



Complementary
Medicine

The Bobath Concept in Adult Neurology

Bente E. Bassøe Gjelsvik



 Thieme



The Bobath Concept in Adult Neurology

Bente E. Bassøe Gjelsvik

Physiotherapist
Advanced Bobath Instructor IBITA
Bergen, Norway

241 Illustrations

Thieme
Stuttgart · New York

Library of Congress Cataloging-in-Publication Data
Gjelsvik, Bente E. Bassøe.

The Bobath concept in adult neurology /
Bente E. Bassøe Gjelsvik.

p. ; cm.

Includes bibliographical references.

ISBN 978-3-13-145451-5

(TPS, rest of world : alk. paper) -

ISBN 978-1-58890-621-2

(TPN, the Americas : alk. paper)

1. Central nervous system—Diseases—Physical therapy.
2. Central nervous system—Diseases—Patients—Rehabilitation.
3. Physical therapy. I. Title.

[DNLM: 1. Central Nervous System Diseases—physiopathology. 2. Central Nervous System Diseases—rehabilitation. 3. Neuronal Plasticity. 4. Neurophysiology—methods. 5. Physical Therapy Modalities.]

WL 300 G5395b 2008]

RC350.P48G54 2008

616.8-dc22

2007030566

Illustrators: Guenter Bosch,
Münsingen-Dottingen, Germany;
Adrian Cornford, Reinheim-Zeilhard, Germany.

Important note: Medicine is an ever-changing science undergoing continual development. Research and clinical experience are continually expanding our knowledge, in particular our knowledge of proper treatment and drug therapy. Insofar as this book mentions any dosage or application, readers may rest assured that the authors, editors, and publishers have made every effort to ensure that such references are in accordance with the **state of knowledge at the time of production of the book**.

Nevertheless, this does not involve, imply, or express any guarantee or responsibility on the part of the publishers in respect to any dosage instructions and forms of applications stated in the book. **Every user is requested to examine carefully** the manufacturers' leaflets accompanying each drug and to check, if necessary in consultation with a physician or specialist, whether the dosage schedules mentioned therein or the contraindications stated by the manufacturers differ from the statements made in the present book. Such examination is particularly important with drugs that are either rarely used or have been newly released on the market. Every dosage schedule or every form of application used is entirely at the user's own risk and responsibility. The authors and publishers request every user to report to the publishers any discrepancies or inaccuracies noticed. If errors in this work are found after publication, errata will be posted at www.thieme.com on the product description page.

© 2008 Georg Thieme Verlag,
Rüdigerstrasse 14, 70469 Stuttgart, Germany
<http://www.thieme.de>
Thieme New York, 333 Seventh Avenue,
New York, NY 10001, USA
<http://www.thieme.com>

Typesetting by Hagedorn Kommunikation, Viernheim,
Germany

Printed in Germany by GCC—Grafisches Centrum
Cuno, Calbe

ISBN 978-3-13-145451-5 (TPS, Rest of World)
ISBN 978-1-58890-621-2 (TPN, The Americas)

Some of the product names, patents, and registered designs referred to in this book are in fact registered trademarks or proprietary names even though specific reference to this fact is not always made in the text. Therefore, the appearance of a name without designation as proprietary is not to be construed as a representation by the publisher that it is in the public domain.

This book, including all parts thereof, is legally protected by copyright. Any use, exploitation, or commercialization outside the narrow limits set by copyright legislation, without the publisher's consent, is illegal and liable to prosecution. This applies in particular to photostat reproduction, copying, mimeographing, preparation of microfilms, and electronic data processing and storage.

Preface

About the Author

I trained as a physiotherapist at the School of Physiotherapy, Royal Victoria Infirmary, Newcastle-upon-Tyne, England, in 1978. For a short time I worked in a newly opened hospital in Newcastle, before I moved back to Norway with my husband. Since July 1978 I have worked in Haukeland University Hospital in Bergen; for the first 7 years in medical wards, from 1985 to 1996 in the Neurology Department, and since then in the Department of Physical Medicine and Rehabilitation. I have been involved in different projects, one of which was the development of a rehabilitation network in Norway. This led to the opening of what is now the Department of Physical Medicine and Rehabilitation in 1996.

I have taken part in many research projects and I have co-authored several articles.

I trained as a Bobath Instructor with the British Bobath Tutor Association (BBTA). I was approved by the International Bobath Instructors Training Association (IBITA) as a Bobath Instructor in 1991, and as an Advanced Bobath Instructor in 2004. I am a specialist in neurorehabilitation and have been a member of the Norwegian Physiotherapist Association since 1995. I have extensive teaching experience and have taught courses in many countries in Europe. Currently, I am the Chairperson of IBITA.

Aims of This Book

To improve the therapist's competency in the treatment of individuals with neurologic conditions by:

- building bridges between:
 - the structure and function of the central nervous system (CNS), the neuromusculoskeletal systems, and the ability for change (plasticity);
 - postural control and movement; and the
 - treatment of neurologic conditions.

- enabling the reader to form hypotheses through clinical reasoning in treatment situations based on a conceptual understanding of the interaction between humans and the environment, and between the CNS, the musculoskeletal systems, movement, and function. Clinical reasoning cannot be learned through reading a book: it is developed through continuous critical evaluation of one's own practice, by pursuing answers through experimenting and by improving one's evidence-based knowledge. I hope that this book may help the reader in this process.

The book is written for physiotherapists and occupational therapists, students, and qualified professionals. It is mainly aimed at the clinician working with neurologically impaired individuals.

Structure of the Book

The book is meant to be read in the way it is structured. The chapters build on each other, and the reader may miss important information and discussions if it is primarily used as a reference book. I do hope, however, that it will be useful as a reference once read through.

Chapter 1, Applied Neurophysiology, is divided into three parts:

- 1.1 Systems Control: This deals with some systems and structures concerned with movement and sensorimotor integration. This part takes a brief look at the structure and function of parts of the CNS, and describes the interaction between CNS function, muscle function, function, and movement. Consequences of CNS lesions and clinical reflections are discussed throughout.
- 1.2 Plasticity: This part outlines changes in the CNS as a result of nature and nurture and as a consequence of CNS lesions. These changes form the basis for learning and are therefore important to understand. Implications for therapy are discussed.

- 1.3 Consequences of and Reorganization after CNS Lesions: This part attempts to put the consequences of CNS lesions and plasticity into a clinical context. This part discusses and formulates hypotheses about the cause and effect of lesions and the sensorimotor problems that patients experience. Clinical symptoms such as spasticity and associated reactions are discussed.

Chapter 2, Physiotherapy, discusses normal balance and movement, and deviations from normal movement as well as choices therapists may have for interventions.

Chapter 3, Assessment, looks at the International Classification of Functioning, Disability, and Health as a basis for assessment. Some outcome measures are also briefly described.

Chapter 4, Case Histories, presents the case studies of two individuals, Sissel and Lisa.

I have used the pronoun "he" for the patient and "she" for the therapist except where photos show differently, although in real life the situation is often reversed.

Foreword

At this time we know more about the central nervous system than ever before, but to bring about the reality of a functional recovery after a lesion is still a very serious clinical challenge to both patients and therapists. In this book Bente Gjelsvik, an acknowledged Bobath Instructor and Clinical Specialist in Neurology, brings all her skills to explain a concept which has evolved over decades to address the complexities of neuro-disability. She adopts a problem-solving approach consistent with the definition of the

Bobath Concept and its current interpretation of movement control. The basis of the text is to understand the structure and function of the organism, which is expressed through an understanding of posture and movement control. It is a clinically oriented text concluding with two detailed case histories that will be of significant interest to all professions involved in the management of neurologic disability.

Mary Lynch-Ellerington

Acknowledgments

The first book that I published with Thieme was in German: *Form und Funktion*. It is thanks to my German editor, Eva Maria Grünwald, that her colleague Angelika-M. Findgott at Thieme Publishers, the English branch of Thieme Verlag, became interested in publishing an English edition—the book you are holding in your hands now. My thanks go to both my editors.

This English edition is based on the German one, but it has been revised and updated. A second German version with the same updates and revisions has already been published. I have done the revision and translation (from Norwegian) into English myself, with great help from my English colleagues at the British Bobath Tutors Association (BBTA): Lynne Fletcher, Janice Champion, and Linzi Smith.

