Physical Rehabilitation

FIFTH EDITION

Susan B. O'Sullivan, PT, EdD
Professor and Chair
Department of Physical Therapy
School of Health and Environment
University of Massachusetts Lowell
Lowell, Massachusetts

Thomas J. Schmitz, PT, PhD
Professor
Division of Physical Therapy
School of Health Professions
Long Island University
Brooklyn Campus
Brooklyn, New York

F. A. DAVIS COMPANY • Philadelphia

F.A. Davis Company
1915 Arch Street
Philadelphia, PA 19103
www.fadavis.com

Printed in the United States of America

Last digit indicates print number: 10 9 8 7 6 5 4 3 2 1

Acquisitions Editor: Margaret Biblis
Manager, Content Development: Deborah Thorp
Developmental Editor: Jennifer Pine
Design Manager: Carolyn O'Brien
Photographer: Jaime Eric Eisman, ASMP
Models: Frank J. Ciuba, DPT, MS; Clifford Weller

Library of Congress Cataloging-in-Publication Data

Physical rehabilitation / [edited by] Susan B. O'Sullivan, Thomas J. Schmitz.—5th ed.
p. ; cm.
Includes bibliographical references and index.
ISBN-13: 978-0-8036-1247-1
ISBN-10: 0-8036-1247-8
1. Physical therapy. I. O'Sullivan, Susan B. II. Schmitz, Thomas J.
[DNLM: 1. Physical Therapy Modalities. 2. Disability Evaluation. WB 460 P57763 2007]
RM700.O88 2007
615.8′2—dc22

2006019334

Preface

With the fifth edition of *Physical Rehabilitation*, we continue a tradition of striving for excellence that began more than 20 years ago. We are gratified by the continuing wide acceptance of *Physical Rehabilitation* by both faculty and students.

The text is designed to provide a comprehensive approach to the rehabilitation management of adult patients. As such, it is intended to serve as a primary textbook for professional-level physical therapy students, and as an important resource for practicing therapists as well as for other rehabilitation professionals. This fifth edition recognizes the continuing growth of the profession and integrates basic and applied clinical research to guide and inform evidence-based clinical practice. It also integrates terminology, practice patterns, specific tests and measures, and interventions presented in the American Physical Therapy Association's *Guide to Physical Therapist Practice.*

Physical Rehabilitation is organized into four sections. Section One (Chapters 1–3) provides an introduction to patient care and includes chapters on clinical decision making, psychosocial factors, and values. Section Two (Chapters 4–12) focuses on examination of the sensory, motor, and cardiovascular systems as well as examination of functional status and the environment. Section Three (Chapters 13–30) addresses the common diseases, disorders, or conditions seen in the rehabilitation setting. Appropriate examination and intervention strategies are discussed for related impairments, functional limitations, and disabilities. Emphasis is placed also on parameters of learning critical to ensuring the patient/client can achieve an independent and active lifestyle. The final section, Section Four (Chapters 31–33) includes orthotics, prosthetics, and the prescriptive wheelchair.

A central element of the text is a strong pedagogical format designed to facilitate and reinforce the learning of key concepts. Each chapter of *Physical Rehabilitation* includes an initial content outline, learning objectives, an introduction and summary, study questions for self-assessment, and extensive references. Additional supplemental readings and recommended resources are also provided. Key terms are bolded throughout each chapter indicating their inclusion in a master glossary toward the end of the text. Application of important concepts is promoted through case study examples and problem-oriented guiding questions. New to the fifth edition, many chapters contain *Evidence Summary Boxes* that summarize and critically appraise research focused on a particular topic or intervention relevant to the chapter content. Our hope is that the boxes may provide a model for readers to continue to critically examine clinical practice using validated clinical methodologies. We also hope it will inspire enthusiasm about the importance of continuous, lifelong self-directed learning, without which practice may become rapidly and dangerously out-of-date.

The visual illustrations have been substantially enhanced with the addition of many new line drawings and photographs. Changes in design and the introduction of a two-color format provide a more reader-friendly environment as well as augment understanding of content.

Without question, our greatest asset in preparing the 5th edition of *Physical Rehabilitation* has been an outstanding group of contributing authors. We are most fortunate to have this group of talented individuals whose breadth and scope of professional knowledge and experience seems unparalleled. These individuals are recognized experts from a variety of specialty areas who have graciously shared their knowledge and clinical practice expertise by providing relevant, up-to-date, and practical information within their respective content areas.

The fifth edition has also benefited from the input of numerous individuals engaged in both academic and clinical practice settings who have used and reviewed the content. We are grateful for their constructive feedback and have instituted many of their suggestions and changes. As always, we welcome suggestions for improvements from our colleagues and students.

As physical therapists continue to take on more and greater professional responsibilities and challenges, the very nature of this text makes it a perpetual "work in progress." We are grateful for the opportunity to contribute to the academic literature in physical therapy as well as to the professional development of those preparing to enter a career devoted to improving the quality of life of those we serve.

We acknowledge the very important contributions that physical therapists make in the lives of their patients. This book is dedicated to those therapists—past, present, and future—who guide and challenge their patients to lead a successful and independent life.

Susan B. O'Sullivan
Thomas J. Schmitz

Acknowledgments

The on-going development of *Physical Rehabilitation* has been in all aspects a collaborative venture. Its fruition made possible only through the expertise and gracious contributions of many talented individuals. Our appreciation is considerable.

Heartfelt thanks are extended to our contributing authors. Each has brought a unique body of knowledge as well as distinct clinical practice expertise to their respective chapters. Their commitment to physical therapist education is collectively displayed in content presentations that carefully reflect the scope of knowledge and skills required of a dynamic, evolving physical therapy practice environment. We are extremely grateful to each of our contributors as well as heartened by the excellence they bring to the fifth edition.

Our gratitude is also extended to our guest editors and reviewers. Their collective expertise provided critical input during various phases of project development. We are particularly indebted to them for their content suggestions, pedagogical insights, and unconditional positive regard for the academic pursuits of physical therapy students.

We thank those individuals and companies who contributed new photographs to the individual chapters and to those patients/clients who allowed their photographs to be used throughout the text.

Our appreciation goes to the dedicated professionals at F.A. Davis Company, Philadelphia, PA: Margaret M. Biblis, Publisher, Jennifer A. Pine, Developmental Editor, Bob Butler, Production Manager, and Ron Moser, Marketing Manager. These individuals are recognized for their continued support, encouragement, and unwavering commitment to excellence. We would also like to thank Jean-Francois Vilain, Former Publisher, FA Davis, for his support throughout previous editions of this text. Thanks also are extended to Berta Steiner, Director of Production, Bermedica Production Ltd, and to Deborah Lynam for her considerable talents in preparing many of the new line illustrations.

We wish to thank the numerous students, faculty, and clinicians who over the years have used *Physical Rehabilitation* and provided us with meaningful and constructive comments that have greatly enhanced this edition. It is our sincere hope that this feedback will continue with the fifth edition.

Our thanks go also to the following individuals who provided assistance during different phases of the project: Diana Agoston, Elizabeth Blas, Ryanne Glasper, Ellen Godwin, Kim Harris, Cristiana Kahl Collins, Katherine Li, Mary Maloney, Eileen McAulay, Michele Mills, Evangelos Pappas, Alexander Rosado, Alexis Sams, and Gideon F. Shapiro. A particular note of thanks is extended to Stephen A. Caronia who contributed several Evidence Summary Boxes as well as considerable editorial assistance; to Alisa Yalan-Murphy, Faculty Liaison, Faculty Media Resource Center, Long Island University, for her great patience in creating many high quality photographs; and to Ivaldo Costa whose support has truly been immeasurable.

Finally, we are grateful for the continuing strong and productive working relationship that we maintain that has allowed us to complete a project of this scope through five editions.

Susan B. O'Sullivan
Thomas J. Schmitz

Guest Editors

Sharon A. Gutman, PhD, OTR
Associate Professor
Columbia University
Programs in Occupational Therapy
New York, New York

Raymond Marx, CPO, FAAOP
President
Ortho-Bionics Laboratory, Inc.
South Ozone Park, New York

Anne Hiller Scott, PhD, OTR, FAOTA
Associate Professor
Division of Occupational Therapy
School of Health Professions
Long Island University
Brooklyn Campus
Brooklyn, New York

Contributing Authors

Andrea L. Behrman, PT, PhD
Associate Professor
Department of Physical Therapy
University of Florida
Gainesville, Florida

Research Health Scientist
VA Brain Rehabilitation Research Center
Malcom Randall VA Medical Center
Gainesville, Florida

Adrienne Falk Bergen, PT, ATP
Seating Specialist
Delray Beach, Florida

Lisa Janice Cohen, PT, MS, OCS
The Body Mind Integration Center
Watertown, Massachusetts

Vanina Dal Bello-Haas, BScPT, PhD
Associate Professor
School of Physical Therapy
University of Saskatchewan
Saskatoon, Saskatchewan
Canada

Carol M. Davis, PT, EdD, MS, FAPTA
Professor and Assistant Chair
Department of Physical Therapy
University of Miami Miller School of Medicine
Coral Gables, Florida

John L. Echternach, PT, EdD, ECS, FAPTA
Professor and Eminent Scholar
School of Physical Therapy
Old Dominion University
Norfolk, Virginia

Joan E. Edelstein, PT, MA, FISPO
Special Lecturer
Program in Physical Therapy
Columbia University
New York, New York

George D. Fulk, PT, PhD
Assistant Professor
Department of Physical Therapy
Clarkson University
Potsdam, New York

Kate Grimes, DPT, MS, CCS
Clinical Assistant Professor
Graduate Programs in Physical Therapy
MGH Institute of Health Professions
Boston, Massachusetts

Andrew A. Guccione, DPT, PhD, FAPTA
Senior Vice President
Practice and Research Division
American Physical Therapy Association
Alexandria, Virginia

Deborah Graffis Kelly, PT, MSEd
Associate Professor
Division of Physical Therapy
University of Kentucky
Lexington, Kentucky

Bella J. May, PT, EdD, FAPTA
Professor Emerita
Medical College of Georgia
Augusta, Georgia

President, BJM Enterprises
Dublin, California

Marian A. Minor, PT, PhD
Professor and Chair
Department of Physical Therapy
School of Health Professions
University of Missouri
Columbia, Missouri

Cynthia C. Norkin, PT, EdD
Former Director and Associate Professor
School of Physical Therapy
Ohio University
Athens, Ohio

Sandra J. Olney, BSc (P & OT), MEd, PhD
Professor and Director
School of Rehabilitation Therapy
Queen's University
Kingston, Ontario
Canada

Susan B. O'Sullivan, PT, EdD
Professor and Chair
Department of Physical Therapy
School of Health and Environment
University of Massachusetts Lowell
Lowell, Massachusetts

Leslie G. Portney, DPT, PhD, FAPTA
Professor and Director
Graduate Programs in Physical Therapy
MGH Institute of Health Professions
Boston, Massachusetts

Pat Precin, OTR/L, MS, ABD
Assistant Professor
Department of Occupational Therapy
New York Institute of Technology
Old Westbury, New York

Executive Director
The Fostering Connection, Inc.
Brooklyn, New York

Consultant
Pathways to Housing, Inc.
New York, New York

Reginald L. Richard, PT, MS
Burn Clinical Specialist
Physical Therapy Department
Miami Valley Hospital Regional Burn Center
Dayton, Ohio

Serge H. Roy, PT, ScD
Research Professor
NeuroMuscular Research Center
Boston University
Boston, Massachusetts

Martha Taylor Sarno, MA, MD (hon)
Professor of Rehabilitation Medicine
School of Medicine
New York University
New York, New York

Director
Speech-Language Pathology Department
Rusk Institute of Rehabilitation Medicine
New York University Medical Center
New York, New York

David A. Scalzitti, PT, MS, OCS
Associate Director
Research Services
American Physical Therapy Association
Alexandria, Virginia

Thomas J. Schmitz, PT, PhD
Professor
Division of Physical Therapy
School of Health Professions
Long Island University
Brooklyn Campus
Brooklyn, New York

Michael C. Schubert, PT, PhD
Assistant Professor
The Department of Otolaryngology Head and Neck Surgery
Johns Hopkins University School of Medicine
Baltimore, Maryland

Julie Ann Starr, PT, MS, CCS
Clinical Associate Professor
Department of Physical Therapy and Athletic Training
Sargent College of Health and Rehabilitation Sciences
Boston University
Boston, Massachusetts

Carolyn A. Unsworth, OTR, PhD
Associate Professor
School of Occupational Therapy
La Trobe University
Bundoora, Victoria
Australia

R. Scott Ward, PT, PhD
Professor and Chair
Division of Physical Therapy
University of Utah
Salt Lake City, Utah

D. Joyce White, PT, DSc
Associate Professor
Department of Physical Therapy
School of Health and Environment
University of Massachusetts Lowell
Lowell, Massachusetts

Reviewers

Candy Bahner, PT, MS
Director and Assistant Professor
Physical Therapist Assistant Program
Washburn University
Topeka, Kansas

Marja Beaufait, PT, MA
Associate Professor
Physical Therapist Assistant Program
St. Petersburg College
St. Petersburg, Florida

Wendy Bircher, PT, EdD
Director
Physical Therapist Assistant Program
San Juan College
Farmington, New Mexico

Jill W. Bloss, PT, MPH
Former Associate Professor
Physical Therapy Program
Nazareth College
Rochester, New York

Kathy Brewer PT, MEd, GCS
Adjunct Faculty
Department of Physical Therapy
Arizona School of Health Sciences
A.T. Still University
Mesa, Arizona

Suzanne Robben Brown, PT, MPH
Director and Associate Professor
Department of Physical Therapy
Arizona School of Health Sciences
A.T. Still University
Mesa, Arizona

Alice F. Cain, PT
Clinical Coordinator
Physical Therapist Assistant Technology
Stark State College
North Canton, Ohio

Susan D. Calise, PT, MA
Brookdale Department of Geriatrics and Adult Development
Mount Sinai School of Medicine
New York, New York

Sean M. Collins, PT, ScD, CCS
Associate Professor
Department of Physical Therapy
School of Health and Environment
University of Massachusetts Lowell
Lowell, Massachusetts

Sam M. Coppoletti, MPT, BS, CSCS
Director and Assistant Professor
Physical Therapist Assistant Program
Shawnee State University
Portsmouth, Ohio

Linda Denney, PT, M.Appl.Sci. (Manip)
Adjunct Assistant Professor
Department of Physical Therapy
Arizona School of Health Sciences
A.T. Still University
Mesa, Arizona

George D. Fulk, PT, PhD
Assistant Professor
Department of Physical Therapy
Clarkson University
Potsdam, New York

Christine Kowalski, PTA, EdD
Director
Physical Therapist Assistant Program
Great Falls College of Technology
Montana State University
Great Falls, Montana

Steven G. Lesh, PT, PhD, MPA, SCS, ATC
Chair and Associate Professor
Department of Physical Therapy
Southwest Baptist University
Bolivar, Missouri

Marilyn Maxwell, PhD
Instructor, English Department
Hewlett High School
Hewlett, New York

Kathy Mercuris, PT, MGS
Former Associate Professor
Division of Physical Therapy
Des Moines University-Osteopathic Medical Center
Des Moines, Iowa

Robert J. Nelson, PT, MS, MPhil
H & D Physical Therapy
New York, New York

Susan Roehrig, PT, PhD
Associate Professor
Program in Physical Therapy
Arkansas State University
Jonesboro, Arkansas

Lynda L. Spangler, PT, MS, ACCE
Assistant Professor
Department of Physical Therapy
The College of St. Scholastica
Duluth, Minnesota

Doreen Stiskal, PT, PhD
Assistant Chair
Graduate Programs in Health Sciences
Seton Hall University
South Orange, New Jersey

Kristin von Nieda, DPT, MEd
Associate Professor
Director, Transitional DPT Program
Department of Physical Therapy
Temple University
Philadelphia, Pennsylvania

Denise Wise, PT, PhD
Chair and Assistant Professor
Department of Physical Therapy
The College of St. Scholastica
Duluth, Minnesota

Peter Zawicki, PT, MS
Director
Physical Therapist Assistant Program
Gateway Community College
Phoenix, Arizona

Table of Contents

Section Three: Intervention Strategies for Rehabilitation 469

CHAPTER

13

LEARNING OBJECTIVES

1. Describe a model for clinical decision making that incorporates components of normal motor control and motor learning.
2. Identify factors critical to motor control and describe intervention strategies designed to optimize the acquisition of motor control.
3. Identify key factors in recovery of function and describe intervention strategies designed to optimize recovery.
4. Identify factors critical to motor learning and describe intervention strategies designed to optimize learning.
5. Differentiate among the following: functional training, neurofacilitation training, and compensatory training.
6. Analyze and interpret patient data, formulate anticipated goals and expected outcomes, and develop a plan of care that presents an integrated approach to treatment when presented with a clinical case study.

Strategies to Improve Motor Function

Susan B. O'Sullivan, PT, EdD

OUTLINE

Developing strategies to improve motor function (motor control, motor recovery, and motor learning) requires a thorough understanding of the neural processes involved in producing movement and learning, and the pathologies that may affect the central nervous system (CNS). In addition, knowledge of recovery processes following CNS insult is essential. Treatment models based on theories of motor control, recovery, and learning allow the therapist to organize thinking and approach clinical decision making in a coherent manner. Patients with disorders of motor function frequently demonstrate a wide variety of impairments, functional limitations, and disabilities. Careful examination of sensorimotor and learning behaviors and the environmental contexts in which they occur provides an appropriate base for planning. Different intervention strategies and techniques have been developed by physical therapists to address disorders of motor function. An optimal plan of care (POC) must address the individual needs of the patient, maintain a focus on minimizing or eliminating functional limitations and physical disabilities, and enhance overall quality of life.

Motor Control

Motor control has been defined as "an area of study dealing with the understanding of the neural, physical, and behavioral aspects of movement."[1, p 465] Information processing of human motor behavior occurs in stages (Fig. 13.1). The initial stage is *stimulus identification.* Relevant stimuli concerning current body state and environmental context are selected and identified. Meaning is attached based on past sensorimotor experiences. Perceptual and cognitive processes including memory, attention, motivation, and emotional control all play an integral role in ensuring the ease and accuracy of information processing during this stage. Selection of relevant sensory input is sensitive to the clarity and intensity of the stimuli received. Thus, stronger and crisper stimuli result in enhanced attentional mechanisms and information processing. Processing is also influenced by stimulus pattern complexity. Complicated, novel patterns of stimuli prolong stimulus identification. An intrinsic knowledge of movement (e.g., position of limb,

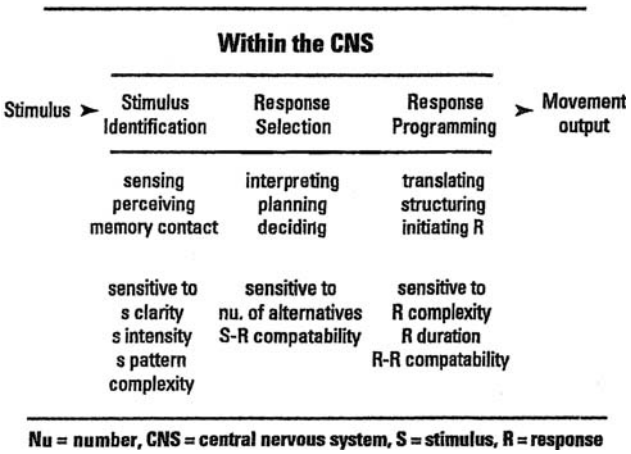

Figure 13.1 Model of information-processing stages of movement control.

length of limb, distance to goal, and so forth) is a critical characteristic of motor behavior. In the *response selection stage* the plan for movement is developed.

A **motor plan** is defined as an idea or plan for purposeful movement that is made up of component motor programs. A general rather than detailed response is selected; that is, a prototype of the final movement. Decision making during this stage is sensitive to the number of different movement alternatives possible and the overall compatibility between the stimulus and response. The more natural the association between stimulus and response, the easier the decision making. For example, in a well-learned movement like crossing at a street light, an individual easily responds to the green light by moving forward. If a crossing guard signals the individual to move forward even though the light is red, the individual is likely to be more hesitant in responding.

The final stage is termed *response programming.* Neural control centers translate and change the idea for movement into muscular actions defined by a motor program. A **motor program** is defined as "an abstract representation that, when initiated, results in the production of a coordinated movement sequence."[1, p 466] The structuring of motor programs includes attention to specific parameters such as synergistic component parts, force, direction, timing, duration, and extent of movement. Parametric specification is based on the constraints of the individual, the task, and the environment. Information processing during this stage is sensitive to the complexity of the desired movement and duration. Thus, complex and lengthier movement sequences increase the duration of processing during this stage. Programming can also be affected by response–response compatibility. This is the compatibility for dual movement tasks that either occur simultaneously (e.g., bouncing a ball while walking) or when choices are required (e.g., one paired movement response must occur before another). During response execution (movement output), muscles are selected against an appropriate background of postural control. **Feedforward**, the sending of signals in advance of movement to ready the system, allows for anticipatory adjustments in postural activity. **Feedback**, response-produced information received during or after the movement, is used to monitor output for corrective actions. Although this simplified model gives the appearance that the information flow is linear, actual processing by the CNS is both serial and parallel. Thus, both single and multiple pathways are engaged to process information.[1] Figure 13.2 provides a schematic depiction of the major directions of information flow within the CNS during voluntary movement.

Theories of Motor Control

A theory is the orderly explanation of observations. Different theories of motor control have been developed over time, and reflect current understanding and interpretation of nervous system function. Because theories provide an important framework for clinical practice, a brief overview is warranted. The reader is also referred to the excellent works of Schmidt[1] and Shumway-Cook and Woollacott[2] for further review and study.

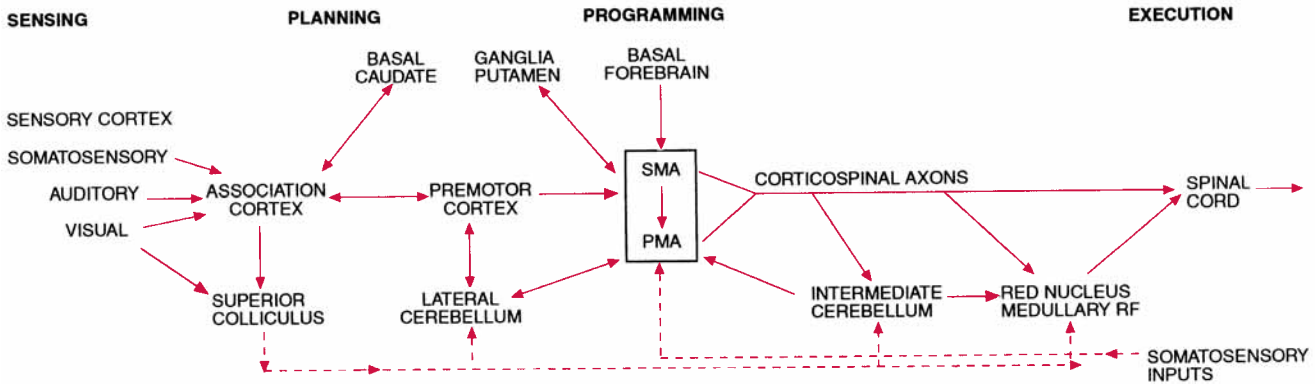

Figure 13.2 Major directions of information flow during a voluntary movement to and from the primary motor areas (PMA) and supplementary motor areas (SMA), only some of the connections of the superior colliculus are shown. RF = reticular formation. (From Brooks,[5, p 199] with permission.)

An early theory of motor control, *reflex theory*, was established by Sherrington.[3] His research on sensory receptors led to the view that movement was the result of a stimulus–response sequence of events or **reflex** based. Complex movements were nothing more than the coupling or chaining together of a number of reflexes to produce the final outcome. Thus, sensation assumed a primary role in the initiation and production of movement. Limitations in reflex theory abound. It fails to consider that voluntary movements can be activated in the absence of a sensory stimulus. It also fails to consider that some movements occur so fast as to not allow use of available feedback. Finally, it does not consider the infinite variability that allows for different movements in response to the same stimulus.[2]

Hierarchical theory dates back to the work of Hughlings Jackson.[4] This theory is based on the assumption that the CNS is organized into three primary levels of control: high, middle, and low centers. Control was viewed as proceeding in a descending direction from higher to lower centers, a "top-down" progression. Reflex theory integrated with hierarchical theory presents the view that reflexes are components of the lower centers that became integrated during normal maturation and development as higher centers assumed control. Conversely, reflexes reemerge in control of movement when higher centers become damaged. A more current interpretation of this model proposes a theory of *flexible hierarchies*.[5] Within this modification, the command hierarchies have been more fully elaborated. The association cortex operates as the highest level (elaborating perceptions and planning strategies), while the sensorimotor cortex in association with portions of the basal ganglia, brainstem, and cerebellum function as the middle level (converting strategies into motor programs and commands). The spinal cord functions at the lowest level, translating commands into muscle actions resulting in the execution of movement. Modern hierarchical theory proposes that the three levels do not operate in a rigid, top-down order as originally described but rather as a flexible system in which each level can exert control on the others. Shifts in control are dependent on the demands and complexity of the task with the higher centers always assuming control whenever the task demands are high.

Systems theory, proposed by Bernstein,[6] is based on the view that motor control is the result of the cooperative actions of many interacting systems, working to accommodate the demands of the specific task. Both internal factors (joint stiffness, inertia, movement-dependent forces) and external factors (gravity) must be taken into consideration in the planning of movements. It assumes a shifting locus of neural control, referred to as a *distributed model of control*. Thus, large areas of the CNS may be engaged for complex motor tasks while relatively few centers are engaged for more discrete movements. This type of multilevel control allows for the control of a number of separate independent dimensions of movement, termed *degrees of freedom*. The executive level is freed from the responsibility of control for simple movements or the demands of having to control many degrees of freedom at one time. *Coordinative structures* are used to simplify control, and to initiate coordinated patterns or synergies to produce movements. The use of *synergies* for the control of locomotion (central pattern generators) and posture (postural synergies) is well documented.[7]

Motor Programming Theory

Motor programs allow for movements to occur in the absence of sensation (deafferentation) or in situations in which limitations in speed of processing feedback negate control (rapid movements). Motor programs also free the nervous system from conscious decisions about movement, reducing the problem of multiple degrees of freedom. Motor programs can be run off virtually without the influence of peripheral feedback or error detection processes, termed an **open-loop control system** (Fig. 13.3). This is in contrast to a **closed-loop control system** (Fig. 13.4), which employs feedback and a reference for correctness to compute error and initiate subsequent corrections. Feedback and closed-loop processes play a critical role in the learning of new motor skills (response selection) and in the shaping and correction of ongoing movements (response execution). Feedback is also essential for the ongoing maintenance of body posture and balance.

The complexity of human movement negates any simplistic model of movement control. An *intermittent control hypothesis* described by Schmidt[1] proposes a blending of both open-loop and closed-loop processes, in

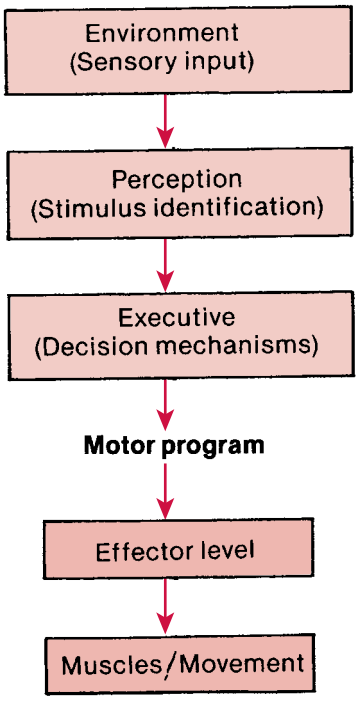

Figure 13.3 Open-loop control system.

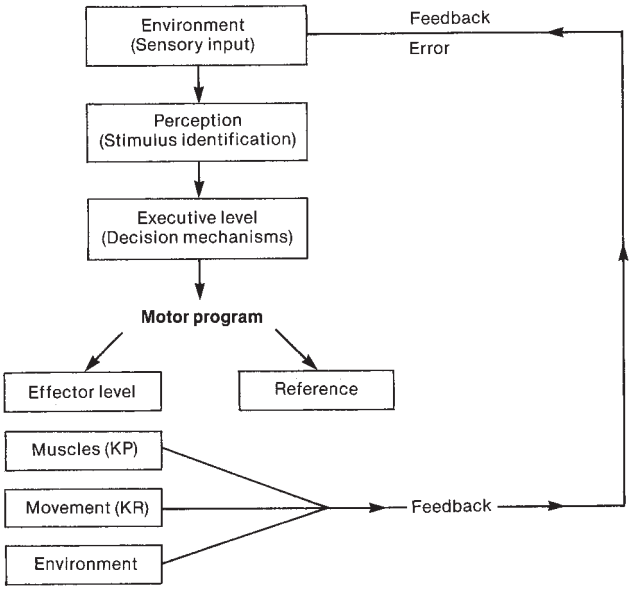

Figure 13.4 Closed-loop control system. KP = knowledge of performance, KR = knowledge of results.

which both operate in concert as part of the larger system. Motor programs provide the generalized code for motor events **(schema)**, while feedback is used to refine and perfect movements. Either may assume a dominant role, depending on the task at hand. Both may operate within a given movement but at different times and with different functions. Generalized motor programs include both invariant characteristics and parameters. *Invariant characteristics* are the unique features of the stored code: relative force, relative timing, and order of components. *Parameters* are the changeable features that ensure flexibility of motor programs and variations in movements from one performance to the next. These include overall force and overall duration of the movement. For example, walking performance can be changed by speeding up or slowing down (changes in overall duration) while the basic order of stepping cycle and relative timing of the components (invariant characteristics) are maintained.[8] Patients with deficits in motor function may demonstrate impairments in voluntary movements (impaired motor planning or programming) or the corrective actions (feedback adjustments) needed to initially learn and coordinate movements.

Motor Learning

Motor learning has been defined as "a set of internal processes associated with practice or experience leading to relatively permanent changes in the capability for skilled behavior."[1, p 466] Learning a motor skill is a complex process that requires spatial, temporal, and hierarchical organization of the CNS. Changes in the CNS are not directly observable but rather are inferred from changes in motor behavior. Improvements in **performance** result from practice or experience and are a frequently used measure of learning. For example, with practice an individual is able to develop appropriate sequencing of movement components with improved timing and reduced effort and concentration. Performance, however, is not always an accurate reflection of learning. It is possible to practice enough to temporarily improve performance but not retain the learning. Conversely, factors such as fatigue, anxiety, poor motivation, or medications may cause performance to deteriorate while learning may still occur. Because performance can be affected by a number of factors, it can be reasonably defined as a "temporary change in motor behavior seen during practice sessions."[2, p 24] **Retention** provides a better measure of learning. Retention refers to the ability of the learner to demonstrate the skill over time and after a period of no practice **(retention interval)**. Performance after a retention interval may decrease slightly, but should return to original performance levels within relatively few practice trials. For example, riding a bike is a well-learned skill that is generally retained even though an individual may not have ridden for years. The ability to apply a learned skill to the learning of other similar tasks, termed **generalizability**, is another important measure of learning. Individuals who learn to transfer from wheelchair to platform mat can apply that learning to other types of transfers (e.g., wheelchair to car, wheelchair to tub). The time and effort required to organize and learn these new types of transfers is reduced. Finally, learning can be measured by **resistance to contextual change**. This is the adaptability required to perform a motor task in altered environmental situations. Thus, an individual who has learned a skill (e.g., walking with a cane on indoor level surfaces) should be able to apply that learning to new and variable situations (e.g., walking outdoors, walking on a busy sidewalk). Motor learning is the direct result of practice and is highly dependent on sensory information and feedback processes. The relative importance of the different types of sensory information varies according to task and to the phase of learning. Individual differences exist **(motor capacity)** and may influence both the rate and degree of learning possible. Impairments in learning are common for the patient with CNS dysfunction.

Theories of Motor Learning

Adams[9] developed a theory of motor learning based on closed-loop control (closed-loop theory). He postulated that sensory feedback from ongoing movement is compared with stored memory of the intended movement (perceptual trace) to provide the CNS with a **reference of correctness** and error detection. Memory traces are then used to produce

an appropriate action and to evaluate outcomes. The stronger the perceptual trace developed through practice, the greater the capability of the learner to use closed-loop processes for learning movements. Adams concentrated on examining slow, linear-positioning responses. This theory does not adequately explain learning under conditions of rapid movements (open-loop control processes). It also does not explain learning that can occur in the absence of sensory feedback (deafferentation studies).

Schema theory, proposed by Schmidt,[10] is based on the concepts that slow movements are feedback-based while rapid movements are program-based. He proposed that schema were used for storage into memory. Schema is defined as "a rule, concept, or relationship formed on the basis of experience."[1, p 467] Schema can include such things as initial conditions (body position, weight of objects, and so forth), relationships between parameters of movement, environmental outcomes, and sensory consequences of movement. *Recall schema* are used to select and define the initial movement conditions while *recognition schema* are used to evaluate movement responses based on expected sensory consequences. Clinically, this theory supports the concept that practicing a variety of movement outcomes would improve learning through the development of expanded rules or schema. It also provides a plausible explanation for the learning of novel and open skills performed in a variable and changing environment.

Stages of Motor Learning

The process of motor learning has been described by Fitts and Posner[11] as occurring in relatively distinct stages, termed cognitive, associated, and autonomous. These stages provide a useful framework for describing the learning process and for organizing training strategies. Table 13.1 provides a summary.

Cognitive Stage

During the initial **cognitive stage** of learning, the major task at hand is to develop an overall understanding of the skill, termed the *cognitive map* or cognitive plan. This decision making phase of *"what to do"* requires a high level of cognitive processing as the learner performs successive approximations of the task, discarding strategies that are not successful and retaining those that are. The resulting trial-and-error practice initially yields uneven performance with frequent errors. Processing of sensory cues and perceptual–motor organization eventually leads to the selection of a motor strategy that proves reasonably successful. Because the learner progresses from an initially disorganized and often clumsy pattern to more organized movements, improvements in performance can be readily observed during this acquisition phase. The learner relies heavily on vision to guide early learning and movement. A stable environment free from distractors optimizes learning during this initial stage.

Associative Stage

During the middle or **associative stage** of learning, refinement of the motor strategy is achieved through continued practice. Spatial and temporal aspects become organized as the movement develops into a coordinated pattern. As performance improves, there is greater consistency and fewer errors and extraneous movements. The learner is now concentrating on "how to do" the movement rather than on what to do. Proprioceptive cues become increasingly important, while dependence on visual cues decreases. The learning process takes varying lengths of time depending on a number of factors. The nature of the task, prior experience and motivation of the learner, available feedback, and organization of practice can all influence acquisition of learning.

Autonomous Stage

The final or **autonomous stage** of learning is characterized by motor performance that after considerable practice is largely automatic. There is only a minimal level of cognitive monitoring, with motor programs so refined they can almost "run themselves." The spatial and temporal components of movement are becoming highly organized, and the learner is capable of coordinated motor patterns. The learner is now free to concentrate on other aspects, such as *"how to succeed"* at a competitive sport. Movements are largely error-free with little interference from environmental distractions. Thus the learner can perform equally well in a stable, predictable environment (termed **closed motor skills**) or in a changing, unpredictable environment (termed **open motor skills**).

Strategies to Enhance Motor Learning

Motor learning involves a significant amount of practice and feedback, with a high level of information processing related to control, error detection, and correction. Motor learning can be facilitated through the use of effective training strategies (summarized in Table 13.1).

Strategy Development

The overall goal during the early cognitive stage of learning is to facilitate task understanding and organize early practice. The learner's knowledge of the skill and any existing problems must be ascertained. The therapist should highlight the purpose of the skill in a functionally relevant context. The task should seem important, desirable, and realistic to learn. The therapist should demonstrate the task exactly as it should be done (i.e., coordinated action with smooth timing and ideal performance speed). This helps the learner develop an internal cognitive map or *reference of correctness*. Attention should be directed to the desired outcome and critical task elements. The therapist should point out similarities to other learned tasks so that subroutines that are part of other motor programs can be retrieved from memory. Features of the environment critical to performance should also be highlighted.

Table 13.1 Characteristics of Motor Learning Stages and Training Strategies

Cognitive Stage Characteristics	Training Strategies
The learner develops an understanding of task; **cognitive mapping** assesses abilities, task demands; identifies stimuli, contacts memory; selects response; performs initial approximations of task; structures motor program; modifies initial responses "*What to do*" decision	Highlight purpose of task in functionally relevant terms. Demonstrate ideal performance of task to establish a **reference of correctness**. Have patient verbalize task components and requirements. Point out similarities to other learned tasks. Direct attention to critical task elements. Select appropriate feedback. • Emphasize intact sensory systems, intrinsic feedback systems. • Carefully pair extrinsic feedback with intrinsic feedback. • High dependence on vision: have patient watch movement. • **Knowledge of Performance** (KP): focus on errors as they become consistent; do not cue on large number of random errors. • **Knowledge of Results** (KR): focus on success of movement outcome. Ask learner to evaluate performance, outcomes; identify problems, solutions. Use reinforcements (praise) for correct performance, continuing motivation. Organize feedback schedule. • Feedback after every trial improves performance during early learning. • Variable feedback (summed, fading, bandwidth designs) increases depth of cognitive processing, improves retention; may decrease performance initially. Organize initial practice. • Stress controlled movement to minimize errors. • Provide adequate rest periods (distributed practice) if task is complex, long, or energy costly or if learner fatigues easily, has short attention, or poor concentration. • Use manual guidance to assist as appropriate. • Break complex tasks down into component parts, teach both parts and integrated whole. • Utilize bilateral transfer as appropriate. • Use blocked (repeated) practice of same task to improve performance. • Use variable practice (serial or random practice order) of related skills to increase depth of cognitive processing and retention; may decrease performance initially. • Use mental practice to improve performance and learning, reduce anxiety. Assess, modify arousal levels as appropriate. • High or low arousal impairs performance and learning. • Avoid stressors, mental fatigue. Structure environment. • Reduce extraneous environmental stimuli, distractors to ensure attention, concentration. • Emphasize closed skills initially gradually progressing to open skills.
Associated Stage Characteristics	**Training Strategies**
The learner practices movements, refines motor program: spatial and temporal organization; decreases errors, extraneous movements Dependence on visual feedback decreases, increases for use of proprioceptive feedback; cognitive monitoring decreases "*How to do*" decision	Select appropriate feedback. • Continue to provide KP; intervene when errors become consistent. • Emphasize proprioceptive feedback, "feel of movement" to assist in establishing an internal reference of correctness. • Continue to provide KR; stress relevance of functional outcomes. • Assist learner to improve self-evaluation, decision making skills. • Facilitation techniques, guided movements may be counterproductive during this stage of learning.

Table 13.1 Characteristics of Motor Learning Stages and Training Strategies (continued)

	Organize feedback schedule. • Continue to provide feedback for continuing motivation; encourage patient to self-assess achievements. • Avoid excessive augmented feedback. • Focus on use of variable feedback (summed, fading, bandwidth) designs to improve retention. Organize practice. • Encourage consistency of performance. • Focus on variable practice order (serial or random) of related skills to improve retention. Structure environment. • Progress toward open, changing environment. • Prepare the learner for home, community, work environments.
Autonomous Stage Characteristics	**Training Strategies**
The learner practices movements, continues to refine motor responses, spatial and temporal highly organized, movements are largely error-free, minimal level of cognitive monitoring "*How to succeed*" decision	Assess need for conscious attention, automaticity of movements. Select appropriate feedback. • Learner demonstrates appropriate self-evaluation, decision making skills. • Provide occasional feedback (KP, KR) when errors evident. Organize practice. • Stress consistency of performance in variable environments, variations of tasks (open skills). • High levels of practice (massed practice) are appropriate. Structure environment. • Vary environments to challenge learner. • Ready the learner for home, community, work environments. Focus on competitive aspects of skills as appropriate, e.g., wheelchair sports.

Highly skilled individuals who have been successfully discharged from rehabilitation can be expert models. Their success in returning to the "real world" will also have a positive effect in motivating patients new to rehabilitation. For example, it is very difficult for a therapist with full use of muscles to accurately demonstrate appropriate transfer skills to an individual with C6 complete tetraplegia. A former patient with a similar level injury can accurately demonstrate how the skill should be performed. Demonstration has also been shown to be effective in producing learning even with unskilled patient models. In this situation, the learner/patient benefits from the cognitive processing and problem solving used while watching the unskilled model attempt to correct errors and arrive at the desired movement.[12] Demonstrations can be live or videotaped. Developing a video library of demonstrations of skilled former patients is a useful strategy to ensure availability of effective models.

During initial practice, the therapist should give clear and concise verbal instructions and not overload the patient with excessive or wordy commands. It is important to reinforce correct performance and intervene when movement errors become consistent or when safety is an issue. The therapist should *not* attempt to correct all the numerous errors that characterize this stage but rather allow for some trial-and-error learning. Feedback, particularly visual feedback, is important during early learning. The learner should be directed to watch the movements closely. The learner's initial performance trials can also be recorded for later viewing and analysis.

Guidance

Guidance involves physically assisting the learner during the task. It is effective during early learning in improving performance of an unfamiliar skill by preventing or limiting errors. The therapist's hands can effectively substitute for missing elements, holding part of a limb stable while constraining unwanted movements and guiding the patient toward correct performance.[13] It also allows the learner to experience the tactile and kinesthetic inputs inherent in task performance, that is, to learn the "sensations of movement."[14] The supportive use of hands can allay fears and instill confidence while ensuring safety. The key to success in using guided movements is to intersperse active practice with guided movements, providing only as much assistance as needed and removing assistance as soon as possible. As manual guidance is reduced, verbal guidance can be increased. Overuse of guided movements is likely to result in dependence on the therapist for assistance, thus

becoming a "crutch." Guidance is most effective for slow postural responses (positioning tasks) and less effective during rapid or ballistic tasks.[1] Once guidance is removed, studies have shown that performance gains are not well maintained on retention tests.[15]

Active Decision Making

As learning progresses, the patient should be actively involved in self-monitoring, analysis, and self-correction of movements. The therapist can prompt the patient in early decision making by posing key questions. Specifically, the patient can be asked:

- What is the intended outcome of movement?
- What problems were present during the movement?
- What do you need to do to correct the problems in order to achieve a successful outcome?
- For complex movements, what are the components or steps of the task?
- How should the components be sequenced?

The therapist should confirm the accuracy of the patient's responses. If movement errors are consistent, the patient's efforts can be redirected. For example, if the patient consistently falls to the right while standing, questions can be more directed (In what direction did you fall? What do you need to do to correct this problem?). The therapist can also use augmented cues (i.e., tapping or light resistance) to assist the patient in correcting postural responses. The development of decision-making skills is critical in ensuring continued learning.

Strategy Refinement

During the associated and autonomous phases of learning, the patient continues to refine movement strategies with high levels of practice. Random errors decrease. As consistent errors are identified, solutions are generated. The focus is on refinement of skills and movement consistency in varied environments. This will ensure an overall range of movement patterns that are adaptable and match the changing demands of open environments. The patient's attention should be now focused on proprioceptive feedback, the "feel of the movement." Thus, the patient is directed to attend to the sensations intrinsic to the movement itself and to associate those sensations with the motor actions. Guided movements and facilitation techniques are counterproductive at this stage because they maintain dependence on the therapist and detract from active control. During late-stage learning, the use of distracters such as ongoing conversation or dual task training (e.g., ball skills during standing and walking) can yield important evidence of a developing level of autonomous control. It is important to remember that many patients undergoing active rehabilitation do not reach the final stage of learning. For example, in patients with traumatic brain injury, performance may reach consistent levels within structured environments, while safe, consistent performance in more open environments is not possible.

Feedback

The vast body of motor learning and therapeutic literature stresses the critical role of feedback in promoting motor learning. Feedback can be either **intrinsic**, occurring as a natural result of the movement, or provided by **extrinsic**, **augmented** sensory cues not typically received in the task. Proprioceptive, visual, vestibular, and cutaneous signals are examples of types of intrinsic feedback, while visual, auditory, or tactile cues are forms of extrinsic feedback (e.g., verbal cues, manual cues, biofeedback devices—EMG, pressure-sensing devices—forceplates, foot pad). During therapy, both intrinsic and extrinsic feedback can be manipulated to enhance motor learning. *Concurrent feedback* is given during task performance while *terminal feedback* is given at the end of task performance. Augmented feedback about the end result or overall outcome of the movement is termed **knowledge of results (KR)**. Augmented feedback about the nature or quality of the movement pattern produced is termed **knowledge of performance (KP)**.[1, p 465] The relative importance of KP and KR varies according to the skill being learned and the availability of feedback from intrinsic sources.[16–20] For example, tracking tasks are highly dependent on intrinsic visual and kinesthetic feedback (KP) while KR has less influence on the accuracy of the movements. In other tasks (e.g., transfers) KR provides the key information about how to shape the overall movements for the next attempt while KP may not be as useful. Performance cues (KP) should focus on key task elements that lead to a successful final outcome. Clinical decisions about feedback include:

- What type of feedback should be employed (mode)?
- How much feedback should be used (intensity)?
- When should feedback be given (scheduling)?

Choices about type of feedback involve the selection of which intrinsic sensory systems to highlight, what type of augmented feedback to use, and how to pair extrinsic feedback to intrinsic feedback. The selection of sensory systems depends on specific examination findings of sensory integrity. The sensory systems selected must provide accurate and usable information. If an intrinsic sensory system is impaired and provides distorted or incomplete information (e.g., impaired proprioception with diabetic neuropathy) then use of alternate sensory systems (vision) should be emphasized. Supplemental augmented feedback can be used to enhance learning. Decisions are also based on stage of learning. Early in learning, visual feedback is easily brought to conscious attention and therefore is important. Less consciously accessible sensory information such as proprioception is more useful during the middle and end stages of learning.

Decisions about frequency and scheduling of feedback (when and how much) must be reached. Frequent feedback (e.g., given after every trial) quickly guides the learner to the correct performance but slows retention. Conversely, feedback that is varied (not given after every trial) slows initial acquisition of the skill while improving performance on a

retention test.[21–25] This is most likely due to the increased depth of cognitive processing that accompanies the variable presentation of feedback. Varied feedback schedules include (1) *summed feedback*, feedback given after a set number of trials (e.g., after every other trial or every third trial); (2) *faded feedback*, feedback given at first after every trial and then less frequently (e.g., after every second trial, progressing to every fifth trial); and (3) *bandwidth feedback*, feedback given only when performance is outside a given error range. *Delayed feedback*, feedback given after a brief time delay (e.g., a 3-second delay), can also be beneficial in allowing the learner a brief time for introspection and self-assessment.[26] In contrast, the therapist who bombards the patient immediately after task completion with excessive augmented verbal feedback may preclude active information processing by the learner. The patient's own decision-making skills are minimized, while the therapist's verbal skills dominate. Winstein[27] points out that this may well explain why many studies on the effectiveness of therapeutic approaches cite minimal carryover and limited retention of newly acquired motor skills. A feedback delay interval that is prolonged or filled with practice of other movements results in interference that may decrease learning. Finally, the withdrawal of augmented feedback should be gradual and carefully paired with the patient's efforts to correctly utilize intrinsic feedback systems.

Practice

The second major influence on motor learning is **practice**. In general, the more the practice, the greater the learning. The therapist's role is to ensure that the patient practices the desired movements. Practice of incorrect movement patterns can lead to a negative learning situation in which "faulty habits and postures" must be unlearned before the correct movements can be mastered. The organization of practice will depend on several factors, including the patient's motivation, attention span, concentration, endurance, and the type of task. An additional factor is the frequency of allowable therapy sessions, which is often dependent on hospital scheduling and availability of services and payment. For outpatients, practice at home is highly dependent on motivation, family support, and suitable environment.

Clinical decisions about practice include:

- How should practice periods and rest periods be spaced (distribution of practice)?
- What tasks and task variations should be practiced (variability of practice)?
- How should the tasks be sequenced (practice order)?
- How should the environment be structured (closed vs open)?

Massed versus Distributed Practice

Massed practice refers to a sequence of practice and rest times in which the rest time is much less than the practice time.[1, p 465] Fatigue, decreased performance, and risk of injury are factors that must be considered when using massed practice. **Distributed practice** refers to spaced practice intervals in which the practice time is equal to or less than the rest time.[1, p 463] Although learning occurs with both, distributed practice results in the most learning per training time although the total training time is increased.[1] It is the preferred mode for many patients undergoing active rehabilitation who demonstrate limited performance capabilities and endurance. With adequate rest periods, performance can be improved without the interfering effects of fatigue or increasing safety issues. Distributed practice is also of benefit if motivation is low or if the learner has a short attention span, poor concentration, or motor planning deficits (e.g., dyspraxia). Distributed practice should also be considered if the task itself is complex, long, or has a high energy cost. Massed practice can be considered when motivation and skill levels are high and when the patient has adequate endurance, attention, and concentration. For example, the patient with spinal cord injury in the final stages of rehabilitation may spend long practice sessions acquiring the wheelchair skills needed for community access.

Blocked versus Random Practice

Blocked practice refers to a practice sequence organized around one task performed repeatedly, uninterrupted by practice of any other task.[1, p 462] **Random practice** refers to a practice sequence in which a variety of tasks are ordered randomly across trials.[1, p 466] While both allow for motor skill acquisition, random practice has been shown to have superior long-term effects in terms of retention.[28–30] For example, a variety of different transfers (e.g., bed to wheelchair, wheelchair to toilet, wheelchair to tub transfer seat) can be practiced all within the same training session. Although skilled performance of individual tasks may be initially delayed, improved retention of transfer skills can be expected. The constant challenge of varying the task demands provides high *contextual interference* and increases the depth of cognitive processing through retrieval practice from memory stores. The acquired skills can then be applied more easily to other task variations or environments. Constant practice will result in superior initial performance because of low contextual interference and may be required in certain situations (e.g., the patient with traumatic brain who requires a high degree of structure and consistency for learning).

Practice Order

Practice order refers to the sequence in which tasks are practiced. *Blocked order* refers to the repeated practice of a task or group of tasks in order (three trials of task 1, three trials of task 2, three trials of task 3: 111222333). *Serial order* refers to a predictable and repeating order (practice of multiple tasks in the following order: 123123123). *Random order* refers to a nonrepeating and nonpredictable order (123321312). Although skill acquisition can be achieved with all three, differences have been found. Blocked order produces improved early acquisition of skills (performance) while serial and random order produce better retention and generalizability of skills. This is again due to contextual

interference and increased depth of cognitive processing.[31,32] The key element here is the degree to which the learner is actively involved in memory retrieval. For example, a treatment session can be organized to include practice of a number of different tasks (e.g., forward-, backward- and sidestepping and stairclimbing). Random ordering of the tasks may initially delay acquisition of the desired stepping movements but over the long term will result in improved retention and generalizability.

Mental Practice

Mental practice is a practice strategy in which performance of the motor task is imagined or visualized without overt physical practice.[1, p 465] Beneficial effects result from the cognitive rehearsal of task elements. It is theorized that underlying motor programs for movement are activated but with subthreshold motor activity.[1] Brain mapping techniques have also revealed activation of similar brain areas during imagined movements as those activated during actual movement.[33,34] Mental practice has consistently been found to facilitate the acquisition of motor skills.[35–38] It should be considered for patients who fatigue easily and are unable to sustain physical practice. Mental practice is also effective in alleviating anxiety associated with initial practice by previewing the upcoming movement experience. Mental practice when combined with physical practice has been shown to increase the accuracy and efficiency of movements at significantly faster rates than physical practice alone.[39] When using mental practice, it is important to make sure the patient understands the task and is actively rehearsing the correct movements. This can be ensured by having the patient verbalize aloud the steps being rehearsed. It is generally contraindicated in patients with profound cognitive, communication, and/or perceptual deficits.

Part versus Whole Practice

Complex motor skills can be broken down into component parts for practice. The component parts are practiced before practice of the whole task is attempted. For example, during initial wheelchair transfer training the transfer steps are practiced in isolation before practicing the whole transfer (e.g., locking the brakes, lifting the foot pedals, moving forward in the chair, standing up, pivoting, and sitting down). It is important to identify the key steps through accurate task analysis and to sequence them in the required order. It is also important to practice the integrated whole in conjunction with the parts practice so that the learner develops the whole idea for the required task (i.e., cognitive map). Delaying practice of the integrated whole can interfere with transfer effects and learning.[1] Part–whole practice is most effective with discrete or serial motor tasks that have highly independent parts. Part–whole practice is not as effective for continuous movement tasks (e.g., walking) or for complex tasks with highly integrated parts. Both require a high degree of coordination with spatial and temporal sequencing of elements. For these tasks, practice of the integrated whole will result in superior learning.

Transfer of Learning

Transfer of learning refers to the gain (or loss) in the capability of task performance as a result of practice or experience on some other task.[1, p 436] Learning can be promoted through practice using contralateral extremities, termed **bilateral transfer**. For example, a patient with stroke first practices the desired movement pattern using the more normal (unaffected) extremity. This initial practice enhances formation or recall of the necessary motor program, which can then be applied to the opposite, involved extremity. This method cannot, however, substitute for lack of movement potential of the affected extremities (e.g., a flaccid limb on the hemiplegic side). Transfer effects are optimal with similarity of the tasks (e.g., identical components and actions) and environments.[40] For example, optimal transfer can be expected with practice of an upper extremity flexion pattern first on one side, then with an identical pattern on the other side.

Practice of *lead-up activities* is commonly used in physical therapy. Lead-ups are simpler task versions of a required complex task. The subtasks are practiced, typically in easier postures with significantly reduced degrees of freedom. Anxiety is also reduced and safety is ensured. Thus initial upright postural control can be practiced in kneeling, half-kneeling, or plantigrade before standing. The patient develops the required hip extension/abduction stabilization control required for upright stance but without the demands of the standing position or fear of falling. The more closely the lead-ups (subskills) resemble the final task, the better the transfer.

Closed versus Open Environments

Altering the environmental context is an important consideration in structuring practice sessions. Early learning benefits from practice in a stable or predictable, *closed environment.* As learning progresses the environment should be varied, and incorporate more variable features consistent with real world, *open environments.* Practicing walking only within the physical therapy clinic might lead to successful performance in that setting (context-specific learning) but does little to prepare the patient for ambulation at home or in the community. The therapist should begin to gradually modify the environment as soon as performance becomes consistent. It is important to remember that some patients (e.g., a patient with traumatic brain injury and limited recovery) may never be able to function in anything but a highly structured environment.

Functional Skills

The development of functional skills is a continuously evolving process that proceeds throughout the lifespan. Foundational skills are learned in infancy and childhood with the emergence of specific markers of developmental

maturation.[41–44] These skills are often referred to as *developmental motor skills* although they remain *essential functional skills* throughout the lifespan. Examples include rolling, supine-to-sit or sit-to-stand, maintaining stability or moving in progressively more challenging antigravity postures (i.e., sitting, kneeling, standing), upper extremity manipulation, and locomotion. Essential functional skills can be grouped into four broad categories of motor skills: mobility, static postural control (stability), dynamic postural control (controlled mobility), and skill. See Table 13.2 for a description of these categories together with examples of activities/postures and impairments. These activities and postures are a focus of functional training during rehabilitation. Careful attention to the demands of the postures can effectively address the degrees of freedom problem in controlling body segments. For example, the prone-on-elbows posture focuses on development of shoulder, upper trunk, and head control while eliminating all demands for movement control in the lower body. Because the center of mass (COM) is low and the base of support (BOS) wide it is inherently safe. Kneeling and half-kneeling postures can be used to improve trunk and hip control without the demands for control of the knee and ankle. As with prone-on-elbows posture, the low COM and wide BOS reduce the likelihood of falls and injury. Table 13.3 presents a list of developmental postures and possible treatment benefits. It is clear that considerable variability exists in the development and refinement of these motor skills in both children and adults and a specific sequence for acquisition of skills cannot be applied.

Age-related factors result in modification and adaptation of functional skills in adults.[45–48] All stages of information processing are affected by aging.[49] Sensory losses (decline in receptor sensitivity, recognition, and sensory encoding) affect stimulus identification. Response selection and programming are also affected by CNS changes.[50] An age-related slowing of movements is well documented with increases noted in both reaction and movement times.[51] Coordination changes result from changes in motor unit size with deficits particularly noticeable in fine motor control. Older adults are also more sensitive to complexity of movement.[52,53] The principle of *speed–accuracy tradeoff* typically applies as adults age, that is, the accuracy of a movement is decreased as its speed is increased. To accommodate for this change, older adults typically move slower, especially when accuracy is required. Decreasing levels of cardiovascular fitness and strength and increased weight commonly associated with a sedentary lifestyle can also affect the performance of motor skills.[54] Finally, older adults often experience multiple disease pathologies that affect their ability to move and learn.[55] For example, an older adult may alter the method used to roll over and sit up secondary to an increase in body weight, a decrease in overall strength and fitness, or an emerging pathology such as Parkinson's disease.

Table 13.2 Motor Skills

Categories	Characteristics	Examples	Impairments
Mobility	Ability to move from one position to another	Rolling; supine-to-sit; sit-to-stand; transfers	Failure to initiate or sustain movements through the range; poorly controlled movements
Static postural control (stability, static equilibrium, or static balance)	Ability to maintain postural stability and orientation with the COM over the BOS with the body not in motion	Holding in antigravity postures: prone-on-elbows, quadruped, sitting, kneeling, half-kneeling, plantigrade, or standing	Failure to maintain a steady body position; excessive postural sway; wide BOS; high guard position or handhold; loss of balance
Dynamic postural control (controlled mobility, dynamic equilibrium, or dynamic balance)	Ability to maintain postural stability and orientation with the COM over the BOS while parts of the body are in motion	Weight shifting and reaching in any of the above postures	Failure to control posture during weight shifting or reaching tasks; loss of balance
Skill	Ability to consistently perform coordinated movement sequences for the purposes of investigation and interaction with the physical and social environment	Upper extremity reach and manipulation Bipedal ambulation	Poorly coordinated movements; lack of precision, control, consistency, and economy of effort

BOS = base of support; COM = center of mass.

Table 13.3 Neurodevelopment Postures and Potential Treatment Benefits

Posture	Treatment Benefits
1. Prone-on-elbows	• Improve upper trunk, UE, and neck/head control • Weightbearing through shoulders, elbows flexed • Increase extensor ROM at hip extensors • Improve head/neck and shoulder stabilizers strength • Wide BOS, low COG
2. Quadruped	• Improve upper trunk, lower trunk, LE, UE, and neck/head control • Weightbearing through hips and shoulders and extended UEs • Improve hip, shoulder, and elbow stabilizers strength • Decrease extensor tone at knees by prolonged weightbearing • Decrease flexor tone at elbows, wrists, and hands by prolonged weightbearing • Increase extensor ROM at elbows, wrists and fingers • Wide BOS, low COG
3. Bridging	• Improve lower trunk and LE control • Increase hip stabilizers strength • Weightbearing through feet and ankles • Lead-up activity for bed mobility, sit-to-stand • Wide BOS, low height of COM
4. Sitting	• Improve upper trunk, lower trunk, LE, and head/neck control • Weightbearing in upright, antigravity position; can include weightbearing through extended UEs • Functional posture, important for reaching and ADL skills • Improve balance reactions • Medium BOS, medium height of COM
5. Kneeling and half-kneeling	• Improve head/neck, upper trunk, lower trunk, and LE control • Weightbearing through hips in upright, antigravity position • Decrease extensor tone at knees by prolonged weightbearing • Increase hip and trunk stabilizers strength • Improve balance reactions • Weightbearing through ankle in half-kneeling • Narrow BOS, intermediate height of COM (kneeling) • Wide BOS, intermediate height of COM (half-kneeling)
6. Modified plantigrade	• Improve head/neck, upper trunk, lower trunk, and UE and LE control • Weightbearing through extended UEs and LEs, upright antigravity position • Improve balance reactions • Functional posture, lead-up for standing, stepping and reaching • Decrease tone in elbow, wrist, and finger flexors by prolonged weightbearing • Increase extensor ROM at wrists and fingers • Wide BOS, high COM
7. Standing	• Improve head/neck, upper trunk, lower trunk, and LE control • Weightbearing through extended LEs, full upright, antigravity position • Improve balance reactions • Functional posture, important for ADL skills; lead-up for gait • Narrow BOS, high COM

BOS = base of support; COM = center of mass; LE = lower extremity; ROM = range of motion; UE = upper extremity;

Recovery

Recovery is the "re-acquisition of movement skills lost through injury."[2, p 23] In complete recovery the performance of the reacquired skills is identical in every way to preinjury performance. It is far more likely that the individual with CNS insult will demonstrate recovery using preinjury skills that are modified in some way. **Compensation** is defined as "behavioral substitution, that is, alternative behavioral strategies are adopted to complete a task."[2, p 38] For example, the patient recovering from stroke learns to dress independently using the nonaffected upper extremity; the patient with a complete T1 level spinal cord injury is taught to roll using upper extremities and momentum.

Immediately after insult, a cascade of events occurs, producing a prolonged but reversible depression of neuronal activity. Changes at a cellular level occur in the immediate area of damaged brain tissue. Disruption of the blood–brain barrier results in edema, with an accumulation of intracellular fluid and leakage of blood cells, proteins, and other toxic substances that disrupt nerve function. There is a release of neurotransmitters, glutamate, and calcium that activate enzymes associated with neuron death and neuronal degeneration. Free radical damage from toxic particles of oxygen and iron also are associated with cell death. *Denervation supersensitivity,* defined as postsynaptic neuronal hypersensitivity, results in decreased synaptic efficiency. Changes also occur in areas remote from the injured brain. Blood flow changes suggestive of depressed neural activity have been found to exist on both sides of the brain and in both cortical and subcortical structures, areas that are remote from the injured site.[56] *Injury-related cortical reorganization* is evidenced by a reduction in motor cortex excitability of the involved areas, a decrease in the cortical representation area of paretic muscles, and impairment of motor function.[57–60]

Brain injury was for a long time thought to be permanent with little potential for brain repair and recovery. This is now viewed as incorrect and can represent a dangerous *self-fulfilling prophecy* when applied to the individual who suffers from such injuries. **Neuroplasticity** (plasticity) has been defined as "the ability of the brain to change and repair itself."[56, p 134] Mechanisms of neuroplasticity include neuroanatomical, neurochemical, and neuroreceptive changes. Anatomical changes include nerve growth (*neural regeneration*). Trophic molecules (*nerve growth factors*) have been shown to play a key role in growth and repair processes. Nerve cells also change their interactions with each other, with physiological changes occurring at the level of the synapses. *Regenerative synaptogenesis* refers to sprouting of the injured axons to innervate (reclaim) previously innervated synapses. *Reactive synaptogenesis (collateral sprouting)* refers to the reclaiming of synaptic sites of the injured axon by dendritic fibers from neighboring axons. Neurotransmitter release and receptor sensitivity are improved (*synaptic plasticity*). Changes in synaptic strength, *long-term potentiation (LTP),* firm up neuronal connections and serve as a basis for all memory and learning. It is important to remember that these neuroplastic changes may be adaptive (functional) or maladaptive (nonfunctional). Neural regeneration, repair, and reorganization is a topic of intense ongoing research. For excellent reviews, the reader is referred to the work of Stein, Brailowsky, and Will[56] and Ploughman.[61]

Recovery can be categorized into two main types: (1) *spontaneous recovery* resulting from repair processes occurring immediately after the insult and (2) **function-induced recovery**, the neural reorganization that occurs as a result of increased use of involved body segments in behaviorally relevant tasks. Initial spontaneous recovery is influenced by the return to function of undamaged parts of the brain with the resolution of temporary blocking factors (i.e., shock, edema, decreased blood flow, decreased glucose utilization). This process has been termed *diaschisis* and takes place over a relatively short time frame, typically 3 to 4 weeks. For example, the patient with cerebral edema following stroke is likely to demonstrate early worsening of clinical signs as edema develops followed by spontaneous improvement within a few weeks as the edema resolves.

Function-Induced Recovery

Function-induced recovery (*use-dependent cortical reorganization*) refers to the ability of the nervous system to modify itself in response to changes in activity and the environment. It is important to remember that the brain is organized with parallel and distributed circuits that provide multiple inputs to many areas and overlapping function. Different and underutilized areas of the brain (e.g., cortical supplementary and association areas) can take over the functions of damaged tissue, a process that has been called *vicariance.* Another possibility is that the CNS has backup or fail-safe systems (parallel cortical maps) that become operational when the primary system breaks down. The unmasking of new, redundant neuron pathways permits cortical map reorganization and maintenance of function. Whole different areas of the brain are also capable of becoming reprogrammed, a process termed *substitution.* An example of substitution is the increased sensitivity of the hands as a sensory information system for the person who becomes blind. In this example, the changes in sensory strategy lead to structural reorganization within the brain. Newer techniques in brain mapping have led to better understanding of these processes. These include (1) positron emission tomography (PET) scanning used to measure regional cerebral blood flow (rCBF), (2) focal transcranial magnetic stimulation (TMS) used to measure responses in motor cortical regions to focal magnetic field stimulation, and (3) functional magnetic resonance imaging (fMRI) used to measure small changes in blood flow during brain activation.[57–59] Recovery

is a complex and dynamic process, and likely involves all of the above processes.

