in stroke rehabilitation whenever possible. Patients who do not require additional rehabilitation services and are discharged to home or who are profoundly and permanently disabled and discharged to a long-term care setting can be managed by a primary care provider.

One recent systematic review of 9 RCTs looked at the effectiveness of various models of primary care-based follow-up after stroke. The studies included interventions using stroke support workers, care coordinators, or case managers. As a result of the wide variability of the methodological quality of the studies, interpretation was limited. The authors noted that although patients and caregivers receiving follow-up were generally more satisfied with some aspects of communication and had a greater knowledge of stroke, there did not appear to be any gains in physical function, mood, or quality of life compared with those who did not.⁸⁵³ Another systematic review examining transitional care models after stroke or myocardial infarction showed that hospital-initiated transitional care could improve some outcomes in adults hospitalized for stroke or myocardial infarction.⁸⁵⁴

Although not specific to stroke, a 2012 Cochrane study to determine the effectiveness of discharge planning for patients moving from an acute hospital stay to a home setting evaluated the results of 24 RCTs comparing individualized discharge plans with routine discharge care that was not tailored to the individual patient. Using data from 8098 patients, the investigators found that hospital length of stay and hospital readmissions were “statistically significantly reduced for patients admitted to hospital with a medical diagnosis and who were allocated to discharge planning (mean difference length of stay -0.91, 95% CI -1.55 to -0.27, 10 trials; readmission rates RR 0.82, 95% CI 0.73 to 0.92, 12 trials).” For elderly patients with a medical condition, they found no significant difference between groups with

respect to mortality (RR, 0.99; 95% CI, 0.78–1.25, 5 trials) or being discharged from hospital to home (RR, 1.03; 95% CI, 0.93–1.14, 2 trials). The authors concluded that a “discharge plan tailored to the individual patient probably brings about reductions in hospital length of stay and readmission rates for older people admitted to hospital with a medical condition” but that the impact of discharge planning on mortality, health outcomes, and cost remained unclear.⁸⁵⁵ For patients who have suffered a stroke and are being discharged from acute care, the discharge planning should include rehabilitation professionals who can identify long-term needs and help organize provision of those services.

Alternative methods of communication and support such as telephone visits, telehealth, or Web-based support are newer options that should be considered, particularly for patients in rural settings who may have difficulty traveling for medical care once they are discharged from formal rehabilitation services.⁸⁵⁶ These technologies can be used for long-distance counseling, problem solving, and educational sessions, as well as for transmitting critical data such as blood pressure readings, weight, or laboratory results.

Recommendation: Ensuring Medical and Rehabilitation Continuity Through the Rehabilitation Process and Into the Community	Class	Level of Evidence
It is reasonable to consider individualized discharge planning in the transition from hospital to home.	Ila	B
It is reasonable to consider alternative methods of communication and support (eg, telephone visits, telehealth, or Web-based support), particularly for patients in rural settings.	Ila	B

Social and Family Caregiver Support

As a result of the complexity of the disease, the deficits and disability, and the change in family and significant other dynamics, the caregiver and family are integral to the post-stroke treatment plan. A major challenge is that 12% to 55% of caregivers suffer from some emotional distress,²⁰⁹ most commonly depression.²³⁸ A growing body of research is focused on the caregiver’s quality of life and on treatment strategies to benefit both the caregiver and the stroke survivor.

Families and caregivers of stroke survivors sustain a significant impact on their psychosocial health. Worldwide, depression is observed not only in the patient but also in the caregiver. Untreated depression is associated with a lower quality of life and increased burden for the caregiver and survivor.⁸⁵⁷ In Korea, increased burden was related to increased patient depression and insufficient support. In contrast, an American study found that increased caregiver burden is more closely correlated with lack of time for self.⁸⁵⁸ Smith and colleagues⁸⁵⁹ found that the caregiver needs varied as a function of age. Younger caregivers want information and training and are more inclined to criticize the healthcare system, whereas older caregivers need support to maintain a positive outlook and are less inclined to criticize the healthcare system.

Since the previous guidelines published in 2005, many researchers have investigated the caregiver perspective and better understand the interventions most likely to improve quality of life and to decrease burden. The Cochrane Collaboration

found that information improved the patient's and caregiver's knowledge while also slightly decreasing patient depression. The most effective educational programs included active involvement and follow-up by the educator. Education programs for caregiver and stroke participant should include supportive problem solving and skill development,⁸⁶⁰ "how to's" of physical care needs and financial assistance,⁸⁶¹ medications,⁸⁶² respite, domestic assistance, and reassurance.⁸⁶³ Ongoing support for the caregiver favorably affects the stroke survivor and caregiver. This support comes in many different actions. Steiner et al⁸⁶⁴ studied physical and emotional support, whereas Campos de Oliveira⁸⁶⁵ more clearly defined the support as a needed support structure. The caregivers need either family or friends to provide emotional and physical assistance, and the caregivers need the healthcare providers to help them establish and maintain this over time.⁸⁶⁶ Counseling can also be a helpful intervention.⁸⁶⁷ In summary, healthcare professionals need to consider the patient, along with a diverse set of support options and treatments for the family and primary caregiver.

Recommendations: Social and Family Caregiver Support	Class	Level of Evidence
It may be useful for the family/caregiver to be an integral component of stroke rehabilitation.	IIb	A
It may be reasonable that family/caregiver support include some or all of the following on a regular basis:	IIb	A
Education		
Training		
Counseling		
Development of a support structure		
Financial assistance		
It may be useful to have the family/caregiver involved in decision making and treatment planning as early as possible and throughout the duration of the rehabilitation process.	IIb	B

Referral to Community Resources

Successful transition to the community requires careful assessment of the match between patient needs and the availability of formal and informal resources. Referral to appropriate local community resources can help to support the needs and priorities of the patient and the family or caregiver. Some services can be organized and in place before hospital discharge, whereas referral to some community resources may be provided on transition to the community. A range of community resources are available that patients and their families/caregivers may desire to access immediately or in the future as their needs change.

Formal referral may be required for services such as vocational counseling, psychological services, social services, sexual health counseling, driver evaluation, or home environment assessment. Referral to a day service program may be appropriate for a patient who may benefit from a structured program and for caregivers who need respite time.

Multiple potential resources may assist stroke patients and their families/caregivers in the management of the long-term effects of stroke such as local stroke survivor and caregiver

support groups, leisure and exercise programs, respite care, self-management programs, and home support (eg, Meals on Wheels).

More than 50% of stroke survivors require support with IADLs.⁸⁶⁸ A high proportion of stroke survivors 1 to 5 years after injury use community services, with the most frequently accessed being household services (housework, lawn/garden care, and Meals on Wheels) and then therapy services (eg, PT).⁸⁶⁸

Caregivers have identified that it is important to know what resources are available and to be able to access them.⁸⁶⁹ Stroke patients and their caregivers can be active in managing their chronic condition if they have appropriate information and resources. If stroke survivors and caregivers are to be active in their decision making and the management of the long-term effects of stroke, appropriate information delivered in a timely and effective format is necessary. It is critical that the process involve assessment of an individual's needs, education about available resources, linking of patient and resources, referrals, and follow-up to ensure the individual receives the necessary services. Health providers may wish to use a checklist to identify whether referral to other services is warranted.⁸⁷⁰ A meta-analysis of 21 trials showed that the provision of information (including local resources) to patients and their caregivers may improve aspects of patient satisfaction, improve knowledge of stroke, and reduce patient depression scores.⁸⁷¹

A systematic review⁸⁷² and meta-analysis⁸⁷³ demonstrated the growing recognition that functional outcomes (including motor, cognitive, and psychosocial function) can be improved or at least maintained in chronic stroke with community interventions. In addition, a meta-analysis of 17 RCTs showed that lifestyle interventions (eg, health promotion or education, lifestyle counseling) may reduce the risks leading to another stroke or cardiovascular event.⁸⁷⁴ A meta-analysis of 8 RCTs showed that exercise referral schemes that provide a clear referral by primary care professionals to third-party professionals to increase exercise or physical activity can increase the number of participants who achieve 90 to 150 min/wk of moderate physical activity and reduce depressive symptoms in sedentary individuals with or without a medical diagnosis (obesity, hypertension, depression, diabetes mellitus).⁸⁷⁵ In a qualitative study, stroke survivors described great physical and psychological well-being after participation in an exercise referral scheme.⁸⁷⁶

Recommendations: Referral to Community Resources	Class	Level of Evidence
It is recommended that acute care hospitals and rehabilitation facilities maintain up-to-date inventories of community resources.	I	C
Patient and family/caregiver preferences for resources should be considered.	I	C
It is recommended that information about local resources be provided to the patient and family.	I	C
It is recommended that contact with community resources be offered through formal or informal referral.	I	C
Follow-up is recommended to ensure that the patient and family receive the necessary services.	I	C

Rehabilitation in the Community

The Centers for Medicare & Medicaid Services define community as one of the following settings: home, board and care, transitional living, intermediate care, or assisted living residence. More than 80% of the >6 million survivors of stroke in the United States live in the community, most of them at home, and the majority with some residual functional limitations. Studies have documented that 35% to 40% of individuals have limitations in basic ADLs 6 months after a stroke. More than 50% have limitations in ≥ 1 IADLs.^{794,877}

There is substantial evidence that rehabilitation services, particularly exercise-based programs, provided in the community after discharge from acute or institutional care can improve cardiovascular health and decrease the risk of cardiovascular events, leading to increased short-term survival rates for individuals who have experienced a stroke.^{878,879} Other community-based intervention trials have demonstrated enhanced ambulation and mobility, better self-care, and greater functional independence.⁸⁸⁰

Benefits associated with community- and home-based rehabilitation programs have been reported for a variety of outcomes, including reduced costs, decreased length of stay in hospitals or institutional settings, more opportunity for patient and family involvement in the treatment process, and less stress on caregivers and family members.^{881,882}

It has also been consistently reported that individuals recovering from a stroke and their family members or caregivers prefer home- or community-based rehabilitation programs over center- or institutionally located rehabilitation services for a variety of practical and personal reasons.⁸⁸¹ Patient satisfaction with home-based rehabilitation programs is generally higher than for institutionally based alternatives.⁸⁸² Because the potential for recovery exists regardless of age and time after stroke and because fewer financial resources appear to be dedicated to providing optimal care during the later phases of stroke recovery, family caregiver education and support are recommended. Intervention, referrals, and follow-up care based on detailed caregiver assessments conducted during the survivor's inpatient stay are likely to smooth the transition of care to the home setting.¹¹ There is growing evidence for the effectiveness of stroke family caregiver and dyad (caregiver and patient) interventions.⁸⁸³ Among the Class I, Level of Evidence A recommendations about caregiver and dyad interventions were the following: (1) Interventions that combine skill building with psycho-educational strategies should be chosen over interventions that only use psycho-educational strategies; (2) interventions that are tailored or individualized on the basis of the needs of stroke caregivers should be chosen over nontailored, one-size-fits-all interventions; (3) postdischarge assessments with tailored interventions based on changing needs should be performed to improve caregiver outcomes; (4) interventions that are delivered face to face or by telephone are recommended; and (5) interventions consisting of 5 to 9 sessions are recommended.

The ability to translate these findings into targeted intervention programs and guidelines for the care of individuals with stroke is complicated by several factors.^{884,885} There is substantial variability in the timing of the initiation of home-based treatment programs. Home-based rehabilitation may

not be appropriate for all individuals with stroke, depending on level of severity, comorbidities, or the need for specialized treatment or equipment. Existing studies comparing community- and home-based rehabilitation vary substantially in the duration and intensity of the intervention and in the nature and complexity of the treatment programs provided.⁸⁸¹ For example, some treatment programs are single interventions such as exercise; other programs involve multiple components requiring levels of specialized expertise.

Issues related to the fidelity and integrity of the treatment, patient safety, and the lack of equipment and capacity to provide selected interventions in a home or community setting have been identified as concerns associated with home-based rehabilitation.⁸⁸⁶ Research-based evidence on potential adverse effects associated with rehabilitation programs conducted in the home and community is limited.

The majority of trials and reviews of community-based rehabilitation programs have compared home-based intervention programs with programs provided in centers or hospital/clinic-based outpatient programs.⁸⁸¹ Several studies published since the 2005 stroke rehabilitation clinical practice guidelines have examined a combination of ESD programs and community rehabilitation and compared these programs with standard inpatient and outpatient rehabilitation services. Langhorne and colleagues^{17,18} found that the combination of ESD and community rehabilitation reduced inpatient length of stay and hospital readmission rates and increased functional independence and the ability of patients to live at home and participate in the community.

A systematic review by Hillier and Inglis-Jassiem⁸⁸¹ examined data comparing the benefits of home-based programs and programs in rehabilitation centers for individuals with stroke living in the community. Eleven trials met the inclusion criteria. Functional outcome data were pooled for the Barthel Index across the majority of the trials. Functional status was significantly improved for the home-based cohort at 6 weeks and 3 to 6 months. The difference between home-based and rehabilitation center groups was less clear after 6 months. Cost benefits and caregiver satisfaction were secondary measures and favored the home-based intervention trials.

A widely cited Cochrane Collaboration review^{887,888} examined therapy-based rehabilitation services for stroke patients at home (Outpatient Service Trialists). The review examined trials meeting the Cochrane Collaboration criteria and compared home-based therapy with conventional care or no care within 1 year of hospital discharge for individuals with stroke. The primary outcomes were adverse events, deterioration in ability to perform ADLs, and level of improvement in ADL outcomes. The authors concluded that home-based therapy reduced the odds of a poor outcome, that is, death or deterioration in the ability to perform ADLs. Patients in the home-based therapy program also demonstrated improved ADL abilities compared with individuals in the usual or no treatment groups.^{887,888}

The majority of trials and reviews examining community- and home-based rehabilitation programs in individuals with stroke have focused on functional, mobility, or motor outcomes. A recent meta-analysis by Graven and others⁷⁹⁴ examined the impact of community-based rehabilitation on reducing

depression and increasing participation and health-related quality of life in individuals with stroke. The 54 studies included in the review were divided into 9 intervention categories. Analyses revealed significant reductions in depressive symptoms. The reduction in depressive symptoms was associated with exercise interventions. Treatments involving leisure and recreational activities showed moderate effects for the outcomes of participation and health-related quality of life. Comprehensive, multifactorial rehabilitation interventions demonstrated limited evidence for depression and participation but showed strong evidence for health-related quality-of-life outcomes.⁷⁹⁴

Recommendations: Rehabilitation in the Community	Class	Level of Evidence
Patients with stroke receiving comprehensive ADL, IADL, and mobility assessments, including evaluation of the discharge living setting, should be considered candidates for community- or home-based rehabilitation when feasible. Exclusions include individuals with stroke who require daily nursing services, regular medical interventions, specialized equipment, or interprofessional expertise.	I	A
It is reasonable that caregivers, including family members, be involved in training and education related directly to home-based rehabilitation programs and be included as active partners in the planning and implementation or treatment activities under the supervision of professionals.	Ila	B
A formal plan for monitoring compliance and participation in treatment activities may be useful for individuals with stroke referred for home- or community-based rehabilitation services. A case manager or professional staff person should be assigned to oversee implementation of the plan.	IIb	B

Sexual Function

Sexuality is an important aspect of poststroke quality of life for both patients and their significant others. Although there is substantial individual variation, overall stroke survivors tend to experience a high prevalence of sexual dysfunction. Comorbid medical conditions (eg, diabetes mellitus, hypertension, depression), medication side effects, stroke-related physical and functional deficits, lack of knowledge, and concerns about safety, role changes, and change in libido can affect the patient's sexual function. Healthcare workers need to help the patient and significant other navigate through the issues surrounding sexual function.

Multiple studies indicate that stroke survivors and their significant others have concerns about sexuality but are frequently reluctant to ask their healthcare providers about these concerns.⁸⁸⁹ This reluctance may stem from the patient's embarrassment or other cultural barriers, as well as a lack of knowledge on the part of the healthcare provider. The greater the patient's disability is, the greater is the likelihood of sexual dysfunction and decreased sexual life satisfaction.⁸⁹⁰ Stroke survivors report a desire for more information about sexuality from healthcare providers, physicians in particular.⁸⁹¹ It is important for the patient and significant other to know

that sex is not contraindicated after stroke. The most common sexual dysfunctions after stroke are decreased libido, erection and ejaculation disorders in men, lubrication and orgasm in women, and self-image and role changes for both men and women. Interventions and education about sexuality that address these concerns such as positioning, timing, open communication, and functional treatments can be helpful. Additional training for healthcare providers on this topic, including methods of appropriately approaching patients and their partners to discuss sexuality, may be needed.⁸⁹²

Recommendation: Sexual Function	Class	Level of Evidence
An offer to patients and their partners to discuss sexual issues may be useful before discharge home and again after transition to the community. Discussion topics may include safety concerns, changes in libido, physical limitations resulting from stroke, and emotional consequences of stroke.	IIb	B

Recreational and Leisure Activity

Engagement in leisure and recreational pursuits is important to health.^{893–896} Active leisure and recreational activities have been targeted as particularly important.^{894,895,897} However, individuals with stroke are limited in their ability to engage in leisure and recreational activities, particularly active ones.^{779,898–900}

In general, poststroke rehabilitation in the United States provides little attention to leisure and recreation.⁹⁰² Individuals with stroke report that they engage in significantly fewer leisure and recreation activities than they did before the stroke.^{898,899} In addition, the leisure activities in which they do engage have shifted from active to sedentary activities such as television watching and reading.⁸⁹⁸ Limited research examines the efficacy of rehabilitation for increasing participation in leisure and recreation activities. However, several studies (1 qualitative study, 2 RCTs, and 2 systematic reviews) suggest that therapy targeted at leisure/recreation and the provision of some adaptive equipment may facilitate increased engagement in leisure or recreation activities.^{794,903,904,906} Although therapy was variable across the studies, in several, the therapy consisted of education about the importance of being physically active, education on community resources, and training in problem solving around barriers to being physically active.^{794,903} One study that showed that such programming facilitated long-term increased physical activity engagement offered this kind of programming during rehabilitation, suggesting that such programming could begin early during rehabilitation.^{908,909} It must be noted, however, that this study took place in Europe, involved much longer durations of rehabilitation than individuals experience in the United States, and involved individuals with a variety of disabling conditions (only 26% were individuals with stroke); in addition, results were not broken down by disability condition. The provision of a wheelchair may be critical because many individuals with stroke who are able to ambulate do not have the endurance to ambulate for long periods in the community.⁹⁰⁶

Recommendations: Recreational and Leisure Activity	Class	Level of Evidence
It is reasonable to promote engagement in leisure and recreational pursuits, particularly through the provision of information on the importance of maintaining an active and healthy lifestyle.	Ila	B
It is reasonable to foster the development of self-management skills for problem solving for overcoming barriers to engagement in active activities.	Ila	B
It is reasonable to start education and self-management skill development about leisure/recreation activities during and in conjunction with in-patient rehabilitation.	Ila	B

Return to Work

In the United States, ≈20% of strokes occur in individuals who are of vocational age.⁹¹⁰ Vocational roles provide a social identity and contribute to increased self-esteem and life satisfaction.⁹¹¹ It is estimated that about one third of the economic burden of stroke through the year 2050 will be attributable to lost earnings after stroke.⁹¹²

The percentage of individuals who were working before their stroke who return to work after stroke varies widely across studies, from 20%⁹¹³ to 66%.⁹¹⁴ This stems from large differences in sample characteristics, healthcare and social system differences in different countries, various definitions of work, and variable follow-up periods. It is clear, however, that a large percentage of individuals with stroke who are of vocational age do not return to work. It is estimated that one third of the \$1.75 trillion in annual costs¹ associated with stroke are attributable to lost earnings in the United States alone.⁹¹² The factors associated with return to work have also varied across different studies. Factors most frequently found to be associated with return to work are younger age, less severe impairments, independence in ADLs, good communication skills, good higher-level cognitive skills and processing speed, and a white collar profession.^{915–921} Some of those who do return to work have been able to return full-time to their previous jobs; some have required job modifications or alternative jobs; and others were able to return only part-time.^{890,917,919} The ability to resume driving may also be an important factor in being able to return to employment.⁹¹⁵

Because several of the variables presenting barriers to return to work are modifiable, therapy targeted at vocational goals has the potential to increase return-to-work rates for individuals with stroke. However, no controlled trials have examined the efficacy or effectiveness of therapy targeted at vocational goals or vocational rehabilitation programs, and a structured review found insufficient evidence to support or refute the efficacy of any specific vocational rehabilitation program.⁹²² Several case studies suggest that for some individuals, therapy targeted at vocational goals can result in successful return to work.^{923,924} Chan and colleagues⁹²⁵ reported that their vocational rehabilitation program facilitates 55% of their enrollees to return to work. However, the lack of enrollee description makes it unclear how to interpret their success rate because several studies have found similar return-to-work rates without formal vocational rehabilitation. Although evidence is limited, many clinicians advise that for individuals

considering return to work, an assessment of cognitive, perception, physical, and motor abilities be performed to determine readiness and the needed accommodations to return to work. This assessment should be tailored to the individual's needs and capabilities for the specified job situation and may include executive functions, high-level oral and written communication, and fatigue. Once performance under the best conditions has been assessed, further assessment under conditions of fatigue and stress may be useful to mimic potential job situations.

Discrimination against individuals with disabilities remains common in the workplace and may not be identified by the prospective employer as a reason for denying a disabled candidate employment. Familiarity with the provisions of the Americans With Disabilities Act and its requirements for “reasonable accommodation” is important for individuals seeking to return to a job after stroke or seeking a new position. Rehabilitation professionals can serve as a resource for motivated employers to help overcome workplace barriers for employees with disabilities.

Recommendations: Return to Work	Class	Level of Evidence
Vocationally targeted therapy or vocational rehabilitation is reasonable for individuals with stroke considering a return to work.	Ila	C
An assessment of cognitive, perception, physical, and motor abilities may be considered for stroke survivors considering a return to work.	IIb	C

Return to Driving

Driving is an essential IADL for many individuals in that it has a major impact on participation in activities outside the home.⁹²⁶ Between one third and two thirds of individuals after stroke resume driving after 1 year.^{927,928} However, because driving is a highly complex activity that requires skills in cognition, perception, emotional control, and motor control,⁹²⁹ the ability to drive is often affected by stroke.⁹²⁸ State law determines whether someone with a stroke is eligible to drive. The law concerning this topic varies by state. For example, in some states, individuals who have a neurological condition (stroke, traumatic brain injury, Parkinson disease, multiple sclerosis), among other non-neurological health conditions, are required to report their health condition to the appropriate state agency (eg, Department of Transportation or Department of Public Safety). After this reporting, the physician should assess patients' physical or mental impairments that might adversely affect driving abilities. Each case must be evaluated individually because not all impairments may give rise to an obligation on the part of the physician. In other states without self-reporting, physicians must take several initial steps before reporting: have a tactful but candid discussion with the patient and family about the risks of driving, suggest to the patient that he or she seek further treatment such as substance abuse treatment or OT, and encourage the patient and the family to decide on a restricted driving schedule. Efforts made by physicians to inform patients and families, to advise them of their options, and to negotiate a workable plan may render reporting unnecessary. Physicians should use their best judgment

when determining when to report impairments that could limit a patient's ability to drive safely. The physician's role is to report medical conditions that would impair safe driving as dictated by his or her state's mandatory reporting laws and standards of medical practice. Physicians should disclose and explain to their patients this responsibility to report. Physicians should protect patient confidentiality by ensuring that only the minimal amount of information is reported and that reasonable security measures are used in handling that information. Physicians should work with their state medical societies to create statutes that uphold the best interests of patients and community and that safeguard physicians from liability when reporting in good faith.⁹³⁰ The appropriate state agency determines whether the individual is allowed to keep his/her license or obtain a restricted license or whether another option is necessary. However, the decision about return to driving should happen with the physiatrist or primary care provider, patient with stroke, and family. If necessary, a driving rehabilitation specialist can perform a formal driving evaluation. The ASA Driving after Stroke Web site provides information on life after stroke.^{930a}

The majority of individuals who sustain a stroke want to and do return to driving within a year after stroke.^{927,928,931} Despite a significant number of individuals in whom driving ability is reduced^{928,932} and the incidence of reduced self-awareness of driving difficulties after stroke,⁹³³ very few individuals are ever formally assessed for driving, nor is return to driving discussed with them.^{72,928,934} This is clearly a neglected area in the current healthcare system surrounding rehabilitation services after stroke.

There are no standardized driving assessment batteries. Many assessments contain both neuropsychological tests and on-the-road testing. There is no clear consensus on whether neuropsychological tests adequately predict the ability to drive. Two recent reviews (1 systematic review,⁹³⁶ 1 meta-analysis⁹³⁷) examined the ability of neuropsychological tests to predict on-the-road driving test performance or voluntary cessation of driving across 37 studies (8 overlapping studies). The only neuropsychological test that was a significant predictor of fitness to drive in both reviews was the Trail Making Test B. There is great variation across studies in sample selection and in which neuropsychological tests were used to predict fitness to drive. For example, finding no effect for vision is likely the result of a biased sample excluding subjects with visual impairments consistent with state laws restricting such individuals from driving.⁹³⁷ Driving simulators offer the ability to test an individual for fitness to drive in dynamic environments that are safer than on-the-road tests.⁹³⁸ One cautionary note is that currently few studies have tested to what degree (if any) driving simulator performance is a sufficient predictor of on-the-road driving to determine the safety of return to driving. One study of 23 participants⁹³⁹ showed that the simulator performance variables of complex reaction time and distance to collision were able to correctly classify 85% of the participants as fit to drive or not. Because there is no single set of neuropsychological tests that can accurately predict fitness to drive, an on-the-road driving test should also be strongly considered, especially for individuals who possess the cognitive ability and are eligible on the basis of local laws.

Several studies have shown that some individuals with stroke who are unable to pass fitness-to-drive tests can do so after intervention.^{938,940–942} Intervention programs may involve adaptive equipment and training for the specific impairments interfering with driving (eg, infrared controls for 1-handed driving, cognitive training, vision training) or simulator training, on-road training, or their combination. Although few studies have tested the efficacy of driving training on driving ability, 2 studies have found simulator training to be superior to traditional cognitive training.^{938,941} One study showed that visual training with the Dynavision system (Dynavision LLC, West Chester, OH) did not result in increased driving ability.⁹⁴³ Unfortunately, other studies that investigated vision training and showed improved driving-related visual skills did not include measures of actual driving ability.⁹⁴⁴ Thus, the evidence is insufficient to determine whether visual training improves driving performance in those individuals with insufficient visual skills. In general, studies examining the efficacy of driver training suffer from small, heterogeneous samples. In addition, intervention programs in these studies do not appear to be specific to the impairments of the participants.

Recommendations: Return to Driving	Class	Level of Evidence
Individuals who appear to be ready to return to driving, as demonstrated by successful performance on fitness-to-drive tests, should have an on-the-road test administered by an authorized person.	I	C
It is reasonable that individuals be assessed for cognitive, perception, physical, and motor abilities to ascertain readiness to return to driving according to safety and local laws.	IIa	B
It is reasonable that individuals who do not pass an on-the-road driving test be referred to a driver rehabilitation program for training.	IIa	B
A driving simulation assessment may be considered for predicting fitness to drive.	IIb	C

Conclusions

Stroke rehabilitation requires a sustained and coordinated effort from a large team, including the patient and his or her goals, family and friends, other caregivers (eg, personal care attendants), physicians, nurses, physical and occupational therapists, speech-language pathologists, recreation therapists, psychologists, nutritionists, social workers, and others. Communication and coordination among these team members are paramount in maximizing the effectiveness and efficiency of rehabilitation and underlie this entire guideline. Without communication and coordination, isolated efforts to rehabilitate the stroke survivor are unlikely to achieve their full potential.

The evidence base on specific stroke rehabilitation interventions has expanded considerably in recent years, although many gaps remain. In addition to summarizing the current evidence base, this document serves to highlight areas where additional research is needed to clarify the most effective treatment strategies.

Treatment gaps and future research directions identified include the following:

- Investigate multimodal interventions (eg, drug and therapy, brain stimulation, and therapy)
- Consider including multiple outcomes such as patient-centered, self-report outcomes in future intervention effectiveness trials (Patient Reported Outcomes Measurement Information System [PROMIS²⁹⁰])
- Consider computer-adapted assessments for personalized and tailored interventions
- Explore effective models of care that consider stroke as a chronic condition rather than simply a single acute event
- Capitalize on newer technologies such as virtual reality, body-worn sensors, and communication resources, including social media

- Develop interventions for individuals with severe stroke
- Develop better predictor models to identify responders and nonresponders to different therapies

As systems of care evolve in response to healthcare reform efforts, postacute care and rehabilitation are often considered a costly area of care to be trimmed, but without recognition of their clinical impact and their ability to reduce the risk of downstream medical morbidity caused by immobility, depression, loss of autonomy, and reduced functional independence. The provision of comprehensive rehabilitation programs with adequate resources, dose, and duration is an essential aspect of stroke care and should be a priority in these redesign efforts. We hope that these guidelines help inform these efforts.

Appendix 1. Structure and Organization of Stroke Rehabilitation Care in the United States

Setting	Admission	Median Length of Stay	Specialist Involvement
Acute inpatient facility (hospital)	Near onset	4 d for ischemic stroke 7 d for hemorrhagic stroke	Major: MD, RN More limited: OT, PT, SLT, SW
IRF	5–7 d	15 d (range, 8–30 d)	Major: MD, RN, OT, PT, SLT More limited: SW
SNF	5–7 d	Highly variable (maximum, 100 d)	Major: LPN/LVN, NA, OT, PT, SLT More limited: MD, RN
Long-term care (nursing home)	Highly variable	Prolonged and highly variable	Major: LPN/LVN, NA More limited: RN, OT, PT, SLT, MD
Long-term care hospital	Variable	25-d average (required)	Major: RN, MD More limited: OT, PT, SLT
HHCA	Variable (typically 5–30 d)	Maximum 60-d episode	Major: NA, RN More limited: OT, PT, SLT, MD
Outpatient office	Variable (typically 5–30 d)	Variable	Major: OT, PT, SLT, MD

HHCA indicates home healthcare agency; IRF, inpatient rehabilitation facility; LPN/LVN, licensed practical or vocational nurse; MD, medical doctor; NA, nurse assistant; OT, occupational therapist; PT, physical therapist; RN, registered nurse (preferably with training in rehabilitation); SLT, speech-language therapist; SNF, skilled nursing facility; and SW, social worker. Modified from Miller et al.¹¹ Copyright © 2010, American Heart Association, Inc.

Appendix 2. Recommended* Measures Table

Construct/Measure	Comments	Approximate Time to Administer, min	References for Further Information
Impairment			
Paresis/strength			
Motricity Index	Consists of strength testing via manual muscle testing at 3 key UE segments and 3 key LE segments; yields a score from 0–100 indicating strength of each limb	<5 for UEs; <5 for LEs	294–299
Muscle strength	Via manual muscle testing, graded on a 0–5 scale or handheld dynamometry	<5	
Grip, pinch dynamometry	Grip and pinch dynamometers are available in most rehabilitation clinics and hospitals; normative data are available for comparison	<5	
Tone			
Modified Ashworth scale	Quantifies spasticity on a scale measuring resistance to passive movement from 0–4, with higher numbers indicating greater severity; can assess at all joints or only a few	10	294, 298, 299

(Continued)

Appendix 2. Continued

Construct/Measure	Comments	Approximate Time to Administer, min	References for Further Information
Sensorimotor impairment measures			
Fugl-Meyer	Quantifies sensorimotor impairment of the UE (0–66 points) and LE (0–34 points) on separate subscales; items are rated on ability to move out of abnormal synergies	25	298–302
Chedoke McMaster Stroke Assessment, impairment inventory	Quantifies impairments in 6 dimensions of shoulder pain, postural control, arm, hand, leg, and foot, each on a 7-point scale, with higher scores equalling less impairment	45	
Activity			
UE function			
Action Research Arm Test	Criteria based with 19 items; scores are from 0–57, with normal=57; allows observation of multiple grasps, grips, and pinches	10	294, 298–300, 302–306
Box and Block Test	Score is the number of blocks moved in 1 min; higher scores equal better performance; normative data are available for comparison	<5	
Chedoke Arm and Hand Activity Index	Criterion based with functional items requiring bilateral UE movement; available in 7-, 8-, 9-, and 13-item versions	25	
Wolf Motor Function Test	Time- and criterion-based scores on 15 items; contains some isolated joint movements and some functional tasks	15	
Balance			
Berg Balance Scale	Criterion-based assessment of static and dynamic balance; widely used in multiple settings	15	307–311
Functional Reach Test	A single-item test that measures how far one can reach in standing; normative data are available for comparison	<5	
Mobility			
Walking speed†	Brief and widely used; categories based on speed are: <0.4 m/s=household ambulation 0.4–0.8 m/s=limited community ambulation >0.8 m/s=community ambulation; normative data available for comparison	<5	307, 308, 312–314
Timed Up and Go	Quantifies more than straight walking, including sit/stand and a turn; scored by time to complete; criterion values available for comparison	<5	
6-Min walk test	Quantifies walking endurance; normative and criterion values for community ambulation distances available	<10	
Functional ambulation category	Classification made after observation or self-report of walking ability; 6-point scale with higher equals better walking ability; this tool allows assessment of walking ability in people who are not independent ambulators	<5	
Observational gait analysis	Commonly used in many clinics to plan treatment programs; several standardized formats are available; appropriate to use in conjunction with one of the above more quantifiable measures	5	
Participation			
Self-reported impairments, limitations, and restrictions			
Stroke Impact Scale: Strength, Mobility, ADL, and Hand Function subscales	These 4 subscales measure different aspects of physical performance; people rate their perceived ability to do different items; each subscale ranges from 0–100, with higher scores indicating better abilities	5 per subscale	294, 304, 307, 315
Motor Activity Log	14 or 28 questions about how the affected UE is used in daily life; scores range from 0–5, with 5 equal to similar to before the stroke	20	
Activities-specific Balance Confidence Scale	16 questions in which people with stroke rate their balance confidence during routine activities; scores range from 0–100, with higher scores indicating more confidence	20	316–319

(Continued)

Appendix 2. Continued

Construct/Measure	Comments	Approximate Time to Administer, min	References for Further Information
Technology for monitoring activity and participation			
Accelerometers, step activity monitors, pedometers	Numerous commercially available options; issues to consider when purchasing: cost, expected wear and tear, accompanying software, ease of use, wearing comfort; pedometers are the most economic option but need to be checked for ability to register steps of individuals with slow walking speeds	<5 to don/doff; additional processing time	7, 294, 321–328, 350

ADL indicates activity of daily living; LE, lower extremity; and UE, upper extremity.

*Note that it is recommended that clinicians select a single measure for each construct; it is often unnecessary to use >1 measure.

†Generally tested on 5- or 10-m walkways.