My husband Olav Gjelsvik is a close and critical colleague. He is a physiotherapist and Bobath Instructor and has given me valuable support, encouragement, and input throughout this process.

My great friend and mentor Mary Lynch-Ellington is a Senior Bobath Instructor and the professional leader of BBTA. She has, over many years, given me the basis for my conceptual understanding of the Bobath Concept. She is an extremely generous person, sharing her insight and knowledge with colleagues and course participants all over the world.

Last but not least, a special thank you to the patients and colleagues who were willing to be included in this book.

Bente E Bassøe Gjelsvik

Contents

Introduction	1	Active Movement; Learned Nonuse; Neglect; Passive Movement	125
The Bobaths: A Historical Overview ..	1	Control of Associated Reactions	127
The International Bobath Instructors Training Association—IBITA	1	Feedback	128
IBITA's Theoretical Assumptions and Clinical Practice	3	Carry-over	130
1 Applied Neurophysiology	5	2.3 Other Interventions: Some Key Points	132
1.1 Systems Control: Systems and Structures Concerned with Movement and Sensorimotor Integration	6	Strength Training	132
The Neuromuscular System	6	Treadmill Training	133
The Somatosensory System, Vision, and Balance	14	Multidisciplinary Teamwork	135
The Brain and Spinal Cord	23	Assistive Devices	135
1.2 Plasticity	47	Medical Intervention for the Treatment of Spasticity	141
Neuroplasticity	49	3 Assessment	145
1.3 Consequences of and Reorganization after CNS Lesions	56	3.1 The International Classification of Functioning, Disability, and Health ..	145
Upper Motor Neuron Lesions	58	3.2 Physiotherapy Assessment	147
The Complex Problem of the Upper Motoneuron Syndrome	60	History	148
2 Physiotherapy	67	Functional Activity	149
2.1 Balance and Movement	68	Body Functions and Structures	151
Human Movement Control	68	Sensation, Perception, and Learned Nonuse	154
Balance	69	Pain	156
Deviations from Normal Movement and Balance Control	81	Clinical Reasoning	157
2.2 Intervention—Considerations and Choices	90	The Aim of Assessment	158
Postural Sets	90	3.3 Outcome Measures	163
Analysis of Basic Postures and Postural Sets	91	Body Structure and Function Measures ..	163
Key Areas	108	Activity Measures	164
Selective Movement and Functional Activity	110	Self-Report Measures	165
The Relationship between Automatic and Voluntary Movement	112	Objective Goal Setting	165
Handling	116	Assessment Diagram	165
		Evaluation and Documentation	166
		Conclusion	166
		4 Case Histories	169
		4.1 Case History: Sissel	169
		Past Medical History, Social History, Activities, and Participation	169
		History of Present Illness	169
		Assessment	170
		Clinical Reasoning and Hypothesis ..	175

Physiotherapy and Clinical Reasoning	176	Lisa's Goals	196
Physiotherapy: Assessment and Treatment as a Continuous Process	177	Assessment	196
Evaluation at Discharge	194	Clinical Reasoning and Hypotheses	201
4.2 Case History: Lisa	195	Physiotherapy	204
Social History, Activity, and Participation	195	Evaluation	217
Medical History	195	Follow-up	217
Previous Training Experience and Treatment	195		
Current Problems	196		
		Bibliography	219
		Index	231

Introduction

The Bobaths: A Historical Overview

The following paragraphs are extracts from *The Bobaths. A Biography of Berta and Karel Bobath* by Jay Schleichkorn, PhD, PT (1992).

Karel Bobath and Berta Otilie Busse were born in Berlin, Karel in 1906, Berta in 1907. He studied medicine and qualified as doctor in 1932. She graduated as gymnastic teacher from the Anna Herrmann school where she learned about normal movements and different relaxation methods. Berta and Karel left for London before the 2nd World War.

The development of the Bobath Concept for adults started 1943 when Berta was asked to treat Simon Elwes, a 43-year-old portrait painter who had suffered a stroke. "When I arrived I found him in bed, his arm and hand extremely stiff in flexion, his hand swollen, a bad shoulder-hand syndrome, and his leg covered up ..." [p. 20] "Instead of doing what I had been taught—exercises, I observed the patient. Slowly, by trial and error, by observation and deduction, I began relating things he was doing in response to what I was doing. It worked better than anything before." [s. xi] ... "I realised for the first time that the patient's pulling into flexion produced his spasticity, and that spasticity was not an unalterable state which could only be treated by stretching spastic muscles." [p. 20] Simon Elwes recovered well and started painting again. Berta treated Simon Elwes for 18 months, and discovered that this form of treatment only was a beginning. It took many years to develop the treatment from this simple way of reducing spasticity to the problem of making the patient active and participating without returning to a spastic state.

Berta graduated as a physiotherapist in 1950 from The Chartered Society for Physiotherapists. Karel and Berta's first Center opened in 1951, and in 1957 "The Western Cerebral Palsy Centre" was established. Both children and adults with different neurological disorders were treated there, with the main emphasis on children with cerebral palsy. Berta Bobath educated the parents in handling of their children through daily activities like bathing, dressing, and how they should

carry their children as living human beings and not lifeless dolls. She strongly advocated the importance of a multidisciplinary approach, especially between physiotherapists, occupational therapists, and speech therapists. The physiotherapist Jenny Bryce, later to become the leader of the Centre for a long period, said that "the aspect which most impressed me was Berta's deep understanding of normal movement, and she applied that understanding in the treatment of both children and adults." [p. 35] In 1990 she said that "The lasting fascination of the concept is that it is constantly under discussion and never in danger of standing still..." [p. 36].

Karel sought to explain the neurophysiological background for Berta's observations and treatment. About the Bobath Concept, they both stated in 1990: "It was based purely on empirical lines by Mrs. Bobath's observation of children and adults with neurological lesions and their response to treatment... The concept is hypothetical in nature, although to some extent it has been confirmed and strengthened by recent research which we hope will continue in the future." [p. ix].

From 1958 Berta and Karel Bobath travelled widely over large parts of the USA, Sour-Afrika, Canada, Europe, Asia, Australia, and Latin-America to teach, lecture, and demonstrate treatment. Berta Bobath was given an M.B.E. (Member of the Order of British Empire) in 1978, and received many international honorary awards. Together, they have produced more than 70 publications and many unpublished congress articles from 1948 till 1990.

They both died on the 20th of January 1991.

The International Bobath Instructors Training Association—IBITA

There have been large and significant developments in the Bobath Concept since Berta and Karel's time. Technological developments have revolutionized assessment and examination procedures, although there are still many aspects of

the function, communication, and plasticity of the central nervous system (CNS) that remain unknown. The problems of patients whom professionals meet today are partly different from the problems patients formerly had: many patients survive due to improved acute care, they are treated in specialized units, and they are discharged earlier from hospitals and rehabilitation units. They are exposed to different demands and to many different treatment concepts or regimens. There is a continuous development of theory and clinical practice, and the demand for evidence-based practice is increasing. Theoretical assumptions change as new knowledge becomes available, demonstrating that the profession is in a dynamic state of development. Emerson Pugh stated in 1977 that "If the brain were so simple that we could understand it, we would be so simple that we could not." Medical "truths" have a short life. As clinicians we need to be humble, accept that science changes, and develop our knowledge. At the same time, we have to be careful not to throw away clinical knowledge based on reasoning and experience, even if the interventions have not yet been proved. Many of our interventions are not documented or researched. Changes witnessed by the therapist and experienced by the patient together in clinical practice may not show on the clinical scales that are used today due to the insensitivity of many of the outcome measures that exist. Qualitative methods are still lacking in clinical practice and research (Bhakta et al. 1996, Sampaio et al. 1997, Gelber and Jozefczyk 1999, Mant 1999, Lagalla et al. 2000, Malterud 2001a, b).

The International Bobath Instructors Training Association (IBITA) was founded in 1984 and is a worldwide organization of qualified IBITA instructors. Today there are approximately 250 members of IBITA.