There is an accumulating body of research on **constraint-induced movement therapy** (*CI therapy* or *forced-use therapy*) in patients following stroke that has demonstrated significant and large improvements of upper extremity (UE) function.[62–71] Treatment-induced cortical reorganization has been demonstrated in studies using TMS[64] and fMRI.[65] Box 13.1 presents a summary of evidence from selected research in this area. Two factors are critical to the successful outcomes achieved in these studies. The first is the concentrated and repetitive practice of the involved UE. Training was intensive (averaging 6 hours/day) and focused on practice of common functional tasks. All subjects started with some voluntary movement (wrist and finger extension) in their affected limb. Second, movement was restricted in the sound UE through the use of mitts or splints and slings for up to 90 percent of waking hours. Behavioral shaping techniques (operant conditioning) were used in which the patients were rewarded for improvement with verbal reinforcement but not blamed (punished) for failure. The tasks were selected and tailored to address the specific motor deficits of the patient and ordered to allow for improving movement control and appropriate rest intervals.

Locomotor training using partial body weight support (BWS), a treadmill (TM), and manually assisted limb movements has also been shown to promote function-induced recovery.[72–75] As in CI therapy, practice is intense and task-specific. The limbs are maximally loaded to tolerance while movements are coordinated to stimulate actual walking. Compensatory strategies are minimized or eliminated. Training is progressed by decreasing the amount of loading (body weight support) and assistance and moving toward overground and community ambulation. See discussion in Chapter 14 and Evidence Summary Boxes in Chapters 18 and 23.

Effect of the Environment on Recovery of Function

Beneficial effects on brain function (increased cortical depth, brain weight, dendritic branching, and enzyme activity) have been demonstrated in rodents exposed to enriched environments.[76–79] The enriched environments consisted of manipulative toys for play, and structures for climbing, running, or swinging. In contrast, intact rodents raised in impoverished or small cage environments did not demonstrate the same level of brain development. When lesions were induced in rodents, exposure to enriched environments and activity prior to surgical insult had a protective effect with greater sparing of function and improved recovery.[80,81] Lesioned rodents exposed post-surgery to enriched environments also demonstrated improved recovery and performance when compared to impoverished, rodents.[82–84] Finally, socialization influenced outcomes. Rats housed in social groups in enriched environments demonstrated superior recovery over isolated rats.[85] Animal studies have also revealed that there may exist an optimal time period for such exposure. When rats with brain lesions less than 7 days were given intensive training, additional neuronal injury was demonstrated. These findings suggest that during the early post-lesion period, the damaged brain may be vulnerable to the stress imposed by intense training with additional injury.[86]

In humans, an unfamiliar and unpredictable hospital or rehabilitation environment may contribute to depression, disorientation, and decline of function. This same environment may be overly structured and protective to the point that it contributes to learned helplessness and disuse.[87] Carr and Shepherd argue that poor recovery after stroke may be partially explained by the impoverished and nonchallenging environments that many individuals recovering from stroke are exposed to.[88–89] While there are few environmental studies, there is evidence that patients recovering from stroke who were treated on an acute stroke unit demonstrated better recovery and functional outcomes than patients who received a comparable amount of physical therapy while on a general medical unit.[90–92] As Carr and Shepherd[93] point out, an important consideration is the amount of "down time" patients typically experience while in rehabilitation. Upwards of 30 to 40 percent of the day can be spent in passive pursuits while time in therapy is limited (e.g., for patients receiving stroke rehabilitation, only 93 minutes/day were spent in physical therapy and occupational therapy).[94–96] During nontherapy time, there is often little attention to self-directed practice, thereby further limiting the potential for optimal recovery of function.

Framework for Intervention

Different neurorehabilitation approaches and therapeutic techniques have evolved for patients with disorders of motor function. Historically many practical treatment ideas have evolved from empirical knowledge and clinical practice. Theory has been applied to explain the success of these interventions and to organize them into a coherent treatment philosophy. Recent emphasis on *evidence-based practice* has resulted in increased efforts to validate therapeutic interventions through research. Figure 13.5 presents a framework of current neurorehabilitation intervention strategies.

Functional Training

Evidence from research on function-induced recovery has led to **functional/task-oriented training**.[62–75] Central to this approach is the idea that specific task-oriented training with extensive practice is essential to reacquiring skill and

(text continues on page 488)

Evidence Summary Box 13.1

Constraint-Induced Movement Therapy

Reference	Subjects	Design/ Intervention	Duration	Results	Comments
Levy, CE, et al[65] 2001	2 patients, post-stroke (3–4 months); post-rehab, with moderate motor deficit; convenience sample. IC: 20°, voluntary wrist ext. and 10° finger ext	Nonrandomized cohort design; CI training of affected UE with restrictive mitt worn on nonaffected hand; behavioral shaping techniques; Pre- and posttreatment testing using: WMFT MAL fMRI	6 hours/day, 5 days/week for 2 weeks	Significant and large improvement in motor function and performance time. fMRI : ↑ motor cortex activity near lesion site; ↑ bilateral hemisphere activation	Small N, no controls; Findings support use of CI to reverse deficits and prevent learning nonuse; fMRI is a useful tool to monitor recovery and treatment effects.
Liepert, J, et al[64] 2000	13 patients, post-stroke (>6 months); post-rehab, with moderate motor deficit; convenience sample; IC: 20°, voluntary wrist ext. and 10° finger ext; no balance or cognitive problems	Nonrandomized cohort design; CI training of affected UE with restrictive hand splint and sling worn on nonaffected UE during 90% of waking hours; behavioral shaping techniques; Pre- and posttreatment testing using: MAL TMS	6 hours/day, 4 days/week for 2 weeks	Significant and large improvement in motor functions; Significant enlargement of cortical motor ouput area in the affected hemisphere; Treatment gains maintained up to 6 months post-treatment.	Small N, no controls; Supports CI therapy as a powerful treatment for improving UE function post-stroke; significant brain changes seen with short time course.
Dromerick, A, Edwards, D; and Hahn, M[66] 2000	20 patients, post-stroke within 14 days of ischemic stroke, with moderate motor deficit and in active rehab; screening tools for inclusion: NIHSS MAS IC: acute stroke, preserved cognitive function, presence of UE protective response	Single randomized clinical trial; Treatment group: CI training of affected UE with restrictive hand mitt worn on nonaffected UE at least 6 hours/day; Control group: traditional OT treatment; behavioral shaping techniques. Pre- and posttreatment testing using: ARA	2 hours/day, 5 days/week for 2 weeks	Mean total ARA score was significantly higher in treatment group ($r = 0.66$); No difference in disability measure (BI).	Small *N*, with controls; Supports CI therapy as a powerful treatment for improving UE function post-stroke; Lower treatment intensities than other studies; BI not a sensitive measure of UE changes; Average age of control group was 10 years greater than treatment group; No long-term follow-up.

(*continued*)

Evidence Summary Box 13.1

Constraint-Induced Movement Therapy (continued)

Reference	Subjects	Design/ Intervention	Duration	Results	Comments
Blanton, S, and Wolf, S[67] 1999	Single patient 4 months post-stroke; post-rehab, with moderate motor deficit; Screening tools for inclusion: MAL MMSE IC: 10°, voluntary wrist ext. and finger ext; no balance or cognitive problems	Single case report design; CI training of affected UE with restrictive mitten worn on nonaffected UE during waking hours; behavioral shaping techniques; Pre- and posttreatment, 3 months follow-up testing using: WMFT MAL	6 hours/day 5 days/week for 2 weeks	Significant improvement of motor abilities and timed abilities on WMFT; ↑ self-report use of UE on MAS; Treatment gains maintained 3 months posttreatment.	Small *N*, no controls; Supports CI therapy as a powerful treatment for improving UE function post-stroke; May reverse effects of learned non-use.
Miltner, W, et al[68] 1999	15 patients, post-stroke, post-rehab. with moderate motor deficit (mean time = 5.1 years); Convenience sample; Screening tools for inclusion: EMG, EEG, MRI, TMS, Cognitive battery IC: 20°, voluntary wrist ext, 10° finger ext; no balance or cognitive problems	Nonrandomized cohort design; CI training of affected UE with restrictive hand splint and sling worn on nonaffected UE during 90% of waking hours; behavioral shaping techniques; Pre and post-treatment testing using: WMFT MAL	7 hours/day, 4 days/week for 12 days	Significant and very large improvement on motor tests (MAL, WMFT); Gains maintained 6 months post-treatment	Small *N*, no controls; Supports CI therapy as a powerful treatment for improving UE function post-stroke; Mean chronicity of 5.1 years discounts effects of spontaneous recovery or prior therapy.
Kunkel, A, et al[69] 1999	5 patients, post-stroke, post-rehab with moderate motor deficit (time post-stroke = 3–15 yrs); convenience sample. Screening tools for inclusion: MRI Cognitive battery IC: 20°, voluntary wrist ext, 10° finger ext; no cognitive problems	Nonrandomized cohort design; CI training of affected UE with restrictive hand splint and sling worn on nonaffected UE during 90% of waking hours; behavioral shaping techniques; Pre- and posttreatment testing using: WMFT MAL AMAT AAUT	6 hours/day 5 days/week for 2 weeks	Significant and large improvement in performance times (AMAT, WMFT); quality of movement (AMAT, WMFT, MAL); and use of UE in real world (AAUT); Gains maintained 3 mo. post-treatment	Small *N*, no controls; Supports CI therapy as a powerful treatment for improving UE function post-stroke; More than 100% in active use of UE.

Evidence Summary Box 13.1

Constraint-Induced Movement Therapy (continued)

Reference	Subjects	Design/ Intervention	Duration	Results	Comments
Van der Lee, J, et al[70] 1999	66 patients, post-stroke (median time = 3 years) with moderate motor deficit; Screening tools for inclusion: ARA IC: at least 1 year post-stroke; 20° voluntary wrist ext, 10° finger ext; no cognitive or balance problems	Single randomized clinical trial; Treatment group: CI training of affected UE with restrictive splint/sling worn on nonaffected UE; Control group: traditional PT treatment according to NDT method (bimanual training); behavioral shaping techniques; Pre- and posttreatment testing using: ARA RAP FMA (UE motor subtest) MAL	6 hours/day, 5 days/week for 2 weeks	Small and significant improvement in motor performance (dexterity measured by ARA) and use of UE (MAL); Gains in ARA maintained 1 yr. post-treatment	Larger *N* than other studies; Supports CI therapy as a treatment for improving UE function post-stroke; Benefits found in those patients with sensory disorders; Both RAP and FMA failed to reveal any differences suggesting inadequate responsiveness of instruments to chronic stroke.
Taub, E, et al[71] 1993	9 patients, post-stroke with moderate motor deficit (median post-stroke time = 4.1 years); Screening tools for inclusion: Cognitive tests IC: at least 1 year post-stroke; 20° voluntary wrist ext, 10° finger ext; no cognitive or balance problems	Single randomized clinical trial Treatment group: CI training of affected UE with restrictive splint/sling worn on nonaffected UE 90% of waking hours Control group: Received training strategies to focus attention on using affected UE; traditional PT (PROM) behavioral shaping techniques Pre- and posttreatment testing using: WMFT MAT	7 hours/day, 5 days/week for 2 weeks	Significant and large improvement in motor performance (performance time, quality of movement on WMFT) and use of UE (MAT); Gains maintained during 2-year follow-up.	Small *N*; Supports CI therapy as a treatment for improving UE function post-stroke; Long follow-up period Attention training of control group reduces likelihood improvement due to attention/placebo factors.

↑ = increased; AAUT = Actual Amount of Use Test (includes 21 items of self-report of arm use); AMAT = Arm Motor Ability Test (includes 13 complex tasks); ARA = Action Research Arm Test (includes 19 items of UE strength, dexterity, and coordination); BI = Barthel Index (measure of basic ADL and disability); CI = Constraint-Induced Movement Therapy: intensive, supervised task-specific practice of affected upper extremity (UE) with restriction of non-affected UE; EEG = electroencephalography; EMG = electromyography; Ext = extension; fMRI = functional magnetic resonance imaging; FMA = Fugl-Meyer Assessment Scale (stroke-specific instrument with upper extremity motor section); IC = inclusion criteria; MAL = Motor Activity Log (structured interview that identifies performance on 30 daily activities); MAS = Motor Assessment Scale (stroke-specific instrument); MMSE = Mini-Mental State Exam (Folstein); N = number of subjects; NDT = Neurodevelopmental Treatment; NIHSS = National Institutes of Health Stroke Scale (stroke-specific instrument); OT = occupational therapy; RAP = Rehabilitation Activities Profile (based on ICIDH, semistructured interview that assesses disabilities and handicaps and consists of 21 items in 5 domains); Rehab = rehabilitation; TMS = focal transcranial magnetic stimulation; WMFT = Wolf Motor Function Test, an UE functional test that includes 14 timed activities and 2 strength tests.

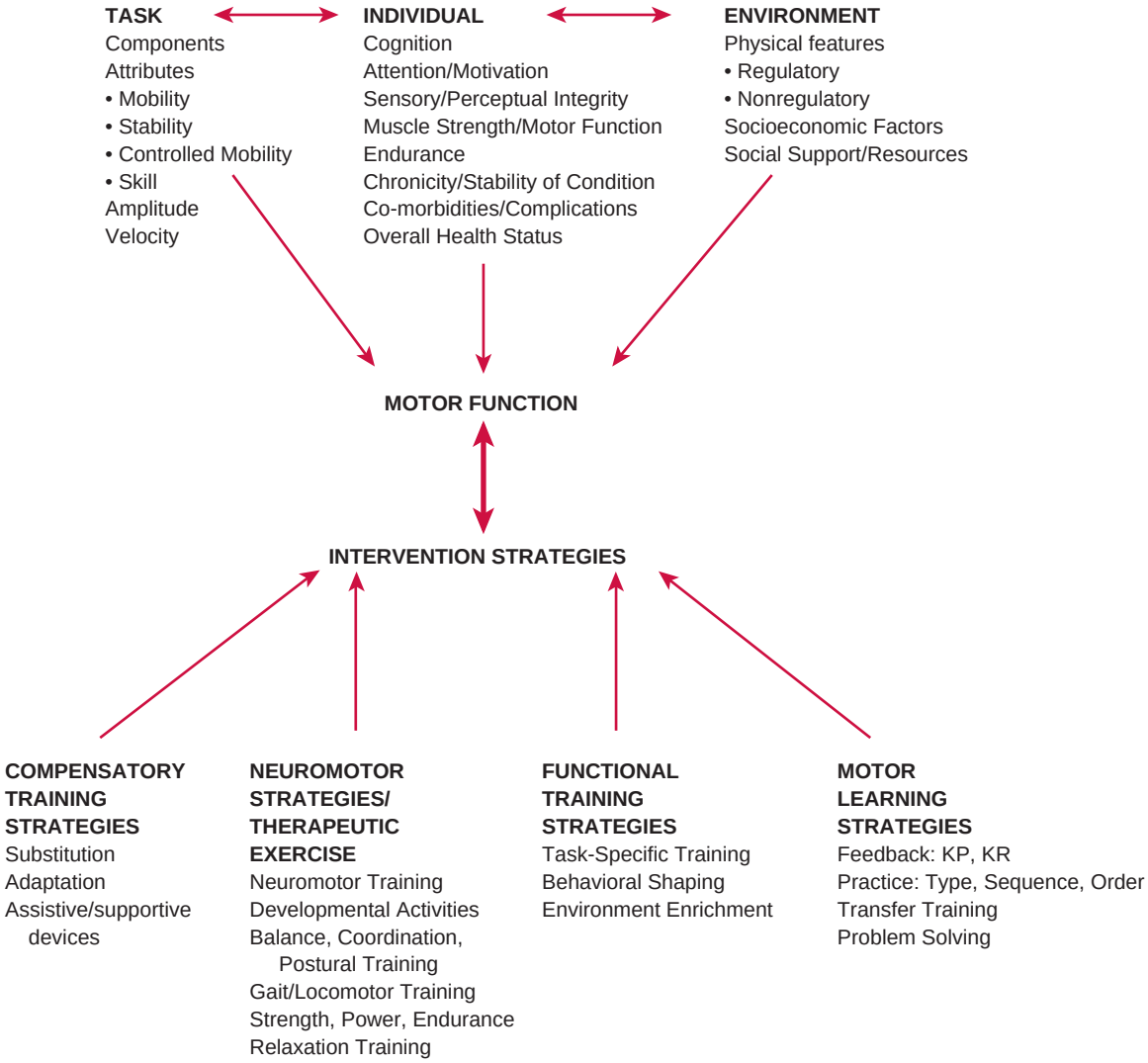

Figure 13.5 Motor function emerges from interaction among the task, the individual, and the environment. Deficits in motor function can be improved using *Neuromotor, Functional/Task Oriented, Motor Learning,* and/or *Compensatory* intervention strategies.

enhancing recovery. Theoretically, the interacting systems of the CNS are viewed as organized around essential functional tasks. Thus, an understanding of tasks, the essential elements within each task, and the context or environment in which tasks occur is key to structuring appropriate training (see Chapter 8, Box 8.1). Tasks important for daily function (e.g., grasp and release, standing and walking) are emphasized. Involved segments are targeted for training (i.e., constraint-induced movement therapy). Level of difficulty is varied and initial tasks are selected to ensure patient motivation and success. Motor learning strategies are used to enhance function, including **behavioral shaping techniques** that use reinforcement and reward to promote skill development. This approach represents a shift away from a traditional neurotherapeutic focus that utilizes extensive hands-on therapy (e.g., facilitated movements). While initial movements can be assisted or guided, active movements are the overall goal. The therapist serves as coach, providing appropriate feedback and encouraging the patient. Task-oriented training effectively counteracts the effects of immobility and the development of indirect impairments such as muscle weakness or loss of flexibility. It also prevents *learned nonuse* of the involved segments while stimulating CNS recovery. Box 13.2 presents a summary of task-oriented training strategies to promote function-induced recovery.

Task-oriented training is not an appropriate for every patient. Its selection is dependent on the degree of recovery and severity of motor deficits. Animal studies have suggested that early overemphasis on use-dependent training may increase the vulnerability of the brain to additional damage.[85] Patients who are not able to participate in task-oriented training include those who lack voluntary control or cognitive function. For example, a patient with traumatic brain injury who is in the early recovery stages has limited

Box 13.2 Task-Oriented Training Strategies to Promote Function-Induced Recovery

Focus on early training as soon as possible after brain damage to utilize specific windows of opportunity and avoid learned nonuse.

Consider the individual's past history, health status, age, and experience in designing appropriate, interesting and stimulating activities.

Involve the patient in goal-setting and decision making, thereby enhancing motivation and promoting active commitment to recovery.

Structure practice utilizing task-related training activities.
- Select tasks important for independent function; include tasks that are important to the patient.
- Identify the patient's abilities/strengths and level of recovery/learning; choose tasks that have potential for patient success.
- Target active movements involving the affected extremities; constrain or limit compensatory strategies.
- Utilize repetition and extensive (massed) practice.
- Include both supervised and unsupervised practice using an activity log.
- Assist (guide) the patient to successfully carry out initial movements as needed.
- Provide augmented verbal feedback and verbal reward for small improvements in task performance.
- Provide modeling of task performance as needed.
- Promote practice of variable behaviors to facilitate adaptation of skills.

Structure context-specific practice.
- Promote initial practice in a supportive environment, free of distractors.
- Progress to variable practice in real-world environments.

Maintain focus on role as *training coach* while minimizing hands-on therapy.

Continue to monitor recovery closely and document progress using valid and reliable functional outcome measures.

Be cautious about timetables and predictions as recovery may take longer than expected.

potential to succeed with this type of intensive training. Similarly, patients with stroke who experience profound upper extremity paralysis would not be eligible for constraint-induced (CI) UE training. One of the consistent exclusion criteria for CI therapy has been inability to perform voluntary wrist and finger extension of the involved hand. Thus threshold abilities to perform the basic components of the task need to be identified. Careful analysis of underlying impairments with a focus on intervention (e.g., strength, ROM) complements task-oriented training. For example, during locomotor training using BWS and a TM system, patients are guided in their stepping and pelvic motions into an efficient motor pattern. Patients need to demonstrate essential prerequisites of basic head and trunk stability during upright positioning in order to be considered appropriate candidates for this type of training.

Neuromotor Development Training

Neuromotor development training includes developmental activities training, motor training, movement pattern training, and neuromuscular education or reeducation. Neuromuscular/sensory stimulation techniques and therapeutic exercises are used to alter sensorimotor impairments and promote functional movements. The affected body segments are targeted for training while compensatory movements by intact segments are not allowed. A hands-on approach is used to stimulate, guide, or assist movements for correct performance.

Gordon[97] points out the dominant treatment philosophy at the time these approaches were developed was one of muscular reeducation (e.g., Kenny method), which could not be easily adapted to patients with CNS disorders. As therapists sought to develop more appropriate approaches for this group of patients, these approaches evolved. A central concept was the use of sensory inputs to modify the CNS and stimulate motor output. These approaches evolved in a time when hierarchical theory of motor control was the prevailing motor control theory during the 1950s and 1960s. Thus the goal of treatment was initially viewed as assisting the higher centers of the CNS in regaining control of motor function. Acquisition of function was viewed as modeling the acquisition of movement patterns seen in normal development. Thus a common base was developmental activities training (e.g., prone-on-elbows, quadruped, sitting). Following remediation of impairments (e.g., reduction of tone and abnormal reflexes) treatment progressed to functional movement patterns. Modifications in these approaches have emerged from the changing scientific evidence and theories of motor control. Currently there is a much greater emphasis on functional training. Motor learning strategies have always been a strong component. Two of the most popular approaches in current use are *Neurodevelopmental Treatment (NDT)* and *Proprioceptive Neuromuscular Facilitation (PNF).*

These approaches are not appropriate for every patient. Patients who demonstrate sufficient recovery and consistent voluntary movement would not benefit from neuromuscular

stimulation and an intensive hands-on approach. Rather these patients are candidates for active task-oriented training. Interventions organized around a behavioral goal provide a better vehicle for promoting continuing functional recovery and long-term retention than interventions that target remediation of a impairments (e.g., spasticity). Also, continued use of interventions (e.g., guided movement) may result in the patient becoming dependent on the therapist, a phenomenon appropriately labeled the "my therapist syndrome." For example, a therapist who is covering for another therapist hears the patient remark that he or she is not helping correctly. It has to be done the way "my therapist" does it. This patient is demonstrating an overreliance on the therapist for movement. In summary, these techniques can provide important choices for treatment. They can help the patient bridge the gap between absent or severely disordered movements and active movements. Once the patient develops independent voluntary control of movement, these treatment approaches are generally counterproductive.

Neurodevelopmental Treatment (NDT)

Neurodevelopmental treatment (NDT) is a treatment approach developed in the late 1940s and early 1950s by Dr. Karel Bobath, an English physician, and Berta Bobath, a physiotherapist.[14,98] Their work focused on patients with neurological dysfunction (cerebral palsy and stroke). The essential problems of these patient groups were identified as a release of abnormal tone (spasticity) and abnormal postural reflexes (primitive spinal cord and brainstem reflexes) from higher center CNS control with resulting loss of the normal postural reflex mechanism (righting, equilibrium, protective extension reactions) and normal movements. The role of sensory feedback was viewed as critical in inhibiting abnormal reactions and facilitating more normal movement patterns.

Current NDT has realigned itself with newer theories of motor control (systems theory and a distributed model of CNS control).[99] Many different factors are recognized as contributing to loss of motor function in patients with neurological dysfunction including the full spectrum of sensory and motor deficits (weakness, limited ROM, impaired tone and coordination). Emphasis is on the use of both feedback and feedforward mechanisms to support postural control. Postural control is viewed as the foundation for all skill learning. Normal development in children and normal movement patterns in all patients are stressed. The patient learns to control posture and movement through a sequence of progressively more challenging postures and activities. NDT uses physical *handling techniques* and *key points of control* (e.g., shoulders, pelvis, hands, and feet) directed at supporting body segments and assisting the patient in achieving active control. Sensory stimulation (facilitation and inhibition via primarily proprioceptive, and tactile inputs) is used during treatment. Postural alignment and stability are facilitated while excessive tone and abnormal movements are inhibited. For example, in the patient with stroke, abnormal obligatory synergy movements are restricted while out-of-synergy movements are facilitated. Activities are selected that are functionally relevant and varied in terms of difficulty and environmental context. Compensatory training strategies (use of the less involved segments) are avoided. Carryover is promoted through a strong emphasis on patient, family, and caregiver education. NDT is taught today in recognized training courses. Appendix A presents an overview of NDT principles and techniques.

Proprioceptive Neuromuscular Facilitation (PNF)

Motor function can be improved using *Proprioceptive Neuromuscular Facilitation (PNF)*, an approach initially developed by Dr. Herman Kabat and Maggie Knott (a physical therapist).[100] Synergistic patterns of movement were identified as components of normal movement. A developmental emphasis was added later by Voss to include practice of various different activities (rolling, prone-on-elbows, quadruped, kneeling, half-kneeling, modified plantigrade, standing, and gait). Extremity patterns of movement are rotational and diagonal in nature (labeled Diagonal 1 [D1] and Diagonal 2 [D2]) rather than straight plane movements. Coordination within and between patterns is stressed. For example, the technique of slow reversals is used to establish smooth linkages between agonist and antagonist actions during reversing patterns. Patterns can be unilateral or bilateral and combined with various trunk patterns and postures. A number of different facilitation techniques, largely proprioceptive, are utilized to facilitate movement (e.g., stretch, resistance, traction, approximation, and so forth). Precise manual contacts are used to provide important directional cues and enhance the function of underlying muscles. PNF also incorporates a number of important motor learning strategies (e.g., practice, repetition, visual guidance of movement, verbal commands, and so forth). It is directed at improving functional performance and coordinated patterns of movement and has been used effectively to treat patients with both neuromuscular or musculoskeletal deficits.[101] PNF is taught today in recognized training courses. Appendix B presents an overview of PNF principles and techniques.

Neuromuscular/Sensory Stimulation Techniques

A number of therapeutic techniques can be used to facilitate, activate, or inhibit muscle contraction. These have been collectively called facilitation techniques, although this term is a misnomer, because they also include techniques used for inhibition. The term **facilitation** refers to the enhanced capacity to initiate a movement response through increased neuronal activity and altered synaptic potential. An applied stimulus may lower the synaptic threshold of the alpha motor neuron but may not be sufficient to produce an observable movement response. **Activation** on the other hand refers to the actual production of a movement response and implies reaching a critical threshold level for neuronal firing. **Inhibition** refers to

the decreased capacity to initiate a movement response through altered synaptic potential. The synaptic threshold is raised, making it more difficult for the neuron to fire and produce movement. The combination of spinal inputs and supraspinal inputs acting on the alpha motor neuron (final common pathway) will determine whether a muscle response is facilitated, activated, or inhibited.

Several general guidelines are important. First, facilitative techniques can be additive. That is, several inputs applied simultaneously, such as quick stretch, resistance, and verbal commands commonly combined in PNF patterns, produce the desired motor response, whereas use of a single stimulus may not. This demonstrates the property of spatial summation within the CNS. Repeated application of the same stimulus (e.g., repeated quick stretches) may also produce the desired motor response owing to temporal summation within the CNS, whereas a single stimulus does not. Thus, stretch is used repeatedly to ensure that the patient with a weak muscle is able to move from the lengthened to the shortened range. The response to stimulation or inhibition is unique to each patient and dependent on a number of different factors, including level of intactness of the CNS, arousal, and the specific level of activity of the motoneurons in question. For example, a patient who is depressed and hypoactive may require large amounts of stimulation to achieve the desired response. Stimulation is generally contraindicated for the patient with hyperactivity while inhibition/relaxation techniques are of benefit. The intensity, duration, and frequency of simulation need to be adjusted to meet individual patient needs. Unpredicted responses can result from inappropriate application of techniques. For example, stretch applied to a spastic muscle may increase spasticity and negatively impact voluntary movement. Facilitation techniques are not appropriate for patients who demonstrate adequate voluntary control; they should be viewed primarily as a bridge to voluntary movement control. Appendix C presents an overview of neuromuscular/sensory stimulation techniques.

Compensatory Training

The focus of *compensatory training* is early resumption of functional skills using the uninvolved or less involved segments for function. For example, the patient with hemiplegia is taught to dress using the less-affected upper extremity; the patient with paraplegia regains functional mobility and wheelchair independence using the upper extremities. Central to this approach is the concept of substitution. The patient is made aware of movement deficiencies (cognitive awareness is developed). Changes are then made in the patient's overall approach to functional tasks. Alternate ways to accomplish the task are suggested, simplified, and adopted. The patient practices and relearns the task using the new pattern. The patient then practices the new pattern in the environment in which the function is expected to occur. Energy conservation techniques are incorporated to ensure the patient can successfully complete all daily tasks. A second central tenet of this approach is modification of the task and environment **(adaptation)** to facilitate relearning of skills, ease of movement, and optimal performance. For example, the patient with unilateral neglect is assisted in dressing by color coding of the shoes (red tape on the left shoe, yellow tape on the right shoe). The wheelchair brake toggle is extended and color coded to allow the easy identification by the patient.

One of the major criticisms of this approach is the focus on uninvolved segments may suppress recovery for certain patients and contribute to learned nonuse of impaired segments.[102] For example, the patient with stroke fails to learn to use the involved extremities. Compensatory training can also lead to development of *splinter skills,* which are skills acquired in a manner inconsistent with skills the individual already possesses. Splinter skills cannot be easily generalized to other task variations or to other environments.

A compensatory training approach may be the only realistic approach possible when recovery is limited or the patient presents with significant impairments and functional limitations with little or no expectation for additional recovery. Examples include the patient with complete spinal cord injury or the patient recovering from stroke with severe sensorimotor deficits and extensive co-morbidities (e.g., severe cardiac and respiratory compromise or memory deficits associated with Alzheimer's disease). The latter is severely limited in the ability to actively participate in rehabilitation and to relearn motor skills.

Integrating Approaches

An understanding of how the brain regulates movement is essential to making sound clinical decisions in selecting interventions. Therapists must be cautious not to develop a "disciple-like" adherence to one approach. Therapists who become certified in, and a proponent of, a particular approach must be particularly cautious about overreliance on a single group of treatment interventions. The diversity of problems experienced by patients with disordered motor function negates the idea that any one approach could be successful for all patients. As patients recover, their abilities and needs change. Therapists must be attuned to the patient's changing status and recognize anticipated goals and expected outcomes may change. A variety of interventions is likely to be the most effective approach in meeting the diverse needs of patients. A common base for all intervention is training to improve functional skills and motor learning. Interventions also need to promote adaptability of skills for function in real-world environments. In selecting interventions, the therapist must consider those that have the greatest chance of success. The choice of interventions must also take into consideration other factors, including ability to deliver care, cost-effectiveness in terms of length of stay and number of allotted physical therapy visits, age of the patient and number of co-morbidities, social support, and potential discharge placement. Examples of

Box 13.3 Examples of General Goals and Outcomes for Patients with Disorders of Motor Function

Impact of pathology/pathophysiology is reduced.
- Risk of recurrence of condition is reduced.
- Risk of secondary impairment is reduced.
- Intensity of care is decreased.

Impact on impairments is reduced.
- Alertness, attention, and memory are improved.
- Joint integrity and mobility are improved
- Sensory awareness and discrimination are improved.
- Motor control is improved.
- Coordination is improved.
- Muscle performance (strength, power, and endurance) is improved.
- Postural control and balance improved.
- Gait and locomotion are improved.
- Endurance is increased.

Ability to perform physical actions, tasks, or activities is improved.
- Functional independence in activities of daily living (ADL) and instrumental activities of daily living (IADL) is increased.
- Level of supervision for task performance is decreased.
- Tolerance of positions and activities is increased.
- Flexibility for varied tasks and environments is improved.
- Motor learning skills are improved.
- Decision making is improved.
- Safety of patient/client, family, and caregivers is improved.

Disability associated with acute or chronic illness is reduced.
- Ability to assume/resume self-care, home management, work (job/school/play), community, and leisure roles is improved.

Health status is improved.
- Sense of well-being is increased.
- Insight, self-confidence, and self-image are improved.
- Health, wellness, and fitness are improved.

Satisfaction, access, availability, and services are acceptable to patient/client.

Patient/client, family, and caregiver knowledge and awareness of the diagnosis, prognosis, anticipated goals/expected outcomes, and interventions are increased.

Adapted from *Guide to Physical Therapist Practice*[103]

general goals and outcomes for improving motor function adapted from the *Guide to Physical Therapist Practice*[103] are presented in Box 13.3. These can serve as a basis for developing specific anticipated goals and expected outcomes for an individual patient.

Intervention Strategies to Improve Motor Control

Strength, Power, and Endurance Training

Muscle performance is defined as "the capacity of a muscle or group of muscles to generate forces."[103, p 688] **Muscle strength** is the "muscle force exerted by a muscle or a group of muscles to overcome a resistance under a specific set of circumstances."[103, p 688] **Muscle power** is "the work produced per unit of time or the product of strength and speed."[103, p 688] **Muscle endurance** is "the ability to sustain forces repeatedly or to generate forces over a period of time."[103, p 688] Muscle performance is regulated by a number of factors, including motor unit recruitment, motoneuron firing patterns, muscle length and tension, muscle fiber composition, fuel storage and delivery, speed and type of contraction, and movement arm.[104] Techniques that optimize these factors while addressing the specific demands of the task and environment will yield maximum functional outcomes. Improved sense of well-being and confidence are also important outcomes.

Patients undergoing neurorehabilitation commonly present with disruption of motoneurons from central pathways, the direct result of upper motor neuron lesions. Weakness or paralysis can affect one side of the body (hemiparesis, hemiplegia), both lower limbs (paraparesis, paraplegia), all four limbs (tetraparesis, tetraplegia), or a single limb or segments of a limb. As recovery progresses, the status of muscle strength and performance may change (e.g., the patient recovering from incomplete spinal cord injury). In addition, prolonged periods of disuse and immobility result in diminished neural activity, atrophy, and weakness. Older adults typically demonstrate a preferential loss of type II fibers. It is also important to recognize that the patient may have been inactive prior to insult or injury resulting in preexisting deconditioning.

Muscle Strengthening

Strength training produces a number of neuromuscular changes. There is an increase in the production of maximal force due to changes in neural drive (increased motor unit recruitment, increased rate, and synchronization of firing rate) and changes in muscle (hypertrophy of muscle fibers, improved metabolic/enzymatic adaptations, increased size and number of myofibrils, muscle fiber type adaptation with conversion of type IIB to type IIA). Connective tissue tensile strength and bone mineral density are increased. Body composition is improved in terms of body mass ratio

of fat to lean. Reaction time, functional performance, and sense of well-being are also improved. The effectiveness of a strengthening program is dependent on achieving an adequate training stimulus. For strength and power training, an *exercise prescription* should include the following elements: *mode* of exercise (type of muscle contraction, application of resistance, arc of movement), *intensity* (exercise load or level of resistance), *frequency* (number of repetitions and sets; number of exercises per set), *rest interval* (recuperation time between sets and exercise sessions), and *duration* (total time of resistance training). Additional determinants include use of *correct alignment* and *stabilization* and consideration of replication of *functional demands.*[105]

Basic principles of effective strengthening programs include overload, specificity, cross training, and reversibility.[106] The loads placed on muscle must be greater than those normally incurred **(overload principle)**. Application of a progressive resistance sequence that achieves 80 percent maximum voluntary strength is a generally accepted criteria for effective strengthening. Free weights, pulley systems, elastic resistance bands, mechanical resistance machines, isokinetic dynamometers, and manual resistance all provide sources of external load on muscle. Training effects are specific to the mode of exercise stress imposed on the exercising muscles **(specificity principle)**. Thus, the training effects from an isometric protocol are specific to the exercising muscle and the point in the range that the muscle is holding. Effects do not carry over to improved dynamic performance (concentric or eccentric contractions). Nor will exercise training of the upper extremities transfer to improved lower extremity performance. **Cross training** refers to a training program that includes a variety of training elements (e.g., isometric, concentric, eccentric, and endurance). Cross training is used to place broadest possible demands on the neuromuscular system and overcome the effects of specificity. **Reversibility principle** refers to the failure to sustain the benefits of strength training if muscles are not regularly used in a maintenance program of resistance or functional exercises. Detraining effects include a reduction in muscle performance, decreased neural recruitment, and muscle fiber atrophy.[107]

Strength training methods have been well described and documented.[105,108] Selection of a particular training sequence must be based on the specific needs of the patient and the potential benefits of a particular method. For example, isometric training will result in gains in static strength without added joint motion. This may be important during early rehabilitation when pain is a factor or when postural stability is the focus of treatment. Dynamic exercise (concentric and eccentric) allows for joint movement and excursion of a body segment and is essential to develop the strength, power, and endurance required for functional mobility skills.

Patients with deficits in motor function may demonstrate deficits in muscle activation. Early training should focus on isometric and eccentric contractions because muscle tension is better maintained than with concentric contractions. This is due primarily to the improved peripheral reflex support of contraction as opposed to the spindle unloading that occurs as the muscle moves into the shortened range of a concentric contraction. The patient is initially asked to actively hold at midrange where the greatest tension can be generated. The patient is then asked to slowly lower the limb (an eccentric contraction) and hold (an isometric contraction). Once control is achieved in both of these types of contractions, concentric contractions can be attempted. For isotonic contractions prestretching the muscle by starting the contraction in the lengthened range optimizes tension development through increased use of viscoelastic forces *(length–tension relationship)* and peripheral reflex support. Weak muscles can be initially lightly resisted (*tracking resistance*) to facilitate contraction through proprioceptive loading of the muscle spindle. Control of velocity is also important to ensure efficiency of initial movement attempts. During concentric contractions, total tension decreases as velocity increases. Thus, patients may be able to generate a contraction at slow speeds but not at high speeds. For example, the patient with stroke who demonstrates limited control should be instructed to begin with slow and controlled movements. As movements become more efficient, they can be progressed to faster speeds.

Open-chain exercises involve the distal segment of the limb moving in space without simultaneous motions at adjacent joints. Muscle activation occurs predominately in the prime mover(s) crossing the moving joint. Resistance is applied to the distal moving segment, typically in nonweightbearing positions. *Closed-chain exercises* involve motions in which the distal part is fixed (foot or hand) while the proximal segments are moving (e.g., weight shifting in standing, bilateral short-arc squats). They are performed in weightbearing postures and involve simultaneous actions of synergistic muscles at multiple joints. The added joint approximation and stimulation of joint and muscle proprioceptors enhances neuromuscular control and joint stabilization (co-contraction). A limitation of closed chain exercise is the substitution of other agonist muscles for specific muscle weakness. In comparison, open-chain exercises can be used to isolate contraction of a muscle or muscle group and enhance specific training.[105] *Pylometric training* involves quick powerful movements with prestretching of the muscle for improved neuromuscular responses (using stretch-shortening principles). For example, when performing a vertical jump, slightly lowering the body in a partial squat first provides increased stretch on the quadriceps and heelcords and enhances the push-off. Plyometric exercise drills are used during the advanced stages of rehabilitation to prepare individuals for explosive movements required for certain sports.[108]

Gains in strength can be obtained through progressive resistive exercises (PRE) using free weights or fixed mechanical resistance machines. A major disadvantage of PRE is that the weight selected is determined by the amount

that can be lifted by the muscle at the weakest point of the range. Isokinetic training devices offer the advantage of providing accommodating resistance throughout the range. Muscle performance is therefore not limited to the weakest part of the range. The amount of force generated is recorded, providing an important objective measure of performance. Different isokinetic protocols using concentric and eccentric contractions have been developed. The speed of movement can be predetermined. This is an important consideration for training the patient who demonstrates neuromuscular impairments in timing and velocity control. For example, the patient recovering from stroke may be unable to generate the acceleration and deceleration forces needed during the different phases of gait. This results in delayed sequencing of muscle components and a general slowing of gait. Isokinetic training that focuses on the timing of these various components can improve gait function. Carryover to improved functional performance with any of these resistance training methods is not assured.[109] Additional functional training is necessary to ensure effective transference of strength and timing parameters to functional skills (specificity principle).

Manually resisted patterns of movement (PNF patterns) offer the advantage of functionally based, synergistic movements. Patterns of motion are spiral and diagonal in nature as opposed to straight planes of motion. The therapist can accommodate to the patient's specific level of weakness by providing repetitive graded resistance throughout the range and by adding additional facilitation as needed to improve or maintain performance. Effective verbal commands improve the magnitude of muscle contraction. Stretch is applied in the lengthened range to assist in the initiation of contraction and throughout the range as needed to sustain contraction. Approximation is applied to assist extensor patterns while traction is applied to assist flexor patterns. Specific PNF techniques (e.g., slow reversal, repeated contractions, and so forth) are described in Appendix B. Elastic resistance bands or pulley weights can also be used to provide resistance in PNF patterns.

Strength gains can be achieved through functional training that uses task-related practice.[110,111] Resistance is provided by gravity and body weight and is applied simultaneously to multiple moving segments. It can be supplemented with manual resistance of the therapist, weights, elastic resistance bands, or resistance of water during pool therapy. Activities can be selected that focus on specific body segments and progressed to involve increasingly larger segments of the body. This serves to increase the level of difficulty and the degrees of freedom that must be controlled during the movement. Benefits of functional training include improved coordination of muscles, improved postural control and balance, and improved muscle extensibility and flexibility. Functional training helps the patient develop control of synergistic muscle groups acting in multiple axes and planes of movements. It also fosters the control of varying types and combinations of muscle contractions (concentric, eccentric, isometric) that are used interchangeably during normal movement. This is a very different focus from the straight planes of motion and isolated movements commonly employed during PRE and isokinetic training. Intrinsic sensory input (somatosensory, vestibular, visual) is maximized during functional training.

Combining strength training protocols with task-specific practice is an effective strategy to maximize transfer gains to functional skills. For example, strengthening of weak lower limb extensor muscles can be first achieved using an isokinetic machine that targets both eccentric and concentric contractions of the quadriceps. This training can effectively be followed up with repetitive practice of functional activities also demanding similar extensor control (e.g., partial squats, sit-to-stand transfers, and stair climbing). The important consideration here is to match the strength training protocol to the requirements of the functional task in terms of range of motion achieved and type, magnitude, and speed of contraction.[109]

Endurance Training

Muscle endurance can be improved with exercise using dynamic contractions of large muscle groups repeated over time. Effects of endurance training are both central (cardiovascular) and peripheral (muscular). Peripheral adaptations include improved oxygen delivery to the exercising muscles, improved metabolic exchange, an increase in the number and size of mitochondria, increased myoglobin, and improved enzymatic activity. Essential components of an exercise prescription include the following interdependent elements: frequency, intensity, time or duration, and type or exercise mode (the *FITT equation*) along with progression of physical activity.[112] Training modes and equipment include walking and jogging (overground, treadmill, pool), cycling (ergometers), stepping (steps), or swimming (pools).

Patients with deficits in motor function may demonstrate poor muscular endurance and fatigue. **Fatigue** is defined as the inability to contract muscle repeatedly over time. Thus exercise cannot be sustained and exercise tolerance is reduced. The onset of fatigue is variable from patient to patient. Although many different factors may play a role, among the most important are the type and intensity of exercise. With the onset of fatigue, patients will demonstrate a decrement in force production progressing to total exhaustion (a ceiling effect). Fatigue can arise from neuromuscular disease affecting three primary sites: (1) the CNS (central fatigue), (2) the peripheral nerves or neuromuscular junction, or (3) the muscle itself.[113] Examples of conditions that can produce debilitating fatigue include multiple sclerosis, Guillain-Barré syndrome, chronic fatigue syndrome, and post-polio syndrome. The real danger of exercise training with these patients is the risk of injury and **overwork weakness**, defined as a prolonged decrease in absolute strength and endurance as a result of excessive activity.[114] For example, following an exercise

session a patient with post-polio syndrome may demonstrate prolonged weakness and fatigue that does not recover with rest. If exercise is exhaustive, the patient may be unable to get out of bed the next day or perform normal ADL. Even a simple conditioning program should be carefully monitored and progressed slowly to avoid overexertion and injury.[115–117] In general, moderate intensities of exercises in the range of 60 to 70 percent of maximal oxygen consumption or "somewhat hard" Ratings of Perceived Exertion are recommended while high intensities are contraindicated. A frequency of 3 days/week or alternate days is ideal using a discontinuous protocol that carefully balances exercise with rest.[118,119] Energy conservation, activity pacing, stress management, and lifestyle modification are essential components of the educational program.

Flexibility Exercises

Joint range of motion (ROM) and muscle flexibility must be adequate to allow for normal functional excursions of muscle and biomechanical alignment. Prolonged periods of disuse and immobility can lead to changes in muscle and joint function, postural alignment, and a host of indirect impairments (e.g., muscle tightness, atrophy, fibrosis, contracture, joint ankylosis, postural deformity). Older adults demonstrate age-related changes affecting joint flexibility. These include increased viscosity of synovial fluid, stiffening of the joint capsule and ligaments, and calcification of articular cartilage.[120] Early intervention is critical in maintaining joint motion, tissue extensibility, physical ability, and function. Additional benefits include improved circulation and tissue nutrition to the limbs and pain inhibition.

Techniques include ROM exercises, muscular stretching, and joint mobilization. The use of a preliminary therapeutic heat modality (e.g., hot pack or ultrasound) increases muscle temperature and elasticity, and collagen extensibility.[121,122] A warm-up period of exercise can also be used. For example, calisthenics or low-resistance cycling will gradually increase tissue temperatures and elasticity, thereby enhancing the safety of stretching. Cold modalities can be used to cool muscles and decrease muscle spasm and physiological splinting.[123] Patients with spasticity may benefit from preliminary prolonged icing and relaxation techniques (e.g., rhythmic rotation, cognitive relaxation). Icing can also be added following stretching, if necessary, to reduce tissue inflammation.

ROM Exercises

Range of motion is "the arc through which movement occurs at a joint or a series of joints."[103, p 690] ROM can be *active (AROM)*, performed and controlled entirely by the voluntary muscular efforts of the patient), *active-assisted* (*AAROM*, requiring some degree of external assistance for voluntary efforts) or *passive (PROM)*, performed solely by therapist or caregiver). The *Guide to Physical Therapist Practice* classifies the first two as therapeutic exercise while passive ROM is classified as manual therapy.[103] PROM is typically used when active movement is not possible (e.g., due to pain, paralysis, or unresponsiveness). AROM and AAROM have additional benefits of improving circulation, decreasing atrophy, and improving motor function. Progression should be to AROM exercises whenever possible as they are an important component of the home exercise program (HEP). ROM exercises are performed through the patient's full available range. The limb should be well supported with stable positioning of the patient to prevent joint trauma. The movements should be slow and rhythmic and within the patient's tolerance. Excess force and pain are contraindicated. This is especially important when working with the patient at risk for osteoporosis and heterotopic ossifications. External force during PROM or AAROM can also be applied mechanically (e.g., continuous passive motion machine, pulleys).

ROM exercises can be administered in anatomic planes of motion or in diagonal patterns of motion (PNF patterns). The latter may be more efficient as ROM can be administered throughout a limb, combining motions at more than one joint. ROM can also be achieved during functional training activities (e.g., shoulder ROM is achieved during weight shifting in quadruped or plantigrade positions). An added benefit may be the patient's lack of attention to joint motion during the activity with less protective splinting. Functional activity training should follow ROM exercises. The adage "use it or lose it" holds true for maintaining the benefits of both strengthening and ROM exercises.

Stretching Techniques

Stretching involves the application of manual or mechanical force to elongate (lengthen) structures that have adaptively shortened and are hypomobile.[105] The term *static stretching* refers to a method of stretching in which the muscle is slowly elongated to tolerance and the end position (greatest tolerated length) is held at least 20 to 30 seconds or longer depending upon the patient's tolerance. The use of slow, prolonged stretch minimizes muscle spindle activation and reflex contraction of the muscle being stretched. Maintaining the position at maximal end range results in the firing of the Golgi tendon organs (GTOs) with resulting inhibition of the muscle being stretched through mechanisms of autogenic inhibition. The combined neurophysiological effects result in improved muscle elongation. Additional benefits of static stretching using low loads include less danger of soft tissue tearing, less muscle soreness, and decreased energy requirements.[124–127] Low-load, long duration mechanical stretching can be applied for 30 minutes up to several hours using mechanical pulleys and weights or specialized orthotic devices. Prolonged positioning on a tilt table with wedges and straps can also be used to effectively to improve lower extremity range.[128,129]

Ballistic stretching involves the use of a high-load, short-duration, intermittent stretch. For example, bouncing movements of the body during sitting toe touch can be used to increase range in young, healthy individuals as part of a conditioning program. They can be appropriate as part of an advanced training program for high-velocity sports, particularly when used in conjunction with static stretching.[130] Ballistic stretching is generally contraindicated for the elderly, the chronically ill, or for patients undergoing active rehabilitation with neuromuscular impairments. It involves high-velocity, high-intensity movements that are not easily controlled. In addition, activation of muscle stretch receptors (muscle spindle Ia endings) results in reflex contraction and limits muscle elongation. Also in chronic contractures, connective tissue is more brittle and tears easily. Thus it is associated with high rates of microtrauma and injury.[105]

Facilitated stretching refers to the use of neuromuscular inhibition techniques to relax (inhibit) and elongate muscles when used in conjunction with stretching. PNF facilitated stretching techniques include Hold-Relax (HR), Contract-Relax (CR), and Active Contraction (AC)[100] (see Appendix B). The limb is actively moved to the lengthened, range-limited position. The patient is then instructed to perform a maximal isometric contraction of the tight muscles in the antagonist pattern. The prestretch contraction results in muscle inhibition from activation of the GTO (autogenic inhibition). This is followed by voluntary relaxation. The limb can then be passively moved into the elongated position (HR) or can be actively moved into the elongated position (Hold-Relax with Active Contraction, HRAC). The CR technique utilizes strong, small range isotonic contraction of the restricting muscles (antagonists) with emphasis on the rotators followed by an isometric hold. Movement into the newly gained range of the agonist pattern is active, not passive (CR with Active Contraction, CRAC). AC produces additional reciprocal inhibition effects (i.e., agonist contraction further inhibits the tight muscle via muscle spindle activity). Although these techniques were originally meant to be applied while using PNF patterns, clinicians have also used them in anatomical planes of motion. Research has demonstrated the effectiveness and superiority of facilitated stretching techniques over static and ballistic stretching techniques.[131–136] An additional benefit is that patients frequently report less discomfort with the application of facilitated stretching techniques as compared to other stretching methods. Because the inhibitory mechanisms affect primarily muscle and depend on voluntary contraction, these techniques are not effective with very weak or paralyzed muscles or range limitation associated with substantial connective tissue changes (chronic contracture).

Frequency of stretching (number of sessions per day or per week) varies according to underlying cause, the chronicity and severity of contracture, the patient's age and level of tissue integrity and healing, and medical management (use of corticosteroids).[105] Optimally multiple sessions per week (i.e., two to five sessions/week) are balanced with adequate rest in between to minimize tissue soreness. Exercises should be followed by active functional movements that maximize the mobility gained. Patients and/or their families/caregivers should be taught stretching exercises (i.e., self-stretching) as part of the HEP to maintain carryover outside of the clinic setting.

Strategies to Manage Tone

Tonal abnormalities are one of a number of features that can affect motor function. **Muscle tone** refers to firmness of the tissue and is the resistance to passive elongation or stretch. It is a function of both mechanical–elastic properties of muscle and neural drive. The term **postural tone** refers to the overall level of tension in the body musculature necessary to maintain body posture against gravity. Changes in tone can vary from higher than normal tone (hypertonia) to lower than normal tone (hypotonia) or fluctuating tone (dystonia). See Chapter 8 for a complete discussion of tonal abnormalities and examination procedures.

Patients with upper motor neuron (UMN) syndrome may exhibit spasticity. **Spasticity** is defined as velocity-dependent hypertonia and hyperactive tendon reflexes (increased deep tendon reflexes, DTRs). Additional *positive signs* associated with UMN syndrome include clonus, spasms, mass reflex responses (exaggerated cutaneous and autonomic reflexes, flexor reflex afferents [FRAs]), and pathological reflexes (e.g., Babinski, Hoffman). *Negative signs* include muscle weakness, slowness of muscle activation, abnormal motor unit recruitment, dyssynergic patterns or obligatory synergies, and loss of coordination and dexterity. Functionally the patient demonstrates poor volitional control of movements and limitations in functional skills. The limbs are typically held in fixed, abnormal postures with antigravity muscles primarily affected. For example, the upper extremity typically assumes an abnormal flexor posture while the lower extremity assumes an abnormal extensor posture. If left untreated, spasticity can lead to the development of secondary impairments such as contracture, postural asymmetries, and deformity. In contrast, the patient with hypotonia typically demonstrates loss of tone with weak or paralyzed muscles, joint instability, and deformity. Following neurological insult, tone varies relative to recovery stage. For example, the patient with a new or recent stroke or spinal cord injury will present with initial flaccidity during the stage of cerebral or spinal shock while the same patient in the post-acute stage will often demonstrate emerging spasticity. Asymmetries of tone between limbs, between the two sides of the body, or between limbs and the trunk are common. Asymmetries may also occur within a limb from muscle to muscle. For example, the proximal muscles are spastic while the distal hand muscles are flaccid.

Strategies for Managing Hypertonia

A number of interventions can be used to manage spasticity. These include prolonged icing, prolonged stretch, inhibitory pressure, and neutral warmth. See Appendix C for a complete description of these techniques. Rhythmic rotation (RRo) is a highly effective exercise technique that can be used to reduce hypertonicity and increase range (see Appendix B). Precise handling of a spastic limb is important. The therapist should use constant, firm manual contacts positioned over nonspastic areas to avoid directly stimulating spastic muscles. To maintain soft tissue range, limbs are slowly moved out of the spastic pattern. Positioning out of the spastic pattern is effective in sustaining the inhibition and range achieved. For example, the patient with stroke is positioned in sitting. Rhythmic rotation is used to move the affected upper limb out of a flexed, adducted position into weightbearing with the elbow extended, hand open, and wrist and fingers extended. Weight shifting during sitting can then be used to maintain inhibition and range. Johnstone[137] advocates the use of inflatable pressure splints to decrease hypertonicity and maintain limbs in optimal tone-reducing positions during functional training. For example, an inflatable pressure cuff is applied to maintain the upper limb in an extended position during weightbearing activities in sitting. At best, inhibitory techniques can be expected to produce a temporary reduction in tone, with results lasting 20 to 30 minutes or up to a few hours. They do not permanently alter tone. Thus, these techniques must be viewed as preparatory techniques to enhance ROM, stretching, and functional movement training and not as the primary focus of treatment. For example, ROM exercise for the patient with severe lower extremity spasticity resulting from multiple sclerosis will likely be ineffective without first applying a tone-reducing technique. Relaxation can be obtained through RRo with both lower extremities positioned on a ball (patient supine with the hips and knees flexed to 90°) and gently rocked side-to-side. Once relaxation occurs, the therapist can then effectively range the limbs to ensure adequate length of muscle and joint position. Resting splints can then be applied to maintain the muscles in an elongated state with positioning of the ankle in a neutral position.

Exercise training is key to lasting improvement in tone and motor function. The following guidelines can be used:

- Primary focus should be on first activating contraction of antagonist muscles (muscles opposite the spastic muscles) to provide inhibition and lengthen spastic muscles.
- Assistance (RRo, active assistive or guided movements) can be used initially as needed but withdrawn as soon as active movements are possible.
- Reciprocal actions are then attempted. Agonist (spastic muscle) contractions are initiated first in small ranges (short arcs) progressing to larger arcs of movement. Smooth, reciprocal movements are practiced.
- Highly stressful and effortful activities should be minimized during early training as they may reinforce (heighten) tone and activate abnormal associated reactions.
- Important functional skills are targeted for training. For example, the patient practices reciprocal reaching, sit-to-stand movements, stepping, or walking.
- Isokinetic exercises are effective in improving function in patients with spasticity.[138–141]
- Strength training can be used and is helpful in improving motor function in patients with spasticity.[142–145]
- Aerobic conditioning can be implemented and is beneficial in improving motor function in patients with spasticity.[146–148]
- Patients and their family or caregivers should be educated about the need to maintain length of spastic muscles. Daily ROM exercises are stressed as well as effective use of stretching, positioning, and splinting techniques.

Serial Casting

Serial casts combined with stretching are effective in reducing hypertonicity, improving range, and reducing deformity.[149–161] *Serial casting* is used when traditional techniques fail and the patient is at risk for development of contractures and deformity, or demonstrates ineffective movement patterns or severe limitations in hygiene and skin care. Inhibitory and ROM techniques are first used to move the limb into its fully lengthened range. Nerve blocks can also be used to improve range prior to casting. The cast is then applied while the limb is held at the end of available range. The sustained position produces relaxation of the spastic muscles, thought to be the result of GTO autogenic inhibition and adaptation of stretch receptors.[162] Neutral warmth and continuous even pressure may also be contributing factors. Inhibitory casting has been found to promote changes in muscle or tendon length and sarcomere distribution.[163] The casts are typically changed every 5 to 7 days (serial application) to gradually increase available range. Poor casting techniques include lack of end-range positioning of the limb, loose-fitting cast, or insufficient padding. Faulty technique may result in a lack of improvement or even increased tone, skin breakdown especially on bony prominences, or nerve compression. An overly restrictive cast can result in decreased circulation and peripheral edema. Highly agitated patients may potentially injure themselves and demonstrate increased risk of skin breakdown and cast breakage. Patients with cognitive or communication impairments should be monitored closely because they will be unable to indicate pain or discomfort and potential skin breakdown. Casting is contraindicated in patients with severe heterotropic ossification; muscle rigidity; skin conditions such as open wounds, blisters, or abrasions; impaired circulation and edema; uncontrolled hypertension; unstable intracranial pressure; pathological inflammatory conditions such as arthritis or gout; or in individuals at risk for compartment syndrome

or nerve impingement. Application to individuals with long-standing contractures (longer than 6 to 12 months) is also contraindicated.[164,165]

Adjustable orthoses have also been used to provide passive, sustained stretch with the added benefits of easy removal for hygiene and observation. These devices use a rotating adjustable dial attached to metal rods and a flexible acrylic thermoplastic base.[166,167] The required adjustments are easier and less time consuming than fabricating an entirely new serial cast. Dynamic orthoses, primarily used on elbow or knee flexion contractures, use a spring-loaded or hydraulic mechanism to provide nearly constant pressure.[168,169] Reported outcomes of studies using these devices include reduction of contractures with minimal complications.

Modalities

Neuromuscular electrical stimulation (NMES) has been used to reduce spasticity and improve motor function.[170] Applications to the tibialis anterior muscle or to the common peroneal nerve have been shown to reduce spasticity in the plantarflexor muscles and ankle clonus.[171–173] Electrical stimulation of forearm muscles has been shown to reduce flexor tone and posturing of the hand.[174,175] Spinal cord stimulators have been utilized to reduce severe flexor and extensor spasms, with variable results.[170] Transcutaneous electrical nerve stimulation (TENS) has been used to improve motor function and reduce tone in patients with UMN syndrome.[176,177] Finally, EMG biofeedback has been used to relax spastic muscles by monitoring muscle activity of during slow, passive stretch. Patients are encouraged to decrease EMG activity during passive or active stretch. Conversely, antagonists to the spastic muscles have also been monitored, with patients encouraged to increase EMG activity and muscle contraction.[178–180]

Strategies for Managing Hypotonia

Intervention techniques to increase tone for patients with hypotonia (flaccidity) can include quick stretch, tapping, resistance, approximation, and positioning (see Appendix C). Patients typically also demonstrate weakness and at times it is difficult to differentiate between the two states. Strengthening exercises that do not overload the weak, hypotonic muscles are indicated. Postural instability is a common problem. Interventions should be designed to improve postural stability in functional positions (see following section on postural stability training). Supportive and protective devices may be necessary to prevent injury to limbs and postural asymmetries (e.g., a Swedish knee cage can be used to prevent hyperextension). NMES can also be used to activate hypotonic muscles, improve strength, and generate movement in paralyzed limbs while preventing disuse atrophy. It is important to focus the patient's attention on the desired movement and verbally cue the patient's movement attempts at volitional contraction. Without such cueing and movement attempts, carryover to volitional control is not possible. Electrical stimulation should ideally be coupled with functional training activities to optimize outcomes.

Postural Control Training

Postural control (balance) is the ability to maintain the body in equilibrium or to control the body's position in space for stability and orientation. **Postural orientation** is the ability to maintain normal alignment relationships between the various body segments and between the body and environment. **Postural stability control** (static equilibrium, static balance, or stability) is the ability to maintain stability and orientation with the center of mass (COM) over the base of support (BOS) with the body at rest (no motion). **Dynamic postural control** (dynamic equilibrium, dynamic balance, or controlled mobility) is the ability to maintain stability and orientation with the COM over the BOS while parts of the body are in motion (see Table 13.2).[2] An intervention program to improve postural control must be based on an accurate evaluation of data obtained during examination of deficits (see Chapter 8). Training activities can be used to:

- Improve static postural control, biomechanical alignment, and symmetrical weight distribution.
- Improve dynamic postural control including musculoskeletal responses necessary for movement and balance.
- Improve adaptation of balance skills for varying task and environmental conditions.
- Improve sensory function including sensory integration and sensory compensation.
- Improve safety awareness and compensatory strategies for effective fall prevention.

Strategies to Improve Static Postural Control

Patients who demonstrate impairments in static postural control are unable to maintain or hold a steady position for a number of reasons, including decreased strength, tonal imbalances (hypotonia, spasticity), impaired voluntary control and hypermobility (ataxia, athetosis), sensory hypersensitivity (tactile-avoidance reactions), or increased anxiety or arousal (high sympathetic state). Instability is associated with excessive postural sway, wide BOS, a high guard hand position or handhold, and loss of balance.

The therapist can select any of a number of weight bearing (antigravity) postures to develop stability control (see Table 13. 3). Postures are selected on the basis of (1) patient safety and level of control and (2) variety in terms of functional tasks. It is important to remember that some activities may cause the patient distress initially. The patient will feel threatened when placed in situations where he or she is in jeopardy of losing balance. The therapist should ensure patient confidence by providing a clear explanation of what is going to happen, and what is expected of the patient in terms that are easy to understand. Support may be given initially to reduce fear if using a new posture, but should be withdrawn as soon as possible to

allow focus on active control. The therapist varies the level of activities, selecting activities that both provide success as well as appropriately challenge the patient.

In sitting or standing, the patient is instructed to "hold steady" while sitting or standing tall and maintaining a visual focus on a forward target. Progression is to holding for longer and longer durations. Neuromuscular/sensory stimulation techniques that can be used to enhance stabilizing muscle contractions include quick stretch, tapping, resistance, approximation, manual contacts, and verbal cues (see Appendix C). For the patient unable to actively stabilize the body, the therapist can begin with resisted isometric contractions of antagonist postural muscle groups using the technique of Rhythmic Stabilization (RS) (see Appendix B). For example, the patient with severe instability following traumatic brain injury who is unable to sit independently may need to practice holding first in the side-lying position during application of RS. The therapist can then progress training through postures that demand increasing amounts of upright (antigravity) postural control—prone-on-elbows to quadruped and finally sitting. In each position the therapist carefully provides matching resistance using RS. If an imbalance exists, the stabilizing activity can be followed by a strengthening activity for the weak muscles.[101] As the trunk becomes more stable, the patient is expected to assume active control in stabilizing in the posture. For a patient with hyperkinetic disorders (e.g., ataxia, athetosis), the PNF technique of Stabilizing Reversals (Slow Reversals) is appropriate (see Appendix B). Alternating isotonic contractions are used, allowing only very small range movements. Progression is toward decreasing range (decrements of range) until finally the patient is asked to stabilize and hold steady in the posture.

Additional strategies to improve stability include the use of elastic resistance bands or weights to enhance *proprioceptive loading* and contraction of stabilizing muscles. For example, in the prone-on-elbows position, bands can be placed around the forearms. The patient is instructed to push out against the band and maintain the forearms apart against the resistance. This selectively loads and facilitates contraction of the shoulder stabilizers (abductors and rotator cuff muscles). In bridging, kneeling, or standing, elastic resistance bands can be placed around the thighs. The patient is instructed to maintain the thighs apart against the resistance of the bands. This selectively loads and facilitates contraction of the hip stabilizers (abductors, extensors), improving stability control at the hips.

The therapist can have the patient stabilize while sitting on a therapy ball (also known as Swiss or stability ball). Gentle bouncing provides joint approximation through the vertebral joints, facilitating extensors and an upright posture. For patients requiring more assistance, sitting control can first be practiced on a compliant surface (foam, wobble board, or Dynadisc™) placed on a platform mat or sitting on a ball with a ball holder underneath the therapy ball. Task difficulty can be increased by reducing the BOS (feet apart to feet together to single limb support).

Aquatic therapy can also be used to enhance proprioceptive loading. The water provides a degree of unweighting and resistance to movement. This can be quite effective in reducing hyperkinetic movements and enhancing postural stability. For example, a patient recovering from traumatic brain injury who demonstrates significant ataxia may be able to sit or stand in the pool with minimal assistance while these same activities outside the pool are not possible.