Disclosures

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Catherine E. Lang	Washington University School of Medicine (St. Louis)	NIH (grant to test interventions for individuals with stroke)†; NIH (coinvestigator on grant investigating brain connectivity after stroke)*; Barnes Jewish Hospital Foundation*; NIH (coinvestigator on grant to investigate postacute rehabilitation for general medical population)*	None	None	None	None	Neuroolutions, Inc*; Rehabilitation Institute of Chicago's NIDRR National Center for Rehabilitation Robotics*; Centers of Excellence in Stroke Collaborative Research for Regeneration, Resilience, and Secondary Prevention*; American Heart/American Stroke Association*; Bugher Foundation*	Royalties for book, AOTA Press Inc*

(Continued)

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*Modest.

†Significant.

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*Modest.

References

- Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Judd SE, Kissela BM, Lackland DT, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Matchar DB, McGuire DK, Mohler ER 3rd, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Willey JZ, Woo D, Yeh RW, Turner MB; on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2015 update: a report from the American Heart Association [published corrections appear in *Circulation*. 2015;131:e535 and *Circulation*. 2016;133:e417]. *Circulation*. 2015;131:e29–e322. doi: 10.1161/CIR.000000000000152.
- Buntin MB, Colla CH, Deb P, Sood N, Escarce JJ. Medicare spending and outcomes after postacute care for stroke and hip fracture. *Med Care*. 2010;48:776–784. doi: 10.1097/MLR.0b013e3181e359df.
- World Health Organization. *ICF: International Classification of Functioning, Disability and Health*. Geneva, Switzerland: World Health Organization; 2011.
- Management of Stroke Rehabilitation Working Group. *Management of Stroke Rehabilitation*. Washington, DC: Veterans Affairs/Department of Defense; 2010.
- American Heart Association. Stroke statements & guidelines. http://professional.heart.org/professional/GuidelinesStatements/UCM_316885_Guidelines-Statements.jsp. Accessed March 5, 2016.
- Mayo NE, Fellows LK, Scott SC, Cameron J, Wood-Dauphinee S. A longitudinal view of apathy and its impact after stroke. *Stroke*. 2009;40:3299–3307. doi: 10.1161/STROKEAHA.109.554410.
- Duncan F, Kutlubaev MA, Dennis MS, Greig C, Mead GE. Fatigue after stroke: a systematic review of associations with impaired physical fitness. *Int J Stroke*. 2012;7:157–162. doi: 10.1111/j.1747-4949.2011.00741.x.
- Gebruers N, Vanroy C, Truijien S, Engelborghs S, De Deyn PP. Monitoring of physical activity after stroke: a systematic review of accelerometer-based measures. *Arch Phys Med Rehabil*. 2010;91:288–297. doi: 10.1016/j.apmr.2009.10.025.
- Lincoln NB, Brinkmann N, Cunningham S, Dejaeger E, De Weerd W, Jenni W, Mahdiz A, Putman K, Schupp W, Schuback B, De Wit L. Anxiety and depression after stroke: a 5 year follow-up. *Disabil Rehabil*. 2013;35:140–145. doi: 10.3109/09638288.2012.691939.
- Gadidi V, Katz-Leurer M, Carmeli E, Bornstein NM. Long-term outcome poststroke: predictors of activity limitation and participation restriction. *Arch Phys Med Rehabil*. 2011;92:1802–1808. doi: 10.1016/j.apmr.2011.06.014.
- National Quality Forum. *Measurement Framework: Evaluation Efficiency Across Patient-Focused Episodes of Care*. Washington, DC: National Quality Forum; 2009.
- Miller EL, Murray L, Richards L, Zorowitz RD, Bakas T, Clark P, Billinger SA; on behalf of the American Heart Association Council on Cardiovascular Nursing and the Stroke Council. Comprehensive overview of nursing and interdisciplinary rehabilitation care of the stroke patient: a scientific statement from the American Heart Association. *Stroke*. 2010;41:2402–2448. doi: 10.1161/STR.0b013e3181e7512b.
- Prvu Bettger JA, Kaltenbach L, Reeves MJ, Smith EE, Fonarow GC, Schwamm LH, Peterson ED. Assessing stroke patients for rehabilitation during the acute hospitalization: findings from the Get With The Guidelines-Stroke program. *Arch Phys Med Rehabil*. 2013;94:38–45. doi: 10.1016/j.apmr.2012.06.029.
- Buntin MB. Access to postacute rehabilitation. *Arch Phys Med Rehabil*. 2007;88:1488–1493. doi: 10.1016/j.apmr.2007.07.023.
- MedPAC. *Healthcare Spending and the Medicare Program: A Data Book*. Washington, DC: Medicare Payment Advisory Commission; 2008.
- Liu K BC, Wissoker D, Maxwell S, Haley J, Long S. Long-term care hospitals under Medicare: facility-level characteristics. *Health Care Financ Rev*. 2001;23:1–8.
- Early Supported Discharge Trialists. Services for reducing duration of hospital care for acute stroke patients. *Cochrane Database Syst Rev*. 2005:CD000443.
- Langhorne P, Taylor G, Murray G, Dennis M, Anderson C, Bautz-Holter E, Dey P, Indredavik B, Mayo N, Power M, Rodgers H, Ronning OM, Rudd A, Suwanwela N, Widen-Holmqvist L, Wolfe C. Early supported discharge services for stroke patients: a meta-analysis of individual patients' data. *Lancet*. 2005;365:501–506. doi: 10.1016/S0140-6736(05)17868-4.
- Langhorne P, Holmqvist LW; Early Supported Discharge Trialists. Early supported discharge after stroke [published correction appears in *J Rehabil Med*. 2007;39:269]. *J Rehabil Med*. 2007;39:103–108. doi: 10.2340/16501977-0042.
- Olson DM, Bettger JP, Alexander KP, Kendrick AS, Irvine JR, Wing L, Coeytaux RR, Dolor RJ, Duncan PW, Graffagnino C. Transition of care for acute stroke and myocardial infarction patients: from hospitalization to rehabilitation, recovery, and secondary prevention. *Evid Rep Technol Assess (Full Rep)*. 2011:1–197.
- Rousseaux M, Daveluy W, Kozlowski R. Value and efficacy of early supported discharge from stroke units. *Ann Phys Rehabil Med*. 2009;52:224–233.
- Brady BK, McGahan L, Skidmore B. Systematic review of economic evidence on stroke rehabilitation services. *Int J Technol Assess Health Care*. 2005;21:15–21.
- Fisher RJ, Gaynor C, Kerr M, Langhorne P, Anderson C, Bautz-Holter E, Indredavik B, Mayo NE, Power M, Rodgers H, Rønning OM, Widen Holmqvist L, Wolfe CD, Walker MF. A consensus on stroke: early supported discharge. *Stroke*. 2011;42:1392–1397. doi: 10.1161/STROKEAHA.110.606285.
- Buntin MB, Escarce JJ, Hoverman C, Paddock SM, Totten M, Wynn BO. *Effects of Payment Changes on Trends in Access to Post-Acute Care*. Baltimore, MD: Centers for Medicare & Medicaid Services, US Department of Health and Human Services; 2005. TR-259-CMS.
- Kramer A, Holthaus D, Goodrich G, Epstein A. *A Study of Stroke Post-Acute Care Costs and Outcomes: Final Report*. Washington, DC: US Department of Health and Human Services; 2006.
- Berg K, Intrator O. Postacute care following stroke or hip fracture: single services and combinations used by Medicare beneficiaries (1987–1992). *J Aging Health*. 1999;11:27–48.
- Buntin MB, Colla CH, Escarce JJ. Effects of payment changes on trends in post-acute care. *Health Serv Res*. 2009;44:1188–1210. doi: 10.1111/j.1475-6773.2009.00968.x.
- Segal M, Pedersen AL, Freeman K, Fast A. Medicare's new restrictions on rehabilitation admissions: impact on the elderly. *Am J Phys Med Rehabil*. 2008;87:872–882. doi: 10.1097/PHM.0b013e31818a67b3.
- Gage B, Morley M, Spain P, Ingber M. *Examining Post Acute Care Relationships in an Integrated Hospital System*. Waltham, PA: US Department of Health and Human Services; 2009.
- Kane RL, Lin WC, Blewett LA. Geographic variation in the use of post-acute care. *Health Serv Res*. 2002;37:667–682.
- Buntin MB, Garten AD, Paddock S, Saliba D, Totten M, Escarce JJ. How much is postacute care use affected by its availability? *Health Serv Res*. 2005;40:413–434. doi: 10.1111/j.1475-6773.2005.00365.x.
- Liu K, Wissoker D, Rimes C. Determinants and costs of Medicare post-acute care provided by SNFs and HHAs. *Inquiry*. 1998;35:49–61.
- Report to Congress: *Variations and Innovation in Medicare*. Washington, DC: Medicare Payment Advisory Commission (MedPAC); 2003.
- Report to Congress: *Medicare Payment Policy*. Washington, DC: Medicare Payment Advisory Commission (MedPAC); 2003.
- Blewett LA, Kane RL, Finch M. Hospital ownership of post-acute care: does it increase access to post-acute care services? *Inquiry*. 1995–1996;32:457–467.
- Bronskill SE, Normand SL, McNeil BJ. Post-acute service use following acute myocardial infarction in the elderly. *Health Care Financ Rev*. 2002;24:77–93.
- Ellis C, Breland HL, Egede LE. Racial/ethnic differences in utilization of post-stroke rehabilitation services: a systematic review. *Ethn Dis*. 2008;18:365–372.
- Finlayson M. Changes predicting long-term care use among the oldest-old. *Gerontologist*. 2002;42:443–453.
- Shatto A. Comparing Medicare beneficiaries, by type of post-acute care received: 1999. *Health Care Financ Rev*. 2002;24:137–142.
- Deutsch A, Granger CV, Heinemann AW, Fiedler RC, DeJong G, Kane RL, Ottenbacher KJ, Naughton JP, Trevisan M. Poststroke rehabilitation: outcomes and reimbursement of inpatient rehabilitation facilities and subacute rehabilitation programs. *Stroke*. 2006;37:1477–1482. doi: 10.1161/01.STR.0000221172.99375.5a.
- Kramer AM, Steiner JF, Schlenker RE, Eilertsen TB, Hrincevich CA, Tropea DA, Ahmad LA, Eckhoff DG. Outcomes and costs after hip fracture and stroke: a comparison of rehabilitation settings. *JAMA*. 1997;277:396–404.
- Kane RL, Chen Q, Finch M, Blewett L, Burns R, Moskowitz M. Functional outcomes of posthospital care for stroke and hip fracture patients under Medicare. *J Am Geriatr Soc*. 1998;46:1525–1533.

42. Keith RA, Wilson DB, Gutierrez P. Acute and subacute rehabilitation for stroke: a comparison. *Arch Phys Med Rehabil*. 1995;76:495–500.
43. Prvu Bettger J, Liang L, Xian Y, Peterson ED, Bushnell C, Duncan PW, Federspiel JJ, Stein J, Montalvo C, Lutz BJ, Hoenig H, Schwamm LH, Wu J, Stafford J, Thomas L. Inpatient rehabilitation facility care reduces the likelihood of death and re-hospitalization after stroke compared with skilled nursing facility care [abstract]. *Stroke*. 2015;46:A146.
44. Kane RL, Chen Q, Blewett LA, Sangl J. Do rehabilitative nursing homes improve the outcomes of care? *J Am Geriatr Soc*. 1996;44:545–554.
45. Wang H, Sandel ME, Terdiman J, Armstrong MA, Klatsky A, Camicia M, Sidney S. Postacute care and ischemic stroke mortality: findings from an integrated health care system in northern California. *PM R*. 2011;3:686–694. doi: 10.1016/j.pmrj.2011.04.028.
46. Petri H, Urquhart J. Channeling bias in the interpretation of drug effects. *Stat Med*. 1991;10:577–581.
47. Stroke Unit Trialists' Collaboration. Organised inpatient (stroke unit) care for stroke. *Cochrane Database Syst Rev*. 2007;CD000197.
48. Kalra L, Langhorne P. Facilitating recovery: evidence for organized stroke care. *J Rehabil Med*. 2007;39:97–102. doi: 10.2340/16501977-0043.
49. Maulden SA, Gassaway J, Horn SD, Smout RJ, DeJong G. Timing of initiation of rehabilitation after stroke. *Arch Phys Med Rehabil*. 2005;86(suppl 2):S34–S40. doi: 10.1016/j.apmr.2005.08.119.
50. Prvu Bettger JA, Stineman MG. Effectiveness of multidisciplinary rehabilitation services in postacute care: state-of-the-science: a review. *Arch Phys Med Rehabil*. 2007;88:1526–1534. doi: 10.1016/j.apmr.2007.06.768.
51. Duncan PW, Sullivan KJ, Behrman AL, Azen SP, Wu SS, Nadeau SE, Dobkin BH, Rose DK, Tilson JK, Cen S, Hayden SK; LEAPS Investigative Team. Body-weight-supported treadmill rehabilitation after stroke. *N Engl J Med*. 2011;364:2026–2036. doi: 10.1056/NEJMoa1010790.
52. Wolf SL, Winstein CJ, Miller JP, Taub E, Uswatte G, Morris D, Giuliani C, Light KE, Nichols-Larsen D; EXCITE Investigators. Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: the EXCITE randomized clinical trial. *JAMA*. 2006;296:2095–2104. doi: 10.1001/jama.296.17.2095.
53. West T, Langhorne P, Bernhardt J, Chan DKY, Guidetti D. How do comprehensive and acute stroke units differ? A critical review. *Int J Ther Rehabil*. 2013;20:41–53.
54. Hakkennes SJ, Brock K, Hill KD. Selection for inpatient rehabilitation after acute stroke: a systematic review of the literature. *Arch Phys Med Rehabil*. 2011;92:2057–2070. doi: 10.1016/j.apmr.2011.07.189.
55. Tistad M, Ytterberg C, Sjöstrand C, Holmqvist LW, von Koch L. Shorter length of stay in the stroke unit: comparison between the 1990s and 2000s. *Top Stroke Rehabil*. 2012;19:172–181. doi: 10.1310/tsr1902-172.
56. Rinere O'Brien S. Trends in inpatient rehabilitation stroke outcomes before and after advent of the prospective payment system: a systematic review. *J Neurol Phys Ther*. 2010;34:17–23. doi: 10.1097/NPT.0b013e3181cfd3ac.
57. Bernhardt J, Thuy MN, Collier JM, Legg LA. Very early versus delayed mobilisation after stroke. *Cochrane Database Syst Rev*. 2009;CD006187. doi: 10.1002/14651858.CD006187.pub2.
58. AVERT Trial Collaboration Group, Bernhardt J, Langhorne P, Lindley RJ, Thrift AG, Ellery F, Collier J, Churilov L, Moodie M, Dewey H, Donnan G. Efficacy and safety of very early mobilisation within 24 h of stroke onset (AVERT): a randomised controlled trial [published correction appears in *Lancet*. 2015;386:30]. *Lancet*. 2015;386:46–55.
59. Chan L, Sandel ME, Jette AM, Appelman J, Brandt DE, Cheng P, Teselle M, Delmonico R, Terdiman JF, Rasch EK. Does postacute care site matter? A longitudinal study assessing functional recovery after a stroke. *Arch Phys Med Rehabil*. 2013;94:622–629. doi: 10.1016/j.apmr.2012.09.033.
60. Wang H, Camicia M, Terdiman J, Mannava MK, Sidney S, Sandel ME. Daily treatment time and functional gains of stroke patients during inpatient rehabilitation. *PM R*. 2013;5:122–128. doi: 10.1016/j.pmrj.2012.08.013.
61. Zhuang LX, Xu SF, D'Adamo CR, Jia C, He J, Han DX, Lao LX. An effectiveness study comparing acupuncture, physiotherapy, and their combination in poststroke rehabilitation: a multicentered, randomized, controlled clinical trial. *Altern Ther Health Med*. 2012;18:8–14.
62. van Nes IJ, Latour H, Schils F, Meijer R, van Kuijk A, Geurts AC. Long-term effects of 6-week whole-body vibration on balance recovery and activities of daily living in the postacute phase of stroke: a randomized, controlled trial. *Stroke*. 2006;37:2331–2335. doi: 10.1161/01.STR.0000236494.62957.f3.
63. Pressure Ulcer Prevention and Treatment Protocol: Health Care Protocol. Bloomington, MN; Institute for Clinical Systems Improvement (ICS); 2012.
64. Deleted in proof.
65. Malhotra S, Pandyan AD, Rosewilliam S, Roffe C, Hermens H. Spasticity and contractures at the wrist after stroke: time course of development and their association with functional recovery of the upper limb. *Clin Rehabil*. 2011;25:184–191. doi: 10.1177/0269215510381620.
66. Sackley C, Brittle N, Patel S, Ellins J, Scott M, Wright C, Dewey ME. The prevalence of joint contractures, pressure sores, painful shoulder, other pain, falls, and depression in the year after a severely disabling stroke. *Stroke*. 2008;39:3329–3334. doi: 10.1161/STROKEAHA.108.518563.
67. Ada L, O'Dwyer N, O'Neill E. Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disabil Rehabil*. 2006;28:891–897. doi: 10.1080/09638280500535165.
68. Harvey L, de Jong I, Goehl G, Mardwedel S. Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. *Aust J Physiother*. 2006;52:251–258.
69. Lannin NA, Cusick A, McCluskey A, Herbert RD. Effects of splinting on wrist contracture after stroke: a randomized controlled trial. *Stroke*. 2007;38:111–116. doi: 10.1161/01.STR.0000251722.77088.12.
70. *National Clinical Guideline for Stroke*. 3rd ed. London, UK: Royal College of Physicians Intercollegiate Stroke Working Party; 2008.
71. Mayer NH, Harvey RL. Use of a resting hand orthosis for the hemiparetic hand after stroke. *PM R*. 2014;6:188–195. doi: 10.1016/j.pmrj.2014.01.012.
72. Hesse S, Mach H, Fröhlich S, Behrend S, Werner C, Melzer I. An early botulinum toxin A treatment in subacute stroke patients may prevent a disabling finger flexor stiffness six months later: a randomized controlled trial. *Clin Rehabil*. 2012;26:237–245. doi: 10.1177/0269215511421355.
73. Ada L, Goddard E, McCully J, Stavrinou T, Bampton J. Thirty minutes of positioning reduces the development of shoulder external rotation contracture after stroke: a randomized controlled trial. *Arch Phys Med Rehabil*. 2005;86:230–234. doi: 10.1016/j.apmr.2004.02.031.
74. de Jong LD, Nieuwboer A, Aufdemkampe G. Contracture preventive positioning of the hemiplegic arm in subacute stroke patients: a pilot randomized controlled trial [published correction appears in *Clin Rehabil*. 2010;24:767]. *Clin Rehabil*. 2006;20:656–667.
75. Basaran A, Emre U, Karadavut KI, Balbaloglu O, Bulmus N. Hand splinting for poststroke spasticity: a randomized controlled trial. *Top Stroke Rehabil*. 2012;19:329–337. doi: 10.1310/tsr1904-329.
76. Tyson SF, Kent RM. The effect of upper limb orthotics after stroke: a systematic review. *NeuroRehabilitation*. 2011;28:29–36. doi: 10.3233/NRE-2011-0629.
77. Namdari S, Horneff JG, Baldwin K, Keenan MA. Muscle releases to improve passive motion and relieve pain in patients with spastic hemiplegia and elbow flexion contractures. *J Shoulder Elbow Surg*. 2012;21:1357–1362. doi: 10.1016/j.jse.2011.09.029.
78. Robinson W, Smith R, Aung O, Ada L. No difference between wearing a night splint and standing on a tilt table in preventing ankle contracture early after stroke: a randomized trial. *Australian J Physiotherapy*. 2008;54:33–38.
79. Kelly J, Rudd A, Lewis R, Hunt BJ. Venous thromboembolism after acute stroke. *Stroke*. 2001;32:262–267.
80. Lansberg MG, O'Donnell MJ, Khatri P, Lang ES, Nguyen-Huynh MN, Schwartz NE, Sonnenberg FA, Schulman S, Vandvik PO, Spencer FA, Alonso-Coello P, Guyatt GH, Akl EA; American College of Chest Physicians. Antithrombotic and thrombolytic therapy for ischemic stroke: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(suppl):e601S–e636S. doi: 10.1378/chest.11-2302.
81. Kamphuisen PW, Agnelli G. What is the optimal pharmacological prophylaxis for the prevention of deep-vein thrombosis and pulmonary embolism in patients with acute ischemic stroke? *Thromb Res*. 2007;119:265–274. doi: 10.1016/j.thromres.2006.03.010.
82. Shorr AF, Jackson WL, Sherner JH, Moores LK. Differences between low-molecular-weight and unfractionated heparin for venous thromboembolism prevention following ischemic stroke: a metaanalysis. *Chest*. 2008;133:149–155. doi: 10.1378/chest.07-1826.

83. Sandercock PA, Counsell C, Tseng MC. Low-molecular-weight heparins or heparinoids versus standard unfractionated heparin for acute ischaemic stroke. *Cochrane Database Syst Rev*. 2008;CD000119.
84. McGarry LJ, Thompson D, Weinstein MC, Goldhaber SZ. Cost effectiveness of thromboprophylaxis with a low-molecular-weight heparin versus unfractionated heparin in acutely ill medical inpatients. *Am J Manag Care*. 2004;10:632–642.
85. Naccarato M, Chiodo Grandi F, Dennis M, Sandercock PA. Physical methods for preventing deep vein thrombosis in stroke. *Cochrane Database Syst Rev*. 2010;CD001922. doi: 10.1002/14651858.CD001922.pub3.
86. CLOTS Trials Collaboration, Dennis M, Sandercock PA, Reid J, Graham C, Murray G, Venables G, Rudd A, Bowler G. Effectiveness of thigh-length graduated compression stockings to reduce the risk of deep vein thrombosis after stroke (CLOTS trial 1): a multicentre, randomised controlled trial. *Lancet*. 2009;373:1958–1965.
87. Roderick P, Ferris G, Wilson K, Halls H, Jackson D, Collins R, Baigent C. Towards evidence-based guidelines for the prevention of venous thromboembolism: systematic reviews of mechanical methods, oral anti-coagulation, dextran and regional anaesthesia as thromboprophylaxis. *Health Technol Assess*. 2005;9:iii-iv, ix-x, 1–78.
88. Gould MK, Garcia DA, Wren SM, Karanicolas PJ, Arcelus JJ, Heit JA, Samama CM; American College of Chest Physicians. Prevention of VTE in nonorthopedic surgical patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines [published correction appears in *Chest*. 2012;141:1369]. *Chest*. 2012;141(suppl):e227S–e277S. doi: 10.1378/chest.11-2297.
89. Boer A, Voth E, Henze T, Prange HW. Early heparin therapy in patients with spontaneous intracerebral haemorrhage. *J Neurol Neurosurg Psychiatry*. 1991;54:466–467.
90. Thomas LH, Cross S, Barrett J, French B, Leathley M, Sutton CJ, Watkins C. Treatment of urinary incontinence after stroke in adults. *Cochrane Database Syst Rev*. 2008;CD004462. doi: 10.1002/14651858.CD004462.pub3.
91. Kovindha A, Wattanapan P, Dejpratham P, Permsirivanich W, Kuptniratsaikul V. Prevalence of incontinence in patients after stroke during rehabilitation: a multi-centre study. *J Rehabil Med*. 2009;41:489–491. doi: 10.2340/16501977-0354.
92. Pettersen R, Saxby BK, Wyller TB. Poststroke urinary incontinence: one-year outcome and relationships with measures of attentiveness. *J Am Geriatr Soc*. 2007;55:1571–1577. doi: 10.1111/j.1532-5415.2007.01396.x.
93. Pettersen R, Wyller TB. Prognostic significance of micturition disturbances after acute stroke. *J Am Geriatr Soc*. 2006;54:1878–1884. doi: 10.1111/j.1532-5415.2006.00984.x.
94. Centers for Disease Control and Prevention. Guidelines for prevention of catheter-associated urinary tract infections: CDC Guidelines. 2009. http://www.cdc.gov/hicpac/cauti/001_cauti.html. Accessed March 5, 2016.
95. Myint PK, Vowler SL, Redmayne O, Fulcher RA. Cognition, continence and transfer status at the time of discharge from an acute hospital setting and their associations with an unfavourable discharge outcome after stroke. *Gerontology*. 2008;54:202–209. doi: 10.1159/000126491.
96. National Institute of Health, National Institute of Diabetes and Digestive and Kidney Disease. Fecal incontinence. 2013. <http://www.digestive.niddk.nih.gov/ddiseases/pubs/fecalincontinence/>. Accessed July 13, 2013.
97. Lindgren I, Jönsson AC, Norrving B, Lindgren A. Shoulder pain after stroke: a prospective population-based study. *Stroke*. 2007;38:343–348. doi: 10.1161/01.STR.0000254598.16739.4e.
98. O'Donnell MJ, Diener HC, Sacco RL, Panju AA, Vinisko R, Yusuf S; PROfESS Investigators. Chronic pain syndromes after ischemic stroke: PROfESS trial. *Stroke*. 2013;44:1238–1243. doi: 10.1161/STROKEAHA.111.671008.
99. Chae J, Mascarenhas D, Yu DT, Kirsteins A, Elovic EP, Flanagan SR, Harvey RL, Zorowitz RD, Fang ZP. Poststroke shoulder pain: its relationship to motor impairment, activity limitation, and quality of life. *Arch Phys Med Rehabil*. 2007;88:298–301. doi: 10.1016/j.apmr.2006.12.007.
100. Paci M, Nannetti L, Taiti P, Baccini M, Rinaldi L. Shoulder subluxation after stroke: relationships with pain and motor recovery. *Physiother Res Int*. 2007;12:95–104.
101. Dromerick AW, Edwards DF, Kumar A. Hemiplegic shoulder pain syndrome: frequency and characteristics during inpatient stroke rehabilitation. *Arch Phys Med Rehabil*. 2008;89:1589–1593. doi: 10.1016/j.apmr.2007.10.051.
102. Lindgren I, Lexell J, Jönsson AC, Brogårdh C. Left-sided hemiparesis, pain frequency, and decreased passive shoulder range of abduction are predictors of long-lasting poststroke shoulder pain. *PM R*. 2012;4:561–568. doi: 10.1016/j.pmrj.2012.04.007.
103. Niessen MH, Veeger DH, Meskers CG, Koppe PA, Konijnenbelt MH, Janssen TW. Relationship among shoulder proprioception, kinematics, and pain after stroke. *Arch Phys Med Rehabil*. 2009;90:1557–1564. doi: 10.1016/j.apmr.2009.04.004.
104. Rajaratnam BS, Venketasubramanian N, Kumar PV, Goh JC, Chan YH. Predictability of simple clinical tests to identify shoulder pain after stroke. *Arch Phys Med Rehabil*. 2007;88:1016–1021. doi: 10.1016/j.apmr.2007.05.001.
105. Roosink M, Renzenbrink GJ, Buitenweg JR, Van Dongen RT, Geurts AC, IJzerman MJ. Persistent shoulder pain in the first 6 months after stroke: results of a prospective cohort study. *Arch Phys Med Rehabil*. 2011;92:1139–1145. doi: 10.1016/j.apmr.2011.02.016.
106. Huang YC, Liang PJ, Pong YP, Leong CP, Tseng CH. Physical findings and sonography of hemiplegic shoulder in patients after acute stroke during rehabilitation. *J Rehabil Med*. 2010;42:21–26. doi: 10.2340/16501977-0488.
107. Pong YP, Wang LY, Wang L, Leong CP, Huang YC, Chen YK. Sonography of the shoulder in hemiplegic patients undergoing rehabilitation after a recent stroke. *J Clin Ultrasound*. 2009;37:199–205. doi: 10.1002/jcu.20573.
108. Lee IS, Shin YB, Moon TY, Jeong YJ, Song JW, Kim DH. Sonography of patients with hemiplegic shoulder pain after stroke: correlation with motor recovery stage. *AJR Am J Roentgenol*. 2009;192:W40–W44. doi: 10.2214/AJR.07.3978.
109. Shah RR, Haghpanah S, Elovic EP, Flanagan SR, Behnegar A, Nguyen V, Page SJ, Fang ZP, Chae J. MRI findings in the painful poststroke shoulder. *Stroke*. 2008;39:1808–1813. doi: 10.1161/STROKEAHA.107.502187.
110. Yi Y, Lee KJ, Kim W, Oh BM, Chung SG. Biomechanical properties of the glenohumeral joint capsule in hemiplegic shoulder pain. *Clin Biomech (Bristol, Avon)*. 2013;28:873–878. doi: 10.1016/j.clinbiomech.2013.09.002.
111. Koog YH, Jin SS, Yoon K, Min BI. Interventions for hemiplegic shoulder pain: systematic review of randomised controlled trials. *Disabil Rehabil*. 2010;32:282–291. doi: 10.3109/09638280903127685.
112. Roosink M, Renzenbrink GJ, Geurts AC, IJzerman MJ. Towards a mechanism-based view on post-stroke shoulder pain: theoretical considerations and clinical implications. *NeuroRehabilitation*. 2012;30:153–165. doi: 10.3233/NRE-2012-0739.
113. Jones AK, Brown CA. Post-stroke shoulder pain: nociceptive or neuropathic? *Pain*. 2013;154:189. doi: 10.1016/j.pain.2012.11.009.
114. Roosink M, Renzenbrink GJ, Geurts AC, IJzerman MJ. RE: Underlying pathology and associated factors of hemiplegic shoulder pain. *Am J Phys Med Rehabil*. 2012;91:279–280. doi: 10.1097/PHM.0b013e3182412145.
115. Gamble GE, Barberan E, Laasch HU, Bowsher D, Tyrrell PJ, Jones AK. Poststroke shoulder pain: a prospective study of the association and risk factors in 152 patients from a consecutive cohort of 205 patients presenting with stroke. *Eur J Pain*. 2002;6:467–474.
116. Roosink M, Renzenbrink GJ, Buitenweg JR, van Dongen RT, Geurts AC, IJzerman MJ. Somatosensory symptoms and signs and conditioned pain modulation in chronic post-stroke shoulder pain. *J Pain*. 2011;12:476–485. doi: 10.1016/j.jpain.2010.10.009.
117. Zeilig G, Rivel M, Weingarden H, Gaidoukov E, Defrin R. Hemiplegic shoulder pain: evidence of a neuropathic origin. *Pain*. 2013;154:263–271. doi: 10.1016/j.pain.2012.10.026.
118. Soo Hoo J, Paul T, Chae J, Wilson RD. Central hypersensitivity in chronic hemiplegic shoulder pain. *Am J Phys Med Rehabil*. 2013;92:1–9; quiz 10–13.
119. Roosink M, Buitenweg JR, Renzenbrink GJ, Geurts AC, IJzerman MJ. Altered cortical somatosensory processing in chronic stroke: a relationship with post-stroke shoulder pain. *NeuroRehabilitation*. 2011;28:331–344. doi: 10.3233/NRE-2011-0661.
120. *Management of Patients With Stroke: Rehabilitation, Prevention and Management of Complications, and Discharge Planning: A National Guideline*. Edinburgh, Scotland: Scottish Intercollegiate Guidelines Network; 2010.
121. Griffin A, Bernhardt J. Strapping the hemiplegic shoulder prevents development of pain during rehabilitation: a randomized controlled trial. *Clin Rehabil*. 2006;20:287–295.
122. Hanger HC, Whitewood P, Brown G, Ball MC, Harper J, Cox R, Sainsbury R. A randomized controlled trial of strapping to prevent post-stroke shoulder pain. *Clin Rehabil*. 2000;14:370–380.