IBITA's roles are:

- To develop and document guidelines and rules and regulations for international courses in the treatment of adults with neurologic conditions
- To develop standards for evaluation of international courses
- To develop competency standards for instructors and course participants
- To develop guidelines for the training and qualification of instructors

- To develop an evidence base for the theoretical assumptions of the Bobath Concept

The following vision and mission statements are from IBITA's bylaws:

Vision

Throughout the world, adults with neurological dysfunction will be assured of the services of an interdisciplinary team trained in neurological rehabilitation, originating in the Bobath concept and developed in accordance with current knowledge.

Mission

1. Members of IBITA plan, organise and run courses worldwide to train physical, occupational and speech therapists, medical doctors and registered nurses in the assessment and treatment of adults with lesions of the central nervous system.
2. Members of IBITA ensure that their teaching and clinical practice is founded upon and reflects current understanding of motor control, neural and muscle plasticity, motor learning and biomechanics, integrated within the Bobath concept.
3. Members of IBITA recognise the importance of evidence-based practice and evaluate the research literature critically in order to implement such practice.
4. Members of IBITA strive constantly to improve their own standards of clinical expertise and to impart their knowledge and skills.
5. Members of IBITA play an active role in the training of new instructors.
6. Members of IBITA are aware of the need for research into the theoretical assumptions and clinical outcomes of treatment, and undertake to publish their findings.
7. Members of IBITA accept their role in education and empowerment of the patient, family and other caregivers.
8. Members of IBITA promote at all times the vision, mission and objectives of IBITA, in their clinical and teaching practice as well as in interaction with other professionals, with national and international organisations and with the public.

IBITA's Theoretical Assumptions and Clinical Practice

IBITA is continuously discussing its theoretical assumptions in the light of new knowledge, and seeks to bridge the gap between theory and clinical practice. Therefore, theoretical assumptions and statements regarding clinical practice are regularly reviewed and revised. The reader is recommended to read the current document on IBITA's website (www.ibita.org).

1 Applied Neurophysiology

1.1 Systems Control: Systems and Structures Concerned with Movement and Sensorimotor Integration	6
1.2 Plasticity	47
1.3 Consequences of and Reorganization after CNS Lesions.....	56

Knowledge of the functions of the central nervous system (CNS) has historically been drawn from experimental studies on animals. In recent years, developments in movement science have led to studies being undertaken mostly on normal, healthy people. More recently, advances in noninvasive neuroimaging techniques have made it possible not only to study local changes in the brain function of people with CNS lesions but also to follow changes in the CNS over time. Techniques such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), transcranial magnetic stimulation (TMS), electroencephalography (EEG), and magnet encephalography (MEG) show changes in the structure of the brain and how these correlate with changes in the patient's physical functioning post-lesion (Academy of Medical Sciences 2004, Ward and Cohen 2004).

Knowledge about neurophysiology, normal movement, and deviations from normal movement forms the basis for clinical reasoning. This chapter therefore aims to discuss movement and the post-lesion alterations in movement and covers:

- **Systems control:** Systems and structures concerned with movement and sensorimotor integration
- **The neuromuscular system:** The muscular system, plasticity, communication with the spinal cord
- **The somatosensory system, vision, and balance:** The connection between somatosensory system, vision, and movement in relation to stereognostic sense and balance

- **Systems within the spinal cord and brain:** Systems important for movement and analysis of movement in a clinical situation
- **Plasticity:** How the brain is structurally and functionally modified by the information it receives
- **Lesions and reorganization:** Consequences of damage to the CNS

The chapter includes a simplified presentation of the aspects of CNS function that are particularly relevant in the production and development of sensorimotor function. The reader is advised to review other relevant publications for revision and more in-depth information (e.g., Brodal's *The Central Nervous System* and Kandel et al.'s *Principles of Neural Science*). The integrated model of human movement, in which all systems—sensory, motor, perceptual, and cognitive—have important roles in making movement efficient and in the context of the individual will be discussed. No system works in isolation; all systems—network with other systems, they receive, integrate, and pass on information, and they affect and are affected by other systems. "Movement is the output of a hybrid functional system interlinked to its environment in which sensory, cognitive and motor processes interact" (Mulder et al. 1996). Motor behavior is the result of integration between the individual, the task, and the environment in which action is being performed. Different systems have different roles in different contexts to make behavior appropriate to the moment.

Localizing CNS function is a complex task and has undergone huge advancements from the

science of phrenology, in which “bumps” on a person’s skull were thought to represent a specially developed brain area, to imaging techniques that permit us to see the human brain in action. Different regions of the brain are specialized for different functions and yet the organization of the CNS is known as *parallel distributed processing*. Many sensory, motor, and cognitive functions are served by more than one pathway. To some degree, this helps regions or pathways to partially compensate for each other if damage occurs (Kandel et al. 2000).

1.1 Systems Control: Systems and Structures Concerned with Movement and Sensorimotor Integration

The Neuromuscular System

The structure and function of the muscular and nervous systems have to be discussed together. CNS and motor activity influence each other. Movement is the end result of the action of skeletal muscles as a result of CNS processing. Processing within the CNS is a result of information sent to the system about the desire or requirement to act, based on a need to interact with the environment. The individual, the functional task, and the environment in which movement is being performed require appropriate processing and function within the CNS and muscular systems. The muscular system and the CNS exchange information and demands continuously. The muscular system is specialized in its structure and function to meet the needs of a variety of movements in different settings to perform a multitude of tasks. The neuromuscular system has an adaptive capacity. Changes in the information sent by the CNS to the muscular system may alter the structure and function of the musculature, and vice versa (altered use of the muscles due to CNS lesions may result in changes in the structure and function of the CNS).

Thus, on the basis of the interdependence between these systems, this chapter is called “The Neuromuscular System.”

Structure and Function of Skeletal Muscle

Skeletal muscle contains contractile and noncontractile elements and specialized sense organs or receptors. The contractile elements are the extrafusal muscle fibers and ends of the muscle spindles. The noncontractile elements are the connective tissue and sense organs (the Golgi tendon organ and muscle spindles). Muscle tone is related to the state of muscle fibers, the activity of the sense organs, viscosity, and the state of the fibrous tissue.

Skeletal Muscle Fibers

These are divided into three main groups and several subgroups. The three main groups are:

- Type 1, also called ST (slow twitch; Brodal 2001) or SO (slow oxidative; Kidd et al. 1992, Rothwell 1994). This fiber type is often described as red due to its high content of myoglobin. Type 1 fibers have a high level of endurance, are precise in their action, and produce a moderate amount of power. The action of these fibers is often referred to as *tonicity* due to the ability of the fibers to maintain dynamic contraction over time; they are mostly found in the areas of the body where maintenance of activity against gravity is the main function. They have a stabilizing function through their precise grading of activity. Tonic activity is dynamic, and the word tonicity refers to something that is “characterized by tension or contraction, esp. muscular tension” (Taber’s Cyclopedic Medical Dictionary 1997). The motor units containing type 1 fibers are characterized as S (slow to fatigue; Rothwell 1994).

Examples

The soleus muscle is continuously active during standing and walking. Therefore it needs to have high endurance and consists of tonic/type 1 motor units. The soleus muscle is characterized as a postural muscle.

The small muscles of the hand, the interossei, and the lumbricals also mostly have type 1 fibers (Rothwell 1994). The small muscles of the hand stabilize the palm and the metacarpophalangeal joints and provide the hand with a postural background for individual finger movement and precise fine motor control.

The small muscles of the foot have a stabilizing function for maintenance of equilibrium of the body. The small muscles of the back are important for postural control of the trunk and therefore core stability.

- Type 2 fibers are also called FT (fast twitch) because they have a faster contraction speed than type 1 fibers. These muscle fiber types are described as white and have a low oxidative capacity with little endurance but increased tempo and force production. They are phasic by nature, and their main function is production of movement. The motor units are classified as FF (fast, fatigable). Type 2 fibers are further subdivided into:
 - Type 2A or FOG (fast oxidative glycolytic; Rothwell 1994). The motor units are classified as FR (fatigue resistant) because they have more endurance

Example

The gastrocnemius muscle has an important role in force production during locomotion, running, jumping, moving on uneven ground, and climbing steps, which require both endurance and force. The muscle therefore has a larger proportion of FOG muscle fibers.