To improve standing control, the patient is directed to practice neuromuscular *fixed-support strategies* that occur at the ankle and hip joints. Feedback is provided to assist the patient in recruiting the correct pattern. To recruit *ankle strategies*, the patient practices small-range, slow-velocity shifts. Attention is directed to the action of ankle muscles to move the body (COM) over the fixed feet (BOS). Standing on a wobble board or foam roller with the flat side down progressing to flat side up are effective activities to recruit ankle strategies. The patient is also directed to practice tasks that normally recruit *hip strategies*. These are recruited with larger shifts in the COM, that approach the limits of stability (LOS), and/or faster body sway motions and are characterized by early activation of proximal hip and trunk muscles. Hip flexion and extension responses are generated during anterior–posterior (AP) displacements and lateral hip motions are generated during lateral displacements. Patients can be instructed to move their upper body forward and backward while standing on a foam roller. Tandem standing or tandem standing on a foam roller can be used to recruit lateral hip strategies.

Anticipatory postural adjustments should also be practiced, because predictive control must be operational for functional balance. The patient is provided with advance information about the upcoming demands of the task. For example, "I want you to catch this 5-pound weighted ball while maintaining your sitting position. The prior knowledge serves as an important source of information in initiating the correct postural pattern. To promote generalizabilty, practice should occur in a variety of environments. For example, training can progress from a closed or fixed environment to a more variable environment such as the physical therapy gym. Balance training must ultimately be context specific to real-life settings of home or community to ensure functional carryover.

Strategies to Improve Dynamic Postural Control

Patients who demonstrate impairments in dynamic postural control are unable to control postural stability and orientation while moving segments of the body. A number of impairments may be contributing factors, including tonal imbalances (spasticity, rigidity, hypotonia), ROM restrictions, impaired voluntary control and hypermobility (ataxia, athetosis), impaired reciprocal actions of the antagonists

(cerebellar dysfunction), or impaired proximal stabilization. Clinically, the patient demonstrates difficulty weight shifting from side-to-side, forward–backward, or diagonally. Difficulties are also apparent in moving one or more limbs while maintaining a posture (sometimes referred to as *static-dynamic control*). For example, one limb is freed for movement (reaching or stepping) while the patient maintains the sitting or standing posture. Or from the quadruped position the patient is asked to lift one arm or leg or to lift the opposite arm and leg. These added movements increase the demand for stabilization control because the overall BOS is reduced and the COM must shift over the remaining support segments before the dynamic limb movement can be successful.

The therapist can select any of a number of weightbearing (antigravity) postures to develop dynamic postural control. Practice begins with movements emphasizing smooth directional changes that engage antagonist actions (e.g., weight shifts). As control improves, the movements are gradually expanded through an increasing range (increments of range). Dynamic movements can be facilitated using quick stretch, tapping, light tracking resistance, manual contacts, and dynamic verbal commands (see Appendix C). Although active movement is the goal, assistance may be required during initial movement attempts for both the dynamic movements as well as the stabilizing body segments. Specific task-oriented training (e.g., reaching, stepping) are more motivating, especially if the task is important to the patient.

PNF extremity patterns can be used to increase the level of difficulty. For example, in sitting the patient is asked to move the dynamic limbs in a chop/reverse chop pattern while maintaining a stable posture. The patient's full attention is focused on performing the pattern and not on the stabilizing postural components. This ability to redirect cognitive attention is an important measure of developing postural control as intact postural control functions largely on an automatic and unconscious level. Specific PNF techniques appropriate for assisting patients include Dynamic Reversals (Slow Reversals), Repeated Contractions, Rhythmic Initiation, and Combination of Isotonics (Agonist Reversals) (see Appendix B). For example, in bridging the patient's movements are resisted in assuming the bridge posture (isotonic contractions) and during movement from the bridge position to hooklying (eccentric contractions) using the Combination of Isotonics technique. This activity is an important lead-up for other functional activities that require similar combinations including sit-to-stand transitions, moving from kneeling to heel sitting, and ascending–descending stairs.

Therapy ball activities are effective in developing dynamic stability control. For example, the patient sits on a ball and gently moves the ball side-to-side, forward–backward, or in a combination (pelvic clock motions). Or the patient sits on the ball while performing voluntary movements of the arms or legs (alternate leg or arm raises). Progression is from unilateral to bilateral and finally to reciprocal limb movements (e.g., Mexican hat dance). Voluntary trunk motions can be practiced while sitting on the ball (e.g., head and trunk rotations or forward/backward leans).[181] Resistance can be introduced by using elastic resistance bands or weight cuffs on the ankles or wrists. Difficulty can also be increased by adding a second task (*dual task training*) such as catching and throwing a weighted ball, balloon volleyball, or kicking a ball. Group activities can be introduced when patients can safely perform each of the activities individually.[182]

To improve standing control, the patient is directed to practice neuromuscular *stepping strategies*. The traditional view holds that stepping strategies occur when the COM exceeds the LOS.[183–185] Perturbation is used to provide the COM displacement. Stepping movements are accompanied by early activation of hip abductors and ankle co-contraction for medial–lateral stability during single-limb support.[186] Maki and McIlroy[187] investigated the role of limb movements in maintaining upright stance, specifically compensatory stepping and grasping movements of the upper limbs, which they termed *change-in-support strategies* (as opposed to the fixed-support strategies at the ankle and hip). These investigators found that both stepping and arm movements were very common reactions to loss of balance. Moreover they were initiated well before the COM reached the LOS, contradicting the traditional view that they are strategies of last resort. They also found that stepping may actually be a preferred strategy to using a hip strategy. The direction and magnitude of change-in-support strategies were found to vary according to the magnitude and direction of the perturbation. For example, stepping may occur forward or backward in response to anterior or posterior displacements. Lateral displacements typically resulted in cross-stepping pattern (seen in 87 percent of lateral stepping responses) as opposed to straight side-stepping. Lateral destabilization with its increased demands for lateral weight transfer is particularly problematic for a large portion of older adults who experience falls. Arm reactions in response to whole-body instability were also found to be prevalent with activation of shoulder muscles occurring in 85 percent of destabilizing trials. Increased understanding of the range and variability of postural strategies for balance negates any simplistic view of balance based on a developmental perspective of reflex control (i.e., righting and equilibrium reactions). Overall, the organization of balance strategies must be viewed as flexible, not rigid, involving multiple body segments. In that context, patterns will vary according to a number of different factors including initial conditions, perturbation characteristics, learning, and intention.[188] Training should therefore include practice of voluntary stepping movements in all directions (i.e., anterior, posterior, or lateral side steps). Elastic resistance bands positioned around the pelvis can be used to improve the strength of stepping responses. Manual perturbations of increasing force can also be used to elicit stepping responses.[182]

Postural Awareness Training

Faulty postures such as forward head, kyphosis, lordosis, excessive hip and knee flexion, or pelvic asymmetries can result in decreased postural stability, inaccurate kinesthetic awareness of true vertical, and pain. Although mild deficits may not affect balance control, deficits that significantly alter the COM position can impair balance.[189] Patients are typically unable to self-correct faulty postures. Physical therapy interventions should focus first on improving specific musculoskeletal impairments (e.g., limited ROM, weakness). For example, active exercises to improve standing balance can include standing heel-cord stretches, heel-rises, toe-offs, partial wall squats, chair-rises, side-kicks, back-kicks, and marching in place using touch-down support of the hands as needed (sometimes referred to as the "kitchen sink exercises"). Postural reeducation begins with demonstration of the correct posture. Verbal cues should focus on control of essential postural elements, that is, stable (neutral) pelvis, axial extension (e.g., sit or stand tall), and normal alignment (e.g., head erect, shoulders back, weight evenly distributed under both hips [sitting] or feet [standing]). Patients can benefit from tactile cues during initial practice (manual or surface-related). For example, patients can stand with the back positioned against a wall or patients with a lateral lean (e.g., post-stroke patients with pusher syndrome) can sit with their side positioned against the seated therapist or a wall. Corner standing or standing between two plinths can be effective for patients with significant COM distortion. Mirrors provide important visual cues regarding vertical position but are generally contraindicated for the patient with visuospatial perceptual deficits. Application of correct postures to real-life functional situations is important to ensure carryover and lasting change.

Center-of-Mass Control Training

The therapist should focus on obtaining symmetrical, balanced weightbearing. Patients may present with specific directional instabilities, such as weightbearing more on one side than the other. For example, after a stroke the patient typically keeps weight centered toward the sound side. Practice should focus on redirecting the patient into a centered position by moving toward the affected side, both in sitting and standing positions. Limits of stability (LOS) should be explored. For example, in sitting or standing, the patient is instructed to slowly sway forward–backward and side-to-side. The outer point at which the COM is still maintained within the BOS is termed the LOS. Loss of balance occurs when the LOS have been exceeded, for example, when the COM extends beyond the BOS. Practice of volitional body sway is important to assist the patient in developing accurate perceptual awareness of stability limits, an important component of an overall CNS internal model of postural control. Because LOS change with different tasks, a variety of functional activities should be practiced in different environmental settings.

Posturography Feedback

Balance training using augmented visual feedback has become increasingly popular in treating the elderly and other patients at risk for falls. Research reports substantiate its effectiveness in improving balance.[190–196] Force-platform devices are used to measure forces and provide center of pressure (COP) biofeedback or *posturography feedback.* COP displacement is associated with movement of the COM or postural sway. While COP excursion always exceeds COM sway, this relationship is close during ankle motions (ankle strategies) when the body moves like a pendulum over the feet. However, when a hip strategy is used (upper body motion focused at the hips) the COP:COM relationship becomes distorted and does not accurately reflect sway.[197,198] A computer analyzes the data and provides relevant biofeedback concerning sway path and COP position on a visual monitor. Some units also provide auditory feedback.

Posturography training can be used to shape sway movements to enhance symmetry and steadiness. The patient can be instructed to increase or decrease sway movements or move the COP cursor on the computer screen to achieve a designated range or to match a designated target. It is an effective training mode for patients who demonstrate problems in force generation. For example, the patient with decreased force generation (hypometria) as typically demonstrated by individuals with Parkinson's disease is directed toward achieving larger and faster sway movements during posturography training. The patient with too much force (hypermetria), as typically demonstrated by the individual with cerebellar ataxia, is directed toward decreasing sway movements progressing to holding a stable, centered posture.[194,195] It is important to remember that balance retraining using posturography biofeedback does not automatically transfer to functional skills like gait. Winstein[199,200] found that a reduction in standing balance asymmetry did not result in a concomitant reduction in asymmetrical limb movement patterns associated with hemiparetic locomotion. Given the specificity of training principle, this is not a surprising finding. Finally, a set of bathroom scales or limb load monitors can provide a low tech, low cost form of biofeedback weight information to assist patients in achieving symmetrical weightbearing.[201,202]

Strategies to Improve Safety

Prevention of falls for the patient with balance deficiency is an important goal of therapy. Lifestyle counseling is important to help recognize potentially dangerous situations and reduce the likelihood of falls. For example, high-risk activities likely to result in falls include turning, sit-to-stand transfers, reaching and bending over, and stair climbing. Patients should also be discouraged from clearly hazardous activities such as climbing on step stools, ladders, and chairs, or walking on slippery or icy surfaces. The education plan should stress the harmful effects of a sedentary lifestyle. Patients should be encouraged to maintain an

active lifestyle, including a program of regular exercise and walking. Medications should be reviewed and those medications linked to increased risk of falls (e.g., medications that result in postural hypotension) should be addressed. A consult with the physician for medication review may be indicated.

Compensatory training strategies should be utilized. The patient should be instructed in how to maintain an adequate BOS at all times. For example, the patient should widen BOS when turning or sitting down. If a force is expected, the patient should be instructed to widen the BOS in the direction of the expected force (e.g., leaning into the wind). If greater stability is needed, instruction should be provided in how to lower the COM (e.g., crouching down to reduce the likelihood of a fall). Greater stability can also be achieved if friction is increased between the body and the support surface. The patient should therefore be instructed to wear shoes with low heels and rubber-soles for better gripping (e.g., athletic shoes). Assistive devices should be used assist to balance when necessary. Consideration should always be given to using the least restrictive device (LRD) while at the same time ensuring safety. Light touch-down support using a vertical or slant cane (used by individuals who are blind) has been shown to improve balance.[203] A fall prevention program must also address environmental factors that contribute to falls. See Box 13.4 for recommendations for reducing falls in the home environment (see also Chapter 12).

Sensory Training

Several general concepts are important to an understanding of the role of sensation in movement. Sensation allows one to interact with the environment, guiding the selection of movement responses. Sensory inputs are used to modify movements and shape motor programs through feedback for corrective actions. Variability and adaptability of movements to environmental change are made possible by the information processing of sensory inputs. Finally, sensory inputs are used to prevent or minimize injury. Interaction of sensory and motor systems occurs throughout the CNS. Much of the sensory information received is not consciously perceived. Spinal-level interactions are largely reflexive in nature, whereas supraspinal centers modulate more complex levels of sensorimotor behavior. Conscious perception and interpretation of sensory information occurs at the highest level, the cortex.

The various types of sensory receptors demonstrate differential sensitivity. Each receptor is highly sensitive to a preferential stimulus while being relatively insensitive to other stimuli at normal intensities. Use of appropriate intensities of sensory stimulation is important to ensure that the desired receptors are stimulated. Excess stimulation can activate unwanted sensory receptors and produce undesired responses, including generalized arousal and sympathetic fight-or-flight reactions. Another special characteristic of sensory receptors is their adaptation to stimuli over time. Generally, they can be divided into two categories, slow- and fast-adapting receptors. In treatment, fast-adapting, phasic receptors such as touch receptors are generally more effective in initiating dynamic movements, whereas slow-adapting, tonic receptors such as joint receptors, Golgi tendon organs, and muscle spindles are used more in monitoring and regulating postural responses. Velocity of movement is also a consideration. At slow velocities, afferent stimuli can contribute to movement responses, while at high velocities there is insufficient time to allow for afferent information to effect motor control (open skills). Certain body segments such as the face, palms of the hands, and soles of the feet demonstrate both high concentrations of tactile receptors and increased representation in the sensory cortex. These areas are highly responsive to stimulation and are closely linked to both protective and exploratory functions.

Damage to the CNS can produce impairments in sensory function. Alterations in tactile, proprioceptive, visual,

Box 13.4 Fall Prevention Strategies: Modifying the Home Environment

- Adequate lighting is essential. Both low light and glare can be hazardous, particularly for the elderly. Glare can be reduced with translucent shades or curtains.
- Light switches should be positioned at the entrance to a room and fully accessible. Timers can ensure that lights come on routinely at dusk. Clapper devices can be used to enable the patient to turn on lights from across the room. Nightlights typically used in bathrooms or hallways do not provide enough light to ensure adequate balance.
- Carpets with loose edges should be tacked down. Scatter or throw rugs should be removed.
- Furniture that obstructs walkways should be removed or repositioned.
- Chairs should be of adequate height and firmness to assist in sit-to-stand transfers. Chairs with armrests and elevated seat heights may be required. Motorized chairs that elevate the patient into standing may be hazardous for (1) some patients who are unable to initiate active balance responses in a timely manner during initial standing and (2) those with impaired LE strength unable to maintain firm foot contact with the floor as the chair rises.
- Stairs are the site of many falls. Ensure adequate lighting. Contrast tape using bright warm colors (red, orange, or yellow) can be used to highlight steps. Handrails are important for safety on stairs and, if not present, may need to be installed.
- Grab bars or rails reduce the incidence of falls in the bathroom. Nonskid mats or strips in the bathtub along with a tub or shower seat can also improve safety. Toilet seats can be elevated to facilitate independent use.

or vestibular systems can affect a patient's ability to move and learn new activities. Deafferentation in animals and in humans is associated with nonuse of a limb, although gross movements are possible under forced situations. Learning of new movements through corrective actions is impaired. The therapist must focus on forced training of sensory-deficient limbs even though the patient may have little interest in moving the limb. The movements obtained should not be expected to be normal, however, because significant deficits have been noted in fine motor control in deafferentated limbs. Following damage to the CNS, sensory inputs may be reduced or distorted. Perceptions are therefore impaired. Sensory training strategies can be used to sharpen and heighten perceptions and assist in reorganizing the CNS.

Training Strategies for Sensory Loss

Sensory stimulation refers to the structured presentation of stimuli to improve (1) alertness, attention, and arousal; (2) sensory discrimination; or (3) initiation of muscle activity and improvement of movement control. Effects are immediate and specific to the current state of the nervous system. See Appendix C for a complete discussion of these techniques. The techniques are important elements of the neurofacilitation approaches.[98–100] Behaviors are modified using techniques to increase or decrease attention and arousal. Movements are elicited and modified through the use of specific stimuli (e.g., stretching, tapping). The effects do not carry over to subsequent movement attempts. Because the movements rely on augmented inputs, their greatest use is to assist the patient with absent or severely disordered voluntary control (e.g., a patient who sustained a stroke and who is unable to consistently initiate muscle contractions). Once a desired motor response is obtained, focus should shift to active movements that utilize naturally occurring intrinsic sensory information. Thus, sensory stimulation techniques may be an effective *bridge* to assist early attempts at movement but should be withdrawn as soon as possible. Repeated use of sensory stimulation long after it is necessary can result in movements that become stimulus dependent, and can further limit the patient's ability to regain voluntary control.

Sensory integration training refers to "the use of enhanced, controlled sensory stimulation in the context of a meaningful, self-directed activity in order to elicit adaptive behavior"[204, p 23] Varied sensory stimuli are presented (tactile, vestibular-proprioceptive, and visual) in order to engage higher brain centers for central processing of sensory information (see Appendix C). The overall goals are to (1) improve sensory discrimination: identification of specific stimuli (e.g., shapes, weights, texture, numbers written on skin), intensities, and localization of stimuli and (2) improve perception: selection, attention, and response to sensory inputs with appropriate use of information to generate specific motor responses. The key elements are multimodal presentation of various different stimuli combined with functional task training. Focus is also on postural training activities with progression to more difficult adaptive motor responses.

Sensory reeducation has been used successfully to improve sensory function in patients with peripheral nerve damage.[205] Patients with stroke-related impairments have also shown benefits from specific sensory training programs.[206–208] Components of these programs consist of having the patient practice sensory identification tasks (numbers, letters drawn on the hand or arm), discrimination tasks (detecting size, weight and texture of objects placed in the hand), and passive-assisted drawing using a pencil. The tasks are alternated between both affected and unaffected hands. Each training session starts and ends with a sensory task the patient could successfully master. The training group showed a positive and significant improvement in sensory function.[208] An important feature of this study was that the subjects were at least 2 years post-stroke, providing strong evidence that the effects were due to training and not recovery. Continued practice with functionally relevant tasks is necessary to maintain the positive effects of any sensory training program. Important considerations for the therapist include having the patient concentrate on the relevant sensory cues, structuring the environment for optimal success, and providing verbal and visual cues. Yekatiel and Guttman[208] suggest that sensory retraining should be considered as a regular component of rehabilitation programs along with motor training following stroke.

Sensory Training Strategies for Balance

An important focus of balance training is utilization and integration of appropriate sensory systems. Normally three sources of inputs are utilized to maintain balance: somatosensory inputs (proprioceptive and tactile inputs from the feet and ankles), visual inputs, and vestibular inputs.[209] Careful examination can identify the patient's use of inputs to maintain balance (e.g., *Clinical Test for Sensory Interaction and Balance* [CTSIB]). See Chapter 8 for a discussion of this test. Training is directed to using varying sensory conditions to challenge the patient. For example, patients who demonstrate a high degree of dependence on vision can practice balance tasks with eyes open and eyes closed, in reduced lighting, or in situations of inaccurate vision (petroleum-coated lenses or prism glasses). Altering the visual inputs allows the patient to shift focus and reliance to other sensory inputs, in this case to intact somatosensory and vestibular inputs. Patients can practice varying somatosensory inputs by standing and walking on different surfaces, from flat surfaces (floor) to compliant surfaces (low to high carpet pile), to dense foam. A patient who is barefoot or wearing thin-soled shoes is better able to attend to sensation from the feet than if wearing thick-soled shoes. Challenges to the vestibular system can be introduced by reducing both visual and somatosensory inputs through

sensory conflict situations. For example, the patient practices standing on dense foam with the eyes closed. The patient can also be directed to walk on foam with eyes closed, a condition that requires maximum use of vestibular inputs. Patients should also practice varying environmental influences such as walking outside, progressing from relatively smooth terrain (sidewalks) to uneven terrain to moving surfaces (escalator, elevator). Repetition and practice are important factors in assisting CNS adaptation.

Patients with significant sensory loss will require assistance in shifting toward the intact systems to monitor and adjust balance using *compensatory training strategies*. For example, the patient with proprioceptive losses will need to learn to shift focus onto the visual system for functional mobility and balance. Thus, the patient with bilateral amputations learns to rely heavily on visual inputs for control in standing and walking. If deficits exist in more than one of the major sensory systems, compensatory shifts are generally inadequate and balance deficits will be pronounced.[2] Thus, the patient with diabetic neuropathy and retinopathy will be at high risk for loss of balance and falls. Compensatory training with an assistive device is indicated. Other patients must be encouraged to ignore distorted information (e.g., impaired proprioception accompanying stroke) in favor of more accurate sensory information (e.g., vision). Augmented feedback can assist in training (e.g., verbal commands, light-touch finger contact, biofeedback cane with auditory signals, limb load monitor).

Gait and Locomotion Training

Substantial rehabilitation efforts are directed toward improving gait to restore or improve a patient's functional mobility and independence. Walking is frequently the number one goal of patients who "want to walk" above all other considerations. Ability to ambulate independently is often a significant factor in determining discharge placements (e.g., return to home or extended care facility). The alternate to walking is locomotion using a wheelchair. To establish a realistic plan of care, the physical therapist must accurately analyze the patient's walking ability. Comprehensive gait analysis including gait variables and common gait deviations is discussed in Chapter 10. The functional demands of the patient's home, community, or work environment must be considered in planning successful interventions and in predicting a patient's future status.

Gait is a complex skill that requires integrated function of many interacting systems. Basic requirements for walking include (1) establishment of a rhythmic stepping pattern, (2) body support and propulsion in the intended direction, (3) dynamic postural control, and (4) ability to adapt to changing task and environmental demands.[210] Multiple muscle groups are active in alternating synergistic patterns. Stabilizing muscles contribute to stability of the stance limb and trunk during weight acceptance and single limb support. Other muscles contribute to limb advancement of the dynamic limb during the swing phase. The pattern is then reversed as the gait cycle progresses. As the speed of walking increases, the requirements for timing and control increase. Older adults can be expected to have reduced walking speed, shorter strides, shorter steps, increased time in double support, and decreased time in swing phase than young adults.[53]

Interventions must first be directed at improving function of individual gait components. For example, attention is directed first at improving ROM and strength of weak muscles such as hip abductors or knee extensors. Emphasis can then shift to improving synergistic control of muscles and flexibility through functional training activities. Important *lead-up* or *preambulation activities* that improve strength, range, and control necessary for gait include bridging, sit-to-stand transfers, and static and dynamic postural control activities in kneeling, half-kneeling, modified plantigrade, and standing. Finally, stepping first in modified plantigrade and then in standing are important lead-up activities. See Chapter 14 for a detailed discussion on locomotor training.

Task-Specific Training

Walking is typically practiced first under supportive conditions using parallel bars or assistive devices (e.g., walker, cane, crutches). The goal is early mobilization of the patient out-of-bed or out-of-chair is to prevent further indirect impairments (e.g., weakness, decreased endurance, loss of mobility, and so forth). Gait is typically slow and deliberate with a great deal of conscious effort. Therapists often assist required gait elements, including the weight shift, stabilization of the stance limb, or advancement of the dynamic limb. These compensatory strategies are effective in promoting early ambulation but do little to promote the balance and dynamic control needed for independent gait.

Once out of the parallel bars close stand-by guarding may be necessary. In general, a hands-off approach is recommended as soon as possible. Initially, the patient can use light touch-down (fingertip) support walking next to a treatment table or wall to maintain balance. Progression is then to walking away from the wall with no touch-down support. The therapist can verbally cue the patient to maintain the pace and symmetry of gait.

A variety of walking patterns should be practiced, including forwards, backwards, and sidewards. Sidestepping and crossed-stepping can be practiced first holding on to the outside of the parallel bars or to an oval bar progressing to no support. The PNF activity of *braiding* is a skill-level gait activity that involves alternating side-steps with cross-steps. One limb steps out to the

side while the other limb alternates between stepping up and across in front of the other leg (in a D1 flexion pattern) or back and around the other leg (in a D2 extension pattern). Alternating the patterns results in improved pelvic/lower trunk rotation and LE control. Locomotion can also be resisted using the PNF technique of Resisted Progression (RP). Manual contacts placed on the pelvis first lightly stretch and then resist motion. Improved timing and control of pelvic rotation is the goal. Resistance can also be provided using elastic resistance bands wrapped around the pelvis. The therapist walks near the patient, holding the ends of the resistance bands. Speed elements can be controlled using a metronome or brisk marching music. This is an important consideration for the patient with Parkinson's disease who responds well to rhythmic auditory cues (see Evidence Summary Box in Chapter 21).

Walking should be practiced on varying surfaces, from smooth surfaces to uneven terrain outside. Varying the BOS can challenge walking. The patient is instructed to walk first using a normal BOS to narrow BOS to finally semitandem or tandem walking. Having the patient walk with eyes open progressing to eyes closed can vary visual inputs. Having the patient walk while moving the head right and left or up and down can vary vestibular inputs. Walking with directional changes and abrupt starts and stops should be practiced. For example, the patient is asked to make an abrupt start and stop on command and to turn on command. Initially turns are wide progressing to more narrow turns; partial turns (90° turns) are progressed to full turns (180° turns). Attention can be varied by having the patient practice walking with while performing a second task (dual-task training). For example, the patient walks and carries an object or bounces a ball. Talking to the patient while walking can also divert attention (Walkie-Talkie Test). Walking through an obstacle course around and over objects can also be used to challenge control. Patients can be asked to stop and perform a task (march in place on a foam cushion) before proceeding on to the next obstacle.[182]

Climbing stairs is an important functional skill. For many patients, it may mean the difference between going home or going to an alternative living environment. Important lead-up activities for stair climbing include sit-to-stand transfers, standing weight shifting and stepping activities. Step-up and step-down exercises can be practiced using varying step heights from low (4 in. [10 cm]) to high (8 in. [20.3 cm]). The patient initially is instructed to step-up and step-down in the same direction and progressed to stepping up and down in different directions (up and over the step). Practice can begin in the parallel bars or next to a treatment table or wall for light touch-down support. Stair climbing is then practiced, first with limited stairs and progressing to a full flight of stairs. Consideration is given to the number of stairs required in the patient's home. Initial upper extremity support on handrails may be used to compensate for any instability the patient may experience. However, pulling forward with the hand during ascent or pushing during descent masks active control of trunk and lower extremity muscles. As soon as possible stair climbing should be practiced without upper extremity support. Practice on ramps may also be necessary for some patients. For patients with knee extension instability, ramps pose an increased challenge during descent while knee stabilization is enhanced during ascent.

Locomotor Training Using Body Weight Support Treadmill Training (BWSTT)

Locomotor training using partial BWS, a TM, and manually assisted limb movements has already been discussed in a previous section in this chapter as well as in Chapters 14, 18, and 23. There is no underestimating the importance this type of training has had in influencing recovery of gait. Evidence supports its efficacy as a training strategy for diverse groups of patients with deficits in motor function. Studies comparing traditional physical therapy approaches to gait with BWSTT have shown significant improvements in gait speed for the BWSTT group. Once treatment is stopped, the differences between the two groups become less apparent.[72,211,212]

Summary

This chapter outlined a conceptual framework for rehabilitation of the patient with deficits in motor function based on normal processes of motor control, motor learning, and recovery. Clinical decision making is based on a thorough examination of the patient's deficits in terms of impairments, functional limitations, and disability. The unique problems of each patient require that the therapist also recognize a number of interrelated factors, including individual needs, motivation, goals, concerns, and potential for independent function. Given the tremendous variability of patients with deficits in motor function, it is unrealistic to expect that any one intervention can be successful with all patients. Interventions must be carefully chosen to improve function and to minimize injury, future impairments, or disability. The effective use of motor learning strategies can dramatically improve treatment outcomes. Carefully planned and structured education empowers the patient. Patient skills in self-evaluation, problem solving, and decision making are promoted to foster independence. If patient independence is not possible because of the complexity of deficits and limitations in recovery, education of family, friends, and caregivers assumes paramount importance.

Questions for Review

1. Differentiate between the terms *motor control* and *motor learning.* How can impairments in motor control be distinguished from those of motor learning?
2. Describe how information is processed within the CNS to arrive at an appropriate plan for movement. What is the difference between a motor program and a motor plan?
3. Differentiate between the three stages of motor learning. How should training strategies differ during each stage?
4. Discuss motor learning training strategies designed to improve retention and generalizability. How do they differ from strategies that optimize performance?
5. Define neuroplasticity. What are the different theories proposed to explain recovery of function?
6. Define functional/task-specific training. Give three examples of interventions based on this approach.
7. Define the neurofacilitation approaches of NDT and PNF. How do they differ from each other? From compensatory training strategies? Give three examples of interventions of each.
8. Define compensatory training. Identify three interventions that can be considered compensatory training strategies.

Case Study

HISTORY

The patient is a 36-year-old man who sustained a traumatic brain injury following a motorcycle accident. On admission to a local hospital, the patient was found to have a left frontal laceration with an underlying linear skull fracture. CT scan revealed edema, a right basal ganglia contusion, and a left frontal contusion. The patient was comatose on admission. His acute hospital course was complicated by increased intracranial pressure and severe spasticity which required casts and splints. A gastric tube was inserted.

The patient's neurological status did not substantially improve at the acute hospital. He was transferred to a rehabilitation hospital 4 weeks post-injury for intensive rehabilitation. He had a brief readmission to the acute hospital during his sixth week post injury for stabilization of acute hypothermia and hypothyroidism. He was then returned to the rehabilitation facility for continued intensive rehabilitation. His medications consisted of Tegretol (200 mg po qid), multivitamins, and Colace.

PART I: PHYSICAL THERAPY EXAMINATION FINDINGS (INITIAL ADMISSION TO REHAB, 4 WEEKS POST-INJURY)

Cognition: The patient is semicomatose and unresponsive. He inconsistently responds to a command to "look at me" with eye opening and to "lift up your leg" with movement of the right leg only. Otherwise there is no response to auditory or visual stimulation.

Language–Communication: Unable to examine.

Social: Married with no children. Wife is a registered nurse and very supportive of her husband.

Vital Signs: Heart rate 60 bpm; blood pressure 122/70 mm Hg; respiratory rate 14 breaths per minute; O_2 sat level is 92.

Sensation: Localizes to pinprick with withdrawal.

Passive Range of Motion:
- RUE is limited in elbow ROM (0 to 70°); LUE elbow ROM is (10 to 100°).
- Both lower extremities (BLEs) are within normal limits except for ankle dorsiflexion 0 to 5° on R and 0 to 10° on L.

Motor Function

Tone: (Modified Ashworth Scale grades [M-AS]) Severe flexor tone and spasms of the trunk that result in the patient moving in bed from a supine to a left sidelying, curled-up (fetal) position, M-AS = 4
- Right upper extremity (RUE) extensor tone, M-AS = 3
- Right lower extremity (RLE) extensor tone, M-AS = 3
- LUE flexor tone, M-AS = 3
- LLE extensor tone, M-AS = 2

Reflexes:
- Frequent asymmetrical tonic neck reflex posturing with head rotated to the right
- Flexor withdrawal reflexes bilaterally in response to pain (delayed on the left with decreased intensity of response)
- Positive support reflex on the left
- Hyperactive deep tendon reflexes throughout
- At times the patient displays decorticate posturing with mass patterning. The lower extremities scissor at times, especially when upper body flexor tone increases

Voluntary Movements:
- The patient is agitated with restless movements and is frequently diaphoretic.
- No head or trunk control, dependent sitting
- Movement of RUE is spontaneous, purposeful at times, and out-of-synergy.

- Movement of the RLE is spontaneous, nonpurposeful, and out-of-synergy.
- LUE no active movement
- LLE movement is spontaneous, nonpurposeful, with abnormal obligatory synergy.

Coordination: Unable to assess; unresponsive.

Skin: Multiple healed lacerations on the knees and calves and pressure sores bilaterally on the lateral malleoli and calcanei from bivalve positioning splints.

Bladder and Bowel: Incontinent of bowel and bladder, and has external catheter in place.

Functional Activities: Dependent in all activities

PART I (QUESTIONS 1–3)

1. Identify and prioritize the clinical problems presented in this case in terms of direct and indirect impairments, and functional limitations based on initial admission data.
2. Identify the goals and outcomes of physical therapy intervention for this patient at this point in his recovery (initial admission).
3. Identify treatment interventions appropriate for this patient at this point in his recovery (initial admission).

PART II: REEXAMINATION 6 MONTHS POST-INJURY

Cognition:

- **Orientation:** The patient is alert, oriented × 3.
- **Memory:** Memory is impaired; shows limited carryover for relearned tasks such as self-care; past memories show more depth and detail than short-term memory.
- **Attention:** Attention span is reduced; he requires repetition and structure to complete activities.
- **Insight:** He demonstrates little insight into disability or safety awareness.

Behavior:

He is functioning at Rancho Levels of Cognitive Functioning (RLOCF) Level VI Confused-Appropriate. He shows some limited goal-directed behavior but is dependent on external direction. He shows some carryover for new learning but at a significantly decreased rate. Becomes easily frustrated and responds with disinhibited behaviors (name calling and swearing).

Language–Communication: The patient is dysarthric; speech is usually intelligible but difficult to understand and delayed in onset. Auditory comprehension is good.

Vital signs: WNL
Skin: Lacerations are healed.

Sensation:

- **Vision and hearing** are within normal limits.
- **LUE:** Absent sensation
- **LLE:** Impaired sensation, decreased proprioception.
- **RUE and RLE:** Intact

Range of Motion:

- **RUE:** elbow ROM 0 to 90°; LUE: elbow ROM 5 to 110°
- **BLEs:** Within normal limits except for ankle dorsiflexion bilaterally of 0 to 15°

Motor Function

- **Tone:** (modified Ashworth Scale grades, M-AS)
  Trunk: Tone in the trunk is within normal limits except for occasional flexor spasms
  RUE and RLE: Extensor tone, M-AS = 1
  LUE: Flexor tone, M-AS = 2
  LLE: Extensor tone, M-AS = 1

- **Reflexes:** Exhibits strong associated reactions in the LUE and increased flexor posturing with stressful activities.

- **Voluntary Movements:**
  RUE and RLE: Demonstrates purposeful, full, isolated motions through available ROM against gravity. Strength is grossly F+ in the RUE and RLE.
  LUE: No voluntary movement
  LLE: Movement is purposeful and in abnormal synergy; strength is grossly F.
  Head and Trunk: Movement is functional and strength is grossly F.

- **Coordination:**
  Exhibits moderate to severe ataxia in the head, trunk, and extremities.
  Demonstrates moderate impairment in finger-to-nose and toe-tapping test.

- **Balance:**
  Sitting:
  Static: Poor; requires handhold support and moderate assistance; demonstrates sacral siting with posterior tilt of the pelvis
  Dynamic: Poor; unable to accept challenge or move without loss of balance

Standing:
Static: Poor, requires maximal assist of two persons to stand in the parallel bars
Dynamic: Unable

Functional Activities:

- **Bed mobility:** Rolls to right and left with supervision (S)
- **Supine-to-sit:** Mod dependence, requires Min A to close S
- **Transfers:** Mod A × 1 in stand-pivot transfers

- **Wheelchair Locomotion:** Maneuvers power wheelchair with close S for safety
- **Gait:** Unable

PART II (QUESTIONS 4–6)

4. Identify and prioritize this patient's problems in terms of direct and indirect impairments, and functional limitations (6 months post-injury).
5. Identify the goals and outcomes of physical therapy intervention for this patient at this point in his recovery (6 months post-injury).
6. Identify treatment interventions appropriate for this patient at this point in his recovery (6 months post-injury).

References

1. Schmidt, R, and Lee, T: Motor Control and Learning, ed. 4. Human Kinetics, Champaign, IL, 2005.
2. Shumway-Cook, A, and Woollacott, M: Motor Control Theory and Practical Applications, ed 2. Lippincott Williams & Wilkins, Baltimore, 2001.
3. Sherrington, C. The Integrative Action of the Nervous System, ed 2. Yale University Press, New Haven, CT, 1947.
4. Taylor, J (ed): Selected Writings of John Hughlings Jackson. Basic Books, New York, 1958.
5. Brooks, V: The Neural Basis of Motor Control. Oxford University Press, New York, 1986.
6. Bernstein, N: The Coordination and Regulation of Movements. Pergamon Press, Oxford, 1967.
7. Kelso, JA: Dynamic Patterns: The Self-Organization of Brain and Behavior. MIT Press, Cambridge, MA, 1995.
8. Magill, R: Motor Learning Concepts and Applications, ed 4. Brown & Benchmark, Madison, WI, 1993.
9. Adams, J: A closed-loop theory of motor learning. J Motor Behav 3:111, 1971.
10. Schmidt, R: A schema theory of discrete motor skill learning. Psychol Rev 82:225, 1975.
11. Fitts, P, and Posner, M: Human Performance. Brooks/Cole, Belmont, CA, 1967.
12. Lee, T, and Swanson, L: What is repeated in a repetition? Effects of practice conditions on motor skill acquisition. Phys Ther 71:150, 1991.
13. Davies, P: Starting Again—Early Rehabilitation after Traumatic Brain Injury or Other Severe Brain Lesion. Springer-Verlag, New York, 1994.
14. Bobath, B: Adult Hemiplegia: Evaluation and Treatment, ed 2. Heinemann, London, 1978.
15. Singer, R, and Pease, D: A comparison of discovery learning and guided instructional strategies on motor skill learning, retention, and transfer. Res Q 47:788, 1976.
16. Salmoni, A, et al: Knowledge of results and motor learning: A review and critical appraisal. Psychol Bull 95:355, 1984.
17. Lee, T, et al: On the role of knowledge of results in motor learning: Exploring the guidance hypothesis. J Mot Behav 22:191, 1990.
18. Bilodeau, EA, et al: Some effects of introducing and withdrawing knowledge of results early and late in practice. J Exp Psychol 58:142, 1959.
19. Magill, R: Augmented feedback in motor skill acquisition. In Singer, RN, Hausenblas, HA, and Janell, CM (eds). Hanbook of Sport Psychology, ed 2, Wiley, New York, 2001, p 86.
20. Winstein, C, et al: Learning a partial-weight-bearing skill: Effectiveness of two forms of feedback. Phys Ther 76:985, 1996.
21. Bilodeau, E, and Bilodeau, I: Variable frequency knowledge of results and the learning of a simple skill. J Exp Psychol 55:379, 1958.
22. Ho, L, and Shea, J: Effects of relative frequency of knowledge of results on retention of a motor skill. Percept Mot Skills 46:859, 1978.
23. Sherwood, D: Effect of bandwidth knowledge of results on movement consistency. Percept Mot Skills 66:535, 1988.
24. Winstein, C, and Schmidt, R: Reduced frequency of knowledge of results enhances motor skill learning. J Exp Psychol Learn Mem Cogn 16:677, 1990.
25. Lavery, J: Retention of simple motor skills as a function of type of knowledge of results. Can J Psych 16:300, 1962.
26. Swinnen, S, et al: Information feedback for skill acquisition: Instantaneous knowledge of results degrades learning. J Exp Psychol Learn Mem Cogn 16:706, 1990.
27. Winstein, C: Knowledge of results and motor learning: Implications for physical therapy. Phys Ther 71:140, 1991.
28. Shea, J, and Morgan, R: Contextual interference effects on the acquisition, retention, and transfer of a motor skill. J Exp Psychol: Hum Learn 5:179, 1979.
29. Wulf, G, and Schmidt, R: Variability in practice facilitation in retention and transfer through schema formation or context effects? J Mot Behav 20:133, 1988.
30. Wulf, G, and Schmidt, R: Variability of practice and implicit motor learning. J Exp Psychol: Learn, Mem, Cogn 23:987, 1997.
31. Lee, T, Wulf, G, and Schmidt, R: Contextual interference in motor learning: Dissociated effects due to the nature of task variations. Q J Exp Psychol 44A:627, 1992.
32. Lee, T, and Magill, R: The locus of contextual interference in motor skill acquisition. J Exp Psychol Learn Mem Cogn 9:730, 1983.
33. Jeannerod, M: Neural simulation of action: A unifying mechanism for motor cognition. Neuroimage 14:103, 2001.
34. Jeannerod, M, and Frak, V: Mental imaging of motor activity in humans. Curr Opin Neurobiol 9:735, 1999.
35. Feltz, D, and Landers, D: The effects of mental practice on motor skill learning and performance: A meta-analysis. J Sports Psychol 5:25, 1983.
36. Richardson, A: Mental practice: A review and discussion (Part 1). Res Q 38:95, 1967.
37. Richardson, A: Mental practice: A review and discussion (Part 2). Res Q 38:263, 1967.
38. Warner, L, and McNeill, M: Mental imagery and its potential for physical therapy. Phys Ther 68:516, 1988.
39. Maring, J: Effects of mental practice on rate of skill acquisition. Phys Ther 70:165, 1990.
40. Lee, TD: Transfer-appropriate processing: A framework for conceptualizing practice effects in motor learning. In: Meijer O and Roth K (eds). Complex Movement Behavior: The Motor-Action Controversy. North Holland, Amsterdam, 1988, p 201.
41. Bayley, N: The development of motor abilities during the first three years. Monogr Soc Res Child Dev 1 (1, serial no 1), 1935.
42. Gesell, A, and Amatruda, C: Developmental Diagnosis. Harper, New York, 1941.
43. McGraw, M: The Neuromuscular Maturation of the Human Infant. Hafner, New York, 1945.
44. Keogh, J, and Sugden, D: Movement Skill Development. Macmillan, New York, 1985.
45. VanSant, A: Life span development in functional tasks. Phys Ther 70:788, 1990.
46. VanSant, A: Rising from a supine position to erect stance: Description of adult movement and a developmental hypothesis. Phys Ther 69:185, 1988.
47. Woollacott, M, and Shumway-Cook, A: Changes in posture control across the life span: A systems approach. Phys Ther 70:799, 1990.
48. Woollacott, M, and Shumway-Cook, A (eds): Development of Posture and Gait Across the Life span. University of South Carolina Press, Columbia, 1989.
49. Light, K: Information processing for motor performance in aging adults. Phys Ther 70:821, 1990.

50. Schlendorf, S: Effects of aging and exercise on the adult central nervous system: A literature review. Neurology Report (now JNPT) 15:24, 1991.
51. Salthouse, T, and Somberg, B: Isolating the age deficit in speeded performance. J Gerontol 37:59, 1982.
52. Light, K, and Spirduso, W: Effects of adult aging on the movement complexity factor of response programming. J Gerontol 45:107, 1990.
53. Spirduso, W: Physical fitness, aging, and psychomotor speed: A review. J Gerontol 35:850, 1980.
54. Shephard, R: Physical Activity and Aging, ed 2. Aspen, Rockville, MD, 1987.
55. Morris, J, and McManus, D: The neurology of aging: Normal versus pathologic change. Geriatrics 46:47, 1991.
56. Stein, D, Failowsky, B, and Will, B: Brain Repair. Oxford University Press, New York, 1995.
57. Cicinelli, P, Traversa, R, and Rossini, P: Post-stroke reorganization of brain motor output to the hand: A 2–4 month follow-up with focal magnetic transcranial stimulation. Electroenceph Clin Neurophysiol 105:438, 1997.
58. Traversa, R, et al: Mapping of motor cortical reorganization after stroke. Stroke 28:110, 1997.
59. Pohl, P, and Richards, L: Changes in brain activity with motor learning in humans. Neurology Report (now JNPT) 22:79, 1998.
60. van Praag, H, Kemperman, G, and Gage, F: Running increases cell proliferation and neurogenesis in adult mouse dentate gyrus. Nat Neurosci 2:266, 1999.
61. Ploughman, M: A review of brain neuroplasticity and implications for the physiotherapeutic management of stroke. Physiother Can, Summer: 164, 2002.
62. Liepert, J, et al: Motor cortex plasticity during constraint-induced movement therapy in stroke patients. Neurosci Lett 250:5, 1998.
63. Liepert, J, et al: Training-induced changes of motor cortex representations in stroke patients. Acta Neurol Scand 101:321, 2000.
64. Liepert, J, et al: Treatment-induced cortical reorganization after stroke in humans. Stroke 31:1210, 2000.
65. Levy, C, et al: Functional MRI evidence of cortical reorganization in upper-limb stroke hemiplegia treated with constraint-induced movement therapy. Am J Phys Med Rehabil 80:4, 2001.
66. Domerick, A, Edwards, D, and Hahn, M: Does the application of constrain-induced movement therapy during acute rehabilitation reduce arm impairment after ischemic stroke? Stroke 31:2984, 2000.
67. Blanton, S, and Wolf, S: Case Report: An application of upper-extremity constraint-induced movement therapy in a patient with sub-acute stroke. Phys Ther 79:847, 1999.
68. Miltner, W, et al: Effects of constraint-induced movement therapy on patients with chronic motor deficits after stroke: A replication. Stroke 30:586, 1999.
69. Kunkel, A, et al: Constraint-induced movement therapy for motor recovery in chronic stroke patients. Arch Phys Med Rehabil 80:624, 1999.
70. Van der Lee, J, et al: Forced use of the upper extremity in chronic stroke patients: Results from a single-blind randomized clinical trial. Stroke 30:2369, 1999.
71. Taub, E, et al: Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehabil 74:347, 1993.
72. Richards, C, et al: Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Arch Phys Med Rehabil 74:612, 1993.
73. Visitin, M, et al: A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. Stroke 29:1122, 1998.
74. Visintin, M, and Barbeau, H: The effects of body weight support on the locomotor pattern of spastic paretic patients. Can J Neurol Sci 16:315, 1989.
75. Field-Fote, E: Spinal cord control of movement: Implications for locomotor rehabilitation following spinal cord injury. Phys Ther 80:477, 2000.
76. Kempermann, G, Kuhn, H, and Gage, F: More hippocampal neurons in adult mice living in an enriched environment. Nature 386:493, 1997.
77. Comery, T, et al: Increased density of multiple-head dendritic spines on medium-sized spiny neurons of the striatum of rats reared in a complex environment. Neurobiol Learn Mem 66:93, 1996.
78. Rosenzweig, M, and Bennet, E: Effects of differential environments on brain weights and enzyme activities in gerbils, rats, and mice. Dev Psycholbiol 2:87, 1969.
79. Smith, R, Parts, T, and Lynch, G: A comparison of the role of the motor cortex in recovery from cerebellar damage in young and adult rats. Behav Biol 12:77, 1974.
80. Held, J, Gordon, J, and Gentile, A: Environmental influences on locomotor recovery following cortical lesions in rats. Behav Neurosci 99:678, 1985.
81. Held, J: Environmental enrichment enhances sparing and recovery of function following brain damage. NeuroReport 22:74, 1998.
82. Ohlsson, A, and Johansson, B: Environment influences functional outcome of cerebral infarction in rats. Stroke 26:644, 1995.
83. Johansson, B, and Ohlsson, A: Environment, social interaction and physical activity as determinants of functional outcome after cerebral infarction in the rat. Exp Neurol 139:322, 1996.
84. Kozlowski, D, James, D, and Schallert, T: Use-dependent exaggeration of neuronal injury after unilateral sensorimotor cortex lesions. J Neurosci 16:4776, 1996.
85. Humm, J, et al: Use-dependent exaggeration of brain injury: Is glutamate involved? Exp Neurol 157:349, 1999.
86. Humm, J, et al: Use-dependent exacerbation of brain damage occurs during an early post-lesion vulnerable period. Brain Res 783:286, 1988.
87. Lawton, P: Therapeutic environments for the aged. In Canter, S and Canter, D (eds): Designing Therapeutic Environments. A review of Research. New York, John Wiley, p 233.
88. Carr, J, and Shepherd, R: A Motor Relearning Programme for Stroke, ed 2. Aspen, Rockville, MD, 1987.
89. Carr, J, and Shepherd, R: A motor learning model for rehabilitation. In Carr, J and Shephard, R (eds): Movement Science. Foundations for Physical Therapy in Rehabilitation. Aspen, Rockville MD, p 31.
90. Karla, L: The influence of stroke unit rehabilitation on functional recovery following stroke. Stroke 25:821, 1994.
91. Indredavik, B, et al: Stroke unit treatment. Long term effects. Stroke 28:1861, 1997.
92. Langhorne, P, and Duncan, P: Does the organization of post acute stroke care really matter? Stroke 32:268, 2001.
93. Carr, J, and Shepherd, R: Neurological Rehabilitation: Optimizing Motor Performance. Butterworth Heinemann, Oxford, 1998.
94. Lincoln, N, et al: Behavioural mapping of patients on a stroke unit. Int Disabil Studies 11:149, 1989.
95. Tinson, D: How stroke patients spend their days. Int Disabil Stud 11:45, 1989.
96. Mackey, F, et al: Stroke rehabilitation: Are highly structured units more conducive to physical activity than less structured units? Arch Phys Med Rehabil 77:1066, 1996.
97. Gordon, J: Assumptions underlying physical therapy intervention: Theoretical and historical perspectives. In Carr, J, and Shepherd, R (eds): Movement Science Foundations for Physical Therapy Rehabilitation. Aspen, Rockville, MD, 2000, p 1.
98. Bobath, B: The treatment of neuromuscular disorders by improving patterns of coordination. Physiotherapy 55:1, 1969.
99. Howle, J: Neuro-Developmental Treatment Approach. Neuro-Developmental Treatment Association, Laguna Beach CA, 2002.
100. Voss, D, et al: Proprioceptive Neuromuscular Facilitation, ed 3. Harper & Row, Philadelphia, 1985.
101. Adler, S, Beckers, D, and Buck, M: PNF in Practice, ed 2. Springer-Verlag, New York, 2003.
102. Taub, E: Movement in nonhuman primates deprived of somatosensory feedback. Exerc Sports Sci Rev 4:335, 1976.
103. American Physical Therapy Association: Guide to physical therapist practice. Phys Ther 81:1, 2001.
104. Smidt, G, and Rogers, M: Factors contributing to the regulation and clinical assessment of muscular strength. Phys Ther 62:1283, 1982.
105. Kisner, C, and Colby, L: Therapeutic Exercise Foundations and Techniques, ed 4. FA Davis, Philadelphia, 2002.
106. Enoka, R: Chronic adaptations. In Enoka R (ed): Neuromechanical Basis of Kinesiology, ed 3. Human Kinetics, Champaign, IL, 2002.
107. Harris, B, and Watkins, M: Adaptations to strength conditions. In Frontera, W, Dawson, D, and Slovik, D (eds): Exercise in Rehabilitation Medicine. Human Kinetics, Champaign, IL, 1999. p 71.

108. Bandy, W, and Sanders, B: Therapeutic Exercise—Techniques for Intervention. Lippincott Williams & Wilkins, Philadelphia, 2001.
109. Eng, J: Strength training in individuals with stroke. Physiother Can 56:189, 2004.
110. O'Sullivan, S, and Schmitz, T: Physical Rehabilitation Laboratory Manual: Focus on Functional Training. FA Davis, Philadelphia, 1999.
111. Hall, C, and Brody, L: Therapeutic Exercise—Moving Toward Function, ed 2. Lippincott Williams & Wilkins, Philadelphia, 2005.
112. American College of Sports Medicine: ACSM's Guidelines for Exercise Testing and Prescription, ed 6. Lippincott Williams & Wilkins, Philadelphia, 2000.
113. Curtis, C, and Weir, J: Overview of exercise responses in healthy and impaired states. Neurology Report (now JNPT) 20:13, 1996.
114. Bennett, R, and Knowlton, G: Overwork weakness in partially denervated skeletal muscle. Clin Orthop 12:22, 1958.
115. Dean, E: Effect of modified aerobic training on movement energetics in polio survivors. Orthopedics 14:1253, 1991.
116. Fillyaw, M, et al: The effects of long-term non-fatiguing resistance exercise in subjects with post-polio syndrome. Orthopedics 14:1252, 1991.
117. Aitkens, S, et al: Moderate resistance exercise program: Its effects in slowly progressive neuromuscular disease. Arch Phys Med Rehabil 74:711, 1993.
118. American College of Sports Medicine: ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities. Human Kinetics, Champaign, IL, 1997.
119. American College of Sports Medicine: ACSM's Resources for Clinical Exercise Physiology. Lippincott Williams & Wilkins, Philadelphia, 2002.
120. Spirduso,W: Physical Dimensions of Aging. Human Kinetics, Champaign, IL, 1995.
121. Lentell, G, et al: The use of thermal agents to influence the effectiveness of a low-load prolonged stretch. J Orthop Sports Phys Ther 16:200, 1992.
122. Wessling, K, et al: Effects of static stretch versus static stretch and ultrasound combined on triceps surae muscle extensibility in healthy women. Phys Ther 67:674, 1987.
123. Cornelius, W, and Jackson, A: The effects of cryotherapy and PNF techniques on hip extensor flexibility. J Athl Train 19:183, 1984.
124. Kottke, F, et al: The rationale for prolonged stretching of shortened connective tissue. Arch Phys Med Rehabil 47: 345, 1982.
125. Anderson, B, and Burke, E: Scientific, medical, and practical aspects of stretching. Clin Sports Med 10:63, 1991.
126. Bandy, W, and Irion, J: The effects of time on static stretch on the flexibility of the hamstring muscles. Phys Ther 74:845, 1994.
127. Gajdosik, R: Effects of static stretching on the maximal length and resistance to passive stretch of short hamstring muscles. J Orthop Sports Phys Ther 13:126, 1991.
128. Bohannon, R, and Larkin, P: Passive ankle dorsiflexion increases in patients after a regimen of tilt table: Wedge board standing. Phys Ther 65:1676, 1985.
129. Light, K, et al: Low-load prolonged stretch vs. high-load brief stretch in treating knee contractures. Phys Ther 64:330, 1984.
130. Zachazewski, J: Flexiblity for sports. In: Sanders, B (ed): Sports Physical Therapy. Appleton & Lange, Norwalk, CT, 1990, p 201.
131. Sady, S, et al: Flexibility training: Ballistic, static or proprioceptive neuromuscular facilitation. Arch Phys Med Rehabil 63:261, 1982.
132. Markos, P: Ipsilateral and contralateral effects of proprioceptive neuromuscular facilitation techniques on hip motion and electromyographic activity. Phys Ther 59:1366, 1979.
133. Etnyre, B, and Abraham, L: Gains in range of ankle dorsiflexion using three popular stretching techniques. Am J Phys Med 65:189, 1986.
134. Etnyre, B, and Lee, E: Chronic and acute flexibility of men and women using three different stretching techniques. Res Q 59:222, 1988.
135. Cornelius, W, et al: The effects of cold application and modified PNF stretching techniques on hip flexibility in college males. Res Q Exerc Sport 63:311, 1992.
136. Osternig, L, et al: Differential response to proprioceptive neuromuscular facilitation (PNF) stretch technique. Med Sci Sports Exerc 22:106, 1990.
137. Johnstone, M: Restoration of Normal Movement After Stroke. Churchill Livingstone, New York, 1995.
138. Giuliani, C: The relationship of spasticity to movement and consideration for therapeutic interventions. Neurol Report (now JNPT) 21: 78, 1997.
139. Light, K, and Giuliane, C: Effect of isokinetic exercise effort on arm coordination of spastic hemiparetic subjects. Neurology Report (now JNPT) 16:19, 1992.
140. Giuliani, C, Light, K, and Rose, D: The effects of isokinetic exercise program in gait patterns of patients with hemiparesis. Neurology Report (now JNPT) 4:23, 1993.
141. Brown, D, and Kautz, S: Increased workload enhances force output during pedaling exercise in persons with poststroke hemiplegia. Stroke 29:598, 1998.
142. Damiano, D, and Abel, M: Functional outcomes of strength training in spastic cerebral palsy. Arch Phys Med Rehabil 79:119, 1998.
143. Damino, D, Vaughan, C, and Abel, M: Muscle response to heavy resistance exercise in children with spastic cerebral palsy. Dev Med Child Neurol 38:731, 1995.
144. Miller, G, Light, K, and Kellog, R: Comparison of isometric force control measures in spastic muscle of post-stroke individuals before and after graded resistive exercise. Neurol Report 20:92, 1996.
145. Hall, C, and Light, K: Heavy restrictive exercise effect on reciprocal movement coordination of closed-head injured subjects with spasticity. Neurology Report (now JNPT) 14:19, 1990.
146. Hunter, M, Tomberlin, J, and Kuna, S: Progressive exercise testing in closed head-injuried subjects comparison of exercise apparatus in assessment of a physical conditioning program. Phys Ther 70:363, 1990.
147. Jankowski, LW, and Sullivan, SJ: Aerobic and neuromuscular training: Effect on the capacity efficiency and fatigability of patients with traumatic brain injuries. Arch Phys Med Rehab 71(7):500, 1990.
148. Potempa, K, et al: Physiologic outcomes of aerobic exercise training in hemiparetic stroke patients. Stroke 26:101, 1995.
149. Booth, B, Doyle M, and Montgomery J: Serial casting for the management of spasticity in the head-injuried adult. Phys Ther 63:1960, 1983.
150. Barnard, P, et al: Reduction of hypertonicity by early casting in a comatose head-injured individual: A case report. Phys Ther 64:1540, 1984.
151. Booth, BJ, et al: Serial casting for the management of spasticity in the head-injured adult. Phys Ther 63:1960, 1983.
152. Lehmkuhl, L, et al: Multimodality treatment of joint contractures in patients with severe brain injury: Cost, effectiveness, and integration of therapies in the application of serial/inhibitive cases. J Head Trauma Rehabil 5:23, 1990.
153. Hill, J: The effects of casting on upper extremity motor disorders after brain injury. Am J Occup Ther 48:219, 1994.
154. Moseley, A: The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. Phys Ther 77:240, 1997.
155. Singer, B, et al: Evaluation of serial casting to correct equinovarus deformity of the ankle after acquired brain injury in adults. Arch Phys Med Rehabil 84:483, 2003.
156. Bronski, B: Serial casting for the neurological patient. American Physical Therapy Association, Physical Disabilities Special Interest Section Newsletter 18:4, 1995.
157. Zablotny, C, et al: Serial casting: Clinical applications for the adult head-injured patient. J Head Trauma Rehabil 2:46, 1987.
158. Sullivan T, et al: Serial casting to prevent equinus in acute traumatic head injury. Physiother Can 40:346, 1988.
159. Singer, B, Singer, K, and Allison, G: Serial plastering to correct equinovarus deformity of the ankle following acquired brain injury in adults: Review and clinical applications. Disabil Rehabil 23:829, 2001.
160. Kent, H, et al: Case control study of lower extremity serial casting in adult patients with head injury. Physiother Can 42:189, 1990.
161. Rothstein, J, et al: The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. Phys Ther 77:248, 1997.
162. Gracies, J: Pathophysiology of impaiment in patients with spasticity and the use of stretch as a treatment for spastic hypertonia. Phys Med Rehabil Clin North Am 12:747, 2001
163. Tabary, J, et al: Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. J Physiol 224:231, 1972.

164. Feldman, P: Upper extremity casting and splinting. In Glenn, M, and Whyte, J (eds): The Practical Management of Spasticity in Children and Adults. Lea & Febiger, Philadelphia, 1990.
165. Giorgetti, M: Serial and inhibitory casting: Implications for acute care physical therapy management. Neurology Report (now JNPT) 17:18, 1993.
166. Collins, K, et al: Customized adjustable orthosis: Their use in spasticity. Arch Phys Med Rehabil 66:397, 1985.
167. Grissom, S, and Blanton, S: Treatment of upper motoneuron plantarflexion contractures by using an adjustable ankle-foot orthosis. Arch Phys Med Rehabil 82:270, 2001.
168. Hepburn, GR: Case studies: Contracture and stiff joint management with Dynasplint. J Orthop Sports Phys Ther 8:498, 1987.
169. MacKay-Lyons, M: Low-load, prolonged stretch in treatment of elbow flexion contractures, secondary to head trauma: A case report. Phys Ther 69:292, 1988.
170. Stefanovska, A, et al: Effects of electrical stimulation on spasticity. Crit Rev Phys Rehabil Med 3:59, 1991.
171. Sieb, T, et al: The quantitative measurement of spasticity: Effect of cutaneous electrical stimulation. Arch Phys Med Rehabil 75:746, 1994.
172. Levin, M, and Hui-Chan, C: Relief of hemiparetic spasticity by TENS is associated with improvement in reflex and voluntary motor functions. Electroencephalogr Clin Neurophysiol 85:131, 1992.
173. Carmick, J: Managing equines in children with cerebral palsy: Electrical stimulation to strengthen the triceps surae muscle. Dev Med Child Neurol 37:965, 1995.
174. Cauraugh, J, et al: Chronic motor dysfunction after stroke: Recovering wrist and finger extension by electromyography-triggered neuromuscular stimulation. Stroke 31:1360, 2000.
175. Kraft, G, Fitts, S, and Hammond, M: Techniques to improve function of the arm and hand in chronic hemiplegia. Arch Phys Med Rehabil 73:220, 1992.
176. Hui-Chan, C, and Levin, M: Stretch reflex latencies in spastic hemiparetic subjects are prolonged after transcutaneous electrical nerve stimulation. Can J Neurol Sci 20:97, 1993.
177. Belanger, AY: Neuromuscular electrostimulation in physiotherapy: A critical appraisal of controversial issues. Physiother Theo Pract 7:83, 1991.
178. Barton, L: Uses of surface EMG for neuromuscular evaluation and training in patients with neurological impairments. Biofeedback Newsmagazine of the AAPB 24:12, 1996.
179. Colborne, G, et al: Feedback of triceps surae EMG in gait of children with cerebral palsy: A controlled study. Arch Phys Med Rehabil 75:40, 1994.
180. Glanz, M, et al: Biofeedback therapy in post-stroke rehabilitation: A meta-analysis of the randomized controlled trials. Arch Phys Med Rehabil 76:508, 1995.
181. Creager, C: Therapeutic Exercises using the Swiss Ball. Executive Physical Therapy, Boulder, CO, 1994.
182. Rose, D: Fall Proof – A Comprehensive Balance and Mobility Training Program. Human Kinetics, Champaign, IL, 2003.
183. Nashner, L: Fixed patterns of rapid postural responses among leg muscles during stance. Exp Brain Res 30:13, 1977.
184. Nashner, L: Adapting reflexes controlling the human posture. Exp Brain Res 26:59, 1976.
185. Horak, F, and Nashner, L: Central programming of postural movements: Adaptation to altered support surface configurations. J Neurophysiol 55:1369, 1986.
186. McIlroy, W, and Maki, B: Adaptive changes to compensatory stepping responses. Gait Posture 3:43, 1995.
187. Maki, B, and McIlroy, W: The role of limb movements in maintaining upright stance: The "change-in-support" strategy. Phys Ther 77:488, 1997.
188. Horak, F, et al: Postural perturbations: New insight for treatment of balance disorders. Phys Ther 77:517, 1997.
189. Damis, C, et al: Relationship between standing posture and stability. Phys Ther 78:502, 1998.
190. Wannstedt, F, and Herman, R: Use of augmented sensory feedback to achieve symmetrical standing. Phys Ther 58:553, 1978.
191. Hocherman, S, et al: Platform training and postural stability in hemiplegia. Arch Phys Med Rehabil 65:588, 1984.
192. Shumway-Cook, A, Anson, D, and Haller, S: Postural sway biofeedback: Its effect on reestablishing stance stability in hemiplegic patients. Arch Phys Med Rehabil 69:395, 1988.
193. Hammon, R, et al: Training effects during repeated therapy sessions of balance training using visual feedback. Arch Phys Med Rehabil 73:738, 1992.
194. Moore, S, and Woollacott, M: The use of biofeedback devices to improve postural stability. Phys Ther Practice 2:1, 1993.
195. Nichols, D: Balance retraining after stroke using force platform biofeedback. Phys Ther 77:553, 1997.
196. Kasser, S, Rose, D, and Clark, S: Balance training for adults with multiple sclerosis: Multiple case studies. Neurol Report (now JNPT) 23:5, 1999.
197. Nashner, L, and McCollum, G: The organization of human postural movements: A formal basis and experimental synthesis. Behav Brain Sci 8:135, 1985.
198. Benda, B, et al: Biomechanical relationship between center of gravity and center of pressure during standing. IEEE Trans Rehab Eng 2:3, 1994.
199. Winstein, C, et al: Standing balance training: Effect on balance and locomotion in hemiparetic adults. Arch Phys Med Rehabil 70:755, 1989.
200. Winstein, C: Balance retraining: Does it transfer? In Duncan, P (ed): Balance. American Physical Therapy Association, Alexandria, VA, 1990, p 95.
201. Gapsis, J, et al: Limb load monitor: Evaluation of a sensory feedback device for controlling weight-bearing. Arch Phys Med Rehabil 63:38, 1982.
202. Gauthier-Gagnon, C, et al: Augmented sensory feedback in the early training of standing balance of below-knee amputees. Physiother Can 38:137, 1986.
203. Jeka, J: Light touch contact as a balance aid. Phys Ther 77:476, 1997.
204. Bundy, AC, Lane, SJ, and Murray, EA: Sensory Integration: Theory and Practice, ed 2. FA Davis, Philadelphia, 2002.
205. Wynn Parry, C, and Salter, M: Sensory re-education after median nerve lesions. The Hand 8:250, 1976.
206. Goldman, H: Improvement of double simultaneous stimulation perception in hemiplegic patients. Arch Phys Med Rehabil 47:681, 1966.
207. Weinberg, S, et al: Training sensory awareness and spatial organization in people with right brain damage. Arch Phys Med Rehabil 60:491, 1979.
208. Yekatiel, M, and Guttman, E: A controlled trial of the retraining of the sensory function of the hand in stroke patients. J Neurol Neurosurg Psychiatry 56:241, 1993.
209. Nashner, L: Sensory, neuromuscular, and biomechanical contributions to human balance. In Duncan, P (ed): Balance. American Physical Therapy Association, Alexandria, VA, 1990, p 5.
210. Forssberg, H: Spinal locomotor functions and descending control. In Sjolund, G and Bjorklund, A (eds) Brain Stem Control of Spinal Mechanisms. Elsevier Biomedical Press, New York, 1982, p 103.
211. Harburn, K, et al: An overhead harness and trolley system for balance and ambulation assessment and training. Arch Phys Med Rehabil 74:220, 1993.
212. Hesse, S, Konrad, M, and Uhlenbroch, D: Treadmill walking with partial body weight support versus floor walking in hemiparetic subjects. Arch Phys Med Rehabil 80:421, 1999.