123. Pandian JD, Kaur P, Arora R, Vishwambaran DK, Toor G, Mathangi S, Vijaya P, Uppal A, Kaur T, Arima H. Shoulder taping reduces injury and pain in stroke patients: randomized controlled trial. *Neurology*. 2013;80:528–532. doi: 10.1212/WNL.0b013e318281550e.
124. Lee JA, Park SW, Hwang PW, Lim SM, Kook S, Choi KI, Kang KS. Acupuncture for shoulder pain after stroke: a systematic review. *J Altern Complement Med*. 2012;18:818–823. doi: 10.1089/acm.2011.0457.
125. Price CI, Pandyan AD. Electrical stimulation for preventing and treating post-stroke shoulder pain: a systematic Cochrane review. *Clin Rehabil*. 2001;15:5–19.
126. Church C, Price C, Pandyan AD, Huntley S, Curless R, Rodgers H. Randomized controlled trial to evaluate the effect of surface neuromuscular electrical stimulation to the shoulder after acute stroke. *Stroke*. 2006;37:2995–3001. doi: 10.1161/01.STR.0000248969.78880.82.
127. Malhotra S, Rosewilliam S, Hermens H, Roffe C, Jones P, Pandyan AD. A randomized controlled trial of surface neuromuscular electrical stimulation applied early after acute stroke: effects on wrist pain, spasticity and contractures. *Clin Rehabil*. 2013;27:579–590. doi: 10.1177/0269215512464502.
128. Yu DT, Chae J, Walker ME, Hart RL, Petroski GF. Comparing stimulation-induced pain during percutaneous (intramuscular) and transcutaneous neuromuscular electric stimulation for treating shoulder subluxation in hemiplegia. *Arch Phys Med Rehabil*. 2001;82:756–760. doi: 10.1053/apmr.2001.23310.
129. Renzenbrink GJ, IJzerman MJ. Percutaneous neuromuscular electrical stimulation (P-NMES) for treating shoulder pain in chronic hemiplegia: effects on shoulder pain and quality of life. *Clin Rehabil*. 2004;18:359–365.
130. Yu DT, Chae J, Walker ME, Kirsteins A, Elovic EP, Flanagan SR, Harvey RL, Zorowitz RD, Frost FS, Grill JH, Feldstein M, Fang ZP. Intramuscular neuromuscular electric stimulation for poststroke shoulder pain: a multicenter randomized clinical trial. *Arch Phys Med Rehabil*. 2004;85:695–704.
131. Chae J, Ng A, Yu DT, Kirsteins A, Elovic EP, Flanagan SR, Harvey RL, Zorowitz RD, Fang ZP. Intramuscular electrical stimulation for shoulder pain in hemiplegia: does time from stroke onset predict treatment success? *Neurorehabil Neural Repair*. 2007;21:561–567. doi: 10.1177/1545968306298412.
132. Chae J, Yu DT, Walker ME, Kirsteins A, Elovic EP, Flanagan SR, Harvey RL, Zorowitz RD, Frost FS, Grill JH, Fang ZP. Intramuscular electrical stimulation for hemiplegic shoulder pain: a 12-month follow-up of a multiple-center, randomized clinical trial. *Am J Phys Med Rehabil*. 2005;84:832–842.
133. Yu DT, Friedman AS, Rosenfeld EL. Electrical stimulation for treating chronic poststroke shoulder pain using a fully implanted microstimulator with internal battery. *Am J Phys Med Rehabil*. 2010;89:423–428. doi: 10.1097/PHM.0b013e3181d8d06f.
134. Chae J, Jedlicka L. Subacromial corticosteroid injection for poststroke shoulder pain: an exploratory prospective case series. *Arch Phys Med Rehabil*. 2009;90:501–506. doi: 10.1016/j.apmr.2008.10.011.
135. Dekker JH, Wagenaar RC, Lankhorst GJ, de Jong BA. The painful hemiplegic shoulder: effects of intra-articular triamcinolone acetonide. *Am J Phys Med Rehabil*. 1997;76:43–48.
136. Lakse E, Gunduz B, Erhan B, Celik EC. The effect of local injections in hemiplegic shoulder pain: a prospective, randomized, controlled study. *Am J Phys Med Rehabil*. 2009;88:805–811; quiz 812–804, 851.
137. Snels IA, Beckerman H, Twisk JW, Dekker JH, De Koning P, Koppe PA, Lankhorst GJ, Bouter LM. Effect of triamcinolone acetonide injections on hemiplegic shoulder pain: a randomized clinical trial. *Stroke*. 2000;31:2396–2401.
138. Rah UW, Yoon SH, Moon do J, Kwack KS, Hong JY, Lim YC, Joen B. Subacromial corticosteroid injection on poststroke hemiplegic shoulder pain: a randomized, triple-blind, placebo-controlled trial. *Arch Phys Med Rehabil*. 2012;93:949–956. doi: 10.1016/j.apmr.2012.02.002.
139. de Boer KS, Arwert HJ, de Groot JH, Meskers CG, Mishre AD, Arendzen JH. Shoulder pain and external rotation in spastic hemiplegia do not improve by injection of botulinum toxin A into the subscapular muscle. *J Neurol Neurosurg Psychiatry*. 2008;79:581–583. doi: 10.1136/jnnp.2007.128371.
140. Yelnik AP, Colle FM, Bonan IV, Vicaute E. Treatment of shoulder pain in spastic hemiplegia by reducing spasticity of the subscapular muscle: a randomised, double blind, placebo controlled study of botulinum toxin A. *J Neurol Neurosurg Psychiatry*. 2007;78:845–848. doi: 10.1136/jnnp.2006.103341.
141. Kong KH, Neo JJ, Chua KS. A randomized controlled study of botulinum toxin A in the treatment of hemiplegic shoulder pain associated with spasticity. *Clin Rehabil*. 2007;21:28–35. doi: 10.1177/0269215506072082.
142. Marciniak CM, Harvey RL, Gagnon CM, Duraski SA, Denby FA, McCarty S, Bravi LA, Polo KM, Fierstein KM. Does botulinum toxin type A decrease pain and lessen disability in hemiplegic survivors of stroke with shoulder pain and spasticity? A randomized, double-blind, placebo-controlled trial. *Am J Phys Med Rehabil*. 2012;91:1007–1019. doi: 10.1097/PHM.0b013e31826ecb02.
143. Marco E, Duarte E, Vila J, Tejero M, Guillen A, Boza R, Escalada F, Espadaler JM. Is botulinum toxin type A effective in the treatment of spastic shoulder pain in patients after stroke? A double-blind randomized clinical trial. *J Rehabil Med*. 2007;39:440–447. doi: 10.2340/16501977-0066.
144. Lim JY, Koh JH, Paik NJ. Intramuscular botulinum toxin-A reduces hemiplegic shoulder pain: a randomized, double-blind, comparative study versus intraarticular triamcinolone acetonide. *Stroke*. 2008;39:126–131. doi: 10.1161/STROKEAHA.107.484048.
145. Adey-Wakeling Z, Crotty M, Shanahan EM. Suprascapular nerve block for shoulder pain in the first year after stroke: a randomized controlled trial. *Stroke*. 2013;44:3136–3141. doi: 10.1161/STROKEAHA.113.002471.
146. Allen ZA, Shanahan EM, Crotty M. Does suprascapular nerve block reduce shoulder pain following stroke: a double-blind randomised controlled trial with masked outcome assessment. *BMC Neurol*. 2010;10:83. doi: 10.1186/1471-2377-10-83.
147. Yasar E, Vural D, Safaz I, Balaban B, Yilmaz B, Goktepe AS, Alaca R. Which treatment approach is better for hemiplegic shoulder pain in stroke patients: intra-articular steroid or suprascapular nerve block? A randomized controlled trial. *Clin Rehabil*. 2011;25:60–68. doi: 10.1177/0269215510380827.
148. Namdari S, Alos H, Baldwin K, Mehta S, Keenan MA. Shoulder tenotomies to improve passive motion and relieve pain in patients with spastic hemiplegia after upper motor neuron injury. *J Shoulder Elbow Surg*. 2011;20:802–806. doi: 10.1016/j.jse.2010.10.023.
149. Duncan PW, Zorowitz R, Bates B, Choi JY, Glasberg JJ, Graham GD, Katz RC, Lamberty K, Reker D. Management of adult stroke rehabilitation care: a clinical practice guideline. *Stroke*. 2005;36:e100–e143. doi: 10.1161/01.STR.0000180861.54180.FF.
150. Cassinari V, Pagni CA. *Central Pain: A Neurological Survey*. Cambridge, UK: Harvard University Press; 1969.
151. Klit H, Finnerup NB, Jensen TS. Central post-stroke pain: clinical characteristics, pathophysiology, and management. *Lancet Neurol*. 2009;8:857–868. doi: 10.1016/S1474-4422(09)70176-0.
152. Andersen G, Vestergaard K, Ingeman-Nielsen M, Jensen TS. Incidence of central post-stroke pain. *Pain*. 1995;61:187–193.
153. Bowsher D. Pain after thalamic stroke: right diencephalic predominance and clinical features in 180 patients. *Neurology*. 1998;51:927; author reply 927–928.
154. Klit H, Finnerup NB, Andersen G, Jensen TS. Central poststroke pain: a population-based study. *Pain*. 2011;152:818–824. doi: 10.1016/j.pain.2010.12.030.
155. Vestergaard K, Nielsen J, Andersen G, Ingeman-Nielsen M, Arendt-Nielsen L, Jensen TS. Sensory abnormalities in consecutive, unselected patients with central post-stroke pain. *Pain*. 1995;61:177–186.
156. Pellicane A, Harvey RL. Central poststroke pain. In: Stein J, Harvey RL, Winstein CJ, Zorowitz RD, Wittenberg GF, eds. *Stroke Recovery and Rehabilitation*. 2nd ed. New York, NY: Demos Medical; 2014:249–266.
157. Jones RC 3rd, Backonja MM. Review of neuropathic pain screening and assessment tools. *Curr Pain Headache Rep*. 2013;17:363. doi: 10.1007/s11916-013-0363-6.
158. Leijon G, Boivie J. Central post-stroke pain: a controlled trial of amitriptyline and carbamazepine. *Pain*. 1989;36:27–36.
159. Vestergaard K, Andersen G, Gotttrup H, Kristensen BT, Jensen TS. Lamotrigine for central poststroke pain: a randomized controlled trial. *Neurology*. 2001;56:184–190.
160. Kim JS, Bashford G, Murphy TK, Martin A, Dror V, Cheung R. Safety and efficacy of pregabalin in patients with central post-stroke pain. *Pain*. 2011;152:1018–1023. doi: 10.1016/j.pain.2010.12.023.
161. Vranken JH, Dijkgraaf MG, Kruis MR, van der Vegt MH, Hollmann MW, Heesen M. Pregabalin in patients with central neuropathic pain: a randomized, double-blind, placebo-controlled trial of a flexible-dose regimen. *Pain*. 2008;136:150–157. doi: 10.1016/j.pain.2007.06.033.
162. Holtom N. Gabapentin for treatment of thalamic pain syndrome. *Palliat Med*. 2000;14:167.

163. Serpell MG; Neuropathic Pain Study Group. Gabapentin in neuropathic pain syndromes: a randomised, double-blind, placebo-controlled trial. *Pain*. 2002;99:557–566.
164. Agnew DC, Goldberg VD. A brief trial of phenytoin therapy for thalamic pain. *Bull Los Angeles Neurol Soc*. 1976;41:9–12.
165. Leijon G, Boivie J. Central post-stroke pain: the effect of high and low frequency TENS. *Pain*. 1989;38:187–191.
166. Katayama Y, Fukaya C, Yamamoto T. Poststroke pain control by chronic motor cortex stimulation: neurological characteristics predicting a favorable response. *J Neurosurg*. 1998;89:585–591. doi: 10.3171/jns.1998.89.4.0585.
167. Nguyen JP, Lefaucher JP, Le Guerinel C, Eizenbaum JF, Nakano N, Carpentier A, Brugières P, Pollin B, Rostaing S, Keravel Y. Motor cortex stimulation in the treatment of central and neuropathic pain. *Arch Med Res*. 2000;31:263–265.
168. Tsubokawa T, Katayama Y, Yamamoto T, Hirayama T, Koyama S. Chronic motor cortex stimulation for the treatment of central pain. *Acta Neurochir Suppl (Wien)*. 1991;52:137–139.
169. Tsubokawa T, Katayama Y, Yamamoto T, Hirayama T, Koyama S. Chronic motor cortex stimulation in patients with thalamic pain. *J Neurosurg*. 1993;78:393–401. doi: 10.3171/jns.1993.78.3.0393.
170. Owen SL, Green AL, Stein JF, Aziz TZ. Deep brain stimulation for the alleviation of post-stroke neuropathic pain. *Pain*. 2006;120:202–206. doi: 10.1016/j.pain.2005.09.035.
171. Rasche D, Rinaldi PC, Young RF, Tronier VM. Deep brain stimulation for the treatment of various chronic pain syndromes. *Neurosurg Focus*. 2006;21:E8.
172. Gillespie LD, Robertson MC, Gillespie WJ, Sherrington C, Gates S, Clemson LM, Lamb SE. Interventions for preventing falls in older people living in the community. *Cochrane Database Syst Rev*. 2012;9:CD007146. doi: 10.1002/14651858.CD007146.pub3.
173. Ashburn A, Hyndman D, Pickering R, Yardley L, Harris S. Predicting people with stroke at risk of falls. *Age Ageing*. 2008;37:270–276. doi: 10.1093/ageing/afn066.
174. Batchelor F, Hill K, Mackintosh S, Said C. What works in falls prevention after stroke? A systematic review and meta-analysis. *Stroke*. 2010;41:1715–1722. doi: 10.1161/STROKEAHA.109.570390.
175. Tilson JK, Wu SS, Cen SY, Feng Q, Rose DR, Behrman AL, Azen SP, Duncan PW. Characterizing and identifying risk for falls in the LEAPS study: a randomized clinical trial of interventions to improve walking poststroke. *Stroke*. 2012;43:446–452. doi: 10.1161/STROKEAHA.111.636258.
176. Truelsen T, Piechowski-Józwiak B, Bonita R, Mathers C, Bogousslavsky J, Boysen G. Stroke incidence and prevalence in Europe: a review of available data. *Eur J Neurol*. 2006;13:581–598. doi: 10.1111/j.1468-1331.2006.01138.x.
177. Eng HS, Das S. Response to Commentary on Watt E, Murphy M, Pascoe E, Scanlon A & Gan S (2011) An evaluation of a structured learning programme as a component of the clinical practicum in final year bachelor of nursing programme: a pre-post test analysis. *Journal of Clinical Nursing* 20, 2286–2293. *J Clin Nurs*. 2012;21:297–298.
178. Andersson AG, Kamwendo K, Appelros P. Fear of falling in stroke patients: relationship with previous falls and functional characteristics. *Int J Rehabil Res*. 2008;31:261–264. doi: 10.1097/MRR.0b013e3282fba390.
179. Friedman SM, Munoz B, West SK, Rubin GS, Fried LP. Falls and fear of falling: which comes first? A longitudinal prediction model suggests strategies for primary and secondary prevention. *J Am Geriatr Soc*. 2002;50:1329–1335.
180. Tinetti ME, Kumar C. The patient who falls: “It’s always a trade-off.” *JAMA*. 2010;303:258–266. doi: 10.1001/jama.2009.2024.
181. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. *N Engl J Med*. 1988;319:1701–1707. doi: 10.1056/NEJM198812293192604.
182. Weerdesteyn V, de Niet M, van Duijnhoven HJ, Geurts AC. Falls in individuals with stroke. *J Rehabil Res Dev*. 2008;45:1195–1213.
183. Hempel S, Newberry S, Wang Z, Booth M, Shanman R, Johnsen B, Shier V, Saliba D, Spector WD, Ganz DA. Hospital fall prevention: a systematic review of implementation, components, adherence, and effectiveness. *J Am Geriatr Soc*. 2013;61:483–494. doi: 10.1111/jgs.12169.
184. Morse JM, Morse RM, Tylko SJ. Development of a scale to identify the fall-prone patient. *Can J Aging*. 1989;8:366–377.
185. Maeda N, Kato J, Shimada T. Predicting the probability for fall incidence in stroke patients using the Berg Balance Scale. *J Int Med Res*. 2009;37:697–704.
186. Batchelor FA, Hill KD, Mackintosh SF, Said CM, Whitehead CH. Effects of a multifactorial falls prevention program for people with stroke returning home after rehabilitation: a randomized controlled trial. *Arch Phys Med Rehabil*. 2012;93:1648–1655. doi: 10.1016/j.apmr.2012.03.031.
187. Taylor-Piliae RE, Hoke TM, Hepworth JT, Latt LD, Najafi B, Coull BM. Effect of Tai Chi on physical function, fall rates and quality of life among older stroke survivors. *Arch Phys Med Rehabil*. 2014;95:816–824. doi: 10.1016/j.apmr.2014.01.001.
188. Stuart M, Benvenuti F, Macko R, Taviani A, Segenni L, Mayer F, Sorkin JD, Stanhope SJ, Macellari V, Weinrich M. Community-based adaptive physical activity program for chronic stroke: feasibility, safety, and efficacy of the Empoli model. *Neurorehabil Neural Repair*. 2009;23:726–734. doi: 10.1177/154596830932734.
189. Bladin CF, Alexandrov AV, Bellavance A, Bornstein N, Chambers B, Coté R, Lebrun L, Pirisi A, Norris JW. Seizures after stroke: a prospective multicenter study. *Arch Neurol*. 2000;57:1617–1622.
190. Richardson EP Jr, Dodge PR. Epilepsy in cerebral vascular disease; a study of the incidence and nature of seizures in 104 consecutive autopsy-proven cases of cerebral infarction and hemorrhage. *Epilepsia*. 1954;3:49–74.
191. Awada A, Omojola MF, Obeid T. Late epileptic seizures after cerebral infarction. *Acta Neurol Scand*. 1999;99:265–268.
192. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, Grubb RL, Higashida RT, Jauch EC, Kidwell C, Lyden PD, Morgenstern LB, Qureshi AI, Rosenwasser RH, Scott PA, Wijdicks EF. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups [published corrections appear in *Stroke*. 2007;38:e38 and *Stroke*. 2007;38:e96]. *Stroke*. 2007;38:1655–1711. doi: 10.1161/STROKEAHA.107.181486.
193. Rumbach L, Sablot D, Berger E, Tatu L, Vuillier F, Moulin T. Status epilepticus in stroke: report on a hospital-based stroke cohort. *Neurology*. 2000;54:350–354.
194. McLean DE. Medical complications experienced by a cohort of stroke survivors during inpatient, tertiary-level stroke rehabilitation. *Arch Phys Med Rehabil*. 2004;85:466–469.
195. Cordonnier C, Hénon H, Derambure P, Pasquier F, Leys D. Influence of pre-existing dementia on the risk of post-stroke epileptic seizures. *J Neurol Neurosurg Psychiatry*. 2005;76:1649–1653. doi: 10.1136/jnnp.2005.064535.
196. Fish DR, Miller DH, Roberts RC, Blackie JD, Gilliatt RW. The natural history of late-onset epilepsy secondary to vascular disease. *Acta Neurol Scand*. 1989;80:524–526.
197. Balami JS, Buchan AM. Complications of intracerebral haemorrhage. *Lancet Neurol*. 2012;11:101–118. doi: 10.1016/S1474-4422(11)70264-2.
198. Connolly ES Jr, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, Hoh BL, Kirkness CJ, Naidech AM, Ogilvy CS, Patel AB, Thompson BG, Vespa P; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; Council on Cardiovascular Surgery and Anesthesia; Council on Clinical Cardiology. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2012;43:1711–1737. doi: 10.1161/STR.0b013e3282587839.
199. Choi KS, Chun HJ, Yi HJ, Ko Y, Kim YS, Kim JM. Seizures and epilepsy following aneurysmal subarachnoid hemorrhage: incidence and risk factors. *J Korean Neurosurg Soc*. 2009;46:93–98. doi: 10.3340/jkns.2009.46.2.93.
200. Naidech AM, Garg RK, Liebling S, Levasseur K, Macken MP, Schuele SU, Batjer HH. Anticonvulsant use and outcomes after intracerebral hemorrhage. *Stroke*. 2009;40:3810–3815. doi: 10.1161/STROKEAHA.109.559948.
201. Messé SR, Sansing LH, Cucchiara BL, Herman ST, Lyden PD, Kasner SE; CHANT Investigators. Prophylactic antiepileptic drug use is associated with poor outcome following ICH. *Neurocrit Care*. 2009;11:38–44. doi: 10.1007/s12028-009-9207-y.
202. Naidech AM, Kreiter KT, Janjua N, Ostapovich N, Parra A, Commichau C, Connolly ES, Mayer SA, Fitzsimmons BF. Phenytoin exposure is associated with functional and cognitive disability after subarachnoid hemorrhage. *Stroke*. 2005;36:583–587. doi: 10.1161/01.STR.0000141936.36596.1e.

203. Goldstein LB. Common drugs may influence motor recovery after stroke: the Sygen In Acute Stroke Study Investigators. *Neurology*. 1995;45:865–871.
204. Lazar RM, Fitzsimmons BF, Marshall RS, Mohr JP, Berman MF. Midazolam challenge reinduces neurological deficits after transient ischemic attack. *Stroke*. 2003;34:794–796. doi: 10.1161/01.STR.0000056540.04159.F3.
205. 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.
206. Kernan WN, Ovbiagele B, Black HR, Bravata DM, Chimowitz MI, Ezekowitz MD, Fang MC, Fisher M, Furie KL, Heck DV, Johnston SC, Kasner SE, Kittner SJ, Mitchell PH, Rich MW, Richardson D, Schwamm LH, Wilson JA; on behalf of the American Heart Association Stroke Council, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Peripheral Vascular Disease. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association [published correction appears in *Stroke*. 2015;46:e54]. *Stroke*. 2014;45:2160–2236. doi: 10.1161/STR.0000000000000024.
207. Wulsin L, Alwell K, Moomaw CJ, Lindsell CJ, Kleindorfer DO, Woo D, Flaherty ML, Khatri P, Adeoye O, Ferioli S, Broderick JP, Kissela BM. Comparison of two depression measures for predicting stroke outcomes. *J Psychosom Res*. 2012;72:175–179. doi: 10.1016/j.jpsychores.2011.11.015.
208. Willey JZ, Disla N, Moon YP, Paik MC, Sacco RL, Boden-Albala B, Elkind MS, Wright CB. Early depressed mood after stroke predicts long-term disability: the Northern Manhattan Stroke Study (NOMASS). *Stroke*. 2010;41:1896–1900. doi: 10.1161/STROKEAHA.110.583997.
209. Chen Y, Lu J, Wong KS, Mok VC, Ungvari GS, Tang WK. Health-related quality of life in the family caregivers of stroke survivors. *Int J Rehabil Res*. 2010;33:232–237. doi: 10.1097/MRR.0b013e328338b04b.
210. Hackett ML, Anderson CS. Predictors of depression after stroke: a systematic review of observational studies. *Stroke*. 2005;36:2296–2301. doi: 10.1161/01.STR.0000183622.75135.a4.
211. Kim JH, Park EY. The factor structure of the Center for Epidemiologic Studies Depression Scale in stroke patients. *Top Stroke Rehabil*. 2012;19:54–62. doi: 10.1310/tsr1901-54.
212. Lam SC, Lee LY, To KW. Depressive symptoms among community-dwelling, post-stroke elders in Hong Kong. *Int Nurs Rev*. 2010;57:269–273. doi: 10.1111/j.1466-7657.2009.00789.x.
213. Paolucci S. Epidemiology and treatment of post-stroke depression. *Neuropsychiatr Dis Treat*. 2008;4:145–154.
214. Vickery CD, Evans CC, Sepehri A, Jabeen LN, Gayden M. Self-esteem stability and depressive symptoms in acute stroke rehabilitation: methodological and conceptual expansion. *Rehabil Psychol*. 2009;54:332–342. doi: 10.1037/a0016434.
215. Hackett ML, Pickles K. Part I: frequency of depression after stroke: an updated systematic review and meta-analysis of observational studies. *Int J Stroke*. 2014;9:1017–1025. doi: 10.1111/ijss.12357.
216. Paolucci S, Gandolfo C, Provinciali L, Torta R, Toso V; DESTRO Study Group. The Italian multicenter observational study on post-stroke depression (DESTRO). *J Neurol*. 2006;253:556–562. doi: 10.1007/s00415-006-0058-6.
217. Hackett ML, Anderson CS, House AO. Management of depression after stroke: a systematic review of pharmacological therapies. *Stroke*. 2005;36:1098–1103. doi: 10.1161/01.STR.0000162391.27991.9d.
218. Ried LD, Jia H, Cameon R, Feng H, Wang X, Tueth M. Does prestroke depression impact poststroke depression and treatment? *Am J Geriatr Psychiatry*. 2010;18:624–633. doi: 10.1097/JGP.0b013e3181ca822b.
219. Tenev VT, Robinson RG, Jorge RE. Is family history of depression a risk factor for poststroke depression? Meta-analysis. *Am J Geriatr Psychiatry*. 2009;17:276–280. doi: 10.1097/JGP.0b013e3181953b6e.
220. Snaaphan L, van der Werf S, Kanselaar K, de Leeuw FE. Post-stroke depressive symptoms are associated with post-stroke characteristics. *Cerebrovasc Dis*. 2009;28:551–557. doi: 10.1159/000247598.
221. Holloway RG, Arnold RM, Creutzfeldt CJ, Lewis EF, Lutz BJ, McCann RM, Rabinstein AA, Saposnik G, Sheth KN, Zahuranec DB, Zipfel GJ, Zorowitz RD; on behalf of the American Heart Association Stroke Council, Council on Cardiovascular and Stroke Nursing, and Council on Clinical Cardiology. Palliative and end-of-life care in stroke: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2014;45:1887–1916. doi: 10.1161/STR.0000000000000015.
222. Schmid AA, Kroenke K, Hendrie HC, Bakas T, Sutherland JM, Williams LS. Poststroke depression and treatment effects on functional outcomes. *Neurology*. 2011;76:1000–1005. doi: 10.1212/WNL.0b013e318210435e.
223. Chollet F, Acket B, Raposo N, Albucher JF, Loubinoux I, Pariente J. Use of antidepressant medications to improve outcomes after stroke. *Curr Neurol Neurosci Rep*. 2013;13:318. doi: 10.1007/s11910-012-0318-z.
224. Vuletić V, Sapina L, Lozert M, Lezačić Z, Morović S. Anxiety and depressive symptoms in acute ischemic stroke. *Acta Clin Croat*. 2012;51:243–246.
225. Shimoda K, Robinson RG. Effects of anxiety disorder on impairment and recovery from stroke. *J Neuropsychiatry Clin Neurosci*. 1998;10:34–40. doi: 10.1176/jnp.10.1.34.
226. Campbell Burton AC, Holmes J, Murray J, Gillespie D, Lightbody EC, Watkins CL, Knapp P. Interventions for treating anxiety after stroke. *Cochrane Database Syst Rev*. 2011;CD008860. doi: 10.1002/14651858.CD008860.pub2.
227. Hackett ML, Anderson CS, House A, Xia J. Interventions for treating depression after stroke. *Cochrane Database Syst Rev*. 2008;CD003437. doi: 10.1002/14651858.CD003437.pub3.
228. de Man-van Ginkel JM, Gooskens F, Schuurmans MJ, Lindeman E, Hafsteinsdóttir TB; Rehabilitation Guideline Stroke Working Group. A systematic review of therapeutic interventions for poststroke depression and the role of nurses. *J Clin Nurs*. 2010;19:3274–3290. doi: 10.1111/j.1365-2702.2010.03402.x.
229. Gainotti G, Antonucci G, Marra C, Paolucci S. Relation between depression after stroke, antidepressant therapy, and functional recovery. *J Neurol Neurosurg Psychiatry*. 2001;71:258–261.
230. Chemerinski E, Robinson RG, Kosier JT. Improved recovery in activities of daily living associated with remission of poststroke depression. *Stroke*. 2001;32:113–117.
231. Sigwalt AR, Budde H, Helmich I, Glaser V, Ghisoni K, Lanza S, Cadore EL, Lhullier FL, de Bem AF, Hohl A, de Matos FJ, de Oliveira PA, Prediger RD, Guglielmo LG, Latini A. Molecular aspects involved in swimming exercise training reducing anhedonia in a rat model of depression. *Neuroscience*. 2011;192:661–674. doi: 10.1016/j.neuroscience.2011.05.075.
232. Kiecolt-Glaser JK, Glaser R. Depression and immune function: central pathways to morbidity and mortality. *J Psychosom Res*. 2002;53:873–876.
233. Woods JA, Lowder TW, Keylock KT. Can exercise training improve immune function in the aged? *Ann NY Acad Sci*. 2002;959:117–127.
234. Mead GE, Morley W, Campbell P, Greig CA, McMurdo M, Lawlor DA. Exercise for depression. *Cochrane Database Syst Rev*. 2009;CD004366. doi: 10.1002/14651858.CD004366.pub4.
235. Sjösten N, Kivela SL. The effects of physical exercise on depressive symptoms among the aged: a systematic review. *Int J Geriatr Psychiatry*. 2006;21:410–418. doi: 10.1002/gps.1494.
236. Eng JJ, Reime B. Exercise for depressive symptoms in stroke patients: a systematic review and meta-analysis. *Clin Rehabil*. 2014;28:731–739. doi: 10.1177/0269215514523631.
237. Saunders DH, Sanderson M, Brazzelli M, Greig CA, Mead GE. Physical fitness training for stroke patients. *Cochrane Database Syst Rev*. 2013;10:CD003316. doi: 10.1002/14651858.CD003316.pub5.
238. Bergersen H, Frøslie KF, Stibrant Sunnerhagen K, Schanke AK. Anxiety, depression, and psychological well-being 2 to 5 years post-stroke. *J Stroke Cerebrovasc Dis*. 2010;19:364–369. doi: 10.1016/j.jstrokecerebrovasdis.2009.06.005.
239. Karaiskos D, Tzavellas E, Spengos K, Vassilopoulos S, Paparrigopoulos T. Duloxetine versus citalopram and sertraline in the treatment of post-stroke depression, anxiety, and fatigue. *J Neuropsychiatry Clin Neurosci*. 2012;24:349–353. doi: 10.1176/appi.neuropsych.11110325.
240. Bhogal SK, Teasell R, Foley N, Speechley M. Heterocyclics and selective serotonin reuptake inhibitors in the treatment and prevention of poststroke depression. *J Am Geriatr Soc*. 2005;53:1051–1057. doi: 10.1111/j.1532-5415.2005.53310.x.
241. Ried LD, Jia H, Feng H, Cameon R, Wang X, Tueth M, Wu SS. Selective serotonin reuptake inhibitor treatment and depression are associated with poststroke mortality. *Ann Pharmacother*. 2011;45:888–897. doi: 10.1345/aph.1P478.
242. Beaupre GS, Lew HL. Bone-density changes after stroke. *Am J Phys Med Rehabil*. 2006;85:464–472. doi: 10.1097/01.phm.0000214275.69286.7a.
243. Celik B, Ones K, Ince N. Body composition after stroke. *Int J Rehabil Res*. 2008;31:93–96. doi: 10.1097/MRR.0b013e3282f7521a.
244. Pluskiewicz W. Skeletal consequences in patients after stroke. *Endokrynol Pol*. 2011;62:48–50.

245. Eng JJ, Pang MY, Ashe MC. Balance, falls, and bone health: role of exercise in reducing fracture risk after stroke. *J Rehabil Res Dev*. 2008;45:297–313.
246. Jørgensen L, Jacobsen BK, Wilsgaard T, Magnus JH. Walking after stroke: does it matter? Changes in bone mineral density within the first 12 months after stroke: a longitudinal study. *Osteoporos Int*. 2000;11:381–387. doi: 10.1007/s001980070103.
247. Pang MY, Eng JJ, McKay HA, Dawson AS. Reduced hip bone mineral density is related to physical fitness and leg lean mass in ambulatory individuals with chronic stroke. *Osteoporos Int*. 2005;16:1769–1779. doi: 10.1007/s00198-005-1925-1.
248. Pang MY, Eng JJ. Muscle strength is a determinant of bone mineral content in the hemiparetic upper extremity: implications for stroke rehabilitation. *Bone*. 2005;37:103–111. doi: 10.1016/j.bone.2005.03.009.
249. U.S. Preventive Services Task Force. Screening for osteoporosis: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2011;154:356–364. doi: 10.7326/0003-4819-154-5-201103010-00307.
250. Marzolini S, McIlroy W, Tang A, Corbett D, Craven BC, Oh PI, Brooks D. Predictors of low bone mineral density of the stroke-affected hip among ambulatory individuals with chronic stroke. *Osteoporos Int*. 2014;25:2631–2638. doi: 10.1007/s00198-014-2793-3.
251. Lin HL, Lin HC, Tseng YF, Liao HH, Worly JA, Pan CY, Hsu CY. Hip fracture after first-ever stroke: a population-based study. *Acta Neurol Scand*. 2015;131:158–163. doi: 10.1111/ane.12301.
252. Drake MT, Murad MH, Mauck KF, Lane MA, Undavalli C, Elraiyah T, Stuart LM, Prasad C, Shahrour A, Mullan RJ, Hazem A, Erwin PJ, Montori VM. Clinical review: risk factors for low bone mass-related fractures in men: a systematic review and meta-analysis. *J Clin Endocrinol Metab*. 2012;97:1861–1870. doi: 10.1210/jc.2011-3058.
253. Borschmann K. Exercise protects bone after stroke, or does it? A narrative review of the evidence. *Stroke Res Treat*. 2012;2012:103697. doi: 10.1155/2012/103697.
254. Borschmann K, Pang MY, Bernhardt J, Iuliano-Burns S. Stepping towards prevention of bone loss after stroke: a systematic review of the skeletal effects of physical activity after stroke. *Int J Stroke*. 2012;7:330–335. doi: 10.1111/j.1747-4949.2011.00645.x.
255. Ryan AS, Ivey FM, Prior S, Li G, Hafer-Macko C. Skeletal muscle hypertrophy and muscle myostatin reduction after resistive training in stroke survivors. *Stroke*. 2011;42:416–420. doi: 10.1161/STROKEAHA.110.602441.
256. Pang MY, Ashe MC, Eng JJ, McKay HA, Dawson AS. A 19-week exercise program for people with chronic stroke enhances bone geometry at the tibia: a peripheral quantitative computed tomography study. *Osteoporos Int*. 2006;17:1615–1625. doi: 10.1007/s00198-006-0168-0.
257. Pang MY, Eng JJ, Dawson AS, McKay HA, Harris JE. A community-based fitness and mobility exercise program for older adults with chronic stroke: a randomized, controlled trial. *J Am Geriatr Soc*. 2005;53:1667–1674. doi: 10.1111/j.1532-5415.2005.53521.x.
258. Gresham G, Duncan P, Stason W, Adams H, Adelman A, Alexander D, Bishop D, Diller L, Donaldson N, Granger C, Holland A, Kelly-Hayes M, McDowell F, Myers L, Phipps M, Roth E, Siebens H, Tarvin G, Trombly C. *Post-Stroke Rehabilitation*. Rockville, MD: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research; 1995.
259. Rathore SS, Hinn AR, Cooper LS, Tyroler HA, Rosamond WD. Characterization of incident stroke signs and symptoms: findings from the atherosclerosis risk in communities study. *Stroke*. 2002;33:2718–2721.
260. Langhorne P, Bernhardt J, Kwakkel G. Stroke rehabilitation. *Lancet*. 2011;377:1693–1702. doi: 10.1016/S0140-6736(11)60325-5.
261. Teasell R, Foley N, Salter K, Bhogal S, Jutai J, Speechley M. Evidence-based review of stroke rehabilitation: executive summary, 12th edition. *Top Stroke Rehabil*. 2009;16:463–488. doi: 10.1310/tsr1606-463.
262. Quinn TJ, Dawson J, Walters MR, Lees KR. Functional outcome measures in contemporary stroke trials. *Int J Stroke*. 2009;4:200–205. doi: 10.1111/j.1747-4949.2009.00271.x.
263. Adams HP Jr, Davis PH, Leira EC, Chang KC, Bendixen BH, Clarke WR, Woolson RF, Hansen MD. Baseline NIH Stroke Scale score strongly predicts outcome after stroke: a report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology*. 1999;53:126–131.
264. Volpe BT, Huerta PT, Zipse JL, Rykman A, Edwards D, Dipietro L, Hogan N, Krebs HI. Robotic devices as therapeutic and diagnostic tools for stroke recovery. *Arch Neurol*. 2009;66:1086–1090. doi: 10.1001/archneurol.2009.182.
265. Dukelow SP, Herter TM, Moore KD, Demers MJ, Glasgow JI, Bagg SD, Norman KE, Scott SH. Quantitative assessment of limb position sense following stroke. *Neurorehabil Neural Repair*. 2010;24:178–187. doi: 10.1177/1545968309345267.
266. Brennan D, Tindall L, Theodoros D, Brown J, Campbell M, Christiana D, Smith D, Cason J, Lee A. A blueprint for telerehabilitation guidelines. *Int J Telerehabil*. 2010;2:31–34. doi: 10.5195/ijt.2010.6063.
267. World Health Organization. *ICF: International Classification of Functioning, Disability and Health*. Geneva, Switzerland: World Health Organization; 2008.
268. Goljar N, Burger H, Vidmar G, Leonardi M, Marinček C. Measuring patterns of disability using the International Classification of Functioning, Disability and Health in the post-acute stroke rehabilitation setting. *J Rehabil Med*. 2011;43:590–601. doi: 10.2340/16501977-0832.
269. Schwamm LH, Pancioli A, Acker JE 3rd, Goldstein LB, Zorowitz RD, Shephard TJ, Moyer P, Gorman M, Johnston SC, Duncan PW, Gorelick P, Frank J, Stranne SK, Smith R, Federspiel W, Horton KB, Magnis E, Adams RJ; American Stroke Association's Task Force on the Development of Stroke Systems. Recommendations for the establishment of stroke systems of care: recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems. *Stroke*. 2005;36:690–703. doi: 10.1161/01.STR.0000158165.42884.4F.
270. Teasell RW, Foley NC, Bhogal SK, Speechley MR. An evidence-based review of stroke rehabilitation. *Top Stroke Rehabil*. 2003;10:29–58. doi: 10.1310/8YNA-1YHK-YMHB-XTE1.
271. Stein J, Bettger JP, Sicklick A, Hedeman R, Magdon-Ismael Z, Schwamm LH. Use of a standardized assessment to predict rehabilitation care after acute stroke. *Arch Phys Med Rehabil*. 2015;96:210–217. doi: 10.1016/j.apmr.2014.07.403.
272. Gillen R, Tennen H, McKee T. Unilateral spatial neglect: relation to rehabilitation outcomes in patients with right hemisphere stroke. *Arch Phys Med Rehabil*. 2005;86:763–767. doi: 10.1016/j.apmr.2004.10.029.
273. Di Monaco M, Trucco M, Di Monaco R, Tappero R, Cavanna A. The relationship between initial trunk control or postural balance and inpatient rehabilitation outcome after stroke: a prospective comparative study. *Clin Rehabil*. 2010;24:543–554. doi: 10.1177/0269215509353265.
274. O'Dell MW, Au J, Schwabe E, Batistick H, Christos PJ. A comparison of two balance measures to predict discharge performance from inpatient stroke rehabilitation. *PM R*. 2013;5:392–399. doi: 10.1016/j.pmrj.2013.02.004.
275. Wee JY, Hopman WM. Stroke impairment predictors of discharge function, length of stay, and discharge destination in stroke rehabilitation. *Am J Phys Med Rehabil*. 2005;84:604–612.
276. Lang CE, Bland MD, Connor LT, Fucetola R, Whitson M, Edmiston J, Karr C, Sturmoski A, Baty J, Corbetta M. The brain recovery core: building a system of organized stroke rehabilitation and outcomes assessment across the continuum of care. *J Neurol Phys Ther*. 2011;35:194–201. doi: 10.1097/NPT.0b013e318235dc07.
277. Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. *Stroke*. 1995;26:982–989.
278. O'Brien SR, Xue Y. Predicting goal achievement during stroke rehabilitation for Medicare beneficiaries. *Disabil Rehabil*. 2014;36:1273–1278. doi: 10.3109/09638288.2013.845253.
279. Chumney D, Nollinger K, Shesko K, Skop K, Spencer M, Newton RA. Ability of Functional Independence Measure to accurately predict functional outcome of stroke-specific population: systematic review. *J Rehabil Res Dev*. 2010;47:17–29.
280. Nakao S, Takata S, Uemura H, Kashiwara M, Osawa T, Komatsu K, Masuda Y, Okahisa T, Nishikawa K, Kondo S, Yamada M, Takahara R, Ogata Y, Nakamura Y, Nagahiro S, Kaji R, Yasui N. Relationship between Barthel Index scores during the acute phase of rehabilitation and subsequent ADL in stroke patients. *J Med Invest*. 2010;57:81–88.
281. Ng YS, Jung H, Tay SS, Pak CW, Chiong Y, Lim PA. Results from a prospective acute inpatient rehabilitation database: clinical characteristics and functional outcomes using the Functional Independence Measure. *Ann Acad Med Singapore*. 2007;36:3–10.
282. Sandel ME, Jette AM, Appelman J, Terdiman J, TeSelle M, Delmonico RL, Wang H, Camicia M, Rasch EK, Brandt DE, Chan L. Designing and implementing a system for tracking functional status after stroke: a feasibility study. *PM R*. 2013;5:481–490; quiz 490.
283. Jette AM, Ni P, Rasch EK, Appelman J, Sandel ME, Terdiman J, Chan L. Evaluation of patient and proxy responses on the activity measure for postacute care. *Stroke*. 2012;43:824–829. doi: 10.1161/STROKEAHA.111.619643.