- Type 2B are the truly white fibers (Brodal 2001). The motor units are classified as FG (fast glycolytic; Rothwell 1994) and have low endurance and produce a lot of force

Example

The tibialis anterior muscle works intermittently during locomotion and in standing. Its use therefore is mainly aimed at phasic activity and low demands of endurance.

Muscle fibers are able to alter their fiber type to some degree in relation to use (Brodal 1998). At birth most muscles are composed of slow (type 1) muscles and only as the body matures does the final proportion of slow and fast muscles emerge (Langton 1998). Athletes have different distributions of muscle fiber types depending on their preferred sport. Long-distance runners, cyclists, and cross-country skiers have a larger percentage of type 1 red fibers, whereas weight-lifters, short-distance runners—sports that require rapid production of force—have a larger percentage of white type 2 fibers. Partly, this is probably due to the individual genetic profiles but muscle plasticity forms a major basis for the physiologic adaptation to our external environment.

Examples of muscle plasticity are adapting to exercise, effects of a microgravity environment, aging, and different pathophysiologic conditions. Muscle plasticity can be both beneficial and maladaptive. Muscle cells display a tremendous ability to adapt to new levels of gene expression in response to a wide range of environmental demands and clinical conditions (Sieck 2001). *Gene expression* is the process by which a gene's information is converted into the structures and functions of a cell (Wikipedia 2006). Alteration of muscle fiber types is a result of changed gene expression. This may be termed *use-dependent plastic adaptation*.

Trials using electrostimulation have demonstrated that muscle fibers may change if the information to the muscle fibers and functional demand are altered (Kidd 1986, Langton 1998).

Muscles are able to alter their fiber type to some degree in relation to use.

Contraction of skeletal muscle is caused by gradual overlap of actin and myosin filaments within the muscle cell or fiber. A muscle fiber is composed of many areas of overlapping myosin and actin called sarcomeres. The numbers of sarcomeres determine the length of a muscle fiber: the more sarcomeres, the longer the muscle. Normally, in the human body the number of sarcomeres, and therefore the length of the muscle, is optimal for the function of the muscle. The force of contraction is therefore at its best within the range of movement where it is most

needed. The sarcomeres are able to produce very little force if the fiber is overstretched or kept in a much-shortened position. The length of the muscle fibers is affected by the way the muscle is being used. If a muscle is kept in a much-shortened position over time, this may lead to an anatomic shortening due to loss of sarcomeres. Sahrman (1992) states that a shortened muscle is more easily recruited than its antagonists which are in a lengthened position, and that as a result of this, the shortened muscle is stronger. She calls this *biased recruitment*.

If a muscle is kept stretched over time, it may start “growing,” that is, the number of sarcomeres may increase. This may cause the muscle to be too long to be able to produce the appropriate amount of force needed for an activity. Sahrman (1992) calls this *stretch weakness*.

The length of a muscle is important for movement and function.

A motor unit comprises of an anterior horn cell (α -motor) with its axon and the muscle fibers it innervates (Fig. 1.1). All muscle fibers in a motor unit are of the same type, and within any muscle there are many other motor units with different fiber type representation. All contact between the CNS and the muscle is through the peripheral nervous system. Brodal (2001) states that motor units vary in size depending on the size and function of the muscle; there

may be fewer than 10 muscle fibers within a unit, such as the external eye muscles, or more than 1000, as in the large muscles of the back. The number and size of the motor units vary. Smaller units are found in muscles that perform precise movements with moderate strength. Mostly the smaller units contain type 1 muscle fibers, whereas the larger units have type 2 representation. The deltoid muscle contains approximately 1000 motor units, 60% of which have type 1 fibers. The first dorsal interosseous muscle has approximately 120 units, of which approximately 57% contain type 1 fibers, but individual differences exist (Rothwell 1994). Motor activity of a muscle is recruited sequentially through the Henneman recruitment principle (Henneman and Mendell 1981), whereby small, slow motor units (containing type 1 muscle fibers) are activated before the larger and faster motor units containing phasic muscle fibers. The recruitment principle has been called the size principle of recruitment by Brodal (2001) and recruitment order by Rothwell (1994).

The force of contraction may be graded in two ways (Brodal 2001):

1. The number of motor units that are recruited. If the number increases, so does the force production
2. An increase in the impulse frequency of the motoneuron, which also leads to increased force production

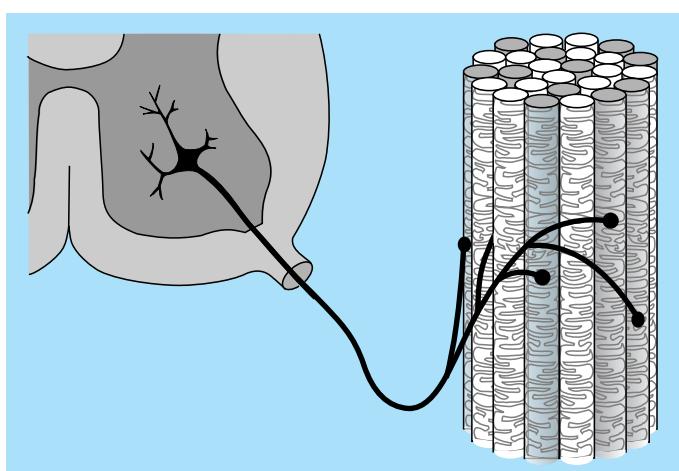


Fig. 1.1 A motor unit is comprised of a number of muscle fibers with the same muscle fiber type, the α -motoneuron that innervates it, and the axonal branches of the α -motoneuron to the individual muscle fibers. Muscle fibers belonging to different motor units are dispersed within the muscle.

Functional Relevance

Small motor units that demonstrate the greatest ability for endurance (“tonic activity,” sustained activity) occur in maximum numbers in muscles whose the main function is postural activity, i.e., sustained activity against gravity. Several authors have described postural activity as the basis for function of the extremities (Dietz 1992, Massion 1992, 1994, Shumway-Cook and Woollacott 2006).

Motor units in a muscle are recruited sequentially, whereby the smaller motor units are activated before the larger motor units. Postural stability is the basis for selective movement control and function.

Most muscles have a mixture of different motor units. The musculature is therefore able to function in relation to different activities: a muscle may have a stabilizing function in cooperation with some muscles or more of a mobility function when working with others. Motor units are recruited sequentially to enable the musculature to grade its activity in relation to strength, synergistic musculature, and required function (Massion 1992). Muscles can vary their activity and function as agonists, antagonists, or synergists depending on how they are being used.

Most muscles have internal selectivity based on the distribution of motor units and muscle fiber type and size. Motor units may be activated differentially; some may work eccentrically at the same time as others are working concentrically to varying degrees. Anatomically defined muscles that cross two joints or more may eccentrically lengthen over one joint while shortening over the other. This ability is called *compartmentalization* (van Ingen Schenau et al. 1990).

Example

The quadriceps muscle continuously varies its activity during locomotion; in stance phase, the proximal part has to contract eccentrically to allow for hip extension, whereas at the same time the distal part has to work concentrically to stabilize the knee for weightbearing. During the initial swing, the activity of the quadriceps is reversed; the proximal part undergoes more concentric contraction to assist in swinging the leg forward whereas the distal part works more eccentrically to allow knee flexion.

Compartmentalization describes the ability of a muscle that crosses more than one joint to perform different functions simultaneously.

Muscle Balance

Muscle balance is the result of cooperation within and between many muscles or muscle groups surrounding a joint: agonists, antagonists, and synergists. In humans with an intact CNS and musculoskeletal system the grading of activity in the different muscle groups is finely tuned and adapted to the relevant function and situation. Maintenance of muscle balance depends on neurologic, muscular, and biomechanical factors (Sahrmann 1992, 2002, see also Stokes 1998):

- Muscular factors—such as the length–tension relationship of the muscle and its ability to produce force appropriately
- Neurologic factors—the sequence of recruitment of motor units within the muscle and the sequence of activation of different muscles or muscle groups
- Biomechanical factors—alignment, structure, and function of the joints

Muscle Imbalance

Muscle imbalance may result if any of the above-mentioned factors are disturbed, for instance neurologic problems leading to malalignment.