Supplemental Readings

Carr, J, and Shephard, R: Neurological Rehabilitation Optimizing Motor Performance. Butterworth-Heinemann, Woburn, MA, 1998.
Frontera, W, Dawson, D, and Slovik, D (eds): Exercise in Rehabilitation Medicine. Human Kinetics, Champaign, IL, 1999.
Schmidt, R, and Lee, T: Motor Control and Learning, ed 4. Human Kinetics, Champaign, IL, 2005.
Shumway-Cook, A, and Woollacott, M: Motor Control Theory and Practical Applications, ed 2. Lippincott Williams & Wilkins, Philadelphia, 2001.

Appendix A: Neurodevelopmental Treatment Approach (NDT)[98,99]

I. NDT Basic Principles

- NDT is based on an ongoing analysis of sensorimotor function and carefully planned interventions designed to improve function. Principles of motor control, motor learning, and motor development guide the planning process.
- Therapy focuses on the client's strengths and competencies while at the same time addressing impairments, functional limitations, and disabilities. Negative signs (weakness, impaired postural control, and paucity of movement) are equally important to address in treatment as positive signs (spasticity, hyperactive reflexes).
- The POC is developed in partnership with the patient, family, and interdisciplinary team.
- Treatment focuses on the relationship between sensory input and motor output.
- Therapeutic handling is the primary NDT intervention strategy. Facilitatory and/or inhibitory inputs are provided to influence the quality of motor responses.
- Training is focused on specific task goals and functional skills. The task and/or environment is modified as needed to enhance function.
- Active participation by the patient is a goal and an expectation of treatment.
- A major role of the therapist is assisting in an accurate analysis of motor problems and development of effective solutions.
- Motor learning principles are adhered to in the therapeutic setting, including verbal reinforcement, repetition, facilitation of error awareness (trial and error learning), an environment conducive to learning, engaging the patient/client/family and ensuring motivation.
- Direct teaching of the patient/client/family/caregiver to ensure carryover of functional activities in the home and community setting is an important component.

II. NDT Intervention Strategies and Techniques

Therapeutic Handling: Therapeutic handling is used to influence the quality of the motor response and is carefully matched to the patient's abilities to use sensory information and adapt movements. It includes neuromuscular facilitation, inhibition, or frequently a combination of the two. Manual contacts are used to:

- "Direct, regulate, and organize tactile, proprioceptive and vestibular input,
- Direct the client's initiation of movement more efficiently and with more effective muscle synergies,
- Support or change alignment of the body in relation to the BOS and with respect to the force of gravity prior to and during movement sequences,
- Decrease the amount of force the client uses to stabilize body segments,
- Guide or redirect the direction, force, speed, and timing of muscle activation for successful task completion,
- Either constrain or increase the flexibility in the degrees of freedom needed to stabilize or move body segments in a functional activity,
- Dense the response of the client to sensory input and the movement outcome and provide nonverbal feedback for reference of correction,
- Recognize when the client can become independent of the therapist's assistance and take over control of posture and movement, and
- Direct the client's attention to meaningful aspects of the motor task."[99, p 259]

Key Points of Control: Key points are parts of the body the therapist chooses as optimal to control (inhibit or facilitate) postures and movement. *Proximal key points* include the shoulders and pelvis and are used to influence proximal segments and trunk. *Distal key points* upper and lower extremities (typically the hands and feet).

Key points of control are also used to provide inhibition of abnormal tone and postures. Examples include:

- Head and trunk flexion decreases shoulder retraction, trunk and limb extension (key points of control: head and trunk).
- Humeral external rotation and flexion to 90 degrees decreases flexion tone of the upper extremity (key point of control: humerus).
- Thumb abduction and extension with forearm supination decreases flexion tone of the wrist and fingers (key point of control: the thumb).
- Femoral external rotation and abduction decreases extensor/adductor tone of the lower extremity (key point of control: hip).

Facilitation: Components of posture and movement that are essential for successful functional task performance are facilitated through therapeutic handling and key points.

Inhibition: Components of posture and movement that are atypical and prevent development of desired motor patterns are inhibited. While originally this term referred strictly to the reduction of tone and abnormal reflexes, in current NDT practice it refers to reduction of any underlying impairment that interferes with functional performance. It can be used to:

- "Prevent or redirect those components of a movement that are unnecessary and interfere with intentional, coordinated movement,
- Constrain the degrees of freedom, to decrease the amount of force the client uses to stabilize posture,
- Balance antagonistic muscle groups, or
- Reduce spasticity or excessive muscle stiffness that interferes with moving specific segments of the body."[99, p 261]

Appendix B: Proprioceptive Neuromuscular Facilitation (PNF)[100,101]

I. PNF Basic Procedures for Facilitation

Patterns of Motion: Normal motor activity occurs in synergistic and functional patterns of movement. PNF patterns are "spiral and diagonal" in character and combine motion in all three planes (flexion/extension, abduction/adduction, and transverse rotation). They closely resemble patterns used in normal functional activity and sports.

Extremity patterns are named for the action occurring at the proximal joint or by the diagonal (antagonist pairs of patterns make up the diagonal).

Upper Extremity (UE) Diagonal 1 = Flexion, Adduction, External Rotation (D1 Flexion) and Extension, Abduction, Internal Rotation (D1 Extension)

UE Diagonal 2 = Flexion, Abduction, External Rotation (D2 Flexion) and Extension, Adduction, Internal Rotation (D2 Extension)

Lower Extremity (LE) Diagonal 1 = Flexion, Adduction, External Rotation (D1 Flexion) and Extension, Adduction, Internal Rotation (D1 Extension)

LE Diagonal 2 = Flexion, Abduction, Internal Rotation (D2 Flexion) and Extension, Adduction, External Rotation (D2 Extension)

Patterns are varied by changing the action of the intermediate joint (i.e., elbow or knee) or by changing the position of the patient (i.e., supine, sitting, standing). Patterns can be unilateral or bilateral (i.e., symmetrical, asymmetrical, or reciprocal).

Trunk patterns include chop and lift patterns, bilateral lower extremity (LE) patterns, scapula and pelvis patterns, and head/neck patterns.

Timing: Normal timing ensures smooth, coordinated movement. In PNF patterns normal timing is from distal to proximal. Distal segments (hand/wrist or foot/ankle) move first followed closely by more proximal components. Rotation occurs throughout the pattern, from beginning to end.

Timing for Emphasis (TE): Maximum resistance is used to elicit a strong contraction and allow overflow to occur from strong to weak components within a synergistic pattern; the strong muscles are resisted isometrically ("locking in") while motion is allowed in the weaker muscles.

Indications: Weakness and/or incoordination

Resistance: Resistance facilitates muscle contraction and motor control. Both intrafusal and extrafusal muscle fibers contract, resulting in recruitment of motor units and improved strength of contraction. Resistance is applied manually and functionally through the use of gravity to all types of contractions (isotonic—concentric and eccentric; isometric).

- **Tracking or Light Resistance** applied to weak muscles is facilitatory and is usually applied in combination with light stretch.
- **Maximal Resistance** (the greatest amount of resistance tolerated by the patient) is used to generate maximal effort and adjusted to ensure smooth, coordinated movement; maximal resistance varies according to the individual patient.

Indications: Facilitate weak muscles to contract; enhance kinesthetic awareness of motion; increase strength; increase motor control and motor learning.

Overflow or Irradiation: Refers to the spread of muscle response from stronger muscles in a synergistic pattern to weaker muscles; maximal resistance is the main mechanism for securing overflow or irradiation. Stronger patterns can also be used to reinforce weaker patterns through mechanisms of overflow or irradiation from one extremity to the other, or extremity to trunk.

Indications: Enhance synergistic actions of muscles, increase strength.

Manual Contacts (MC): Precise manual contacts (grip) are used to provide pressure to tactile and pressure receptors overlying muscles to facilitate contraction and guide direction of movements; pressure is applied opposite to the direction of the desired motion.

Indications: Enhance contraction and synergistic patterns.

Positioning: Muscle positioning at optimal range of function allows for optimal responses of muscles (*length–tension relationship*). The greatest muscle tension is generated in mid-ranges with weak contractile force (*active insufficiency*) occurring in the shortened ranges.

The lengthened range provides optimal stretch for muscle spindle support of contraction while the shortened range with muscle spindle unloading provides the least amount of muscle spindle support for contraction.

Indications: Enhance weak contraction.

Therapist Position and Body Mechanics: Therapist is positioned directly *in line* with the desired motion (facing the direction of the movement) in order to optimize the direction of resistance that is applied.

Indications: Enhance therapist's control of the patient's movements; reduce therapist fatigue through effective use of body weight and position.

Verbal Commands (VCs): Verbal commands allow for the use of well-timed words and appropriate vocal volume to direct the patient's movements.

- *Preparatory commands* ready the patient for movement (what to do) and need to be clear and concise. They are optimally accompanied by demonstration and/or guided movement.
- *Action commands* guide the patient through the movement (when and how to move). Strong, dynamic action commands are used when maximal stimulation of movement is the goal; soft action commands are used when relaxation is the goal. Timing is critical to coordinate the patient's actions with the therapist's VCs, resistance, and MCs.
- *Corrective commands* provide augmented feedback to help the patient modify movements.

Indications: Verbal stimulation to enhance strength of muscle contraction and guide the synergistic actions in patterns of movement; verbal corrections provide augmented feedback to enhance motor learning.

Vision: Vision is used to guide the patient's movements, enhance muscle contractions, and synergistic patterns of movement.

Indications: Enhance initial motor control and motor learning.

Stretch (STR): The elongated position/lengthened range and the stretch reflex are used to facilitate muscle contraction. All muscles in the pattern are elongated to optimize the effects of stretch. Commands for voluntary movement are always synchronized with stretch to enhance the response.

- *Repeated stretch* can be applied throughout the range to reinforce contraction in weak muscles that are fading out.

Indications: Enhance strength of muscle contraction and synergistic patterns of movement.

Approximation (AP): Approximation (compressing the joint surfaces) is used to facilitate extensor/stabilizing muscle contraction and stability; can be applied manually, functionally through the use of gravity acting on body during upright positions, or mechanically using weights or weighted vests or belts. Approximation is applied manually during upright, weightbearing positions and in PNF extensor patterns.

Indications: Weakness, inability of extensor muscle to function in weightbearing for stabilization control

Traction (TR): A distraction force (separating the joint surfaces) is used to facilitate muscle contraction and motion, especially in flexion patterns or pulling motions; force is applied manually during PNF. Gentle distraction is also useful in reducing joint pain.

Indications: Weakness, inability of flexor muscles to function in mobilizing or antigravity patterns

II. PNF Techniques

Reversal of Antagonists: A group of techniques that allow for agonist contraction followed by antagonist contraction without pause or relaxation.

- **Dynamic Reversals (Slow Reversals):** Utilizes isotonic contractions of first agonists, then antagonists performed against resistance. Contraction of stronger pattern is selected first with progression to the weaker pattern. The limb is moved through full ROM.

Indications: Impaired strength and coordination between agonist and antagonist, limitations in ROM, fatigue

- **Stabilizing Reversals:** Utilizes alternating isotonic contractions of first agonists, then antagonists against resistance, allowing only very limited ROM.

Indications: Impaired strength, stability and balance, coordination

- **Rhythmic Stabilization (RS):** Utilizes alternating isometric contractions of first agonists, then antagonists against resistance; no motion is allowed.

Indications: Impaired strength and coordination, limitations in ROM; impaired stabilization control and balance

Repeated Contractions, RC (Repeated Stretch): Repeated isotonic contractions from the lengthened range, induced by quick stretches and enhanced by resistance; performed through the range or part of range at a point of weakness. Technique is repeated (i.e., three or four stretches) during one pattern or until contraction weakens.

Indications: Impaired strength, initiation of movement, fatigue, and limitations in active ROM

Combination of Isotonics (Agonist Reversals, AR): Resisted concentric, contraction of agonist muscles moving through the range is followed by a stabilizing contraction (holding in the position) and then eccentric, lengthening contraction, moving slowing back to the start position; there is no relaxation between the types of contractions. Typically used in antigravity activities/assumption of postures (i.e., bridging, sit-to-stand transitions).

Indications: Weak postural muscles, inability to eccentrically control body weight during movement transitions, poor dynamic postural control

Rhythmic Initiation (RI): Voluntary relaxation followed by passive movements progressing to active-assisted and active-resisted movements to finally active movements. Verbal commands are used to set the speed and rhythm of the movements. Light tracking resistance is used during the resistive phase to facilitate movement.

Indications: Inability to relax, hypertonicity (spasticity, rigidity); difficulty initiating movement; motor planning deficits (apraxia or dyspraxia); motor learning deficits; communication deficits (aphasia)

Contract-Relax (CR): A relaxation technique usually performed at a point of limited ROM in the agonist pattern. Strong, small range isotonic contraction of the restricting muscles (antagonists) with emphasis on the rotators is followed by an isometric hold. The contraction is held for 5 to 8 seconds and is then followed by voluntary relaxation and movement into the new range of the agonist pattern. Movement can be passive but active contraction is preferred.

- **Contract-relax-active-contraction (CRAC):** Active contraction into the newly gained range serves to maintain the inhibitory effects through reciprocal inhibition.

Indications: Limitations in ROM

Hold-Relax (HR): A relaxation technique usually performed in a position of comfort and below a level that causes pain. Strong isometric contraction of the restricting muscles (antagonists) is resisted, followed by voluntary relaxation, and passive movement into the newly gained range of the agonist pattern.

- **Hold-Relax-Active Contraction (HRAC):** Similar to HR except movement into the newly gained range of the agonist pattern is active, not passive. Active contraction serves to maintain the inhibitory effects through reciprocal inhibition.

Indications: Limitations in PROM with pain

Replication (Hold-Relax Active Motion, HRA): The patient is positioned in the shortened range/end position of a movement and is asked to hold. The isometric contraction is resisted followed by voluntary relaxation and passive movement into the lengthened range. The patient is then instructed to move back into the end position; stretch and resistance are applied to facilitate the isotonic contraction. For each repetition, increasing ROM is desired.

Indications: Marked weakness; inability to sustain a contraction in the shortened range

Resisted Progression (RP): Stretch, approximation, and tracking resistance is applied manually to facilitate pelvic motion and progression during locomotion; the level of resistance is light so as to not disrupt the patient's momentum, coordination, and velocity. RP can also be applied using elastic band resistance.

Indications: Impaired timing and control of lower trunk/ pelvic segments during locomotion, impaired endurance

Rhythmic Rotation (RRo): Relaxation is achieved with slow, repeated rotation of a limb at a point where limitation is noticed. As muscles relax the limb is slowly and gently moved into the range. As a new tension is felt, RRo is repeated. The patient can use active movements (voluntary effort) for RRo or the therapist can perform RRo passively. Voluntary relaxation when possible is important.

Indications: Relaxation of excess tension in muscles (hypertonia) combined with PROM of the range-limiting muscles

Appendix C: Neuromuscular/Sensory Stimulation Techniques

The term **neuromuscular technique** refers to the facilitation or inhibition of muscle contraction or motor responses. The term **sensory stimulation** refers to the structured presentation of stimuli to improve (1) alertness, attention, and arousal; (2) sensory discrimination; or (3) initiation of muscle activity and improvement of movement control. Effects are immediate and specific to the current state of the nervous system. Additional practice using relevant inputs and feedback is necessary for meaningful and lasting functional change to occur. Variable perceptions exist among individuals; may see decreased sensitivity in some older adults and with some neurological conditions.

I. Proprioceptive Facilitation Techniques

1. Quick Stretch

Stimulus: Brief stretch applied to a muscle.
Activates muscle spindles (facilitates Ia endings); sensitive to velocity and length changes. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.

Response: Stretch reflex: facilitates or enhances agonist muscle contraction; phasic.

Additional peripheral reflex effects: inhibits antagonists, facilitates synergists (*reciprocal innervation effects*). Influences perception of effort.

Techniques: Quick stretch; more effective when applied in the lengthened range (e.g., PNF patterns); tapping over muscle belly or tendon

Comments: A low-threshold response, relatively short-lived; can add resistance to maintain contraction.

Adverse Effects: May increase spasticity when applied to spastic muscles.

2. Prolonged Stretch

Stimulus: Slow, maintained stretch, applied at maximum available lengthened range
Activates muscle spindles (higher threshold response, primarily IIs), Golgi tendon organs (Ib endings); sensitive to length changes; has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.

Response: Inhibits or dampens muscle contraction and tone due largely to peripheral reflex effects (*stretch-protection reflex*).

Techniques: Positioning; inhibitory splinting, casting; mechanical low-load weights using traction

Comments: Higher threshold response; may be more effective in extensor muscles than flexors due to the added effects of II inhibition. To maintain inhibitory effects, follow with activation of antagonist muscles (*reciprocal inhibition effects*).

3. Resistance

Stimulus: A force exerted to muscle
Activates muscle spindles (Ia and II endings) and golgi tendon organs (Ib endings); sensitive to velocity and length changes. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.

Response: Facilitates or enhances muscle contraction due to: (1) peripheral reflex effects: muscle spindle effects via reciprocal innervation (facilitates agonist, inhibits antagonists, facilitates synergists); Golgi tendon effects via autogenic inhibition: dampens or smooths out the force of contraction and (2) suprasegmental effects: recruits both alpha and gamma motoneurons, additional motor units. Hypertrophies extrafusal muscle fibers; enhances kinesthetic awareness.

Techniques: Manual resistance, carefully graded for optimal muscle function.
Use of body weight and gravity using upright positions.
Mechanical resistance: use of weights, cuffs or vests.
Isokinetic resistance: resistance is applied to a muscle contracting at a constant rate.

Comments: *Tracking (light manual) resistance* is used to facilitate and accommodate to very weak muscles. With weak hypotonic muscles, eccentric and isometric contractions are used before concentric (enhances muscle spindle support of contraction with less spindle unloading). Maximal resistance may produce overflow from strong to weak muscles within the same muscle pattern (synergy) or to contralateral extremities.

Adverse Effects: Too much resistance can easily overpower weak, hypotonic muscles and prevent voluntary movement, encouraging substitution. May possibly increase spasticity in spastic muscles.

4. Joint Approximation

Stimulus: Compression of joint surfaces
Activates joint receptors, primarily static, type I receptors. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.

Response: Facilitates postural extensors and stabilizing responses (co-contraction); enhances joint awareness

Techniques: Manual joint compression
Mechanical using weighted harness, vest, or belt
Elastic tubing with compression of joints during movement
Bouncing while sitting on a Swiss ball
Comments: Used in PNF extensor extremity patterns, pushing actions.
Approximation applied to top of shoulders or pelvis in upright weightbearing positions facilities postural extensors and stability (e.g., sitting, kneeling, or standing).
Adverse Effects: Contraindicated with inflamed joints.

5. Joint Traction

Stimulus: Traction of joint surfaces
Activates joint receptors, possibly phasic, type II. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Response: Facilitates joint motion; enhances joint awareness
Techniques: Manual distraction
Mechanical: wrist or ankle cuffs
Comments: Used in PNF flexor extremity patterns, pulling actions.
Joint mobilization uses slow, sustained traction to improve mobility, relieve muscle spasm, and reduce pain.
Adverse Effects: Contraindicated in hypermobile or unstable joints.

6. Inhibitory Pressure

Stimulus: Deep, maintained pressure applied across the longitudinal axis of tendons; prolonged positioning in extreme lengthened range
Activates muscle receptors (Golgi tendon organs) and tactile receptors (pacinian corpuscles). Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Response: Inhibition, dampens muscle tone.
Techniques: Firm, maintained pressure applied manually or with positioning.
Pressure from prolonged weightbearing on knees (e.g., quadruped or kneeling) dampens extensor tone.
Pressure from prolonged weightbearing on extended arm, wrist, and fingers dampens flexor tone (e.g., sitting, modified plantigrade).
Pressure over calcaneus dampens plantarflexor tone.
Tactile pressure over acupressure points relieves pain and dampens muscle tone.

Mechanical: firm objects (cones) in hand, inhibitory splints or casts (e.g., wrist, lower leg).
Comments: Inhibitory effects can be enhanced by combination with other relaxation techniques (e.g., deep breathing techniques, soothing environment)
Adverse Effects: Sustained positioning may dampen muscle contraction enough to affect functional performance (e.g., difficulty walking after prolonged kneeling).

II. Exteroceptive Stimulation Techniques

1. Manual Contacts

Stimulus: Firm, deep pressure of the hands in contact with the body
Activates tactile receptors and muscle proprioceptors (somatosensation).
Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Response: Can facilitate contraction in muscle directly under the hands.
Provide sensory awareness, directional cues to movement.
Provide security and support to unstable body segments.
Comments: Can be used with or without resistance.
Adverse Effects: *Contraindicated* over spastic muscles, and open wounds.

2. Light Touch

Stimulus: A brief, light contact to skin
Activates fast adapting tactile receptors
Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Potential for interaction with autonomic nervous system, sympathetic division
Response: Protection and alerting responses: protective withdrawal (flexion and adduction) of stimulated extremity withdrawing away from the stimulus; can also see contralateral extension in the lower extremity
Increased arousal
Discriminative responses: identification of touch stimuli, spatial discrimination
Techniques: Brief, light stroke of the fingertips
Brief swipe with ice cube
Light pinch or squeezing or pressure to nail bed
Applied to areas of high tactile receptor density (hands, feet, lips) that are more sensitive to stimulation
Comments: Low threshold response, accommodates rapidly.
Effective in initially mobilizing patients with low response levels (e.g., the patient with traumatic brain injury who is minimally responsive)
Can apply tracking resistance to maintain contraction.
Adverse Effects: Overstimulation may produce sympathetic arousal (rebound effects) with undesirable *fight-or-flight responses.*
Contraindicated for patients with generalized arousal or autonomic instability (e.g., the patient with traumatic brain injury who is agitated and combative).
Brief icing should be used with caution on face, forehead, midline back because of risk of adverse sympathetic and arousal effects.

3. Maintained Touch

Stimulus: Deep, maintained touch/pressure
Activates tactile receptors. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.

Potential for interaction with autonomic nervous system, parasympathetics

Response: Calming effect, generalized inhibition, decreased fight/flight responses; desensitizes skin.

Techniques: Firm manual contacts
Firm pressure to midline abdomen, back, lips, palms, and/or soles of feet
Firm rubbing midline back

Comments: Useful for patients with agitation and high arousal (e.g., the patient with traumatic brain injury)
Useful for patients with hypersensitivity (e.g., the patient with peripheral nerve injury and paresthesias or the patient with tactile defensiveness).
Can be used in combination with other maintained stimuli and sensory discrimination training. Brief touch stimuli should be avoided.

4. Slow Stroking

Stimulus: Slow stroking, applied to paravertebral spinal region
Activates tactile receptors. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Potential for interaction with autonomic nervous system, parasympathetics

Response: Calming effect, generalized inhibition, decreased fight-or-flight responses

Techniques: The patient is placed in a supported position such as prone, or sitting head and arms supported and resting forward on a table top.
A flat hand is used to apply firm, alternating strokes in a downward direction over the paravertebral region for approximately 3 to 5 minutes.

Comments: Useful with patients who demonstrate high arousal, increased sympathetic (fight-or-flight) responses.
Can combine with other relaxation techniques (e.g., deep breathing exercises, quiet environment).
Patients with large amounts of body hair may be less responsive to calming effects; hair follicle stimulation may be irritating.

5. Neutral Warmth

Stimulus: Retention of body heat
Activates tactile and thermoreceptors. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Potential for interaction with autonomic nervous system, primarily parasympathetics

Response: Generalized inhibition of tone; warming produces a calming effect, relaxation, and reduction of pain

Techniques: Wrapping body or body parts: ace wraps, towel wraps
Application of snug fitting clothing (gloves, socks, tights)
Air splints
Tepid baths
Duration variable depending on patient response

Comments: Useful for patients with high arousal, or increased sympathetic activity; spasticity.

Adverse Effects: Overheating should be avoided, may produce rebound effects (increased arousal or tone).

6. Prolonged Icing

Stimulus: Cold applications
Activates thermoreceptors. Has both segmental (spinal cord) and suprasegmental (CNS higher centers) effects.
Potential for interaction with autonomic nervous system, sympathetics

Response: Decreases neural and muscle spindle firing.
Provides inhibition of muscle tone and painful muscle spasm.
Decreases metabolic rate of tissues.

Techniques: Immersion in cold water, ice chips,
Ice towel wraps or ice packs
Ice massage
Cooling suit
Duration variable depending on patient response

Comments: Monitor effects carefully.

Adverse Effects: Can produce sympathetic nervous system arousal, protective withdrawal responses, fight-or-flight responses.
Contraindicated in patients with sensory deficits, generalized arousal, autonomic instability, and vascular problems.

III. Vestibular Stimulation Techniques

1. Slow Vestibular Stimulation

Stimulus: Low-intensity, slow and rhythmic vestibular stimulation
Activates primarily otolith organs (tonic receptors); lesser effects on semicircular canals (phasic receptors) with inputs via CN VIII (vestibulocochlear) to CNS higher centers and spinal cord; potential for interaction with autonomic nervous system, primarily parasympathetics

Response: Generalized relaxation: inhibition or dampening of tone and motor output (vestibulospinal reflexes); decreased arousal, fight/flight responses

Techniques: Passive, manually assisted or active motions: slow, repetitive rolling or rocking movements, e.g., sidelying rolling, sitting rocking,
Mechanical: use of a rocking chair or bed, therapy ball or bolster, equilibrium board, hammock, swing; wheelchair ride

Comments: Useful with patients who are hypertonic, hyperactive, or who demonstrate high arousal, or tactile defensiveness (e.g., the patient with traumatic brain injury who is combative)
Can be combined with other relaxation techniques (e.g., deep breathing, cognitive/imagery techniques, quiet environment).

2. Vestibular Stimulation

Stimulus: Vestibular stimulation via head and body movements

Activates semicircular canals (phasic receptors that detect rotational acceleration and deceleration), otolith organs (tonic receptors that detect head position with respect to gravity and linear acceleration) with inputs via CN VIII (vestibulocochlear) to CNS higher centers (vestibular nucleus, spinal cord, reticular formation, superior colliculus, cerebellum).
Vestibulospinal and vestibuloocular reflexes
Potential for interaction with autonomic nervous system

Response: Postural and tonal adjustments
Head and eye movements
Improvement of retinal image stability (vestibuloocular reflex), decreased post-rotatory nystagmus
Improvement of motor coordination
Generalized arousal and consciousness

Techniques: Change of position or movement
Fast spinning and linear movements with acceleration and deceleration components heightens alertness and motor responses (e.g., spinning in a chair, mesh net or hammock; prone on a scooter board).
Equipment: equilibrium boards, wobble boards, therapy ball, dynamic posturography

Comments: Useful with:
Hypotonic patients (e.g., an individual with Down syndrome)
Patients with sensory integrative dysfunction (e.g., a child with hyperactivity)
Patients with coordination problems (e.g., stroke, cerebral palsy)
Helpful in overcoming the effects of akinesia or bradykinesia (e.g., a patient with Parkinson's disease)

Adverse Effects: Prolonged effects may include behavioral changes, seizures, and sleep disturbances.
Contraindicated for patients with recurrent seizures or who are intolerant to sensory stimulation.

IV. Augmented Visual Stimulation Techniques

Stimuli: Visual objects: pen light, brightly colored blocks, familiar objects, photo card
Visual backgrounds: checkerboard background, moving surround screen
Videotapes
Visual biofeedback
Activates photoreceptors (rods and cones) with inputs via CN II (optic) to CNS higher centers (lateral geniculate nucleus, primary visual cortex in the occipital lobe, association areas)

Response: Visual discrimination: conscious awareness and recognition of objects; visual tracking
Alerting, orienting responses: startle response to an unexpected visual stimulus
Visual proprioception: processes information about body in space and spatial relationships.
Contributes to control motor responses: active movements, postural/tonal adjustments.
Can contribute to relaxation response.
Potential for emotional responses (limbic system)

Techniques: Structured application of visual stimuli: presentation of visual objects: vary colors, size, distance, and orientation
Moving visual targets
Computer programs for visual–perceptual training
Environmental: altered lighting:
Soft lights and cool colors for promotion of relaxation (e.g., the patient with traumatic brain injury and confused/agitated response levels)
Bright lights, bright colors, and repetitive even patterns for generalized stimulation of consciousness, attention, and alertness (e.g., a patient with traumatic brain injury with decreased response levels)
Visual biofeedback can be used to aid movement control, strength of muscle contraction, or muscle relaxation.

Comments: Visual scanning activities are important for patients with hemianopsia and unilateral spatial inattention.
Elimination of extraneous visual stimuli and visual distractors using a quiet or closed environment may be necessary to ensure patient attention and visual perception (e.g., for the patient with traumatic brain injury in the confused recovery stages).
Utilize gradual reintroduction of distracting visual stimuli in a variable or open environment as recovery permits.

Adverse Effects: Avoid sensory overload, irritating stimuli that may cause agitation.
Altered or decreased visual perception occurs with busy, open clinic environments; visual distractors or sudden, unexpected visual stimuli disrupt motor performance.

V. Augmented Auditory Stimulation Techniques

Stimuli: Verbal commands (VCs)
Variable sounds: rattle, cluster bells
Metronome
Audiotapes: familiar music or voices
Auditory biofeedback
Activates cochlear receptors via CN VIII (vestibulocochlear) to CNS higher centers (cochlear nucleus, reticular formation, inferior colliculus, and medial geniculate body)

Response: Auditory discrimination: conscious awareness and recognition of sounds, auditory tracking responses
Alerting, orienting responses: startle response to a loud noise
Motor responses: active movement responses, postural/tonal adjustments
Relaxation responses
Emotional responses (limbic system)

Techniques: Structured application of auditory stimuli: presentation of varying auditory sounds.
With VCs: consideration of pitch, tone, and level, volume/intensity is important; adaptation occurs with constant volume.
Relaxing, soft, familiar music aids relaxation and reduction of tone.
Rhythmic auditory stimulation and brisk music aids movement initiation and the development of timing and rhythm of a movement sequence (e.g., marching music for patients with Parkinson's disease).
Music aids socialization; useful in group classes.
Auditory biofeedback can be used to aid movement control, strength of muscle contraction, or muscle relaxation.
Comments: Precise, dynamic VCs are an important element of PNF.
Positive emotional effects occur with VCs that are motivating and encouraging.
Elimination of extraneous noise and auditory distractors using a quiet or closed environment may be necessary to ensure patient attention and auditory perception (e.g., for the patient with traumatic brain injury in the confused recovery stages).
Utilize gradual reintroduction of distracting auditory stimuli in a variable or open environment as recovery permits.
Adverse Effects: Avoid sensory overload, irritating stimuli that may cause agitation.
Negative emotional effects occur with VCs that express anger and frustration.
Altered or decreased auditory perception occurs with busy, open clinic environments; auditory distractors or sudden loud noises disrupt motor performance.

VI. Augmented Olfactory Stimulation Techniques

Stimuli: Varying odors that stimulate the sense of smell
Pleasant odors: vanilla, perfume, favorite foods
Stimulant odors: ammonia, vinegar
Activates nasal olfactory receptors (fast adapting) to CN I (olfactory) to temporal and frontal lobes without synapsing in thalamus, higher centers.
Limbic system: Emotional responses
Response: Relaxation responses with pleasant, familiar odors:
Pleasure, positive mood
Reduction of tone and hyperkinetic movements

Alerting, orienting, arousal responses with noxious odors:
Alertness, arousal (e.g., after fainting)
In the minimally conscious patient (e.g., traumatic brain injury): alertness, arousal, withdrawal responses
Motor responses: active movement responses, postural/ tonal adjustments
Techniques: Structured application of stimuli: presentation of varying scents.
Comments: Consider patient's premorbid interests and likes as well as odors in the external environment and scents you may be wearing.
Adverse Effects: Avoid sensory overload; irritating stimuli that cause agitation.
Adverse fight/flight or emotional responses can occur.
Contraindicated in patients with hypersensitivity.

VII. Gustatory Stimulation Techniques

Stimuli: Taste stimuli
Activates taste receptors on posterior tongue to CN IX (glossopharyngeal); anterior and sides of tongue to CN X (vagus) and CN VII (facial) to higher centers (temporal lobe)
Response: Recognition of tastes; fast adapting
Techniques: Structured application of stimuli: presentation of varying tastes.
Feeding is a multisensory experience including taste, smell, pressure, texture, and temperature.
Comments: Various foods evoke emotional contexts.
Adverse Effects: Dysphagia management requires careful control of food inputs (e.g., taste, texture, size).

VIII. Sensory Integration Training

Stimuli: Multimodal: varied sensory stimuli are presented in the context of meaningful activities (tactile, vestibular-proprioceptive, and visual).
Activates sensory receptors and higher brain centers: engages central processing areas of sensory information.
Response: Improved sensory discrimination: identification of specific stimuli (e.g., shapes, weights, texture, numbers written on skin), intensities, and improved ability to localize stimuli
Improve perception: selection, attention, and response to sensory inputs with appropriate use of information to generate specific motor responses
Techniques: Multimodal, presentation of various different stimuli is combined with functional task training.
Tactile: deep touch-pressure activities (e.g., stroking or rubbing the skin, use of a vibrator, manipulation and identification of objects, drawing letters on skin, learning to read Braille); one-handed and two-handed (bimanual) activities
Vestibular–proprioceptive: activities designed to stimulate a variety of movement experiences (e.g., linear and accelerated movements, resisted movements, gasping and moving objects, throwing objects, therapy ball or wobble board activities) Emphasis is on functional tasks, interlimb control.
Visual: visual cues, tracking tasks
Focus is on postural training activities with progression to more difficult adaptive motor responses.
Comments: Sensory organization training is an important component of balance training: activities focus on

isolating, suppressing, and combining different inputs under varying conditions.

Used for patients with sensory integrative dysfunction who have a poor ability to discriminate touch, movement, force, or information about their bodies. May underlie disorders of bilateral integration, sequencing, and dyspraxia.

Some patients present with decreased discrimination abilities and intense craving for certain types of sensory inputs (e.g., the hyperactive child with frequent outbursts)

Maintain optimal concentration and attention: provide frequent rests and change of task.

Reduce environmental distractors; combine with relaxation techniques.

Adverse Effects: Sensory overload may produce prolonged after-effects (e.g., pupil dilation, changes in respiratory rate, flushing or pallor, nausea, sleep disturbance).

CHAPTER

18

LEARNING OBJECTIVES

1. Describe the epidemiology, etiology, pathophysiology, symptomatology, and sequelae of stroke.
2. Identify and describe the examination procedures used to evaluate patients with stroke to establish a diagnosis, prognosis, and plan of care.
3. Describe the role of the physical therapist in assisting the patient in recovery from stroke in terms of interventions, patient/client-related instruction, coordination, communication, and documentation.
4. Identify and describe strategies of intervention during inpatient rehabilitation.
5. Analyze and interpret patient data, formulate realistic goals and outcomes, and develop a plan of care when presented with a clinical case study.

Stroke

Susan B. O'Sullivan, PT, EdD

OUTLINE

Stroke or **brain attack** is the sudden loss of neurological function caused by an interruption of the blood flow to the brain. **Ischemic stroke** is the most common type, affecting about 80 percent of individuals with stroke, and results when a clot blocks or impairs blood flow, depriving the brain of essential oxygen and nutrients. **Hemorrhagic stroke** occurs when blood vessels rupture, causing leakage of blood in or around the brain. The term **cerebrovascular accident (CVA)** is used interchangeably with stroke to refer to the vascular conditions of the brain. Clinically, a variety of focal deficits are possible, including changes in the level of consciousness and impairments of sensory, motor, cognitive, perceptual, and language functions. To be classified as stroke, neurological deficits must persist for at least 24 hours. Motor deficits are characterized by paralysis **(hemiplegia)** or weakness **(hemiparesis)**, typically on the side of the body opposite the side of the lesion. The term *hemiplegia* is often used generically to refer to the wide

variety of motor problems that result from stroke. The location and extent of brain injury, the amount of collateral blood flow, and early acute care management determine the severity of neurological deficits in an individual patient. Impairments may resolve spontaneously as brain swelling subsides (reversible ischemic neurological deficit), generally within 3 weeks. Residual neurological impairments are those that persist longer than 3 weeks and may lead to permanent disability. Strokes are classified by etiological categories (thrombosis, embolus, or hemorrhage), specific vascular territory (anterior cerebral artery syndrome, middle cerebral artery syndrome, and so forth), and management categories (transient ischemic attack, minor stroke, major stroke, deteriorating stroke, young stroke).

Epidemiology

Stroke is the third leading cause of death and the most common cause of disability among adults in the United States. It affects approximately 700,000 individuals each year; about 500,000 are new strokes and 200,000 are recurrent strokes. There are an estimated 5,400,000 stroke survivors, or 2.6 percent of the population. The incidence of stroke is about 1.25 times greater for males than females. Compared to whites, African-Americans have twice the risk of first-ever stroke; rates are also higher in Mexican-Americans, American Indians, and Alaska Natives.[1] The incidence of stroke increases dramatically with age, doubling in the decade after 65 years of age. For white men 65 to 74 years of age, the incidence is about 14.4 per 1000 population; for ages 75 to 84 it is 24.6 and for 85 and older it is 27.0. Twenty-eight percent of strokes occur in individuals younger than 65 years of age.[2] About 14 percent of persons who survive an initial stroke or TIA will experience another one within 1 year.[1]

About 22 percent of men and 25 percent of women with an initial stroke will die within 1 year, with these rates increasing among people age 65 and older. After 8 years, only about half of patients under the age of 65 are still alive. The type of stroke is significant in determining survival. Of patients with stroke, hemorrhagic stroke account for the largest number of deaths, with mortality rates of 37 to 38 percent at 1 month while ischemic strokes have a mortality rate of only 8 to 12 percent at 1 month.[1] Survival rates are dramatically lessened by increased age, hypertension, heart disease, and diabetes. Loss of consciousness at stroke onset, lesion size, persistent severe hemiplegia, multiple neurological deficits, and history of previous stroke are also important predictors of mortality.[2]

Stroke is the most common cause of chronic disability.[3] Of survivors, an estimated one third will be functionally dependent after 1 year experiencing difficulty with activities of daily living (ADL), ambulation, speech, and so forth.[4] Stroke survivors represent the largest group admitted to inpatient rehabilitation hospitals.[5] Another indicator of disability is the fact that approximately 26 percent of patients with stroke are institutionalized in a nursing home. Direct and indirect costs of stroke in 2005 dollars are estimated at $56.8 billion.[1]

Etiology

Atherosclerosis is a major contributory factor in cerebrovascular disease. It is characterized by plaque formation with an accumulation of lipids, fibrin, complex carbohydrates, and calcium deposits on arterial walls that leads to progressive narrowing of blood vessels. Interruption of blood flow by atherosclerotic plaques occurs at certain sites of predilection. These generally include bifurcations, constrictions, dilation, or angulations of arteries. The most common sites for lesions to occur are at the origin of the common carotid artery or at its transition into the middle cerebral artery, at the main bifurcation of the middle cerebral artery, and at the junction of the vertebral arteries with the basilar artery (Fig. 18.1).

Ischemic strokes are the result of a thrombus, embolism, or conditions that produce low systemic perfusion pressures. The resulting lack of cerebral blood flow (CBF) deprives the brain of needed oxygen and glucose, disrupts cellular metabolism, and leads to injury and death of tissues. A thrombus

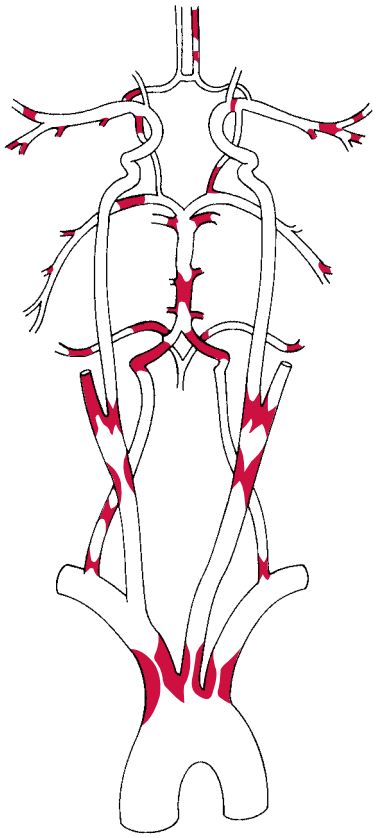

Figure 18.1 Preferred sites for atherosclerotic plaque. (From American Heart Association, Diagnosis and Management of Stroke, 1979, p 4, with permission.)

results from platelet adhesion and aggregation on plaques. **Cerebral thrombosis** refers to the formation or development of a blood clot within the cerebral arteries or their branches. It should be noted that lesions of extracranial vessels (carotid or vertebral arteries) can also produce symptoms of stroke. Thrombi lead to ischemia, or occlusion of an artery with resulting **cerebral infarction** or tissue death (atherothrombotic brain infarction [ABI]). Thrombi can also become dislodged and travel to a more distal site in the form of an intra-artery embolus. **Cerebral embolus (CE)** is composed of bits of matter (blood clot, plaque) formed elsewhere and released into the bloodstream, traveling to the cerebral arteries where they lodge in a vessel, produce occlusion and infarction. The most common source of cerebral embolus is disease of the cardiovascular system. Occasionally systemic disorders may produce septic, fat, or air emboli that affect the cerebral circulation. Ischemic strokes may also result from low systemic perfusion, the result of cardiac failure or significant blood loss with resulting systemic hypotension. The neurological deficits produced with systemic failure are global in nature with bilateral neurological deficits.

Hemorrhagic strokes, with abnormal bleeding into the extravascular areas of the brain are the result of rupture of a cerebral vessel or trauma. Hemorrhage results in increased intracranial pressures with injury to brain tissues and restriction of distal blood flow. **Intracerebral hemorrhage (IH)** is caused by rupture of a cerebral vessel with subsequent bleeding into the brain. Primary **cerebral hemorrhage** (nontraumatic spontaneous hemorrhage) typically occurs in small blood vessels weakened by atherosclerosis producing an **aneurysm. Subarachnoid hemorrhage (SH)** occurs from bleeding into the subarachnoid space typically from a saccular or berry aneurysm affecting primarily large blood vessels. Congenital defects that produce weakness in the blood vessel wall are major contributing factors to the formation of an aneurysm. Hemorrhage is closely linked to chronic hypertension. **Arteriovenous malformation (AVM)** is another congenital defect that can result in stroke. AVM is characterized by a tortuous tangle of arteries and veins with agenesis of an interposing capillary system. The abnormal vessels undergo progressive dilatation with age and eventually bleed in about 50 percent of cases. Sudden and severe cerebral bleeding can result in death within hours, because intracranial pressures rise rapidly and adjacent cortical tissues are compressed or displaced as in brainstem herniation.

Risk Factors and Stroke Prevention

Cardiovascular diseases affecting the brain and heart share a number of common risk factors important to the development of atherosclerosis. Major risk factors for stroke are hypertension, heart disease, and diabetes. In patients with ABI, 70 percent have hypertension, 30 percent coronary heart disease, 15 percent congestive heart disease, 30 percent peripheral arterial disease, and 15 percent diabetes.[2] This coexistence of vascular problems increases significantly with the age of the patient. Stroke risk is increased by four to six times in patients with high blood pressure (elevated above 160/95 mm Hg). Cardiovascular risk is also increased with elevated total blood cholesterol and low-density lipoprotein (LDL) cholesterol and is decreased with higher levels of high-density lipoprotein (HDL) cholesterol.[1] Patients with marked elevations of hematocrits are also at an increased risk of occlusive stroke owing to a generalized reduction of cerebral blood flow. Cardiac disorders such as rheumatic heart valvular disease, endocarditis, or cardiac surgery (e.g., coronary artery bypass graft [CABG]) increase the risk of embolic stroke. Atrial fibrillation is an independent risk factor with five times an increased risk of stroke. Transient ischemic attacks (TIAs) are another important risk factor for stroke. About 10 percent of individuals with TIA will go on to have a major stroke within 90 days; 5 percent will have a major stroke within 2 days.[1,2]

Stroke is largely preventable. Potentially modifiable risk factors include smoking, obesity, lack of exercise, diet, and excess alcohol consumption. Dietary recommendations include control of cholesterol and lipids. Cessation of cigarette smoking significantly decreases risk as does reducing obesity and increasing physical activity. Control of associated diseases, especially diabetes, hypertension, and heart disease, is essential. As with a cardiac risk profile, the more risk factors present or the greater the degree of abnormality of any one factor, the greater the risk of stroke. Stroke risk factors considered nonmodifiable include age (for adults older than 55 the lifetime risk is 1 in 6), gender (the rate for women is slightly higher due to the fact that women live longer), race (African-American), and family history.[1]

Effective stroke prevention also depends on improving public awareness concerning the *early warning signs of stroke.* Only about half of Americans can recognize even one warning sign. Early warning signs identified by The National Stroke Association[6] are listed in Box 18.1.

Box 18.1 Early Warning Signs of Stroke[6]

- Sudden numbness or weakness of the face, arm, or leg, especially on one side of the body
- Sudden confusion, trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden, severe headaches with no known cause

Other important but less common stroke symptoms include:

- Sudden nausea, fever, and vomiting distinguished from a viral illness by the speed of onset (minutes or hours vs several days)
- Brief loss of consciousness or a period of decreased consciousness (fainting, confusion, convulsions, or coma)

The significance of recognizing early warning signs rests with prompt initiation of emergency care under the rule that "*time is brain.*" Patients and families are encouraged to call 911 immediately, even if these symptoms go away quickly or are not painful.[6] Early computed tomography (CT) is used to differentiate between atherothrombotic stroke and hemorrhagic stroke. If the stroke is atherothrombotic, clot-dissolving enzymes (tissue plasminogen activator [t-PA], urokinase, or prourokinase) can be administered. To be effective, thrombolytic therapy such as t-PA must be given within 3 hours of the onset of symptoms and cannot be given with hemorrhagic stroke because the drug may worsen bleeding. Within this window of opportunity, the patient must recognize the situation as a medical emergency, be transported to an appropriate hospital, evaluated by emergency room staff including a CT scan of the brain, and treated. Although this treatment has been available since the mid-1990s and has been shown to be safe and dramatically reduce death and disability,[7,8] current estimates indicate that only 5 percent of individuals experiencing stroke are treated with t-PA.[9] Patients who do receive t-PA are more likely to recover with no disability or minimal disability as compared to those who do not receive the treatment.[10] Major heart and stroke organizations currently promote the use of the term *brain attack* to help individuals recognize the importance of seeking immediate emergency care.

Pathophysiology

Interruption of blood flow for only a few minutes sets in motion a series of pathological events. Complete cerebral circulatory arrest results in irreversible cellular damage with a core area of focal infarction within minutes. The transitional area surrounding the core is termed the *ischemic penumbra* and consists of viable but metabolically lethargic cells. Ischemia triggers a number of damaging and potentially reversible events, termed **ischemic cascade**. The release of excess neurotransmitters (glutamate and aspartate) produces a progressive disturbance of energy metabolism and anoxic depolarization. This results in an inability of brain cells to produce energy, particularly adenosine triphosphate (ATP). This is followed by excess influx of calcium ions and pump failure of the neuronal membrane. Excess calcium reacts with intracellular phospholipids to form free radicals. Calcium influx also stimulates the release of nitric oxide and cytokines. Both mechanisms further damage brain cells. The extension of the infarction into the penumbra area generally takes place over a period of 3 to 4 hours.[11] Research efforts are ongoing toward development of drugs that might restore blood supply and reverse the metabolic changes of the ischemic penumbra area.

Ischemic strokes produce **cerebral edema**, an accumulation of fluids within the brain that begins within minutes of the insult and reaches a maximum by 3 to 4 days. It is the result of tissue necrosis and widespread rupture of cell membranes with movement of water from the blood into brain tissues. The swelling gradually subsides and generally disappears by 2 to 3 weeks. Significant edema can elevate intracranial pressures, leading to intracranial hypertension and neurological deterioration associated with contralateral and caudal shifts of brain structures **(brainstem herniation)**. Clinical signs of elevating intracranial pressures (ICP) include decreasing level of consciousness (stupor and coma), widened pulse pressure, increased heart rate, irregular respirations (Cheyne-Stokes respirations), vomiting, unreacting pupils (cranial nerve [CN] III signs), and papilledema. Cerebral edema is the most frequent cause of death in acute stroke and is characteristic of large infarcts involving the middle cerebral artery and the internal carotid artery.[12]

Management Categories

Transient ischemic attack (TIA) refers to the temporary interruption of blood supply to the brain. Symptoms of focal neurological deficit may last for only a few minutes or for several hours, but do not last longer than 24 hours. After the attack is over there is no evidence of residual brain damage or permanent neurological dysfunction. TIAs may result from a number of different etiological factors including occlusive episodes, emboli, reduced cerebral perfusion (arrhythmias, decreased cardiac output, hypotension, overmedication with antihypertensive medications, **subclavian steal syndrome**) or cerebrovascular spasm. The major clinical significance of TIA is as a precursor to susceptibility for both cerebral infarction and myocardial infarction. Patients are classified as having a *major stroke* in the presence of stable, usually severe, impairments. The term *deteriorating stroke* is used to refer to the patient whose neurological status is deteriorating after admission to the hospital. This change in status may be due to cerebral or systemic causes (e.g., cerebral edema, progressing thrombosis). The category of *young stroke* is used to describe a stroke affecting persons younger than the age of 45. Younger individuals may have potential for better recovery.

Vascular Syndromes

Cerebral blood flow (CBF) varies with the patency of the vessels. Progressive narrowing secondary to atherosclerosis decreases blood flow. As in coronary heart disease, symptomatic changes generally result from a restriction of flow greater than 80 percent. The severity and symptoms of stroke are dependent on a number of factors, including (1) the location of the ischemic process, (2) the size of the ischemic area, (3) the nature and functions of the structures involved, and (4) the availability of collateral blood flow.

Presenting symptoms may also depend on the rapidity of the occlusion of a blood vessel because slow occlusions may allow collateral vessels to take over, whereas sudden events do not.

Cerebral Blood Flow

CBF is controlled by a number of *autoregulatory mechanisms* (cerebral) that modulate a constant rate of blood flow through the brain. These mechanisms provide homeostatic balance, counteracting fluctuations in systolic blood pressure while maintaining a normal flow of 50 to 60 ml/100 g of brain tissue per minute. The brain has high energy requirements and very little metabolic reserves. Thus it requires a continuous, rich perfusion of blood to deliver oxygen and glucose to the tissues. Cerebral flow represents approximately 17 percent of available cardiac output. Chemical regulation of CBF occurs in response to changes in blood concentrations of carbon dioxide or oxygen. Vasodilation and increased CBF are produced in response to an increase in $Paco_2$ or a decrease in Pao_2, while vasoconstriction and decreased CBF are produced by the opposite stimuli. Blood flow is also altered by changes in the blood pH. A fall in pH (increased acidity) produces vasodilation, and a rise in pH (increased alkalinity) produces a decrease in blood flow. Neurogenic regulation alters blood flow by vasodilating vessels in direct proportion to local function of brain tissue. Released metabolites probably act directly on the smooth muscle in local vessel walls. Changes in blood viscosity or intracranial pressures may also influence CBF. Changes in blood pressure produce minor alterations of CBF. As pressure rises, the artery is stretched, resulting in contraction of smooth muscle in the vessel wall. Thus, the patency of the vessel is decreased, with a consequent decrease in CBF. As pressure falls, contraction lessens and CBF increases. Following stroke, autoregulatory mechanisms may be impaired.[13]

Knowledge of cerebral vascular anatomy is essential to understand the symptoms, diagnosis, and management of stroke. Extracranial blood supply to the brain is provided by right and left internal carotid arteries and by the right and left vertebral arteries. The internal carotid artery begins at the bifurcation of the common carotid artery and ascends in the deep portions of the neck to the carotid canal. It turns rostromedially and ascends into the cranial cavity. It then pierces the dura mater and gives off the ophthalmic and anterior choroidal arteries before bifurcating into the middle and anterior cerebral arteries. The anterior communicating artery communicates with the anterior cerebral arteries of either side, giving rise to the rostral portion of the **circle of Willis** (Fig. 18.2). The vertebral artery arises as a branch off the subclavian artery. It enters the vertebral foramen of the sixth cervical vertebra and travels through the foramina of the transverse processes of the upper six cervical vertebrae to the

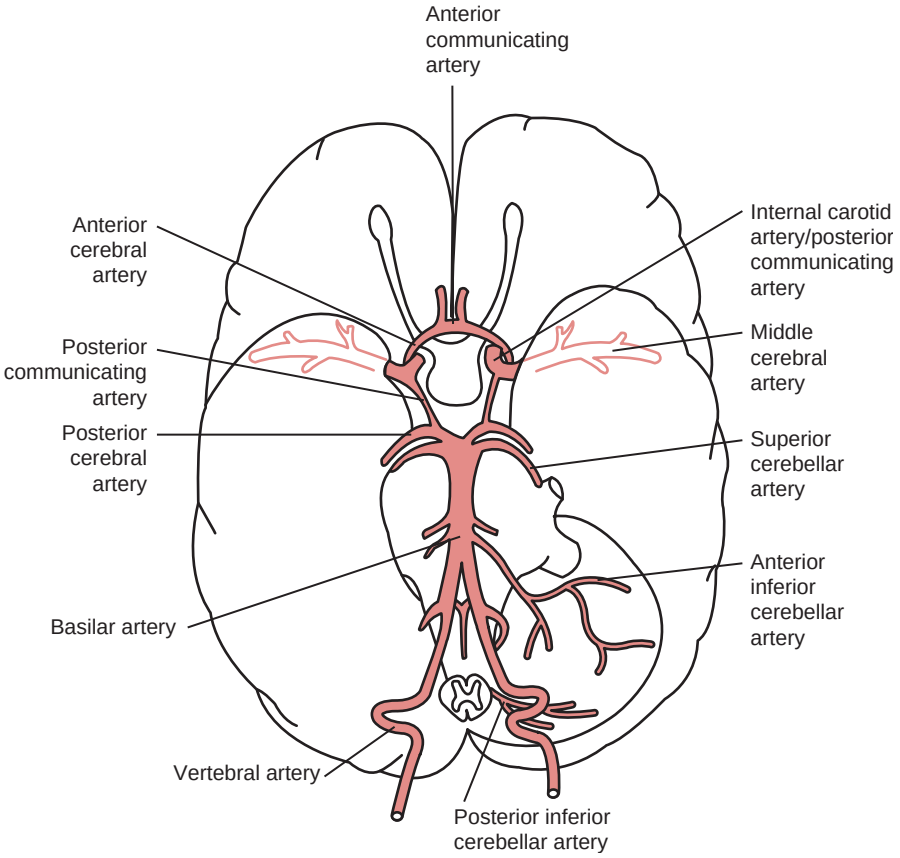

Figure 18.2 Cerebral circulation: Circle of Willis.

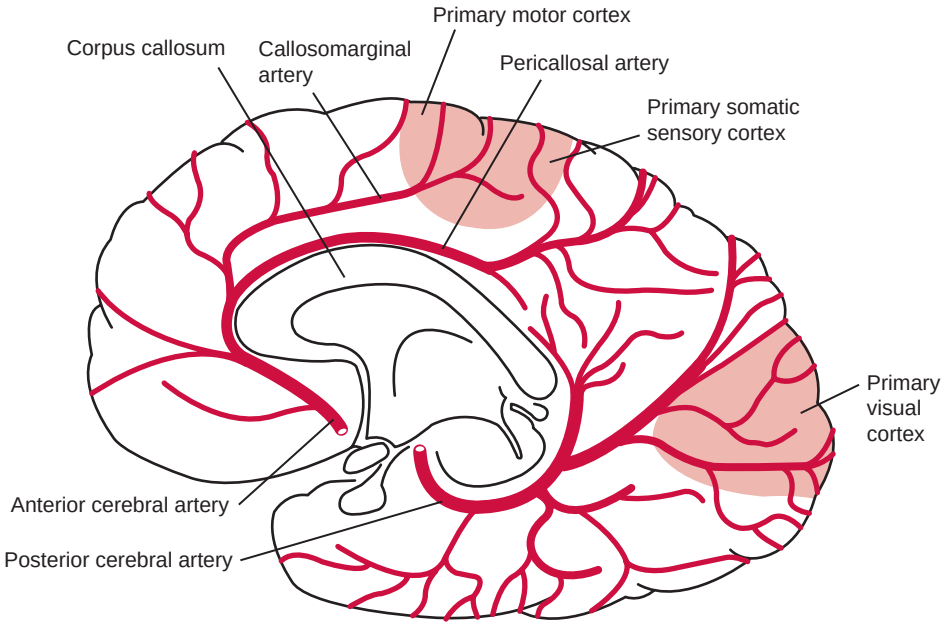

Figure 18.3 Cerebral circulation: A diagram of a midsagittal view of the brain illustrates the distribution of the anterior and posterior cerebral arteries.

foramen magnum and into the brain. There it travels in the posterior cranial fossa ventrally and medially and unites with the vertebral artery from the other side to form the basilar artery at the upper border of the medulla. At the upper border of the pons, the basilar artery bifurcates to form the posterior cerebral arteries and the posterior portion of the circle of Willis. Posterior communicating arteries connect the posterior cerebral arteries with the internal carotid arteries and complete the circle of Willis.

Anterior Cerebral Artery Syndrome

The anterior cerebral artery (ACA) is the first and smaller of two terminal branches of the internal carotid artery. It supplies the medial aspect of the cerebral hemisphere (frontal and parietal lobes) and subcortical structures, including the basal ganglia (anterior internal capsule, inferior caudate nucleus), anterior fornix, and anterior four fifths of the corpus callosum (Fig. 18.3). Because the anterior communicating artery allows perfusion of the proximal anterior cerebral artery from either side, occlusion proximal to this point results in minimal deficit.

More distal lesions produce more significant deficits. Table 18.1 presents the clinical manifestations of *anterior cerebral artery (ACA) syndrome.* The most common characteristic of ACA syndrome is contralateral hemiparesis and sensory loss with greater involvement of the lower extremity because the somatotopic organization of the medial

Table 18.1 Clinical Manifestations of Anterior Cerebral Artery Syndrome

Signs and Symptoms	Structures Involved
Contralateral hemiparesis involving mainly the LE (UE is more spared)	Primary motor area, medial aspect of cortex, internal capsule
Contralateral hemisensory loss involving mainly the LE (UE is more spared)	Primary sensory area, medial aspect of cortex
Urinary incontinence	Posteromedial aspect of superior frontal gyrus
Problems with imitation and bimanual tasks, apraxia	Corpus callosum
Abulia (akinetic mutism), slowness, delay, lack of spontaneity, motor inaction	Uncertain localization
Contralateral grasp reflex, sucking reflex	Uncertain localization

LE = lower extremity; UE = upper extremity.

Adapted from Fredericks, C, and Saladin, L: Pathophysiology of the Motor Systems. FA Davis, Philadelphia, 1996, p 504, with permission.

aspect of the cortex includes the functional area for the lower extremity.

Middle Cerebral Artery Syndrome

The middle cerebral artery (MCA) is the second of the two main branches of the internal carotid artery and supplies the entire lateral aspect of the cerebral hemisphere (frontal, temporal, and parietal lobes) and subcortical structures, including the internal capsule (posterior portion), corona radiata, globus pallidus (outer part), most of the caudate nucleus, and the putamen (Fig. 18.4). Occlusion of the proximal MCA produces extensive neurological damage with significant cerebral edema. Increased intracranial pressures typically lead to loss of consciousness, brain herniation, and possibly death. Table 18.2 presents the clinical manifestations of *middle cerebral artery (MCA) syndrome.* The most common characteristics of MCA syndrome are contralateral spastic hemiparesis and sensory loss of the face, upper extremity (UE), and lower extremity (LE), with the face and UE more involved than the LE. Lesions of the parieto-occipital cortex of the dominant hemisphere (usually the left hemisphere) typically produce aphasia. Lesions of the right parietal lobe of the nondominant hemisphere (usually the right hemisphere) typically produce perceptual deficits (e.g., **unilateral neglect**, **anosognosia, apraxia**, and **spatial disorganization**). **Homonymous hemianopsia** (a visual field defect) is also a common finding. The MCA is the most common site of occlusion in stroke.

Internal Carotid Artery Syndrome

Occlusion of the internal carotid artery (ICA) typically produces massive infarction in the region of the brain supplied by the middle cerebral artery. The ICA supplies both the MCA and the ACA. If collateral circulation to the ACA from the circle of Willis is absent, extensive cerebral infarction in the areas of both the ACA and MCA can occur. Significant edema is common with possible uncal herniation, coma, and death (mass effect).

Posterior Cerebral Artery Syndrome

The two posterior cerebral arteries (PCAs) arise as terminal branches of the basilar artery and each supplies the corresponding occipital lobe and medial and inferior temporal lobe (see Fig. 18.3). It also supplies the upper brainstem, midbrain, and posterior diencephalon, including most of the thalamus. Table 18.3 presents the clinical manifestations of *posterior cerebral artery (PCA) syndrome*. Occlusion proximal to the posterior communicating artery typically results in minimal deficits owing to the collateral blood supply from the posterior communicating artery (similar to ACA syndrome). Occlusion of thalamic branches may produce hemianesthesia (contralateral sensory loss) or **central post-stroke (thalamic) pain**. Occipital infarction produces homonymous hemianopsia, **visual agnosia**, **prosopagnosia**, or, if bilateral, cortical blindness. Temporal lobe ischemia results in amnesia (memory loss). Involvement of subthalamic branches may involve

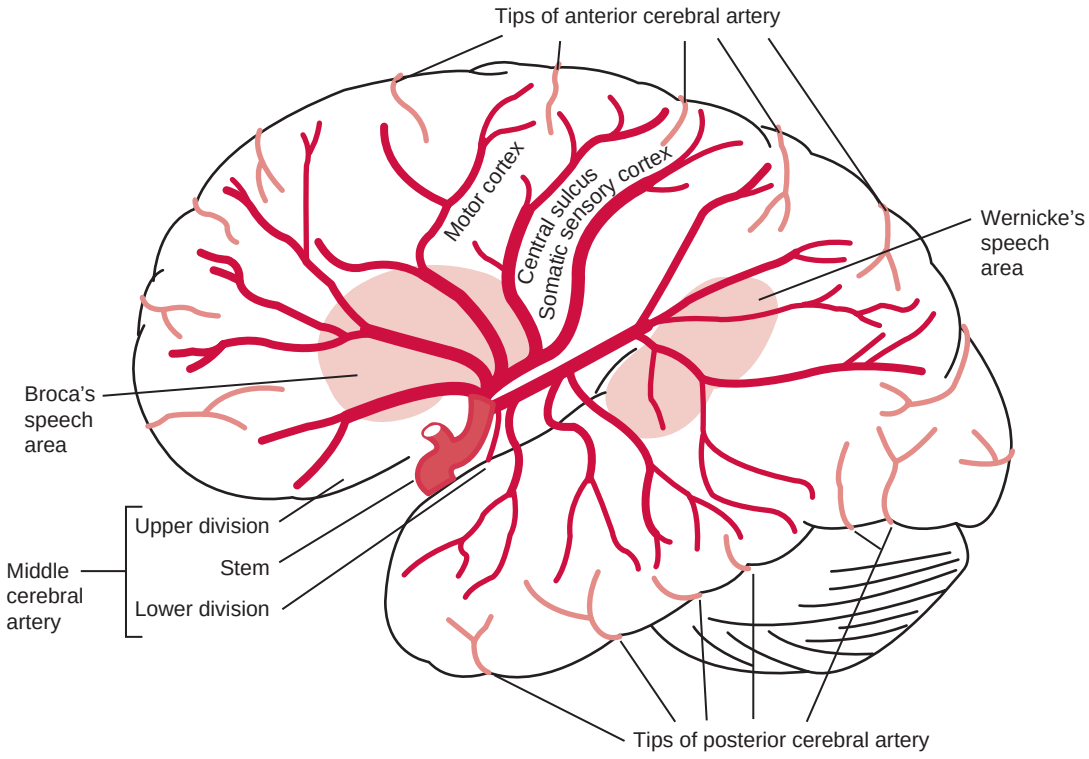

Figure 18.4 Cerebral circulation: Diagram of a lateral view of the brain illustrates the distribution of the middle cerebral artery.