284. Hsieh CL, Hoffmann T, Gustafsson L, Lee YC. The diverse constructs use of activities of daily living measures in stroke randomized controlled trials in the years 2005–2009. *J Rehabil Med*. 2012;44:720–726.
285. Wunderlich G. *Improving the Measurement of Late-Life Disability in Population Surveys: Beyond ADLs and IADLs: Summary of a Workshop*. Washington, DC: National Academies Press; 2009.
286. Butler M, Kane R, Larson S, Jeffery M, Grove M. *Quality Improvement Measurement of Outcomes for People With Disabilities: Closing the Quality Gap: Revisiting the State of the Science*. Rockville, MD: Agency for Healthcare Research and Quality; 2012. Evidence report/technology assessment No. 208.
287. Sumathipala K, Radcliffe E, Sadler E, Wolfe CD, McKevitt C. Identifying the long-term needs of stroke survivors using the International Classification of Functioning, Disability and Health. *Chronic Illn*. 2012;8:31–44. doi: 10.1177/1742395311423848.
288. Kollen B, Kwakkel G, Lindeman E. Functional recovery after stroke: a review of current developments in stroke rehabilitation research. *Rev Recent Clin Trials*. 2006;1:75–80.
289. Patient Protection and Affordable Care Act. PL 111–148. 111th Congress (2010).
290. Broderick JE, Schneider S, Junghaenel DU, Schwartz JE, Stone AA. Validity and reliability of patient-reported outcomes measurement information system instruments in osteoarthritis. *Arthritis Care Res (Hoboken)*. 2013;65:1625–1633. doi: 10.1002/acr.22025.
291. Gershon RC, Wagster MV, Hendrie HC, Fox NA, Cook KF, Nowinski CJ. NIH toolbox for assessment of neurological and behavioral function. *Neurology*. 2013;80(suppl 3):S2–S6. doi: 10.1212/WNL.0b013e3182872e5f.
292. Teasell R. Challenges in the implementation of evidence in stroke rehabilitation. *Top Stroke Rehabil*. 2012;19:93–95. doi: 10.1310/tsr1902-93.
- 292a. Canadian Partnership for Stroke Recovery. Evidence-Based Review of Stroke Rehabilitation. <http://www.ebrsr.com/index.php>. Accessed March 5, 2016.
- 292b. Canadian Partnership for Stroke Recovery. <http://www.ebrsr.com/evidence-review/21-outcome-measures>. Evidence-based review of stroke rehabilitation. <http://www.EBRsr.com>. Accessed March 5, 2016.
293. Fitzpatrick R, Davey C, Buxton M, Jones D. Evaluating patient-based outcome measures for use in clinical trials. *Health Technology Assessment*. 1998;2:i–iv, 1–74.
294. Lang CE, Bland MD, Bailey RR, Schaefer SY, Birkenmeier RL. Assessment of upper extremity impairment, function, and activity after stroke: foundations for clinical decision making. *J Hand Ther*. 2013;26:104–114; quiz 115. doi: 10.1016/j.jht.2012.06.005.
295. Chen HM, Chen CC, Hsueh IP, Huang SL, Hsieh CL. Test-retest reproducibility and smallest real difference of 5 hand function tests in patients with stroke. *Neurorehabil Neural Repair*. 2009;23:435–440. doi: 10.1177/1545968308331146.
296. Mathiowetz V, Kashman N, Volland G, Weber K, Dowe M, Rogers S. Grip and pinch strength: normative data for adults. *Arch Phys Med Rehabil*. 1985;66:69–74.
297. Mathiowetz V, Weber K, Volland G, Kashman N. Reliability and validity of grip and pinch strength evaluations. *J Hand Surg Am*. 1984;9:222–226.
298. Velstra IM, Ballert CS, Cieza A. A systematic literature review of outcome measures for upper extremity function using the *International Classification of Functioning, Disability, and Health* as reference. *PM R*. 2011;3:846–860. doi: 10.1016/j.pmrj.2011.03.014.
299. Sivan M, O'Connor RJ, Makower S, Levesley M, Bhakta B. Systematic review of outcome measures used in the evaluation of robot-assisted upper limb exercise in stroke. *J Rehabil Med*. 2011;43:181–189. doi: 10.2340/16501977-0674.
300. Hsieh YW, Wu CY, Lin KC, Chang YF, Chen CL, Liu JS. Responsiveness and validity of three outcome measures of motor function after stroke rehabilitation. *Stroke*. 2009;40:1386–1391. doi: 10.1161/STROKEAHA.108.530584.
301. Hsueh IP, Hsu MJ, Sheu CF, Lee S, Hsieh CL, Lin JH. Psychometric comparisons of 2 versions of the Fugl-Meyer Motor Scale and 2 versions of the Stroke Rehabilitation Assessment of Movement. *Neurorehabil Neural Repair*. 2008;22:737–744. doi: 10.1177/1545968308315999.
302. Lin JH, Hsu MJ, Sheu CF, Wu TS, Lin RT, Chen CH, Hsieh CL. Psychometric comparisons of 4 measures for assessing upper-extremity function in people with stroke. *Phys Ther*. 2009;89:840–850. doi: 10.2522/ptj.20080285.
303. Beebe JA, Lang CE. Relationships and responsiveness of six upper extremity function tests during the first six months of recovery after stroke. *J Neurol Phys Ther*. 2009;33:96–103. doi: 10.1097/NPT.0b013e3181a33638.
304. Connell LA, Tyson SF. Clinical reality of measuring upper-limb ability in neurologic conditions: a systematic review. *Arch Phys Med Rehabil*. 2012;93:221–228. doi: 10.1016/j.apmr.2011.09.015.
305. Lemmens RJ, Timmermans AA, Janssen-Potten YJ, Smeets RJ, Seelen HA. Valid and reliable instruments for arm-hand assessment at ICF activity level in persons with hemiplegia: a systematic review. *BMC Neurol*. 2012;12:21. doi: 10.1186/1471-2377-12-21.
306. Rowland TJ, Gustafsson L. Assessments of upper limb ability following stroke: a review. *Br J Occup Ther*. 2008;71:427–437.
307. Sullivan JE, Crowner BE, Kluding PM, Nichols D, Rose DK, Yoshida R, Pinto Zipp G. Outcome measures for individuals with stroke: process and recommendations from the American Physical Therapy Association Neurology Section Task Force. *Phys Ther*. 2013;93:1383–1396. doi: 10.2522/ptj.20120492.
308. Pollock C, Eng J, Garland S. Clinical measurement of walking balance in people post stroke: a systematic review. *Clin Rehabil*. 2011;25:693–708. doi: 10.1177/0269215510397394.
309. Tyson SF, Connell LA. How to measure balance in clinical practice: a systematic review of the psychometrics and clinical utility of measures of balance activity for neurological conditions. *Clin Rehabil*. 2009;23:824–840. doi: 10.1177/0269215509335018.
310. Mao HF, Hsueh IP, Tang PF, Sheu CF, Hsieh CL. Analysis and comparison of the psychometric properties of three balance measures for stroke patients. *Stroke*. 2002;33:1022–1027.
311. Blum L, Korner-Bitensky N. Usefulness of the Berg Balance Scale in stroke rehabilitation: a systematic review. *Phys Ther*. 2008;88:559–566. doi: 10.2522/ptj.20070205.
312. Tyson S, Connell L. The psychometric properties and clinical utility of measures of walking and mobility in neurological conditions: a systematic review. *Clin Rehabil*. 2009;23:1018–1033. doi: 10.1177/0269215509339004.
313. Kitsos G, Harris D, Pollack M, Hubbard IJ. Assessments in Australian stroke rehabilitation units: a systematic review of the post-stroke validity of the most frequently used. *Disabil Rehabil*. 2011;33:2620–2632. doi: 10.3109/09638288.2011.575526.
314. Ferrarello F, Bianchi VA, Baccini M, Rubbieri G, Mossello E, Cavallini MC, Marchionni N, Di Bari M. Tools for observational gait analysis in patients with stroke: a systematic review. *Phys Ther*. 2013;93:1673–1685. doi: 10.2522/ptj.20120344.
315. Ashford S, Slade M, Malaprade F, Turner-Stokes L. Evaluation of functional outcome measures for the hemiparetic upper limb: a systematic review. *J Rehabil Med*. 2008;40:787–795. doi: 10.2340/16501977-0276.
316. Kim JH, Park EY. Balance self-efficacy in relation to balance and activities of daily living in community residents with stroke. *Disabil Rehabil*. 2014;36:295–299. doi: 10.3109/09638288.2013.790488.
317. Salbach NM, Mayo NE, Robichaud-Ekstrand S, Hanley JA, Richards CL, Wood-Dauphinee S. Balance self-efficacy and its relevance to physical function and perceived health status after stroke. *Arch Phys Med Rehabil*. 2006;87:364–370. doi: 10.1016/j.apmr.2005.11.017.
318. Salbach NM, Mayo NE, Hanley JA, Richards CL, Wood-Dauphinee S. Psychometric evaluation of the original and Canadian French version of the activities-specific balance confidence scale among people with stroke. *Arch Phys Med Rehabil*. 2006;87:1597–1604. doi: 10.1016/j.apmr.2006.08.336.
319. Botner EM, Miller WC, Eng JJ. Measurement properties of the Activities-specific Balance Confidence Scale among individuals with stroke. *Disabil Rehabil*. 2005;27:156–163. doi: 10.1080/09638280400008982.
320. Deleted in proof.
321. Rand D, Eng JJ. Disparity between functional recovery and daily use of the upper and lower extremities during subacute stroke rehabilitation. *Neurorehabil Neural Repair*. 2012;26:76–84. doi: 10.1177/1545968311408918.
322. Rand D, Eng JJ, Tang PF, Jeng JS, Hung C. How active are people with stroke? Use of accelerometers to assess physical activity. *Stroke*. 2009;40:163–168. doi: 10.1161/STROKEAHA.108.523621.
323. Bailey RR, Lang CE. Upper-limb activity in adults: referent values using accelerometry. *J Rehabil Res Dev*. 2013;50:1213–1222. doi: 10.1682/JRRD.2012.12.0222.
324. Dobkin BH, Dorsch A. The promise of mHealth: daily activity monitoring and outcome assessments by wearable sensors. *Neurorehabil Neural Repair*. 2011;25:788–798. doi: 10.1177/1545968311425908.

325. Dobkin BH, Xu X, Batalin M, Thomas S, Kaiser W. Reliability and validity of bilateral ankle accelerometer algorithms for activity recognition and walking speed after stroke. *Stroke*. 2011;42:2246–2250. doi: 10.1161/STROKEAHA.110.611095.
326. Uswatte G, Foo WL, Olmstead H, Lopez K, Holand A, Simms LB. Ambulatory monitoring of arm movement using accelerometry: an objective measure of upper-extremity rehabilitation in persons with chronic stroke. *Arch Phys Med Rehabil*. 2005;86:1498–1501.
327. Carroll SL, Greig CA, Lewis SJ, McMurdo ME, Snihotta FF, Johnston M, Johnston DW, Scopes J, Mead GE. The use of pedometers in stroke survivors: are they feasible and how well do they detect steps? *Arch Phys Med Rehabil*. 2012;93:466–470. doi: 10.1016/j.apmr.2011.08.047.
328. Roos MA, Rudolph KS, Reisman DS. The structure of walking activity in people after stroke compared with older adults without disability: a cross-sectional study. *Phys Ther*. 2012;92:1141–1147. doi: 10.2522/ptj.20120034.
329. Hsueh IP, Chen JH, Wang CH, Hou WH, Hsieh CL. Development of a computerized adaptive test for assessing activities of daily living in outpatients with stroke. *Phys Ther*. 2013;93:681–693. doi: 10.2522/ptj.20120173.
330. Mahoney FI, Barthel DW. Functional evaluation: the Barthel Index. *Md State Med J*. 1965;14:61–65.
331. Holbrook M, Skilbeck CE. An activities index for use with stroke patients. *Age Ageing*. 1983;12:166–170.
332. Chen HF, Wu CY, Lin KC, Chen CL, Huang PC, Hsieh CJ, Liu JS. Rasch validation of a combined measure of basic and extended daily life functioning after stroke. *Neurorehabil Neural Repair*. 2013;27:125–132. doi: 10.1177/1545968312457828.
333. Sathian K, Buxbaum LJ, Cohen LG, Krakauer JW, Lang CE, Corbetta M, Fitzpatrick SM. Neurological principles and rehabilitation of action disorders: common clinical deficits. *Neurorehabil Neural Repair*. 2011;25(suppl):21S–32S. doi: 10.1177/1545968311410941.
334. Pineiro R, Pendlebury ST, Smith S, Flitney D, Blamire AM, Styles P, Matthews PM. Relating MRI changes to motor deficit after ischemic stroke by segmentation of functional motor pathways. *Stroke*. 2000;31:672–679.
335. Lindenberg R, Renga V, Zhu LL, Betzler F, Alsop D, Schlaug G. Structural integrity of corticospinal motor fibers predicts motor impairment in chronic stroke. *Neurology*. 2010;74:280–287. doi: 10.1212/WNL.0b013e3181ccc6d9.
336. Stinear CM, Barber PA, Smale PR, Coxon JP, Fleming MK, Byblow WD. Functional potential in chronic stroke patients depends on corticospinal tract integrity. *Brain*. 2007;130(pt 1):170–180. doi: 10.1093/brain/awl333.
337. Beebe JA, Lang CE. Active range of motion predicts upper extremity function 3 months after stroke. *Stroke*. 2009;40:1772–1779. doi: 10.1161/STROKEAHA.108.536763.
338. Kwakkel G, Kollen B, Lindeman E. Understanding the pattern of functional recovery after stroke: facts and theories. *Restor Neurol Neurosci*. 2004;22:281–299.
339. Nijland RH, van Wegen EE, Harmeling-van der Wel BC, Kwakkel G; EPOS Investigators. Presence of finger extension and shoulder abduction within 72 hours after stroke predicts functional recovery: Early Prediction of Functional Outcome After Stroke: the EPOS cohort study. *Stroke*. 2010;41:745–750. doi: 10.1161/STROKEAHA.109.572065.
340. Bland MD, Sturmshak A, Whitson M, Connor LT, Fucetola R, Huskey T, Corbetta M, Lang CE. Prediction of discharge walking ability from initial assessment in a stroke inpatient rehabilitation facility population. *Arch Phys Med Rehabil*. 2012;93:1441–1447. doi: 10.1016/j.apmr.2012.02.029.
341. Harris JE, Eng JJ. Paretic upper-limb strength best explains arm activity in people with stroke. *Phys Ther*. 2007;87:88–97. doi: 10.2522/ptj.20060065.
342. Baker K, Cano SJ, Playford ED. Outcome measurement in stroke: a scale selection strategy. *Stroke*. 2011;42:1787–1794. doi: 10.1161/STROKEAHA.110.608505.
343. Barak S, Duncan PW. Issues in selecting outcome measures to assess functional recovery after stroke. *NeuroRx*. 2006;3:505–524. doi: 10.1016/j.nurx.2006.07.009.
344. Duncan PW, Lai SM, Keighley J. Defining post-stroke recovery: implications for design and interpretation of drug trials. *Neuropharmacology*. 2000;39:835–841.
345. Bland MD, Sturmshak A, Whitson M, Harris H, Connor LT, Fucetola R, Edmiaston J, Huskey T, Carter A, Krampner M, Corbetta M, Lang CE. Clinician adherence to a standardized assessment battery across settings and disciplines in a poststroke rehabilitation population. *Arch Phys Med Rehabil*. 2013;94:1048–1053.e1. doi: 10.1016/j.apmr.2013.02.004.
346. Brown RW. Why is quality assurance so difficult? A review of issues in quality assurance over the last decade. *Intern Med J*. 2002;32:331–337.
347. Weinert CR, Mann HJ. The science of implementation: changing the practice of critical care. *Curr Opin Crit Care*. 2008;14:460–465. doi: 10.1097/MCC.0b013e3283079eb5.
348. Burdick KE, Endick CJ, Goldberg JF. Assessing cognitive deficits in bipolar disorder: are self-reports valid? *Psychiatry Res*. 2005;136:43–50. doi: 10.1016/j.psychres.2004.12.009.
349. Adams SA, Matthews CE, Ebbeling CB, Moore CG, Cunningham JE, Fulton J, Hebert JR. The effect of social desirability and social approval on self-reports of physical activity [published correction appears in *Am J Epidemiol*. 2005;161:899]. *Am J Epidemiol*. 2005;161:389–398. doi: 10.1093/aje/kwi054.
350. Moore JL, Roth EJ, Killian C, Hornby TG. Locomotor training improves daily stepping activity and gait efficiency in individuals poststroke who have reached a “plateau” in recovery. *Stroke*. 2010;41:129–135. doi: 10.1161/STROKEAHA.109.563247.
351. National Institute of Deafness and Other Communication Disorders. Aphasia. <http://www.nidcd.nih.gov/health/voice/aphasia.htm>. Accessed March 5, 2016.
352. Hall N, Boisvert M, Steele R. Telepractice in the assessment and treatment of individuals with aphasia: a systematic review. *Int J Telerehabil*. 2013;5:27–38. doi: 10.5195/ijt.2013.6119.
353. Cherney LR, van Vuuren S. Telerehabilitation, virtual therapists, and acquired neurologic speech and language disorders. *Semin Speech Lang*. 2012;33:243–257. doi: 10.1055/s-0032-1320044.
354. Hill AJ, Theodoros DG, Russell TG, Ward EC. The redesign and re-evaluation of an Internet-based telerehabilitation system for the assessment of dysarthria in adults. *Telemed J E Health*. 2009;15:840–850. doi: 10.1089/tmj.2009.0015.
355. McClure JA, Salter K, Foley N, Mahon H, Teasell R. Adherence to Canadian Best Practice Recommendations for Stroke Care: vascular cognitive impairment screening and assessment practices in an Ontario inpatient stroke rehabilitation facility. *Top Stroke Rehabil*. 2012;19:141–148. doi: 10.1310/tsr1902-141.
356. Patel MD, Coshall C, Rudd AG, Wolfe CD. Cognitive impairment after stroke: clinical determinants and its associations with long-term stroke outcomes. *J Am Geriatr Soc*. 2002;50:700–706.
357. Patel MD, Coshall C, Rudd AG, Wolfe CD. Natural history of cognitive impairment after stroke and factors associated with its recovery. *Clin Rehabil*. 2003;17:158–166.
358. Tatemichi TK, Desmond DW, Stern Y, Palk M, Sano M, Bagiella E. Cognitive impairment after stroke: frequency, patterns and relationship to functional abilities. *J Neurol Neurosurg Psychiatry*. 1994;57:202–207.
359. Tatemichi TK, Paik M, Bagiella E, Desmond DW, Pirro M, Hanzawa LK. Dementia after stroke is a predictor of long-term survival. *Stroke*. 1994;25:1915–1919.
360. Mysiw WJ, Beegan JG, Gatens PF. Prospective cognitive assessment of stroke patients before inpatient rehabilitation: the relationship of the Neurobehavioral Cognitive Status Examination to functional improvement. *Am J Phys Med Rehabil*. 1989;68:168–171.
361. Deleted in proof.
362. Klingner CM, Witte OW, Günther A. Sensory syndromes. *Front Neurol Neurosci*. 2012;30:4–8. doi: 10.1159/000333373.
363. Pambakian A, Currie J, Kennard C. Rehabilitation strategies for patients with homonymous visual field defects. *J Neuroophthalmol*. 2005;25:136–142.
364. Rowe FJ, Wright D, Brand D, Jackson C, Harrison S, Maan T, Scott C, Vogwell L, Peel S, Akerman N, Dodridge C, Howard C, Shipman T, Sperring U, Macdiarmid S, Freeman C. A prospective profile of visual field loss following stroke: prevalence, type, rehabilitation, and outcome. *Biomed Res Int*. 2013;2013:719096. doi: 10.1155/2013/719096.
365. Modha DS, Singh R. Network architecture of the long-distance pathways in the macaque brain. *Proc Natl Acad Sci USA*. 2010;107:13485–13490. doi: 10.1073/pnas.1008054107.
366. Mountcastle V. *The Sensory Hand: Neural Mechanisms of Somatic Sensation*. Boston, MA: Harvard University Press; 2005.
367. Tyson SF, Hanley M, Chillala J, Selley AB, Tallis RC. Sensory loss in hospital-admitted people with stroke: characteristics, associated factors, and relationship with function. *Neurorehabil Neural Repair*. 2008;22:166–172. doi: 10.1177/1545968307305523.

368. Parker J, Mountain G, Hammerton J. A review of the evidence underpinning the use of visual and auditory feedback for computer technology in post-stroke upper-limb rehabilitation. *Disabil Rehabil Assist Technol*. 2011;6:465–472. doi: 10.3109/17483107.2011.556209.
369. Baram Y. Virtual sensory feedback for gait improvement in neurological patients. *Front Neurol*. 2013;4:138. doi: 10.3389/fneur.2013.00138.
370. Mousavi Hondori H, Khademi M, Dodakian L, Cramer SC, Lopes CV. A spatial augmented reality rehab system for post-stroke hand rehabilitation. *Stud Health Technol Inform*. 2013;184:279–285.
371. Winward CE, Halligan PW, Wade DT. Somatosensory recovery: a longitudinal study of the first 6 months after unilateral stroke. *Disabil Rehabil*. 2007;29:293–299. doi: 10.1080/09638280600756489.
372. Ruch TC, Fulton JF, German WJ. Sensory discrimination in monkey, chimpanzee and man after lesions of the parietal lobe. *Arch Neurol Psych*. 1938;39:919–938.
373. Xerri C, Merzenich MM, Peterson BE, Jenkins W. Plasticity of primary somatosensory cortex paralleling sensorimotor skill recovery from stroke in adult monkeys. *J Neurophysiol*. 1998;79:2119–2148.
374. Harrison TC, Silasi G, Boyd JD, Murphy TH. Displacement of sensory maps and disorganization of motor cortex after targeted stroke in mice. *Stroke*. 2013;44:2300–2306. doi: 10.1161/STROKEAHA.113.001272.
375. Carey LM, Abbott DF, Puce A, Jackson GD, Syngieniotis A, Donnan GA. Reemergence of activation with poststroke somatosensory recovery: a serial fMRI case study. *Neurology*. 2002;59:749–752.
376. Schaechter JD, Moore CI, Connell BD, Rosen BR, Dijkhuizen RM. Structural and functional plasticity in the somatosensory cortex of chronic stroke patients. *Brain*. 2006;129(pt 10):2722–2733. doi: 10.1093/brain/awl214.
377. Sullivan JE, Hedman LD. Sensory dysfunction following stroke: incidence, significance, examination, and intervention. *Top Stroke Rehabil*. 2008;15:200–217. doi: 10.1310/tsr1503-200.
378. Lin JH, Hsueh IP, Sheu CF, Hsieh CL. Psychometric properties of the sensory scale of the Fugl-Meyer Assessment in stroke patients. *Clin Rehabil*. 2004;18:391–397.
379. Benaim C, Froger J, Cazottes C, Gueben D, Porte M, Desnuelle C, Pellissier JY. Use of the Faces Pain Scale by left and right hemispheric stroke patients. *Pain*. 2007;128:52–58. doi: 10.1016/j.pain.2006.08.029.
380. Simo LS, Ghez C, Botzer L, Scheidt RA. A quantitative and standardized robotic method for the evaluation of arm proprioception after stroke. *Conf Proc IEEE Eng Med Biol Soc*. 2011;2011:8227–8230. doi: 10.1109/IEMBS.2011.6092029.
381. Semrau JA, Herter TM, Scott SH, Dukelow SP. Robotic identification of kinesthetic deficits after stroke. *Stroke*. 2013;44:3414–3421. doi: 10.1161/STROKEAHA.113.002058.
382. Pollock A, Hazelton C, Henderson CA, Angillet J, Dhillon B, Langhorne P, Livingstone K, Munro FA, Orr H, Rowe F, Shahani U. Interventions for visual field defects in patients with stroke. *Stroke*. 2012;43:e37–e38. doi: 10.1161/STROKEAHA.111.639815.
383. Kasten E, Poggel DA, Müller-Oehring E, Gothe J, Schulte T, Sabel BA. Restoration of vision II: residual functions and training-induced visual field enlargement in brain-damaged patients. *Restor Neurol Neurosci*. 1999;15:273–287.
384. Gray CS, French JM, Bates D, Cartlidge NE, Venables GS, James OF. Recovery of visual fields in acute stroke: homonymous hemianopia associated with adverse prognosis. *Age Ageing*. 1989;18:419–421.
385. Ali M, Hazelton C, Lyden P, Pollock A, Brady M; VISTA Collaboration. Recovery from poststroke visual impairment: evidence from a clinical trials resource. *Neurorehabil Neural Repair*. 2013;27:133–141. doi: 10.1177/1545968312454683.
386. Yamasoba T, Kikuchi S, Higo R. Deafness associated with vertebrobasilar insufficiency. *J Neurol Sci*. 2001;187:69–75.
387. Lee H. Recent advances in acute hearing loss due to posterior circulation ischemic stroke. *J Neurol Sci*. 2014;338:23–29. doi: 10.1016/j.jns.2013.12.048.
388. Lee H, Baloh RW. Sudden deafness in vertebrobasilar ischemia: clinical features, vascular topographical patterns and long-term outcome. *J Neurol Sci*. 2005;228:99–104. doi: 10.1016/j.jns.2004.10.016.
389. Hinchey JA, Shephard T, Furie K, Smith D, Wang D, Tonn S; Stroke Practice Improvement Network Investigators. Formal dysphagia screening protocols prevent pneumonia. *Stroke*. 2005;36:1972–1976. doi: 10.1161/01.STR.0000177529.86868.8d.
390. Lakshminarayan K, Tsai AW, Tong X, Vazquez G, Peacock JM, George MG, Luepker RV, Anderson DC. Utility of dysphagia screening results in predicting poststroke pneumonia. *Stroke*. 2010;41:2849–2854. doi: 10.1161/STROKEAHA.110.597039.
391. Foley NC, Martin RE, Salter KL, Teasell RW. A review of the relationship between dysphagia and malnutrition following stroke. *J Rehabil Med*. 2009;41:707–713. doi: 10.2340/16501977-0415.
392. Perry L, Hamilton S, Williams J, Jones S. Nursing interventions for improving nutritional status and outcomes of stroke patients: descriptive reviews of processes and outcomes. *Worldviews Evid Based Nurs*. 2013;10:17–40. doi: 10.1111/j.1741-6787.2012.00255.x.
393. Donovan NJ, Daniels SK, Edmiston J, Weinhardt J, Summers D, Mitchell PH; on behalf of the American Heart Association Council on Cardiovascular Nursing and Stroke Council. Dysphagia screening: state of the art: invitational conference proceeding from the State-of-the-Art Nursing Symposium, International Stroke Conference 2012. *Stroke*. 2013;44:e24–e31. doi: 10.1161/STR.0b013e3182877f57.
394. Jauch EC, Saver JL, Adams HP Jr, Bruno A, Connors JJ, Demaerschalk BM, Khatri P, McMullan PW Jr, Qureshi AI, Rosenfield K, Scott PA, Summers DR, Wang DZ, Wintermark M, Yonas H; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013;44:870–947. doi: 10.1161/STR.0b013e318284056a.
395. Singh S, Hamdy S. Dysphagia in stroke patients. *Postgrad Med J*. 2006;82:383–391. doi: 10.1136/pgmj.2005.043281.
396. McCullough GH, Rosenbek JC, Wertz RT, McCoy S, Mann G, McCullough K. Utility of clinical swallowing examination measures for detecting aspiration post-stroke. *J Speech Lang Hear Res*. 2005;48:1280–1293. doi: 10.1044/1092-4388(2005/089).
397. Garon BR, Sierzant T, Ormiston C. Silent aspiration: results of 2,000 video fluoroscopic evaluations. *J Neurosci Nurs*. 2009;41:178–185; quiz 186–187.
398. Bingjie L, Tong Z, Xinting S, Jianmin X, Guijun J. Quantitative videofluoroscopic analysis of penetration-aspiration in post-stroke patients. *Neurol India*. 2010;58:42–47. doi: 10.4103/0028-3886.60395.
399. National Collaborating Centre for Chronic Conditions. *Stroke: National Clinical Guideline for Diagnosis and Initial Management of Acute Stroke and Transient Ischemic Attack (TIA)*. London, UK: Royal College of Physicians, 2008.
400. Geeganage C, Beavan J, Ellender S, Bath PM. Interventions for dysphagia and nutritional support in acute and subacute stroke. *Cochrane Database Syst Rev*. 2012;10:CD000323.
401. Ashford J, McCabe D, Wheeler-Hegland K, Frymark T, Mullen R, Musson N, Schooling T, Hammond CS. Evidence-based systematic review: oropharyngeal dysphagia behavioral treatments, part III: impact of dysphagia treatments on populations with neurological disorders. *J Rehabil Res Dev*. 2009;46:195–204.
402. Robbins J, Butler SG, Daniels SK, Diez Gross R, Langmore S, Lazarus CL, Martin-Harris B, McCabe D, Musson N, Rosenbek J. Swallowing and dysphagia rehabilitation: translating principles of neural plasticity into clinically oriented evidence. *J Speech Lang Hear Res*. 2008;51:S276–S300. doi: 10.1044/1092-4388(2008/021).
403. Xie Y, Wang L, He J, Wu T. Acupuncture for dysphagia in acute stroke. *Cochrane Database Syst Rev*. 2008;CD006076. doi: 10.1002/14651858.CD006076.pub2.
404. Sørensen RT, Rasmussen RS, Overgaard K, Lerche A, Johansen AM, Lindhardt T. Dysphagia screening and intensified oral hygiene reduce pneumonia after stroke. *J Neurosci Nurs*. 2013;45:139–146. doi: 10.1097/JNN.0b013e31828a412c.
405. Langdon PC, Lee AH, Binns CW. High incidence of respiratory infections in “nil by mouth” tube-fed acute ischemic stroke patients. *Neuroepidemiology*. 2009;32:107–113. doi: 10.1159/000177036.
406. Dennis M, Lewis S, Cranswick G, Forbes J; FOOD Trial Collaboration. FOOD: a multicentre randomised trial evaluating feeding policies in patients admitted to hospital with a recent stroke. *Health Technol Assess*. 2006;10:iii-iv, ix-x, 1–120.
407. Dennis MS, Lewis SC, Warlow C; FOOD Trial Collaboration. Effect of timing and method of enteral tube feeding for dysphagic stroke patients (FOOD): a multicentre randomised controlled trial. *Lancet*. 2005;365:764–772. doi: 10.1016/S0140-6736(05)17983-5.
408. Dennis MS, Lewis SC, Warlow C; FOOD Trial Collaboration. Routine oral nutritional supplementation for stroke patients in hospital (FOOD): a multicentre randomised controlled trial. *Lancet*. 2005;365:755–763. doi: 10.1016/S0140-6736(05)17982-3.