Muscle balance depends on muscular, neurologic, and biomechanical factors. Alterations in recruitment and the distribution of motor activity affect alignment. Altered alignment affects muscle function.

Zackowski et al. (2004) describe *impaired joint individuation* as the inability to fix joints that should have been fixed during movement of another joint, and explain this phenomenon as impairment in motor control. These authors refer to other studies providing further evidence for reduced capacity of an impaired limb to generate certain muscle co-activation patterns. This may be due to abnormal spatial tuning (distribution) of muscle activity.

■ Noncontractile Elements

The connective tissue unites, supports, and holds the structures of the body together. Fibrous tissue is elastic and supports muscles and joints as well as allowing movement. With increasing age, the fibrous tissue loses its strength and elasticity. If fibrous tissue is kept shortened, contractures may result (Tyldesley and Grieve 1996).

■ Sense Organs in Muscle

Muscle Spindles

Muscle spindles are specialized receptor organs found in between and in parallel with muscle fibers in skeletal muscles. The muscle fibers of a muscle spindle are called *intrafusal fibers*. The muscle spindles inform the CNS of the length of the muscle, length changes, and the speed of change via primary (Ia) and secondary (II) afferent nerve fibers (Fig. 1.2). The muscle spindle is innervated by a γ -motoneuron, which can cause contraction and so alter the length of the spindle. Therefore it can modify the sensitivity of the spindle during alterations in length or stretch of

the muscle. Muscle fibers are innervated by α -motoneurons. In a relaxed muscle there is hardly any detectable activity in the muscle spindles, and the probability of impulse activity in the γ -motoneurons is therefore small. The impulse frequency from the Ia afferent fibers increases strongly with isometric contraction of the muscle. This is caused by an increase of γ -activity as the α -motoneurons stimulate the extrafusal muscle fibers to contract. This is called α - γ co-activation. The sensitivity of the muscle spindle seems to increase when the muscle works actively. If a muscle actively contracts and shortens, the impulse frequency of the Ia fibers is maintained at a high level, which indicates that the γ -motoneurons also have to increase their activity (Brodal 2001). The function of the γ -motoneuron is to maintain the sensitivity of the muscle spindle during muscular activity (Kandel et al. 2000). This system seems to be important for the maintenance of tonic contractions, e.g., keeping the knee stable during loading of the leg in stance phase. The musculature depends on a harmonious interplay between afferent information from the spindles and efferent

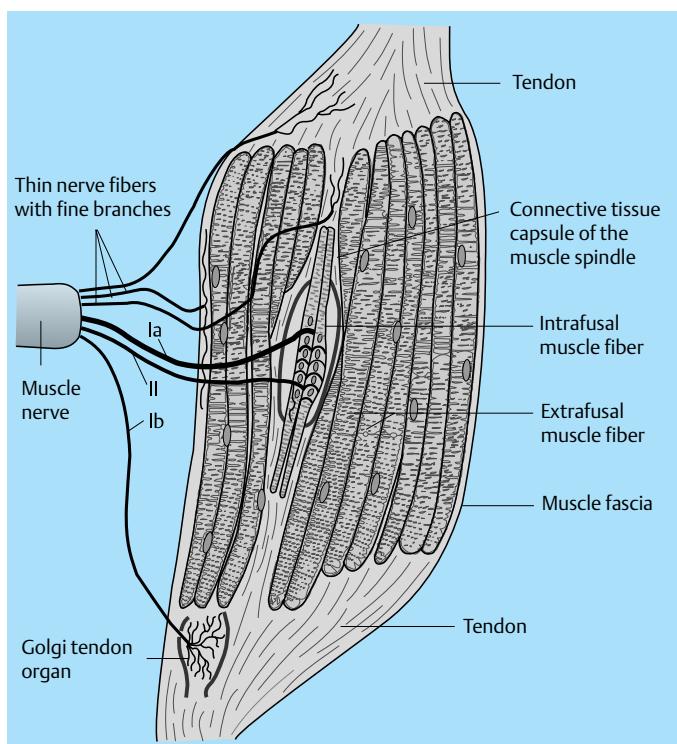


Fig. 1.2 The muscle spindle and the Golgi tendon organ. The muscle spindles are situated in between and in parallel with the extrafusal (skeletal) muscle fibers and are attached to the tendon through connective tissue.

motoneuron activity. Through this, the muscle is always ready for alterations in activity.

The muscle spindles inform the CNS continuously of the state of the muscle (Dietz 1992). The CNS therefore knows, all the time, about the movement that is about to happen, is happening, or has happened, and compares these. If the movement was not performed as anticipated, the CNS may correct the activity. In this way, the CNS may modulate and control motor activity accurately and increase the reaction to unexpected disturbances, for instance in balance (Brodal 2001).

The number of muscle spindles in different muscles vary, with greater numbers present where the need for precise grading of activity is necessary e.g., in the small muscles of the hand or the deep postural muscles of the back.

Golgi Tendon Organs

Golgi tendon organs are specialized receptors that inform the CNS about tension changes within the muscle (Brodal 2001). They are innervated by a sensory nerve fiber, the branches of which encircle the connective tissue fibers in the organ. The connective tissue fibers are attached to muscle fibers at one end and to the tendon proper at the other end (see Fig. 1.2). These muscle fibers belong to *many different motor units*. The tendon organ is therefore able to detect tension changes and the distribution of activity in different motor units within the muscle at the same time.

Muscle Tone

Tone is related to the state of the muscle fibers, the activity within the sense organs, muscle viscosity, and connective tissue. Muscle tone is an expression of the stiffness of the tissue. The relationship between muscle length and tension is called *stiffness* (Brodal 2001). The control of this relationship is called *stiffness control*.

Muscle tone is usually the term used to describe the tension in relaxed muscle, and is also called *resting tone*. Brodal (2001) states that the most important factor in changing the level of tone is muscular contraction. The viscoelastic properties of the muscle fibers, connective tissue in the muscle and the muscle tendon add to this to a lesser degree (see Simons and Mense 1998 for discussion). Shumway-Cook and Woollacott

(2006) define *postural tone* as activity in muscles that counteract the force of gravity in the upright position. They state that "muscles throughout the body, not only those of the trunk, are tonically active to maintain the body in a narrowly confined vertical position during quiet stance." They use the term *ideal alignment* to describe the increase of muscle work needed when the body moves outside a narrowly confined vertical position, i.e., even small ranges of movement increase the demands on muscular activity. To maintain normal function, tone needs to be high enough to allow the body to be *dynamically active* in relation to gravity. Postural tone is influenced by information from somatosensory receptors (skin receptors in the soles of the feet and neck receptors among others) and visual and vestibular input. Other factors that influence tone are pain, fear, and input from other areas of the brain and spinal cord.

Tone is related to the state of the muscle fibers, the activity within the sense organs, muscle viscosity, and connective tissue. The most important cause of alteration of tone is muscular contraction.

Information to the spinal cord comes from all somatosensory receptors in the body, for instance skin, joints, connective tissue, muscles, tendons as well as from other sensory systems (vision, hearing, equilibrium) and other motor systems within the CNS. On any one motoneuron there may be as many as 50 000 synapses from sense organs and receptors, and from all levels and pathways within the brain and spinal cord (Fig. 1.3). Information is modulated continuously, and may result in motor activity. Muscle length, tension, and activity will in most situations be appropriate for the function or activity to be performed because of this integration of information (Brodal 2001).