Table 18.2 Clinical Manifestations of Middle Cerebral Artery Syndrome

Signs and Symptoms	Structures Involved
Contralateral hemiparesis involving mainly the UE and face (LE is more spared)	Primary motor cortex and internal capsule
Contralateral hemisensory loss involving mainly the UE and face (LE is more spared)	Primary sensory cortex and internal capsule
Motor speech impairment: Broca's or nonfluent aphasia with limited vocabulary and slow, hesitant speech	Broca's cortical area (third frontal convolution) in the dominant hemisphere, typically the left hemisphere
Receptive speech impairment: Wernicke's or fluent aphasia with impaired auditory comprehension and fluent speech with normal rate and melody	Wernicke's cortical area (posterior portion of the temporal gyrus) in the dominant hemisphere, typically the left
Global aphasia: nonfluent speech with poor comprehension	Both third frontal convolution and posterior portion of the superior temporal gyrus
Perceptual deficits: unilateral neglect, depth perception, spatial relations, agnosia	Parietal sensory association cortex in the nondominant hemisphere, typically the right
Limb-kinetic apraxia	Premotor or parietal cortex
Contralateral homonymous hemianopsia	Optic radiation in internal capsule
Loss of conjugate gaze to the opposite side	Frontal eye fields or their descending tracts
Ataxia of contralateral limb(s) (sensory ataxia)	Parietal lobe
Pure motor hemiplegia (lacunar stroke)	Upper portion of posterior limb of internal capsule

LE = lower extremity; UE = upper extremity.

Adapted from Fredericks, C, and Saladin, L: Pathophysiology of the Motor Systems. FA Davis, Philadelphia, 1996, p 502, with permission.

subthalamic nucleus or its pallidal connections, producing a wide variety of deficits. Contralateral hemiplegia occurs with involvement of the cerebral peduncle.

Lacunar Syndromes

Lacunar syndromes are caused by small vessel disease deep in the cerebral white mater (penetrating artery disease). They are strongly associated with hypertensive hemorrhage and diabetic microvascular disease. Lacunar syndromes are consistent with specific anatomic sites. *Pure motor lacunar stroke* is associated with involvement of the posterior limb of the internal capsule, pons, and pyramids. *Pure sensory lacunar stroke* is associated with involvement of the ventrolateral thalamus or thalamocortical projections. Other lacunar syndromes include *dysarthria/clumsy hand syndrome* (involving the base of the pons, genu of anterior limb or the internal capsule), *ataxic hemiparesis* (involving the pons, genu of internal capsule, corona radiata, or cerebellum), *sensory/motor stroke* (involving the junction of the internal capsule and thalamus), or *dystonia/involuntary movements* (choreoathetosis with lacunar infarction of the putamen or globus pallidus; hemiballismus with involvement of the subthalamic nucleus). Deficits in consciousness, language, or visual fields are not seen in lacunar strokes as the higher cortical areas are preserved. A hypertensive hemorrhage affecting the thalamus can also produce central post-stroke pain.[11–16]

Vertebrobasilar Artery Syndrome

The vertebral arteries arise from the subclavian arteries and travel into the brain along the medulla where they merge at the inferior border of the pons to form the basilar artery. The vertebral arteries supply the cerebellum (via posterior inferior cerebellar arteries) and the medulla (via the medullary arteries). The basilar artery supplies the pons (via pontine arteries), the internal ear (via labyrinthine arteries), and the cerebellum (via the anterior inferior and superior cerebellar arteries). The basilar artery then terminates at the upper border of the pons giving rise to the two posterior arteries (see Fig. 18.2). Occlusions of the vertebrobasilar system can produce a wide variety of symptoms with both ipsilateral and contralateral signs, because some of the tracts in the brainstem will have crossed and others will not. Numerous cerebellar and cranial nerve abnormalities also are present. Table 18.4 presents the clinical manifestations of *vertebrobasilar artery syndromes*.

Locked-in syndrome (LIS) occurs with basilar artery thrombosis and bilateral infarction of the ventral pons. LIS is a catastrophic event with sudden onset. Patients develop

Table 18.3 Clinical Manifestations of Posterior Cerebral Artery Syndrome

Signs and Symptoms	Structures Involved
Peripheral territory	
Contralateral homonymous hemianopsia	Primary visual cortex or optic radiation
Bilateral homonymous hemianopsia with some degree of macular sparing	Calcarine cortex (macular sparing is due to occipital pole receiving collateral blood supply from MCA)
Visual agnosia	Left occipital lobe
Prosopagnosia (difficulty naming people on sight)	Visual association cortex
Dyslexia (difficulty reading) without agraphia (difficulty writing), color naming (anomia), and color discrimination problems	Dominant calcarine lesion and posterior part of corpus callosum
Memory defect	Lesion of inferomedial portions of temporal lobe bilaterally or on the dominant side only
Topographic disorientation	Nondominant primary visual area, usually bilaterally
Central territory	
Central post-stroke (thalamic) pain Spontaneous pain and dysesthesias; sensory impairments (all modalities)	Ventral posterolateral nucleus of thalamus
Involuntary movements; choreoathetosis, intention tremor, hemiballismus	Subthalamic nucleus or its pallidal connections
Contralateral hemiplegia	Cerebral peduncle of midbrain
Weber's syndrome Oculomotor nerve palsy and contralateral hemiplegia	Third nerve and cerebral peduncle of midbrain
Paresis of vertical eye movements, slight miosis and ptosis, and sluggish pupillary light response	Supranuclear fibers to third cranial nerve

LE = lower extremity; UE = upper extremity.

Adapted from Fredericks, C, and Saladin, L: Pathophysiology of the Motor Systems. FA Davis, Philadelphia, 1996, p 509, with permission.

acute hemiparesis rapidly progressing to tetraplegia and lower bulbar paralysis (CN V through XII are involved). Initially the patient is dysarthric and dysphonic but rapidly progresses to mutism (anarthria). There is preserved consciousness and sensation. Thus the patient cannot move or speak but remains alert and oriented. Horizontal eye movements are impaired but vertical eye movements and blinking remain intact. Communication can be established via these eye movements. Mortality rates are high (59 percent), and those patients that do survive are left with severe impairments associated with brainstem injury.[14,15]

Extracranial injuries to the vertebral arteries as they travel through the cervical spine can also produce vertebrobasilar signs and symptoms. Forceful neck motions (e.g., whiplash or aggressive neck manipulations) are among the more common types of injuries.

Medical Diagnosis of Stroke

History and Examination

An accurate history profiling the timing of neurological events is obtained from the patient or from family members in the case of the unconscious or noncommunicative patient. Of particular importance are the pattern of onset and the course of initial neurological symptoms. An abrupt onset with rapid coma is suggestive of cerebral hemorrhage. Severe headache typically precedes loss of consciousness. An embolus also occurs rapidly, with no warning, and is frequently associated with heart disease and/or heart complications. A more variable and uneven onset is typical with thrombosis. The patient's past history including episodes of TIAs or head trauma, presence of major or

(text continues on page 716)

Table 18.4 Clinical Manifestations of Vertebrobasilar Artery Syndromes

Signs and Symptoms	Structures Involved
Medial medullary syndrome	Occlusion vertebral artery, medullary branch
Ipsilateral to lesion	
Paralysis with atrophy of half the tongue with deviation to the paralyzed side when tongue is protruded	CN XII, hypoglossal, or nucleus
Contralateral to lesion	
Paralysis of UE and LE	Corticospinal tract
Impaired tactile and proprioceptive sense	Medial lemniscus
Lateral medullary (Wallenburg's) syndrome	Occlusion of posterior inferior cerebellar artery or vertebral artery
Ipsilateral to lesion	
Decreased pain and temperature sensation in face	Descending tract and nucleus of CN V, Trigeminal
Cerebellar ataxia: gait and limbs ataxia	Cerebellum or inferior cerebellar peduncle
Vertigo, nausea, vomiting	Vestibular nuclei and connections
Nystagmus	Vestibular nuclei and connections
Horner's syndrome: miosis, ptosis, decreased sweating	Descending sympathetic tract
Dysphagia and dysphonia: paralysis of palatal and laryngeal muscles, diminished gag reflex	CN IX, glossopharyngeal, and CN X, vagus, or nuclei
Sensory impairment of ipsilateral UE, trunk, or LE	Cuneate and gracile nuclei
Contralateral to lesion	
Impaired pain and thermal sense over 50% of body, sometimes face	Spinal lemniscus—spinothalamic tract
Complete basilar artery syndrome (locked-in syndrome)	Basilar artery, ventral pons
Tetraplegia (Quadriplegia)	Corticospinal tracts bilaterally
Bilateral cranial nerve palsy: upward gaze is spared	Long tracts to cranial nerve nuclei bilaterally
Coma	Reticular activating system
Cognition is spared	
Medial inferior pontine syndrome	Occlusion of paramedian branch of basilar artery
Ipsilateral to lesion	
Paralysis of conjugate gaze to side of lesion (preservation of convergence)	Pontine center for lateral gaze paramedian pentine reticular formation (PPRF)
Nystagmus	Vestibular nuclei and connections
Ataxia of limbs and gait	Middle cerebellar peduncle
Diplopia on lateral gaze	CN VI, abducens, or nucleus
Contralateral to lesion	
Paresis of face, UE, and LE	Corticobulbar and corticospinal tract in lower pons
Impaired tactile and proprioceptive sense over 50% of the body	Medial lemniscus

Table 18.4 Clinical Manifestations of Vertebrobasilar Artery Syndromes (continued)

Signs and Symptoms	Structures Involved
Lateral inferior pontine syndrome	Occlusion of anterior inferior cerebellar artery, a branch of the basilar artery
Ipsilateral to lesion	
Horizontal and vertical nystagmus, vertigo, nausea, vomiting	CN VIII, vestibular, or nucleus
Facial paralysis	CN VII, facial, or nucleus
Paralysis of conjugate gaze to side of lesion	Pontine center for lateral gaze (PPRF)
Deafness, tinnitus	CN VIII, cochlear, or nucleus
Ataxia	Middle cerebellar peduncle and cerebellar hemisphere
Impaired sensation over face	Main sensory nucleus and descending tract of fifth nerve
Contralateral to lesion	
Impaired pain and thermal sense over half the body (may include face)	Spinothalamic tract
Medial midpontine syndrome	Occlusion of paramedian branch of the mid-basilar artery
Ipsilateral to lesion	
Ataxia of limbs and gait (more prominent in bilateral involvement)	Middle cerebellar peduncle
Contralateral to lesion	
Paralysis of face, UE, and LE	Corticobulbar and corticospinal tract
Deviation of eyes	PPRF
Lateral midpontine syndrome	Occlusion of short circumferential artery
Ipsilateral to lesion	
Ataxia of limbs	Middle cerebellar peduncle
Paralysis of muscles of mastication	Motor fibers or nucleus of CN V, trigeminal
Impaired sensation over side of face	Sensory fibers or nucleus of CN V, trigeminal
Medial superior pontine syndrome	Occlusion of paramedian branches of upper basilar artery
Cerebellar ataxia	Superior or middle cerebellar peduncle
Internuclear ophthalmoplegia	Medial longitudinal fasciculus
Contralateral to lesion	
Paralysis of face, UE, and LE	Corticobulbar and corticospinal tract

(*continued*)

Table 18.4 Clinical Manifestations of Vertebrobasilar Artery Syndromes (continued)

Signs and Symptoms	Structures Involved
Lateral superior pontine syndrome (occlusion of superior cerebellar artery, a branch of the basilar artery)	
Ipsilateral to lesion	
Cerebellar ataxia of limbs and gait, falling to side of lesion	Middle and superior cerebellar peduncles, superior surface of cerebellum, dentate nucleus
Dizziness, nausea, vomiting	Vestibular nuclei
Horizontal nystagmus	Vestibular nuclei
Paresis of conjugate gaze (ipsilateral)	Uncertain
Loss of optokinetic nystagmus	Uncertain
Horner's syndrome: miosis, ptosis, decreased sweating on opposite side face	Descending sympathetic fibers
Contralateral to lesion	
Impaired pain and thermal sense of face, limbs, and trunk	Spinothalamic tract
Impaired touch, vibration, and position sense, more in LE than UE (tendency to incongruity of pain and touch deficits)	Medial lemniscus (lateral portion)

CN = cranial nerve; LE = lower extremity; UE = upper extremity.

Adapted from Fredericks, C, and Saladin, L: Pathophysiology of the Motor Systems. FA Davis, Philadelphia, 1996, p 505, with permission.

minor risk factors, medications, pertinent family history, and any recent alterations in patient function (either transient or permanent) are thoroughly investigated.

The physical examination of the patient includes a general medical examination as well as a neurological examination. An investigation of vital signs (heart rate, respiratory rate, blood pressure) and signs of cardiac decompensation is essential. The neurological examination stresses function of the cerebral hemispheres, cerebellum, cranial nerves, eyes, and sensorimotor system. The presenting symptoms will help to determine the location of the lesion, and comparison of both sides of the body will reveal the side of the lesion. Bilateral signs are suggestive of brainstem lesions or massive cerebral involvement.

Neurovascular tests are performed. These include:

- *Neck flexion*. Meningeal irritation secondary to subarachnoid hemorrhage will produce resistance or pain with neck flexion.
- *Palpation of arteries*. Both superficial and deep arteries are palpated including temporal, facial, carotid, subclavian, brachial, radial, abdominal aorta, and lower extremity (LE) arteries.
- *Auscultation of heart and blood vessels*. Abnormal heart sounds, murmurs, or bruits may be present and indicate increased flow turbulence and stenosis in a vessel.
- *Ophthalmic pressures*. Abnormal pressures in the ophthalmic artery may indicate problems in the internal carotid artery.

Tests and Measures

A number of routine laboratory and diagnostic tests are performed.[11] These include:

- *Urinalysis*: Detects infection, diabetes, renal failure, or dehydration.
- *Blood analysis*: Provides a complete blood count (CBC), platelet count, prothrombin time, partial thromboplastin time, and erythrocyte sedimentation rate (ESR).
- *Fasting blood glucose level*
- *Blood chemistry profile*: Indicates serum electrolytes and serum cardiac enzyme levels. Electrolyte abnormalities may contribute to extension of infarct to the penumbra area. Elevation of the creatinine phosphokinase isoenzyme (CPK-MB) is indicative of coincidental cardiac infarction.
- *Blood cholesterol and lipid profile*.
- *Thyroid function tests*: Accelerated atherosclerosis can result from hypothyroidism.
- *Full cardiac evaluation*: Includes radiograph of the chest (heart size, lungs); electrocardiograph (ECG) to detect arrhythmias as a source of emboli or coincidental heart

disease. Stroke may also cause ECG abnormalities, typically T-wave inversion, prolonged QT interval, and ST inversion.

- *Echocardiography*: May reveal valvular disease (a source of emboli) or other heart conditions such as congestive heart failure (CHF), recent myocardial infarction (MI).
- *Lumbar puncture*: May be used to diagnosis subarachnoid hemorrhage in the presence of focal neurological deficit and nuchal rigidity.

Imaging

Modern cerebrovascular imaging techniques have vastly improved the accurate diagnosis of stroke. These include:

Computerized Tomography (CT). CT scan is the most commonly used imaging technique. An intravenous iodinated contrast dye may be used to enhance the density of intravascular blood. CT resolution only allows identification of large arteries and veins, and venous sinuses. In the acute phase, the CT scans are used to rule out other brain lesions such as tumor or abscess and to identify hemorrhagic stroke. In the case of suspected stroke, an emergency CT is used to rule out hemorrhage if anticoagulants or clot-busting drugs are to be administered. Many times CT scans during the acute phase are negative with no clear abnormalities. In the subacute phase, CT scans can delineate the development of cerebral edema (within 3 days) and cerebral infarction (within 3 to 5 days) by showing areas of decreased density. It is important to remember that the extent of CT lesion does not necessarily correlate with clinical signs or changes in function (Fig. 18.5).

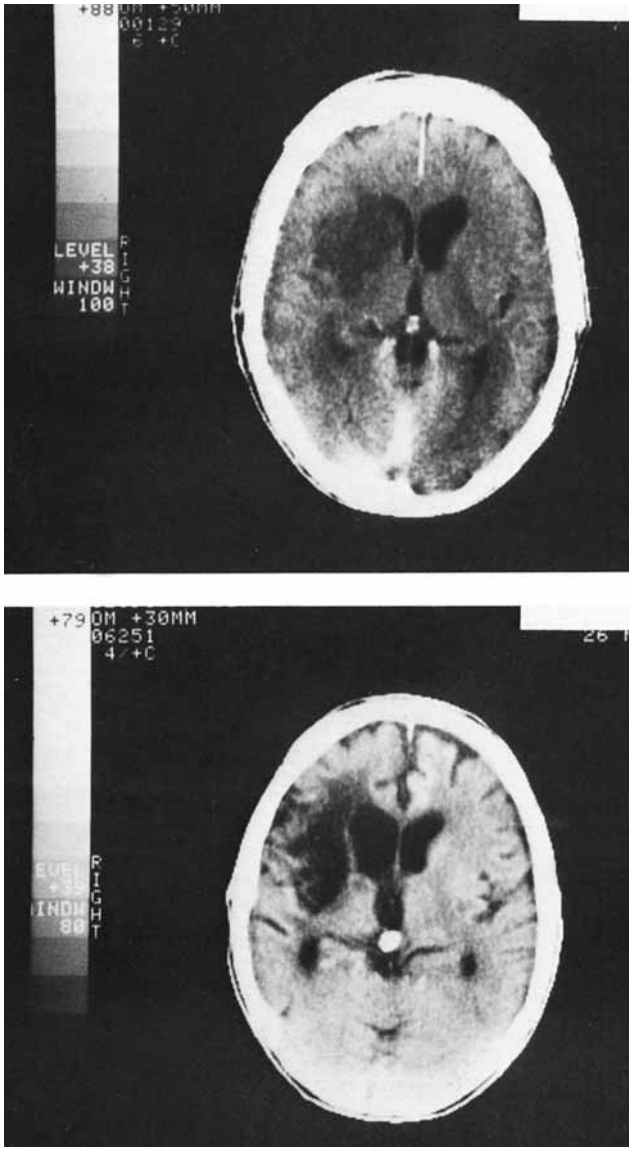

Figure 18.5 Computed tomography scan of a patient with infarction of the middle cerebral artery territory. (From Hackinski, W, and Norris, JW: The Acute Stroke. FA Davis, Philadelphia, 1985, p 194, with permission.)

Magnetic Resonance Imaging (MRI). MRI measures nuclear particles as they interact with a powerful magnetic field. Greater resolution of the brain and its structural detail is obtained with MRI than with a CT scan. Magnetic resonance imaging is more sensitive in the diagnosis of acute strokes, allowing detection of cerebral infarction within 2 to 6 hours after stroke. It is also able to detail the extent of infarction or hemorrhage and can detect smaller lesions than a CT scan. Contrast enhancement (e.g., gadolinium) can be used to document changes in an infarct over the first 2 to 3 weeks.

Positron Emission Tomography (PET). Positron emission from an injected radionuclide is measured. The use of PET allows imaging of regional blood flow and localized cerebral metabolism. PET scanning can be used in the subacute stage to distinguish infracted areas and to identify areas of tissue where ischemia is reversible. The high cost and limited availability of PET scans limit the use in the routine evaluation of stroke.

Transcranial and Carotid Doppler. This technique is used for noninvasive imaging of the neck and chest vessels (carotid, vertebral, and subclavian arteries).

Cerebral Angiography. Cerebral angiography is invasive and involves the injection of radiopaque dye into blood vessels with subsequent radiography. It provides visualization of the vascular system and is often used when surgery is considered (carotid stenosis, arteriovenous malformations). There is associated morbidity and mortality with angiography. CT or MRI scans are more commonly used.

Medical Management

Medical management of completed stroke includes strategies to:

- Improve cerebral perfusion by reestablishing circulation and oxygenation. Oxygen is delivered via mask or nasal cannula. Patients in a coma may require intubation or assisted ventilation and suctioning.
- Maintain adequate blood pressure. Hypotension or extreme hypertension is treated; antihypertension agents have the added risk of inducing hypotension and decreasing cerebral perfusion.
- Maintain sufficient cardiac output. If the causes of stroke are cardiac in origin, medical management focuses on control of arrhythmias and cardiac decompensation.
- Restore/maintain fluid and electrolyte balance.
- Maintain blood glucose levels within the normal range.
- Control seizures and infections.
- Control intracranial pressures and herniation using antiedema agents. Ventriculostomy may be indicated to monitor and drain cerebrospinal fluid.
- Maintain bladder function, which may include urinary catheter. Catheterization is typically short-term but may be long-term with the patient in coma.
- Maintain integrity of skin and joints by instituting protective positioning; a turning schedule every 2 hours; and early physical and occupational therapy.

Pharmacological interventions for completed stroke can include[11]:

- Anticoagulant therapy (heparin, Coumadin): used to improve perfusion and reduce the risk of recurring clots (embolism and thrombosis); clotting times are monitored as there is an increased risk of bleeding.
- Antiplatelet therapy (aspirin): Long-term, low-dose is used to decrease the risk of recurrent stroke; higher doses may be used in place of anticoagulants and may be recommended for patients with atrial fibrillation. Ticlopidine or clopidogrel bisulfate are alternate agents for recurrent stroke prevention.
- Antihypertensive agents.

Neurosurgical intervention may include:

- Endarterectomy which is the surgical removal of the lining and plaque of an artery. Internal carotid artery stenosis affects approximately 25 percent of elderly persons. Endarterectomy is used to prevent strokes but not to treat acute strokes.[11]
- In hemorrhagic stroke, surgery can be used to repair a superficial ruptured aneurysm or AVM, prevent rebleeding, and evacuate a clot (hematoma). Larger, deeper intracranial or brainstem vascular lesions are generally not amendable to surgery.
- Surgery may also be indicated for resection of a superficial unruptured AVM when there is high risk of rupture and stroke.

Primary Impairments

Sensation

Sensation is frequently impaired but rarely absent on the hemiplegic side. Impairments are reported in about 53 percent of patients with stroke and can range from loss of superficial and/or deep sensations to impairments in the combined cortical sensations (see Chapter 5).[2] The type and extent of impairment is related to the location and size of the vascular lesion. Specific localized areas of dysfunction are common with cortical lesions, whereas diffuse involvement throughout one side of the body suggests deeper lesions involving the thalamus and adjacent structures. The most common distribution of loss is a face-UE-LE pattern (reported in 55 percent of cases). Less frequently, impairments may be noted in face-UE (29 percent of cases) or UE-LE (7 percent of cases).[17] Symptoms of crossed anesthesia (ipsilateral facial impairments with contralateral trunk and limb involvement) typify brainstem lesions. Proprioceptive losses are common. In one study, 44 percent of patients with stroke demonstrated significant proprioceptive loss with associated impairments noted in motor control, postural function, and balance.[18] Loss of superficial touch and pain and temperature sensation is also common. Profound hemisensory loss can contribute to unilateral neglect, difficulty with functional tasks, and increased risk of self-injury. The patient may also complain of abnormal sensations such as numbness, dysesthesias, or hyperesthesia.

Pain

Hemorrhagic or ischemic stroke can result in severe headache or neck and face pain. Lesions of the PCA involving the ventral posterolateral thalamus and spinothalamic system can result in central post-stroke (thalamic) pain. This is characterized by constant, severe burning pain with intermittent sharp pains. Patients experience an exaggerated response to stimuli affecting the contralateral half of the body. Paroxysmal spasms of pain may be triggered by simply stroking the skin, pinprick, contact with heat or cold, and pressure (hyperalgesia). Loud noises, bright lights, or other mild irritants may also trigger pain. Thalamic pain is typically delayed in onset and may not appear until a few weeks or months after the onset of stroke. Spontaneous recovery is rare and suffering may be intolerable. Patients may experience little relief from analgesic treatment. The debilitating nature of thalamic pain frequently prevents the patient from actively participating in rehabilitation.[19]

Visual Changes

Homonymous hemianopsia, a visual field defect, occurs with lesions involving the optic radiation in the internal capsule (MCA distribution) or to the primary visual cortex (PCA distribution). It occurs in about 26 percent of patients with stroke. (2) The patient experiences loss of vision in the contralateral half of each visual field; that is, the nasal half of one eye and temporal half of the eye corresponding to the hemiplegic side. Field defects contribute to the patient's overall lack of awareness of the hemiplegic side. Patients who are aware of the problem can compensate with head turning movements. Patients may also experience **visual neglect** (visual inattention) and problems with depth perception, and spatial relationships. See Chapter 29 for a complete discussion of visual and perceptual impairments. Paralysis of conjugate gaze from involvement of frontal lobe eye field area, CN III, or gaze centers in the pontine reticular formation result in **forced gaze deviation**. Unopposed action of eye muscles causes the eyes to deviate in the direction of the intact musculature. Patients with hemispheric lesions may look away from the hemiplegic side, while patients with brainstem lesions may look toward the hemiplegic side. Brainstem strokes affecting the coordination of eye muscles may also produce signs of diplopia, oscillopsia, or visual distortions.

Motor Function

Stages of Motor Recovery

Initially flaccid paralysis is present (stage 1). This is replaced by the development of spasticity, hyperreflexia, and mass patterns of movement, termed **obligatory synergies**. Muscles involved in these synergy patterns are often so strongly linked together that isolated movements outside the obligatory patterns are not possible. During stage 2 (early synergy) facilitatory stimuli will elicit synergies with no or only minimal voluntary movement. As recovery progresses, spasticity is marked with full strong obligatory synergies (stage 3). Synergy influence begins to decline in stage 4 as some movements out-of-synergy emerge, especially if movement takes place in the weaker synergy first. During stage 5, relative independence of synergy, spasticity continues to wane and isolated joint movements become more apparent while during stage 6 patterns of movement are near normal. This general pattern of recovery was initially described by Twitchell[20] and Brunnstrom[21,22] and confirmed by additional investigators[23–26] (Box 18.2). Several important points merit consideration. An overall pattern of motor recovery exists though individual recovery is highly variable. Some patients experience mild involvement with full recovery while other patients demonstrate severe involvement with incomplete recovery. The degree of recovery depends on a number of factors, including lesion location and severity and capacity for adaptation through training. Finally, recovery differs within patients. For example, the UE may be more involved and demonstrate less complete recovery than the LE as is seen in MCA syndrome.

Box 18.2 Sequential Motor Recovery Stages Following Stroke

STAGE 1 Recovery from hemiplegia occurs in a stereotyped sequence of events that begins with a period of *flaccidity* immediately following the acute episode. *No movement of the limbs* can be elicited.

STAGE 2 As recovery begins, the basic limb synergies or some of their components may appear as associated reactions, or *minimal voluntary movement* responses may be present. At this time, spasticity begins to develop.

STAGE 3 Thereafter, the patient gains *voluntary control of the movement synergies*, although full range of all synergy components does not necessarily develop. Spasticity has further increased and may become severe.

STAGE 4 Some *movement combinations that do not follow the paths of either synergy are mastered*, first with difficulty, then with more ease, and *spasticity begins to decline.*

STAGE 5 If progress continues, more *difficult movement combinations are learned* as the basic limb synergies lose their dominance over motor acts.

STAGE 6 With the *disappearance of spasticity, individual joint movements become possible and coordination* approaches normal. From here on, as the last recovery step, normal motor function is restored, but this last stage is not achieved by all, for the recovery process can plateau at any stage.

From Brunnstrom, S: Movement Therapy in Hemiplegia. Harper & Row, New York, 1970, with permission.

Weakness

Weakness (paresis) is found in 80 to 90 percent of all patients after stroke and is a major factor in disability.[17] Patients are unable to generate the force necessary for initiating and controlling movement. The degree of weakness is related to the location and size of the brain injury and varies from a complete inability to achieve any visible contraction to measurable impairments in force production. Deficits on the contralateral, side typically include hemiparesis (opposite UE and LE). Owing to the high incidence of MCA strokes, the UE is frequently more affected than the LE. About 20 percent of individuals with MCA strokes fail to regain any functional use of the affected UE. Typically, distal muscles exhibit greater strength deficits than proximal.[27,28] This can be explained by the greater facilitation of distal muscles than proximal by the corticospinal system.[29] Some researchers have not found this proximal–distal gradient.[30] Mild weakness also occurs on the ipsilateral, "supposedly normal" side.[27,28] This can be explained by the fact that only 75 to 90 percent of the corticospinal fibers cross in the medulla to the contralateral side. The remainder are transmitted to the spinal cord ipsilaterally

in the anterior or ventral corticospinal tract. Once in the spinal cord some of these fibers cross while the rest remain uncrossed, thereby explaining bilateral weakness.[29] The amount of weakness experienced by the patient may also vary according to specific functional tasks. Thus, a patient may appear stronger in some tasks than others.

Observed muscle weakness is also associated with a number of changes in both the muscle and the motor unit. Changes occur in muscle composition, including atrophy of muscle fibers. There is a selective loss of type II fast-twitch fibers with an increase in the percentage of type I fibers (a finding also reported in the elderly). This selective loss of type II fibers results in difficulty with initiation and production of rapid, high-force movements. The number of functioning motor units and discharge firing rates also decrease, in one study by as much as 50 percent at 6 months following stroke.[31] This is explained by the presence of transsynaptic degeneration of alpha motoneurons that occurs with loss of corticospinal innervation.[32] Abnormal recruitment of motor units has also been reported.[33–37] Thus patients demonstrate inefficient patterns of muscle activation and difficulty maintaining a constant level of force production. Patients demonstrate increased effort and fatigability with frequent complaints of feelings of weakness. Denervation potentials are common, also the result of denervation changes in the corticospinal tracts.[38] Overall reaction times are increased, a finding also reported in the stronger extremities and for the elderly in general. Movement times are prolonged, a timing abnormality that contributes to impairment of coordinated motor sequences.[39,40] Finally, there is increased coactivation of agonists and antagonists, a timing disorder that limits force production during voluntary movements.[41,42]

Alterations in Tone

Flaccidity (hypotonicity) is present immediately after stroke and is due primarily to the effects of cerebral shock. It is generally short-lived, lasting a few days, or weeks. Flaccidity may persist in a small number of patients with lesions restricted to the primary motor cortex or cerebellum. **Spasticity** (hypertonicity) emerges in about 90 percent of cases and occurs on the side of the body opposite the lesion. Spasticity in UMN syndrome occurs predominately in antigravity muscles (see Chapter 8, Table 8.1). In the patient with stroke, UE, spasticity is frequently strong in scapular retractors; shoulder adductors, depressors, and internal rotators; elbow flexors and forearm pronators; and wrist and finger flexors. In the neck and trunk, spasticity may cause increased lateral flexion to the hemiplegic side. In the LE, spasticity is often strong in the pelvic retractors, hip adductors and internal rotators, hip and knee extensors, plantarflexors and supinators, and toe flexors. Spasticity results in tight (stiff) muscles that restrict volitional movement. Posturing of the limbs (e.g., a tight fisted hand with the elbow bent and held tightly against the chest or a stiff extended knee with a plantarflexed foot) is common with moderate to severe spasticity. Spastic posturing can lead to the development of painful spasms (similar to muscle cramping), degenerative changes, and fixed contractures.[43] The adjustment of postural muscles that occurs normally in preparation for and during a movement task, termed *automatic postural tone*, is also impaired.[44] Thus, patients with stroke may lack the ability to adjust and stabilize proximal limbs and trunk appropriately during movement, with resulting postural abnormalities, balance impairments, and increased risk for falls.

Abnormal Synergy Patterns

Abnormal and highly stereotyped **obligatory synergies** emerge with spasticity. Thus, the patient is unable to perform an isolated movement of a limb segment without producing movements in the remainder of the limb. For example, efforts to bend the elbow also result in shoulder flexion, abduction, and external rotation. The patient is severely limited in the ability to adapt movements to varying task or environmental demands. Obligatory synergies can be elicited either reflexively, as associated reactions, or as minimal voluntary movements. As recovery progresses they become stronger and are linked to spasticity. Two distinct abnormal synergy patterns have been described for each extremity: a flexion synergy and an extension synergy (Table 18.5). An inspection of the synergy components reveals that certain muscles are not usually involved in either synergy. These muscles include the (1) latissimus dorsi, (2) teres major, (3) serratus anterior, (4) finger extensors, and (5) ankle evertors. These muscles, therefore, are generally difficult to activate while the patient is exhibiting these patterns. Obligatory synergies are often incompatible with normal activities of daily living and functional mobility skills (FMS). For example, the patient with a strong LE extensor synergy will have a difficulty walking owing to foot plantarflexion and inversion when the hip and knee are extended. As recovery progresses, spasticity and obligatory synergies begin to disappear and more normal synergies with isolated joint control become possible.[22,45,46]

Abnormal Reflexes

Reflexes are altered and also vary according to the stage of recovery. Initially, stroke results in hypoflexia with flaccidity. When spasticity and synergies emerge, hyperreflexia is seen. Stretch reflexes are hyperactive and patients may demonstrate clonus, clasp-knife response, and a positive Babinski, all consistent findings of UMN syndrome.

Tonic reflexes may appear in a readily identifiable form similar to that seen in other types of neurological insult (e.g., traumatic brain injury, cerebral palsy). Thus, movement of the head or position of the body may elicit an obligatory change in resting tone or movement of the extremities. The most commonly seen is the asymmetric tonic neck reflex (ATNR) in which head rotation causes elbow extension of the UE on the jaw side with elbow flexion of the opposite skull limb.[22,23,47]

Table 18.5 Obligatory Synergy Patterns Following Stroke

	Flexion Synergy Components	Extension Synergy Components
Upper extremity	Scapular retraction/elevation or hyper-extension	Scapular protraction
	Shoulder abduction, external rotation	Shoulder adduction,[a] internal rotation
	Elbow flexion[a]	Elbow extension
	Forearm supination	Forearm pronation[a]
	Wrist and finger Flexion	Wrist and finger flexion
Lower extremity	Hip flexion,[a] abduction, external rotation	Hip extension, adduction,[a] internal rotation
	Knee flexion	Knee extension[a]
	Ankle dorsiflexion, inversion	Ankle plantarflexion,[a] inversion
	Toe dorsiflexion	Toe plantarflexion

[a]Generally the strongest components

Associated reactions are also typically present in patients with stroke who exhibit strong spasticity and obligatory synergies. These consist of unintentional movements of the hemiparetic limb caused by voluntary action of another limb or by the stimulation of yawning, sneezing, or coughing. For example, the patient vigorously contracts the elbow flexors of the stronger UE; the hemiparetic elbow also flexes. Or the patient flexes the hip and lifts the hemiparetic LE in sitting; the hemiparetic UE also flexes. Associated reactions can limit functional performance, especially in the UE.[22,48]

Altered Coordination

Proprioceptive losses can result in sensory ataxia. Strokes affecting the cerebellum typically produce cerebellar ataxia (e.g., lateral medullary syndrome, basilar artery syndrome, pontine syndromes) and motor weakness. The resulting problems with timing and sequencing of muscles can significantly impair function and limit adaptability to changing task and environmental demands. Basal ganglia involvement (posterior cerebral artery syndrome) may lead to slow movements (bradykinesia) or involuntary movements (choreoathetosis, hemiballismus).

Altered Motor Programming

Motor praxis is the ability to plan and execute coordinated movement. Lesions of the premotor frontal cortex of either hemisphere, left inferior parietal lobe, and corpus callosum can produce apraxia. Apraxia is more evident with left hemisphere damage than right and is commonly seen with aphasia. The patient demonstrates difficulty planning and executing purposeful movements that cannot be accounted for by any other reason (i.e., impaired strength, coordination, sensation, tone, cognitive function, communication, or uncooperativeness). There are two main types of apraxia. **Ideational apraxia** refers an inability of the patient to produce movement either on command or automatically and represents a complete breakdown in the conceptualization of the task. The patient has no idea how to do the movement and thus cannot formulate the required motor programs. With **ideomotor apraxia** the patient is unable to produce a movement on command but is able to move automatically. Thus the patient can perform habitual tasks when not commanded to do so and often perseverates, repeating the activity over and over. See Chapter 29 for a more complete discussion of apraxia.

Postural Control and Balance

Balance is disturbed following stroke with impairments in steadiness, symmetry, and dynamic stability common.[49–51] Problems may exist when reacting to a destabilizing external force (reactive postural control) or during self-initiated movements (anticipatory postural control). Thus, the patient may be unable to maintain balance in sitting or standing or to move in a weightbearing posture without loss of balance. Disruptions of central sensorimotor processing lead to an inability to adapt postural movements to changing task and environmental demands and impair motor learning.[52] Patients with stroke typically demonstrate asymmetry with most of the weight in sitting or standing shifted toward the stronger side. They also demonstrate increased postural sway in standing (a finding characteristic of the elderly in general).[53] Delays in the onset of motor activity, abnormal timing and sequencing of muscle activity, and abnormal co-contraction result in disorganization of postural synergies. For example, proximal muscles may be activated in advance of distal muscles or in some patients, very late (a finding also found in many elderly). Compensatory responses typically include excessive hip

and knee movements. Corrective responses to perturbations or destabilizing forces are frequently inadequate and result in loss of balance and falls. Patients with hemiplegia typically fall in the direction of weakness.[54,55]

Ipsilateral Pushing

Ipsilateral pushing (also known as **pusher syndrome** [56] **or contraversive pushing** [57]) is an unusual motor behavior characterized by active pushing with the stronger extremities toward the hemiparetic side, leading to a lateral postural imbalance. The end result is a tendency to fall toward the hemiparetic side. Pushing can vary in severity and generally increases with the difficulty of the postural challenge. During sitting, the push results in a strong lateral lean toward the weaker side, often pushing the patient onto the wheelchair arm. In standing, a strong push creates an unstable situation with a high risk of falls because the hemiparetic LE typically cannot support the body weight. The patient shows no fear even when active pushing leads to instability and strongly resists any attempts to passively correct posture to mid-line, symmetrical weightbearing. This pattern is totally opposite the expected postural deficiency seen in most patients after stroke, that is, increased weightbearing to the stronger side to compensate for deficits on the hemiparetic side. Pushing is caused by severe misperception of body orientation in relation to gravity. Karnath et al[57] found patients experienced a misperception of subjective postural vertical position, perceiving their body as vertical when it was actually tilted about 18°. They also found that the visual and vestibular inputs for orientation perception to vertical remained intact as patients were able to align their bodies with the help of visual cues and conscious strategies. Ipsilateral pushing is the result of stroke affecting the posterolateral thalamus[57,58] and a deficit in processing of somesthetic information.[58,59] Pedersen et al[60] identified the behavior in 10 percent of 327 patients with stroke. These researchers also found no significant association between ipsilateral pushing and hemineglect, anosognosia, aphasia, or apraxia. Functional mobility skills are significantly impaired for patients with ipsilateral pushing. Typically the patient demonstrates severe problems in transfers, standing, and gait. The use of a cane during ambulation is problematic because patients use the cane to increase push to the hemiplegic side. Peterson et al[60] demonstrated that patients with ipsilateral pushing have poorer rehabilitation outcomes with longer hospital stays and prolonged recovery times (3.6 weeks longer on average). They also had significantly lower functional scores on admission and discharge with increased levels of dependence at discharge. However with training, the brain can compensate well. The syndrome is rarely still evident at 6 months.[61]

Speech, Language, and Swallowing

Patients with lesions involving the cortex of the dominant hemisphere (typically the left hemisphere) demonstrate speech and language impairments. **Aphasia** is the general term used to describe an acquired communication disorder caused by brain damage and is characterized by an impairment of language comprehension, formulation, and use. Aphasia has been estimated to occur in 30 to 36 percent of all patients with stroke.[2] There are many different types of aphasias; major classification categories are fluent, nonfluent, and global. In **fluent aphasia (Wernicke's/sensory/receptive aphasia)**, speech flows smoothly with a variety of grammatical constructions and preserved melody of speech. Auditory comprehension is impaired. Thus, the patient demonstrates difficulty in comprehending spoken language and in following commands. The lesion is located in the auditory association cortex in the left lateral temporal lobe. In **nonfluent aphasia (Broca's/expressive aphasia)** the flow of speech is slow and hesitant, vocabulary is limited, and syntax is impaired. Speech production is labored or lost completely while comprehension is good. The lesion is located in the premotor area of the left frontal lobe. **Global aphasia** is a severe aphasia characterized by marked impairments of both production and comprehension of language. It is often an indication of extensive brain damage. Severe problems in communication may limit the patient's ability to learn and often impedes successful outcomes in rehabilitation. See Chapter 30 for a complete discussion of these impairments and their management.

Patients with stroke commonly present with **dysarthria** with a reported incidence ranging from 48 to 57 percent.[2] This term refers to a category of motor speech disorders caused by lesions in parts of the central or peripheral nervous system that mediate speech production. Respiration, articulation, phonation, resonance, and/or sensory feedback may be affected. The lesion can be located in the primary motor cortex in the frontal lobe, the primary sensory cortex in the parietal lobe, or the cerebellum. Volitional and automatic actions such as chewing and swallowing and movement of the jaw and tongue are impaired resulting in slurred speech. In patients with stroke, dysarthria can accompany aphasia, complicating the course of rehabilitation (see Chapter 30).

Swallowing difficulty, **dysphagia**, occurs in about 12 percent of patients with lesions affecting the medullary brainstem (CN IX and X), large vessel pontine lesions, as well as in acute hemispheric lesions (especially infarcts in the MCA and PCA).[2] Dysfunction of the lips, mouth, tongue, palate, pharynx, larynx, or proximal esophagus all contribute to dysphagia. The most frequent problem seen with dysphagia is delayed triggering of the swallowing reflex (86 percent of patients) followed by reduced pharyngeal peristalsis (58 percent of patients) and reduced lingual control (50 percent of patients).[62] Altered mental status, altered sensation, poor jaw and lip closure, impaired head control, and poor sitting posture also contribute to the patient's swallowing difficulties. Most patients demonstrate multiple problems that can include drooling, difficulty ingesting food, compromised nutritional status, and

dehydration. Dysphagia may be severe enough to require the use of tube feeding, either a nasogastric (NG) tube for short periods of time or an invasive gastrostomy (G) tube for more long-term care. Nutrition can also be provided through an intravenous route (total parenteral nutrition [TPN]). There are numerous risks and complications associated with these feeding methods and difficult clinical decisions are made by the family in conjunction with the medical or dysphagia team.

Perception and Cognition

Stroke can produce visual–perceptual deficits, with a reported incidence ranging from 32 to 41 percent.[2] They are frequently the result of lesions in the right parietal cortex and seen more with left hemiplegia than right. These may include disorders of **body scheme/body image**, **spatial relations**, and **agnosias**. Body scheme refers to a postural model of the body including the relationship of the body parts to each other and the relationship of the body to the environment. Body image is the visual and mental image of one's body that includes feelings about one's body. Both may be distorted following stroke. Specific impairments of body scheme/ body image include **unilateral neglect**, **anosognosia**, **somatoagnosia**, **right–left discrimination**, **finger agnosia**, and **anosognosia**. Spatial relations syndrome refers to a constellation of impairments that have in common a difficulty in perceiving the relationship between the self and two or more objects in the environment. It includes specific impairments in figure–ground discrimination, form discrimination, spatial relations, position in space, and topographical disorientation. Agnosia is the inability to recognize incoming information despite intact sensory capacities. Agnosias can include visual object agnosia, auditory agnosia or tactile agnosia (astereognosis). The reader is referred to Chapter 29 for a more complete discussion of these deficits and their management.

Cognitive deficits are present with lesions involving the cortex and include impairments in alertness, attention, orientation, memory, or executive functions. Premorbid changes associated with pathological aging may also account for some of the dysfunction noted and should be carefully determined from interviews with family, significant others, or caregivers. The patient with acute stroke may be largely unaware of what is going on in the external environment, a problem of impaired alertness that results from lesions in the prefrontal cortex and reticular formation. The patient may also be disoriented and unable to provide information about self, time of day, physical or geographical location, or disability, the result of lesions affecting the prefrontal cortex, limbic system, and limbic cortex. **Attention** is the ability to select and attend to a specific stimulus while simultaneously suppressing extraneous stimuli. Attention disorders include impairments in sustained attention, selective attention, divided attention, or alternating attention. Altered attention results from lesions in the prefrontal cortex and reticular formation. **Memory** is defined as the ability to store experiences and perceptions for later recall. Memory disorders include impairments in immediate recall and short-term or long-term memory. Immediate and short-term memory impairments are common, occurring in about 36 percent of patients with stroke while long-term memory typically remains intact.[2] Thus, the patient cannot remember the instructions for a new task given only minutes or hours ago but can easily remember things done 30 years ago. Short-term memory loss is associated with lesions of the limbic system, limbic association cortex (orbitofrontal areas), or temporal lobes. Long-term memory loss is associated with lesions of the hippocampus of the limbic system. Memory gaps may be filled with inappropriate words or fabricated stories, an impairment termed **confabulation** that also results from lesions in the prefrontal cortex. The patient may be confused, demonstrating disorientation and an inability to understand the specific context of a conversation. Confusion is the result of disruption of the prefrontal cortex and occurs with diffuse, bilateral lesions. **Perseveration** is the continued repetition of words, thoughts, or acts not related to current context. Thus the patient gets "stuck" and repeats words or acts without much success at stopping. Preservation results from lesions in the premotor and/or prefrontal cortex.[63,64]

Executive functions, defined as those capacities that enable a person to engage in purposeful behaviors, include volition, planning, purposeful action, and effective performance. Patients with lesions of the prefrontal cortex typically demonstrate impairments in executive function including some or all of the following: impulsiveness, inflexible thinking, lack of abstract thinking, impaired organization and sequencing, decreased insight, impaired planning ability, and impaired judgment. Patients are unable to realistically appraise their environment and the people and events in it. They also demonstrate difficulty in self-monitoring and self-correcting behaviors, thereby posing enormous safety risks. See Chapter 29 for a complete discussion of these impairments and their management.

Dementia can result from multiple small infarcts of the brain, termed **multi-infarct dementia**. It is characterized by progressive impairments in memory and cognition. These changes are associated with episodes of cerebral ischemia and hypertension. Other contributing factors include arrhythmias, myocardial infarct, TIAs, diabetes, obesity, and smoking. Scattered areas of the brain are involved, evidenced by focal neurological deficits. Onset is frequently abrupt. The patient may fluctuate between periods of impaired function and periods of improved function, demonstrating a stepwise and paroxysmal deterioration of intellectual function. This is in contrast to the gradual onset and more steady, widespread decline seen in Alzheimer's dementia.[65]

Delirium, also known as acute confusional state, can result from a number of factors following acute stroke.

Deprivation of oxygen to the brain, metabolic imbalance, or adverse drug reactions can all induce confusion. Additional contributory factors can include sensory and perceptual losses coupled with an unfamiliar hospital environment and inactivity. Delirium is characterized by a clouding of consciousness or dulling of cognitive processes and impaired alertness. Thus the patient is inattentive, incoherent, and disorganized with fluctuating levels of consciousness. Hallucinations and agitation are also common. Nighttime may be particularly problematic. Patients with significant sensory deficits following stroke may experience sensory deprivation problems evidenced by irritability, confusion, psychosis, delusions, and even hallucinations. These problems are more frequently seen in patients who have been confined to a bed for a long time or whose bed is positioned to limit social interaction (e.g., with the more involved side toward the door). Some patients are equally unable to deal with a sensory overload, produced by too much stimulation. Altered arousal levels are implicated.

Emotional Status

Lesions of the brain affecting the frontal lobe, hypothalamus, and limbic system can produce a number of emotional changes. The patient with stroke may demonstrate **pseudobulbar affect (PBA)**, also known as *emotional lability* or *emotional dysregulation syndrome*. PBA occurs in about 18 percent of cases and is characterized by emotional outbursts of uncontrolled or exaggerated laughing or crying that are inconsistent with mood. The patient quickly changes from laughing to crying with only slight provocation. The patient is typically unable to control these episodes or to inhibit the expression of spontaneous emotions.[66] Frequent crying may also accompany depression. **Apathy** occurs in about 22 percent of cases and is characterized by a shallow affect and blunted emotional responses. In such patients, apathy is frequently misconstrued as depression or poor motivation. Patients can also demonstrate **euphoria** (exaggerated feelings of well-being), increased levels of irritability or frustration, and social inappropriateness. Changes in the ability to sense, move, communicate, think, or act as before are enormously frustrating by themselves and create high stress levels for the patient with stroke. Increased levels of irritability and frustration are the natural outcomes of high stress levels. These behaviors along with a poor social perception of one's self and environment may lead to increasing isolation and social withdrawal.[67,68]

Depression is extremely common, occurring in about one-third of stroke cases.[2] It is characterized by persistent feelings of sadness accompanied by feelings of hopelessness, worthlessness and/or helplessness. Depressed patients may also experience a loss of energy or persistent fatigue, an inability to concentrate, and decreased interest in daily life along with changes in weight and sleep patterns, generalized anxiety, and recurrent thoughts of death or suicide. Depression is seen with lesions in the left frontal lobe (acute stage) and with lesions in the right parietal lobes (subacute stage).[69,70] Most patients remain significantly depressed for many months, with an average time of 7 to 8 months. The period from 6 months to 2 years after a CVA is the most likely time for depression to occur. Depression occurs in both mildly and severely involved patients and thus is not significantly related to the degree of motor impairment. Patients with lesions of the left hemisphere may experience more frequent and more severe depression than patients with right hemisphere or brainstem strokes.[71,72] These findings suggest that post-stroke depression is not simply a result of psychological reaction to disability but rather a direct impairment of the CVA. Prolonged post-stroke depression can interfere with the success of rehabilitation and result in poorer long-term functional outcomes. The reader is referred to Chapter 2 for a more complete discussion of psychosocial impairments and their management.

Hemispheric Behavioral Differences

Individuals with stroke differ widely in their approach to processing information and in their behaviors. Those with *left hemisphere damage* (right hemiplegia) demonstrate difficulties in communication and in processing information in a sequential, linear manner. They are frequently described as cautious, anxious, and disorganized. This makes them more hesitant when trying new tasks and increases the need for feedback and support. They tend, however, to be realistic in their appraisal of their existing problems. Individuals with *right hemisphere damage* (left hemiplegia), on the other hand, demonstrate difficulty in spatial–perceptual tasks and in grasping the whole idea of a task or activity. They are frequently described as quick and impulsive. They tend to overestimate their abilities while acting unaware of their deficits. Safety is therefore a far greater issue with patients with left hemiplegia, where poor judgment is common. These patients also require a great deal of feedback when learning a new task. The feedback should be focused on slowing down the activity, checking sequential steps, and relating it to the whole task. The patient with left hemiplegia frequently cannot attend to visuospatial cues effectively, especially in a cluttered or crowded environment.[73–75] Table 18.6 summarizes the behavioral differences attributed to damage of the left and right hemispheres.

Bladder and Bowel Function

Disturbances of bladder function are common during the acute phase, occurring in about 29 percent of cases.[2] Urinary incontinence can result from bladder hyperreflexia or hyporeflexia, disturbances of sphincter control, and/or sensory loss. A toileting schedule for prompted voiding is often implemented to reduce the incidence of incontinence

Table 18.6 Hemispheric Differences Commonly Seen Following Stroke

Right Brain Injury	Left Brain Injury
Left-side hemiplegia/paresis	Right-side hemiplegia/paresis
Left-side hemisensory loss	Right-side hemisensory loss
Visual–perceptual impairments: Left-side unilateral neglect Agnosia Visuospatial impairments Disturbances of body image and body scheme	Speech and language impairments: (dominant hemisphere/right-handed individuals) Nonfluent (Broca's) aphasia Fluent (Wernicke's) aphasia Global aphasia
Difficulty sustaining a movement	Difficulty planning and sequencing movements Apraxia more common: ideational, ideomotor
Quick, impulsive behavioral style	Slow, cautious behavioral style
Difficulty grasping the overall organization or pattern, problem-solving and synthesizing information	Disorganized problem-solving
Often unaware of impairments Poor judgment Inability to self-correct; increased safety risk	Often very aware of impairments Anxious about poor performance
Rigidity of thought Difficulty with abstract reasoning	Difficulty with processing delays
Difficulty with perception of emotions, expression of negative emotions	Difficulty with expression of positive emotions
Difficulty processing visual cues	Difficulty processing verbal cues, verbal commands
Memory impairments, typically related to spatial-perceptual information	Memory impairments, typically related to language
Dysfunction of either hemisphere depending on lesion location: Visual field defects Emotional abnormalities: lability, apathy, irritability, low frustration levels, depression Cognitive deficits: confusion, short attention span, loss of memory, executive functions	

and to accommodate for factors that cause functional incontinence such as inattention, mental status changes, or immobility. Generally, this problem improves quickly. Persistent incontinence is often due to a treatable medical condition (e.g., urinary tract infection). Absorbent pads and special undergarments or external collection devices may be used if incontinence proves refractory. Urinary retention can be controlled pharmacologically and with intermittent or indwelling catheterization. Early treatment is desirable to prevent further complications such as chronic urinary tract infection and skin breakdown. Patients who are incontinent often suffer embarrassment, isolation, and depression. Persistent incontinence is associated with a poor long-term prognosis for functional recovery.

Disturbances of bowel function can include incontinence and diarrhea or constipation and impaction. Patients who are constipated may require stool softeners and dietary/fluid modifications to resolve this problem. Physical activity is also helpful to improve these problems.

Complications and Indirect Impairments

Musculoskeletal

Loss of voluntary movement and immobility can result in loss of range of movement (ROM) and contractures. Contractures can develop anywhere but are particularly apparent in the paretic limbs. As contractures progress, edema and pain may develop and further restrict mobility. In the UE, limitations in the shoulder motions of flexion, abduction, and external rotation are common. Contractures are likely in the elbow flexors, wrist and finger flexors, and

forearm pronators. In the LE, plantarflexion contractures are common. Alterations in alignment coupled with decreased activity of muscles may lead to postural deformity, altered patterns of movement with increased energy expenditure, and excessive effort.

Disuse atrophy and muscle weakness results from inactivity and immobility. Early mobilization stressing out-of-bed upright postures and weightbearing activities along with forced use of the involved extremities are effective strategies in counteracting these effects. Patients recovering from stroke consistently demonstrate the strong negative impact of weakness on functional outcomes. Impairments in gait, balance, falls, UE functional tasks, and manual dexterity have all been linked directly to impairments of strength.[76–79]

Osteoporosis, a bone disease characterized by a loss of bone mass per unit volume, is common in the elderly and results from decreased physical activity, changes in protein nutrition, hormonal deficiency, and calcium deficiency. Patients with stroke who are immobilized and restricted in weightbearing demonstrate increased risk of osteoporosis. Fall risk is also increased with incidence rates ranging between 23 percent and 50 percent for individuals with chronic stroke.[80] Risk of falls in patients with stroke is multifactorial, arising from sensorimotor deficits, impaired balance, confusion, attention deficits, perceptual deficits, visual impairments, behavioral impulsivity, depression, and communication problems.[81–85] Increased risk of fracture, especially vertebral and hip fracture, is the natural outcome of osteoporosis and falls. In patients with stroke, osteoporosis and hip fracture are more likely on the more involved side.[86] Strategies to improve motor function and prevent falls are indicated (for an overview, see Chapter 13).

Neurological

Seizures

Seizures occur in a small percentage of patients with stroke and are slightly more common in occlusive carotid disease (17 percent) than in MCA disease (11 percent). Seizures are common right after stroke during the acute phase (e.g., in about 15 percent of cases with cerebral hemorrhage); late-onset seizures can also occur several months after stroke. They tend to be of the partial motor type.[87] Seizures are potentially life threatening if not controlled. Anticonvulsant medications may be indicated (e.g., phenytoin [Dilantin], carbamazepine [Tegretol], phenobarbital [Solfoton]. Potential adverse side effects include sedation (drowsiness) and ataxia.

Hydrocephalus

Hydrocephalus, an excessive accumulation of cerebral spinal fluid (CSF) within the cranial cavity, is rare but can occur with subarachnoid or intracerebral hemorrhage. The increasing blood volume results in obstruction of CSF circulation. Patients may experience headache, nausea, vomiting, visual impairment, increasing lethargy, and ataxia. Seizures can also occur. Surgical intervention with ventriculostomy and ventricular drainage is necessary in emergency situations. Long-term management is accomplished with the placement of a ventricular peritoneal shunt.[11]

Cardiovascular/Pulmonary

Thrombophlebitis/Deep Vein Thrombosis

Thrombophlebitis and deep venous thrombosis (DVT) are potential complications for all immobilized patients. The incidence of DVT in patients with stroke is as high as 47 percent with an estimated 10 percent of deaths attributed to pulmonary embolism. (2) The dangers are particularly high during the acute phase when venous stasis from bed rest, limb paralysis and decreased activity, hemineglect, and reduced cognitive status significantly elevate the risks. The hallmark clinical signs of DVT include rapid onset of unilateral leg swelling with dependent edema. The patient may report tenderness, a dull ache, or a tight feeling in the calf; pain is usually not severe. About 50 percent of cases do not present with clinically detectable symptoms and can be identified only by radiocontrast venography (the gold standard), impedance plethysmography, or Doppler ultrasonography. Prompt diagnosis and treatment of acute DVT are necessary to reduce the risk of fatal pulmonary embolism. Pharmacological management consists of anticoagulant therapy (blood thinners). Prophylactic use of low-dose heparin (LDH) or low-molecular-weight (LMW) heparin has been shown to reduce the incidence by 45 percent and 79 percent, respectively. Symptomatic treatment of DVT consists of bed rest and elevation of the limb until tenderness subsides (generally 3 to 5 days) to prevent pressure fluctuations within the venous system and emboli. Edema management may include intermittent pneumatic compression and compression stockings.[88] Careful daily monitoring of the lower limbs is essential. Early mobilization and ambulation are important primary prevention measures.

Cardiac Function

The majority of strokes are caused by vascular disease. Patients who suffer a stroke as a result of underlying coronary artery disease (CAD) may demonstrate impaired cardiac output, cardiac decompensation, and serious rhythm disorders. If these problems persist, they can directly alter cerebral perfusion and produce additional focal signs (e.g., mental confusion).[89] Patients with stroke typically exhibit low peak $\dot{V}O_2$ levels during exercise (about half of that achieved by age-matched healthy individuals).[90] These vary according to age, level of disability, number and severity of comorbidities, secondary complications, and medications. Cardiac limitations in exercise tolerance may restrict rehabilitation potential and requires diligent monitoring and careful exercise prescription by the physical therapist.[91]

Most patients with stroke are significantly deconditioned and exhibit low work capacities, the result of acute illness, bedrest, and limited activity levels. Some individuals may have been inactive prior to the stroke. Changes in the cardiovascular system associated with deconditioning include reduced cardiac output, decreased maximal heart rate, increased resting and exercise blood pressures, decreased maximal oxygen uptake, and decreased vital capacity. Changes in the musculoskeletal systems (e.g., decreased muscle mass and strength, decreased bone mass, decreased flexibility) and decreased glucose tolerance also affect exercise tolerance and endurance levels. Decreased activity levels may also be related to depression, a common finding in stroke.[92]

Pulmonary Function

Pulmonary function is often impaired in individuals with stroke. Decreased lung volume, decreased pulmonary perfusion and vital capacity and altered chest wall excursion are all common findings. The decreased respiratory output is accompanied by increased oxygen demands required during altered movement patterns. For example, walking using an orthosis and assistive device dramatically increases the energy demands of the activity. The end result for the patient with stroke is increased fatigue and decreased endurance.

Aspiration, penetration of food, liquid, saliva, or gastric reflux into the airway, occurs in about one third of patients with dysphagia. It is more common during the acute phase of recovery and can occur during any phase of swallowing. Aspiration is an important complication in that it can lead to acute respiratory distress within hours, aspiration pneumonia, and, if left untreated, death. Dysphagia can also lead to dehydration and compromised nutrition. Early examination and treatment of dysphagia is essential to prevent aspiration. Videofluoroscopic examination (a modified barium swallow [MBS]) is the most commonly used technique to examine the preparatory, oral, pharyngeal, and esophageal phases of swallowing. Fiberoptic endoscopic examination of swallowing (FEES) provides information about laryngeal function, hypopharyngeal residue, and airway protection (aspiration).[93]

Integumentary

Ischemic damage and subsequent necrosis of the skin results in skin breakdown and decubitus ulcers. The incidence in patients with stroke is reported to be 14.5 percent.[94] The skin breaks down typically over bony prominences from pressure, friction, shearing, and/or maceration. Intense pressure for a short time or low pressure for a long time results in pressure sores. Friction occurs as the skin rubs or is dragged against the supporting surface, for example, when the patient slides down in bed or is pulled up. Spasticity and contractures can also contribute to increased friction. Shearing occurs from sliding of adjacent structures in opposite directions (skin vs underlying bone), for example, during transfers from bed to stretcher without a pull sheet. Maceration is caused by excess moisture, for example, with urinary incontinence. Additional risk factors include reduced activity (bedfast or chairfast), immobility, decreased sensation, abnormal patterns of movement, poor nutrition, and decreased level of consciousness. The incidence of pressure sores is increased with comorbid medical conditions such as infections, peripheral vascular disease, edema, and diabetes.[2]

Daily systematic inspection of the skin, particularly over high-risk areas is essential in recognizing the early signs of breakdown. The skin needs to be kept clean, dry, and protected from injury. Proper techniques for positioning, turning, and transferring are essential. A positioning schedule is instituted and the time in each position is limited. Assumption of upright postures (sitting and standing) is promoted as soon as possible. Pressure-relieving devices (PRDs) to minimize high concentrations of pressure are used. These may include foam pads, alternating pressure mattress, water mattress, air-fluidized bed, sheepskin, heel and elbow protectors, multipodus boots, and use of a trapeze. Proper positioning (seating) in the wheelchair and use of pressure relieving devices (gel or air cushions) are also critical. Lubricants, protective dressings, and barrier sprays may also be used. Ensuring the patient has adequate nutrition and hydration will also protect against skin breakdown as will early mobilization by the rehabilitation team.

Recovery and Prognosis

Recovery from stroke is generally fastest in the first weeks after onset, with measurable neurological and functional recovery occurring in the first month after stroke. Much of early recovery can be attributed to the resolution of **diachesis**, or transient inhibition of function, that accompanies acute stroke. Thus the reduction of edema, absorption of damaged tissue, and improved local circulation and cellular metabolism allows intact neurons that were previously inhibited to regain function.[95] Patients can continue to make measurable functional gains generally at a reduced rate for months or years after insult. Late recovery of function has been demonstrated for patients with chronic stroke (defined as greater than 1 year post-stroke) who undergo extensive functional training.[96–101] These changes are due largely to function-induced plasticity. A functional training approach that emphasizes use of the more involved extremities and an enriched environment effectively stimulates neural reorganization of the brain. Prolonged recovery with improvements occurring over a period of years is especially apparent in the areas of language and visuospatial function.[2] A more complete discussion of neuroplasticity and function-induced recovery is presented in Chapter 13.

Rates of motor recovery vary across management categories: patients suffering minor stroke recover rapidly with few or no residual deficits whereas severely impaired individuals demonstrate more limited and prolonged recovery. The initial grade of paresis, measured on initial hospital admission, is an important predictor of motor recovery. Motor function often improves after the first few days. In the case of complete paralysis on admission, complete motor recovery occurs in less than 15 percent of patients.[102] Recovery has been demonstrated in a wide variety of patients, including those with extensive central nervous system (CNS) damage and advanced age.[103–112] Patients with small lacunar strokes demonstrate improved motor recovery over patients with large, hemispheric lesions.[113] In an extensive review of the literature, Hendricks et al[102] found no significant difference in potential for motor recovery between type of stroke (hemorrhage vs infarction) and location (brainstem vs hemispheric infarction).

Functional mobility skills are impaired following stroke and vary considerably from individual to individual. During the acute stroke phase, 70 to 80 percent of patients demonstrate mobility problems in ambulation while 6 months to 1 year later the figures are reversed, with only 20 percent of patients needing help to walk independently. Basic ADL skills such as feeding, bathing, dressing, and toileting are also compromised during acute stroke, with 67 to 88 percent of patients demonstrating partial or complete dependence. Independence in ADL also improves with time with only 31 percent of survivors requiring partial or total assistance a year later.[114,115] The ability to perform functional tasks is influenced by a number of factors. Motor and perceptual impairments have the greatest impact on functional performance, but other limiting factors include sensory loss, disorientation, communication disorders, and decreased cardiorespiratory endurance. Enablement factors include high motivation, stable supportive family, financial resources, and intensive training with repetitive practice.