409. Leys D, Hénon H, Mackowiak-Cordoliani MA, Pasquier F. Poststroke dementia. *Lancet Neurol*. 2005;4:752–759. doi: 10.1016/S1474-4422(05)70221-0.
410. 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. doi: 10.1053/apmr.2000.19240.
411. Zedlitz AM, Rietveld TC, Geurts AC, Fasotti L. Cognitive and graded activity training can alleviate persistent fatigue after stroke: a randomized, controlled trial. *Stroke*. 2012;43:1046–1051. doi: 10.1161/STROKEAHA.111.632117.
412. Toniolo S. Neuropsychological interventions in stroke survivors: implications for evidence based psychological practice. *G Ital Med Lav Ergon*. 2011;33(suppl A):A29–A36.
413. Sofi F, Valecchi D, Bacci D, Abbate R, Gensini GF, Casini A, Macchi C. Physical activity and risk of cognitive decline: a meta-analysis of prospective studies. *J Intern Med*. 2011;269:107–117. doi: 10.1111/j.1365-2796.2010.02281.x.
414. Angevaren M, Aufdemkampe G, Verhaar HJ, Aleman A, Vanhees L. Physical activity and enhanced fitness to improve cognitive function in older people without known cognitive impairment. *Cochrane Database Syst Rev*. 2008;CD005381. doi: 10.1002/14651858.CD005381.pub2.
415. Cumming TB, Tyedin K, Churilov L, Morris ME, Bernhardt J. The effect of physical activity on cognitive function after stroke: a systematic review. *Int Psychogeriatr*. 2012;24:557–567. doi: 10.1017/S1041610211001980.
416. Janssen H, Bernhardt J, Collier JM, Sena ES, McElduff P, Attia J, Pollack M, Howells DW, Nilsson M, Calford MB, Spratt NJ. An enriched environment improves sensorimotor function post-ischemic stroke. *Neurorehabil Neural Repair*. 2010;24:802–813. doi: 10.1177/1545968310372092.
417. Janssen H, Ada L, Bernhardt J, McElduff P, Pollack M, Nilsson M, Spratt NJ. An enriched environment increases activity in stroke patients undergoing rehabilitation in a mixed rehabilitation unit: a pilot non-randomized controlled trial. *Disabil Rehabil*. 2014;36:255–262. doi: 10.3109/09638288.2013.788218.
418. Cicerone KD, Langenbahn DM, Braden C, Malec JF, Kalmar K, Fraas M, Felicetti T, Laatsch L, Harley JP, Bergquist T, Azulay J, Cantor J, Ashman T. Evidence-based cognitive rehabilitation: updated review of the literature from 2003 through 2008. *Arch Phys Med Rehabil*. 2011;92:519–530.
419. Bowen A, Hazelton C, Pollock A, Lincoln NB. Cognitive rehabilitation for spatial neglect following stroke. *Cochrane Database Syst Rev*. 2013;7:CD003586. doi: 10.1002/14651858.CD003586.pub3.
420. Loetscher T, Lincoln NB. Cognitive rehabilitation for attention deficits following stroke. *Cochrane Database Syst Rev*. 2013;5:CD002842. doi: 10.1002/14651858.CD002842.pub2.
421. das Nair R, Lincoln N. Cognitive rehabilitation for memory deficits following stroke. *Cochrane Database Syst Rev*. 2007:CD002293.
422. Barker-Collo SL, Feigin VL, Lawes CM, Parag V, Senior H, Rodgers A. Reducing attention deficits after stroke using attention process training: a randomized controlled trial. *Stroke*. 2009;40:3293–3298. doi: 10.1161/STROKEAHA.109.558239.
423. Winkens I, Van Heugten CM, Wade DT, Habets EJ, Fasotti L. Efficacy of time pressure management in stroke patients with slowed information processing: a randomized controlled trial. *Arch Phys Med Rehabil*. 2009;90:1672–1679. doi: 10.1016/j.apmr.2009.04.016.
424. Cappa S, Benke T, Clarke S, Rossi B, Stemmer B, van Heugten C; Task Force on Cognitive Rehabilitation; European Federation of Neurological Societies. EFNS guidelines on cognitive rehabilitation: report of an EFNS task force. *Eur J Neurol*. 2005;12:665–680.
425. Chung CS, Pollock A, Campbell T, Durward BR, Hagen S. Cognitive rehabilitation for executive dysfunction in adults with stroke or other adult non-progressive acquired brain damage. *Cochrane Database Syst Rev*. 2013;4:CD008391. doi: 10.1002/14651858.CD008391.pub2.
426. Poulin V, Korner-Bitensky N, Dawson DR, Bherer L. Efficacy of executive function interventions after stroke: a systematic review. *Top Stroke Rehabil*. 2012;19:158–171. doi: 10.1310/tsr1902-158.
427. Salter K, Teasell R, Bitensky J, Foley N, Bhogal SK, Mahon H, McClure JA. Cognitive disorders and apraxia: evidence based review of stroke rehabilitation, version 15. 2012. <http://www.ebsr.com/evidence-review/12-cognitive-disorders-and-apraxia>. Accessed March 5, 2016.
428. Man DW, Soong WY, Tam SF, Hui-Chan CW. A randomized clinical trial study on the effectiveness of a tele-analogy-based problem-solving programme for people with acquired brain injury (ABI). *NeuroRehabilitation*. 2006;21:205–217.
429. Fish J, Manly T, Emslie H, Evans JJ, Wilson BA. Compensatory strategies for acquired disorders of memory and planning: differential effects of a paging system for patients with brain injury of traumatic versus cerebrovascular aetiology. *J Neurol Neurosurg Psychiatry*. 2008;79:930–935. doi: 10.1136/jnnp.2007.125203.
430. Chen P, Hartman AJ, Priscilla Galarza C, DeLuca J. Global processing training to improve visuospatial memory deficits after right-brain stroke. *Arch Clin Neuropsychol*. 2012;27:891–905. doi: 10.1093/arclin/acs089.
431. Hildebrandt H, Bussmann-Mork B, Schwendemann G. Group therapy for memory impaired patients: a partial remediation is possible. *J Neurol*. 2006;253:512–519. doi: 10.1007/s00415-006-0013-6.
432. Doornhein K, De Haan EH. Cognitive training for memory deficits in stroke patients. *Neuropsychol Rehabil*. 1998;8:393–400. doi: 10.1080/713755579.
433. Aben L, Heijnenbroek-Kal MH, van Loon EM, Groet E, Ponds RW, Busschbach JJ, Ribbers GM. Training memory self-efficacy in the chronic stage after stroke: a randomized controlled trial. *Neurorehabil Neural Repair*. 2013;27:110–117. doi: 10.1177/1545968312455222.
434. Westerberg H, Jacobaeus H, Hirvikoski T, Clevberger P, Ostensson ML, Bartfai A, Klingberg T. Computerized working memory training after stroke: a pilot study. *Brain Inj*. 2007;21:21–29. doi: 10.1080/02699050601148726.
435. Lemoncello R, Sohlberg MM, Fickas S, Prideaux J. A randomised controlled crossover trial evaluating Television Assisted Prompting (TAP) for adults with acquired brain injury. *Neuropsychol Rehabil*. 2011;21:825–846. doi: 10.1080/09602011.2011.618661.
436. Eghdam A, Scholl J, Bartfai A, Koch S. Information and communication technology to support self-management of patients with mild acquired cognitive impairments: systematic review. *J Med Internet Res*. 2012;14:e159. doi: 10.2196/jmir.2275.
437. Hamilton RH, Chrysikou EG, Coslett B. Mechanisms of aphasia recovery after stroke and the role of noninvasive brain stimulation. *Brain Lang*. 2011;118:40–50. doi: 10.1016/j.bandl.2011.02.005.
438. Monti A, Ferrucci R, Fumagalli M, Mameli F, Cogiamanian F, Ardolino G, Priori A. Transcranial direct current stimulation (tDCS) and language. *J Neurol Neurosurg Psychiatry*. 2013;84:832–842. doi: 10.1136/jnnp-2012-302825.
439. Deleted in proof.
440. McDonnell MN, Smith AE, Mackintosh SF. Aerobic exercise to improve cognitive function in adults with neurological disorders: a systematic review. *Arch Phys Med Rehabil*. 2011;92:1044–1052. doi: 10.1016/j.apmr.2011.01.021.
441. Devine JM, Zafonte RD. Physical exercise and cognitive recovery in acquired brain injury: a review of the literature. *PM R*. 2009;1:560–575. doi: 10.1016/j.pmrj.2009.03.015.
442. Särkämö T, Tervaniemi M, Laitinen S, Forsblom A, Soinila S, Mikkonen M, Autti T, Silvennoinen HM, Erkkilä J, Laine M, Peretz I, Hietanen M. Music listening enhances cognitive recovery and mood after middle cerebral artery stroke. *Brain*. 2008;131(pt 3):866–876. doi: 10.1093/brain/awn013.
443. Kim BR, Chun MH, Kim LS, Park JY. Effect of virtual reality on cognition in stroke patients. *Ann Rehabil Med*. 2011;35:450–459. doi: 10.5535/arm.2011.35.4.450.
444. Gladstone DJ, Danells CJ, Armesto A, McIlroy WE, Staines WR, Graham SJ, Herrmann N, Szalai JP, Black SE; Subacute Therapy With Amphetamine and Rehabilitation for Stroke Study Investigators. Physiotherapy coupled with dextroamphetamine for rehabilitation after hemiparetic stroke: a randomized, double-blind, placebo-controlled trial. *Stroke*. 2006;37:179–185. doi: 10.1161/01.STR.0000195169.42447.78.
445. Tardy J, Pariente J, Leger A, Dechaumont-Palacin S, Gerdelat A, Guiraud V, Conchou F, Albucher JF, Marque P, Franceries X, Cognard C, Rascol O, Chollet F, Loubinoux I. Methylphenidate modulates cerebral post-stroke reorganization. *Neuroimage*. 2006;33:913–922. doi: 10.1016/j.neuroimage.2006.07.014.
446. Berkowitz HL. Modafinil in poststroke depression. *Psychosomatics*. 2005;46:93; author reply 93–94. doi: 10.1176/appi.psy.46.1.93.
447. Brioschi A, Gramigna S, Werth E, Staub F, Ruffieux C, Bassetti C, Schluep M, Annoni JM. Effect of modafinil on subjective fatigue in multiple sclerosis and stroke patients. *Eur Neurol*. 2009;62:243–249. doi: 10.1159/000232927.
448. Chang WH, Park YH, Ohn SH, Park CH, Lee PK, Kim YH. Neural correlates of donepezil-induced cognitive improvement in patients with right

- hemisphere stroke: a pilot study. *Neuropsychol Rehabil.* 2011;21:502–514. doi: 10.1080/09602011.2011.582708.
449. Narasimhalu K, Effendy S, Sim CH, Lee JM, Chen I, Hia SB, Xue HL, Corrales MP, Chang HM, Wong MC, Chen CP, Tan EK. A randomized controlled trial of rivastigmine in patients with cognitive impairment no dementia because of cerebrovascular disease. *Acta Neurol Scand.* 2010;121:217–224. doi: 10.1111/j.1600-0404.2009.01263.x.
 450. Narushima K, Paradiso S, Moser DJ, Jorge R, Robinson RG. Effect of antidepressant therapy on executive function after stroke. *Br J Psychiatry.* 2007;190:260–265. doi: 10.1192/bjp.bp.106.025064.
 451. Bauxbaum LJ, Haaland KY, Hallett M, Wheaton L, Heilman KM, Rodriguez A, Gonzalez Rothi LJ. Treatment of limb apraxia: moving forward to improved action [published correction appears in *Am J Phys Med Rehabil.* 2008;87:424]. *Am J Phys Med Rehabil.* 2008;87:149–161. doi: 10.1097/PHM.0b013e31815e6727.
 452. Zwinkels A, Geusgens C, van de Sande P, Van Heugten C. Assessment of apraxia: inter-rater reliability of a new apraxia test, association between apraxia and other cognitive deficits and prevalence of apraxia in a rehabilitation setting. *Clin Rehabil.* 2004;18:819–827.
 453. De Renzi E, Motti F, Nichelli P. Imitating gestures: a quantitative approach to ideomotor apraxia. *Arch Neurol.* 1980;37:6–10.
 454. Poeck K. The clinical examination for motor apraxia. *Neuropsychologia.* 1986;24:129–134.
 455. Feyereisen P, Barter D, Goossens M, Clerehugh N. Gestures and speech in referential communication by aphasic subjects: channel use and efficiency. *Aphasiology.* 1988;2:21–32.
 456. McDonald S, Tate RL, Rigby J. Error types in ideomotor apraxia: a qualitative analysis. *Brain Cogn.* 1994;25:250–270. doi: 10.1006/brcg.1994.1035.
 457. Blijlevens H, Hocking C, Paddy A. Rehabilitation of adults with dyspraxia: health professionals learning from patients. *Disabil Rehabil.* 2009;31:466–475. doi: 10.1080/09638280802131093.
 458. West C, Bowen A, Hesketh A, Vail A. Interventions for motor apraxia following stroke. *Cochrane Database Syst Rev.* 2008;CD004132. doi: 10.1002/14651858.CD004132.pub2.
 459. Dovern A, Fink GR, Weiss PH. Diagnosis and treatment of upper limb apraxia. *J Neurol.* 2012;259:1269–1283. doi: 10.1007/s00415-011-6336-y.
 460. Cicerone KD, Dahlberg C, Malec JF, Langenbahn DM, Felicetti T, Kneipp S, Ellmo W, Kalmar K, Giacino JT, Harley JP, Laatsch L, Morse PA, Catanese J. Evidence-based cognitive rehabilitation: updated review of the literature from 1998 through 2002. *Arch Phys Med Rehabil.* 2005;86:1681–1692.
 461. Geusgens CA, Winkens I, van Heugten CM, Jolles J, van den Heuvel WJ. Occurrence and measurement of transfer in cognitive rehabilitation: a critical review. *J Rehabil Med.* 2007;39:425–439. doi: 10.2340/16501977-0092.
 462. Wu AJ, Radel J, Hanna-Pladdy B. Improved function after combined physical and mental practice after stroke: a case of hemiparesis and apraxia. *Am J Occup Ther.* 2011;65:161–168.
 463. Corbetta M, Kincade MJ, Lewis C, Snyder AZ, Sapir A. Neural basis and recovery of spatial attention deficits in spatial neglect. *Nat Neurosci.* 2005;8:1603–1610. doi: 10.1038/nn1574.
 464. Kerkhoff G, Schenk T. Rehabilitation of neglect: an update. *Neuropsychologia.* 2012;50:1072–1079. doi: 10.1016/j.neuropsychologia.2012.01.024.
 465. Barrett AM, Buxbaum LJ, Coslett HB, Edwards E, Heilman KM, Hillis AE, Milberg WP, Robertson IH. Cognitive rehabilitation interventions for neglect and related disorders: moving from bench to bedside in stroke patients. *J Cogn Neurosci.* 2006;18:1223–1236. doi: 10.1162/jocn.2006.18.7.1223.
 466. Karnath HO, Rensing J, Johannsen L, Rorden C. The anatomy underlying acute versus chronic spatial neglect: a longitudinal study. *Brain.* 2011;134(pt 3):903–912. doi: 10.1093/brain/awq355.
 467. Rengachary J, He BJ, Shulman GL, Corbetta M. A behavioral analysis of spatial neglect and its recovery after stroke. *Front Hum Neurosci.* 2011;5:29. doi: 10.3389/fnhum.2011.00029.
 468. Bowen A, Lincoln N. Cognitive rehabilitation for spatial neglect following stroke (review). In: *The Cochrane Collaboration.* Vol 4. New York, NY: John Wiley & Sons, Ltd; 2008.
 469. Luauté J, Halligan P, Rode G, Rossetti Y, Boisson D. Visuo-spatial neglect: a systematic review of current interventions and their effectiveness. *Neurosci Biobehav Rev.* 2006;30:961–982. doi: 10.1016/j.neubiorev.2006.03.001.
 470. Yang NY, Zhou D, Chung RC, Li-Tsang CW, Fong KN. Rehabilitation interventions for unilateral neglect after stroke: a systematic review from 1997 through 2012. *Front Hum Neurosci.* 2013;7:187. doi: 10.3389/fnhum.2013.00187.
 471. Cazzoli D, Müri RM, Schumacher R, von Arx S, Chaves S, Gutbrod K, Bohlhalter S, Bauer D, Vanbellingen T, Bertschi M, Kipfer S, Rosenthal CR, Kennard C, Bassetti CL, Nyffeler T. Theta burst stimulation reduces disability during the activities of daily living in spatial neglect. *Brain.* 2012;135(pt 11):3426–3439. doi: 10.1093/brain/awr182.
 472. Fong KN, Yang NY, Chan MK, Chan DY, Lau AF, Chan DY, Cheung JT, Cheung HK, Chung RC, Chan CC. Combined effects of sensory cueing and limb activation on unilateral neglect in subacute left hemiplegic stroke patients: a randomized controlled pilot study. *Clin Rehabil.* 2013;27:628–637. doi: 10.1177/0269215512471959.
 473. Fortis P, Maravita A, Gallucci M, Ronchi R, Grassi E, Senna I, Olgiati E, Perucca L, Banco E, Posteraro L, Tesio L, Vallar G. Rehabilitating patients with left spatial neglect by prism exposure during a visuomotor activity. *Neuropsychologia.* 2010;24:681–697. doi: 10.1037/a0019476.
 474. Kerkhoff G, Keller I, Artinger F, Hildebrandt H, Marquardt C, Reinhart S, Ziegler W. Recovery from auditory and visual neglect after optokinetic stimulation with pursuit eye movements: transient modulation and enduring treatment effects. *Neuropsychologia.* 2012;50:1164–1177. doi: 10.1016/j.neuropsychologia.2011.09.032.
 475. Kim BR, Chun MH, Kim DY, Lee SJ. Effect of high- and low-frequency repetitive transcranial magnetic stimulation on visuospatial neglect in patients with acute stroke: a double-blind, sham-controlled trial. *Arch Phys Med Rehabil.* 2013;94:803–807. doi: 10.1016/j.apmr.2012.12.016.
 476. Lim JY, Kang EK, Paik NJ. Repetitive transcranial magnetic stimulation to hemispatial neglect in patients after stroke: an open-label pilot study. *J Rehabil Med.* 2010;42:447–452. doi: 10.2340/16501977-0553.
 477. Pizzamiglio L, Fasotti L, Jehkonen M, Antonucci G, Magnotti L, Boelen D, Asa S. The use of optokinetic stimulation in rehabilitation of the hemineglect disorder. *Cortex.* 2004;40:441–450.
 478. Polanowska K, Seniów J, Paprot E, Leśniak M, Członkowska A. Left-hand somatosensory stimulation combined with visual scanning training in rehabilitation for post-stroke hemineglect: a randomised, double-blind study. *Neuropsychol Rehabil.* 2009;19:364–382. doi: 10.1080/09602010802268856.
 479. Saevärrsson S, Kristjánsson A, Halsband U. Strength in numbers: combining neck vibration and prism adaptation produces additive therapeutic effects in unilateral neglect. *Neuropsychol Rehabil.* 2010;20:704–724. doi: 10.1080/09602011003737087.
 480. Song W, Du B, Xu Q, Hu J, Wang M, Luo Y. Low-frequency transcranial magnetic stimulation for visual spatial neglect: a pilot study. *J Rehabil Med.* 2009;41:162–165. doi: 10.2340/16501977-0302.
 481. Vangkilde S, Habekost T. Finding Wally: prism adaptation improves visual search in chronic neglect. *Neuropsychologia.* 2010;48:1994–2004. doi: 10.1016/j.neuropsychologia.2010.03.020.
 482. Welfringer A, Leifert-Fiebach G, Babinsky R, Brandt T. Visuomotor imagery as a new tool in the rehabilitation of neglect: a randomised controlled study of feasibility and efficacy. *Disabil Rehabil.* 2011;33:2033–2043. doi: 10.3109/09638288.2011.556208.
 483. Wu CY, Wang TN, Chen YT, Lin KC, Chen YA, Li HT, Tsai PL. Effects of constraint-induced therapy combined with eye patching on functional outcomes and movement kinematics in poststroke neglect. *Am J Occup Ther.* 2013;67:236–245. doi: 10.5014/ajot.2013.006486.
 484. Katz N, Ring H, Naveh Y, Kizony R, Feintuch U, Weiss PL. Interactive virtual environment training for safe street crossing of right hemisphere stroke patients with unilateral spatial neglect. *Disabil Rehabil.* 2005;27:1235–1243. doi: 10.1080/09638280500076079.
 485. Kim YM, Chun MH, Yun GJ, Song YJ, Young HE. The effect of virtual reality training on unilateral spatial neglect in stroke patients. *Ann Rehabil Med.* 2011;35:309–315. doi: 10.5535/arm.2011.35.3.309.
 486. Saevärrsson S, Kristjánsson A, Hildebrandt H, Halsband U. Prism adaptation improves visual search in hemispatial neglect. *Neuropsychologia.* 2009;47:717–725. doi: 10.1016/j.neuropsychologia.2008.11.026.
 487. Tsang MH, Sze KH, Fong KN. Occupational therapy treatment with right half-field eye-patching for patients with subacute stroke and unilateral neglect: a randomised controlled trial. *Disabil Rehabil.* 2009;31:630–637. doi: 10.1080/09638280802240621.
 488. Fong KN, Chan MK, Ng PP, Tsang MH, Chow KK, Lau CW, Chan FS, Wong IP, Chan DY, Chan CC. The effect of voluntary trunk rotation and half-field eye-patching for patients with unilateral neglect in stroke: a randomized controlled trial. *Clin Rehabil.* 2007;21:729–741. doi: 10.1177/0269215507076391.

489. Wilson B, Cockburn J, Halligan P. Development of a behavioral test of visuospatial neglect. *Arch Phys Med Rehabil*. 1987;68:98–102.
490. Tham K. The baking tray task: a test of spatial neglect. *Neuropsychol Rehabil*. 1996;6:19–26. doi: 10.1080/13755496.
491. Azouvi P, Olivier S, de Montety G, Samuel C, Louis-Dreyfus A, Tesio L. Behavioral assessment of unilateral neglect: study of the psychometric properties of the Catherine Bergego Scale. *Arch Phys Med Rehabil*. 2003;84:51–57. doi: 10.1053/apmr.2003.50062.
492. Keith RA, Granger CV, Hamilton BB, Sherwin FS. The functional independence measure: a new tool for rehabilitation. In: Eisenberg MG, Grzesiak RC, eds. *Advances in Clinical Rehabilitation*. Vol 2. New York: Springer; 1987:6–18.
493. Deleted in proof.
494. Tompkins CA. Rehabilitation for cognitive-communication disorders in right hemisphere brain damage. *Arch Phys Med Rehabil*. 2012;93(suppl):S61–S69. doi: 10.1016/j.apmr.2011.10.015.
495. Lehman Blake M, Frymark T, Venedictov R. An evidence-based systematic review on communication treatments for individuals with right hemisphere brain damage. *Am J Speech Lang Pathol*. 2013;22:146–160. doi: 10.1044/1058-0360(2012/12-0021).
496. Godecke E, Hird K, Lalor EE, Rai T, Phillips MR. Very early poststroke aphasia therapy: a pilot randomized controlled efficacy trial. *Int J Stroke*. 2012;7:635–644. doi: 10.1111/j.1747-4949.2011.00631.x.
497. Allen L, Mehta S, McClure JA, Teasell R. Therapeutic interventions for aphasia initiated more than six months post stroke: a review of the evidence. *Top Stroke Rehabil*. 2012;19:523–535. doi: 10.1310/tsr1906-523.
498. Moss A, Nicholas M. Language rehabilitation in chronic aphasia and time postonset: a review of single-subject data. *Stroke*. 2006;37:3043–3051. doi: 10.1161/01.STR.0000249427.74970.15.
499. Brady MC, Kelly H, Godwin J, Enderby P. Speech and language therapy for aphasia following stroke. *Cochrane Database Syst Rev*. 2012;5:CD000425. doi: 10.1002/14651858.CD000425.pub3.
500. Cherney LR, Patterson JP, Raymer A, Frymark T, Schooling T. Evidence-based systematic review: effects of intensity of treatment and constraint-induced language therapy for individuals with stroke-induced aphasia. *J Speech Lang Hear Res*. 2008;51:1282–1299. doi: 10.1044/1092-4388(2008/07-0206).
501. Cherney LR, Patterson JP, Raymer AM. Intensity of aphasia therapy: evidence and efficacy. *Curr Neurol Neurosci Rep*. 2011;11:560–569. doi: 10.1007/s11910-011-0227-6.
502. Sickert A, Anders LC, Münte TF, Sailer M. Constraint-induced aphasia therapy following sub-acute stroke: a single-blind, randomised clinical trial of a modified therapy schedule. *J Neurol Neurosurg Psychiatry*. 2014;85:51–55. doi: 10.1136/jnnp-2012-304297.
503. Bakheit AM, Shaw S, Barrett L, Wood J, Carrington S, Griffiths S, Searle K, Koutsi F. A prospective, randomized, parallel group, controlled study of the effect of intensity of speech and language therapy on early recovery from poststroke aphasia. *Clin Rehabil*. 2007;21:885–894. doi: 10.1177/0269215507078486.
504. Cherney LR, Erickson RK, Small SL. Epidural cortical stimulation as adjunctive treatment for non-fluent aphasia: preliminary findings. *J Neurol Neurosurg Psychiatry*. 2010;81:1014–1021. doi: 10.1136/jnnp.2009.184036.
505. Nobis-Bosch R, Springer L, Radermacher I, Huber W. Supervised home training of dialogue skills in chronic aphasia: a randomized parallel group study. *J Speech Lang Hear Res*. 2011;54:1118–1136. doi: 10.1044/1092-4388(2010/09-0204).
506. Palmer R, Enderby P, Cooper C, Latimer N, Julious S, Paterson G, Dimairo M, Dixon S, Mortley J, Hilton R, Delaney A, Hughes H. Computer therapy compared with usual care for people with long-standing aphasia poststroke: a pilot randomized controlled trial. *Stroke*. 2012;43:1904–1911. doi: 10.1161/STROKEAHA.112.650671.
507. Simmons-Mackie N, Raymer A, Armstrong E, Holland A, Cherney LR. Communication partner training in aphasia: a systematic review. *Arch Phys Med Rehabil*. 2010;91:1814–1837. doi: 10.1016/j.apmr.2010.08.026.
508. Lanyon LE, Rose ML, Worrall L. The efficacy of outpatient and community-based aphasia group interventions: a systematic review. *Int J Speech Lang Pathol*. 2013;15:359–374. doi: 10.3109/17549507.2012.752865.
509. Berthier ML, Green C, Higuera C, Fernández I, Hinojosa J, Martín MC. A randomized, placebo-controlled study of donepezil in post-stroke aphasia. *Neurology*. 2006;67:1687–1689. doi: 10.1212/01.wnl.0000242626.69666.e2.
510. Berthier ML, Green C, Lara JP, Higuera C, Barbancho MA, Dávila G, Pulvermüller F. Memantine and constraint-induced aphasia therapy in chronic poststroke aphasia. *Ann Neurol*. 2009;65:577–585. doi: 10.1002/ana.21597.
511. Hong JM, Shin DH, Lim TS, Lee JS, Huh K. Galantamine administration in chronic post-stroke aphasia. *J Neurol Neurosurg Psychiatry*. 2012;83:675–680. doi: 10.1136/jnnp-2012-302268.
512. Ashtary F, Janghorbani M, Chitsaz A, Reisi M, Bahrami A. A randomized, double-blind trial of bromocriptine efficacy in nonfluent aphasia after stroke. *Neurology*. 2006;66:914–916. doi: 10.1212/01.wnl.0000203119.91762.0c.
513. Güngör L, Terzi M, Onar MK. Does long term use of piracetam improve speech disturbances due to ischemic cerebrovascular diseases? *Brain Lang*. 2011;117:23–27. doi: 10.1016/j.bandl.2010.11.003.
514. Barwood CH, Murdoch BE, Whelan BM, Lloyd D, Riek S, O'Sullivan JD, Coulthard A, Wong A. Improved receptive and expressive language abilities in nonfluent aphasic stroke patients after application of rTMS: an open protocol case series. *Brain Stimul*. 2012;5:274–286. doi: 10.1016/j.brs.2011.03.005.
515. Barwood CH, Murdoch BE, Whelan BM, Lloyd D, Riek S, O'Sullivan JD, Coulthard A, Wong A. Improved language performance subsequent to low-frequency rTMS in patients with chronic non-fluent aphasia post-stroke. *Eur J Neurol*. 2011;18:935–943. doi: 10.1111/j.1468-1331.2010.03284.x.
516. Holland R, Crinion J. Can tDCS enhance treatment of aphasia after stroke? *Aphasiology*. 2012;26:1169–1191. doi: 10.1080/02687038.2011.616925.
517. Seniów J, Waldowski K, Leśniak M, Iwański S, Czepiel W, Członkowska A. Transcranial magnetic stimulation combined with speech and language training in early aphasia rehabilitation: a randomized double-blind controlled pilot study. *Top Stroke Rehabil*. 2013;20:250–261. doi: 10.1310/tsr2003-250.
518. Thiel A, Hartmann A, Rubi-Fessen I, Anglade C, Kracht L, Weiduschat N, Kessler J, Rommel T, Heiss WD. Effects of noninvasive brain stimulation on language networks and recovery in early poststroke aphasia. *Stroke*. 2013;44:2240–2246. doi: 10.1161/STROKEAHA.111.000574.
519. Warlow CP, Dennis MS, Van Gijn J, Hankey GJ, Sandercock PAG, Bamford JG, Wardlaw J, eds. *Stroke: A Practical Guide to Management*. Oxford, UK: Blackwell Scientific; 2000.
520. Mackenzie C. Dysarthria in stroke: a narrative review of its description and the outcome of intervention. *Int J Speech Lang Pathol*. 2011;13:125–136. doi: 10.3109/17549507.2011.524940.
521. Mackenzie C, Lowit A. Behavioural intervention effects in dysarthria following stroke: communication effectiveness, intelligibility and dysarthria impact. *Int J Lang Commun Disord*. 2007;42:131–153. doi: 10.1080/13682820600861776.
522. Wambaugh JL, Duffy JR, McNeil MR, Robin DA, Rogers MA. Treatment guidelines for acquired apraxia of speech: treatment descriptions and recommendations: second of two reports. *J Med Speech Lang Pathol*. 2006b;14:xxxv–Ixxvii.
523. Yorkston KM, Hakel M, Beukelman DR, Fager S. Evidence for effectiveness of treatment of loudness, rate, or prosody in dysarthria: a systematic review. *J Med Speech Lang Pathol*. 2007;15:XI–XXXVI.
524. Sellars C, Hughes T, Langhorne P. Speech and language therapy for dysarthria due to non-progressive brain damage. *Cochrane Database Syst Rev*. 2005;CD002088.
525. West C, Hesketh A, Vail A, Bowen A. Interventions for apraxia of speech following stroke. *Cochrane Database Syst Rev*. 2005;CD004298.
526. Wenke RJ, Theodoros D, Cornwell P. The short- and long-term effectiveness of the LSVT for dysarthria following TBI and stroke. *Brain Inj*. 2008;22:339–352. doi: 10.1080/02699050801960987.
527. Wambaugh JL, Duffy JR, McNeil MR, Robin DA, Rogers MA. Treatment guidelines for acquired apraxia of speech: a synthesis and evaluation of the evidence. *J Med Speech Lang Pathol*. 2006;14:35–37.
528. Wenke RJ, Theodoros D, Cornwell P. A comparison of the effects of the Lee Silverman voice treatment and traditional therapy on intelligibility, perceptual speech features, and everyday communication in nonprogressive dysarthria. *J Med Speech Lang Pathol*. 2011;19:1–24.
529. Palmer R, Enderby P, Hawley M. Addressing the needs of speakers with longstanding dysarthria: computerized and traditional therapy compared. *Int J Lang Commun Disord*. 2007;42(suppl 1):61–79. doi: 10.1080/13682820601173296.
530. Frankoff DJ, Hatfield B. Augmentative and alternative communication in daily clinical practice: strategies and tools for management of severe