Clinical Relevance

Many people who have suffered a CNS lesion have a reduction in balance, selective control of movement and strength. The lesion itself, how the person is being positioned (how he sits, lies), demands for independence, stimulation, training, and the person's ability to activate and control his own body, will influence the neuro-

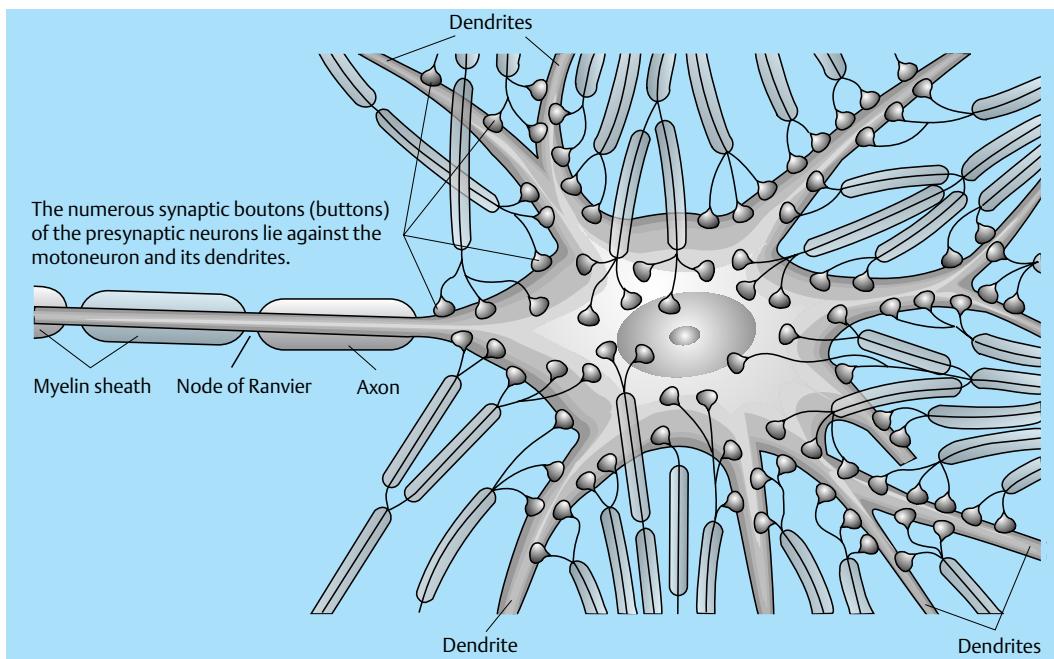


Fig. 1.3 The synaptic connections to the motoneuron. There may be as many as 50 000 synapses on one motoneuron.

muscular adaptations that occur over time. The neuromuscular system adapts to the new situation and how the body is being used. This may lead to:

- Changes in the length–tension relationship, alteration of tone
- Altered recruitment pattern of motor units, which may disrupt the ability to stabilize the body/body part as a background for movement
- Muscle imbalance (reduced interplay between different muscle groups)
- Muscle fiber type changes and the level and constitution of connective/fibrous tissue; increased or decreased length of muscles; the muscle may be too stiff or stretched and cannot be activated efficiently and functionally
- Changes in the way the patient moves and uses his body; the need for new movement strategies to achieve goals. When the patient lacks the ability to initiate the appropriate activity for a required function he finds other ways—compensatory strategies. Patients use the movement strategies available to them to be functional here and now. Inappropriate activity may strengthen the above factors

and complicate and limit the patient's choice of movement in both the short and the long term

- Alignment problems as a result of tonal factors, use, changes in contractile and noncontractile tissue will affect muscle function
- Altered somatosensory information or perception may affect the patient's ability to move

Hypotonia and *hypertonia* are terms used to describe tonic changes within muscle. Brodal (2001) describes *hypotonia* as reduced tone in the musculature. In a clinical situation, this is perceived as reduced ability to activate muscles appropriately and as a lower muscular tension or stiffness than expected in the same situation when compared with people without a CNS lesion. *Hypertonia* is described as a continuous increase in tension or stiffness even if the person attempts to relax (Brodal 2001). Clinically this is perceived as an inability to grade and modulate tone, or as a higher muscular tension or stiffness than expected in the same situation when compared with people without a CNS lesion.

In the early stages of an acute brain lesion, patients may display a more generalized paresis, and in the setting of acute traumatic brain injury patients may have severe hypertonia throughout the body (opisthotone). Lesions of certain parts of the brain stem may give a clinical picture of severe low tone/paralysis, often bilaterally. However, the therapist rarely encounters patients who are either totally hypo- or hypertonic. In most patients, there is a mixture of muscles and muscle groups presenting with high and low tone and some areas with more normalized activity. Increased tone causes more stiffness of muscle, less pliability and flexibility, whereas low tone may result in instability, when the precise grading of activity is inappropriate.

The musculature seems to lose its variability and flexibility in CNS lesions. Altered distribution of motor unit activity and changes in alignment may affect muscle function negatively—the muscles have new working conditions. The patient therefore may be unable to align and recruit the neuromuscular activity necessary to reach the desired goal efficiently. *Efficient* in this context means that the patient does not need to use more force or exertion than would be normal for a person without a CNS lesion.

The latissimus dorsi is anatomically described as one muscle, but comprises many segments working together to create function. When all of the latissimus contracts, it brings about extension of the spine, increased lumbar lordosis combined with internal rotation, adduction, and extension of the arm. During normal conditions, the latissimus is able to activate differentially by increasing lumbar extension at the same time as the arms are stretched above the head, as a jumper is pulled over the neck and head while undressing (Fig. 1.4), i.e., it contracts concentrically distally and eccentrically in relation to arm function proximally—both at the same time. In my clinical experience with CNS lesions, it seems as if the ability of the muscle to perform differential actions is disturbed. The patient's ability to compartmentalize muscle activity may be reduced, and the muscles seem to contract in their total range of movement when activated. In the case of the latissimus dorsi, the arm tends to rotate internally, adduct and extend together with lumbar extension when activated. If this total pattern of movement is learned, the patient's functional ability and independence will be reduced.



Fig. 1.4 Activation of the latissimus dorsi.

Muscle fiber changes have been shown in CNS lesions (Ada and Canning 1990). Inactivity due to immobilization, denervation, or reduced activation makes the musculature prone to atrophy. Muscle fiber type changes may also be present: Hufschmidt and Mauritz (1985) state that a tonic transformation of muscle fibers may be one reason for the increased resistance to stretch experienced in spastic muscles. Phasic muscle fibers may be transformed to undergo more tonic activity.

In many neurologic conditions it seems as if the patient's postural control is mostly affected. Clinical experience suggests that the patient uses the strategies available to him to maintain balance, for instance by fixing with the arms, increasing arm support, or flexing and adducting the hips. When the arms are used for balance, they are being recruited to support the body and are not free for functional use. Normally,

phasic activity dominates in arms due to the need for rapid movement in a variety of different contexts. When the arm muscles are recruited to maintain stability, the functional demands on arm musculature change, and a gradual transformation of muscle fiber type may ensue, contributing to the increased stiffness of arm musculature experienced by some patients.

Muscle length changes occur in pathologic conditions of the CNS (Goldspink and Williams 1990). When patients are kept sitting for many hours each day, the hip flexors are kept in a shortened position. The muscle fibers shorten and adapt to the position in which they are being held. This may lead to a reduction of sarcomeres so the muscle becomes anatomically shortened. When the patient attempts to stand up or is being transferred through standing by helpers, the shortened muscles experience stretch. The muscle spindles and Golgi tendon organs inform the spinal cord of the stretch and tension, the α -motoneurons are activated to contract the hip flexors to take the tension off the spindles, and the hip flexors contract too early due to predisposed recruitment. So the patient either lifts the leg off the floor and is thereby destabilized, or is pulled down in the hips and pelvis and is not able to reach a standing position.

Conversely, the patient's hip extensors are in a lengthened position while sitting. As the patients may sit for many hours each day, the hip extensors are passively stretched and are stimulated to "grow" in length. As a result, the number of sarcomeres may increase, which leads to the muscle being unable to produce an appropriate amount of force to allow the patient to stand up, maintain standing, or stabilize the hip during the stance phase of walking. This is due to overstretch weakness. As a result, the patient may have to use his arms for support during transfers, in standing, and walking. The use of arm support increases the patient's flexor activity (pressing down) through the arms and trunk—the patient therefore attempts to maintain standing through flexor activity and negate extension.

If a patient's arm is kept positioned on a table in front of him or in his lap for long periods every day, there is a danger of the biceps shortening distally. Proximally the length changes will depend on the position of the shoulder. Triceps will experience prolonged stretch distally and

usually shortening proximally. Both lose their ability to be activated functionally (Ada and Canning 1990).