Physical Rehabilitation

Acute Phase

Low-intensity rehabilitation can begin in the acute care facility as soon as the patient is medically stabilized, typically within 72 hours. Patients may be admitted to a stroke unit or a neurological unit that provides comprehensive rehabilitation services. Evidence supports the benefits of such specialized units in significantly improving functional outcomes when compared to patients not receiving specialized care.[116–120] Early mobilization prevents or minimizes the harmful effects of deconditioning and the potential for secondary impairments. Functional reorganization is promoted through early stimulation and use of the hemiparetic side. *Learned nonuse* of the hemiparetic extremities and maladaptive patterns of movement are minimized. Mental deterioration, depression, and apathy can be reduced through the fostering of a positive outlook toward the rehabilitation process. Patients need to be presented early on with an organized plan for rehabilitation that addresses their individual goals and stresses resumption of ADL and independent function. It is equally important that patients and their families receive accurate information about stroke and available support. Current trends are toward shorter acute care hospital stays (average stay is about 7 days). However, early discharge has resulted in an increase in the number of serious medical complications seen during inpatient rehabilitation or at home. The rate of serious medical complications on rehabilitation admission ranges from 22 to 48 percent.[121] These complications in turn may result in delays during active rehabilitation and for some temporary cessation of therapy or transfer back to the acute hospital until medical complications are resolved. Therapists need to be vigilant in monitoring patients for potential risk of medical emergencies (e.g., cardiac arrhythmias, DVT, uncontrolled blood pressure, stroke, and so forth).

Post-Acute Phase

Patients with moderate or severe residual impairments or functional limitations may benefit from intensive inpatient rehabilitation provided in a freestanding rehabilitation facility or in a rehabilitation unit within the acute care hospital. Rehabilitation programs certified by the Commission on Accreditation of Rehabilitation Facilities (CARF) and the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) can be expected to adhere to uniform standards and provide high-quality care.[2] Evidence supports the value of inpatient rehabilitation programs in producing improved functional outcomes for patients with stroke.[122–126] Patients are referred to inpatient rehabilitation if they can tolerate an intensity of services consisting of two or more rehabilitation disciplines, 5 days a week for a minimum of 3 hours of active rehabilitation per day. If the patient requires less intensive services, transfer to a transitional care unit (TCU) within the acute care facility or a skilled nursing facility can be requested. Here rehabilitation services are less intense, ranging from 1 hour of therapy services two to three times per week to daily, short sessions.[2]

The timing of rehabilitation services is an important factor in predicting outcome. In general, a shorter onset-to-admission interval, within the first 20 days, has been shown to significantly improve functional outcomes when compared to longer intervals.[127–130] There is also some evidence to suggest that patients with left hemiplegia who suffer profound cognitive–perceptual deficits may respond less favorably to early rehabilitation. These patients may benefit from additional preadmission time to allow for cognitive and perceptual–motor reorganization so critical

for learning.[131] Additional factors that influence the timing of rehabilitation efforts include medical stability, motivation, patient endurance, and recovery. In an era of time-limited payment for comprehensive rehabilitation services, selecting the optimal time for rehabilitation services may prevent unnecessary patient failures and improve long-term functional outcomes.

The preferred practice pattern for patients with stroke from the *Guide to Physical Therapist Practice* is 5 D, *Impaired Motor Function and Sensory Integrity Associated with Nonprogressive Disorders of the Central Nervous System—Acquired in Adolescence or Adulthood.*[132, p 365] In this document the reader will find relevant information on patient/client diagnostic classification; ICD-9-CM codes; examination components; considerations for evaluation, diagnosis, and prognosis; and suggested interventions. Thus the Guide serves as a primary resource to help physical therapists design an appropriate plan of care and document the services provided and outcomes achieved.

A team of rehabilitation specialists including the physician, nurse, physical therapist, occupational therapist, speech–language pathologist, medical social worker, and case manager best provides comprehensive services for the patient with stroke. Additional disciplines may include a neuropsychologist, dietician, ophthalmologist, and recreational or vocational therapist. The patient/client, family, and caregivers are also important members of the team and should be involved in all decision making regarding health, wellness, and fitness needs. Interdisciplinary communication is critical for effective team function and occurs through case conferences, informal interactions, patient care rounds, and patient/client family meetings. Critical tasks for the team include the development of an integrated plan of care with unified goals, interventions, and outcomes that are mutually reinforced by all team members. Effective case management also includes a coordinated education plan, and accurate and effective documentation.

Rehabilitation services during the chronic phase, generally defined to be more than 6 months post-stroke, are typically delivered in an outpatient rehabilitation facility or at home. These services are prescribed for the patient who is discharged from inpatient rehabilitation and in need of continuing rehabilitation. Many of the interventions begun during inpatient rehabilitation are continued and progressed in order to sustain the gains made and improve functional performance. Some patients with mild involvement who did not require intense inpatient rehabilitation may also benefit from outpatient rehabilitation services. A complete record of past medical and rehabilitation services should be made available to these agencies. The intensity of services provided varies but is generally less than that of inpatient rehabilitation (e.g., 60 to 90 minutes per visit, two to three times per week). Outpatient intervention programs that target progressive improvements in flexibility, strength, balance, gait, endurance, and UE function have been shown to be effective in producing meaningful outcomes.[133–138] The patient and family are instructed in a home exercise program (HEP) and educated about the importance of maintaining exercise levels, health promotion, fall prevention, and safety.

The patient can also receive rehabilitation services at home. The challenges of being home can impose additional daily stresses for the patient and family. Difficulties should be addressed promptly as they arise. The therapist needs to emphasize the development of problem-solving skills to ensure successful adaptation to variable home and community environments. Fall risk factors should be eliminated or minimized as appropriate or possible. Examination of the environment and recommendations for modification of the environment are important parts of the preparation for return to home (see Chapter 12).

Finally, the patient should be assisted in the resumption of social and recreational participation. With increasing activity levels, it is important to monitor the patient's endurance levels carefully and provide instruction in energy conservation techniques as needed. A small number of stroke survivors can be evaluated and assisted in return to work. As the patient becomes successful in the home and community environments, services should be gradually phased out. Follow-up visits at periodic intervals are recommended to identify problems as they develop and to ensure long-term maintenance of function.[2]

Examination

The three basic components of a comprehensive physical therapy examination include patient/client history, systems review, and tests and measures. The selection of examination procedures will vary based on a number of factors including patient's age, location and severity of stroke, stage of recovery, data from initial screenings, phase of rehabilitation, home/community/work situation, as well as other factors.

The purposes are to:

- Determine the diagnosis and classification within a specific practice pattern.
- Monitor recovery from stroke.
- Identify patients who are most likely to benefit from rehabilitation services and the most appropriate choice of a setting.
- Develop a specific plan of care, including anticipated goals, expected outcomes, prognosis, and interventions.
- Monitor progress toward projected goals and outcomes through periodic reevaluation.
- Determine if referral to another practitioner is indicated.
- Plan for discharge.

The comprehensive examination provides the main source of information for clinical decision making. Examination findings should be coordinated with those of the rehabilitation team in order to arrive at an integrated plan of care. Box 18.3 presents *Elements of the Examination of*

Box 18.3 Elements of the Examination of the Patient with Stroke[132]

Patient/Client History

- Age, sex, race/ethnicity, primary language, education
- Social history: cultural beliefs and behaviors, family and caregiver resources, social support systems
- Occupation/employment/work
- Living environment: home/ work barriers
- Hand dominance
- General health status: physical, psychological, social, and role function, health habits
- Family history
- Medical/surgical history
- Current conditions/chief complaints
- Medications
- Medical/laboratory test results
- Functional activity level: premorbid

Systems Review

- Neuromuscular
- Musculoskeletal
- Cardiovascular/pulmonary
- Integumentary

Tests and Measures/Impairments

Tests and measures are selected based on their ability to quantify or describe each of the following:

- Level of consciousness, arousal, attention, and cognition: mental status, insight, motivation.
  Primary impairments: impaired alertness and attention, perseveration, confabulation, confusion, disorientation, distractibility, memory deficits, impaired judgment
- Emotional status
  Primary impairments: depression, pseudobulbar affect, apathy, euphoria
- Behavioral style
  Primary impairments: impulsive or cautious behavioral styles; frustration, irritability
- Communication and language: coordinate efforts with the speech–language pathologist
  Primary impairments: fluent, nonfluent or global aphasia, dysarthria.
- Circulation; cardiovascular signs and symptoms.
  Common comorbidities: hypertension, CAD, CHF, diabetes, DVT
- Ventilation and respiration/gas exchange: pulmonary signs and symptoms
  Common comorbidities: chronic pulmonary disease
- Anthropometric characteristics: body mass index, girth, length
  Secondary impairments: edema, common in hand and foot
- Integumentary integrity: skin condition, pressure sensitive areas; effectiveness of protective pressure-relieving devices
  Secondary impairments: altered skin integrity, decubitus ulcers
- Pain: intensity and location
  Primary impairments: central post-stroke pain.
  Secondary impairments: hemiplegic shoulder and/or hand pain
- Cranial and peripheral nerve integrity.
  Primary impairments: dysphagia
- Sensory integrity and integration
  Primary impairments: homonymous hemianopsia, tactile/proprioceptive/kinesthetic losses, astereognosis.
- Perceptual function: coordinate efforts with occupational therapist
  Primary impairments: spatial relations syndrome, body scheme/body image disorders, unilateral neglect, agnosia, topographical disorientation.
- Joint integrity, alignment, and mobility: ROM (active and passive); muscle length and soft tissue extensibility
  Secondary impairments: altered biomechanical alignment; loss of joint ROM, muscle and soft tissue length
- Posture: alignment and position, symmetry (static and dynamic, sitting and standing); ergonomics and body mechanics
  Secondary impairment: altered biomechanical alignment
- Motor function: motor control and motor learning
  Primary impairments:
  Altered reflex integrity: hyperreflexia, tonic reflexes, associated reactions.
  Abnormal tone: flaccidity initially; spasticity: spastic posturing.
  Abnormal (obligatory) synergies: flexion and extension synergy patterns.
  Altered voluntary movement patterns: altered initiation, sequencing, timing of muscle contractions; altered force production.
  Coordination, dexterity, agility: coordination deficits
  Motor planning: ideomotor or ideational apraxia
- Muscle performance: strength, power, and endurance.
  Primary impairments: paralysis or weakness; fatigue
  Secondary impairments: disuse atrophy
- Postural control and balance: sensorimotor integration, balance strategies (static and dynamic); safety
  Primary impairments: altered balance, increased fall risk
- Gait and locomotion: gait pattern and speed, use of assistive devices/orthotic devices, safety
  Primary impairments: altered sequencing, timing, balance, endurance
- Wheelchair management and mobility: safety and endurance
- Aerobic capacity and endurance: functional activity testing, graded exercise testing.
  Secondary impairments: decreased endurance
- Orthotic, protective and supportive devices: fit, alignment, function, use, safety
- Functional status and activity level: performance-based examination of functional skills (FIM level), basic and instrumental ADL; functional mobility skills; home management skills. Assistive or adaptive devices: fit, alignment, function, use; safety
  Primary impairments: loss of independent function
- Work, community, and leisure activities: ability to assume/resume activities, safety

the Patient with Stroke[132] and possible impairments. Many of these examination procedures, tests and measures are discussed in earlier chapters; of special relevance, the reader is referred to Chapter 8. This section discusses relevant tests and measures and *disability-specific instruments* developed for the patient with stroke.

Patient and Client History

Data obtained through interview with the patient/family and review of the medical record should include information on general demographics, medical/surgical history, social and employment history, family history, living environment, general health status, and social and health habits. The patient's current/chief complaints and current functional status, and activity level should be ascertained. Coexisting health problems and medications should also be identified. Data obtained from the history will help focus further in-depth examination and systems review.[79]

Levels of Consciousness

Altered level of consciousness (coma, decreased arousal levels) may occur with extensive brain damage. The Glasgow Coma Scale developed by Teasdale and Jennett[139] is the gold standard used to document level of coma. Three areas of function are examined: eye opening, best motor response, and verbal responses. The therapist should document levels of consciousness using standard descriptive terms: *normal*, *lethargy*, *obtundation*, *stupor*, and *coma*. See Chapter 8. Since the patient's behaviors can be expected to fluctuate widely, frequent repeat observations are necessary.

Communication

The patient's communication abilities should be fully ascertained before proceeding on with other examination procedures. It is not uncommon for family and staff to overestimate the patient's abilities to understand language especially if the patient is cooperative. Close collaboration with the speech–language pathologist is important in making an accurate determination of the patient's communication impairments. Receptive language functions (auditory comprehension, reading comprehension) and expressive language function (word finding, fluency, writing) should be carefully examined. Neuromotor disorders (dysarthria, apraxia) need to be clearly differentiated from aphasia. If communication is severely limited and alternate forms required (gestures, demonstration, communication boards), therapists should be fully knowledgeable of such methods prior to the examination. See Chapter 30.

Cognitive, Emotional, and Behavioral States

It is important to examine cognitive abilities early because it may affect the validity of other tests and measures. An examination of orientation (to person, place, time, and circumstance), attention (selective, sustained, alternating, divided), memory (immediate, short- and long-term), and ability to follow instructions (one-, two-, and three-level commands) can be made from observations of the patient's interactions and responses to specific questions. Higher cortical functions can be examined using tests of simple arithmetic and abstract reasoning (grasp of information, abstract thinking and problem-solving, calculating ability, constructional ability). The *Mini-Mental Status Examination (MMSE)* provides a valid and reliable quick screen of cognitive function.[140] A determination of learning impairments (retention and generalization) usually requires repeat sessions with the patient before a complete picture can be ascertained. Difficulties arise in reaching an accurate determination of cognition when the patient presents with impairments in communication. Close collaboration with the occupational therapist and speech–language pathologist is essential. See Chapters 29 and 30.

Emotional states and behavioral styles can best be examined through observation of the patient in a variety of situations over a number of sessions. It is important to correlate findings with those reported by the family regarding premorbid behaviors and emotional characteristics. Families who report a "personality change" after stroke are likely responding to presenting emotional impairments and disinhibition. Episodes of euphoria and crying should be carefully documented and links to situational or environmental circumstances explored. Duration and frequency of these episodes should also be documented along with strategies that are successful in bringing about an end to the episode. The patient's response to new and stressful situations should also be carefully observed. Depression is common. The *Beck Depression Inventory*[141] is a useful instrument for screening. It consists of 21 statements that are scored on a scale from zero to three (the short version has 13 questions and takes 5 minutes to complete).

Cranial Nerve Integrity

The therapist should examine for facial sensation (CN V), facial movements (CN V, VII), and labyrinthine/auditory function (CN VIII). The presence of swallowing difficulties and drooling necessitates an examination of the motor nuclei of the lower brainstem cranial nerves (CN IX, X, and XII) affecting the muscles of the face, tongue, larynx, and pharynx. This includes determination of motor function of the lips, mouth, tongue, palate, pharynx, and larynx. The gag reflex should be examined because hypoactivity may lead to aspiration into the airway. Adequacy of cough mechanisms should also be carefully examined. A team of specialists, the dysphagia team which typically includes the speech–language pathologist, occupational therapist, and physical therapist, performs detailed examination of swallowing. Therapists need to be able to recognize the presence of swallowing difficulties and initiate prompt referral.

The visual system should be carefully investigated, including tests for visual field defects (CN II, optic radiation, visual cortex), acuity (CN II), pupillary reflexes

(CN II, III), and extraocular movements (CN III, IV, VI). Ocular motility disturbances may be present with brainstem strokes, such as diplopia, oscillopsia, visual distortions, or paralysis of conjugate gaze. Visual field defects (homonymous hemianopsia) need to be differentiated from visual neglect, a perceptual deficit characterized by an inattention to or neglect of visual stimuli presented on the involved side. The patient with pure hemianopsia is typically aware of the deficit and will spontaneously compensate by moving the eyes or head toward the side of deficit; the patient with visual neglect will be unaware (inattentive) of the deficit (see Chapter 29). The use of prescriptive eyeglasses should be determined prior to any testing; the therapist should ensure eyeglasses are worn and clean.

Sensory Integrity

A sensory examination should include testing of superficial sensations (e.g., touch, pressure, sharp or dull discrimination, temperature) and deep sensations (proprioception, kinesthesia, vibration). Combined (cortical) sensations such as stereognosis, tactile localization, two-point discrimination, texture recognition should also be examined (see Chapter 5). Impairments may be evident in one sensory modality and not in others. Differences can also be expected between upper and lower hemiplegic extremities. Comparisons with the intact side can be made, but the therapist should be cognizant that impairments may exist in the supposedly "normal" extremities secondary to effects of comorbid conditions (e.g., neuropathy) or aging. Sensory testing may be difficult or need to be deferred owing to cognitive or communication deficits. Profound sensory impairments will negatively impact on rehabilitation outcomes and goals.

Perception

Significant information on sensory and perceptual deficits will be provided by close collaboration with the occupational therapist. Many tests and formalized test batteries have been developed to examine body scheme, body image, spatial relations, agnosia, and apraxia. These are discussed fully in Chapter 29. Because the patient with left hemiplegia may behave in ways that tend to minimize his or her disabilities, it is easy for staff to overestimate the patient's perceptual abilities. The use of gestures or visual cues may decrease this patient's ability to perform specific perceptual tests, whereas verbal cues may increase chances for success. Carefully structuring the environment to minimize clutter and activity, provide clear boundaries and reference points, as well as provide adequate lighting will also improve performance of the patient who exhibits significant visuospatial impairments.

Problems in unilateral neglect (lack of awareness of part of the body or the external environment) will limit movement and use of the involved extremities (usually the nondominant left side). The patient typically does not react to sensory stimuli (visual, auditory, or somatosensory) presented on the involved side. Careful observation of spontaneous use of affected limbs as well as specific responses to inquiries for movement on or toward the hemiplegic side will provide important information about neglect. Persistent neglect may result in bruising or trauma to the hemiplegic limbs during activity and negatively impacts rehabilitation outcomes.

Joint Integrity and Mobility

An examination of joint integrity and mobility should include both passive and active ROM (AROM), joint hypermobility/hypomobility, and soft-tissue changes (swelling, inflammation, or restriction). The shoulder and wrist should be examined closely because joint malalignment problems are common. Edema of the wrist often produces malaligned carpal bones with resulting impingement during wrist extension. Problems with spasticity may result in inconsistent ROM findings, because fluctuations in tone may occur from one testing session to the next. Thus tonal abnormalities should be noted at the time of examination. AROM tests are invalid for the patient in early or middle recovery when paresis, tonal changes, or obligatory synergies influence performance and preclude the isolated movements required in AROM tests. ROM limitations, developing contracture, and pain should be carefully documented. The therapist needs to carefully identify the nature of the pain and how the pain relates to joint movement and limitation (see Chapter 6).

Tone/Reflexes

For the patient in early and middle recovery, examination of tone and reflexes is essential. Passive motion testing can be used to determine hypotonicity, or spasticity. Patterns of spastic muscles are identified (see Table 8.1). Severity of spasticity can be graded on the basis of resistance to passive stretch using the *modified Ashworth Scale (mAS)*.[142] The position of the affected limbs at rest (resting postures) and during voluntary movements should be observed for tonal influences. An examination of stretch reflexes (hyperreflexia) and pathological reflexes (e.g., Babinski, tonic reflex activity, associated reactions) should also be performed.

Voluntary Movement Patterns

Voluntary movement patterns should be examined for control. Abnormal, obligatory synergies can be expected to dominate performance during early recovery. The therapist must base the examination of synergy dominance on knowledge of the typical components of the synergies (see Table 18.5). It is possible for one limb to vary significantly from the other (e.g., the UE may demonstrate more synergistic dominance than the LE). Synergistic dominance versus isolated joint control may also vary within a limb (e.g., the shoulder may demonstrate more isolated control than the wrist and hand).

During later recovery, movements demonstrate isolated joint control and appear more normal in the absence of spasticity and synergy restrictions. Coordination tests can

be used to examine control. The therapist focuses on elements of speed/rate control, steadiness, response orientation, and reaction and movement times. Fine motor control and dexterity should be examined using writing, dressing, and feeding tasks (see Chapter 7). While more significant impairments can be expected on the hemiparetic side, it is important to remember that subtle deficits can occur on the less involved side. Thus, it is important to examine both unilateral and bilateral movements, including symmetrical, asymmetrical, unrelated movements. Performance may vary as the patient moves from supine to sitting to standing positions with the resultant increased postural demands and greater degrees of freedom. Slower than normal movements (bradykinesia), or abnormal involuntary movements (chorea, hemiballismus) may occur with lesions affecting the basal ganglia and should be carefully examined.

Strength

Although an examination of strength is necessary, the traditional manual muscle test may pose problems of validity in the presence of strong spasticity, reflex, and synergy dominance. The patient is often not able to move into the required standard positions or to isolate specific joint actions. In this situation, an estimation of strength can be made from observation of active movements during functional activities (functional strength testing).[143] The patient's self-report can also yield important indicators of weakness and fatigue. The patient in later recovery with improving motor control can be examined using more traditional strength tests and measures (e.g., manual muscle testing [MMT], handheld or isokinetic dynamometry) (see Chapter 6). These can provide accurate information on residual impairments in strength, power, and endurance.

Postural Control and Balance

Postural control and balance should be examined in a variety of postures, especially sitting and standing. The patient's ability to maintain a position (steadiness) as well as postural alignment and position (symmetry) within the base of support are determined. Common asymmetries assumed after stroke include increased weightbearing on the stronger side. Dynamic stability control can be examined by having the patient move within a given posture (weight shift) within his or her limits of stability. The patient should be encouraged to shift weight in all directions, especially to the paretic side where impairments are expected. Functional tasks that utilize moving from one posture to another (e.g., supine-to-sit, sit-to-stand) can also be used to examine dynamic postural control. Both reactive postural control (response to perturbations) and anticipatory postural control (response to voluntary extremity movements) should be examined.[144,145]

Performance-based balance tests and measures can be used to determine balance function following stroke. Stroke specific tests are discussed here while generic tests are discussed in Chapter 8. These include:

- The *Berg Balance Scale (BBS)* was initially developed for use with the acute stroke patient and is now in widespread use. It includes 14 functional tasks that are scored using a 5-point ordinal scale. It examines unsupported sitting and standing, transfers, functional reach, picking objects off the floor, turning, single leg stance and stepping. Descriptive criteria are provided for each scoring level: a score of 4 indicates independent function while a score of 0 indicates unable to perform. A maximum score of 56 points is possible. Both intrarater and interrater reliability are high (r = 0.95).[146,147]
- Balance subscale of the *Fugl-Meyer Test (FM-B)*, a subtest of the Fugl-Meyer Assessment of Physical Performance that was developed for use with the acute stroke patient. It includes items of unsupported sitting, standing (with and without support), parachute reactions to both sides, and single limb stance both sides. It is scored using a 3-point ordinal scale (see Appendix A).[148]
- *Postural Assessment Scale for Stroke Patients (PASS)* was developed to examine the postural abilities of the acute stroke patient. It includes 12 items that examine sitting and standing without support, standing on the paretic LE, and changing posture (supine-to-affected side, supine-to-unaffected side, supine-to-sitting, sitting-to-standing, and standing picking a pencil off the floor). It is scored using an ordinal scale with descriptors ranging from cannot perform to perform with little help, to perform without help. It demonstrates good construct validity and high interrater and intrarater reliability (0.88 and 0.72, respectively).[149]

Other performance-based tests and measures (described in Chapter 8) include: *Functional Reach Test (FRT)*[150]; *Performance-Oriented Mobility Assessment—Tinetti (POMA)*[151]; *Timed Up and Go Test*[152]; *Clinical Test of Sensory Interaction and Balance (CTSIB)*[153]; and Dynamic Posturography: *Limits of Stability (LOS) Test*®.[154]

Ambulation and Functional Mobility

Gait is altered following stroke owing to a number of factors. Some of the more common problems in hemiplegic gait and their possible causes are summarized in Box 18.4. An examination of gait typically includes an observational gait analysis (OGA). The therapist examines the movements occurring at the ankle, foot, knee, hip, pelvis, and trunk during walking (kinematic gait analysis). Gait is observed from the different planes of motion and deviations are identified. Videotaping an OGA improves identification of gait deviations, provides a visual record of performance, and offers a useful teaching tool in assisting the patient in remediation of gait problems. Quantitative measures of distance and time, cadence, velocity, and stride times should also be obtained using measured walkways and a stopwatch. Kinetic gait analysis involves the forces

Box 18.4 Gait Deviations Commonly Seen Following Stroke

Stance Phase

Trunk/pelvis

Unawareness of affected side: poor proprioception

Forward trunk:

- Weak hip extension
- Flexion contracture

Hip

Poor hip position (typically adduction or flexion): poor proprioception

Trendelenburg limp: weak abductors

Scissoring: spastic adductors

Knee

Flexion during forward progression:

- Flexion contracture
- Weak hip and knee extensors
- Poor proprioception
- Ankle dorsiflexion range past neutral
- Weakness in extension pattern or in selective motion of hip and knee extensors and plantarflexors

Hyperextension during forward progression:

- Plantarflexion contracture past 90°
- Impaired proprioception: knee wobbles or snaps back into recurvatum
- Severe spasticity in quadriceps
- Weak knee extensors: compensatory locking of knee in hyperextension

Ankle/foot

Equinus gait (heel does not touch the ground): spasticity or contractures of gastrocnemius soleus

Varus foot (patient bears weight on the lateral surface of the foot): hyperactive or spastic anterior tibialis, post tibialis, toe flexors, and soleus

Unequal step lengths: hammer toes caused by spastic toe flexors prevent the patient from stepping forward onto the opposite foot because of pain/weightbearing on flexed toes

Lack of dorsiflexion range on the affected side (approximately 10° is needed)

Swing Phase

Trunk/pelvis

Insufficient forward pelvic rotation (pelvic retraction): weak abdominal muscles

Inclination to sound side for foot clearance: weakness of flexor muscles

Hip

Inadequate flexion:

- Weak hip flexors
- Poor proprioception
- Spastic quadriceps
- Abdominal weakness (hip hikers)
- Hip abductor weakness of opposite site

Abnormal substitutions include circumduction, external rotation/adduction, backward leaning of trunk/dragging toes; momentum/uncontrolled swing

Exaggerated hip flexion: strong flexor synergy

Knee

Inadequate knee flexion:

- Inadequate hip flexion and poor foot clearance
- Spastic quadriceps

Exaggerated but delayed knee flexion: strong flexor synergy

Inadequate knee extension at weight acceptance

- Spastic hamstrings
- Sustained total flexor pattern

Weak knee extensors or poor proprioception

Ankle/foot

Persistent equinus and/or equinovarus

- Plantarflexor contracture or spasticity
- Weak dorsiflexors
- Delayed contraction of dorsiflexors
- Toes drag during midswing

Varus: spastic anterior tibialis, weak peroneals, and toe extensors

Equinovarus: spasticity of post tibialis and/or gastrocnemius soleus

Exaggerated dorsiflexion: strong flexor synergy pattern

Adapted from educational materials used at Rancho Los Amigos Medical Center, Downey, CA, and Spaulding Rehabilitation Hospital, Boston, MA.

involved in gait and requires sophisticated equipment (force plates) to obtain data.[155–157] Stroke specific tests are discussed here while generic tests are discussed in Chapter 10.

Performance-based gait tests can be used to determine gait function following stroke. These include:

- *The 10-Meter Walk Test*: Gait speed is timed using a stopwatch to determine velocity.[158,159]
- The 6-Minute Walk Test: Walking distance is recorded in a specified 6-minute time interval to determine functional gait endurance.[160–163] Shorter distances, e.g., a 2-Minute Walk Test,[163] have been used for patients with acute stroke.
- *Energy expenditure*: Oxygen consumed is measured during a 5-Minute Walk Test.
- *Emory Functional Ambulation Profile (EFAP)*: a walking test that examines walking ability in the patient with stroke[164]; a modified version is also available (mFAP).[165] It includes timed tasks performed over different environmental terrains (hard floor, carpeted floor, obstacle course) as well as items of rising from a chair, timed "up & go", and stair climbing. High interrater and intrarater reliability is reported (ICC = 0.99 and ICC = 0.998, respectively).
- *Walkie-Talkie Test*: determines the ability to divide attention while walking (i.e., holding a conversation while walking).

Box 18.5 Functional Walking Categories

Physiological Walker
- Walks for exercise only either at home or in parallel bars during physical therapy.

Household Walker

Limited household walker:
- Relies on walking to some extent for home activities.
- Requires assistance for some walking activities, uses a wheelchair, or is unable to perform others.

Unlimited household walker:
- Able to use walking for all household activities without any reliance on a wheelchair.
- Encounters difficulty with stairs and uneven terrain.
- May not be able to enter or leave the house independently.

Community Walker

Most-limited community walker:
- Can enter and leave the home independently.
- Can ascend and descend a curb independently.
- Can manage stairs to some degree.
- Independent in at least one moderate community activity (i.e., appointments, restaurants) and needs assistance or is unable in no more than one other low-challenge activity (i.e., church, neighborhood, visiting friend).

Least-limited community walker:
- Demonstrates independent stair management.
- Independent in all moderate community activities without assistance or use of wheelchair.
- Independent in either local stores or uncrowded shopping centers.
- Independent in at least two other moderate community activities.

Community walker:
- Independent in all home and community activities.
- Can accept crowds and uneven terrain.
- Demonstrates complete independence in shopping centers.

Note: Patients in each higher category can perform all activities of the previous group as well as the additional level of challenge listed.

From Perry, J, et al[166] p. 985, with permission.

Perry and co-workers[166] constructed a walking ability questionnaire and surveyed a group of 147 patients with chronic stroke about the effects of their limited walking ability. They then developed a classification of walking handicap after stroke (see Box 18.5). The use of functional categories (physiological walker, household walker, and community walker) provides a useful method of identifying customary level of walking at home and in the community. *Walking handicap*, defined as the social disadvantage as a result of limitations in walking ability, can also be identified. Factors that differentiated household from community ambulators included strength, proprioception, isolated knee control (flexion and extension), and velocity. The findings of this study can be used to improve communication among clinicians, treatment planning, and documentation.

Functional Status

Functional measures are used to determine the impact of impairments and the plan of care, monitor progress, ascertain efficacy of stroke rehabilitation efforts, and make recommendations for long-term care or placement. Instruments can include items to examine functional mobility skills (bed mobility, movement transitions, transfers, locomotion, stairs), basic ADL skills (feeding, hygiene, dressing), and instrumental ADL skills (communication, home chores). Information on functional disability following stroke is typically gained through performance-based measures. The *Barthel Index*[167] and its later form the *Functional Independence Measure (FIM)*[168] have been extensively tested and demonstrate excellent reliability, validity, and sensitivity. The FIM is now in widespread use in rehabilitation facilities across the United States. Higher FIM scores have been correlated to successful outcomes, discharge home, and return to the community for patients with stroke.[169] See Chapter 11 for a more detailed discussion of these instruments.

Disability-Specific Instruments

Fugl-Meyer Assessment of Physical Performance (FMA)

The pioneering work of Signe Brunnstrom[21,22] on motor recovery and motor behavior following stroke led to the development of the FMA.[24] This is an impairment-based test with items organized by sequential recovery stages. A three-point ordinal scale is used to measure impairments of volitional movement with grades ranging from 0 (item cannot be performed) to 2 (item can be fully performed). Specific descriptions for performance accompany individual test items. Subtests exist for UE function, LE function, balance, sensation, ROM, and pain. The cumulative test score for all components is 226 with availability of specific subtest scores (e.g., UE maximum score is 66, LE score 34; balance score 14). This instrument has good construct validity and high reliability ($r = 0.99$) for determining motor function and balance.[170] Quantifiable outcome data allow this instrument to be accurately used for research purposes (a gold standard) and document recovery over time. The instrument requires an estimated 30 to 40 minutes to administer (see Appendix A).

National Institutes of Health Stroke Scale (NIHSS)

National Institutes of Health (NIH) developed the NIHSS for initial and serial examination of impairments following

acute stroke.[171] The NIH NINDS t-PA Stroke Trial Study Group promotes it use for patients with cerebral infarction. It is an 11-item impairment-based test that uses a variable ordinal scale. Some items are scored 0–2 or 0–3 (level of consciousness, best gaze, visual fields, facial palsy, limb ataxia, sensory, best language, dysarthria, extinction, and inattention) other items items are scored 0–4 (motor arm and motor leg). Specific descriptors are attached to each score.[172] An exam scoring service for the NIHSS is maintained by the National Stroke Association.

Stroke Rehabilitation Assessment of Movement (STREAM)

The STREAM is a clinical measure of voluntary movements and basic mobility following stoke. It consists of 30 items (test movements) distributed equally among three subscales: upper-limb movements, lower-limb movements, and basic mobility items. Voluntary movement items explore out-of-synergy control and are scored using a 3-point ordinal scale (unable to perform, partial performance, complete performance). The basic mobility section includes a variety of items (rolling, bridging, sit-to-stand, standing, stepping, walking, and stairs) and is scored using a 4-point ordinal scale (unable, partial, complete/with aid, complete/no aid). The maximum STREAM score is 70 with each limb subscore worth 20 points and the functional mobility subscore worth 30 points.[173] The instrument has good construct validity and high reliability ($r = 0.99$).[174] It has been used to document motor recovery over time and predict discharge destination following stroke.[175]

Motor Assessment Scale (MAS)

The MAS was developed by Carr et al[176] to examine functional mobility skills following stroke. It uses a 6-point ordinal scale with descriptors for each item score. It includes eight items of motor function, including movement transitions (supine-to-sidelying, supine-to-sit, sit-to-stand), balanced sitting, walking, upper-arm function, hand movements, and advanced hand function. The ninth item is an impairment item examining muscle tone. This instrument has been shown to be highly reliable ($r = 0.89$ to .99) with high concurrent validity (0.88 correlated with the Fugl-Meyer Assessment).[177] It can be used to document motor recovery over time.

Goals and Outcomes

Examples of general goals and outcomes for patients with stroke (Preferred Practice Pattern 5D) as adapted from the *Guide to Physical Therapist Practice*[132] are presented in Box 18.6. These general goals will provide the basis for development of specific anticipated goals and expected outcomes for an individual patient.

Box 18.6 Examples of General Goals and Outcomes for Patients with Stroke, Adapted from the *Guide to Physical Therapist Practice*[132]

Impact of pathology/pathophysiology is reduced.
- Patient/client, family, and caregiver knowledge and awareness of the disease, prognosis, and plan of care is enhanced.
- Symptom management is enhanced.
- Changes associated with recovery are monitored.
- Risk of secondary impairments and reoccurrence of condition is reduced.
- Intensity of care is decreased.

Impact of impairments is reduced.
- Cognitive function is improved.
- Communication is improved.
- Sensory awareness and skin integrity are improved.
- Perceptual function is improved.
- Awareness and use of the hemiplegic side is improved.
- Pain is decreased.
- Joint integrity and mobility are improved.
- Motor function (motor control and motor learning) is improved.
- Muscle performance (strength, power, and endurance) is improved.
- Postural control and balance are improved.
- Gait and locomotion are improved.
- Aerobic capacity is increased.

Ability to perform physical actions, tasks, or activities is improved.
- Independence in ADL is increased.
- Tolerance of upright postures and activities is increased.
- Problem-solving and decision making skills are enhanced.
- Safety of patient/client, family, and caregivers is improved.

Disability associated with chronic illness is reduced.
- Ability to assume/resume self-care and home management is improved.
- Ability to assume work (job/school/play), community, and leisure roles is improved.
- Awareness and use of community resources are improved.

Health status and quality of life are improved.
- Sense of well-being is enhanced.
- Stressors are reduced.
- Insight, self-confidence, and self-management skills are improved.
- Health, wellness, and fitness are improved.

Patient/client satisfaction is enhanced.
- Access and availability of services are acceptable to patient/client and family.
- Quality of rehabilitation services is acceptable to patient/client and family.
- Care is coordinated with patient/client, family, caregivers, and other professionals.
- Discharge placement needs are determined.

Physical Therapy Interventions

Framework for Intervention

Neurorehabilitation approaches and therapeutic techniques for the patient with stroke have evolved over the years from a major emphasis on muscle reeducation initially developed for patients with poliomyelitis in the 1940s (e.g., Kenny method) to the neurophysiological/neurodevelopmental approaches first popularized in the 1950s and 1960s. These include *Neurodevelopmental Treatment (NDT),*[23,56] *Movement Therapy in Hemiplegia—Brunnstrom Approach,*[22] *Proprioceptive Neuromuscular Facilitation (PNF),*[178,179] and sensory stimulation techniques. Of these, NDT and PNF remain in popular use today. Currently there is increased emphasis on functional/task specific training using intense practice of functional tasks along with behavioral shaping and environmental enrichment (e.g., constraint-induced movement therapy [CIMT] for the paretic UE or locomotor training using *body weight support and treadmill training [BWSTT]*). Compensatory training strategies are also used in some circumstances to promote resumption of function using the less involved extremities. These are indicated for patients who demonstrate severe motor impairment and limited recovery. Motor learning strategies provide a common base for all functional training. These are discussed fully in Chapter 13.

Evidence-Based Practice

There is increasing emphasis on evidence-based practice (EBP) that promotes the use of current best research evidence along with individual clinical expertise in order to reach informed decisions about patient care.[180] EBP allows therapists to identify the best (most effective) techniques and to take responsibility for evaluating their practice on an ongoing basis. Early attempts to validate the success neurodevelopmental/facilitation approaches for the patient with stroke failed to provide convincing evidence. Studies designed to delineate differences between conventional exercise approaches (ROM, functional training) and neurodevelopmental/facilitation approaches failed to demonstrate significant advantages of one approach over another.[181–187] Patients improved regardless of the type of intervention, thus providing evidence about the value of physical therapy as a whole. It should be pointed out that many of these early studies were subject to serious methodological flaws.[188] For example, studies used small sample sizes, failed to include a control group or to control for experimenter bias and co-interventions, and/or utilized poorly defined treatments and/or inappropriate outcome measures. Randomized controlled trials (RCT) that utilize both experimental and control groups and larger sample sizes offer the clinician a higher level of confidence in study findings. Several recent RCTs evaluated the effectiveness of neurodevelopmental/facilitation approaches versus other therapeutic approaches. These are summarized in Box 18.7. Evidence concerning the effectiveness of task-oriented training is presented in Evidence Summary Box 13.1 (Post-Stroke Constraint-Induced Movement Therapy) and Evidence Summary Box 18.8 (Post-Stroke Locomotor Training Using Body Weight Support and Treadmill Training).

Important conclusions to be drawn from these studies are that: (1) collectively they provide consistent evidence for the beneficial effects of physical therapy; (2) studies investigating neurodevelopmental/facilitation approaches have failed to consistently demonstrate superiority over other approaches; and (3) studies on task-oriented training have yielded positive results in terms of improving locomotor function (post-stroke locomotor training studies) and UE function (CIMT training studies). Specificity of training and increased intensity of training are important factors in the positive results of this latter group. It is clear that additional large, multicenter RCT studies (Level I studies) are needed. Finally, it is important to point out that there is no one intervention optimal for all patients with stroke. Because patients with stroke are a diverse group with variable levels of function, interventions must be carefully selected based on individual abilities. Therapists need to select interventions that have the greatest chance of successfully remediating existing impairments and promoting functional recovery. The choice of interventions must also take into consideration a number of other factors, including phase of post-stroke recovery (acute, post-acute, chronic), age of the patient, number of comorbidities, social and financial resources, and potential discharge placement. Early emphasis on improving functional independence provides an important source of motivation for both the patient and family.

Strategies to Improve Sensory Function

Patients who have significant sensory impairments may demonstrate impaired or absent spontaneous movement because of the lack of sensory inputs before and during movement. The more the patient can be encouraged to use the affected side, the greater the chance of increased awareness and function. Conversely, the patient who refuses to use the hemiplegic side contributes to the problems imposed by lack of sensorimotor experience. Without attention during treatment, this learned nonuse phenomenon can contribute to further deterioration.[202] Sensory stimulation is important for recovery. Training focuses on restoring sensitivity of the more affected extremities and requires some residual sensory function. The presentation of repeated sensory stimuli stimulates tactile, mechano- and muscle receptors. For example, stroking, stretch, superficial and deep pressure, and approximation can be used. The selection of sensory inputs should be directly related to a functional task and provided to those body

(text continues on page 740)

Evidence Summary Box 18.7

Post-Stroke Neurodevelopmental/Facilitation Training

Reference	Subjects	Design/Intervention	Duration	Results	Comments
Sunderland, KJ, et al[189] 1992	132 subjects Post-acute stage Mildly affected	Single-blind RCT Compared NDT approach (hands-on PT) with behavioral therapy/motor learning approach Outcome measures: Extended Motricity Index; Motor Club Assessment; PROM; pain; Frenchay Arm Test	4 weeks inpatient therapy and 6 weeks outpatient therapy	At 6 months, behavioral group demonstrated ↑ strength, ROM, and speed of movement No differences in attainment of functional skills	Examinations at 1, 3, and 6 months after stroke Large sample size
Feys, HM, et al[190] 1998	100 subjects post-acute stage, ischemic strokes	Single-blind RCT Exp Gr: received movement training, sensory stimulation, RIP Contr Gr: sham shock wave therapy Outcome measures: FMA Action Research Arm Test BI	5 × week for 6 weeks	No significant difference: improvement noted in both groups Exp Gr better scores on FMA at 6 months No difference on functional tests	Examinations at 1 and 6 weeks, 6 and 12 months Good sample size
Langhammer, B, and Stanghelle, J[191] 2000	61 subjects, acute stage	Double-blind RCT Compared NDT approach with motor learning approach Outcome measures: MAS; BI Nottingham Health Profile Sodring Motor Evaluation Scale	5 × week, 40-minute sessions	Motor learning group had decreased length of stay and improved functional outcome	Examinations at 2 weeks and 3 months Good sample size
Mudie, M, et al[192] 2002	40 subjects	Double-blind RCT Used a reaching task Compared 3 groups: (1) visual feedback group (monitor) (2) NDT group (ROM, tone normalization, balance training); (3) task-training group (functional training) Outcome measures: Weight distribution in sitting and standing; BI	5 × week for 2 weeks	NDT group: improved sitting symmetry Feedback and task training groups: improved sitting symmetry at 12 weeks Task-training group: improved functional gains	Examinations at 2 and 12 weeks Small sample size Outcomes consistent with specificity of training
Basmajian, J, et al[193] 1987	29 subjects post-acute stage	RCT Compared 2 groups receiving behavioral/biofeedback training with NDT training Outcome measures: UE Function Test; Health Belief Survey; Beck's Depression Inventory	3 × week for 5 weeks	Both groups improved; no differences found	Small sample size

BI = Barthel Index; Contr Gr = control group; Exp Gr = experimental group; FMA = Fugl-Meyer Assessment of Physical Performance; MAS = Motor Assessment Scale; PT = physical therapy; RCT = randomized controlled trial; RIP = reflex inhibiting postures; UE = upper extremity.

Evidence Summary Box 18.8

Post-Stroke Locomotor Training Using Body Weight Support and Motorized Treadmill Training

Reference	Subjects	Design/Intervention	Duration	Results	Comments
Visintin, M, et al[101] 1998	100 subjects postacute phase	RCT Treadmill gait training:compared BWS with no-BWS Outcome measures: balance (BBS): motor recovery (STREAM); OG walking speed and endurance	4× week for 6 weeks	BWS group demonstrated significant improvement over no-BWS in functional balance (BBS), motor recovery, OG walking speed and endurance	79% progressed to full weightbearing OG walking; improvements were sustained Good sample size
Hesse, S, et al[194] 1994	9 subjects postacute phase	Nonrandomized cohort design Outcome measures: FAC, Standing Balance Test, Rivermead Motor Assessment, Motricity Index, mAS	15 min/session, 30 min/day, 5×/wk for 5 weeks	Gait capacity improved in all subjects (gait velocity by 3 times, cadence and stride length by 2 times); all but 1 subject progressed to OG walking	No control group; fails to account for recovery and effect of regular ongoing PT Small sample size
Hesse, S, et al[195] 1995	7 subjects, MCA stroke at least 3 mo post-stroke	Case series design: A-B-A Compared TT with BWS with PT based on Bobath approach Outcome measures: FAC, Rivermead Motor Assessment, Motricity Index, mAS	Each session (A-B-A) lasted 3 weeks, 5×/week, 30 min daily	Training using BWS and a treadmill was more effective in improving gait ability and walking velocity	Weekly examinations 5 patients demonstrated unilateral neglect and 3 patients demonstrated pusher syndrome Small sample size
Malouin, F, et al[196] 1992	10 subjects MCA stroke acute stroke (7–14 days post-stroke)	Nonrandomized cohort design Intervention program: early standing (tilt table); weight shifting exercises (limb-load monitor), TT, Kinetron Outcome measures: BI, FMA, treadmill velocity	2× day, 5×/week for 5 weeks	Both treadmill velocity and duration increased	Intensive and graded locomotor training activities were well tolerated Small sample size No control group Double the therapy time of normal
Richards, C, et al[197] 1993	27 subjects	RCT Compared early task-based PT (standing, weightshifting and Kinetron exercises; TT), 1.74 hours/day with conventional PT intervention groups (1.79 hours/day and 0.73 hours/day) Outcome measures: FMA, BI, BBS, gait velocity at 6-month followup	Daily	Significant improvement in gait velocity for task-based therapy group	Small sample size

(continued)

Evidence Summary Box 18.8

Post-Stroke Locomotor Training Using Body Weight Support and Motorized Treadmill Training (continued)

Reference	Subjects	Design/Intervention	Duration	Results	Comments
Sullivan, K, Knowlton, B, and Dobkin, B[198] 2002	24 subjects, chronic stroke	Nonrandomized cohort design Intervention program: walking using BWS and a treadmill, 2 groups of varying speeds (slow speeds 0.22 m/sec; variable speeds 0.22 m/sec to 0.89 m/sec; fast speeds 0.89) Outcome measures: FMA 10-m walk: Self-selected walking speed OG	12 sessions, 20-min duration, over 4–5 weeks	Training at fast speeds was more effective at improving speeds of OG walking than training at slow or variable speeds.	No control group Gains maintained at 3 months Small sample size
Smith, G, et al[199] 1999	14 subjects chronic stroke	Nonrandomized cohort design Intervention program: low-intensity walking using BWS and a treadmill Outcome measures: Dynamometer—reflexive and volitional torque	3× week for 3 months	Training improved volitional torque for both concentric and eccentric contractions	No control group Lacked functional outcome measures Small sample size
Nilsson, L, et al[200] 2001	60 subjects post-acute stage	RCT Compared walking using BWS and a treadmill with OG walking training (motor relearning approach) Outcome measures: FIM FMA FAC 10-m walk test BBS	30 minute/day, 5× week for 2 months	Both groups improved on function (FIM, FAC), balance (BBS), and walking speed	10-month followup Good sample size
Laufer, Y, et al[201] 2001	25 subjects postacute stage	Nonrandomized cohort design Compared walking using BWS and a treadmill wih OG walking Outcome measures: FAC Speed (10-m walk test) stride length (foot switch) EMG activity	3 weeks	Training using BWS and a treadmill improved gait function, stride length and stance on paretic limb	No control group Small sample size

BBS = Berg Balance Scale; BI = Barthel Index; BWS = Body Weight Support using an overhead harness; FAC = Functional Ambulation Category; FIM = Functional Independence Measure; FMA = Fugl-Meyer Assessment of Physical Performance; mAS = Modified Ashworth Scale; MCA = Middle cerebral artery syndrome; OG = overground; PT = physical therapy; RCT = randomized controlled trial; STREAM = Stroke Rehabilitation Assessment of Movement; TT = treadmill training.

surfaces directly used in the task. For example, UE tasks can include stroking the hand with different textured fabrics, pressing objects into the hand (coin, button, key), or drawing shapes/letters/numbers on the skin. During functional training, approximation can be provided to extended UE during weightbearing (e.g., in sitting or standing/modified plantigrade position). Approximation can be added to LE tasks such as standing and stepping. Stimulation should be of sufficient intensity to engage the system but not so strong as to produce adverse effects (e.g., withdrawal). During stimulation, the patient's attention should be focused directly on the stimulation and task. Initial attempts are with

eyes closed (EC). If the patient fails to recognize the stimulus, the patient can be allowed to look with eyes open (EO) while the stimulus is made more intense. The therapist needs to provide feedback and encouragement in order to direct the patient's attention and shape the patient's responses.[203,204] Johnstone suggests using inflatable pressure splints to provide additional sensory stimulation (deep pressure, muscle, and joint sensations) during functional training. In more severe cases, she also recommends a program of intermittent pressure therapy to provide alternating stimulation of a limb and overcome the problems of sensory accommodation.[205,206]

A safety education program should be instituted early for patients, family, and caregivers to improve awareness of sensory impairments and ensure protection of anesthetic limbs. This is particularly important for preventing UE trauma during transfer and wheelchair activities.

Patients with hemianopsia or unilateral neglect demonstrate a lack of awareness of the contralesional side. The impairments are more pervasive in patients with neglect and in its most severe form (anosognosia) may extend to a totally unawareness of the disability or the extent of the problems. These patients benefit from training strategies that encourage awareness and use of the environment on the hemiparetic side and use of the hemiparetic extremities. It is important to teach active visual scanning movements through turning of the head and axial trunk rotation to the more involved side. Cueing (e.g., visual, verbal, or motor cues) is used to direct the patient's attention. For example, a red anchor line can be taped on the floor and the patient directed to visually follow the line from one side to the other. Or a red ribbon can be attached to the patient's hemiparetic wrist and the patient directed to keep the red ribbon in sight. Scanning movements can also be stimulated using visual tracking tasks using a computer. Patients are given feedback about the success of their efforts and reinforcement for each successful performance (shaping). Imagery has also been shown to help (e.g., "imagine you are a lighthouse beam; use your beam to sweep and scan the floor from one side to the other"). During therapy, the therapist stimulates and encourages active voluntary movements of the hemiparetic limbs while encouraging the patient to look at his or her limbs while moving. UE exercises that involve crossing the midline toward the hemiparetic side (e.g., reaching activities or PNF chop or lift patterns) are important. Functional activities that encourage bilateral interaction are also valuable (e.g., pouring a drink and drinking from a cup; picking up an object with the more involved hand and placing it in the other; "dusting a tabletop" with a cloth held by both hands). The therapist needs to maximize the patient's attention by providing visual, tactile, or proprioceptive stimuli on the more affected side. These can include stroking, brushing, icing, or vibrating the hemiparetic limbs. The therapist also needs to consistently reorient the patient as inattention develops. Patients with very low levels of arousal are likely to be less responsive to therapy efforts.[207–209]

Strategies to Improve Motor Function

Strategies to Improve Flexibility and Joint Integrity

Soft tissue/joint mobilization and ROM exercises are initiated early to maintain joint integrity and mobility and prevent contractures. AROM and passive ROM (PROM) with terminal stretch should be performed daily in all motions. If a contracture is developing, more frequent ROM (twice daily or more) is necessary and sustained low-load stretching can be considered.

Positioning strategies are also important in maintaining soft tissue length. Effective positioning of the hemiparetic extremities encourages proper joint alignment while positioning the limbs out of the abnormal postures typically assumed. The use of protective devices such as resting splints may be necessary. Coordination with family and caregivers is essential for long-term management (Box 18.9).

In the UE, correct PROM techniques require careful attention to external rotation and distraction of the humerus, especially as ranges approach 90° of flexion or more. The scapula should be mobilized on the thoracic wall with an emphasis on upward rotation and protraction to prevent soft tissue impingement in the subacromial space during overhead movements of the arm (Fig. 18.6) and to prepare for forward reach patterns. The use of overhead pulleys for self-ROM is generally contraindicated because of failure to achieve the above requirements for scapulohumeral movement. Full extension of the elbow is important because the majority of patients with stroke develop tightness in elbow flexors as a result of excess flexor spasticity. Normal length of wrist and finger extensors should also be maintained as tightness is typical in flexion. This can be achieved functionally through sitting, weightbearing on the extended paretic UE with the wrist extended and fingers open and extended. Edema and tonal changes may produce impingement with wrist extension. In this situation, the carpal bones should be mobilized prior to stretching at the wrist. Strategies to teach patients safe self-ROM techniques should be instituted early. Suggested activities include:

- *Arm cradling*: The more affected UE cradles and lifts the less affected UE to 90°; the arm is moved into positions of horizontal abduction and adduction. Active trunk rotation is combined with the arm movements.
- *Table-top polishing*: The affected extremity is positioned in humeral flexion with scapular protraction and elbow extension; both hands are positioned on a towel. The less affected hand moves the paretic hand by pulling on the towel (forward, and side-to-side). Trunk movements and ROM are optimized by placing the chair slightly back from the table.

Box 18.9 Common Malalignments Following Stroke and Positioning Strategies

Common Malalignments

Pelvis/trunk. An asymmetric pelvic position is assumed with more weight borne on the ischial tuberosity on the sound side. This results in lateral flexion of the trunk with the head shifted toward the affected side. A posterior pelvic tilt is common. This results in sacral sitting with a flattened lumbar curve and an exaggerated thoracic curve (kyphosis) and forward head.

Scapula. A position of scapular downward rotation is assumed. Scapular instability (winging) may also be present, especially in weightbearing postures.

Glenohumeral joint. Lateral flexion of the trunk and downward rotation of the scapula result in depression and subluxation.

Upper extremity (UE). The limb is typically held in internal rotation and adduction with elbow flexion, forearm pronation, wrist flexion and ulnar deviation, and finger flexion.

Lower extremity (LE). In standing, a position of pelvic retraction and elevation is assumed, with hip and knee extension and hip adduction/internal rotation (i.e., a scissoring position); in sitting the hip and knee are flexed with hip abduction and external rotation (i.e., a flexor synergy pattern). Ankle plantarflexion is common to both.

Positioning Strategies

Lying in the supine position.
Head/neck: neutral and symmetrical; supported on pillow.
Trunk: aligned in midline.
Affected UE: scapular protracted, shoulder forward; arm supported on a pillow; elbow extended with hand resting on a pillow; wrist neutral, fingers extended, and thumb abducted.
Affected LE: hip forward (pelvis protracted); knee on a small towel roll to prevent hyperextension; nothing against the soles of feet. For persistent plantarflexion, a splint can be used to position the foot and ankle in neutral position.

Sidelying on the nonhemiplegic side.
Head/neck: neutral and symmetrical.
Trunk: aligned in midline; small pillow or towel can be placed under the rib cage to elongate the hemiplegic side.
Affected UE: scapular protracted, shoulder forward; arm on a supporting pillow: elbow extended, wrist neutral, fingers extended, and thumb abducted.
Affected LE: hip and knee flexed, supported on a pillow.

Sidelying on the hemiplegic side.
Head/neck: neutral and symmetrical.
Trunk: aligned in midline.
Affected UE: scapular protracted; shoulder forward; arm placed in slight abduction and external rotation; elbow extended, forearm supinated, wrist neutral, fingers extended, and thumb abducted.
Affected LE: aligned with hip extended and knee flexed. An alternate position is slight hip and knee flexion with pelvic protraction.

Sitting in an armchair or wheelchair.
Head/neck: neutral and symmetrical; head directly above pelvis.
Trunk: spine extension, aligned in midline; equal weightbearing on both buttocks.
Affected UE: shoulder protracted and forward; elbow supported on an arm trough or lapboard; wrist neutral, fingers extended, and thumb abducted.
Both LEs: hips flexed to 90°, positioned in neutral with respect to rotation.

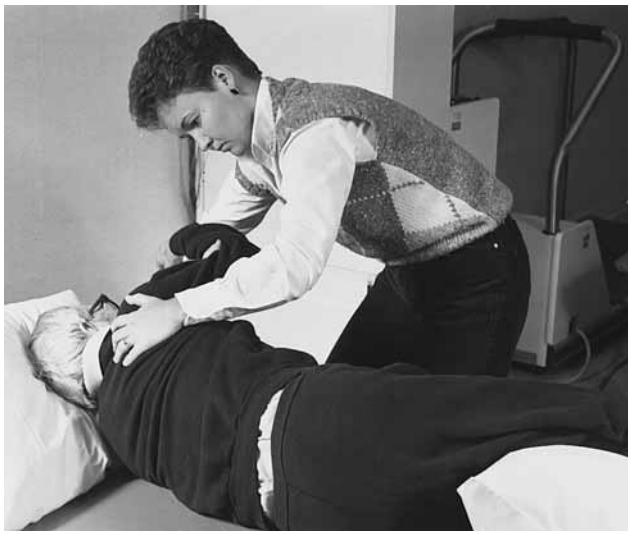

Figure 18.6 Range of motion exercises for the more affected upper extremity. The therapist carefully mobilizes the scapula during arm elevation.

- Sitting, the patient leans forward and reaches both hands down to the floor. This position encourages forward flexion of the humerus with scapular protraction, and extension of the elbow, wrist, and fingers.
- Supine, hands are clasped together and placed behind the head, the elbows fall flat to the mat. This activity should be considered only if scapula upward mobility is present. Hands clasped, self-overhead movements are contraindicated if scapulohumeral rhythm is lacking.

While sitting in a wheelchair, the patient's paretic UE can be positioned on a lap tray or on an arm trough (shallow elbow/forearm support) attached to the armrest. The shoulder is positioned in 5° of abduction and flexion and neutral rotation; elbow in 90° flexion and slightly forward; forearm pronated; and hand in a functional resting position. Splinting the hand can also be considered. A volar resting (pan) splint is commonly used. The forearm, wrist, and fingers are positioned in a functional position (20 to 30° of wrist extension, MP flexion 40 to 45°, IP flexion

10 to 20°, and thumb opposition). A resting splint is more appropriate for nighttime use than daytime when spontaneous function is desired. In the presence of spasticity, tone-reducing devices can be considered (e.g., finger abduction splint, firm cone, spasticity reduction splint, or inflatable pressure splint).

As most patients regain some use of their LEs early in recovery, ROM techniques focus on individual patient needs with attention to several common areas of impairment. For many patients, voluntary movement in the foot and ankle is limited owing to plantarflexor spasticity. Weight-shifting activities in modified plantigrade (forward shift stretches the plantarflexors) or prolonged static positioning using adaptive equipment (i.e., tilt table with toe wedges) can be used to gain range. Facilitation of active contraction of dorsiflexors can also be combined with stretching to provide reciprocal inhibition to plantarflexors. If synergistic influence is strong, the patient can be effectively positioned while supine on a mat with the paretic LE abducted off the side with knee flexed and foot flat on the floor or a stool. This position of hip abduction and extension with knee flexion serves to break-up synergistic dominance and position the limb out of the typical spastic scissoring posture. If the patient spends considerable time sitting in a wheelchair, care should be taken to stretch the hip flexors. If hip flexor contractures are allowed to develop, they can lead to increased difficulty with standing, transfers, and ambulation.

Strategies to Improve Strength

Muscle weakness is a major impairment following stroke. *Active restraint* arising from spastic antagonist muscles has been suggested as a negative influence on movement and a cause of agonist weakness. Bobath,[23] whose theories formed the basis of *Neurodevelopmental Treatment (NDT)*, advocated that strengthening techniques for agonists and excessive effort should be avoided in favor of inhibition techniques designed to reduce spasticity in spastic muscles. This conclusion has been challenged by a number of investigators who have demonstrated that the problems clearly lie with inadequate motor unit recruitment and paresis of agonists.[32,42,43] Patients with stroke can undergo graded strength training without any detrimental increase in spasticity.[210,211] Significant improvements in strength have been demonstrated.[123,210–217] Evidence of carryover effects to improved function is less convincing. Some studies have indicated improvements in function[123,210,215–217] while other studies have failed to demonstrate significant carryover to improved function.[212,214,218,219] Specificity of training may well explain the lack of significant transfer to functional tasks. For excellent comprehensive reviews on this subject the reader is referred to the work of Eng[32] and Morris et al.[79]

Exercise modalities for strengthening include free weights, elastic bands or tubing, and machines (PRE, isokinetics). For patients who are very weak (<3/5), gravity-eliminated exercises using powder boards, sling suspension, or aquatic exercise is indicated. The therapist may need to assist initial movement attempts using active-assisted or facilitated movements (stretch and tracking resistance). Gravity-resisted active movements are indicated for patients who demonstrate 3/5 strength (e.g., arm lifts, leg lifts). Patients who demonstrate adequate strength in gravity-resisted exercise (e.g., 8 to 12 reps) can be progressed to exercise using added resistance (e.g., free weights, bands, or machines). Combining resistance training with functional activities provides additional benefits in terms of improving function (e.g., step-ups or stair climbing while the patient is wearing weighted ankle cuffs). Lifting free weights or using elastic bands places added demands for postural stability and is an important element of training to improve postural control. Many patients with stroke demonstrate poor hand function with no effective grasp. Specially designed gloves may be necessary to ensure maintained contact with exercise equipment (e.g., leather mitts with Velcro®, wrist cuffs). Patients with impaired sensation are at increased risk for injury and should be monitored closely.[91]

In determining a safe exercise prescription, it is important to remember the high incidence of hypertension and cardiac disease in patients with stroke. Exercise is contraindicated in patients with recent stroke and unstable blood pressure (BP). High-intensity strengthening exercises (sustained maximal effort) is generally contraindicated. Isometric exercise accompanied by the Valsalva maneuver and dangerous elevations in BP is also contraindicated. Concentric or eccentric exercises can be used; eccentric exercises will produce less cardiovascular stress than concentric.[220] Dynamic exercises performed in an upright position (sitting) produce less elevations in BP than recumbent/supine exercises. For patients at risk, submaximal protocols using low-intensity exercises (e.g., 30 to 50 percent of maximal voluntary contraction) are appropriate for initial exercise. Varying the exercises (e.g., bench press, leg press, hamstring curl, shoulder press, triceps push-down, biceps curl) is also an effective strategy to reduce risks. The therapist needs to ensure that warm-ups and cool-downs are adequate and the overall exercise progression is gradual.

Careful monitoring of exercise is essential. For patients at risk, BP, heart rate (HR), and ratings of perceived exertion (RPE) should be taken after each exercise set initially. As exercise progresses, less frequent monitoring can be implemented. The therapist also needs to monitor breathing rate and form, ensuring breath holding and Valsalva do not occur. Patients should be instructed in how to measure their own HR and RPE. They should also be taught the warning signs for when to stop exercising. These include:[91]

- Lightheadedness or dizziness.
- Chest heaviness, pain, or tightness; angina.
- Palpitations or irregular heart beat.

- Sudden shortness of breath not due to increased activity.
- Discomfort or stiffness in muscles persisting for several days after exercise.

Patients who are on medications that limit cardiac output (e.g. beta-blockers) will demonstrate reduced heart rate responses and lower peak heart rates. Patients taking diuretics to reduce fluid volume may demonstrate altered electrolyte balance with resulting dysrhythmias. Patients taking vasodilators may require a longer cool-down period after exercise to prevent post-exercise hypotension.[90]

Patients with stroke and older adults who have been immobilized for long periods of time demonstrate increased risk of muscle injury with exercise. Heavy-resistance strength training is therefore contraindicated. The risk of muscle injury is greatest with eccentric exercise.[221] In order to reduce this risk, the therapist needs to begin training with low-intensity exercises and gradually increase intensity to patient tolerance. It is important to ensure adequate rest periods using distributed practice to start, and provide a variety of exercises. Careful monitoring of fatigue and muscle soreness is necessary.[32] Episodes of prolonged or unusual fatigue and delayed-onset muscle soreness (DOMS, 24 to 48 hours after exercise) should be avoided. Progression to higher training levels should proceed only in the absence of DOMS and fatigue.

Strategies to Manage Spasticity

Patients who demonstrate spasticity benefit from interventions designed to modify or reduce tone. These include early mobilization combined with elongation of spastic muscles and sustained stretch through positioning. The technique of rhythmic rotation (a passive manual technique) can be effective in gaining initial range. The therapist slowly moves the limb into the lengthened range while gently rotating it back and forth. Once full range is achieved, the limb is positioned in the lengthened position. For example, the shoulder is extended, abducted, and externally rotated with the elbow, wrist, and fingers extended and positioned in weightbearing (see Fig. 18.7). The patient then needs to maintain the position for an extended time (e.g., 5 to 10 minutes of sustained stretching). Additional inhibitory effects are attained from prolonged pressure on the long flexor tendons of the hand. Slow rocking movements (rocking the body over the elongated limb) also increase the inhibitory effects through adding influences of slow vestibular stimulation. Spasticity in the quadriceps can be similarly inhibited through prolonged pressure and weightbearing in kneeling or quadruped positions (Fig. 18.8). A reduction in truncal tone can be promoted using techniques of rhythmic rotation or rhythmic initiation combined with axial trunk rotation (e.g., in sidelying, sitting or hooklying, segmental trunk rotation). PNF upper trunk patterns (chopping or lifting) that emphasize rotational movements of the trunk can also be effective in maintaining reduced trunk tone.[179] Sidesitting on the hemiparetic side provides sustained stretch to the spastic side flexors. The patient and family member or caregiver should be taught specific stretching techniques that focus on spastic muscles.

Figure 18.7 Sitting, with extended arm support. The patient is wearing a humeral cuff sling to prevent subluxation of the shoulder. The therapist assists in stabilizing the elbow and fingers in extension.