- communication disorders. *Top Stroke Rehabil.* 2011;18:112–119. doi: 10.1310/tsr1802-112.
531. Hanson E, Yorkston K, Beukelman D. Speech supplementation techniques for dysarthria: a systematic review. *J Med Speech Lang Pathol.* 2004;12:IX–XXIX.
 532. Brady MC, Clark AM, Dickson S, Paton G, Barbour RS. The impact of stroke-related dysarthria on social participation and implications for rehabilitation. *Disabil Rehabil.* 2011;33:178–186. doi: 10.3109/09638288.2010.517897.
 533. Dickson S, Barbour RS, Brady M, Clark AM, Paton G. Patients' experiences of disruptions associated with post-stroke dysarthria. *Int J Lang Commun Disord.* 2008;43:135–153. doi: 10.1080/13682820701862228.
 534. Mackenzie C, Paton G, Kelly S, Brady M, Muir M. The Living With Dysarthria Group: implementation and feasibility of a group intervention for people with dysarthria following stroke and family members. *Int J Lang Commun Disord.* 2012;47:709–724. doi: 10.1111/j.1460-6984.2011.00180.x.
 535. Baylor C, Burns M, Eadie T, Britton D, Yorkston K. A qualitative study of interference with communicative participation across communication disorders in adults. *Am J Speech Lang Pathol.* 2011;20:269–287. doi: 10.1044/1058-0360.2011.010084.
 536. Dykstra AD, Hakel ME, Adams SG. Application of the ICF in reduced speech intelligibility in dysarthria. *Semin Speech Lang.* 2007;28:301–311. doi: 10.1055/s-2007-986527.
 537. Whitehill TL, Ma EPM, Tse FCM. Environmental barriers to communication for individuals with dysarthria. *J Med Speech Lang Pathol.* 2010;18:141–144.
 538. American Speech-Language Hearing Association. Speech-language pathologists providing clinical services via telepractice [position statement]. 2005. <http://www.asha.org/policy>. Accessed August 4, 2014.
 539. Doan QV, Brashear A, Gillard PJ, Varon SF, Vandenburgh AM, Turkel CC, Elovic EP. Relationship between disability and health-related quality of life and caregiver burden in patients with upper limb poststroke spasticity. *PM R.* 2012;4:4–10. doi: 10.1016/j.pmrj.2011.10.001.
 540. Lundström E, Smits A, Borg J, Terént A. Four-fold increase in direct costs of stroke survivors with spasticity compared with stroke survivors without spasticity: the first year after the event. *Stroke.* 2010;41:319–324. doi: 10.1161/STROKEAHA.109.558619.
 541. Shackley P, Shaw L, Price C, van Wijck F, Barnes M, Graham L, Ford GA, Steen N, Rodgers H. Cost-effectiveness of treating upper limb spasticity due to stroke with botulinum toxin type A: results from the Botulinum Toxin for the Upper Limb After Stroke (BoTULS) trial. *Toxins (Basel).* 2012;4:1415–1426.
 542. Moura Rde C, Fukujima MM, Aguiar AS, Fontes SV, Dauar RF, Prado GF. Predictive factors for spasticity among ischemic stroke patients. *Arg Neuropsychiatr.* 2009;67:1029–1036.
 543. Urban PP, Wolf T, Uebele M, Marx JJ, Vogt T, Stoeter P, Bauermann T, Weibrich C, Vucurevic GD, Schneider A, Wissel J. Occurrence and clinical predictors of spasticity after ischemic stroke. *Stroke.* 2010;41:2016–2020. doi: 10.1161/STROKEAHA.110.581991.
 544. Wissel J, Schelosky LD, Scott J, Christe W, Faiss JH, Mueller J. Early development of spasticity following stroke: a prospective, observational trial. *J Neurol.* 2010;257:1067–1072. doi: 10.1007/s00415-010-5463-1.
 545. Lundström E, Smits A, Terént A, Borg J. Time-course and determinants of spasticity during the first six months following first-ever stroke. *J Rehabil Med.* 2010;42:296–301. doi: 10.2340/16501977-0509.
 546. Ryu JS, Lee JW, Lee SI, Chun MH. Factors predictive of spasticity and their effects on motor recovery and functional outcomes in stroke patients. *Top Stroke Rehabil.* 2010;17:380–388. doi: 10.1310/tsr1705-380.
 547. Kong KH, Lee J, Chua KS. Occurrence and temporal evolution of upper limb spasticity in stroke patients admitted to a rehabilitation unit. *Arch Phys Med Rehabil.* 2012;93:143–148. doi: 10.1016/j.apmr.2011.06.027.
 548. Carda S, Invernizzi M, Baricich A, Cisari C. Casting, taping or stretching after botulinum toxin type A for spastic equinus foot: a single-blind randomized trial on adult stroke patients. *Clin Rehabil.* 2011;25:1119–1127. doi: 10.1177/0269215511405080.
 549. Karadag-Saygi E, Cubukcu-Aydogan K, Kablan N, Ofluoglu D. The role of kinesiotaping combined with botulinum toxin to reduce plantar flexors spasticity after stroke. *Top Stroke Rehabil.* 2010;17:318–322. doi: 10.1310/tsr1704-318.
 550. Sabut SK, Sikdar C, Kumar R, Mahadevappa M. Functional electrical stimulation of dorsiflexor muscle: effects on dorsiflexor strength, plantarflexor spasticity, and motor recovery in stroke patients. *NeuroRehabilitation.* 2011;29:393–400. doi: 10.3233/NRE-2011-0717.
 551. Caliendo P, Celletti C, Padua L, Minciotti I, Russo G, Granata G, La Torre G, Granieri E, Camerota F. Focal muscle vibration in the treatment of upper limb spasticity: a pilot randomized controlled trial in patients with chronic stroke. *Arch Phys Med Rehabil.* 2012;93:1656–1661. doi: 10.1016/j.apmr.2012.04.002.
 552. Noma T, Matsumoto S, Etoh S, Shimodono M, Kawahira K. Anti-spastic effects of the direct application of vibratory stimuli to the spastic muscles of hemiplegic limbs in post-stroke patients. *Brain Inj.* 2009;23:623–631. doi: 10.1080/02699050902997896.
 553. Noma T, Matsumoto S, Shimodono M, Etoh S, Kawahira K. Anti-spastic effects of the direct application of vibratory stimuli to the spastic muscles of hemiplegic limbs in post-stroke patients: a proof-of-principle study. *J Rehabil Med.* 2012;44:325–330. doi: 10.2340/16501977-0946.
 554. Department of Veterans Affairs, Department of Defense, American Heart Association/American Stroke Association. *VA/DoD Clinical Practice Guideline for the Management of Stroke Rehabilitation.* Washington, DC: Veterans Affairs/Department of Defense; 2010.
 555. Brainin M, Norrving B, Sunnerhagen KS, Goldstein LB, Cramer SC, Donnan GA, Duncan PW, Francisco G, Good D, Graham G, Kissela BM, Olver J, Ward A, Wissel J, Zorowitz R; International PSS Disability Study Group. Poststroke chronic disease management: towards improved identification and interventions for poststroke spasticity-related complications. *Int J Stroke.* 2011;6:42–46. doi: 10.1111/j.1747-4949.2010.00539.x.
 556. Olvey EL, Armstrong EP, Grizzle AJ. Contemporary pharmacologic treatments for spasticity of the upper limb after stroke: a systematic review. *Clin Ther.* 2010;32:2282–2303. doi: 10.1016/j.clinthera.2011.01.005.
 557. Teasell R, Foley N, Pereira S, Sequeira K, Miller T. Evidence to practice: botulinum toxin in the treatment of spasticity post stroke. *Top Stroke Rehabil.* 2012;19:115–121. doi: 10.1310/tsr1902-115.
 558. Foley N, Pereira S, Salter K, Fernandez MM, Speechley M, Sequeira K, Miller T, Teasell R. Treatment with botulinum toxin improves upper-extremity function post stroke: a systematic review and meta-analysis. *Arch Phys Med Rehabil.* 2013;94:977–989. doi: 10.1016/j.apmr.2012.12.006.
 559. Shaw LC, Price CI, van Wijck FM, Shackley P, Steen N, Barnes MP, Ford GA, Graham LA, Rodgers H; BoTULS Investigators. Botulinum Toxin for the Upper Limb after Stroke (BoTULS) Trial: effect on impairment, activity limitation, and pain. *Stroke.* 2011;42:1371–1379. doi: 10.1161/STROKEAHA.110.582197.
 560. Wolf SL, Milton SB, Reiss A, Easley KA, Shenvi NV, Clark PC. Further assessment to determine the additive effect of botulinum toxin type A on an upper extremity exercise program to enhance function among individuals with chronic stroke but extensor capability. *Arch Phys Med Rehabil.* 2012;93:578–587. doi: 10.1016/j.apmr.2011.10.026.
 561. Doan QV, Gillard P, Brashear A, Halperin M, Hayward E, Varon S, Lu ZJ. Cost-effectiveness of onabotulinumtoxinA for the treatment of wrist and hand disability due to upper-limb post-stroke spasticity in Scotland. *Eur J Neurol.* 2013;20:773–780. doi: 10.1111/ene.12062.
 562. Cousins E, Ward A, Roffe C, Rimington L, Pandyan A. Does low-dose botulinum toxin help the recovery of arm function when given early after stroke? A phase II randomized controlled pilot study to estimate effect size. *Clin Rehabil.* 2010;24:501–513. doi: 10.1177/0269215509358945.
 563. Rosales RL, Kong KH, Goh KJ, Kumthornthip W, Mok VC, Delgado-De Los Santos MM, Chua KS, Abdullah SJ, Zakine B, Maisonnobe P, Magis A, Wong KS. Botulinum toxin injection for hypertonicity of the upper extremity within 12 weeks after stroke: a randomized controlled trial. *Neurorehabil Neural Repair.* 2012;26:812–821. doi: 10.1177/1545968311430824.
 564. Kaji R, Osako Y, Suyama K, Maeda T, Uechi Y, Iwasaki M; GSK1358820 Spasticity Study Group. Botulinum toxin type A in post-stroke lower limb spasticity: a multicenter, double-blind, placebo-controlled trial [published correction appears in *J Neurol.* 2010;257:1416]. *J Neurol.* 2010;257:1330–1337. doi: 10.1007/s00415-010-5526-3.
 565. Santamato A, Micello MF, Panza F, Fortunato F, Pilotto A, Giustini A, Testa A, Fiore P, Ranieri M, Spidalieri R. Safety and efficacy of incobotulinum toxin type A (NT 201-Xeomin) for the treatment of post-stroke lower limb spasticity: a prospective open-label study. *Eur J Phys Rehabil Med.* 2013;49:483–489.
 566. Santamato A, Panza F, Ranieri M, Frisardi V, Micello MF, Filoni S, Fortunato F, Intiso D, Basciani M, Logroscino G, Fiore P. Efficacy and safety of higher doses of botulinum toxin type A NT 201 free from complexing proteins in the upper and lower limb spasticity after

- stroke. *J Neural Transm (Vienna)*. 2013;120:469–476. doi: 10.1007/s00702-012-0892-x.
567. Foley N, Murie-Fernandez M, Speechley M, Salter K, Sequeira K, Teasell R. Does the treatment of spastic equinovarus deformity following stroke with botulinum toxin increase gait velocity? A systematic review and meta-analysis. *Eur J Neurol*. 2010;17:1419–1427. doi: 10.1111/j.1468-1331.2010.03084.x.
 568. Tok F, Balaban B, Yaşar E, Alaca R, Tan AK. The effects of onabotulinum toxin A injection into rectus femoris muscle in hemiplegic stroke patients with stiff-knee gait: a placebo-controlled, nonrandomized trial. *Am J Phys Med Rehabil*. 2012;91:321–326. doi: 10.1097/PHM.0b013e3182465feb.
 569. Meythaler JM, Clayton W, Davis LK, Guin-Renfroe S, Brunner RC. Orally delivered baclofen to control spastic hypertonia in acquired brain injury. *J Head Trauma Rehabil*. 2004;19:101–108.
 570. Meythaler JM, Guin-Renfroe S, Johnson A, Brunner RM. Prospective assessment of tizanidine for spasticity due to acquired brain injury. *Arch Phys Med Rehabil*. 2001;82:1155–1163. doi: 10.1053/apmr.2001.25141.
 571. Chyatte SB, Birdsong JH, Bergman BA. The effects of dantrolene sodium on spasticity and motor performance in hemiplegia. *South Med J*. 1971;64:180–185.
 572. 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.
 573. Bes A, Eyssette M, Pierrot-Deseilligny E, Rohmer F, Warter JM. A multi-centre, double-blind trial of tizanidine, a new antispastic agent, in spasticity associated with hemiplegia. *Curr Med Res Opin*. 1988;10:709–718. doi: 10.1185/03007998809111122.
 574. Medici M, Pebet M, Ciblis D. A double-blind, long-term study of tizanidine (“Sirdalud”) in spasticity due to cerebrovascular lesions. *Curr Med Res Opin*. 1989;11:398–407. doi: 10.1185/03007998909110141.
 575. 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. doi: 10.1185/03007998409109576.
 576. 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.
 577. Medaer R, Hellbuyk H, Van Den Brande E, Saxena V, Thijs M, Kovacs L, Eerdekens M, Dehaen F. Treatment of spasticity due to stroke: a double-blind, cross-over trial comparing baclofen with placebo. *Acta Ther*. 1991;17:323–331.
 578. Meythaler JM, DeVivo MJ, Hadley M. Prospective study on the use of bolus intrathecal baclofen for spastic hypertonia due to acquired brain injury. *Arch Phys Med Rehabil*. 1996;77:461–466.
 579. Francisco GE, Boake C. Improvement in walking speed in poststroke spastic hemiplegia after intrathecal baclofen therapy: a preliminary study. *Arch Phys Med Rehabil*. 2003;84:1194–1199.
 580. Horn TS, Yablon SA, Stokic DS. Effect of intrathecal baclofen bolus injection on temporospatial gait characteristics in patients with acquired brain injury. *Arch Phys Med Rehabil*. 2005;86:1127–1133. doi: 10.1016/j.apmr.2004.11.013.
 581. Ivanhoe CB, Francisco GE, McGuire JR, Subramanian T, Grissom SP. Intrathecal baclofen management of poststroke spastic hypertonia: implications for function and quality of life. *Arch Phys Med Rehabil*. 2006;87:1509–1515. doi: 10.1016/j.apmr.2006.08.323.
 582. Rémy-Nérès O, Tiffreau V, Bouilland S, Bussel B. Intrathecal baclofen in subjects with spastic hemiplegia: assessment of the antispastic effect during gait. *Arch Phys Med Rehabil*. 2003;84:643–650.
 583. Francisco GE, Yablon SA, Schiess MC, Wiggs L, Cavalier S, Grissom S. Consensus panel guidelines for the use of intrathecal baclofen therapy in poststroke spastic hypertonia. *Top Stroke Rehabil*. 2006;13:74–85. doi: 10.1310/tsr1304-74.
 584. Davenport RJ, Dennis MS, Wellwood I, Warlow CP. Complications after acute stroke. *Stroke*. 1996;27:415–420.
 585. Forster A, Young J. Incidence and consequences of falls due to stroke: a systematic inquiry. *BMJ*. 1995;311:83–86.
 586. Pouwels S, Lalmohamed A, Leufkens B, de Boer A, Cooper C, van Staa T, de Vries F. Risk of hip/femur fracture after stroke: a population-based case-control study. *Stroke*. 2009;40:3281–3285. doi: 10.1161/STROKEAHA.109.554055.
 587. Yiu J, Miller WC, Eng JJ, Liu Y. Longitudinal analysis of balance confidence in individuals with stroke using a multilevel model for change. *Neurorehabil Neural Repair*. 2012;26:999–1006. doi: 10.1177/1545968312437941.
 588. Bronstein AM, Pavlou M. Balance. In: Barnes MP, Good DC, eds. *Handbook of Clinical Neurology, Neurological Rehabilitation*. New York, NY: Elsevier; 2013;110:189–208.
 589. Shumway-Cook A, Woolacott MH. *Motor Control: Translating Research Into Clinical Practice*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.
 590. Deleted in proof.
 591. Campbell GB, Matthews JT. An integrative review of factors associated with falls during post-stroke rehabilitation. *J Nurs Scholarsh*. 2010;42:395–404. doi: 10.1111/j.1547-5069.2010.01369.x.
 592. Lubetzky-Vilnai A, Kartin D. The effect of balance training on balance performance in individuals poststroke: a systematic review. *J Neurol Phys Ther*. 2010;34:127–137. doi: 10.1097/NPT.0b013e3181ef764d.
 593. Mehrholz J, Kugler J, Pohl M. Water-based exercises for improving activities of daily living after stroke. *Cochrane Database Syst Rev*. 2011;CD008186. doi: 10.1002/14651858.CD008186.pub2.
 594. Kim IC, Lee BH. Effects of augmented reality with functional electric stimulation on muscle strength, balance and gait of stroke patients. *J Phys Ther Sci*. 2012;24:755–762.
 595. Kim BH, Lee SM, Bae YH, Yu JH, Kim TH. The effect of a task-oriented training on trunk control ability, balance and gait of stroke patients. *J Phys Ther Sci*. 2012;24:519–522.
 596. Jung JC, Goo BO, Lee DH, Yu JH, Kim TH. Effects of 3D visual feedback exercise on the balance and walking abilities of hemiplegic patients. *J Phys Ther Sci*. 2011;23:859–862.
 597. Byun SD, Jung TD, Kim CH, Lee YS. Effects of the sliding rehabilitation machine on balance and gait in chronic stroke patients: a controlled clinical trial. *Clin Rehabil*. 2011;25:408–415.
 598. Karthikbabu S, Nayak A, Vijayakumar K, Misri Z, Suresh B, Ganesan S, Joshua AM. Comparison of physio ball and plinth trunk exercises regimens on trunk control and functional balance in patients with acute stroke: a pilot randomized controlled trial. *Clin Rehabil*. 2011;25:709–719.
 599. Lau KW, Mak MK. Speed-dependent treadmill training is effective to improve gait and balance performance in patients with sub-acute stroke. *J Rehabil Med*. 2011;43:709–713.
 600. Saeyns W, Vereeck L, Truijen S, Lafosse C, Wuyts FP, Heyning PV. Randomized controlled trial of truncal exercises early after stroke to improve balance and mobility. *Neurorehabil Neural Repair*. 2012;26:231–238.
 601. Schmid AA, Van Puymbroeck M, Altenburger PA, Schalk NL, Dierks TA, Miller KK, Damush TM, Bravata DM, Williams LS. Poststroke balance improves with yoga: a pilot study. *Stroke*. 2012;43:2402–2407. doi: 10.1161/STROKEAHA.112.658211.
 602. Aruin AS, Rao N, Sharma A, Chaudhuri G. Compelled body weight shift approach in rehabilitation of individuals with chronic stroke. *Top Stroke Rehabil*. 2012;19:556–563.
 603. Fisher S, Lucas L, Thrasher TA. Robot-assisted gait training for patients with hemiparesis due to stroke. *Top Stroke Rehabil*. 2011;18:269–276.
 604. Schuster C, Butler J, Andrews B, Kischka U, Ettlin T. Comparison of embedded and added motor imagery training in patients after stroke: results of a randomised controlled pilot trial. *Trials*. 2012;13:11.
 605. Tyson SF, Kent RM. Effects of an ankle-foot orthosis on balance and walking after stroke: a systematic review and pooled meta-analysis. *Arch Phys Med Rehabil*. 2013;94:1377–1385. doi: 10.1016/j.apmr.2012.12.025.
 606. Stoykov ME, Stojakovich M, Stevens JA. Beneficial effects of postural intervention on prehensile action for an individual with ataxia resulting from brainstem stroke. *NeuroRehabilitation*. 2005;20:85–89.
 607. Bastian AJ, Martin TA, Keating JG, Thach WT. Cerebellar ataxia: abnormal control of interaction torques across multiple joints. *J Neurophysiol*. 1996;76:492–509.
 608. Chua KS, Kong KH. Functional outcome in brain stem stroke patients after rehabilitation. *Arch Phys Med Rehabil*. 1996;77:194–197.
 609. Teasell R, Foley N, Doherty T, Finestone H. Clinical characteristics of patients with brainstem strokes admitted to a rehabilitation unit. *Arch Phys Med Rehabil*. 2002;83:1013–1016.
 610. Kelly PJ, Stein J, Shafqat S, Eskey C, Doherty D, Chang Y, Kurina A, Furie KL. Functional recovery after rehabilitation for cerebellar stroke. *Stroke*. 2001;32:530–534.
 611. Hatakenaka M, Miyai I, Mihara M, Yagura H, Hattori N. Impaired motor learning by a pursuit rotor test reduces functional outcomes

- during rehabilitation of poststroke ataxia. *Neurorehabil Neural Repair*. 2012;26:293–300. doi: 10.1177/1545968311412053.
612. Molinari M, Leggio MG, Solida A, Ciorra R, Misciagna S, Silveri MC, Petrosini L. Cerebellum and procedural learning: evidence from focal cerebellar lesions. *Brain*. 1997;120(pt 10):1753–1762.
 613. Richards L, Senesac C, McGuirk T, Woodbury M, Howland D, Davis S, Patterson T. Response to intensive upper extremity therapy by individuals with ataxia from stroke. *Top Stroke Rehabil*. 2008;15:262–271. doi: 10.1310/tsr1503-262.
 614. van de Port IG, Wood-Dauphinee S, Lindeman E, Kwakkel G. Effects of exercise training programs on walking competency after stroke: a systematic review. *Am J Phys Med Rehabil*. 2007;86:935–951.
 615. Veerbeek JM, Koolstra M, Ket JC, van Wegen EE, Kwakkel G. Effects of augmented exercise therapy on outcome of gait and gait-related activities in the first 6 months after stroke: a meta-analysis. *Stroke*. 2011;42:3311–3315. doi: 10.1161/STROKEAHA.111.623819.
 616. Langhorne P, Coupar F, Pollock A. Motor recovery after stroke: a systematic review. *Lancet Neurol*. 2009;8:741–754. doi: 10.1016/S1474-4422(09)70150-4.
 617. French B, Thomas LH, Leathley MJ, Sutton CJ, McAdam J, Forster A, Langhorne P, Price CI, Walker A, Watkins CL. Repetitive task training for improving functional ability after stroke. *Cochrane Database Syst Rev*. 2007;CD006073.
 618. Eng JJ, Tang PF. Gait training strategies to optimize walking ability in people with stroke: a synthesis of the evidence. *Expert Rev Neurother*. 2007;7:1417–1436. doi: 10.1586/14737175.7.10.1417.
 619. Dobkin BH, Duncan PW. Should body weight-supported treadmill training and robotic-assistive steppers for locomotor training trot back to the starting gate? *Neurorehabil Neural Repair*. 2012;26:308–317. doi: 10.1177/1545968312439687.
 620. Ada L, Dean CM, Lindley R. Randomized trial of treadmill training to improve walking in community-dwelling people after stroke: the AMBULATE trial. *Int J Stroke*. 2013;8:436–444. doi: 10.1111/j.1747-4949.2012.00934.x.
 621. Dickstein R. Rehabilitation of gait speed after stroke: a critical review of intervention approaches. *Neurorehabil Neural Repair*. 2008;22:649–660. doi: 10.1177/15459683080220060201.
 622. Dean CM, Rissel C, Sherrington C, Sharkey M, Cumming RG, Lord SR, RN, Kirkham C, O'Rourke S. Exercise to enhance mobility and prevent falls after stroke: the Community Stroke Club randomized trial. *Neurorehabil Neural Repair*. 2012;26:1046–1057. doi: 10.1177/1545968312441711.
 623. English C, Hillier S. Circuit class therapy for improving mobility after stroke: a systematic review. *J Rehabil Med*. 2011;43:565–571. doi: 10.2340/16501977-0824.
 624. Mudge S, Barber PA, Stott NS. Circuit-based rehabilitation improves gait endurance but not usual walking activity in chronic stroke: a randomized controlled trial. *Arch Phys Med Rehabil*. 2009;90:1989–1996. doi: 10.1016/j.apmr.2009.07.015.
 625. Wevers L, van de Port I, Vermue M, Mead G, Kwakkel G. Effects of task-oriented circuit class training on walking competency after stroke: a systematic review. *Stroke*. 2009;40:2450–2459. doi: 10.1161/STROKEAHA.108.541946.
 626. Polese JC, Ada L, Dean CM, Nascimento LR, Teixeira-Salmela LF. Treadmill training is effective for ambulatory adults with stroke: a systematic review. *J Physiother*. 2013;59:73–80. doi: 10.1016/S1836-9553(13)70159-0.
 627. Høyer E, Jahnsen R, Stanghelle JK, Strand LI. Body weight supported treadmill training versus traditional training in patients dependent on walking assistance after stroke: a randomized controlled trial. *Disabil Rehabil*. 2012;34:210–219. doi: 10.3109/09638288.2011.593681.
 628. Ada L, Dean CM, Vargas J, Ennis S. Mechanically assisted walking with body weight support results in more independent walking than assisted overground walking in non-ambulatory patients early after stroke: a systematic review. *J Physiother*. 2010;56:153–161.
 629. Mehta S, Pereira S, Viana R, Mays R, McIntyre A, Janzen S, Teasell RW. Resistance training for gait speed and total distance walked during the chronic stage of stroke: a meta-analysis. *Top Stroke Rehabil*. 2012;19:471–478. doi: 10.1310/tsr1906-471.
 630. Pak S, Patten C. Strengthening to promote functional recovery post-stroke: an evidence-based review. *Top Stroke Rehabil*. 2008;15:177–199. doi: 10.1310/tsr1503-177.
 631. Pereira S, Mehta S, McIntyre A, Lobo L, Teasell RW. Functional electrical stimulation for improving gait in persons with chronic stroke. *Top Stroke Rehabil*. 2012;19:491–498. doi: 10.1310/tsr1906-491.
 632. Robbins SM, Houghton PE, Woodbury MG, Brown JL. The therapeutic effect of functional and transcutaneous electric stimulation on improving gait speed in stroke patients: a meta-analysis. *Arch Phys Med Rehabil*. 2006;87:853–859. doi: 10.1016/j.apmr.2006.02.026.
 633. Daly JJ, Roenigk K, Holcomb J, Rogers JM, Butler K, Gansen J, McCabe J, Fredrickson E, Marsolais EB, Ruff RL. A randomized controlled trial of functional neuromuscular stimulation in chronic stroke subjects. *Stroke*. 2006;37:172–178. doi: 10.1161/01.STR.0000195129.95220.77.
 634. Ambrosini E, Ferrante S, Pedrocchi A, Ferrigno G, Molteni F. Cycling induced by electrical stimulation improves motor recovery in post-acute hemiparetic patients: a randomized controlled trial. *Stroke*. 2011;42:1068–1073. doi: 10.1161/STROKEAHA.110.599068.
 635. Sabut SK, Sikdar C, Mondal R, Kumar R, Mahadevappa M. Restoration of gait and motor recovery by functional electrical stimulation therapy in persons with stroke. *Disabil Rehabil*. 2010;32:1594–1603. doi: 10.3109/09638281003599596.
 636. Yamaguchi T, Tanabe S, Muraoka Y, Masakado Y, Kimura A, Tsuji T, Liu M. Immediate effects of electrical stimulation combined with passive locomotion-like movement on gait velocity and spasticity in persons with hemiparetic stroke: a randomized controlled study. *Clin Rehabil*. 2012;26:619–628. doi: 10.1177/0269215511426803.
 637. Yan T, Hui-Chan CW, Li LS. Functional electrical stimulation improves motor recovery of the lower extremity and walking ability of subjects with first acute stroke: a randomized placebo-controlled trial. *Stroke*. 2005;36:80–85. doi: 10.1161/01.STR.0000149623.24906.63.
 638. Everaert DG, Stein RB, Abrams GM, Dromerick AW, Francisco GE, Hafner BJ, Huskey TN, Munin MC, Nolan KJ, Kufta CV. Effect of a foot-drop stimulator and ankle-foot orthosis on walking performance after stroke: a multicenter randomized controlled trial. *Neurorehabil Neural Repair*. 2013;27:579–591. doi: 10.1177/1545968313481278.
 639. Ottawa Panel, Khadilkar A, Phillips K, Jean N, Lamothe C, Milne S, Sarnecka J. Ottawa Panel evidence-based clinical practice guidelines for post-stroke rehabilitation. *Top Stroke Rehabil*. 2006;13:1–269.
 640. Kluding PM, Dunning K, O'Dell MW, Wu SS, Ginosian J, Feld J, McBride K. Foot drop stimulation versus ankle foot orthosis after stroke: 30-week outcomes. *Stroke*. 2013;44:1660–1669. doi: 10.1161/STROKEAHA.111.000334.
 641. Kottink AI, Hermens HJ, Nene AV, Tenniglo MJ, Groothuis-Oudshoorn CG, IJzerman MJ. Therapeutic effect of an implantable peroneal nerve stimulator in subjects with chronic stroke and footdrop: a randomized controlled trial. *Phys Ther*. 2008;88:437–448. doi: 10.2522/ptj.20070035.
 642. Sheffler LR, Taylor PN, Gunzler DD, Buurke JH, IJzerman MJ, Chae J. Randomized controlled trial of surface peroneal nerve stimulation for motor relearning in lower limb hemiparesis. *Arch Phys Med Rehabil*. 2013;94:1007–1014. doi: 10.1016/j.apmr.2013.01.024.
 643. Sheffler LR, Hennessey MT, Naples GG, Chae J. Peroneal nerve stimulation versus an ankle foot orthosis for correction of footdrop in stroke: impact on functional ambulation. *Neurorehabil Neural Repair*. 2006;20:355–360. doi: 10.1177/1545968306287925.
 644. Chollet F, Tardy J, Albucher JF, Thalamez C, Berard E, Lamy C, Bejot Y, Deltour S, Jaillard A, Niclot P, Guillon B, Moulin T, Marque P, Pariente J, Arnaud C, Loubinoux I. Fluoxetine for motor recovery after acute ischaemic stroke (FLAME): a randomised placebo-controlled trial [published correction appears in *Lancet Neurol*. 2011;10:205]. *Lancet Neurol*. 2011;10:123–130. doi: 10.1016/S1474-4422(10)70314-8.
 645. 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.
 646. Fruehwald S, Gatterbauer E, Rehak P, Baumhackl U. Early fluoxetine treatment of post-stroke depression—a three-month double-blind placebo-controlled study with an open-label long-term follow up. *J Neurol*. 2003;250:347–351. doi: 10.1007/s00415-003-1014-3.
 647. Pariente J, Loubinoux I, Carel C, Albucher JF, Leger A, Manelfe C, Rascol O, Chollet F. Fluoxetine modulates motor performance and cerebral activation of patients recovering from stroke. *Ann Neurol*. 2001;50:718–729.
 648. Miyai I, Reding R. Effects of antidepressants on functional recovery following stroke: a double blind study. *J Neuro Rehabil*. 1998;12:5–13.
 649. Mead GE, Hsieh CF, Lee R, Kutlubaev M, Claxton A, Hankey GJ, Hackett M. Selective serotonin reuptake inhibitors for stroke recovery: a systematic review and meta-analysis. *Stroke*. 2013;44:844–850. doi: 10.1161/STROKEAHA.112.673947.

650. Martinsson L, Hardemark H, Eksborg S. Amphetamines for improving recovery after stroke. *Cochrane Database Syst Rev*. 2007;CD002090.
651. Scheidtmann K, Fries W, Müller 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. doi: 10.1016/S0140-6736(01)05966-9.
652. Shifflett SC. Does acupuncture work for stroke rehabilitation: what do recent clinical trials really show? *Top Stroke Rehabil*. 2007;14:40–58. doi: 10.1310/tsr1404-40.
653. Ng SS, Hui-Chan CW. Transcutaneous electrical nerve stimulation combined with task-related training improves lower limb functions in subjects with chronic stroke. *Stroke*. 2007;38:2953–2959. doi: 10.1161/STROKEAHA.107.490318.
654. Ng SS, Hui-Chan CW. Does the use of TENS increase the effectiveness of exercise for improving walking after stroke? A randomized controlled clinical trial. *Clin Rehabil*. 2009;23:1093–1103. doi: 10.1177/0269215509342327.
655. Tyson SF, Sadeghi-Demneh E, Nester CJ. The effects of transcutaneous electrical nerve stimulation on strength, proprioception, balance and mobility in people with stroke: a randomized controlled cross-over trial. *Clin Rehabil*. 2013;27:785–791. doi: 10.1177/0269215513478227.
656. Wittwer JE, Webster KE, Hill K. Rhythmic auditory cueing to improve walking in patients with neurological conditions other than Parkinson's disease: what is the evidence? *Disabil Rehabil*. 2013;35:164–176. doi: 10.3109/09638288.2012.690495.
657. Doğan A, Mengüllüoğlu M, Özgürin N. Evaluation of the effect of ankle-foot orthosis use on balance and mobility in hemiparetic stroke patients. *Disabil Rehabil*. 2011;33:1433–1439. doi: 10.3109/09638288.2010.533243.
658. Tyson S, Sadeghi-Demneh E, Nester C. A systematic review and meta-analysis of the effect of an ankle-foot orthosis on gait biomechanics after stroke. *Clin Rehabil*. 2013;27:879–891. doi: 10.1177/0269215513486497.
659. Tyson SF, Kent RM. Orthotic devices after stroke and other non-progressive brain lesions [retracted in *Cochrane Database Syst Rev*. 2009;CD003694] *Cochrane Database Syst Rev*. 2009;CD003694. doi: 10.1002/14651858.CD003694.pub3.
660. Thijssen DH, Paulus R, van Uden CJ, Kooloos JG, Hopman MT. Decreased energy cost and improved gait pattern using a new orthosis in persons with long-term stroke. *Arch Phys Med Rehabil*. 2007;88:181–186. doi: 10.1016/j.apmr.2006.11.014.
661. Mehrholz J, Elsner B, Werner C, Kugler J, Pohl M. Electromechanical-assisted training for walking after stroke: updated evidence. *Stroke*. 2013;44:e127–e128. doi: 10.1161/STROKEAHA.113.003061.
662. Hornby TG, Campbell DD, Kahn JH, Demott T, Moore JL, Roth HR. Enhanced gait-related improvements after therapist- versus robotic-assisted locomotor training in subjects with chronic stroke: a randomized controlled study [published correction appears in *Stroke*. 2008;39:e143]. *Stroke*. 2008;39:1786–1792. doi: 10.1161/STROKEAHA.107.504779.
663. Swinnen E, Beckwée D, Meeusen R, Baeyens JP, Kerckhofs E. Does robot-assisted gait rehabilitation improve balance in stroke patients? A systematic review. *Top Stroke Rehabil*. 2014;21:87–100. doi: 10.1310/tsr2102-87.
664. Stein J, Bishop L, Stein DJ, Wong CK. Gait training with a robotic leg brace after stroke: a randomized controlled pilot study. *Am J Phys Med Rehabil*. 2014;93:987–994. doi: 10.1097/PHM.0000000000000119.
665. Woodford H, Price C. EMG biofeedback for the recovery of motor function after stroke. *Cochrane Database Syst Rev*. 2007;CD004585.
666. Laver KE, George S, Thomas S, Deutsch JE, Crotty M. Virtual reality for stroke rehabilitation. *Cochrane Database Syst Rev*. 2011;CD008349. doi: 10.1002/14651858.CD008349.pub2.
667. Moreira MC, de Amorim Lima AM, Ferraz KM, Benedetti Rodrigues MA. Use of virtual reality in gait recovery among post stroke patients: a systematic literature review. *Disabil Rehabil Assist Technol*. 2013;8:357–362. doi: 10.3109/17483107.2012.749428.
668. Langhammer B, Stanghelle JK. Can physiotherapy after stroke based on the Bobath concept result in improved quality of movement compared to the motor relearning programme. *Physiother Res Int*. 2011;16:69–80. doi: 10.1002/prj.474.
669. Deleted in proof.
670. Nakayama H, Jørgensen HS, Raaschou HO, Olsen TS. Compensation in recovery of upper extremity function after stroke: the Copenhagen Stroke Study. *Arch Phys Med Rehabil*. 1994;75:852–857.
671. Gresham GE, Duncan PW, Stason WB, Adams HP, Adelman AM, Alexander DN, Bishop DS, Diller L, Donaldson NE, Granger CV. Post-stroke rehabilitation. *Clinical Practice Guideline*. Rockville, MD: US Department of Health and Human Services, Public Health Service, Agency for Healthcare Policy and Research; 1995.
672. Lang CE, Beebe JA. Relating movement control at 9 upper extremity segments to loss of hand function in people with chronic hemiparesis. *Neurorehabil Neural Repair*. 2007;21:279–291. doi: 10.1177/1545968306296964.
673. Faria-Fortini I, Michaelsen SM, Cassiano JG, Teixeira-Salmela LF. Upper extremity function in stroke subjects: relationships between the *International Classification of Functioning, Disability, and Health* domains. *J Hand Ther*. 2011;24:257–264; quiz 265.
674. Kwakkel G, Kollen BJ, van der Grond J, Prevo AJ. Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. *Stroke*. 2003;34:2181–2186. doi: 10.1161/01.STR.0000087172.16305.CD.
675. Bayona NA, Bitsensky J, Salter K, Teasell R. The role of task-specific training in rehabilitation therapies. *Top Stroke Rehabil*. 2005;12:58–65. doi: 10.1310/BQM5-6YGB-MVJ5-WVCR.
676. Hubbard JJ, Parsons MW, Neilson C, Carey LM. Task-specific training: evidence for and translation to clinical practice. *Occup Ther Int*. 2009;16:175–189. doi: 10.1002/oti.275.
677. Levin MF, Michaelsen SM, Cirstea CM, Roby-Brami A. Use of the trunk for reaching targets placed within and beyond the reach in adult hemiparesis. *Exp Brain Res*. 2002;143:171–180. doi: 10.1007/s00221-001-0976-6.
678. Wu CY, Chen YA, Lin KC, Chao CP, Chen YT. Constraint-induced therapy with trunk restraint for improving functional outcomes and trunk-arm control after stroke: a randomized controlled trial. *Phys Ther*. 2012;92:483–492. doi: 10.2522/ptj.20110213.
679. Corti M, McGuirk TE, Wu SS, Patten C. Differential effects of power training versus functional task practice on compensation and restoration of arm function after stroke. *Neurorehabil Neural Repair*. 2012;26:842–854. doi: 10.1177/1545968311433426.
680. Harris JE, Eng JJ. Strength training improves upper-limb function in individuals with stroke: a meta-analysis. *Stroke*. 2010;41:136–140. doi: 10.1161/STROKEAHA.109.567438.
681. Bonaiuti D, Rebasti L, Sioli P. The constraint induced movement therapy: a systematic review of randomised controlled trials on the adult stroke patients. *Eura Medicophys*. 2007;43:139–146.
682. Taub E, Miller NE, Novack TA, Cook EW 3rd, Fleming WC, Nepomuceno CS, Connell JS, Crago JE. Technique to improve chronic motor deficit after stroke. *Arch Phys Med Rehabil*. 1993;74:347–354.
683. Taub E, Uswatte G, King DK, Morris D, Crago JE, Chatterjee A. A placebo-controlled trial of constraint-induced movement therapy for upper extremity after stroke. *Stroke*. 2006;37:1045–1049. doi: 10.1161/01.STR.0000206463.66461.97.
684. Taub E, Uswatte G, Mark VW, Morris DM, Barman J, Bowman MH, Bryson C, Delgado A, Bishop-McKay S. Method for enhancing real-world use of a more affected arm in chronic stroke: transfer package of constraint-induced movement therapy. *Stroke*. 2013;44:1383–1388. doi: 10.1161/STROKEAHA.111.000559.
685. Wolf SL, Thompson PA, Winstein CJ, Miller JP, Blanton SR, Nichols-Larsen DS, Morris DM, Uswatte G, Taub E, Light KE, Sawaki L. The EXCITE stroke trial: comparing early and delayed constraint-induced movement therapy. *Stroke*. 2010;41:2309–2315. doi: 10.1161/STROKEAHA.110.588723.
686. Dromerick AW, Lang CE, Birkenmeier RL, Wagner JM, Miller JP, Videon TO, Powers WJ, Wolf SL, Edwards DF. Very Early Constraint-Induced Movement during Stroke Rehabilitation (VECTORS): a single-center RCT. *Neurology*. 2009;73:195–201. doi: 10.1212/WNL.0b013e3181ab2b27.
687. Boake C, Noser EA, Ro T, Baraniuk S, Gaber M, Johnson R, Salmeron ET, Tran TM, Lai JM, Taub E, Moye LA, Grotta JC, Levin HS. Constraint-induced movement therapy during early stroke rehabilitation. *Neurorehabil Neural Repair*. 2007;21:14–24. doi: 10.1177/1545968306291858.
688. Page SJ, Levine P, Leonard A, Szaflarski JP, Kissela BM. Modified constraint-induced therapy in chronic stroke: results of a single-blinded randomized controlled trial. *Phys Ther*. 2008;88:333–340. doi: 10.2522/ptj.20060029.
689. Page SJ, Levine P, Leonard AC. Modified constraint-induced therapy in acute stroke: a randomized controlled pilot study. *Neurorehabil Neural Repair*. 2005;19:27–32. doi: 10.1177/1545968304272701.