Inactivity causes the amount of fibrous tissue within the muscle to increase and the muscle becomes stiffer (Goldspink and Williams 1990). The opposing muscle groups, joint capsule, and ligaments may become stretched and the stiffness decreases in the stretched muscle. There may be, as a result, an imbalance in the supporting tissues and therefore loss of stability. This may negatively affect the patient's ability to move.

The Somatosensory System, Vision, and Balance

The Somatosensory System

The term **somatosensory** is related to sensory experiences within the body (*soma*). In this chapter the discussion will be limited to sensory information from the skin, joints, and muscles.

Somatosensory information is received and carried through the peripheral nervous system; from receptors in the body to the spinal cord. Somatosensory information is to some degree modulated and integrated in the spinal cord. Information is transmitted through ascending fibers that synapse with interneurons and motoneurons at spinal level as they ascend to the brain. Somatosensory information is transmitted via the dorsal root ganglion of the spinal cord and two ascending pathways: the anterolateral system and the dorsal column medial lemniscus pathway (Fig. 1.5) (Kandel et al. 2000).

- The anterolateral pathway comprises of the spinothalamic tract and the spinoreticular tract, which transmit information related to pain and pain/temperature, respectively. Information ascends contralaterally in the spinal cord to the thalamus.
- The dorsal columns and the medial lemniscus pathway: the gracile fascicle and cuneate fascicle and the spinocerebellar tract. The fasciculi cuneatus and gracilis pass on information from receptors in the skin and joints in the upper and lower part of the body, respectively, which travel ipsilaterally to the cuneate and gracile nuclei in the brain stem. Here, they cross over to form the medial lemniscus. As the fibers ascend further, they gradually move

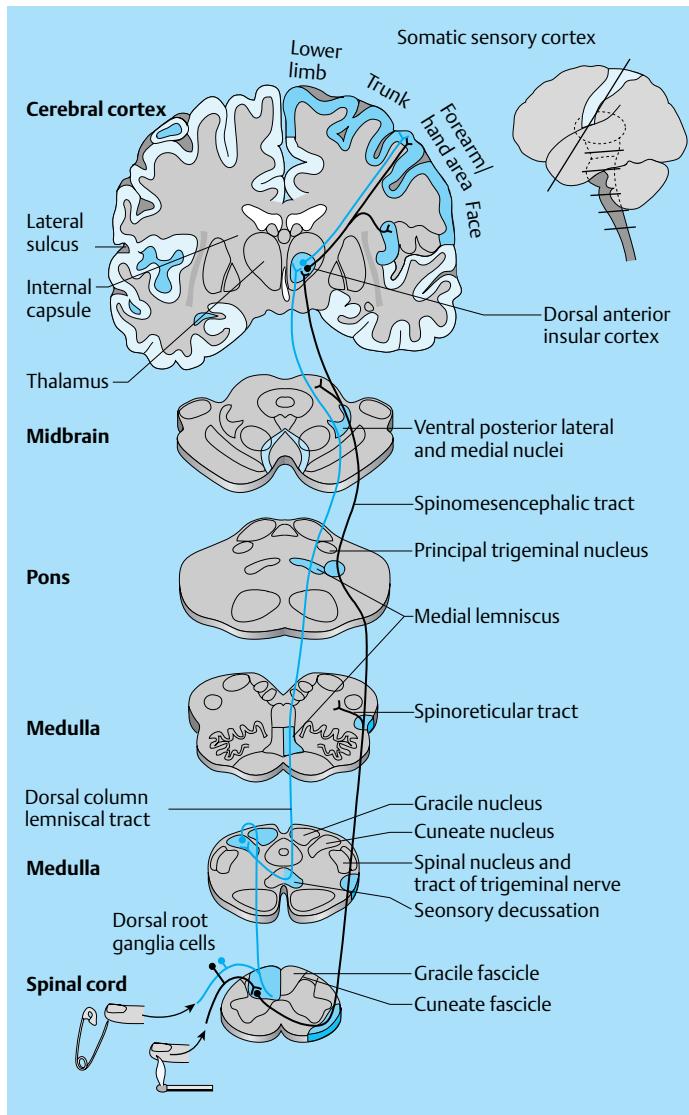


Fig. 1.5 The figure illustrates the dorsal column medial lemniscus system—the sensory nerve entering the spinal cord, and the ipsilateral pathway to the nuclei cuneate and gracilis in the brain stem. Here the nerves cross over to form the medial lemniscus. The spino-

reticular and the spinothalamic tracts cross over as they enter the spinal cord to form the anterolateral system. From the brain stem these two systems ascend together to the thalamus and to the cortex. (Redrawn with permission from Kandel et al., 2000, p. 447.)

more laterally and join fibers of the spinothalamic tract in the thalamus.

Somatosensory impulses are modulated in the thalamus, which also receives information from the basal ganglia, cerebellum, the eye, and auditory nuclei. Therefore it has an important role

in making the cortex aware of sensation. The thalamus has connections to the basal ganglia, the cortex, the red nucleus, and cerebellum. As a result of this network, it is able to process different types of information and has a close relationship to motor and sensory areas of the brain and to

the basal ganglia. Thus it has a role in interpreting the world. The somatosensory impulses leave the thalamus and travel to somatosensory areas of the cortex via the internal capsule.

Direct pathways originate in the interneurons within the spinal cord. Two such important pathways are the *ventral* and *dorsal spinocerebellar tracts*, which terminate in the spinocerebellum as

mossy fibers (see Cerebellum, page 34). They send proprioceptive information to the cerebellum from the legs—from joints and the activity within muscle, and from the muscle spindles and tendon organs, i.e., information about movement and about *descending commands* reaching the interneurons.

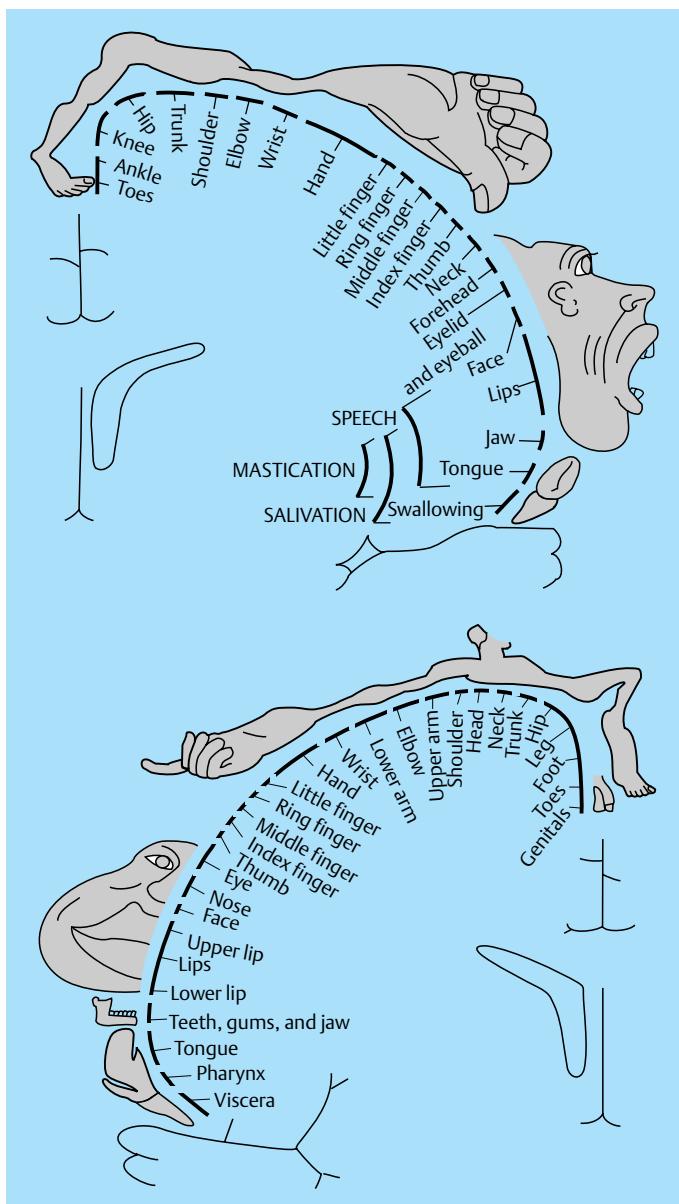


Fig. 1.6 The sensory and motor homunculi show the somatotopic organization of sensory and motor representation within the cortex.