Training strategies should include activation of the antagonist muscles using slow and controlled movements. Local facilitation techniques can be added to activate very weak antagonist muscles and are effective in reducing agonist tone through the effects of reciprocal inhibition. Thus in the UE efforts are directed toward active contractions of the elbow extensors in the presence of flexor spasticity while in the LE efforts are directed toward active contractions of the knee flexors with extensor spasticity. Reciprocal relationships are not always within a normal range, however, particularly in the presence of strong spasticity and spastic co-contraction.[23] Additional modalities and/or splinting can be effective in reducing tone. Cold in the

Figure 18.8 Use of a pressure splint applied to support the elbow in extension during weightbearing in the quadruped position. Weightbearing is on the hands and knees.

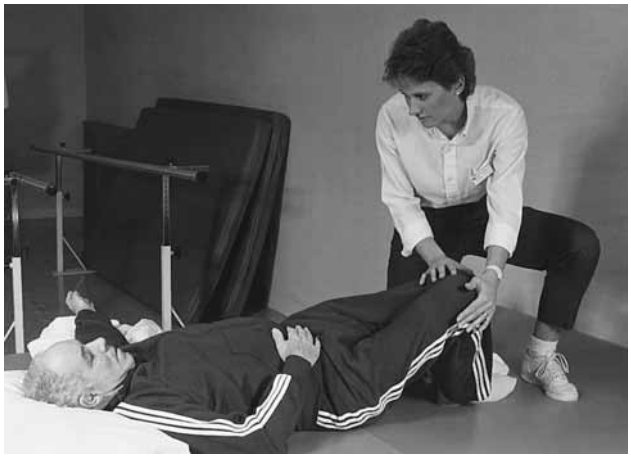

Figure 18.9 Inhibition of truncal tone through lower trunk rotation. The therapist uses the technique of rhythmic initiation to increase mobility.

form of ice wraps or ice packs can be used to temporarily dampen neural firing rates and therefore spasticity. Electrical stimulation to the antagonist muscles or vibration have also been used with some success. Soothing verbal commands and cognitive relaxation techniques (mental imagery) provide an overall calming influence and generally relax tone. Pain syndromes have the opposite effect.

As mentioned earlier, Johnstone[206] advocates the use of inflatable pressure splints (air splints) to stabilize and maintain an extremity in an elongated position and provide tone inhibition. Splints also help to control unwanted synergistic movements and associated reactions, and assist in early weightbearing. Figure 18.8 demonstrates the use of an air splint to stabilize elbow extension in the quadruped position while Figure 18.10 demonstrates its use in the modified plantigrade position. Patients with a flaccid, hypotonic limb may also benefit from the use of pressure splints to provide sensory input and initial stabilization. Long or full limb pressure splints also assist in controlling edema, a common problem of paralyzed limbs. Positioning the splinted limb in elevation can assist in reducing edema.

Strategies to Improve Initial Movement Control

Activities that promote normal postural alignment and control and functional use of the extremities are the primary focus of initial movement training. Patients with stroke typically present with loss of dissociated or fractionated movements with obligatory synergy patterns and associated reactions.[222] For example, coordinated grasp and manipulation are lost as the fingers respond with strong flexion when lifting the arm results in elbow flexion with flexion, abduction, and external rotation of the shoulder. Inter- and intralimb control is also abnormal with movements of one limb strongly linked to movements of the other. During initial

Figure 18.10 Use of a pressure splint applied to support the elbow in extension during weightbearing in the modified plantigrade position. Weightbearing is on the hands and feet.

training, the therapist needs to focus on *dissociation* of different body segments (the ability to move the different parts of the body or limb separately) and *selective* (out-of-synergy) movement patterns. For example, the more affected UE is stabilized in an extended weightbearing position while the patient practices stepping movements in modified plantigrade position.

The linking together of the proper components of movement and the refinement of isolated control requires a great deal of mental concentration and volitional control. Movements that are performed too quickly or with too much force will be ineffective in producing the control needed. Thus the therapist needs to instruct the patient to avoid excessive effort and unnecessary force during movement. The therapist should aim for as much normalcy in movement as possible and select postures that assist the desired movements through optimal biomechanical stabilization and/or use of the optimal point in the range. As control develops, postures can be changed to more difficult ones that challenge developing control. For example, initial elbow extension can be first attempted in sidelying with the shoulder flexed to 90°. The posture can then be changed to sitting, and finally standing. Movements may start out assisted (guided) but should shift to active control as soon as possible. Often the resistance of gravity acting on the

body, or slight manual (tracking) resistance, is enough to initiate or facilitate correct movement responses through proprioceptive loading. If the patient's motor responses are very weak and or unable to activate, direct facilitation using a variety of different stimuli may be necessary to assist the patient in initiation of movement. For example, the patient who lacks adequate control of elbow extension can be positioned in sitting with the affected UE weightbearing. Tapping can be applied over the triceps to facilitate holding in extension. The work of Davies provides an excellent resource for early training activities.[56,223]

Functional tasks are the main focus of training (e.g., reaching and manipulation, walking, stair climbing). Normal function implies variability of movements. Muscles need to be activated in varied activities using varied types of contractions. All three types—eccentric, isometric, and concentric—are important to include in an exercise program. For the patient with stroke who demonstrates very weak movements, eccentric contractions should be practiced before concentric contractions as they utilize elastic elements and muscle spindle support more efficiently. For the same amount of tension, fewer motor units are required. Practice of functional tasks that utilize variations of contractions should also be implemented. For example, the patient who practices sit-to-stand, stabilizing in stance, and then stand-to-sit is utilizing sequences of concentric-isometric-eccentric contractions. Weak muscles (typically antagonistic to strong spastic muscles) should be activated first in unidirectional movements. As control develops, exercises can shift to include slow active reciprocal contractions of agonist and antagonist muscles first in limited ranges, then in full range. This emphasis on balanced interaction of both agonists and antagonists is crucial for normal coordination and function. Proprioceptive neuromuscular facilitation (PNF) patterns using reversal of antagonists and proprioceptive loading through light, tracking resistance are ideal for this.[179] See Chapter 13 for an expanded discussion.

Strategies to Improve Motor Learning

Motor skill learning is based on the brain's capacity for recovery through mechanisms of reorganization, and adaptation. An effective rehabilitation plan capitalizes on this potential and encourages active participation—*the patient must be fully engaged*. Activities are selected that are meaningful and important to the patient. Optimal motor learning can be promoted through attention to a number of factors, most importantly, strategy development, feedback, and practice. Carr and Shepherd[224] describe many of these strategies in their book entitled *Motor Relearning Programme for Stroke*.

Strategy Development

The therapist first assists the patient in learning the desired task (cognitive stage). More specifically, critical task elements and successful goals and outcomes are identified. The desired task is demonstrated at the ideal performance speeds. The patient then begins to practice. If the task has a number of interrelated steps, practice of component parts may precede practice of the whole task. It is important, however, not to delay practice of the integrated task because this may interfere with effective transfer of learning. The therapist should give clear, simple verbal instructions and not overload the patient with excessive or wordy commands. Correct performance should be reinforced and intervention provided when movement errors become consistent. Active participation is essential for learning; *there is no learning with passive movements*. Practicing the movements on the less affected side first can yield important transfer effects. Simultaneous practice of similar movements on both sides (bimanual task practice) have also been shown to improve learning and promote integration of the two sides of the body. Visualization of the movement components (mental practice) can help some patients in initially organizing the movement if cognition is intact.

As initial practice progresses, the patient is asked to self-examine performance and identify problems, specifically, what difficulties exist, what can be done to correct the difficulties, and what movements can be eliminated or refined. If a complex task is practiced, the patient is asked to identify if the correct components were performed, how the individual components fit together, and if they were appropriately sequenced. If the patient is unable to provide an accurate assessment of problems, the therapist can prompt the patient in decision making using guiding questions and utilize demonstration to help identify problems. For example, if the patient consistently falls to the right while standing, questions can be directed toward this problem (e.g., "In what direction did you fall?" "What do you need to do to prevent yourself from falling?"). The patient is thus actively involved in developing problem-solving skills (self-monitoring and self-correction of movements). These skills are essential in ensuring independence and generalizability of learning to other environments and variations.

Feedback

Feedback can be intrinsic (naturally occurring as part of the movement response) or extrinsic (provided by the therapist). During early motor learning the therapist provides extrinsic feedback (e.g., verbal cueing, manual cueing) to shape performance. It is important to monitor performance carefully and provide accurate feedback. The patient's attention should be directed to naturally occurring intrinsic feedback. During early intervention visual inputs are critical for motor learning. This can be facilitated by having the patient look at the movement (a central concept of PNF). Use of a mirror can be an effective adjunct for some patients to improve visual feedback, especially during postural and positioning activities. It is, however, contraindicated in patients with marked visuospatial perceptual impairments. During later learning (associative phase), proprioception becomes

important for movement refinement. This can be encouraged by early and carefully reinforced weightbearing (approximation) on the more affected side during upright activities. Additional proprioceptive inputs (manual contacts, tapping, stretch, tracking resistance, antigravity postures, or vibration) can be used to improve feedback and stimulate learning. The patient should be encouraged to "feel the movement" while learning to distinguish correct movement responses from incorrect ones. Surface electromyography (EMG) can also be used to provide augmented feedback. Exteroceptive inputs (light rubbing, stroking) may be used to provide additional sources of sensory inputs, particularly where distortions of proprioception exist. As treatment progresses, the emphasis again shifts from extrinsic to intrinsic feedback and to self-monitoring and correcting movement responses. Great care must be taken to avoid sensory bombardment or feedback dependence (i.e., movements that occur only if stimulated). This requires careful consideration during each treatment session. Therapists should also limit use of immediate feedback to allow the patient adequate time for introspection. Pain and fatigue (either mental or physical) should be avoided, as each can result in decreased performance and learning.

Practice

Practice, practice, and more practice is essential for motor skill learning and recovery. The therapist needs to organize the patient's therapy session to ensure optimal practice. Constant repetition of the desired task (blocked practice) will improve initial performance and motivation. Most hospitalized patients initially require a distributed practice schedule with adequate rest periods owing to limited endurance. The patient should be encouraged to self-monitor practice sessions and recognize when fatigue may be setting in and rest is required. The therapist needs to progress the patient to variable practice (practice of similar or related tasks using serial or random practice orders) as soon as possible. Variable practice also improves performance and more importantly results in better retention of learned skills, adaptability (modification of tasks), and generalizability to different contexts (environments). Patient, staff, and family efforts should be coordinated to ensure continued and consistent practice during off-therapy times.

Careful attention to the learning environment will also yield important therapeutic gains. Distractions should be reduced and a consistent and comfortable environment provided in which the patient can learn. For many patients with stroke, this will initially be a closed environment with limited distractions. Later the environment can be varied, providing an appropriate level of *contextual interference*. Thus the patient is progressed toward performing the same skill in more open, variable and real-life environments. The addition of *Easy Street Environments* to many rehabilitation centers provides an important tool to simulate community environments.

Motivation is key to successful learning. The patient should be fully involved in collaborative goal-setting from the beginning and continually reminded of the goal, the task, where they are, and what they are striving for (expected outcomes). Treatment sessions should include positive experiences, ensuring the patient experiences success in therapy and instilling self-confidence. Beginning and ending the therapy session on a positive note is a helpful strategy. Self-efficacy ratings can be used to monitor progress (e.g., "What successes did you achieve in therapy today?"). Supportive strategies should be discussed with family and caregivers. Finally, the therapist should continually communicate support and encouragement to the patient. Recovery from stroke is an extremely stressful experience and will challenge the coping abilities of both patient and family.

Strategies to Improve Postural Control and Functional Mobility

The loss of sensory and motor function on one side will present a tremendous challenge for the patient struggling to relearn postural control and functional mobility. Initial treatment strategies should focus on trunk symmetry and use of both sides of the body. Progression is from guided movements to active movements as soon as the patient is able to assume independent control.

Suggested functional training activities include:

Rolling

Rolling to both sides should be practiced; rolling onto the less affected side will prove more difficult. Extremity movement patterns (e.g., PNF D1 flexion of the LE) can be used to enhance the movement. Care must be take to ensure the patient does not leave the more affected UE behind but rather brings it forward. This can be accomplished by having the patient clasp the hands together in a prayer position first. The more affected LE can also be used to assist in rolling by pushing off from a flexed and adducted, hooklying position (Fig. 18.11). Rolling onto the

Figure 18.11 Early mobility activities: rolling onto the unaffected side. The therapist guides the movement and assists the upper extremity pattern of prayer position.

more affected side and into a sidelying-on-elbow position is important to promote early weightbearing. This position also has the added benefit of elongating the lateral trunk flexors, which may be spastic.

Supine-to-Sit and Sit-to-Supine

The patient should practice moving from supine-to-sitting from both sides, with an emphasis on rising from the more involved side. The therapist can provide assistance from sidelying on the more affected side by shifting the LEs over the edge of the bed or mat while the patient pushes up into sitting using both UEs for support. Controlled lowering should also be practiced.

Sitting

Early training in sitting should focus on achieving a symmetrical posture with proper spine and pelvic alignment. The pelvis should be neutral, spine straight. Feet should be flat on the support surface. Typically, patients with stroke will sit asymmetrically with weight borne more on the less affected side, pelvis in a posterior tilt, and upper trunk flexed (kyphotic). Lateral flexion to the affected side is also common. The therapist can manually guide the patient into the correct sitting position and provide verbal and tactile cues. Early sitting can be assisted by having the patient use the UEs for bilateral support (at sides or in front: on table top, a large ball, or the therapist's shoulders with the therapist sitting directly in front of the patient). Sitting on a therapy ball can also be used to promote pelvic alignment and mobility (pelvic rotations) and trunk upright alignment (gentle bouncing). Sitting control should be progressed from first holding steady in the posture (stability) to moving in the posture (dynamic stability), and finally to dynamic challenges (reaching). A common problem with hemiplegia is the inability of the upper trunk to move independently of the lower trunk (dissociate). Upper trunk mobility with reciprocal flexion/extension, lateral flexion, and rotation movements should therefore be practiced. Lateral weight shifts to the more affected side typically are the most difficult. Manual contacts in the direction of the movement combined with gentle resistance can provide important early learning cues. PNF patterns of chop/reverse chop or lift/reverse lift are excellent examples of patterns that promote trunk rotation, bilateral UE activity, and crossing the midline (important for unilateral neglect). The patient should also practice scooting in sitting ("butt walking") to ensure mobility for dressing (putting pants on) and sit-to-stand transitions (coming to the edge of the seat to place the feet back and under the body).

Bridging

Bridging activities help develop trunk and hip extensor control important for use of a bedpan, pressure relief on the buttocks, initial bed mobility (scooting), and sit-to-stand transfers. It also develops advanced LE out-of-synergy control (hip extension with knee flexion), and stimulates early weightbearing through the foot (Fig. 18.12). Bridging activities include independent assumption of the posture, holding in the posture, and moving in the posture (lateral weight shifts, bridge-and-placing hips to one side). If the more affected LE is unable to hold in a hooklying position, the therapist will need to assist by stabilizing the foot. Lifting the less affected foot off the surface (placing it on a small ball) while maintaining the pelvis level significantly increases the difficulty and can be used to increase demands on the more affected side. Difficulty can also be increased by varying the position of the UEs, from extended and abducted at the sides, to arms folded across the chest or hands clasped together overhead in a prayer position.

Figure 18.12 Early mobility activities: bridging. The patient combines hip extension with knee flexion. The therapist assists in stabilizing the affected knee in flexion with foot flat.

Sit-to-Stand (STS) and Sit-Down Transfers

STS transfers should be practiced with a focus on symmetrical weightbearing, coordinated muscular responses, and adequate timing (Fig. 18.13). Initially the patient must actively flex the trunk and use momentum to shift the body mass forward (*flexion-momentum phase*). The feet should be placed well back to allow ankle dorsiflexors to assist with forward rotation. The patient with stroke typically demonstrates decreased forward movement and momentum. The therapist should focus the patient's eyes on a visual target directly in front at eye level and use verbal cues to facilitate the desired movements ("move your shoulders forward and stand up"). The patient can be assisted in this phase by swinging both hands forward or reaching forward with both UEs, hands clasped together in a prayer position. If the patient is apprehensive of falls, both hands can be positioned on a large therapy ball while the therapist stabilizes and moves the ball forward in time with the forward weight shift. Pushing off with both hands on the support surface is not effective in producing the forward weight shift and should be discouraged. The patient's movements must then be directed into the *extension phase* which requires hip and knee extensors to produce vertical movement into the upright position. Weakness and incoordination of these muscles typically result in incomplete

Figure 18.13 Sit-to-stand movement transitions. The therapist assists the patient in straightening his affected knee while he brings his center of mass forward. Hands are held together in a prayer position.

Figure 18.14 Early weightbearing in modified plantigrade with extended UEs. The therapist assists elbow and finger extension of the affected right UE.

extension and inability to stand up independently. The height of the seat can be elevated at first to decrease the extensor force required. Progression is then to lower seat heights. Increased weightbearing on the stronger LE can be achieved by varying the initial foot position, placing the stronger foot slightly behind the weaker foot. As the patient improves, the position of the feet can be reversed to focus attention on increased use of the weaker side. The patient with stroke typically accomplishes standing up very slowly. With repetitive practice, the patient should be encouraged to focus on increasing the speed of the movement and to not pause between the two phases. Using a prayer position (hands clasped together and held straight ahead with elbows extended) reduces UE push-off. The patient with stroke also demonstrates decreased control in sitting down owing to lack of eccentric control and will sit down abruptly after moving partially through the range. Eccentric movements (small range movements) can be practiced with the patient positioned back against a wall doing partial wall squats. Lower trunk rotation can be promoted by having the patient practice STS using a platform mat. From standing, the patient shifts the pelvis laterally to the more affected side, and then sits down. By using this activity, the patient can move all the way around the mat alternating standing and controlled sitting, focusing on moving toward the weaker side.

Standing, Modified Plantigrade

Modified plantigrade is an ideal early standing posture to develop postural and extremity control. The more affected UE is extended and weightbearing (an out-of- synergy posture), while the more affected LE is holding in extension (also an out-of-synergy pattern of hip flexion with knee extension). The forward trunk position creates an extension moment at the knee, thus assisting weak knee extensors. In addition, the posture has a wide (four-limb) base of support and is very stable (Fig. 18.14). Progression should again be from holding in the posture to moving in the posture (weight shifts) to reaching tasks.

Standing

Initial upright standing can be enhanced using fingertip light touch down support on a high table or wall. As soon as possible, the patient should be encouraged to practice standing with unilateral UE support (more affected side) and then free standing (no UE support). As in other postures, an appropriate progression includes first holding in the posture, to moving in the posture (weight shifts), and finally withstanding challenges to dynamic balance (e.g., reaching in all directions, stepping). The patient is instructed in proper symmetry and alignment. Gentle resistance can be applied to assist in holding, using the PNF technique of rhythmic stabilization. Weight shifts should incorporate moving forward-backward, side-to-side, and diagonally (incorporating upper trunk rotation). Lateral weight shifts to the more affected side are the most difficult. Manual contacts in the direction of the movement combined with gentle resistance can provide important early learning cues. Early weightbearing on the more affected limb can be achieved using a half-sitting position (Fig. 18.15).

Transfers

During early transfers, the patient may require maximal assistance. Adjusting the hospital bed to the height of the chair or wheelchair will help to decrease the difficulty of the transfer. Staff often emphasize the sound

Figure 18.15 Early weightbearing on the affected LE. The therapist assists controlled, small-range flexion and extension movements of the knee. The affected UE is maintained in an inhibitory position.

Figure 18.16 Transferring to the affected side. The patient learns to control standing up and pivoting with the affected LE leading. The therapist assists in balance.

side by placing the chair to that side and having the patient stand and pivot a quarter turn on the stronger LE before sitting down. Although this compensatory strategy promotes early transfers, it neglects the weaker side and may make subsequent training more difficult. The patient should be taught to transfer to both sides, with emphasis on moving toward the more affected side. Practice to both sides has functional significance, as most bathrooms are not large enough to allow positioning of the wheelchair on both sides of a tub or toilet. Also, the patient is not likely to be able to reposition the wheelchair once he or she transfers into bed so that a transfer toward the same side can be achieved when getting out of bed. When transferring, the patient's affected arm can be stabilized in extension and external rotation against the therapist's body. Alternately, the patient's UEs (hands in prayer position) can be placed in front or to one side on the therapist's shoulders. (Fig. 18.16). The therapist can then assist in STS by using manual contacts, either at the upper trunk or pelvis. The more affected LE may be stabilized by the therapist's knee exerting a counterforce on the patient's knee as needed. Transfer training should include practice in transferring to various different surfaces and heights (e.g., wheelchair, toilet, tub seat, car).

Functional mobility training is begun early and continued throughout the course of rehabilitation. Training activities and postures are varied according to individual needs. Additional postures such as modified prone-on-elbows (table top weightbearing), quadruped, side-sitting, kneeling, and half-kneeling may be appropriate and can be used to increase the level of difficulty and focus on specific body segments and deficiencies in control. Some postures may not be appropriate (e.g., prone-on-elbows for the patient with cardiorespiratory compromise or a flaccid, subluxed UE, or kneeling for the patient with osteoarthritis). Advanced functional training should include practice in getting down to and up from the floor in the event of a fall. See Davies[56,223] and O'Sullivan and Schmitz[225] for additional functional training activities.

The Patient with Ipsilateral Pushing (Pusher Syndrome)

The patient with ipsilateral pushing presents with an entirely different set of postural control problems. The patient sits or stands asymmetrically, but with most of the weight shifted toward the weaker side. The patient uses the stronger UE or LE to push over to the weaker side, often resulting in instability and falls. Efforts by the therapist to passively correct

the patient's tilted posture often result in the patient pushing stronger. Training needs to emphasize vertical positions with *active* movement shifts to the stronger side. Use of visual stimuli is effective as patients retain the ability to correct posture with such stimuli but may not be able to do so spontaneously. Patients should be asked to look at their posture and see if they are upright. Environmental prompts can be used to assist orientation. These can include use of a mirror if visuospatial deficits are not present or vertical structures in the environment. For example, the therapist can sit on the patients less involved side and instruct the patient to "lean over to me." Or the patient can be positioned with the stronger side next to a wall and instructed to "lean toward the wall."[61] Therapists can provide verbal and tactile cues for postural orientation. To improve sitting posture, training activities can include sitting on a therapy ball to promote symmetry and sitting, crossing the weaker LE over the stronger LE. In early standing the weaker LE is often flexed and has difficulty supporting the body on that side. Extension can be assisted by the use of an airsplint or a posterior leg splint or by direct tapping over the quadriceps muscle.[56] The modified plantigrade position is effective for early supported standing; however, the therapist should focus on unilateral support using the weaker UE. Again, an airsplint can be used to assist extension of the weaker arm. If a cane is used, it can be shortened to encourage weight shift to the stronger side. An environmental boundary can be used to achieve symmetrical standing (e.g., standing in a doorway or corner standing). It is important to limit pushing with the sound extremities. For example, in sitting or standing, the therapist should block the stronger limb from drifting laterally into abduction and extension and pushing. Motor learning strategies are very effective in reducing the effects of this disorder and enhancing recovery. In particular, the therapist should demonstrate correct orientation to vertical, provide consistent feedback about body orientation, and practice correct orientation and weight shifts. The patient should be fully involved in problem-solving. For example, the therapist should ask questions such as "what direction are you tilted? "and "what direction do you have to move in order to achieve vertical?". Karnath and Broetz[61] indicate that the potential for minimizing the impact of ipsilateral pushing is good with effective training.

Strategies to Improve Upper Extremity Function

Patients with severe sensory, motor, and functional impairments (recovery stage 3 or less) are generally less responsive to remedial interventions than patients demonstrating more advanced recovery. These patients will benefit from early mobilization, ROM, and positioning strategies, previously discussed in this chapter. Compensatory training strategies and environmental adaptations should be considered to maximize function. For patients with more advanced recovery (stage 4 or higher), training strategies that focus on repetitive and intense use of tasks can be expected to produce meaningful improvements in function. Activities to retrain UE postural support, reaching, and manipulation are essential elements of training. Enhanced training programs along with behavioral training methods (e.g., constraint-induced movement therapy) have demonstrated promising gains in recovery of function. The greatest treatment effects are seen in those patients with some initial active movement control while those with more severe involvement demonstrate poorer final outcomes.[226,227] Training to improve UE function should be closely coordinated with the occupational therapist.

UE as a Postural Support

Extended arm weightbearing with stabilized hand on a support surface is an important early activity to promote proximal stabilization and counteract the effects of excess flexor hypertonus and a dominant flexion synergy. Approximation can be used to increase stimulation of shoulder/scapular stabilizers and elbow extensors. Weightbearing activities are performed in sitting (see Fig. 18.7), modified plantigrade (see Fig. 18.14), and standing positions. The quadruped posture provides the greatest challenge for UE stabilization control but may be too difficult for some patients. Control should progress from holding to dynamic stabilization activities. For example, the patient stabilizes with the more affected UE while performing weight shifts and functional tasks with the stronger UE (e.g., reaching).[228] As previously mentioned, the more affected UE should also be recruited for postural assistance during functional training activities (e.g., pushing up from sidelying into sitting).

Reaching

Patients with stroke have difficulty regaining control of scapular upward rotation and protraction, elbow extension, and wrist and finger extension. Reaching and manipulation also requires accurate processing and use of visual–perceptual information. Patients with minimal voluntary control can practice initial reaching forward in a sideling position, where the patient's UE is supported by the therapist in shoulder flexion with elbow extension. The UE is mobilized forward and the patient is asked to hold this position. If holding is successful, then eccentric and reciprocal movements are attempted. Supported reaching can be also practiced in sitting with the hand resting on a tabletop. The patient is encouraged to slide the hand forward over tabletop, recruiting shoulder flexors, scapular protractors, and elbow extensors. A cloth can be used to decrease friction effects as the patient practices wiping or polishing a table. The patient can also practice reaching forward and downward touching the floor. Advanced reaching activities include independent lifting and reaching forward (e.g., UE placed into a shirt sleeve), overhead, or sidewards. A PNF D1 thrust pattern can be practiced (reverse thrust is contraindicated as the limb is moving into a flexion synergy pattern). Combining reaching with increased balance challenges in standing should

also be incorporated (e.g., standing and reaching to pick an object up off a shelf, a low stool or the floor; modified push-ups in standing). Varying the height and distance reached, increasing the weight of objects held in the hand or increasing the speed and accuracy requirements can increase difficulty. Strategies used for substitution when the patient is unable to reach include trunk or head lateral movements and should not be allowed. Excessive shoulder elevation should also be discouraged.[228,229]

Manipulation and Dexterity

Meaningful task-oriented practice involving grasp and manipulation is important for stimulating recovery. Initial hand movements typically include gross grasp and release while advanced hand patterns (fine motor control) may not be present unless there is more advanced recovery. Voluntary release is generally much more difficult to achieve than voluntary grasp, and stretching/positioning and inhibitory techniques may be necessary to facilitate extension movements. Initial hand tasks can include using the more affected hand to stabilize (e.g., hand stabilizes paper while the stronger hand writes, hand stabilizes food while the stronger hand cuts) or holding a book with both hands for reading. The patient should be encouraged to use the weaker hand to assist in ADL (e.g., washing the upper body with a washcloth, bringing food to mouth). Forks, toothbrushes, and pens may need to have built-up handles for grasp. Task training should combine reach patterns with hand activity (e.g., picking sock off floor, reaching for an object off a shelf). Advanced hand activities include practice of wrist and finger extension, opposition, and manipulation of objects (e.g., using utensils to eat, drinking from a cup, writing, picking up and reorienting coins, paperclips or other objects). Pronation often predominates while active supination without elbow and shoulder flexion is difficult to achieve. The therapist must observe movements carefully and assist in eliminating those aspects of movement patterns that interfere with effective and efficient control. Graded physical assist and use of mental practice/imagery techniques can be helpful to improve learning and performance.[228]

Enhanced Training Activities

Constraint-induced movement therapy (CIMT) is discussed in Chapter 13 (see Evidence Summary Box 13.1). Gains in motor function following CIMT have been demonstrated in patients with stroke and are associated with changes in brain organization as evidenced by functional magnetic resonance imaging (fMRI). These include an apparent shift in motor cortical activation toward other ipsilateral areas and the contralesional hemisphere.[230] Figure 18.17 illustrates a training session using CIMT.

Bilateral arm training with rhythmic auditory cueing (BATRAC) has been shown to improve motor function in patients with chronic stroke.[231] This repetitive training program utilizes a customized bilateral arm trainer in which the patient holds onto T-bar handles and moves them in a nearly friction-free environment forward in the transverse plane (simulating forward reaching and its reverse motion). The bilateral movements are timed to an auditory metronome set at the patient's preferred speed. Significant improvements were noted in functional motor performance of the weaker UE that were sustained 2 months after training.

Figure 18.17 Constraint-induced-movement therapy (CIMT). The patient practices a pegboard task using the affected UE while the sound hand is constrained with a mitt. The therapist times the activity while encouraging the patient.

Electromyographic biofeedback (EMG-BFB) has been used to improve motor function in patients following stroke. This technique allows patients to alter motor unit activity based on augmented audio and visual feedback information. Training can focus on voluntary inhibition of spastic muscles (e.g., reducing firing frequency of spastic finger flexors), or on increasing kinesthetic awareness and recruitment of motor units in weak, hypoactive muscles (e.g., wrist/forearm extensor muscles). Patients in the chronic stage (1 year post-stroke) or patients in late recovery for whom spontaneous recovery is more or less complete (6 months post-stroke) have demonstrated positive results that have been attributed to biofeedback therapy.[232–236] Reported benefits include improvements in ROM, voluntary control, and function. Most researchers indicate that effectiveness of biofeedback neuromuscular reeducation is greatest when used as an adjunct to task-specific training.

Neuromuscular electrical stimulation (NMES) has been used with patients recovering from stroke to reduce spasticity, improve sensory awareness, and volitional limb movements.[237] NMES has been shown to increase the ability of muscle to exert force by preferentially activating the fast-contracting motor units.[238] Effective treatment results

have been reported for improving function in wrist extensors,[239] and the deltoid and supraspinatus muscles. In the latter example, glenohumeral alignment was improved and subluxation reduced.[240,241] As with the biofeedback research, optimal results have been obtained when combined with task-specific training.

Management of Shoulder Pain

Hemiplegic shoulder pain (HSP) is a common complication after stroke, with incidence rates ranging from 38 to 84 percent of cases.[242,243] Pain is described as sharp and stabbing and is more common on movement than rest. Early on, pain can be intermittent and limited to just the shoulder. During later stages pain is constant and can progress to severe pain in more than just the shoulder. Several causes of HSP have been identified, that can be broadly divided into flaccid and spastic presentations. In the flaccid stage, proprioceptive impairment, lack of tone, and muscle paralysis reduce the support and normal seating action of the rotator cuff muscles, particularly the supraspinatus. The ligaments and capsule thus become the shoulder's sole support. The normal orientation of the glenoid fossa is upward, outward, and forward, so that it keeps the superior capsule taut and stabilizes the humerus mechanically. In the absence of supporting musculature, any abduction or forward flexion of the humerus, or scapular depression and downward rotation, reduces this stabilization and causes the humerus to sublux. Initially the *subluxation* is not painful, but mechanical stresses resulting from traction and gravitational forces produce persistent malalignment and pain. Glenohumeral friction–compression stresses also occur between the humeral head and superior soft tissues during flexion or abduction movements in the absence of normal scapulohumeral rhythm (*shoulder impingement syndrome*). During the spastic stage, abnormal muscle tone may contribute to poor scapular position (depression, retraction, and downward rotation) and contributes to subluxation and restricted movement. Secondary tightness in ligaments, tendons, and joint capsule can develop quickly. *Adhesive capsulitis* (intracapsular inflammation: "frozen shoulder") is a common finding. Poor handling and positioning of the more affected UE have been implicated in producing joint microtrauma and pain. Activities that traumatize the shoulder include PROM without adequate mobilization of the scapula (promoting normal scapulohumeral rhythm), pulling on the UE during a transfer, or using reciprocal pulleys.[244,245] An incorrectly aligned joint can significantly impair the patient's ability to move.

Prolonged soft tissue injury can result in *chronic regional pain syndrome (CRPS-type I)*, also known as reflex sympathetic dystrophy (RSD). This pain typically has a diffuse onset and is characterized as aching throughout the limb. CRPS-1 is associated with a range of other symptoms. The wrist tends to assume a flexed position with intense pain likely during wrist extension movements. The elbow is not involved. Early *stage 1* vasomotor changes include discoloration (pale pink or cool) and alterations in temperature. The skin may be hypersensitive to touch, pressure, or temperature variations. The patient typically guards against movement attempts. *Stage 2* is characterized by subsiding pain and early dystrophic changes: muscle and skin atrophy, vasospasm, hyperhidrosis (increased sweating), and course hair and nails. There is radiographic evidence of early osteoporosis. In *stage 3*, the atrophic phase, pain and vasomotor changes are rare. There is progressive atrophy of the skin, muscles, and bones (severe osteoporosis is evident). Pericapsular fibrosis and articular changes become pronounced. The hand typically becomes contracted in a clawed position with metacarpophalangeal (MP) extension and interphalangeal (IP) flexion (similar to the intrinsic minus hand). There is marked atrophy of thenar and hypothenar muscles with flattening of the hand. Chances of reversal of signs and symptoms are high for stage 1 and variable for stage 2, while stage 3 changes are largely irreversible.[246]

Early diagnosis and identification of factors that cause HSP is essential. Interventions are selected based examination findings. Because of close daily contact with the patient, the therapist is frequently one of the first to recognize and report early signs and symptoms. In the flaccid stage, the arm should be supported at all times. Proper positioning and handling is essential. In supine and sitting the scapula/shoulder should be protracted with the arm forward in slight abduction and neutral rotation. Interventions aimed at reducing subluxation include NMES therapy, and use of supportive devices (see section below). Interventions aimed at normalizing tone and reducing pain include appropriate mobilization techniques (gentle grade 1 to 2 mobilizations, gentle stretching), cryotherapy, EMG biofeedback, and relaxation training. Interventions for adhesive capsulitis include mobilization and PROM techniques, and ultrasound. The therapist needs to ensure that everyone involved in assisting the patient (e.g., family member, caregiver, nurses, and aides) has been instructed in proper handling/mobilization of the UE, and recognizes the importance of avoiding trauma and traction injuries during PROM, transfers, and wheelchair activities. Active assisted range of motion (AAROM) and active movements of the UE and trunk are facilitated in order to optimize functional recovery. Interventions to manage edema may also be a consideration. Persistent pain may be managed with oral analgesics or local injection techniques (e.g., triamcinolone acetonide). Repeat steroid injections are not recommended due to likely weakening of the rotator cuff. With intractable pain, surgical nerve blocks may also be considered.[244,245]

Supportive Devices

A patient with hypotonia is at increased risk of *traction injury*. Care must be taken not to pull on a flaccid UE during position changes or let it hang unsupported. Slings can prevent soft tissue stretching (e.g., capsular stretching) and

relieve pressure on the neurovascular bundle. However, slings do little to reduce subluxation or improve shoulder function, especially if scapular and trunk malalignment are not adequately addressed. Most slings have the additional negative feature of positioning the arm close to the body in adduction, internal rotation, and elbow flexion. With prolonged use, contractures and increased flexor tone may develop. Slings also impair trunk mobility, balance, sensory input, and body image and may increase body neglect. Slings block spontaneous use of the UE and can contribute to learned nonuse. There are considerable differences in the effectiveness of the various types of supports.[247] A pouch sling or single strap hemisling with two cuffs that support the elbow and wrist provides minimal mechanical support of the humerus. An alternate approach to the traditional sling is a humeral cuff sling. This device has an arm cuff on the distal humerus supported by a figure-eight harness. It provides humeral support with slight external rotation while allowing elbow extension, and may also provide some reduction of subluxation (see Fig. 18.7). This style of sling can be worn for longer periods because it does not restrict the elbow in a flexed position or limit distal function.[248,249]

Gillen[228] suggests the following guidelines are appropriate to consider when prescribing a sling:

- Slings are appropriate for initial transfer and gait training, but overall use should be minimized during rehabilitation.
- Slings that position the UE in flexion are less desirable and should be used only for select upright activities and only for short time periods.
- No one sling is appropriate for all patients; selection and use should be carefully evaluated.
- Effective alternatives to use of a sling should be considered: taping (strapping) to facilitate or inhibit musculature surrounding the scapula; NMES. The hand can also be positioned in a garment pocket.

The patient, family members, and caregivers should be instructed in and allowed to practice proper use of the support. As recovery progresses and spasticity and voluntary movement emerge, spontaneous reduction of shoulder subluxation typically occurs. Slings have no value at this point in recovery.

For patients using a wheelchair, an arm board or lap tray can provide support for the flaccid arm. A lateral elbow guard and/or straps may be necessary if the patient's arm slips off the side. Patients with decreased sensation are at risk for hand injury if the hand becomes stuck in the spokes of the wheelchair; elbow trauma can occur if the elbow slips off the side (e.g., the elbow hits as the patient is going through a doorway).

Strategies to Improve Lower Extremity Function

LE training activities essentially prepare the patient for the appropriate gait. This requires breaking up the obligatory synergy patterns. For example, during midstance hip and knee extensors need to be activated with hip abductors and dorsiflexors. Suggested activities include PNF LE D1 extension pattern; holding against elastic band resistance around the upper thighs in supine or standing positions; and standing, lateral side-steps. Hip adduction should be stressed during flexion movements of the hip and knee. Suggested activities include supine, PNF LE D1flexion pattern; sitting, crossing and uncrossing the more affected LE over the less affected; and standing, step-ups. Hip extension with knee flexion is needed to allow for toe-off at the end of stance. Activities that can be used to promote knee flexion with hip extension include bridging, supine hip extension with knee flexion over the side of the mat pushing down through the heel, or standing, posterior foot rises. Pelvic control is important and can be promoted through lower trunk rotation activities that emphasize forward pelvic rotation (protraction); post-stroke, the patient typically demonstrates a retracted and elevated pelvis. Rotation can be practiced in sidelying; supine, modified hooklying; kneeling; or standing. Sitting on a therapy ball, pelvic shifting is another useful activity to promote pelvic control. Control of knee motions is often problematic; post-stroke, the patient with knee weakness typically exhibits hyperextension when standing. Reciprocal action (smooth reversals of flexion and extension movements) should be stressed early, beginning first in supine (e.g., foot slides in hooklying), sitting (e.g., foot slides under the chair), partial sitting (see Fig. 18.15), or partial wall squats in standing.

An effective progression increases the challenge to the patient gradually by modifying postures while reducing synergy influence (e.g., hip abduction can be performed first in hooklying, then supine, sidelying, modified plantigrade, and finally standing). Dorsiflexors can be activated in sitting by first having the patient hold and slowly let the forefoot move down, then pull the forefoot up. This simulates the functional expectations of the normal gait cycle as the foot goes from swing phase through stance. The sequence can then be repeated in standing, a much more difficult position in which to control dorsiflexors. Voluntary control of eversion is often the most difficult motion to achieve because these muscles do not function in either synergy. The application of stretch and resistance to these muscles during an activity that recruits dorsiflexors and evertors may be effective in initiating a response (e.g., in bridging, knee rocks side-to-side).

Strategies to Improve Balance

Balance Training

Stroke results in significant changes in balance. Patients typically exhibit delayed, varied, or absent balance responses with impairments in latency, amplitude, and timing of muscle activity. It is therefore important to proceed slowly in training and to select challenges appropriate for the patient's level of control. Once postural alignment and

static stability is achieved in upright postures, the patient is ready for center-of-mass (COM) control training. In sitting and standing, the patient is instructed to explore his or her limits of stability (LOS) through low-frequency weight shifting. The patient learns how far in any one direction he or she can safely move and how to align the COM within the base of support (BOS) to maintain upright stability. The therapist needs to stress symmetrical weightbearing, as well as activities that promote shifting toward the more affected side. Weightbearing on the more affected hip (sitting) and foot (standing) is encouraged while unnecessary activity of the less affected limbs (grabbing for support) is discouraged. The therapist can increase the difficulty of the activity by manipulating:

- Base of support: sitting, LEs uncrossed to crossed; standing, wide to narrow to tandem position; standing on one LE
- Support surface: sitting on a mat to sitting on a therapy ball; standing on the floor to standing on dense foam
- Sensory inputs: EO to EC; feet on firm surface or foam
- UE position/support: light touch down support; UEs extended out to the side to UEs across the chest
- UE movements: single UE raises to bilateral UE raises (symmetrical, asymmetrical); reaching; picking objects off table, stool, floor
- LE movements: single LE raises, stepping (forward–backward, side; step-ups); marching in place; foot on ball, moving ball
- Trunk movements: head and trunk rotations; looking up at ceiling or down to floor
- Destabilizing functional activities: sit-to-stand, sit-down, turning, floor-to-standing
- Dual task training: standing while catching or kicking a ball; standing while talking; standing while holding a tray with a glass of water
- Environmental conditions: closed to open environments

The goals of training are to increase the consistency, range, and speed of self-initiated movements while encouraging symmetry and maximum use of the more affected side. Supportive devices such as a posterior leg splint, gait belt, or body-weight support harness can be used to assist in early standing to instill confidence and prevent falls.

Postural strategy training is an important component of intervention. Ankle strategies can be promoted through small range anterior–posterior shifts or by applying a small perturbation at the hips (forward–backward). Standing on a half-foam roller or wobble board also promotes ankle strategies, but may be too advanced for some patients during early rehab. Hip strategies can be promoted through larger anterior–posterior shifts or stronger perturbations. Medial–lateral hip strategies are promoted by tandem stance (on floor or foam roller). Stepping strategies are promoted by increased displacements of the COM (e.g., forward, backward, or sideward leans that move the COM outside of the BOS). The therapist can apply an elastic band around the hips, offering resistance to the forward lean. Resistance that is quickly released once the patient achieves the desired lean will necessitate a step to control balance. Step-ups (small step to large; foam surface) should also be practiced.[250]

Figure 18.18 Balance training in kneeling. The therapist assists in maintenance of the kneeling posture while encouraging the patient in the weight shift and cone-stacking task.

The patient's full attention and concentration is required and should be directed toward completion of the task at hand (task-specific training vs a focus on balance in general) (Fig. 18.18). The therapist provides well-timed feedback to help the patient correct alignment and adjust postural control while minimizing hands-on support (only as needed). Balance training should encourage active problem-solving. The patient is presented with challenges, is able to identify potential problems, and recruits safe strategies to maintain balance. Thus adaptability of skills needed for successful community reentry is promoted. Safety education about fall prevention is a critical factor in ensuring maintenance of the patient's hard-won functional independence (see Chapter 13).

Enhanced Training Activities. Force platform biofeedback (center-of-pressure biofeedback) provided to the patient while standing on a computerized force-plate system can be used to improve balance. The patient practices voluntary movement shifts in response to computer generated visual feedback. Patients can also practice responding to unexpected platform tilts (perturbations) in order to improve reactive balance control. A safety harness may be required during early training; holding on with one or both hands is discouraged. Improvements with biofeedback/forceplate training have been found in steadiness (reduced sway),[251,252] postural symmetry,[251,252] and dynamic stability.[252-254] Nichols[255] points out that the evidence is stronger

and more consistent for the latter two parameters than for changes in steadiness. There is limited evidence of carryover of improved balance during functional skills, specifically transfer skills and endurance,[254] functional reach,[256] and measures of ADL and mobility.[252] Carryover to improved locomotor performance has not been demonstrated.[251,254,256] Failure to find significant correlations to gait is most likely related to specificity of training, specifically a dissimilarity between training mode and outcome measure. Studies comparing conventional balance training with biofeedback/forceplate training based on improvements on functional balance measures (Berg Balance Scale, Timed Up-and-Go) have failed to show any differences between the training modes; both interventions were effective in improving balance.[257,258]

Strategies to Improve Locomotion

Locomotor Training

While locomotor control is distributed across discrete regions of the CNS, walking is primarily a brainstem and spinal cord function. For example, locomotor central pattern generators (CPG) have been identified as existing in the ventral spinal cord while integrating command centers have been identified in the medial medullary reticular formation. Thus patients with cortical stroke may be able to regain the ability to walk. The CNS is responsive to training-induced plastic changes in locomotor function and recovery. Thus, patients with limited recovery who lack voluntary isolated control can still be trained to walk. While sensation is normally used for walking, patients can also learn to walk with limited sensation.

Locomotor training using body weight support from an overhead harness and a motorized treadmill allows the clinician to stimulate automatic walking using intensive task-oriented training (Fig. 18.19). Normal kinematics and phase relationships of the full gait cycle are promoted, including limb loading in midstance and unweighting and stepping during swing. Initially manual assistance is provided by trainers to normalize gait in the presence of muscle weakness and impaired balance. For example, one therapist provides manual assistance to foot placement during stepping movements of the weaker LE while a second therapist stands behind the patient and provides manual assistance to pelvic rotation movements. An overhead harness is used to support a portion of the patient's weight (e.g., 30 percent progressing down to 20 percent, and 10 percent). The harness controls the upright position of the patient in the absence of good postural stability and reduces fear of falling. The use of a harness also eliminates the need for adaptive UE support to compensate for LE weakness (e.g., as seen with the use of a walker). As improvements in walking occur, the harness is removed and full weight-bearing is allowed. At this point, the patient is practicing supervised walking on a treadmill. Initially the treadmill speeds are slow (e.g., 0.52 mph [0.23 m/sec]) and are gradually increased as the patient's walking ability is improved (e.g., 0.95 mph [0.42 m/sec)[101] Sullivan et al[198] trained three groups at different speeds and found that training at fast speeds (0.98 m/sec) was more effective at improving speeds of overground walking than training at slow (0.22 m/sec) or variable speeds (0.22 m/sec to 0.89 m/sec). It is important to keep in mind that the functional speeds required for community ambulation (normal healthy population) average 2.8 mph (1.3 m/sec).[166] Stepping movements are triggered by LE muscle and joint receptors (e.g., stretch on hip flexors and plantarflexors at the end of the support phase triggers stepping) while load receptors in the stance limb trigger extensors.[259] Patients train on average 30 minutes/day, 5 days per week for durations of 6 to 12 weeks. This form of locomotor training has been shown to be a safe and effective task-oriented training activity that allows the patient to regain walking ability. Specificity and intensity of training is directly related to reported outcomes. These include improvements in walking speed,[101,194–198,200] distance,[101,196] endurance,[101] functional balance (Berg Balance Scale)[101,200] and motor recovery (STREAM).[101] Walking also results in improvements in muscular and cardiovascular endurance.[260] See Evidence Summary Box 18.8. Progression is to overground walking.

Figure 18.19 Locomotor training using body weight support and a treadmill. One therapist manually assists pelvic motions while a second therapist assists stepping of the affected left LE.

Figure 18.20 Assisted ambulation using a plastic ankle-foot orthosis and a quad cane. A resting pan splint supports the patient's affected hand.

Gait Training

Conventional gait training focuses on improving the mechanics and quality of walking. Parallel bars and ambulation aids (e.g., walkers, hemiwalkers, quad canes) can assist in early gait stability and safety (see Fig. 18.20). However, prolonged use of these devices can be problematic for a patient who has the potential to walk without the device. There is increased loading on UE and the stronger LE. With prolonged use, the patient also fails to develop appropriate balance mechanisms while asymmetry is promoted. There is an excessive weight shift toward the less affected side with the use of a hemiwalker or quad cane. Prolonged use of a walker encourages a forward trunk position with maximum loading on the UEs. Gait is typically slower with assistive devices and overall locomotor rhythm is impaired. It is important to progress patients as quickly as possible to the least restrictive device and to no device whenever possible. Gait practice with an overhead harness and partial body weight support provides the least interference with balance and walking. Consideration should be given to maintaining the natural rhythm of walking and speed. The patient should be encouraged to take even steps. This can be facilitated by the use of rhythmic auditory cues (e.g., verbal cues, metronome) and foot markers placed on the floor. Progression is to longer steps and increased overall distances with faster speeds. The patient should also practice walking in varying environments that encourage adaptation to natural environments.

An accurate analysis of gait abnormalities is critical (see Box 18.5 Gait Deviations Commonly Seen Following Stroke). These abnormalities arise as a result of impairments in flexibility, strength, coordination, and balance. Gait activities should focus on improving specific gait impairments and modifying key elements. Critical areas of stance phase control that will need to be addressed include initial weight acceptance, midstance control, and forward weight advancement during stance on the more involved limb. During swing, control of knee and foot for toe clearance and foot placement are key requirements. Finally, persistent posturing of the UE in flexion and adduction during gait should be addressed. This latter problem can be effectively controlled through positioning the hemiplegic UE in extension and abduction with the hand open (Fig. 18.21).

The patient should practice functional, task-specific locomotor skills: walking forward, backward, sideward, and in crossed stepping (e.g., sidestepping, braiding). Elevation activities (e.g., step-up/step-down activities; lateral step-ups; stair climbing, step-over-step) and

Figure 18.21 Assisted ambulation. The therapist provides support and assists in the lateral weight transfer onto the affected side. The UE is maintained in an inhibitory position (extension abduction and external rotation).

community activities (e.g., walking on ramps, curbs, uneven terrain, over and around obstacles) and those with coincident timing requirements (e.g., crossing the street; stepping on and off elevators or escalators; walking through automatic doors) should be practiced. Initially gait will be slow and deliberate. As control develops, the patient should be encouraged to improve gait speed while maintaining safety. Dual-task activities (e.g., holding a ball, bouncing a ball, carrying a tray, carrying on a conversation) and balance activities (e.g., tandem walking on a line, walking on foam) can be incorporated into the training program as control improves. Timing and reciprocity of LE movements can be improved with the use of progressive treadmill, cycle ergometer, and Kinetron® isokinetic training.

An important goal of training is to have the patient be able to monitor his or her performance and recognize and initiate corrective actions. The patient should be able to vary walking speed and direction, navigate changes in the support surface and the environment while walking. Functional practice in real-life environments will assist the patient in developing the confidence needed for meeting the demands of community reentry.

Enhanced Training Activities. *Limb load monitors* provide biofeedback about the amount of loading or weightbearing on the hemiparetic limb and have been effective in improving stance and gait. Patients receiving this training demonstrate more symmetrical weightbearing and increased stance times on their more affected limb with increased swing times on their less affected limb.[261]

NMES has been used to improve dorsiflexor function and prevent drop foot.[262-264] Multichannel FES (MFES) uses a program developed from individual profiles of EMG and anthropometric measurements to stimulate antagonistic groups of muscles. Significant improvements of gait in patients with stroke have been reported.[265–268] Because the patients had limbs that had been paralyzed for more than 6 months, the results suggest a significant training effect as a result of the intervention.

Orthotics

An orthosis may be required when persistent problems prevent safe ambulation (e.g., inadequate ankle dorsiflexion during swing, mediolateral ankle instability, and insufficient push-off during late stance). Prescription will depend on the unique problems each patient presents. The pattern of instability and weakness at the ankle and knee, and the extent and severity of spasticity and sensory deficits of the limb are major considerations when prescribing an orthosis. Temporary devices (e.g., dorsiflexor assists) may be used during the early stages while recovery is proceeding to allow the patient to practice standing and walking. Use of a temporary orthosis also provides insight into the type of components that will most effectively address the patient's needs. Permanent devices are prescribed once the patient's status stabilizes. Consultation with a certified orthotist and clinic team is initiated if a permanent orthosis is needed.

Foot–Ankle Controls. An ankle–foot orthosis (AFO) is commonly prescribed to control impaired ankle/foot function. These may include a custom molded polypropylene AFO (posterior leaf spring, modified AFO, or solid ankle AFO), or conventional double upright/ dual channel AFO. The least restrictive AFO is the posterior leaf spring (PLS) used to control drop foot. A modified AFO has a slightly wider lateral brim and can provide additional control of calcaneal and forefoot inversion and eversion. A solid ankle molded AFO provides maximum stabilization through its lateral trim lines that project more anteriorly. Movement in all planes (dorsiflexion, plantarflexion, inversion, and eversion) is limited. The conventional double upright metal AFO may be indicated for patients who cannot tolerate plastic AFOs owing to sensory impairments, girth fluctuations, or diabetic neuropathy, or who require additional controls. A posterior stop can be added to limit plantarflexion while a spring assist can be added to assist dorsiflexion (Klenzak joint). Advantages of a conventional AFO include better stabilization of the ankle, allowing improved heelstrike and push-off.[269] Disadvantages include heavier weight, less cosmetic appearance, and increased difficulty donning and doffing. An air-stirrup brace can be used to provide medial–lateral stability at the subtalar joint while allowing dorsiflexion and plantarflexion.[270]

Knee Controls. Knee instability following stroke can be controlled with an AFO by adjusting the position of the ankle. An ankle set in 5° dorsiflexion limits knee hyperextension, while an ankle set in 5° plantarflexion decreases the flexor moment and stabilizes the knee during midstance.[271] A patient with knee hyperextension without foot and/or ankle instability may benefit from the application of a Swedish Knee Cage to protect the knee. Extensive bracing using a knee–ankle–foot orthosis (KAFO) is rarely indicated or successful. The added weight and restrictions in normal knee joint motion significantly increase energy costs and limit independent function.

The therapist must frequently reexamine the patient's motor function and recovery. The need for an orthosis or a particular type of orthosis may change with continuing recovery. The therapist may need to recommend a change in prescription or discontinuing the use of a device. With limited reimbursements, ordering a new orthosis may prove problematic and speaks to the need to anticipate changes when ordering the initial device. For example, a good option for the patient who needs a custom molded solid AFO is to order a hinged AFO with a plantarflexion stop. As the patient regains sufficient knee and dorsiflexor control, the device can be adjusted to remove the stop and allow the hinges to work. Orthotic training should be initiated and includes donning and doffing, skin inspections, and education in safe use

of the device during gait. See Chapter 31 for a more complete description of orthotic devices.

Wheelchairs

Most patients require the use of a wheelchair for mobility at some point during their recovery. Patients with stroke exhibit typical postural asymmetries, which need to be carefully evaluated. These include:

- Trunk laterally flexed to the weaker side; head may also be flexed to the weaker side.
- Pelvic posterior tilt with some obliquity (lower on the unaffected side).
- LE rolled out into abduction and external rotation; if spasticity is present, increased hip extension, adduction, and internal rotation with knee extension may occur; foot is typically plantarflexed and inverted.
- UE held flexed and adducted to the trunk with increased elbow, wrist, and finger flexion. With flaccidity, the shoulder is subluxed with the hand dangling in a dependent position.

Positioning in a wheelchair needs to correct for these postural asymmetries and ensure correct sitting posture. The reader is referred to Chapter 33 for a more complete discussion of general seating principles and wheelchair adaptations.

A patient with stroke can learn to propel a wheelchair using the stronger UE and LE. The seat-to-floor height is critical in ensuring successful use of the foot for steering and propulsion. A *hemi-height wheelchair* with a lower seat to floor height (17.5 in.) may be required. A standard wheelchair has a seat to floor height of 19.5 in. One-arm drive chairs in which both handrims are placed on one wheel were designed for individuals with only one functional UE. The patient with stroke is rarely successful in using this type of chair as it takes a great deal of strength and coordination to propel the wheelchair in a forward direction. It is contraindicated in patients with significant perceptual and coordination impairments. A power wheelchair may be required for some individuals who cannot successfully use a manual wheelchair and will depend upon a wheelchair as their primary means of locomotion. The therapist needs to consider individual needs and reimbursement policies when ordering a wheelchair. It is important to balance both present and future needs as providers restrict frequent reordering of a new wheelchair. It is also important to remember that prolonged used of a wheelchair contributes to learned non-use and may limit recovery, especially if walking is a primary goal of therapy.

Wheelchair training activities include patient and caregiver instruction in the use, maintenance, and safety of all parts of the wheelchair (e.g., brakes, legrests, removable armrests). The patient needs to be instructed in methods of propulsion and given the opportunity to practice on level and varied surfaces (e.g., ramps, outdoor terrain). Transfers (to and from bed, toilet, tub, car) should also be practiced once the patient receives the prescriptive wheelchair.

Strategies to Improve Aerobic Function

Patients with stroke demonstrate decreased levels of physical conditioning following periods of prolonged immobility and reduced activity. The energy costs to complete many functional tasks are higher than normal owing to the abnormal ways in which the activities are performed.[272–275] Many patients also demonstrate concomitant cardiovascular disease and may be recovering from acute cardiac events at the same time.[276] These patients require careful determination of cardiopulmonary responses during exercise and appropriate monitoring. Vital signs, heart rate, and ratings of perceived exertion (RPE) are all important measures of cardiopulmonary adaptation to exercise. Signs and symptoms of exertional intolerance (i.e., excessive fatigue, dyspnea, dizziness, diaphoresis, nausea, vomiting, chest pain) should be monitored closely and can signal an inappropriate level of exercise intensity and cardiac decompensation.[277] Some patients may also require electrocardiographic monitoring to ensure safety during training.

Individuals recovering from stroke can benefit from endurance (aerobic) training to improve cardiovascular function. During the early stages of inpatient rehabilitation, functional activities are appropriate for training (e.g., overground walking). During the postacute stage, patients/clients may be able to engage in more traditional exercise training modes such as treadmill walking or stationary cycling. Patients with balance impairments will benefit from treadmill training or overground walking with a safety harness, or a recumbent cycle ergometer. To ensure safety, patients should receive a thorough examination and supervised exercise test before starting a training program (e.g., symptom-limited graded exercise test). Prescriptive elements include mode (type of exercise), frequency, intensity, and duration (see Chapter 16: Heart Disease). Choice of training mode depends upon the individual's abilities and interests. Intensities are typically in the range of 40 percent to 70 percent of maximal oxygen uptake. Suggested frequency is three times per week for 20 to 60 minutes per session. Frequency may be increased to daily if lower intensities are used. Because of the level of deconditioning, patients with stroke should begin with intermittent training protocols but can be progressed to 30 minutes of continuous exercise.[90,91] The use of a training log or exercise diary is an excellent way to keep track of prescriptive elements, objective measurements (heart rate, blood pressure), and subjective reactions (RPE, perceived enjoyment). Adequate supervision, monitoring, and safety education about warning

signs for impending stroke and heart attack are critical components.

Endurance training programs for patients with stroke have been shown to yield significant improvements in physical fitness, functional status, psychological outlook, and self-esteem.[278–280] Regular exercise may also have the additional benefit of reducing risk of recurrent stroke or heart attack. Finally, patients who participate in a regular conditioning program may be more successful in adopting continuing, life-long exercise habits and in moving beyond the disability associated with stroke.

Strategies to Improve Feeding and Swallowing

A multidisciplinary team, including the speech-language pathologist, occupational therapist, physical therapist, nutritionist, and physician manages dysphagia. The goals/outcomes of dysphagia management are to (1) improve strength, coordination, and range of oral musculature; (2) promote normal feeding through graduated resumption of activities; (3) promote volitional control through effective verbal coaching; and (4) provide effective education and support.

A critical component of dysphagia management is positioning. Posture should be normally aligned with the head held in a chin-down (tucked) position rather than extended or tipped back. This reduces the chances of aspiration or choking and promotes normal swallowing through appropriate alignment of the necessary structures. If the patient lacks adequate head control, the head should be supported either manually or with supports.

Oral exercises including movements of the lips, tongue, cheeks, and jaw should be practiced. The patient is instructed to purse lips and hold a tongue depressor between the lips. Tongue movements in all directions are practiced and can be resisted manually (using a sterile gauze or glove to cover finger or with a moist tongue depressor). Firm pressure applied to the anterior third of the tongue with a tongue depressor can be used to stimulate posterior elevation of the tongue. Cheek exercises include practice in puffing, blowing bubbles, and drinking thick liquids through a straw. Vibrating or pressing above the upper lip for closure and under the lower lip for opening can stimulate jaw movements.[281]

Food presentation is an important part of dysphagia management. Food should be positioned at an appropriate height and distance from the patient and within the patient's visual field. Adapted utensils, plate guards, and nonslip mats can be used to assist in the transfer of food to the mouth. Some guiding may be required to self-feed with more involved UE. Food should be at first semimoist (e.g., pureed food, pasta, boiled chicken), progressing to regular textures and foods rich in taste, smell, and texture, qualities that assist in facilitating the swallowing reflex. Favorite foods should be employed whenever possible. Verbal cues should be given, encouraging the patient to swallow each mouthful and to eat slowly. Stroking the neck can be used to stimulate swallowing. Massaging the check may be helpful in clearing the cheek of a food bolus. Resisted sucking can be promoted using a straw and very thick liquids (slushes, shakes), or by holding the open end of the straw against the finger. As sucking ability improves, the patient can be presented with thinner liquids. Modification of the eating environment is also an important consideration. Every effort should be made to ensure that the environment is pleasant, free from distraction, devoid of unpleasant sights and smells, and provides adequate lighting. The patient's full attention should be directed to the task by using appropriate and consistent verbal cues. The importance of mealtime for social interaction should not be overlooked.[93,281]

Patient/Client-Related Instruction

Stroke represents a major health crisis for patients and their families. Ignorance about the cause of the illness or the recovery process and misconceptions concerning the rehabilitation program and potential outcomes can negatively influence coping responses and progress in rehabilitation. Frequently the problems seem unmanageable and overwhelming for the family, especially when faced with alterations in the patient's behavior, cognition, and emotion. Patients may feel depressed, isolated, irritable, or demanding. Families often demonstrate reactions that include initial relief and hope for full recovery, followed by feelings of entrapment, depression, anger, or guilt when complete recovery does not occur. These changes and feelings can strain even the best of relationships. Therapists can often have a dramatic influence on this situation because of the high frequency of contact and the often close relationships that develop with patients and their families. There are a number of important guidelines to follow when planning educational interventions[2]:

- Give accurate, factual information; counsel family members about the patient's capabilities and limitations; *avoid* predictions that categorically define expected function or future recovery.
- Structure interventions carefully, giving only as much information as the patient or family need or can assimilate; provide reinforcement and repetition.
- Adapt interventions to ensure they are appropriate to the educational and cultural background of the patient and family.
- Offer a variety of educational interventions: didactic sessions, books, brochures, and videotapes, and family participation in therapy. (See Appendix B.)
- Provide a forum for open discussion and communication.

- Be supportive, sensitive, and maintain a positive, hopeful manner.
- Assist patients and families in confronting alternatives and developing problem-solving abilities.
- Motivate and provide positive reinforcement in therapy; enhance patient satisfaction and self-esteem.
- Refer patients and families to support and self-help groups such as the following national associations:

  American Stroke Association—A Division of the American Heart Association
  7272 Greenville Avenue
  Dallas, TX 75231
  1–800–AHA–USA1 (1–800–242–8721)

  National Stroke Association
  9707 East Easter Lane
  Englewood, CO 80112
  1–800–STROKES (1–800–787–6537)

Psychotherapy and counseling (e.g., sexual, leisure, vocational) can assist in improving overall quality of life and should be recommended as needed.

Discharge Planning

Planning for discharge begins early in rehabilitation and involves the patient and family. Potential placement (safe place of residence), level of family and community support, and need for continued medical and rehabilitation services should all be explored. Family members should regularly participate in therapy sessions to learn exercises and activities designed to support the patient's independence. Discharge should be considered when reasonable treatment goals/outcomes are attained. Indication of the attainment of a functional ceiling can be considered when there is lack of evidence of progress at two successive evaluations over a period of 2 weeks. Home visits should be made prior to discharge to determine the home's physical structure and accessibility. Potential problems can be identified and corrective measures initiated. Home adaptations, assistive devices, and supportive services should be in place before the patient is discharged to home. Several trial home stays may be helpful in smoothing the transition from rehabilitation center to home. Patients with residual impairments or functional limitations who will be receiving outpatient or home therapy should be given all the necessary information concerning these services. Community services should be identified and information provided to the patient and family. Long-term follow-up at regularly scheduled intervals should be initiated in order to maintain patients at their highest possible functional level.

Stroke Rehabilitation Outcomes

Most patients with stroke regain their independent living status following discharge. The Copenhagen Stroke Study, based on 1197 patients, revealed that 64 percent of patients were discharged to home, 15 percent were discharged to a nursing home, and 21 percent of patients died during their hospital stay. After rehabilitation, 11 percent of survivors still exhibited severe or very severe deficits, 11 percent had moderate deficits, and 78 percent had mild or no deficits.[282] Functional recovery was completed within 12.5 weeks of stroke onset in 95 percent of patients.[110] Recovery of walking function occurred in 61 percent of survivors (50 percent were independent while 11 percent required assistance).[111] Only 30 to 60 percent of stroke survivors regain independence in ADL. Major problems in stroke outcome studies include the heterogeneity of patients admitted for rehabilitation, lack of consistency in outcome measures, and differences in duration, type, and onset of rehabilitation programs. Patients who demonstrate less successful rehabilitation outcomes tended to include those with (1) advanced age; (2) severe motor impairments (prolonged paralysis, apraxia); (3) persistent medical problems (incontinence); (4) impaired cognitive function (decreased alertness, poor attention span, judgment, memory), severe language disturbances, and an inability to learn new tasks or follow simple commands; (5) severe visuospatial hemineglect; and (6) other less well-defined social and economic problems.[283–285]

Summary

Stroke can result from a number of different vascular events that interrupt cerebral circulation and impair brain function, including cerebral thrombosis, emboli, or hemorrhage. The location and size of the ischemic process, the nature and functions of the structures involved, the availability of collateral blood flow, and effectiveness of early emergency medical management all influence the symptomatology that evolves. For many patients, stroke represents a major cause of disability, with diffuse problems affecting widespread areas of function. From a practical standpoint, patients with stroke present a tremendous challenge for clinicians. Effective rehabilitation should take advantage of the brain's capacity for repair and recovery. Rehabilitation interventions seek to promote recovery and independence through neurofacilitation, functional, and compensatory training strategies. Interventions also focus on the prevention of secondary impairments. The utilization of effective motor learning strategies with task-oriented training for real-life environments is critical for the successful attainment of functional outcomes.