690. Page SJ, Sisto S, Johnston MV, Levine P. Modified constraint-induced therapy after subacute stroke: a preliminary study. *Neurorehabil Neural Repair*. 2002;16:290–295.
691. Page SJ, Sisto S, Levine P, McGrath RE. Efficacy of modified constraint-induced movement therapy in chronic stroke: a single-blinded randomized controlled trial. *Arch Phys Med Rehabil*. 2004;85:14–18.
692. Wang Q, Shao JL, Zhu QX, Li J, Meng PP. Comparison of conventional therapy, intensive therapy and modified constraint-induced movement therapy to improve upper extremity function after stroke. *J Rehabil Med*. 2011;43:619–625. doi: 10.2340/16501977-0819.
693. Shi YX, Tian JH, Yang KH, Zhao Y. Modified constraint-induced movement therapy versus traditional rehabilitation in patients with upper-extremity dysfunction after stroke: a systematic review and meta-analysis. *Arch Phys Med Rehabil*. 2011;92:972–982. doi: 10.1016/j.apmr.2010.12.036.
694. Smania N, Gandolfi M, Paolucci S, Iosa M, Ianes P, Recchia S, Giovanzana C, Molteni F, Avesani R, Di Paolo P, Zaccala M, Agostini M, Tassorelli C, Fiaschi A, Primon D, Ceravolo MG, Farina S. Reduced-intensity modified constraint-induced movement therapy versus conventional therapy for upper extremity rehabilitation after stroke: a multicenter trial. *Neurorehabil Neural Repair*. 2012;26:1035–1045. doi: 10.1177/1545968312446003.
695. Coupar F, Pollock A, van Wijck F, Morris J, Langhorne P. Simultaneous bilateral training for improving arm function after stroke. *Cochrane Database Syst Rev*. 2010;CD006432. doi: 10.1002/14651858.CD006432.pub2.
696. Latimer CP, Keeling J, Lin B, Henderson M, Hale LA. The impact of bilateral therapy on upper limb function after chronic stroke: a systematic review. *Disabil Rehabil*. 2010;32:1221–1231. doi: 10.3109/09638280903483877.
697. Cauraugh JH, Naik SK, Lodha N, Coombes SA, Summers JJ. Long-term rehabilitation for chronic stroke arm movements: a randomized controlled trial. *Clin Rehabil*. 2011;25:1086–1096.
698. Morris JH, Van Wijck F. Responses of the less affected arm to bilateral upper limb task training in early rehabilitation after stroke: a randomized controlled trial. *Arch Phys Med Rehabil*. 2012;93:1129–1137.
699. Whittall J, Waller SM, Sorkin JD, Forrester LW, Macko RF, Hanley DF, Goldberg AP, Luft A. Bilateral and unilateral arm training improve motor function through differing neuroplastic mechanisms: a single-blinded randomized controlled trial. *Neurorehabil Neural Repair*. 2011;25:118–129. doi: 10.1177/1545968310380685.
700. Wu CY, Chuang LL, Lin KC, Chen HC, Tsay PK. Randomized trial of distributed constraint-induced therapy versus bilateral arm training for the rehabilitation of upper-limb motor control and function after stroke. *Neurorehabil Neural Repair*. 2011;25:130–139. doi: 10.1177/1545968310380686.
701. Hayner K, Gibson G, Giles GM. Comparison of constraint-induced movement therapy and bilateral treatment of equal intensity in people with chronic upper-extremity dysfunction after cerebrovascular accident. *Am J Occup Ther*. 2010;64:528–539.
702. Brunner IC, Skouen JS, Strand LI. Is modified constraint-induced movement therapy more effective than bimanual training in improving arm motor function in the subacute phase post stroke? A randomized controlled trial. *Clin Rehabil*. 2012;26:1078–1086.
703. Mehrholz J, Pohl M. Electromechanical-assisted gait training after stroke: a systematic review comparing end-effector and exoskeleton devices. *J Rehabil Med*. 2012;44:193–199. doi: 10.2340/16501977-0943.
704. Klamroth-Marganska V, Blanco J, Campen K, Curt A, Dietz V, Ettlin T, Felder M, Fellinghauer B, Guidali M, Kollmar A, Luft A, Nef T, Schuster-Amft C, Stahl W, Riener R. Three-dimensional, task-specific robot therapy of the arm after stroke: a multicentre, parallel-group randomised trial. *Lancet Neurol*. 2014;13:159–166. doi: 10.1016/S1474-4422(13)70305-3.
705. Lo AC, Guarino PD, Richards LG, Haselkorn JK, Wittenberg GF, Federman DG, Ringer RJ, Wagner TH, Krebs HI, Volpe BT, Bever CT Jr, Bravata DM, Duncan PW, Corn BH, Maffucci AD, Nadeau SE, Conroy SS, Powell JM, Huang GD, Peduzzi P. Robot-assisted therapy for long-term upper-limb impairment after stroke [published correction appears in *N Engl J Med*. 2011;365:1749]. *N Engl J Med*. 2010;362:1772–1783. doi: 10.1056/NEJMoa0911341.
706. Lo AC, Guarino P, Krebs HI, Volpe BT, Bever CT, Duncan PW, Ringer RJ, Wagner TH, Richards LG, Bravata DM, Haselkorn JK, Wittenberg GF, Federman DG, Corn BH, Maffucci AD, Peduzzi P. Multicenter randomized trial of robot-assisted rehabilitation for chronic stroke: methods and entry characteristics for VA ROBOTICS. *Neurorehabil Neural Repair*. 2009;23:775–783. doi: 10.1177/1545968309338195.
707. Mehrholz J, Hadrach A, Platz T, Kugler J, Pohl M. Electromechanical and robot-assisted arm training for improving generic activities of daily living, arm function, and arm muscle strength after stroke. *Cochrane Database Syst Rev*. 2012;6:CD006876. doi: 10.1002/14651858.CD006876.pub3.
708. Masiero S, Armani M, Rosati G. Upper-limb robot-assisted therapy in rehabilitation of acute stroke patients: focused review and results of new randomized controlled trial. *J Rehabil Res Dev*. 2011;48:355–366.
709. Kwakkel G, Kollen BJ, Krebs HI. Effects of robot-assisted therapy on upper limb recovery after stroke: a systematic review. *Neurorehabil Neural Repair*. 2008;22:111–121. doi: 10.1177/1545968307305457.
710. Kutner NG, Zhang R, Butler AJ, Wolf SL, Alberts JL. Quality-of-life change associated with robotic-assisted therapy to improve hand motor function in patients with subacute stroke: a randomized clinical trial. *Phys Ther*. 2010;90:493–504. doi: 10.2522/ptj.20090160.
711. Hsieh YW, Wu CY, Liao WW, Lin KC, Wu KY, Lee CY. Effects of treatment intensity in upper limb robot-assisted therapy for chronic stroke: a pilot randomized controlled trial. *Neurorehabil Neural Repair*. 2011;25:503–511. doi: 10.1177/1545968310394871.
712. Conroy SS, Whittall J, Dipietro L, Jones-Lush LM, Zhan M, Finley MA, Wittenberg GF, Krebs HI, Bever CT. Effect of gravity on robot-assisted motor training after chronic stroke: a randomized trial. *Arch Phys Med Rehabil*. 2011;92:1754–1761. doi: 10.1016/j.apmr.2011.06.016.
713. Abdullah HA, Tarry C, Lambert C, Barreca S, Allen BO. Results of clinicians using a therapeutic robotic system in an inpatient stroke rehabilitation unit. *J Neuroeng Rehabil*. 2011;8:50. doi: 10.1186/1743-0003-8-50.
714. Pomeroy VM, King LM, Pollock A, Baily-Hallam A, Langhorne P. Electrostimulation for promoting recovery of movement or functional ability after stroke: systematic review and meta-analysis. *Cochrane Database Syst Rev*. 2006;(2):CD003241.
715. Alon G, Levitt AF, McCarthy PA. Functional electrical stimulation (FES) may modify the poor prognosis of stroke survivors with severe motor loss of the upper extremity: a preliminary study. *Am J Phys Med Rehabil*. 2008;87:627–636. doi: 10.1097/PHM.0b013e31817fab1.
716. Hara Y, Ogawa S, Tsujiuchi K, Muraoka Y. A home-based rehabilitation program for the hemiplegic upper extremity by power-assisted functional electrical stimulation. *Disabil Rehabil*. 2008;30:296–304.
717. Van Peppen RP, Kwakkel G, Wood-Dauphinee S, Hendriks HJ, Van der Wees PJ, Dekker J. The impact of physical therapy on functional outcomes after stroke: what's the evidence? *Clin Rehabil*. 2004;18:833–862.
718. Butler AJ, Page SJ. Mental practice with motor imagery: evidence for motor recovery and cortical reorganization after stroke. *Arch Phys Med Rehabil*. 2006;87(suppl 2):S2–S11. doi: 10.1016/j.apmr.2006.08.326.
719. Page SJ, Levine P, Leonard A. Mental practice in chronic stroke: results of a randomized, placebo-controlled trial. *Stroke*. 2007;38:1293–1297. doi: 10.1161/01.STR.0000260205.67348.2b.
720. Page SJ, Levine P, Sisto SA, Johnston MV. Mental practice combined with physical practice for upper-limb motor deficit in subacute stroke. *Phys Ther*. 2001;81:1455–1462.
721. Liu KP, Chan CC, Lee TM, Hui-Chan CW. Mental imagery for promoting relearning for people after stroke: a randomized controlled trial. *Arch Phys Med Rehabil*. 2004;85:1403–1408.
722. Liu KP, Chan CC, Wong RS, Kwan IW, Yau CS, Li LS, Lee TM. A randomized controlled trial of mental imagery augment generalization of learning in acute poststroke patients. *Stroke*. 2009;40:2222–2225. doi: 10.1161/STROKEAHA.108.540997.
723. Bovennd Eerd TJ, Dawes H, Sackley C, Izadi H, Wade DT. An integrated motor imagery program to improve functional task performance in neurorehabilitation: a single-blind randomized controlled trial. *Arch Phys Med Rehabil*. 2010;91:939–946. doi: 10.1016/j.apmr.2010.03.008.
724. Page SJ, Dunning K, Hermann V, Leonard A, Levine P. Longer versus shorter mental practice sessions for affected upper extremity movement after stroke: a randomized controlled trial. *Clin Rehabil*. 2011;25:627–637. doi: 10.1177/0269215510395793.
725. Kowalczyk J, Chong SL, Galea M, Prochazka A. In-home tele-rehabilitation improves tetraplegic hand function. *Neurorehabil Neural Repair*. 2011;25:412–422. doi: 10.1177/1545968310394869.
726. Deleted in proof.
727. Clinical Trials.gov. Efficacy of Virtual Reality Exercises in Stroke Rehabilitation: A Multicentre Study (EVREST Multicentre). <https://clinicaltrials.gov/ct2/show/NCT01406912?term=NCT01406912&rank=1>. Accessed August 27, 2015.

728. Kiper P, Piron L, Turolla A, Stozek J, Tonin P. The effectiveness of reinforced feedback in virtual environment in the first 12 months after stroke. *Neurol Neurochir Pol*. 2011;45:436–444.
729. da Silva Cameirao M, Bermudez I, Badia S, Duarte E, Verschure PF. Virtual reality based rehabilitation speeds up functional recovery of the upper extremities after stroke: a randomized controlled pilot study in the acute phase of stroke using the rehabilitation gaming system. *Restor Neurol Neurosci*. 2011;29:287–298. doi: 10.3233/RNN-2011-0599.
730. Deleted in proof.
731. van Vliet PM, Wulf G. Extrinsic feedback for motor learning after stroke: what is the evidence? *Disabil Rehabil*. 2006;28:831–840. doi: 10.1080/09638280500534937.
732. Subramanian SK, Massie CL, Malcolm MP, Levin MF. Does provision of extrinsic feedback result in improved motor learning in the upper limb poststroke? A systematic review of the evidence. *Neurorehabil Neural Repair*. 2010;24:113–124. doi: 10.1177/1545968309349941.
733. Molier BI, Van Asseldonk EH, Hermens HJ, Jannink MJ. Nature, timing, frequency and type of augmented feedback; does it influence motor relearning of the hemiparetic arm after stroke? A systematic review. *Disabil Rehabil*. 2010;32:1799–1809. doi: 10.3109/09638281003734359.
734. Celnik P, Hummel F, Harris-Love M, Wolk R, Cohen LG. Somatosensory stimulation enhances the effects of training functional hand tasks in patients with chronic stroke. *Arch Phys Med Rehabil*. 2007;88:1369–1376.
735. Hunter SM, Hammett L, Ball S, Smith N, Anderson C, Clark A, Tallis R, Rudd A, Pomeroy VM. Dose-response study of mobilisation and tactile stimulation therapy for the upper extremity early after stroke: a phase I trial. *Neurorehabil Neural Repair*. 2011;25:314–322. doi: 10.1177/1545968310390223.
736. Kliaiput A, Kitisomprayoonkul W. Increased pinch strength in acute and subacute stroke patients after simultaneous median and ulnar sensory stimulation. *Neurorehabil Neural Repair*. 2009;23:351–356. doi: 10.1177/1545968308324227.
737. Bowen A, Knapp P, Gillespie D, Nicolson DJ, Vail A. Non-pharmacological interventions for perceptual disorders following stroke and other adult-acquired, non-progressive brain injury. *Cochrane Database Syst Rev*. 2011;CD007039. doi: 10.1002/14651858.CD007039.pub2.
738. Sullivan JE, Hurley D, Hedman LD. Afferent stimulation provided by glove electrode during task-specific arm exercise following stroke. *Clin Rehabil*. 2012;26:1010–1020.
739. Pomeroy V, Aglioti SM, Mark VW, McFarland D, Stinear C, Wolf SL, Corbett M, Fitzpatrick SM. Neurological principles and rehabilitation of action disorders: rehabilitation interventions. *Neurorehabil Neural Repair*. 2011;25(suppl):33S–43S. doi: 10.1177/1545968311410942.
740. Chang WH, Kim YH, Bang OY, Kim ST, Park YH, Lee PK. Long-term effects of rTMS on motor recovery in patients after subacute stroke. *J Rehabil Med*. 2010;42:758–764. doi: 10.2340/16501977-0590.
741. Kim DY, Lim JY, Kang EK, You DS, Oh MK, Oh BM, Paik NJ. Effect of transcranial direct current stimulation on motor recovery in patients with subacute stroke. *Am J Phys Med Rehabil*. 2010;89:879–886. doi: 10.1097/PHM.0b013e3181f70aa7.
742. Lindenberg R, Renga V, Zhu LL, Nair D, Schlaug G. Bihemispheric brain stimulation facilitates motor recovery in chronic stroke patients. *Neurology*. 2010;75:2176–2184. doi: 10.1212/WNL.0b013e318202013a.
743. Nair DG, Renga V, Lindenberg R, Zhu L, Schlaug G. Optimizing recovery potential through simultaneous occupational therapy and non-invasive brain-stimulation using tDCS. *Restor Neurol Neurosci*. 2011;29:411–420. doi: 10.3233/RNN-2011-0612.
744. Hesse S, Waldner A, Mehrholz J, Tomelleri C, Pohl M, Werner C. Combined transcranial direct current stimulation and robot-assisted arm training in subacute stroke patients: an exploratory, randomized multicenter trial. *Neurorehabil Neural Repair*. 2011;25:838–846. doi: 10.1177/1545968311413906.
745. Bolognini N, Vallar G, Casati C, Latif LA, El-Nazer R, Williams J, Banco E, Macea DD, Tesio L, Chessa C, Fregni F. Neurophysiological and behavioral effects of tDCS combined with constraint-induced movement therapy in poststroke patients. *Neurorehabil Neural Repair*. 2011;25:819–829. doi: 10.1177/1545968311411056.
746. Avenanti A, Coccia M, Ladavas E, Provinciali L, Ceravolo MG. Low-frequency rTMS promotes use-dependent motor plasticity in chronic stroke: a randomized trial. *Neurology*. 2012;78:256–264. doi: 10.1212/WNL.0b013e3182436558.
747. Deleted in proof.
748. Winter J, Hunter S, Sim J, Crome P. Hands-on therapy interventions for upper limb motor dysfunction following stroke. *Cochrane Database Syst Rev*. 2011;CD006609. doi: 10.1002/14651858.CD006609.pub2.
749. Coupar F, Pollock A, Legg LA, Sackley C, van Vliet P. Home-based therapy programmes for upper limb functional recovery following stroke. *Cochrane Database Syst Rev*. 2012;5:CD006755. doi: 10.1002/14651858.CD006755.pub2.
750. Chaiyawat P, Kulkantrakorn K. Effectiveness of home rehabilitation program for ischemic stroke upon disability and quality of life: a randomized controlled trial. *Clin Neurol Neurosurg*. 2012;114:866–870. doi: 10.1016/j.clineuro.2012.01.018.
751. United Nations. Convention on the Rights of Persons with Disabilities. <http://www.un.org/disabilities/convention/facts.shtml>. 2006. Accessed March 5, 2016.
752. Legg LA, Drummond AE, Langhorne P. Occupational therapy for patients with problems in activities of daily living after stroke. *Cochrane Database Syst Rev*. 2006;CD003585.
753. Jutai J, Coulson S, Teasell R, Bayley M, Garland J, Mayo N, Wood-Dauphinee S. Mobility assistive device utilization in a prospective study of patients with first-ever stroke. *Arch Phys Med Rehabil*. 2007;88:1268–1275. doi: 10.1016/j.apmr.2007.06.773.
754. Polese JC, Teixeira-Salmela LF, Nascimento LR, Faria CD, Kirkwood RN, Laurentino GC, Ada L. The effects of walking sticks on gait kinematics and kinetics with chronic stroke survivors. *Clin Biomech (Bristol, Avon)*. 2012;27:131–137. doi: 10.1016/j.clinbiomech.2011.08.003.
755. Tyson SF, Rogerson L. Assistive walking devices in nonambulant patients undergoing rehabilitation after stroke: the effects on functional mobility, walking impairments, and patients' opinion. *Arch Phys Med Rehabil*. 2009;90:475–479. doi: 10.1016/j.apmr.2008.09.563.
756. Laufer Y. Effects of one-point and four-point canes on balance and weight distribution in patients with hemiparesis. *Clin Rehabil*. 2002;16:141–148.
757. Mountain AD, Kirby RL, MacLeod DA, Thompson K. Rates and predictors of manual and powered wheelchair use for persons with stroke: a retrospective study in a Canadian rehabilitation center. *Arch Phys Med Rehabil*. 2010;91:639–643. doi: 10.1016/j.apmr.2009.11.025.
758. Rehabilitation Engineering and Assistive Technology Society of North America. RESNA Wheelchair Service Provision Guide. 2011. <http://www.resna.org/dotAsset/22485.pdf>. Accessed June 28, 2013.
759. Barrett JA, Watkins C, Plant R, Dickinson H, Clayton L, Sharma AK, Reston A, Gratton J, Fall S, Flynn A, Smith T, Leathley M, Smith S, Barer DH. The COSTAR wheelchair study: a two-centre pilot study of self-propulsion in a wheelchair in early stroke rehabilitation: Collaborative Stroke Audit and Research. *Clin Rehabil*. 2001;15:32–41.
760. Mountain AD, Kirby RL, Eskes GA, Smith C, Duncan H, MacLeod DA, Thompson K. Ability of people with stroke to learn powered wheelchair skills: a pilot study. *Arch Phys Med Rehabil*. 2010;91:596–601. doi: 10.1016/j.apmr.2009.12.011.
761. Barker DJ, Reid D, Cott C. The experience of senior stroke survivors: factors in community participation among wheelchair users. *Can J Occup Ther*. 2006;73:18–25.
762. Pettersson I, Ahlström G, Törnquist K. The value of an outdoor powered wheelchair with regard to the quality of life of persons with stroke: a follow-up study. *Assist Technol*. 2007;19:143–153. doi: 10.1080/10400435.2007.10131871.
763. Erel S, Uygur F, Engin Simsek I, Yakut Y. The effects of dynamic ankle-foot orthoses in chronic stroke patients at three-month follow-up: a randomized controlled trial. *Clin Rehabil*. 2011;25:515–523. doi: 10.1177/0269215510390719.
764. de Sèze MP, Bonhomme C, Daviet JC, Burguete E, Machat H, Rousseaux M, Mazaux JM. Effect of early compensation of distal motor deficiency by the Chignon ankle-foot orthosis on gait in hemiplegic patients: a randomized pilot study. *Clin Rehabil*. 2011;25:989–998. doi: 10.1177/0269215511410730.
765. Smith AC, Saunders DH, Mead G. Cardiorespiratory fitness after stroke: a systematic review. *Int J Stroke*. 2012;7:499–510. doi: 10.1111/j.1747-4949.2012.00791.x.
766. Shephard RJ. Maximal oxygen intake and independence in old age. *Br J Sports Med*. 2009;43:342–346. doi: 10.1136/bjism.2007.044800.
767. Touzé E, Varenne O, Chatellier G, Peyrard S, Rothwell PM, Mas JL. Risk of myocardial infarction and vascular death after transient ischemic attack and ischemic stroke: a systematic review and meta-analysis. *Stroke*. 2005;36:2748–2755. doi: 10.1161/01.STR.0000190118.02275.33.
768. Burn J, Dennis M, Bamford J, Sandercock P, Wade D, Warlow C. Long-term risk of recurrent stroke after a first-ever stroke: the Oxfordshire

- Community Stroke Project [published correction appears in *Stroke*. 1994;25:1887]. *Stroke*. 1994;25:333–337.
769. Dhamoon MS, Sciacca RR, Rundek T, Sacco RL, Elkind MS. Recurrent stroke and cardiac risks after first ischemic stroke: the Northern Manhattan Study. *Neurology*. 2006;66:641–646. doi: 10.1212/01.wnl.0000201253.93811.f6.
 770. Leoo T, Lindgren A, Petersson J, von Arbin M. Risk factors and treatment at recurrent stroke onset: results from the Recurrent Stroke Quality and Epidemiology (RESQUE) Study. *Cerebrovasc Dis*. 2008;25:254–260. doi: 10.1159/000113864.
 771. Hartman-Maeir A, Soroker N, Ring H, Avni N, Katz N. Activities, participation and satisfaction one-year post stroke. *Disabil Rehabil*. 2007;29:559–566. doi: 10.1080/09638280600924996.
 772. Hildebrand M, Brewer M, Wolf T. The impact of mild stroke on participation in physical fitness activities. *Stroke Res Treat*. 2012;2012:548682. doi: 10.1155/2012/548682.
 773. Wellwood I, Langhorne P, McKevitt C, Bernhardt J, Rudd AG, Wolfe CD. An observational study of acute stroke care in four countries: the European Registers of Stroke Study. *Cerebrovasc Dis*. 2009;28:171–176. doi: 10.1159/000226116.
 774. MacKay-Lyons MJ, Makrides L. Cardiovascular stress during a contemporary stroke rehabilitation program: is the intensity adequate to induce a training effect? *Arch Phys Med Rehabil*. 2002;83:1378–1383.
 775. Kuys S, Brauer S, Ada L. Routine physiotherapy does not induce a cardiorespiratory training effect post-stroke, regardless of walking ability. *Physiother Res Int*. 2006;11:219–227.
 776. Kaur G, English C, Hillier S. How physically active are people with stroke in physiotherapy sessions aimed at improving motor function? A systematic review. *Stroke Res Treat*. 2012;2012:820673. doi: 10.1155/2012/820673.
 777. Manns PJ, Tomczak CR, Jelani A, Cress ME, Haennel R. Use of the Continuous Scale Physical Functional Performance Test in stroke survivors. *Arch Phys Med Rehabil*. 2009;90:488–493. doi: 10.1016/j.apmr.2008.08.219.
 778. Alzahrani MA, Ada L, Dean CM. Duration of physical activity is normal but frequency is reduced after stroke: an observational study. *J Physiother*. 2011;57:47–51. doi: 10.1016/S1836-9553(11)70007-8.
 779. Ashe MC, Miller WC, Eng JJ, Noreau L; Physical Activity and Chronic Conditions Research Team. Older adults, chronic disease and leisure-time physical activity. *Gerontology*. 2009;55:64–72. doi: 10.1159/000141518.
 780. Resnick B, Michael K, Shaughnessy M, Nahm ES, Kobunek S, Sorkin J, Orwig D, Goldberg A, Macko RF. Inflated perceptions of physical activity after stroke: pairing self-report with physiologic measures. *J Phys Act Health*. 2008;5:308–318.
 781. Dogra S, Stathokostas L. Sedentary behavior and physical activity are independent predictors of successful aging in middle-aged and older adults. *J Aging Res*. 2012;2012:190654. doi: 10.1155/2012/190654.
 782. Healy GN, Dunstan DW, Salmon J, Cerin E, Shaw JE, Zimmet PZ, Owen N. Breaks in sedentary time: beneficial associations with metabolic risk. *Diabetes Care*. 2008;31:661–666. doi: 10.2337/dc07-2046.
 783. Fletcher GF, Ades PA, Kligfield P, Arena R, Balady GJ, Bittner VA, Coke LA, Fleg JL, Forman DE, Gerber TC, Gulati M, Madan K, Rhodes J, Thompson PD, Williams MA; on behalf of the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee of the Council on Clinical Cardiology, Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular and Stroke Nursing, and Council on Epidemiology and Prevention. Exercise standards for testing and training: a scientific statement from the American Heart Association. *Circulation*. 2013;128:873–934. doi: 10.1161/CIR.0b013e31829b5b44.
 784. Stoller O, de Bruin ED, Knols RH, Hunt KJ. Effects of cardiovascular exercise early after stroke: systematic review and meta-analysis. *BMC Neurol*. 2012;12:45. doi: 10.1186/1471-2377-12-45.
 785. Pang MY, Eng JJ, Dawson AS, Gylfadóttir S. The use of aerobic exercise training in improving aerobic capacity in individuals with stroke: a meta-analysis. *Clin Rehabil*. 2006;20:97–111.
 786. Brazzelli M, Saunders DH, Greig CA, Mead GE. Physical fitness training for patients with stroke: updated review. *Stroke*. 2012;43:e39–e40. doi: 10.1161/STROKEAHA.111.647008.
 787. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Bassett DR Jr, Tudor-Locke C, Greer JL, Vezina J, Whitt-Glover MC, Leon AS. 2011 Compendium of Physical Activities: a second update of codes and MET values. *Med Sci Sports Exerc*. 2011;43:1575–1581. doi: 10.1249/MSS.0b013e31821ee12.
 788. Duncan P, Studenski S, Richards L, Gollub S, Lai SM, Reker D, Perera S, Yates J, Koch V, Rigler S, Johnson D. Randomized clinical trial of therapeutic exercise in subacute stroke. *Stroke*. 2003;34:2173–2180. doi: 10.1161/01.STR.0000083699.95351.F2.
 789. Mackay-Lyons M, McDonald A, Matheson J, Eskes G, Klus MA. Dual effects of body-weight supported treadmill training on cardiovascular fitness and walking ability early after stroke: a randomized controlled trial. *Neurorehabil Neural Repair*. 2013;27:644–653. doi: 10.1177/1545968313484809.
 790. Hambrecht R, Walther C, Möbius-Winkler S, Gielen S, Linke A, Conradi K, Erbs S, Kluge R, Kendziorra K, Sabri O, Sick P, Schuler G. Percutaneous coronary angioplasty compared with exercise training in patients with stable coronary artery disease: a randomized trial. *Circulation*. 2004;109:1371–1378. doi: 10.1161/01.CIR.0000121360.31954.1F.
 791. Myers J. Physical activity: the missing prescription. *Eur J Cardiovasc Prev Rehabil*. 2005;12:85–86.
 792. Pang MY, Lau RW. The effects of treadmill exercise training on hip bone density and tibial bone geometry in stroke survivors: a pilot study. *Neurorehabil Neural Repair*. 2010;24:368–376. doi: 10.1177/1545968309353326.
 793. Deleted in proof.
 794. Graven C, Brock K, Hill K, Joubert L. Are rehabilitation and/or care co-ordination interventions delivered in the community effective in reducing depression, facilitating participation and improving quality of life after stroke? *Disabil Rehabil*. 2011;33:1501–1520. doi: 10.3109/09638288.2010.542874.
 795. Lai SM, Studenski S, Richards L, Perera S, Reker D, Rigler S, Duncan PW. Therapeutic exercise and depressive symptoms after stroke. *J Am Geriatr Soc*. 2006;54:240–247. doi: 10.1111/j.1532-5415.2006.00573.x.
 796. Deleted in proof.
 797. Mehta S, Pereira S, Janzen S, Mays R, Viana R, Lobo L, Teasell RW. Cardiovascular conditioning for comfortable gait speed and total distance walked during the chronic stage of stroke: a meta-analysis. *Top Stroke Rehabil*. 2012;19:463–470. doi: 10.1310/tsr1906-463.
 798. Deleted in proof.
 799. Schönberger M, Hansen NR, Pedersen DT, Zeeman P, Jørgensen JR. The relationship between physical fitness and work integration following stroke. *Brain Impairment*. 2010;11:262–269.
 800. Chen MD, Rimmer JH. Effects of exercise on quality of life in stroke survivors: a meta-analysis. *Stroke*. 2011;42:832–837. doi: 10.1161/STROKEAHA.110.607747.
 801. Ivey FM, Ryan AS, Hafer-Macko CE, Goldberg AP, Macko RF. Treadmill aerobic training improves glucose tolerance and indices of insulin sensitivity in disabled stroke survivors: a preliminary report. *Stroke*. 2007;38:2752–2758. doi: 10.1161/STROKEAHA.107.490391.
 802. Takatori K, Matsumoto D, Okada Y, Nakamura J, Shomoto K. Effect of intensive rehabilitation on physical function and arterial function in community-dwelling chronic stroke survivors. *Top Stroke Rehabil*. 2012;19:377–383. doi: 10.1310/tsr1905-377.
 803. Rimmer JH, Rauworth AE, Wang EC, Nicola TL, Hill B. A preliminary study to examine the effects of aerobic and therapeutic (nonaerobic) exercise on cardiorespiratory fitness and coronary risk reduction in stroke survivors. *Arch Phys Med Rehabil*. 2009;90:407–412. doi: 10.1016/j.apmr.2008.07.032.
 804. Billinger SA, Mattlage AE, Ashenden AL, Lentz AA, Harter G, Rippee MA. Aerobic exercise in subacute stroke improves cardiovascular health and physical performance. *J Neurol Phys Ther*. 2012;36:159–165. doi: 10.1097/NPT.0b013e318274d082.
 805. Hackam DG, Spence JD. Combining multiple approaches for the secondary prevention of vascular events after stroke: a quantitative modeling study. *Stroke*. 2007;38:1881–1885. doi: 10.1161/STROKEAHA.106.475525.
 806. Mackay-Lyons M, Thornton M, Ruggles T, Che M. Non-pharmacological interventions for preventing secondary vascular events after stroke or transient ischemic attack. *Cochrane Database Syst Rev*. 2013;3:CD008656.
 807. Lennon O, Carey A, Gaffney N, Stephenson J, Blake C. A pilot randomized controlled trial to evaluate the benefit of the cardiac rehabilitation paradigm for the non-acute ischaemic stroke population. *Clin Rehabil*. 2008;22:125–133. doi: 10.1177/0269215507081580.
 808. Prior PL, Hachinski V, Unsworth K, Chan R, Mytka S, O'Callaghan C, Suskin N. Comprehensive cardiac rehabilitation for secondary prevention after transient ischemic attack or mild stroke, I: feasibility and risk factors. *Stroke*. 2011;42:3207–3213. doi: 10.1161/STROKEAHA.111.620187.

809. Cumming TB, Thrift AG, Collier JM, Churilov L, Dewey HM, Donnan GA, Bernhardt J. Very early mobilization after stroke fast-tracks return to walking: further results from the phase II AVERT randomized controlled trial. *Stroke*. 2011;42:153–158. doi: 10.1161/STROKEAHA.110.594598.
810. Sundseth A, Thommesen B, Rønning OM. Outcome after mobilization within 24 hours of acute stroke: a randomized controlled trial. *Stroke*. 2012;43:2389–2394. doi: 10.1161/STROKEAHA.111.646687.
811. Fihn SD, Blankenship JC, Alexander KP, Bittl JA, Byrne JG, Fletcher BJ, Fonarow GC, Lange RA, Levine GN, Maddox TM, Naidu SS, Ohman EM, Smith PK. 2014 ACC/AHA/AATS/PCNA/SCAI/STS focused update of the guideline for the diagnosis and management of patients with stable ischemic heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines, and the American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *Circulation*. 2014;130:1749–1767. doi: 10.1161/CIR.0000000000000095.
812. Kwan G, Balady GJ. Cardiac rehabilitation 2012: advancing the field through emerging science. *Circulation*. 2012;125:e369–e373. doi: 10.1161/CIRCULATIONAHA.112.093310.
813. Billinger SA, Arena R, Bernhardt J, Eng JJ, Franklin BA, Johnson CM, MacKay-Lyons M, Macko RF, Mead GE, Roth EJ, Shaughnessy M, Tang A; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular and Stroke Nursing; Council on Lifestyle and Cardiometabolic Health; Council on Epidemiology and Prevention; Council on Clinical Cardiology. Physical activity and exercise recommendations for stroke survivors: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2014;45:2532–2553. doi: 10.1161/STR.0000000000000022.
814. Management of Stroke Rehabilitation Working Group. VA/DOD clinical practice guideline for the management of stroke rehabilitation. *J Rehabil Res Dev*. 2010;47:1–43.
815. Ivey FM, Hafer-Macko CE, Macko RF. Exercise training for cardiometabolic adaptation after stroke. *J Cardiopulm Rehabil Prev*. 2008;28:2–11. doi: 10.1097/01.HCR.0000311501.57022.a8.
816. American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription*. 9th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2013.
817. Deleted in proof.
818. Danielsson A, Willén C, Sunnerhagen KS. Measurement of energy cost by the physiological cost index in walking after stroke. *Arch Phys Med Rehabil*. 2007;88:1298–1303. doi: 10.1016/j.apmr.2007.06.760.
819. Eng JJ, Dawson AS, Chu KS. Submaximal exercise in persons with stroke: test-retest reliability and concurrent validity with maximal oxygen consumption. *Arch Phys Med Rehabil*. 2004;85:113–118.
820. Hurkmans HL, Ribbers GM, Streur-Kranenburg MF, Stam HJ, van den Berg-Emons RJ. Energy expenditure in chronic stroke patients playing Wii Sports: a pilot study. *J Neuroeng Rehabil*. 2011;8:38. doi: 10.1186/1743-0003-8-38.
821. Chang WH, Kim MS, Huh JP, Lee PK, Kim YH. Effects of robot-assisted gait training on cardiopulmonary fitness in subacute stroke patients: a randomized controlled study. *Neurorehabil Neural Repair*. 2012;26:318–324. doi: 10.1177/1545968311408916.
822. Stewart KJ, Bacher AC, Turner KL, Fleg JL, Hees PS, Shapiro EP, Tayback M, Ouyang P. Effect of exercise on blood pressure in older persons: a randomized controlled trial [published correction appears in *Arch Intern Med*. 2006;166:1813]. *Arch Intern Med*. 2005;165:756–762. doi: 10.1001/archinte.165.7.756.
823. Stewart KJ, Bacher AC, Turner K, Lim JG, Hees PS, Shapiro EP, Tayback M, Ouyang P. Exercise and risk factors associated with metabolic syndrome in older adults. *Am J Prev Med*. 2005;28:9–18. doi: 10.1016/j.amepre.2004.09.006.
824. Deleted in proof.
825. Hill TR, Gjellesvik TI, Moen PM, Tørhaug T, Fimland MS, Helgerud J, Hoff J. Maximal strength training enhances strength and functional performance in chronic stroke survivors. *Am J Phys Med Rehabil*. 2012;91:393–400. doi: 10.1097/PHM.0b013e31824ad5b8.
826. Durstine JL. *ACSM's Exercise Management for Persons With Chronic Diseases and Disabilities*. Champaign, IL: Human Kinetics; 2009.
827. Gordon NF, Gulianick M, Costa F, Fletcher G, Franklin BA, Roth EJ, Shephard T. Physical activity and exercise recommendations for stroke survivors: an American Heart Association scientific statement from the Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention; the Council on Cardiovascular Nursing; the Council on Nutrition, Physical Activity, and Metabolism; and the Stroke Council. *Circulation*. 2004;109:2031–2041. doi: 10.1161/01.CIR.0000126280.65777.A4.
828. Swain DP, Franklin BA. VO(2) reserve and the minimal intensity for improving cardiorespiratory fitness. *Med Sci Sports Exerc*. 2002;34:152–157.
829. Globas C, Becker C, Cerny J, Lam JM, Lindemann U, Forrester LW, Macko RF, Luft AR. Chronic stroke survivors benefit from high-intensity aerobic treadmill exercise: a randomized control trial. *Neurorehabil Neural Repair*. 2012;26:85–95. doi: 10.1177/1545968311418675.
830. Gjellesvik TI, Brurok B, Hoff J, Tørhaug T, Helgerud J. Effect of high aerobic intensity interval treadmill walking in people with chronic stroke: a pilot study with one year follow-up. *Top Stroke Rehabil*. 2012;19:353–360. doi: 10.1310/tsr1904-353.
831. Bruce RA, Kusumi F, Hosmer D. Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J*. 1973;85:546–562.
832. Rimmer JH, Wang E, Smith D. Barriers associated with exercise and community access for individuals with stroke. *J Rehabil Res Dev*. 2008;45:315–322.
833. Nicholson S, Sniehotta F, Van Wijck F, Greig CA, Johnston M, McMurdo MET, Dennis M, Mead GE. A systematic review of perceived barriers and motivators to physical activity after stroke. *Int J Stroke*. 2013;8:357–364. doi: 10.1111/j.1747-4949.2012.00880.x.
834. Morris J, Oliver T, Kroll T, MacGillivray S. The importance of psychological and social factors in influencing the uptake and maintenance of physical activity after stroke: a structured review of the empirical literature. *Stroke Res Treatment*. 2012;2012:195249. doi: 10.1155/2012/195249.
835. Holman H, Lorig K. Patient self-management: a key to effectiveness and efficiency in care of chronic disease. *Public Health Rep*. 2004;119:239–243. doi: 10.1016/j.phr.2004.04.002.
836. van Veenendaal H, Grinspun DR, Adriaanse HP. Educational needs of stroke survivors and their family members, as perceived by themselves and by health professionals. *Patient Educ Couns*. 1996;28:265–276.
837. Simpson LA, Eng JJ, Tawashy AE. Exercise perceptions among people with stroke: barriers and facilitators to participation. *Int J Ther Rehabil*. 2011;18:520–530.
838. Damush TM, Plue L, Bakas T, Schmid A, Williams LS. Barriers and facilitators to exercise among stroke survivors. *Rehabil Nurs*. 2007;32:253–260, 262.
839. Banks G, Bernhardt J, Churilov L, Cumming TB. Exercise preferences are different after stroke. *Stroke Res Treat*. 2012;2012:890946. doi: 10.1155/2012/890946.
840. van der Ploeg HP, Streppel KR, van der Beek AJ, van der Woude LH, Vollenbroek-Hutten MM, van Harten WH, van Mechelen W. Successfully improving physical activity behavior after rehabilitation. *Am J Health Promot*. 2007;21:153–159.
841. Boysen G, Krarup LH, Zeng X, Oskebra A, Körv J, Andersen G, Gluud C, Pedersen A, Lindahl M, Hansen L, Winkel P, Truelsen T; ExStroke Pilot Trial Group. ExStroke Pilot Trial of the effect of repeated instructions to improve physical activity after ischaemic stroke: a multinational randomised controlled clinical trial. *BMJ*. 2009;339:b2810.
842. Jones F, Mandy A, Partridge C. Changing self-efficacy in individuals following a first time stroke: preliminary study of a novel self-management intervention. *Clin Rehabil*. 2009;23:522–533. doi: 10.1177/0269215508101749.
843. Joubert J, Reid C, Barton D, Cumming T, McLean A, Joubert L, Barlow J, Ames D, Davis S. Integrated care improves risk-factor modification after stroke: initial results of the Integrated Care for the Reduction of Secondary Stroke model. *J Neurol Neurosurg Psychiatry*. 2009;80:279–284. doi: 10.1136/jnnp.2008.148122.
844. Barrett BT. A critical evaluation of the evidence supporting the practice of behavioural vision therapy. *Ophthalmic Physiol Opt*. 2009;29:4–25.
845. Deleted in proof.
846. Pollock A, Hazelton C, Henderson CA, Angilly J, Dhillon B, Langhorne P, Livingstone K, Munro FA, Orr H, Rowe FJ, Shahani U. Interventions for disorders of eye movement in patients with stroke. *Cochrane Database Syst Rev*. 2011;CD008389. doi: 10.1002/14651858.CD008389.pub2.
847. Riggs RV, Andrews K, Roberts P, Gilewski M. Visual deficit interventions in adult stroke and brain injury: a systematic review. *Am J Phys Med Rehabil*. 2007;86:853–860. doi: 10.1097/PHM.0b013e318151f907.

848. Keller I, Lefin-Rank G. Improvement of visual search after audiovisual exploration training in hemianopic patients. *Neurorehabil Neural Repair*. 2010;24:666–673. doi: 10.1177/1545968310372774.
849. Kihoon J, Jaeho Y, Jinhwa J. Effects of virtual reality-based rehabilitation on upper extremity function and visual perception in stroke patients: a randomized control trial. *J Phys Ther Sci*. 2012;24:1205–1208.
850. Mödden C, Behrens M, Damke I, Eilers N, Kastrup A, Hildebrandt H. A randomized controlled trial comparing 2 interventions for visual field loss with standard occupational therapy during inpatient stroke rehabilitation. *Neurorehabil Neural Repair*. 2012;26:463–469. doi: 10.1177/1545968311425927.
851. O'Halloran R, Worrall LE, Hickson L. The number of patients with communication related impairments in acute hospital stroke units. *Int J Speech Lang Pathol*. 2009;11:438–449. doi: 10.3109/17549500902741363.
852. Edwards DF, Hahn MG, Baum CM, Perlmuter MS, Sheedy C, Dromerick AW. Screening patients with stroke for rehabilitation needs: validation of the post-stroke rehabilitation guidelines. *Neurorehabil Neural Repair*. 2006;20:42–48. doi: 10.1177/1545968305283038.
853. Allison R, Shelling L, Dennett R, Ayers T, Evans PH, Campbell JL. The effectiveness of various models of primary care-based follow-up after stroke: a systematic review. *Prim Health Care Res Dev*. 2011;12:214–222. doi: 10.1017/S146342361100003X.
854. Prvu Bettger J, Alexander KP, Dolor RJ, Olson DM, Kendrick AS, Wing L, Coeytaux RR, Graffagnino C, Duncan PW. Transitional care after hospitalization for acute stroke or myocardial infarction: a systematic review. *Ann Intern Med*. 2012;157:407–416. doi: 10.7326/0003-4819-157-6-201209180-00004.
855. Shepperd S, Lannin NA, Clemson LM, McCluskey A, Cameron ID, Barras SL. Discharge planning from hospital to home. *Cochrane Database Syst Rev*. 2013;1:CD000313. doi: 10.1002/14651858.CD000313.pub4.
856. Lutz BJ, Chumbler NR, Lyles T, Hoffman N, Kobb R. Testing a home-telehealth programme for US veterans recovering from stroke and their family caregivers. *Disabil Rehabil*. 2009;31:402–409. doi: 10.1080/09638280802069558.
857. Oupra R, Griffiths R, Pryor J, Mott S. Effectiveness of Supportive Educative Learning programme on the level of strain experienced by caregivers of stroke patients in Thailand. *Health Soc Care Community*. 2010;18:10–20. doi: 10.1111/j.1365-2524.2009.00865.x.
858. Choi-Kwon S, Mitchell PH, Veith R, Teri L, Buzaitis A, Cain KC, Becker KJ, Tirschwell D, Fruin M, Choi J, Kim JS. Comparing perceived burden for Korean and American informal caregivers of stroke survivors. *Rehabil Nurs*. 2009;34:141–150.
859. Smith SD, Gignac MA, Richardson D, Cameron JI. Differences in the experiences and support needs of family caregivers to stroke survivors: does age matter? *Top Stroke Rehabil*. 2008;15:593–601. doi: 10.1310/tsr1506-593.
860. Perrin PB, Johnston A, Vogel B, Heesacker M, Vega-Trujillo M, Anderson J, Rittman M. A culturally sensitive Transition Assistance Program for stroke caregivers: examining caregiver mental health and stroke rehabilitation. *J Rehabil Res Dev*. 2010;47:605–617.
861. Levine C, Albert SM, Hokenstad A, Halper DE, Hart AY, Gould DA. "This case is closed": family caregivers and the termination of home health care services for stroke patients. *Milbank Q*. 2006;84:305–331. doi: 10.1111/j.1468-0009.2006.00449.x.
862. Salter K, Foley N, Teasell R. Social support interventions and mood status post stroke: a review. *Int J Nurs Stud*. 2010;47:616–625. doi: 10.1016/j.ijnurstu.2009.12.002.
863. Lurbe-Puerto K, Leandro ME, Baumann M. Experiences of caregiving, satisfaction of life, and social repercussions among family caregivers, two years post-stroke. *Soc Work Health Care*. 2012;51:725–742. doi: 10.1080/00981389.2012.692351.
864. Steiner V, Pierce L, Drachusak S, Nofziger E, Buchman D, Szirony T. Emotional support, physical help, and health of caregivers of stroke survivors. *J Neurosci Nurs*. 2008;40:48–54.
865. Campos de Oliveira B, Garanhani ML, Garanhani MR. Caregivers of people with stroke: needs, feeling and guidelines provided. *Acta Paulista de Enfermagem*. 2011;24:43–49.
866. Thomas M, Greenop K. Caregiver experiences and perceptions of stroke. *Health SA Gesondheid*. 2008;13:29–40.
867. Visser-Meily A, van Heugten C, Post M, Schepers V, Lindeman E. Intervention studies for caregivers of stroke survivors: a critical review. *Patient Educ Couns*. 2005;56:257–267. doi: 10.1016/j.pec.2004.02.013.
868. White JH, Alston MK, Marquez JL, Sweetapple AL, Pollack MR, Attia J, Levi CR, Sturm J, Whyte S. Community-dwelling stroke survivors: function is not the whole story with quality of life. *Arch Phys Med Rehabil*. 2007;88:1140–1146. doi: 10.1016/j.apmr.2007.06.003.
869. White CL, Komer-Bitensky N, Rodrigue N, Rosmus C, Sourial R, Lambert S, Wood-Dauphinee S. Barriers and facilitators to caring for individuals with stroke in the community: the family's experience. *Can J Neurosci Nurs*. 2007;29:5–12.
870. Philp I, Brainin M, Walker MF, Ward AB, Gillard P, Shields AL, Norrving B; Global Stroke Community Advisory Panel. Development of a poststroke checklist to standardize follow-up care for stroke survivors. *J Stroke Cerebrovasc Dis*. 2013;22:e173–e180. doi: 10.1016/j.jstrokecerebrovasdis.2012.10.016.
871. Forster A, Brown L, Smith J, House A, Knapp P, Wright JJ, Young J. Information provision for stroke patients and their caregivers. *Cochrane Database Syst Rev*. 2012;11:CD001919. doi: 10.1002/14651858.CD001919.pub3.
872. Teasell R, Mehta S, Pereira S, McIntyre A, Janzen S, Allen L, Lobo L, Viana R. Time to rethink long-term rehabilitation management of stroke patients. *Top Stroke Rehabil*. 2012;19:457–462. doi: 10.1310/tsr1906-457.
873. Ferrarello F, Baccini M, Rinaldi LA, Cavallini MC, Mossello E, Masotti G, Marchionni N, Di Bari M. Efficacy of physiotherapy interventions late after stroke: a meta-analysis. *J Neurol Neurosurg Psychiatry*. 2011;82:136–143. doi: 10.1136/jnnp.2009.196428.
874. Lennon O, Galvin R, Smith K, Doody C, Blake C. Lifestyle interventions for secondary disease prevention in stroke and transient ischaemic attack: a systematic review. *Eur J Prev Cardiol*. 2014;21:1026–1039. doi: 10.1177/2047487313481756.
875. Pavey TG, Taylor AH, Fox KR, Hillsdon M, Anokye N, Campbell JL, Foster C, Green C, Moxham T, Mutrie N, Searle J, Trueman P, Taylor RS. Effect of exercise referral schemes in primary care on physical activity and improving health outcomes: systematic review and meta-analysis. *BMJ*. 2011;343:d6462.
876. Sharma H, Bulley C, van Wijck FM. Experiences of an exercise referral scheme from the perspective of people with chronic stroke: a qualitative study. *Physiotherapy*. 2012;98:336–343. doi: 10.1016/j.physio.2011.05.004.
877. Ryan T, Enderby P, Rigby AS. A randomized controlled trial to evaluate intensity of community-based rehabilitation provision following stroke or hip fracture in old age. *Clin Rehabil*. 2006;20:123–131.
878. Battersby M, Hoffmann S, Cadilhac D, Osborne R, Lalor E, Lindley R. "Getting your life back on track after stroke": a phase II multi-centered, single-blind, randomized, controlled trial of the Stroke Self-Management Program vs. the Stanford Chronic Condition Self-Management Program or standard care in stroke survivors. *Int J Stroke*. 2009;4:137–144. doi: 10.1111/j.1747-4949.2009.00261.x.
879. Harrington R, Taylor G, Hollinghurst S, Reed M, Kay H, Wood VA. A community-based exercise and education scheme for stroke survivors: a randomized controlled trial and economic evaluation. *Clin Rehabil*. 2010;24:3–15. doi: 10.1177/0269215509347437.
880. Thorsén AM, Holmqvist LW, de Pedro-Cuesta J, von Koch L. A randomized controlled trial of early supported discharge and continued rehabilitation at home after stroke: five-year follow-up of patient outcome. *Stroke*. 2005;36:297–303. doi: 10.1161/01.STR.0000152288.42701.a6.
881. Hillier S, Inglis-Jassiem G. Rehabilitation for community-dwelling people with stroke: home or centre based? A systematic review. *Int J Stroke*. 2010;5:178–186. doi: 10.1111/j.1747-4949.2010.00427.x.
882. Lee HS, Ann CS, Kim MC, Choi JH, Yuk GC. Patient preference for community-based rehabilitation programs after stroke. *J Phys Ther Sci*. 2011;23:137–140.
883. Bakas T, Clark PC, Kelly-Hayes M, King RB, Lutz BJ, Miller EL; on behalf of the American Heart Association Council on Cardiovascular and Stroke Nursing and the Stroke Council. Evidence for stroke family caregiver and dyad interventions: a statement for healthcare professionals from the American Heart Association and American Stroke Association. *Stroke*. 2014;45:2836–2852. doi: 10.1161/STR.0000000000000033.
884. Hartley S, Finkenflugel H, Kuipers P, Thomas M. Community-based rehabilitation: opportunity and challenge. *Lancet*. 2009;374:1803–1804. doi: 10.1016/S0140-6736(09)62036-5.
885. Barker LN, Ziino C. Community rehabilitation: "home versus centre" guidelines for choosing the optimal treatment location. *Int J Rehabil Res*. 2010;33:115–123. doi: 10.1097/MRR.0b013e32832e6c73.

886. Reed MC, Wood V, Harrington R, Paterson J. Developing stroke rehabilitation and community services: a meta-synthesis of qualitative literature. *Disabil Rehabil*. 2012;34:553–563. doi: 10.3109/09638288.2011.613511.
887. Aziz NA, Leonardi-Bee J, Phillips M, Gladman JR, Legg L, Walker MF. Therapy-based rehabilitation services for patients living at home more than one year after stroke. *Cochrane Database Syst Rev*. 2008;CD005952. doi: 10.1002/14651858.CD005952.pub2.
888. Legg LA, Quinn TJ, Mahmood F, Weir CJ, Tierney J, Stott DJ, Smith LN, Langhorne P. Non-pharmacological interventions for caregivers of stroke survivors. *Cochrane Database Syst Rev*. 2011;CD008179. doi: 10.1002/14651858.CD008179.pub2.
889. Schmitz MA, Finkelstein M. Perspectives on poststroke sexual issues and rehabilitation needs. *Top Stroke Rehabil*. 2010;17:204–213. doi: 10.1310/tsr1703-204.
890. Passier PE, Visser-Meily JM, Rinkel GJ, Lindeman E, Post MW. Life satisfaction and return to work after aneurysmal subarachnoid hemorrhage [published correction appears in *J Stroke Cerebrovasc Dis*. 2011;20:487]. *J Stroke Cerebrovasc Dis*. 2011;20:324–329. doi: 10.1016/j.jstrokecerebrovasdis.2010.02.001.
891. Stein J, Hillinger M, Clancy C, Bishop L. Sexuality after stroke: patient counseling preferences. *Disabil Rehabil*. 2013;35:1842–1847. doi: 10.3109/09638288.2012.754953.
892. Gianotten WL, Bender JL, Post MW. Training in sexology for medical and paramedical professionals: a model for the rehabilitation setting. *Sex Relationship Ther*. 2006; 21:303–317. <http://dx.doi.org/10.1080/14681990600754559>. Accessed December 29, 2014.
893. Primack BA, Carroll MV, McNamara M, Klem ML, King B, Rich M, Chan CW, Nayak S. Role of video games in improving health-related outcomes: a systematic review. *Am J Prev Med*. 2012;42:630–638. doi: 10.1016/j.amepre.2012.02.023.
894. Taylor AH, Cable NT, Faulkner G, Hillsdon M, Narici M, Van Der Bij AK. Physical activity and older adults: a review of health benefits and the effectiveness of interventions. *J Sports Sci*. 2004;22:703–725. doi: 10.1080/02640410410001712421.
895. Aoyagi Y, Shephard RJ. Habitual physical activity and health in the elderly: the Nakanajo Study. *Geriatr Gerontol Int*. 2010;10(suppl 1):S236–S243. doi: 10.1111/j.1447-0594.2010.00589.x.
896. Schwarzenegger A, Chrisman M, Coleman R. *The Health and Social Benefits of Recreation*. Sacramento, CA: California State Parks; 2005.
897. Thompson Coon J, Boddy K, Stein K, Whear R, Barton J, Depledge MH. Does participating in physical activity in outdoor natural environments have a greater effect on physical and mental wellbeing than physical activity indoors? A systematic review. *Environ Sci Technol*. 2011;45:1761–1772. doi: 10.1021/es102947t.
898. O'Sullivan C, Chard G. An exploration of participation in leisure activities post-stroke. *Aust Occup Ther J*. 2010;57:159–166. doi: 10.1111/j.1440-1630.2009.00833.x.
899. McKenna K, Liddle J, Brown A, Lee K, Gustafsson L. Comparison of time use, role participation and life satisfaction of older people after stroke with a sample without stroke. *Aust Occup Ther J*. 2009;56:177–188. doi: 10.1111/j.1440-1630.2007.00728.x.
900. Eriksson G, Aasnes M, Tistad M, Guidetti S, von Koch L. Occupational gaps in everyday life one year after stroke and the association with life satisfaction and impact of stroke. *Top Stroke Rehabil*. 2012;19:244–255. doi: 10.1310/tsr1903-244.
901. Deleted in proof.
902. Richards LG, Latham NK, Jette DU, Rosenberg L, Smout RJ, DeJong G. Characterizing occupational therapy practice in stroke rehabilitation. *Arch Phys Med Rehabil*. 2005;86(suppl 2):S51–S60. doi: 10.1016/j.apmr.2005.08.127.
903. Desrosiers J, Noreau L, Rochette A, Carbonneau H, Fontaine L, Viscogliosi C, Bravo G. Effect of a home leisure education program after stroke: a randomized controlled trial. *Arch Phys Med Rehabil*. 2007;88:1095–1100. doi: 10.1016/j.apmr.2007.06.017.
904. Walker MF, Leonardi-Bee J, Bath P, Langhorne P, Dewey M, Corr S, Drummond A, Gilbertson L, Gladman JR, Jongbloed L, Logan P, Parker C. Individual patient data meta-analysis of randomized controlled trials of community occupational therapy for stroke patients. *Stroke*. 2004;35:2226–2232. doi: 10.1161/01.STR.0000137766.17092.fb.
905. Deleted in proof.
906. Barker DJ, Reid D, Cott C. The experience of senior stroke survivors: factors in community participation among wheelchair users. *Can J Occup Ther*. 2006;73:18–25.
907. Deleted in proof.
908. van der Ploeg HP, Streppel KR, van der Beek AJ, van der Woude LH, Vollenbroek-Hutten MM, van Harten WH, van Mechelen W. Counselling increases physical activity behaviour nine weeks after rehabilitation. *Br J Sports Med*. 2006;40:223–229. doi: 10.1136/bjsm.2005.021139.
909. van der Ploeg HP, Streppel KR, van der Beek AJ, van der Woude LH, Vollenbroek-Hutten MM, van Harten WH, van Mechelen W. Successfully improving physical activity behavior after rehabilitation. *Am J Health Promot*. 2007;21:153–159.
910. Treger I, Shames J, Giaquinto S, Ring H. Return to work in stroke patients. *Disabil Rehabil*. 2007;29:1397–1403. doi: 10.1080/09638280701314923.
911. Vestling M, Tufvesson B, Iwarsson S. Indicators for return to work after stroke and the importance of work for subjective well-being and life satisfaction. *J Rehabil Med*. 2003;35:127–131.
912. Brown DL, Boden-Albala B, Langa KM, Lisabeth LD, Fair M, Smith MA, Sacco RL, Morgenstern LB. Projected costs of ischemic stroke in the United States. *Neurology*. 2006;67:1390–1395. doi: 10.1212/01.wnl.0000237024.16438.20.
913. Hofgren C, Björkdahl A, Esbjörnsson E, Sunnerhagen KS, Stibrant-Sunnerhagen K. Recovery after stroke: cognition, ADL function and return to work [published correction appears in *Acta Neurol Scand*. 2007;115:210]. *Acta Neurol Scand*. 2007;115:73–80. doi: 10.1111/j.1600-0404.2006.00768.x.
914. Hommel M, Trabucco-Miguel S, Joray S, Naegele B, Gonnet N, Jaillard A. Social dysfunctioning after mild to moderate first-ever stroke at vocational age. *J Neurol Neurosurg Psychiatry*. 2009;80:371–375. doi: 10.1136/jnnp.2008.157875.
915. Doucet T, Muller F, Verdun-Esquer C, Debelleix X, Brochard P. Returning to work after a stroke: a retrospective study at the Physical and Rehabilitation Medicine Center La Tour de Gassies. *Ann Phys Rehabil Med*. 2012;55:112–127. doi: 10.1016/j.rehab.2012.01.007.
916. Kauranen T, Turunen K, Laari S, Mustanoja S, Baumann P, Poutiainen E. The severity of cognitive deficits predicts return to work after a first-ever ischaemic stroke. *J Neurol Neurosurg Psychiatry*. 2013;84:316–321. doi: 10.1136/jnnp-2012-302629.
917. Andersen G, Christensen D, Kirkevold M, Johnsen SP. Post-stroke fatigue and return to work: a 2-year follow-up. *Acta Neurol Scand*. 2012;125:248–253. doi: 10.1111/j.1600-0404.2011.01557.x.
918. Hannerz H, Holbaek Pedersen B, Poulsen OM, Humle F, Andersen LL. A nationwide prospective cohort study on return to gainful occupation after stroke in Denmark 1996–2006. *BMJ Open*. 2011;1:e000180.
919. Busch MA, Coshall C, Heuschmann PU, McKevitt C, Wolfe CD. Sociodemographic differences in return to work after stroke: the South London Stroke Register (SLSR). *J Neurol Neurosurg Psychiatry*. 2009;80:888–893. doi: 10.1136/jnnp.2008.163295.
920. Saeki S, Toyonaga T. Determinants of early return to work after first stroke in Japan. *J Rehabil Med*. 2010;42:254–258. doi: 10.2340/16501977-0503.
921. Hackett ML, Glozier N, Jan S, Lindley R. Returning to paid employment after stroke: the Psychosocial Outcomes In Stroke (POISE) cohort study. *PLoS One*. 2012;7:e41795. doi: 10.1371/journal.pone.0041795.
922. Baldwin C, Brusco NK. The effect of vocational rehabilitation on return-to-work rates post stroke: a systematic review. *Top Stroke Rehabil*. 2011;18:562–572. doi: 10.1310/tsr1805-562.
923. Morris R. The psychology of stroke in young adults: the roles of service provision and return to work. *Stroke Res Treat*. 2011;2011:534812. doi: 10.4061/2011/534812.
924. Lasker J, LaPointe L, Kodras J. Helping a professor with aphasia resume teaching through multimodal approaches. *Aphasiology*. 2005;19:399–410.
925. Chan ML. Description of a return-to-work occupational therapy programme for stroke rehabilitation in Singapore. *Occup Ther Int*. 2008;15:87–99.
926. Lister R. Loss of ability to drive following a stroke: the early experiences of three elderly people on discharge from hospital. *Br J Occup Ther*. 1999;62:514–520.
927. Perrier MJ, Komer-Bitensky N, Mayo NE. Patient factors associated with return to driving poststroke: findings from a multicenter cohort study. *Arch Phys Med Rehabil*. 2010;91:868–873. doi: 10.1016/j.apmr.2010.03.009.
928. Fisk GD, Owsley C, Pulley LV. Driving after stroke: driving exposure, advice, and evaluations. *Arch Phys Med Rehabil*. 1997;78:1338–1345.
929. Anstey KJ, Wood J, Lord S, Walker JG. Cognitive, sensory and physical factors enabling driving safety in older adults. *Clin Psychol Rev*. 2005;25:45–65. doi: 10.1016/j.cpr.2004.07.008.
930. Yale SH, Hansotia P, Knapp D, Ehrfurth J. Neurologic conditions: assessing medical fitness to drive. *Clin Med Res*. 2003;1:177–188.

- 930a. American Stroke Association. Driving after stroke. http://www.strokeassociation.org/STROKEORG/LifeAfterStroke/RegainingIndependence/Driving/Driving-After-Stroke_UCM_311016_Article.jsp#.Vtu80BjiTY8. Accessed March 5, 2016.
931. Logan PA, Dyas J, Gladman JR. Using an interview study of transport use by people who have had a stroke to inform rehabilitation. *Clin Rehabil*. 2004;18:703–708.
932. Akinwuntan AE, Feys H, De Weerd W, Baten G, Arno P, Kiekens C. Prediction of driving after stroke: a prospective study. *Neurorehabil Neural Repair*. 2006;20:417–423. doi: 10.1177/1545968306287157.
933. McKay C, Rapport LJ, Bryer RC, Casey J. Self-evaluation of driving simulator performance after stroke. *Top Stroke Rehabil*. 2011;18:549–561. doi: 10.1310/tsr1805-549.
934. Petzold A, Korner-Bitensky N, Rochette A, Teasell R, Marshall S, Perrier MJ. Driving poststroke: problem identification, assessment use, and interventions offered by Canadian occupational therapists. *Top Stroke Rehabil*. 2010;17:371–379. doi: 10.1310/tsr1705-371.
935. Deleted in proof.
936. Marshall SC, Molnar F, Man-Son-Hing M, Blair R, Brosseau L, Finestone HM, Lamothe C, Korner-Bitensky N, Wilson KG. Predictors of driving ability following stroke: a systematic review. *Top Stroke Rehabil*. 2007;14:98–114. doi: 10.1310/tsr1401-98.
937. Devos H, Akinwuntan AE, Nieuwboer A, Truijen S, Tant M, De Weerd W. Screening for fitness to drive after stroke: a systematic review and meta-analysis. *Neurology*. 2011;76:747–756. doi: 10.1212/WNL.0b013e31820d6300.
938. Akinwuntan AE, De Weerd W, Feys H, Pauwels J, Baten G, Arno P, Kiekens C. Effect of simulator training on driving after stroke: a randomized controlled trial. *Neurology*. 2005;65:843–850. doi: 10.1212/01.wnl.0000171749.71919.fa.
939. Lundqvist A, Gerdle B, Rönnberg J. Neuropsychological aspects of driving after stroke: in the simulator and on the road. *Appl Cogn Psychol*. 2000;14:135–150.
940. Devos H, Akinwuntan AE, Nieuwboer A, Ringoot I, Van Berghen K, Tant M, Kiekens C, De Weerd W. Effect of simulator training on fitness-to-drive after stroke: a 5-year follow-up of a randomized controlled trial. *Neurorehabil Neural Repair*. 2010;24:843–850. doi: 10.1177/1545968310368687.
941. Devos H, Akinwuntan AE, Nieuwboer A, Tant M, Truijen S, De Wit L, Kiekens C, De Weerd W. Comparison of the effect of two driving retraining programs on on-road performance after stroke. *Neurorehabil Neural Repair*. 2009;23:699–705. doi: 10.1177/1545968309334208.
942. Söderström ST, Pettersson RP, Leppert J. Prediction of driving ability after stroke and the effect of behind-the-wheel training. *Scand J Psychol*. 2006;47:419–429. doi: 10.1111/j.1467-9450.2006.00550.x.
943. Crotty M, George S. Retraining visual processing skills to improve driving ability after stroke. *Arch Phys Med Rehabil*. 2009;90:2096–2102. doi: 10.1016/j.apmr.2009.08.143.
944. Bergsma DP, Leenders MJ, Verster JC, van der Wildt GJ, van den Berg AV. Oculomotor behavior of hemianopic chronic stroke patients in a driving simulator is modulated by vision training. *Restor Neurol Neurosci*. 2011;29:347–359. doi: 10.3233/RNN-2011-604.