Questions for Review

1. Differentiate between each of the following vascular syndromes: anterior cerebral artery, middle cerebral artery, internal carotid artery, posterior cerebral artery, lacunar, and vertebrobasilar artery. What are the differences that can be expected between hemispheric lesions?
2. What are the major causes of stroke? Define and explain each.
3. What diagnostic measures are used to confirm stroke? Describe the role of the CT scan in the implementation of emergency medical measures.
4. Describe the normal recovery process in stroke including expected stages of motor recovery. Give an example of how this knowledge may influence selection of interventions.
5. What are the major communication, cognitive, and emotional impairments that can result from stroke? Where are the likely lesions?
6. Differentiate between the following stroke-specific instruments: the Fugl-Meyer Assessment of Physical Performance (FMA) and the Stroke Rehabilitation Assessment of Movement (STREAM).
7. Identify three motor learning strategies important for treating the patient with stroke.
8. What are the major goals for reestablishing postural control? Identify three training activities that could be used during the post-acute stage to reestablish postural control and functional mobility.
9. What are the essential elements of an intervention program designed to improve UE function? Identify three training activities.
10. What are the essential elements of an intervention program designed to improve LE function? Identify three training activities.
12. Describe the benefits of locomotor training using body weight support and a treadmill. How should training be progressed?
13. Identify and describe common LE orthotic devices used for the patient with stroke. What are the major indications and contraindications for each?
14. Differentiate between the focus of rehabilitation efforts during the acute and postacute phases of stroke rehabilitation.
15. What are the essential elements included in an educational program for the patient with stroke and family members?

Case Study

HISTORY

The patient/client is a 41-year-old man admitted to an acute care hospital with a diagnosis of CVA with R hemiparesis (L MCA). Admitted to a rehabilitation facility 10 days later.

PAST MEDICAL HISTORY

- Seizure disorder since childhood. Dilantin was discontinued 5 years ago.
- History of mild hypertension well controlled with medication.
- Smokes 1 pack/day; 20-year history.

MEDICATIONS

Persantine 50 mg po tid
Tenormin 25 mg po qd
Aspirin 10 grains po bid

TESTS

- Carotid angiography: complete occlusion of left internal carotid artery.
- Cardiac ultrasound: intermittent mitral valve prolapse.
- EKG: nonspecific ST wave changes.
- CT scan: initial scan unremarkable; repeat CT scan consistent with a large left middle cerebral artery ischemic infarction.

SOCIAL HISTORY

Patient lives with his wife and three teenage children and was independent and active prior to CVA. He has a college education and has worked for 20 years as a computer programmer. There is a two-step access to a rented, single-family house.

COGNITION

- Disoriented to time.
- Good attention span for 30- to 45-minute treatment session.
- Difficult to examine further owing to language impairment; cognitive deficits likely.
- Patient has difficulty following directions for motor responses; two- or three-step commands.

LANGUAGE/COMMUNICATION

- Auditory comprehension: moderate to severe decrease in understanding words and simple concrete sentences; unreliable yes/no responses.
- Verbal expression: severely decreased to nonfunctional; limited to only occasional automatic words.
- Reading comprehension: severely decreased to nonfunctional at a word level. Unable to match word to object.

- Written expression: to be determined.
- Gestures: spontaneous use of gestures not evident.

PHYSICAL THERAPY EXAMINATION

PROM

- BUEs WNL; R shoulder pain at end ranges
- BLEs WNL except R dorsiflexion 0 to 5°

Tone

- RUE increased tone (moderate to severe) in elbow flexors; shoulder adductors and internal rotators.
- RLE increased tone (moderate) in hip and knee extensors, plantar flexors.

Motor Control

- RUE: partial motion (1/2 range) in extensor synergy pattern (shoulder and elbow extension); no voluntary motion of hand; cannot perform flexor synergy pattern.
- Demonstrates RUE neglect and often lets UE drop off the wheelchair.
- RLE: full motion in both extensor and flexor synergy patterns with extensor pattern dominating; extensor synergy achieved with associated reaction of RUE (extensor synergy).
- LUE and LLE: full isolated movement with G+ to N strength.

Sensory

- RUE: impaired, severity difficult to determine owing to communication deficits.
- No apparent sensation to sharp/dull stimuli; moderate decrease in light touch sensitivity.
- RLE: few consistent responses to sharp/dull stimuli proximally; moderate to severe decrease distally; no sensation noted on dorsum of foot.
- Patient reports pain in both RUE and RLE.
- Proprioception: inappropriate responses; difficult to determine owing to patient's difficulty with understanding of questions being asked.

Coordination

- LUE and LLE intact.
- RUE and RLE unable to test.

Postural Control/Balance

- Head control: good
- Sitting static control: good, able to maintain balance without support; maintains centered alignment (COM) for 5 minutes.
- Sitting dynamic control: fair, able to maintain balance; weight shifting with reduced limits of stability (LOS); shifts to R are reduced 50%; shifts to L are normal.
- Standing static control: fair, able to maintain independent standing in parallel bars for up to 1 minute with LUE handhold.
- Standing dynamic control: poor; unable to weight shift to R without loss of balance; weight shifts to L are reduced 50%.

Motor Planning

Appears to have mild motor apraxia; difficult to determine owing to communication impairments.

Endurance

Tolerates 3/4-hour treatment session with occasional rests.

Functional Status

- Rolls to R: Independent with bed rail
- Rolls to L: Min assist
- Scoots up in bed: Supervision
- Supine-to-sit: Min assist
- Sit-to-supine: Min assist
- Transfers bed-to-chair: Stand pivot transfer, Mod assist (FIM 3)
- W/C mobility: propels 150 ft with supervision (FIM 5); uses LUE and L foot for propulsion
- Locomotion: ambulates 10 ft in parallel bars with max assist of one (FIM 1).
  Requires assist in initiation of movement of RLE.
  Requires assist with R knee control for extension.
  R foot is plantarflexed and supinated during stance, foot drop during swing.
  AFO ordered (solid ankle AFO)
- Stairs: to be determined at a later date.
- Eating: Supervision (FIM 5)
- Bathing: Mod assist RUE and RLE (FIM 3)
- Dressing: Mod assist RUE and RLE (FIM 3)

(FIM = Functional Independence Measure)

PSYCHOSOCIAL

Patient is motivated and cooperative. He appears anxious about his future and exhibited a brief episode of crying during the initial therapy session. Family is supportive and anxious to have him home again.

GUIDING QUESTIONS

1. Identify/categorize this patient's problems in terms of:
   a. Direct impairments.
   b. Indirect impairments.
   c. Functional limitations/disabilities
2. Identify anticipated goals (remediation of impairments) and expected outcomes (remediation of functional limitations/disability) for this patient.
3. Formulate five treatment interventions with one progression that could be used during the first 3 weeks of therapy. Provide a brief rationale that justifies your choice.
4. Identify relevant motor learning strategies appropriate for the initial physical therapy sessions with this patient.

References

1. American Heart Association: Heart and Stroke Statistical—2005 Update. American HeartAssociation, Dallas, 2005.
2. Post-Stroke Rehabilitation Guideline Panel: Post-Stroke Rehabilitation Clinical Practice Guideline. Aspen, Gaithersburg, MD, 1996 (formerly published as AHCPR Publication No. 95-0662, May 1995).
3. Wolfe, C: The impact of stroke. Br Med Bul 56:275, 2000.
4. Murray, C, and Lopez, A: The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020. Harvard University Press, Boston, 1996.
5. Granger, C, and Hamilton, B: The Uniform Data System for Medical Rehabilitation report of first admissions for 1992. Am J Phys Med Rehabil 73:51, 1994.
6. American Stroke Association: Stroke Warning Signs. American Heart Association, Dallas Texas, 2000.
7. The National Institute of Neurological Disorders and Stroke rt-PA Study Group: Tissue plasminogen activator for acute ischemic stroke. N Engl J Med 333:1581, 1995.
8. Hack, W, et al, for the European Cooperative Acute Stroke Study (ECASS) Group: Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke. JAMA 274:1017, 1995.
9. Wehrmacher, W, Iqbal A, and Messmore, H: Brain attack: Who will write the orders for thrombolytics? Arch Int Med 160:119, 2000.
10. Kwiatkowski, T, et al: Effects of tissue plasminogen activator or acute ischemic stroke at one year. N Engl J Med 340:1781, 1999.
11. Gilroy, J: Basic Neurology, ed 3. McGraw-Hill, New York, 2000.
12. Hachinski, V and Norris, J: The Acute Stroke. FA Davis, Philadelphia, 1985.
13. Curtis, S, and Porth, C: Disorders of brain function. In Porth, C (ed): Pathophysiology, ed. 5. Lippincott, Philadelphia, 1998, p 879.
14. Haig, A, et al: Locked-in syndrome: A review. Curr Concepts Rehabil Med 2:12, 1986.
15. Haig, A, et al: Mortality and complications of the locked-in syndrome. Arch Phys Med Rehabil 68:24, 1987.
16. Kaplan, P, Cailliet, R, and Kaplan, C: Rehabilitation of Stroke. Butterworth-Heinemann, Woburn, MA, 2003.
17. Bogousslavsky, J, et al: The Lausanne stroke registry: Analysis of 1,000 consecutive stroke patients. Stroke 19: 1083, 1988.
18. Smith, D, et al: Proprioception and spatial neglect after stroke. Age Ageing 12:63, 1983.
19. Fields, H: Pain. McGraw-Hill, New York, 1987.
20. Twitchell, T: The restoration of motor function following hemiplegia in man. Brain 47:443, 1951.
21. Brunnstrom, S: Motor testing procedures in hemiplegia based on recovery stages. J Am Phys Ther Assoc 46:357, 1966.
22. Brunnstrom, S: Movement Therapy in Hemiplegia. Harper & Row, New York, 1970.
23. Bobath, B: Adult Hemiplegia: Evaluation and Treatment, ed 2. Heinemann, London, 1978.
24. Fugl-Meyer, A, et al: The post stroke hemiplegic patient, 1. A method for evaluation of physical performance. Scand J Rehabil Med 7:13, 1976.
25. Gray, C, et al: Motor recovery following acute stroke. Age Ageing 19:179, 1990.
26. Wade, D, et al: Recovery after stroke: The first 3 months. J Neurol Neurosurg Psychiatry 48:7, 1985.
27. Colebatch, J, and Gandevia, S: The distribution of muscular weakness in upper motor neuron lesions affecting the arm. Brain 112:749, 1989.
28. Adams, R, Gandevia, S, and Skuse, N: The distribution of muscle weakness in upper motoneuron lesions affecting the lower limb. Brain 113: 1459, 1990.
29. Davidoff, R: The pyramidal tract. Neurology 40:332, 1990.
30. Andrews A, and Bohannon, R: Distribution of muscle strength impairments following stroke. Clinical Rehab 14:79, 2000.
31. McComas, A, et al: Functional changes in motoneurons of hemiparetic patients. J Neurol Neurosurg Psychiatry 36: 183, 1973.
32. Eng, J: Strength training in individuals with stroke. Physiother Can, 56:189, 2004.
33. Weightman, M: Motor unit behavior following cerebrovascular accident. Neurology Report (now JNPT) 18:26, 1994.
34. Dattola, R, et al: Muscle rearrangement in patients with hemiparesis after stroke: An electrophysiological and morphological study. Eur Neurol 33:109, 1993.
35. Tang, A, and Rymer, W: Abnormal force: EMG relations in paretic limbs of hemiparetic human subjects. J Neurol Neurosurg Psychiatry 44:690, 1981.
36. Rosenfalck, A, and Andreassen, S: Impaired regulation of force and firing pattern of single motor units in patients with spasticity. J Neurol Neurosurg Psychiatry 43:907, 1980.
37. Canning, C, Ada, L, and O'Dwyer, N: Slowness to develop force contributes to weakness after stroke. Arch Phys Med Rehabil 80:66, 1999.
38. Spaans, F, and Wilts, G: Denervation due to lesions of the central nervous system: An EMG study in cases of cerebral contusion and cerebrovascular accidents. J Neurol Sci 57:291, 1982.
39. Dickstein, R, et al: Reaction and movement times in patients with hemiparesis for unilateral and bilateral elbow flexion. Phys Ther 73:37, 1993.
40. Buonocore, M, et al: Psychomotor skills in hemiplegic patients: Reaction time differences related to hemispheric lesion side. Neurophysiol Clin 20:203, 1990.
41. Gowland, C, et al: Agonist and antagonist activity during voluntary upper-limb movement in patients with stroke. Phys Ther 72:624, 1992.
42. Knutsson, E, and Martensson, C: Dynamic motor capacity in spastic paresis and its relationship to prime mover dysfunction, spastic reflexes and antagonistic co-ordination. Scand J Rehabil Med 12:93, 1980.
43. Hufschmidt, A, and Mauritz, K: Chronic transformation of muscle in spasticity: A peripheral contribution to increased tone. J Neurol Neurosurg Psychiatry 48:676, 1985.
44. Schenkman, M, and Butler, R: Automatic postural tone in posture, movement, and function. Forum on physical therapy issues related to cerebrovascular accident. American Physical Therapy Association, Alexandria, VA, 1992.
45. Michels, E: Synergies in hemiplegia. Clin Manage 1:9, 1981.
46. Sahrmann, S, and Norton, B: The relationship of voluntary movement to spasticity in the upper motor neuron syndrome. Ann Neurol 2:460, 1977.
47. Bobath, B: Abnormal Postural Reflex Activity Caused by Brain Lesions, ed 3. Heinemann, London, 1985.
48. Mulley, G: Associated reactions in the hemiplegic arm. Scand J Rehabil Med 14:17, 1982.
49. Horak, F, et al: The effects of movement velocity, mass displaced and task certainty on associated postural adjustments made by normal and hemiplegic individuals. J Neurol Neurosurg Psychiatry 47:1020, 1984.
50. Dickstein, R, et al: Foot-ground pressure pattern of standing hemiplegic patients: Major characteristics and patterns of movement. Phys Ther 64:19, 1984.
51. Mizrahi, J, et al: Postural stability in stroke patients: Vectorial expression of asymmetry, sway activity, and relative sequence of reactive forces. Med Bio Eng Comput 27:181, 1989.
52. DiFabio, R, and Badke, M: Relationship of sensory organization to balance function in patients with hemiplegia. Phys Ther 70:543, 1990.
53. Shumway-Cook, A, Anson, D, and Haller, S: Postural sway biofeedback: Its effect on reestablishing stance stability in hemiplegic patients. Arch Phys Med Rehabil 69:395, 1988.
54. Badke, M, and DiFabio, R: Balance deficits in patients with hemiplegia: Considerations for assessment and treatment. In Duncan, P (ed): Balance: Proceedings of the APTA Forum. American Physical Therapy Association, Alexandria, VA, 1990, p 73.
55. Duncan, P, and Badke, M: Determinants of abnormal motor control. In: Duncan, P, and Badke, M (eds): Stroke Rehabilitation: The Recovery of Motor Control. Year Book Medical, Chicago, 1987, p 135.

56. Davies, P: Steps to Follow: The Comprehensive Treatment of Patients with Hemiplegia, ed 2. Springer-Verlag, New York, 2000.
57. Karnath, H, Ferber, S, and Dichgans, J: The origin of contraversive pushing: Evidence for a second graviceptive system in humans. Neurology 55:1298, 2000.
58. Karnath, H, Ferber, S, and Dichgans, J: The neural representation of postural control in humans. PNAS 97:13931, 2000.
59. Perennou, D, et al: Understanding the pusher behavior of some stroke patients with spatial deficits: A pilot study. Arch Phys Med Rehabil 83:570, 2002.
60. Pedersen, P, et al: Ipsilateral pushing in stroke: Incidence, relation to neuropsychological symptoms, and impact on rehabilitation. The Copenhagen stroke study. Arch Phys Med 77:25, 1996.
61. Karnath, H, and Broetz, D: Understanding and treating "pusher syndrome." Phys Ther 83:1119, 2003.
62. Veis, S, and Logemann, J: Swallowing disorders in persons with cerebrovascular accident. Arch Phys Med Rehabil 66:372, 1985.
63. Arnadottir, G: Impact of neurobehavioral deficits on activities of daily living. In Gillen, G, and Burkhardt, A (eds): Stroke Rehabilitation: A Function-Based Approach, ed 2. CV Mosby, St. Louis, 2005, p 285.
64. Starkstein, S, and Robinson, R: Neuropsychiatric aspects of stroke. In Coffey, C, and Cummings, J (eds): Textbook of Geriatric Neuropsychiatry. American Psychiatric Press, Washington, DC, 1994, p 457.
65. Hibbard, MR, and Gordon, WA: The comprehensive psychological assessment of individuals with stroke. J Neurol Rehabil 2:9, 1992.
66. Robinson, R, et al: Pathological laughing and crying following stroke: Validation of a measurement scale and a double-blind study. Am J Psychiatry 150:286, 1993.
67. Binder, L: Emotional problems after stroke. Stroke 15:174, 1984.
68. Robinson, R, et al: A two-year longitudinal study of post-stroke mood disorders: Diagnosis and outcome at one and two years. Stroke 18:837, 1987.
69. Remer-Osborn, J: Psychological, behavioral, and environmental influences on post-stroke recovery. Top Stroke Rehabil 5(2) 45, 1998.
70. Robinson, R: An 82-year-old woman with mood changes following a stroke. JAMA 283(12):1607, 2000.
71. Robinson, R, and Price, T: Post-stroke depressive disorders: A follow-up study of 103 patients. Stroke 13:635, 1982.
72. Robinson, R, and Benson, D: Depression in aphasic patients: Frequency, severity, and clinical-pathological correlations. Brain Lang 14:282, 1981.
73. Diller, L: Perceptual and intellectual problems in hemiplegia: Implications for rehabilitation. Med Clin North Am 53:575, 1969.
74. American Heart Association: How Stroke Affects Behavior. American Heart Association, Dallas, 1994.
75. Geschwind, N: Specialization of the human brain. Sci Am 241:80, 1979.
76. Bohannon, R: Selected determinants of ambulation capacity in patients with hemiplegia. Clin Rehab 3:47, 1989.
77. Bohannon, R: Correlation of lower limb strengths and other variables in standing performance in stroke patients. Physiother Can 41:198, 1989.
78. Hamrin, E, et al: Muscle strength and balance in post stroke patients. Ups J Med Sci 87:11, 1982.
79. Morris, S, Dodd, K, and Morris, M: Outcomes of progressive resistance strength training following stroke: A systematic review. Clin Rehab 18:27, 2004.
80. Harris, J, et al: Relationship of balance and mobility to fall incidence in people with chronic stroke. Phys Ther 85:150, 2005.
81. Byers, V, Arrington, M, and Finstuen, K: Predictive risk factors associated with stroke patient falls in acute care settings. J Neurosci Nurs 22: 147, 1990.
82. Nyberg, L, and Gustafson, Y: Patient falls in stroke rehabilitation: A challenge to rehabilitation strategies. Stroke 26:838, 1995.
83. Teasall, R, et al: The incidence and consequences of falls in stroke patients during inpatient rehabilitation: Factors associated with high risk. Arch Phys Med Rehabil 83:329, 2002.
84. Tutuarima, J, et al: Risk factors for falls of hospitalized stroke patients. Stroke 28:297, 1997.
85. Forster, A, and Young, J: Incidence and consequences of falls due to stroke: A systematic inquiry. Br Med J 311:83, 1995.
86. Ramnemark, A, et al.: Fractures after stroke. Osteoporos Int 8:92, 1998.
87. Cocito, L, et al: Epileptic seizures in cerebral arterial occlusive disease. Stroke 13:189, 1982.
88. Clagett, GP, et al: Prevention of venous thromboembolism. Chest 102(Suppl):391, 1992.
89. Buck, L: The coincidence of heart disease and stroke: Pathogenesis and considerations for therapy. Neurology Report (now JNPT) 18:29, 1994.
90. Palmer-McLean, K, and Wilberger, J: Stroke and head injury. In American College of Sports Medicine: ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities. Human Kinetics, Champaign IL, 1997, p 169.
91. Rimmer, J, and Nicola, T: Stroke. In American College of Sports Medicine: ACSM's Resources for Clinical Exercise Physiology: Musculoskeletal, Neuromuscular, Neoplastic, Immunologic, and Hematologic Conditions. Lippincott, Williams & Wilkins, Philidelphia, 2002, p 3.
92. Mol, V, and Baker, C: Activity intolerance in the geriatric stroke patient. Rehabil Nurs 16:337, 1991.
93. Avery-Smith, W: Dysphagia management. In Gillen, G, and Burkhardt, A (eds): Stroke Rehabilitation: A Function-Based Approach, ed 2. CV Mosby, St. Louis, 2004, p 514.
94. Roth, EJ: Medical complications encountered in stroke rehabilitation. Phys Med Rehabil Clin North Am 2:563, 1991.
95. Stein, D, Brailowsky, S, and Will, B: Brain Repair. Oxford University Press, New York, 1995.
96. Wolf, S, et al: Forced use of hemiplegic upper extremities to reverse the effect of learned nonuse among chronic stroke and head-injured patients. Exp Neurol 104:125, 1989.
97. Liepert, J, et al: Motor cortex plasticity during constraint-induced movement therapy in stroke patients. Neurosci Lett 250:5, 1998.
98. Miltner, W, et al: Effects of constraint-induced movement therapy on patients with chronic motor deficits after stroke: A replication. Stroke 30: 586, 1999.
99. Kunkel, A, et al: Constraint-induced movement therapy for motor recovery in chronic stroke patients. Arch Phys Med Rehabil 80:624, 1999.
100. Taub, E, et al: Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehabil 74:347, 1993.
101. Visintin, M, et al.: A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. Stroke 29:1122, 1998.
102. Hendricks, H, et al.: Motor recovery after stroke: A systematic review of the literature. Arch Phys Med Rehabil 83:1629, 2002.
103. Dombovy, M, and Bach-y-Rita, P: Clinical observations on recovery from stroke. Adv Neurol 47:265, 1988.
104. Kelly-Hayes, M, et al: Time course of functional recovery after stroke: The Framingham Study. J Neurol Rehabil 3:65, 1989.
105. Duncan, P, et al: Measurement of motor recovery after stroke. Outcome assessment and sample size requirements. Stroke 23:1084, 1992.
106. Kwakkel, G, et al: Predicting disability in stroke—A critical review of the literature. Age Ageing 25:479, 1996.
107. Andrews, K, et al: The rate of recovery from stroke—and its measurement. Int Rehabil Med 3:155, 1981.
108. Binkofski, F: Recovery of motor functions following hemiparetic stroke—a clinical and magnetic resonance morphometric study. Cerebrovasc Dis 11:273, 2001.
109. Bonita, R, and Beaglehole, R: Recovery of motor function after stroke. Stroke:19:1497, 1988.
110. Jorgenson, H, et al: Outcome and time course of recovery. Part II: Time course of recovery. The Copenhagen Stroke Study. Arch Phys Med Rehabil 76:406, 1995.
111. Jorgensen, H, et al: Recovery of walking function in stroke patients: The Copenhagen Stroke Study. Arch Phys Med Rehabil 76:27, 1995.
112. Nakayama, H, et al: Compensation in recovery of upper extremity function after stroke: The Copenhagen Stroke Study. Arch Phys Med Rehabil 75:852, 1994.
113. Samuelsson, M, Soderfeldt, B, and Olsson, G: Functional outcome in patients with lacunar infarction. Stroke 27:842, 1996.
114. Ferrucci, L, et al: Recovery of functional status after stroke: A postrehabilitation follow-up study. Stroke 24:200, 1993.

115. National Stroke Association: The Complete Guide to Stroke. National Stroke Association, Englewook, CO, 2003.
116. Langhorne, P, et al: Do stroke units save lives? Lancet 342:395, 1993.
117. Hayes, S, and Carroll, S: Early intervention care in the acute stroke patient. Arch Phys Med Rehabil 67:319, 1986.
118. Strand, T, et al: A non-intensive stroke unit reduces functional disability and the need for long-term hospitalization. Stroke 16:29, 1985.
119. Hamrin, E: Early activation in stroke: Does it make a difference? Scand J Rehabil Med 14:101, 1982.
120. Stroke Unit Trialists' Collaboration: Organised inpatient (stroke unit) care after stroke (Cochrane Review). The Cochrane Library, Issue 3. John Wiley & Sons, Ltd, Chichester, UK, retrieved August 20, 2005 from: http://www.cochrane.org/reviews/en/ab000197.html.
121. Kalra, L, et al: Medical complications during stroke rehabilitation. Stroke 26:990, 1995.
122. Dombovy, M, Sandok, B, and Basford, J: Rehabilitation for stroke: A review. Stroke 17:363, 1986.
123. Inaba, M, et al: Effectiveness of functional training, active exercise, and resistive exercise for patients with hemiplegia. Phys Ther 53:28, 1973.
124. Ernst, E. A review of stroke rehabilitation and physiotherapy. Stroke, 21:1081, 1990.
125. Kwakkel, G, et al: Intensity of leg and arm training after primary middle-cerebral-artery stroke: A randomized trial. Lancet 354:191, 1999.
126. Miller, K, Garland, S, and Koshland, G: Techniques and efficacy of physiotherapy poststroke. Phys Med Rehabil 12:473, 1998.
127. Paolucci, S, et al: Early versus delayed inpatient stroke rehabilitation: A matched comparison conducted in Italy. Arch Phys Med Rehabil 81:695, 2000.
128. Feigenson, J, et al: Factors influencing outcome and length of stay in a stroke rehabilitation unit. Part 1: Analysis of 248 unscreened patients. Medical and functional prognostic indicators. Stroke 8:651, 1977.
129. Shay, S, Vanclay, F, and Cooper, B: Predicting discharge status at commencement of stroke rehabilitation. Stroke 20:766, 1989.
130. Novack, T, Satterfield, W, and Connor, M: Stroke onset and rehabilitation: Time lag as a factor in treatment outcome. Arch Phys Med Rehabil 65:316, 1984.
131. Johnston, M, and Keister, M: Early rehabilitation for stroke patients: A new look. Arch Phys Med Rehabil 65:437, 1984.
132. American Physical Therapy Association: Guide to Physical Therapist Practice, ed 2. Phys Ther 81:1, 2001.
133. Duncan, P, et al: Randomized clinical trial of therapeutic exercise in subacute stroke. Stroke 34:2173, 2003.
134. Werner, R, and Kessle, S: Effectiveness of an intensive outpatient rehabilitation program for post acute stroke patients. Am J Phys Med Rehabil 75:114, 1996.
135. Rodriquez, A, et al: Gait training efficacy using a home-based practice model in chronic hemiplegia. Arch Phys Med Rehabil 77:801, 1996.
136. von Koch, L, et al: A randomized controlled trial of rehabilitation at home after stroke in southwest Stockholm: Outcome at six months. Scand J Rehab Med 32:80, 2000.
137. Geddes, J, and Chamberlain, A: Home-based rehabilitation for people with stroke: A comparative study of six community services providing co-ordinated, multidisciplinary treatment. Clin Rehabil 15:589, 2001.
138. Eng, J, et al: A community-based group exercise program for persons with chronic stroke. Med Sci Sports Exerc 35:1271, 2003.
139. Teasdale, G, and Jennett, B: Assessment of coma and impaired consciousness: A practical scale. Lancet 13:2, 1974.
140. Folstein, MF, et al: Mini mental state: A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 12:189, 1975.
141. Beck, A, and Beck, R: Screening depressed patients in family practice: A rapid technique. Postgrad Med 52:81, 1972.
142. Bohannon, R, and Smith, M: Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 67:206, 1987.
143. Bohannon, R: Measurement and treatment of paresis in the geriatric patient. Top Geriatr Rehabil 7:15, 1991.
144. Badke, M, and DiFabio, R: Balance deficits in patients with hemiplegia: Considerations for assessment and treatment. In Duncan, P (ed): Balance: Proceedings of the APTA Forum. American Physical Therapy Association, Alexandria, VA, 1990, p 23.
145. Badke, M, and Duncan, P: Patterns of rapid motor responses during postural adjustments when standing in healthy subjects and hemiplegic patients. Phys Ther 63:13, 1983.
146. Berg, KO, et al: Measuring balance in the elderly: Preliminary development of an instrument. Physiother Can 41:304, 1989.
147. Berg, KO, et al: Measuring balance in the elderly: Validation of an instrument. Can J Public Health 83 (Suppl):7, 1992.
148. Fugl-Meyer, A: Post-stroke hemiplegia assessment of physical properties. Scand J Rehabil Med 7:85, 1980.
149. Benaim, C, et al: Validation of a standardized assessment of postural control in stroke patients: The Postural Assessment Scale for Stroke Patients (PASS). Stroke 30 (9): 1862, 1999.
150. Duncan, P, et al: Functional reach: A new clinical measure of balance. J Gerontol 45:192, 1990.
151. Tinetti, ME: Performance-oriented assessment of mobility problems in elderly patients. J Am Geriatr Soc 34:119, 1986.
152. Podsiadlo, D, and Richardson, S: The timed "Up and Go": A test of basic functional mobility for frail elderly persons. J Am Geriatr Soc 39:142, 1991.
153. Shumway-Cook, A, and Horak, F: Assessing the influence of sensory interaction on balance: Suggestion from the field. Phys Ther 66:1548, 1986.
154. Nashner, LM: Sensory, neuromuscular, and biomechanical contributions to human balance. In Duncan, P (ed): Balance: Proceedings of the APTA Forum. American Physical Therapy Association, Alexandria VA, 1990, p 4.
155. Richards, C, Malouin, F, and Dean, C: Gait in stroke: Assessment and rehabilitation. Clin Geriatr Med 15:833, 1999.
156. Turnbull, G, and Wall, J: The development of a system for the clinical assessment of gait following a stroke. Physiotherapy 71:294, 1985.
157. Holden, M, et al: Clinical gait assessment in the neurologically impaired: Reliability and meaningfulness. Phys Ther 64:35, 1984.
158. Salbach, N, et al: Responsiveness and predictability of gait speed and other disability measures in acute stroke. Arch Phys Med Rehabil 82:1204, 2001.
159. Dean, C, Richards, C, and Malouin, F: Walking speed over 10 meters overestimates locomotor capacity after stroke. Clin Rehabil 15:415, 2001.
160. Sadaria, K, and Bohannon, R: The 6-Minute Walk Test: A brief review of literature. Clin Exerc Physiol 3:127, 2001.
161. Harada, N, Chiu, V, and Stewart, A: Mobility-related function in older adults: Assessment with a 6-Minute Walk Test. Arch Phys Med Rehabil 80:837, 1999.
162. Kervio, G, Carre, F, and Ville, N: Reliability and intensity of the Six-Minute Walk Test in healthy elderly subjects. Med Sci Sports Exer 35:169, 2003
163. Miller, P, Moreland, J, and Stevenson, T: Measurement properties of a standardized version of the Two-Minute Walk Test for Individuals with Neurological Dysfunction. Physiother Can 54(4):241, 2002.
164. Wolf, S, et al: Establishing the reliability and validity of measurements of walking time using the Emory Functional Ambulation Profile. Phys Ther 79:1122, 1999.
165. Baer, H, and Wolf, S: Modified Emory Functional Ambulation Profile. Stroke 32:973, 2001.
166. Perry, J, et al: Classification of walking handicap in the stroke population. Stroke 26:982. 1995.
167. Mahoney, F, and Barthel, D: Functional evaluation: Barthel Index. Md State Med J 14:61, 1965.
168. Keith, RA, et al: The Functional Independence Measure. Adv Clin Rehabil 1:6, 1987.
169. Uniform Data Service, Data Management Service: UDS Update. State University of New York at Buffalo, 1993.
170. Duncan, P, et al: Reliability of the Fugl-Meyer Assessment of Sensorimotor Recovery following cerebrovascular accident. Phys Ther 63:1606, 1983.
171. Wityk, RJ, Pessin, MS, and Kaplan, RF: Serial assessment of acute stroke using the NIH stroke scale. Stroke 25(2):362, 1994.

172. Goldstein, LB, and Samsa, GP: Reliability of the national institutes of health stroke scale. Stroke 28: 307, 1997.
173. Daley, K, et al.: The Stroke Rehabilitation Assessment of Movement (STREAM): Refining and validating the content. Physiother Can 49:269, 1997.
174. Daley, K, Mayo, N, and Wood-Dauphinee, S: Reliability of scores on the Stroke Rehabilitation Assessment of Movement (STREAM) measure. Phys Ther 79:8, 1999.
175. Ahmed, S, et al: The Stroke Rehabilitation Assessment of Movement (STREAM): A comparison with other measures used to evaluate effects of stroke and rehabilitation. Phys Ther 83: 617, 2003.
176. Carr, J, et al: Investigation of a new motor assessment scale for stroke patients. Phys Ther 65:175, 1985.
177. Pool, J, and Whitney, S: Motor Assessment Scale for stroke patients: Concurrent validity and interrater reliability. Arch Phys Med Rehabil 69:195, 1988.
178. Voss, D, et al: Proprioceptive Neuromuscular Facilitation, ed 3. Harper & Row, Philadelphia, 1985.
179. Adler, S, et al: PNF in Practice, ed 2. Springer-Verlag, Berlin, 2003.
180. Sackett, D, et al: Evidence-based Medicine, ed 2. Churchill Livingstone, New York, 2000.
181. Ernst, E: A review of stroke rehabilitation and physiotherapy. Stroke 21:1082, 1990.
182. Logigian, M, et al: Clinical exercise trial for stroke patients. Arch Phys Med Rehabil 64:364, 1983.
183. Stern, P, et al: Effects of facilitation exercise techniques in stroke rehabilitation. Arch Phys Med Rehabil 51:526, 1970.
184. Lord, J, and Hall, K: Neuromuscular reeducation versus traditional programs for stroke rehabilitation. Arch Phys Med Rehabil 67:88, 1986.
185. Dickstein, R, et al: Stroke rehabilitation: Three exercise therapy approaches. Phys Ther 66:1233, 1986.
186. Jongbloed, L, et al: Stroke rehabilitation: Sensorimotor integrative treatment versus functional treatment. Am J Occup Ther 43:391, 1989.
187. Wagenaar, R, et al: The functional recovery of stroke: A comparison between neuro-developmental treatment and the Brunnstrom method. Scand J Rehabil Med 22:1, 1990.
188. Ashburn, A, et al: Physiotherapy in the rehabilitation of stroke: A review. Clin Rehabil 7:337, 1993.
189. Sunderland, KJ, et al: Enhanced physical therapy improves recovery of arm function after stroke: A randomized controlled trial. J Neurol Neurosurg 55(7):530, 1992.
190. Feys, HM, et al: Effect of a therapeutic intervention for the hemiplegic upper limb in the acute phase after stroke. Stroke 29 (4): 785, 1998.
191. Langhammer, B, and Stanghelle, J: Bobath or motor relearning programme? A comparison of two different approaches of physiotherapy in stroke rehabilitation: A randomized controlled study. Clin Rehabil 14 (4):361, 2000.
192. Mudie, M, et al: Training symmetry of weight distribution after stroke: A randomized controlled pilot study comparing task-related reach, Bobath and feedback training approaches. Clin Rehabil 16(4):361, 2002.
193. Basmajian, J, et al: Stroke treatment: Comparison of integrated behavioral physical therapy vs traditional physical therapy programs. Arch Phys Med Rehabil 68:267, 1987.
194. Hesse, S, et al: Restoration of gait in nonambulatory hemiparetic patients by treadmill training with partial body-weight support. Arch Phys Med Rehabil 75:1087, 1994.
195. Hesse, S, et al: Treadmill training with partial body weight support compared with physiotherapy in nonambulatory hemiparetic patients. Stroke 26:976, 1995.
196. Malouin, F, et al: Use of an intensive task-oriented gait training program in a series of patients with acute cerebrovascular accidents. Phys Ther 72:781, 1992.
197. Richards, C, et al: Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Arch Phys Med Rehabil 74 (6) 612, 1993.
198. Sullivan, K, Knowlton, B, and Dobkin, B: Step training with body weight support: Effect of treadmill speed and practice paradigms on poststroke locomotor recovery. Arch Phys Med Rehabil 83(5):683, 2002.
199. Smith, G, et al: Task-oriented exercise improves hamstring strength and spastic reflexes in chronic stroke patients. Stroke 30(10):2112, 1999.
200. Nilsson, L, et al: Walking training of patients with hemiparesis at an early stage after stroke: A comparison of walking training on a treadmill with body weight support and walking training on the ground. Clin Rehabil 15(5):515, 2001.
201. Laufer Y, et al: The effect of treadmill training on the ambulation of stroke survivors in the early stages of rehabilitation: A randomized study. J Rehabil Res Dev 38 (1):69, 2001.
202. Taub, E: Somatosensory deafferentation research with monkeys. In Ince, L (ed): Behavioral Psychology in Rehabilitation Medicine: Clinical Applications. Williams & Wilkins, Baltimore, 1980, p 371.
203. Dannenbaum, R, and Dykes, R: Sensory loss in the hand after sensory stroke: Therapeutic rationale. Arch Phys Med Rehabil 69:833, 1988.
204. Weinberg, J, et al: Training sensory awareness and spatial organization in people with right brain damage. Arch Phys Med Rehabil 60:491, 1979.
205. Johnstone, M: Therapy for Stroke. Churchill Livingstone, New York, 1991.
206. Johnstone, M: Restoration of Normal Movement After Stroke. Churchill Livingstone, New York, 1995.
207. Bailey, M, Riddoch, M, and Crome, P: Treatment of visual neglect in elderly patients with stroke: A single-subject series using either a scanning and cueing strategy or a left-limb activation strategy. Phys Ther 82(8):782, 2002.
208. Bailey, M, and Riddoch, M: Hemineglect in stroke patients. Part 2. Rehabilitation techniques and strategies: A summary of recent studies. Phys Ther Rev 4:77, 1999.
209. Wiart, L, et al: Unilateral neglect syndrome rehabilitation by trunk rotation and scanning training. Arch Phys Med Rehabil 78:424, 1997
210. Sharp, SA, and Brouwer, BJ: Isokinetic strength training of the hemiparetic knee: Effects on function and spasticity. Arch Phys Med Rehabil 78:1231, 1997
211. Badics, E, et al: Systematic muscle building exercises in the rehabilitation of stroke patients. Neuro Rehab 17:211, 2002.
212. Kim, CM, et al: Effects of isokinetic strength training on walking in persons with stroke: A double-blind controlled pilot study. J Stroke Cerebrovasc Dis 10:265, 2001.
213. Carr, M, and Jones, J: Physiologic effects of exercise on stroke survivors. Top Stroke Rehabil 9:57, 2003.
214. Bourbonnais, D, et al: Effect of force-feedback treatments in patients with chronic motor deficits after a stroke. Am J Phys Med Rehabil 81:890, 2002.
215. Engardt, M, et al: Dynamic muscle strength training in stroke patients: Effects on knee extension torque, electromyographic activity, and motor function. Arch Phys Med Rehabil 76:419, 1995.
216. Weiss, A, et al: High intensity strength training improves strength and functional performance after stroke. Am J Phys Med Rehabil 79:369, 2000.
217. Butefisch, C, et al: Repetitive training of isolated movements improves the outcome of motor rehabilitation of the centrally paretic hand. J Neurol Sci 103:59, 1995.
218. Moreland, JD, et al: Progressive resistance strengthening exercises after stroke: A single-blind randomized controlled trial. Arch Phys Med Rehabil 84:1433, 2003.
219. Glasser, L: Effects of isokinetic training on the rate of movement during ambulation in hemiparetic patients. Phys Ther 66:672, 1986.
220. Overend, TJ, et al: Cardiovascular stress associated with concentric and eccentric isokinetic exercise in young and older adults. J Gerontol A Biol Sci Med Sci 55:177, 2000.
221. McCully, KK: Exercise-induced injury to skeletal muscle. Fed Proc 45:2933, 1986.
222. Howle, J: Neuro-Developmental Treatment Approach—Theoretical Foundations and Principles of Clinical Practice. NDTA Assoc, Laguna Beach, CA, 2002.
223. Davies, P: Right in the Middle—Selective Trunk Activity in the Treatment of Adult Hemiplegia. Springer-Verlag, New York, 1990.

224. Carr, J, and Shepherd, R: A Motor Relearning Programme for Stroke, ed 2. Aspen, Gaithersville, MD, 1987.
225. O'Sullivan, S, and Schmitz, T: Physical Rehabilitation Laboratory Manual: Focus on Functional Training. FA Davis, Philadelphia, 1999.
226. Sunderland, A et al: Enhanced physical therapy improves recovery of arm function after stroke: A randomized controlled trial. J Neurol Neurosurg Psychiatry 55:530, 1992.
227. Woldag, H, and Hummelsheim, H: Evidence-based physiotherapeutic concepts for improving arm and hand function in stroke patients: A review. J Neurol 249(5):518, 2003.
228. Gillen, G: Upper extremity function and management. In Gillen, G, and Burkhardt, A (eds): Stroke Rehabilitation: A Function-Based Approach, ed 2. CV Mosby, St. Louis, 2004, p 172.
229. Carr, J, and Shepherd, R: Stroke Rehabilitation—Guidelines for Exercise and Training to Optimize Motor Skill. Butterworth Heinemann, Elsevier, Philadelphia, 2003.
230. Schaechter, JD, et al: Motor recovery and cortical reorganization after constraint-induced movement therapy in stroke patients: A preliminary study. Neurorehabil Neural Repair 16(4):326, 2002.
231. Whitall, J, et al: Repetitive bilateral arm training with rhythmic auditory cueing improves motor function in chronic hemiparetic stroke. Stroke 31:2390, 2000.
232. Wolf, S, and Binder-Macleod, S: Electromyographic biofeedback applications to the hemiplegic patient—changes in lower extremity neuromuscular and functional status. Phys Ther 63:1404, 1983.
233. Prevo, A, et al: Effect of EMG feedback on paretic muscles and abnormal co-contraction in the hemiplegic arm, compared with conventional physical therapy. Scand J Rehabil Med 14:121, 1982.
234. Tries, J: EMG feedback for the treatment of upper extremity dysfunction: Can it be effective? Biofeedback Self Regul 14(1):21, 1989.
235. Schleenbaker, RE, and Mainous, AG: Electromyographic biofeedback for neuromuscular education in the hemiplegic stroke patient: A meta-analysis. Arch Phys Med Rehabil 74(12):1301,1993.
236. Glantz, M, et al: Biofeedback therapy in post-stroke rehabilitation: A meta-analysis of the randomized controlled trials. Arch Phys Med Rehabil 76:508, 1995.
237. Smith, L: Restoration of volitional limb movement in hemiplegia following patterned functional electrical stimulation. Percept Mot Skills 71:851, 1990.
238. Trimble, M, and Enoka, R: Mechanisms underlying the training effects associated with neuromuscular electrical stimulation. Phys Ther 71:273, 1991.
239. Bowman, B, et al: Positional feedback and electrical stimulation: An automated treatment for the hemiplegic wrist. Arch Phys Med Rehabil 60:497, 1979.
240. Baker, L, and Parker, K: Neuromuscular electrical stimulation of the muscles surrounding the shoulder. Phys Ther 66:1930, 1986.
241. Price, C, and Pandyan, A: Electrical stimulation for preventing and treating post-stroke shoulder pain (Cochrane review). The Cochrane Library, Issue 3. John Wiley & Sons, Chichester, UK, retrieved July 20, 2005 from: http://www.cochrane.org/reviews/en/ab000197.html.
242. Roy, C: Shoulder pains in hemiplegia: A literature review. Clin Rehabil 2:35, 1988.
243. Jespersen, HF, et al: Shoulder pain after a stroke. Int J Rehabil Res 18A:273, 1995.
244. Turner-Stokes, L, and Jackson, D: Shoulder pain after stroke: A review of the evidence base to inform the development of an integrated care pathway. Clin Rehabil 16:276, 2002.
245. Snels, I, et al: Treating patients with hemiplegic shoulder pain. Am J Phys Med Rehabil 81(2):150, 2002.
246. Tepperman, P, et al: Reflex sympathetic dystrophy in hemiplegia. Arch Phys Med Rehabil 65:442, 1984.
247. Zorowitz, R, et al: Shoulder subluxation after stroke: A comparison of four supports. Arch Phys Med Rehabil 76:763, 1995.
248. Brooke, M, et al: Shoulder subluxation in hemiplegia: Effects of three different supports. Arch Phys Med Rehabil 72:582, 1991.
249. Bernath, V: Shoulder supports in patients with hypotonicity following stroke. Centre for Clinical Effectiveness, Clayton Australia, Jan 2001. Retrieved August 20, 2005 from http://www.med.monash.edu.au/healthservices/cce/evidence/pdf/c/470.pdf.
250. Rose, D: Fall Proof: A Comprehensive Balance and Mobility Training Program. Human Kinetics, Champaign, IL, 2003.
251. Winstein, C, et al: Standing balance training: Effect on balance and locomotion in hemiparetic adults. Arch Phys Med Rehabil 70:755, 1989.
252. Sackley, C, and Lincoln, N: Single blind randomized controlled trial of visual feedback after stroke: Effects on stance symmetry and function. Disabil Rehabil 19:536, 1997.
253. Hamman, R, et al: Training effects during repeated therapy sessions of balance training using visual feedback. Arch Phys Med Rehabil 73:738, 1992.
254. McRae, J, et al: Rehabilitation of hemiplegia: Functional outcomes and treatment of postural control. Phys Ther 74(Suppl):S119, 1994.
255. Nichols, D: Balance retraining after stroke using force platform biofeedback. Phys Ther 77:553, 1997.
256. Fishman, M, et al: Comparison of functional upper extremity tasks and dynamic standing. Phys Ther 76(Suppl):79, 1996.
257. Walker, C, Brouwer, B, and Culham, E: Use of visual feedback in retraining balance following acute stroke. Phys Ther 80:886, 2000.
258. Geiger, R, et al: Balance and mobility following stroke: Effects of physical therapy interventions with and without biofeedback/force-plate training. Phys Ther 81:995, 2001.
259. Shepherd, R, and Carr, J: Treadmill walking in neurorehabilitation. Neurorehabil Neural Repair 13:171, 1999.
260. Macko, RF, et al: Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients; A preliminary report. Stroke 28(2):326, 1997.
261. Binder, S, et al: Evaluation of electromyographic biofeedback as an adjunct to therapeutic exercise in treating the lower extremities of hemiplegic patients. Phys Ther 61:886, 1981.
262. Cranstam, B, et al: Improvement of gait following functional electrical stimulation. Scand J Rehabil Med 9:7, 1977.
263. Merlitte, R, et al: Clinical experience of electronic peroneal stimulators in 50 hemiparetic patients. Scand J Rehabil Med 11:111, 1979.
264. Cozean, C, et al: Biofeedback and functional electric stimulation in stroke rehabilitation. Arch Phys Med Rehabil 69:401, 1988.
265. Bogataj, U, et al: The rehabilitation of gait in patients with hemiplegia: A comparison between conventional therapy and multichannel functional electrical stimulation therapy. Phys Ther 75:490, 1995.
266. Jacobs-Daly, J, et al: Electrically induced gait changes post stroke, using an FNS system with intramuscular electrodes and multiple channels. J Neurol Rehab 7:17, 1993.
267. Bogataj, U, et al: Restoration of gait during two to three weeks of therapy with multichannel electrical stimulation. Phys Ther 69:319, 1989.
268. Stanic, U, et al: Multichannel electrical stimulation for the correction of hemiplegic gait. Scan J Rehabil Med 10:75, 1978.
269. Gok, H, et al: Effects of ankle-foot orthoses on hemiparetic gait. Clin Rehabil 17:137, 2003.
270. Burdett, R, et al: Gait comparison of subjects with hemiplegia walking unbraced, with ankle-foot orthosis, and with air-stirrup brace. Phys Ther 68:1197, 1988.
271. Lehmann, J, et al: Knee movements: Origin in normal ambulation and their modification by double-stopped ankle-foot orthoses. Arch Phys Med Rehabil 63:345, 1982.
272. Corcoran, P, et al: Effects of plastic and metal braces on speed and energy cost of hemiparetic ambulation. Arch Phys Med Rehabil 5:69, 1970.
273. Hirschberg, G, and Ralston, H: Energy cost of stairclimbing in normal and hemiplegic subjects. Am J Phys Med 44:165, 1965.
274. Fisher, S: Energy cost of ambulation in health and disability: A literature review. Arch Phys Med Rehabil 59:124, 1978.
275. Roth, E, et al: Cardiovascular response to physical therapy in stroke rehabilitation. NeuroRehab 2:7, 1992.
276. Buck, L: The coincidence of heart disease and stroke: Pathogenesis and considerations for therapy. Neurology Report (now JNPT) 18:29, 1994.
277. Monga, T, et al: Cardiovascular response to acute exercise in patients with cerebrovascular accidents. Arch Phys Med Rehabil 69:937, 1988.

278. Macko, RF, et al: Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients. Stroke 28(2):326, 1997.
279. Potempa, K, et al: Physiological outcomes of aerobic exercise training in hemiparetic stroke patients. Stroke 26:101, 1995.
280. Brinkman, J, and Hoskins, T: Physical conditioning and altered self-concept in rehabilitated hemiplegic patients. Phys Ther 59:859, 1979.
281. Carr, E: Assessment and treatment of feeding difficulties after stroke. Top Geriatr Rehabil 7:35, 1991.
282. Jorgensen, H, et al: Outcome and time course of recovery in stroke. Part I: Outcome. The Copenhagen Stroke Study. Arch Phys Med Rehabil 76:406, 1995.
283. Meijer, R, et al: Prognostic factors for ambulation and activities of daily living in the subacute phase after stroke. A systematic review of the literature. Clin Rehabil 17(2): 119, 2003.
284. Galski T, et al: Predicting length of stay, functional outcome, and aftercare in the rehabilitation of stroke patients. Stroke 24:1794, 1993.
285. Granger, C, and Hamilton, B: Measurement of stroke rehabilitation outcome in the 1980s. Stroke 21(Suppl II):1146, 1990.

Supplemental Readings

Carr, J, and Shepherd, R: Stroke Rehabilitation. Butterworth Heinemann, New York, 2003.
Davies, P: Steps to Follow: The Comprehensive Treatment of Patients with Hemiplegia, ed 2. Springer-Verlag, New York, 2000.
Davies, P: Right in the Middle—Selective Trunk Activity in the Treatment of Adult Hemiplegia. Springer-Verlag, New York, 1990.
Gillen, G, and Burkhardt, A (eds): Stroke Rehabilitation: A Function-Based Approach, ed 2. CV Mosby, St. Louis, 2004.
Johnston, M: Restoration of Normal Movement After Stroke. Churchill Livingstone, New York, 1995.
Reyerson, S, and Levit, K: Functional Movement Reeducation. Churchill Livingstone, New York, 1997.
Refshauge, K, Ada, L, and Ellis, E (eds): Science-Based Rehabilitation—Theories into Practice. Butterworth-Heinemann, New York, 2005.

Appendix A: Fugl-Meyer Assessment of Physical Performance

SUMMARY OF SCORES

MOTOR

Upper arm		Maximum Score	36
Wrist & hand		Maximum Score	30
TOTAL UPPER EXTREMITY SCORE		MAXIMUM SCORE	66
TOTAL LOWER EXTREMITY SCORE		MAXIMUM SCORE	34

TOTAL MOTOR SCORE		TOTAL MAXIMUM SCORE	100	PERCENTAGE OF RECOVERY
BALANCE				
TOTAL SCORE		MAXIMUM SCORE	14	
SENSATION				
TOTAL SCORE		MAXIMUM SCORE	24	
JOINT RANGE OF MOTION				
TOTAL SCORE		MAXIMUM SCORE	44	
PAIN				
TOTAL SCORE		MAXIMUM SCORE	44	

TOTAL FUGL-MEYER SCORE		TOTAL MAXIMUM SCORE	226	PERCENTAGE OF RECOVERY

Appendix A: Fugl-Meyer Assessment of Physical Performance (continued)

Area	Test	Scoring Criteria	Maximum Possible Score	Attained Score
	Motor			
UPPER EXTREMITY (sitting)	I. Reflexes a. biceps _____ b. triceps _____	0—No reflex activity can be elicited. 2—Reflex activity can be elicited.	4	
	II. Flexor Synergy elevation _____ shoulder retraction _____ abduction (at least 90°) _____ external rotation _____ elbow flexion _____ forearm supination _____	0—Cannot be performed at all. 1—Performed partly. 2—Performed faultlessly.	12	
	III. Extensor Synergy shoulder adduction/internal rotation _____ elbow extension _____ forearm pronation _____	0—Cannot be performed at all. 1—Performed partly. 2—Performed faultlessly.	6	
	IV. Movement Combining Synergies a. Hand to lumbar spine _____ b. Shoulder flexion to 90° elbow at 0° _____ c. Pronation/supination of forearm with elbow at 90° and shoulder at 0° _____	a. 0—No specific action performed. 1—Hand must pass anterior superior iliac spine. 2—Action is performed faultlessly. b. 0—Arm is immediately abducted or elbow flexes at start of motion. 1—Abduction or elbow flexion occurs in later phase of motion. 2—Faultless motion. c. 0—Correct position of shoulder and elbow cannot be attained, and/or pronation or supination cannot be performed at all. 1—Active pronation or supination can be performed even within a limited range of motion, and at the same time the shoulder and elbow are correctly positioned. 2—Complete pronation and supination with correct positions at elbow and shoulder.	6	
	V. Movement Out of Synergy a. Shoulder abduction to 90° elbow at 0° and forearm pronated _____ b. Shoulder flexion, 90–180° elbow at 0° and forearm in mid position _____ c. Pronation/supination of forearm elbow at 0° and shoulder between 30–90° of flexion _____	a. 0—*Initial* elbow flexion occurs or any deviation from pronated forearm occurs. 1—Motion can be performed partly, or if during motion, elbow is flexed or forearm cannot be kept in pronation. 2—Faultless motion. b. 0—Initial flexion of elbow or shoulder abduction occurs. 1—Elbow flexion or shoulder abduction, occurs during shoulder flexion. 2—Faultless motion. c. 0—Supination and pronation cannot be performed at all or elbow and shoulder position cannot be attained. 1—Elbow and shoulder properly positioned and pronation and supination performed in a limited range. 2—Faultless motion.	6	

(*continued*)

Appendix A: Fugl-Meyer Assessment of Physical Performance (continued)

Area	Test	Scoring Criteria	Maximum Possible Score	Attained Score
	Motor			
UPPER EXTREMITY	VI. Normal Reflex Activity biceps and/or finger flexors and triceps _____	(This stage, which can render the score of two, is included only if the patient has a score of 6 in stage V.) 0—At least 2 of the 3 phasic reflexes are markedly hyperactive. 1—One reflex markedly hyperactive or at least 2 reflexes are lively. 2—No more than one reflex is lively and none are hyperactive.	2	
WRIST	VII. a. Stability, elbow at 90°, shoulder at 0° b. Flexion/extension, elbow at 90°, shoulder at 0° _____ c. Stability, elbow at 0°, shoulder at 30° _____ d. Flexion/extension, elbow at 0°, shoulder at 30° _____ e. Circumduction _____	a. 0—Patient cannot dorsiflex wrist to required 15°. 1—Dorsiflexion is accomplished, but no resistance is taken. 2—Position can be maintained with some (slight) resistance. b. 0—Volitional movement does not occur. 1—Patient cannot actively move the wrist joint throughout the total ROM. 2—Faultless, smooth movement. c. Scoring is the same as for item a. d. Scoring is the same as for item b. e. 0—Cannot be performed. 1—Jerky motion or incomplete circumduction. 2—Complete motion with smoothness.	10	
HAND	VIII. a. Finger Mass Flexion _____ b. Finger Mass Extension _____ c. Grasp #1—MP joints extended and PIPS & DIPS are flexed. Grasp is tested against resistance. d. Grasp #2—Patient is instructed to adduct thumb, 1st carpometacarpophalangeal and interphalangeal joint at 0° e. Grasp #3—Patient opposes the thumb pad against the pad of index finger. A pencil is interposed _____ f. Grasp #4—The patient should grasp a cylinder shaped object (small can), the volar surface of the 1st and 2nd finger against each other _____ g. Grasp #5—A spherical grasp.	a. 0—No flexion occurs. 1—Some flexion, but not full motion. 2—Complete active flexion (compared with unaffected hand). b. 0—No extension occurs. 1—Patient can release an active mass flexion grasp. 2—Full active extension. c. 0—Required position cannot be acquired. 1—Grasp is weak. 2—Grasp can be maintained against relatively great resistance. d. 0—Function cannot be performed. 1—Scrap of paper interposed between the thumb and index finger can be kept in place, but not against a slight tug. 2—Paper is held firmly against a tug. e. Scoring procedures are the same as for Grasp #2. f. Scoring procedures are the same as for Grasp #2 and #3. g. Scoring procedures are the same as for Grasp #2, 3, and 4.	14	
HAND	IX. Coordination/Speed—Finger-to-nose (five repetitions in rapid succession). a. Tremor _____ b. Dysmetria _____ c. Speed _____	a. 0—Marked tremor. 1—Slight tremor. 2—No tremor. b. 0—Pronounced or unsystematic dysmetria. 1—Slight or systematic dysmetria. 2—No dysmetria. c. 0—Activity is more than 6 seconds longer than unaffected hand. 1—2 to 5 seconds longer than unaffected hand. 2—Less than 2 seconds difference.	6	
		TOTAL MAXIMUM UPPER EXTREMITY SCORE	66	

LOWER EXTREMITY (supine)	I. Reflex activity—tested in suprine position. Achilles _____ Patellar _____	0—No reflex activity 2—Reflex activity	4	
Supine	II. a. Flexor Synergy Hip flexion _____ Knee flexion _____ Ankle dorsiflexion _____ b. Extensor synergy—(motion is resisted) Hip extension _____ Adduction _____ Knee extension _____ Ankle plantarflexion _____	a. 0—Cannot be performed 1—Partial motion 2—Full motion b. 0—No motion 1—Weak motion 2—Almost full strength compared to normal	6 8	
SITTING (knees free of chair)	III. Movement Combining Synergies a. Knee flexion beyond 90° _____ b. Ankle dorsiflexion _____	a. 0—No active motion 1—From slightly extended position knee can be flexed but not beyond 90° b. 0—No active flexion 1—Incomplete active flexion 2—Normal dorsiflexion	4	
STANDING	IV. Movement Out of Synergy Hip at 0° a. Knee flexion _____ b. Ankle dorsiflexion _____	a. 0—Knee cannot flex without hip flexion 1—Knee begins flexion without hip flexion, but doesn't get to 90°, or hip flexes during motion 2—Full motion as described b. 0—No active motion 1—Partial motion 2—Full motion	4	
SITTING	V. Normal Reflexes Knee flexors _____ Patellar _____ Achilles _____	0—2 of the 3 are markedly hyperactive 1—One reflex is hyperactive or 2 reflexes are lively 2—No more than 1 reflex lively	2	
(SUPINE)	VI. Coordination/Speed Heel to opposite knee (5 repetitions in rapid succession) a. Tremor _____ b. Dysmetria _____ c. Speed _____	a. 0—Marked tremor 1—Slight tremor 2—No tremor b. 0—Pronounced or unsystematic 1—Slight or systematic 2—No dysmetria c. 0—Six seconds slower than unaffected side 1—Two to 5 seconds slower 2—Less than 2 seconds difference	6	
		TOTAL MAXIMUM LOWER EXTREMITY SCORE	34	

(*continued*)

Appendix A: Fugl-Meyer Assessment of Physical Performance (continued)

Area	Test	Scoring Criteria	Maximum Possible Score	Attained Score
BALANCE	a. Sit without support _____ b. Parachute reaction, non-affected side _____ c. Parachute reaction, affected side _____ d. Supported standing e. Stand without support f. Stand on unaffected side _____ g. Stand on affected side _____	a. 0—Cannot maintain sitting without support 1—Can sit unsupported less than 5 minutes 2—Can sit longer than 5 minutes b. 0—Does not abduct shoulder or extend elbow 1—Impaired reaction 2—Normal reaction c. Scoring is the same as #2 d. 0—Cannot stand 1—Stands with maximum support of others 2—Stands with minimum support of one for 1 minute e. 0—Cannot stand 1—Stands less than 1 minute or sways 2—Stands with good balance more than 1 min. f. 0—Cannot be maintained longer than 1–2 sec. 1—Stands balanced 4–9 seconds 2—Stands balanced more than 10 sec. g. 0—Scoring is the same as #6		
		MAXIMUM BALANCE SCORE	14	
UPPER AND LOWER EXTREMITIES	*Sensation* I. Light Touch a. Upper arm _____ b. Palm of hand _____ c. Thigh _____ d. Sole of foot _____ II. Proprioception a. Shoulder _____ b. Elbow _____ c. Wrist _____ d. Thumb _____ e. Hip _____ f. Knee _____ g. Ankle _____ h. Toe _____	Light Touch Scoring 0—Anesthesia 1—Hyperaesthesia/dyesthesia 2—Normal Proprioception Scoring 0—No sensation 1—Three quarter of answers are correct, but considerable difference in sensation compared with unaffected side. 2—All answers are correct, little or no difference	8 16	

		Motion/Pain				
		Motion	Pain			
SHOULDER	Flexion	_____	_____	Motion Scoring	Motion	
	Abduction to 90°	_____	_____	0—Only a few degrees of motion	44	
	External rotation	_____	_____	1—Decreased passive range of motion		
	Internal rotation	_____	_____	2—Normal passive range of motion		
ELBOW	Flexion	_____	_____			
	Extension	_____	_____	Pain Scoring		
WRIST	Flexion	_____	_____	0—Marked pain at end of range or pain through range	44	
	Extension	_____	_____	1—Some pain		
FINGERS	Flexion	_____	_____	2—No pain		
	Extension	_____	_____			
FOREARM	Pronation	_____	_____			
	Supination	_____	_____			
HIP	Flexion	_____	_____			
	Abduction	_____	_____			
	External rotation	_____	_____			
	Internal rotation	_____	_____			
KNEE	Flexion	_____	_____			
	Extension	_____	_____			
ANKLE	Dorsiflexion	_____	_____			
	Plantarflexion	_____	_____			
FOOT	Pronation	_____	_____			
	Supination	_____	_____			

Appendix B: Web-Based Resources for Clinicians, Families, and Patients with Stroke

American Heart Association	http://www.americanheart.org/
American Stroke Association-a division of the American Heart Association	http://www.strokeassociation.org/
National Stroke Association	http://www.stroke.org/
American Stroke Foundation	http://www.americanstroke.org/
International Stroke Society	http://www.internationalstroke.org
Stroke Association—UK	http://www.stroke.org.uk/
Heart and Stroke Foundation of Canada	http://www.heartandstroke.ca/
Veterans Affairs—stroke	http://www.va.gov/
Americans with Disabilities Act: ADA home page	http://www.usdoj.gov/crt/ada
Medicare information	http://www.cms.hhs.gov
Social Security Online	http://www.ssa.gov
National Institute of Neurological Disorders and Stroke	http://www.ninds.nih.gov
National Library of Medicine	http://www.nlm.nih.gov
American Association of Physical Medicine and Rehabilitation	http://www.aapmr.org/condtreat/rehab/stroke.htm
American Academy of Neurology (ANA)	http://www.aan.com/professionals http://www.aan.com/public (public education) http://www.neurology.org (Journal of Neurology)
National Rehabilitation Information Center (NARIC)	http://www.naric.com
Stroke rehab forum at Med Help	http://www.medhelp.org/forums/stroke Rehab/
Rehabilitation Research & Training Center on Stroke Rehabilitation	http://www.rrtc-stroke.org
Internet Handbook of Neurology	http://www.neuropat.dote.hu/stroke1.htm
Stroke and depression	http://www.nimh.nih.gov/publicat/depstroke.cfm
National Aphasia Association	http://www.aphasia.org
National Easter Seal Society	http://www.easter-seals.org
Disease prevention	http://www.everydaychoices.org
The Neurology Channel—stroke	http://www.neurologychannel.com/stroke/
Agency for Healthcare Research & Quality	http://www.ahrq.gov/consumer/strokecon.htm
Brain attack- stroke prevention & treatment—USFDA	http://www.fda.gov/fdac/features/2005/205_stroke.html
Stroke Information Directory	http://www.stroke-info.com
Clinical trials—National Institutes of Health (NIH)—stroke	http://www.clinicaltrials.gov/search/term=stroke
Stroke survivors	http://www.stroke-survivors.com
Resource center for clinicians and families	http://www.strokehelp.com/
The Stroke Network, Inc	http://www.strokenetwork.org
National Family Caregivers Association (NFCA)	http://www.nfcacares.org
Well Spouse Foundation	http://www.wellspouse.org
Ability Hub—assistive technology solutions	http://www.abilityhub.com
ABLEDATA—assistive technology information	http://www.abledata.com
Disabled Online	http://www.disabledonline.com