Direct pathways synapse in different precerebellar nuclei in the reticular formation of the brain stem. These inputs inform the cerebellum about different versions of the changes in activity in the body and its environment, and permit comparison of signals. Studies in cats walking on a treadmill have shown that these pathways are modulated rhythmically and in phase with the step cycle. The ventral tract carries internally generated information about central locomotor rhythm and the rhythmic discharge of somatosensory receptors. The dorsal tract carries information about ongoing movement only (Kandel et al. 2000).

Clinical Relevance

Data indicate that loading (heel-strike) and unloading (heel-off) in locomotion provide the CNS information from spatial and temporal parameters, proprioceptive information, pressure receptors, and recruitment patterns of the lower limbs (Trew and Everett 1998). Weightbearing regulates the step cycle by influencing stance duration, and sensitivity to loading has been observed in humans. The degree of leg extensor activation is highly correlated with the percentage of body loading and has been found to be functionally phase dependent (Mudge and Rochester 2001). Data demonstrate that the level of loading through the limbs during cyclic activity can provide important information that facilitates the generation of steplike efferent patterns by the human lumbosacral spinal cord, by providing cues that modulate the activity of the motoneurons innervating the lower limb muscles. Limb unloading has been shown to be an important signal for the termination of stance and the initiation of swing in cat locomotion (Harkema et al. 1997).

Heel-strike is important for initiation of stance and therefore locomotion.

Heel-off is an important signal for the termination of stance, and therefore for swing phase.

Patients with CNS lesions often have reduced control and mobility of the ankle and foot; at times hypertonicity or stiffness of the calf muscles prevents heel-strike, and the patient may show inactive dorsiflexion, or activates patterns of inversion and plantarflexion during walking.

An inability to place the heel on the floor as the first component of stance or to lift the heel off the floor as a signal for the termination of stance disrupts the ability to achieve a stable stance phase of locomotion as a prerequisite for an efficient swing phase.

Sensorimotor Integration

More than half of the human cortex contains association areas that coordinate events arising in the motor and sensory parts of the CNS. These areas are involved in planning, thinking, feeling, perception, speech, learning, memory, emotions, and motor skills. Large parts of descending commands modulate sensory information in the spinal cord and brain stem. Sensory information modulates motor activity at all levels within the CNS. The primary sensory cortex (SI) has cortical representations of the different body parts, called cortical maps, and is the first level where humans perceive sensory information. This has been represented as the sensory and motor homunculi (Fig. 1.6). SI, SII, and the posterior parietal cortex (Fig. 1.7) all receive information from the thalamus. The sensory areas send information to both motor and association areas of the cortex, which is important for the ability to recognize and localize sensory stimuli.

Precise motor activity depends on close integration with the sensory systems. Most movements require a constant flow of information

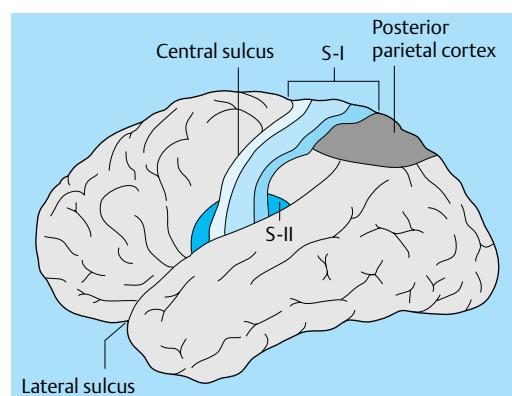


Fig. 1.7 The three major divisions of the somatosensory areas of the cortex: primary sensory cortex S-I, secondary somatosensory cortex S-II, and the posterior parietal cortex.

from receptors in the skin, joints, and muscles to evaluate if the movement is proceeding as planned. Information from the ocular and the vestibular systems can be of vital importance for motor performance. Sensory information enables the CNS to update and correct the outgoing commands to the musculature—either during ongoing movement or the next time it is being performed (Brodal 2001). Activity in ascending fibers may simultaneously affect the activity in the descending fibers, and vice versa. Somatosensory and visual information is crucial for exploration of the environment. Human interaction with the environment forms the basis for muscular activity, movement, and balance. Motor activity is context based.

Vision

Visual information travels from the eyes to the visual cortex in the occipital lobe of the brain for awareness of visual input. Visual impulses are also transmitted to the nucleus superior colliculus in the brain stem, both directly from the optic nerve and indirectly through the visual cortex. Human beings are therefore able to automatically turn their head toward something in the environment and the control eye movements. Visual information is also transmitted to the cerebellum and has a role in coordination.

Vision is essential for mobility, the ability to read, visual orientation, and activities of daily living (ADLs) (Kerty 2005). Visual information is important for anticipatory adjustments to movement, in feedforward motor control and eye-hand contact for precise manipulation of objects. The use of a knife and fork for eating or the ability to thread a fine needle depends on sensory and visual information that enables us to get food on the fork or the thread into the eye of the needle. If vision fails, other somatosensory inputs compensate. People without eyesight or reduced eyesight develop an increased sensitivity to specific receptors in the skin to be able to control movement with good precision.

As we move about we receive visual information, both about the environment and the relative position and movement of the body within the environment. The eyes scan the environment in the direction of movement: the height of the steps on a staircase, the position of furniture or any obstacles, and the position and movement

of people. Vision is important for appropriate placing of our feet if the terrain is unknown, uneven, or complex, or when balancing on a narrow beam or near the edge of a cliff, but it does not dictate the detailed placing of the foot during normal walking. The dependence on visual information reduces as the environment becomes more known.

If vision fails, as when moving through a dark room, then proprioceptive and vestibular inputs become more important for movement. On the other hand, vision may compensate if information from other systems fails, and may become the main contributor in balance control. The in-

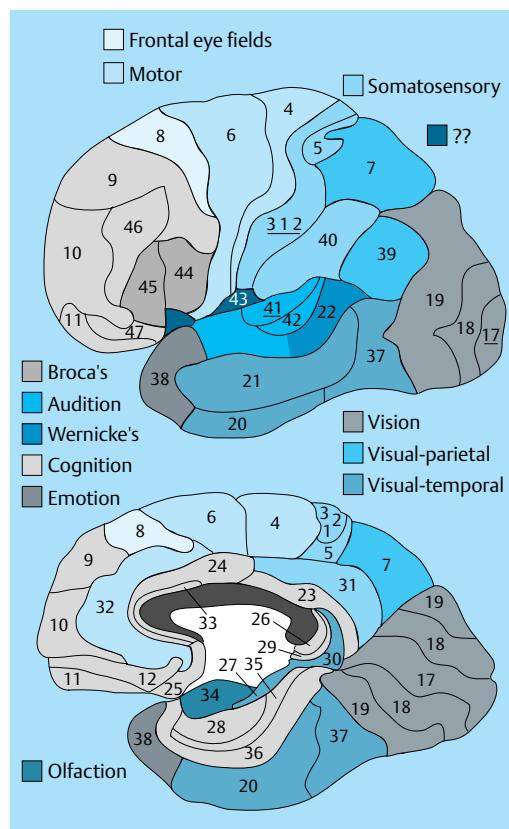


Fig. 1.8 Areas for vision, motor activity, speech, hearing, cognition, somatosensation, and emotion within the brain. Many of these areas receive or integrate information from the visual system. The numbers refer to Brodmann areas, which are histologically defined regions. (Reprinted with permission from Professor Mark W. Dubin, MCD Biology, University of Colorado at Boulder.)

teraction of the different systems involved in movement and balance are adaptable to new conditions by weighing the relative importance of the different afferent systems in relation to what is needed (Brodal 2001, Wade and Jones 1997).

Visual problems are present in as many as 40% of people who have had a stroke and in 50% after traumatic brain injury (Kerty 2005). Visual disturbances following a stroke are a result of the integrative nature of the brain and the fact that visual function involves considerable areas of the brain (Fig. 1.8).

The one-sided visual defect often seen in stroke (hemianopia) is caused by a lesion to the optic nerve tract from the optic chiasma to the occipital cortex. A stroke may also affect association areas in the lower temporal region (recognition of objects or shapes) or the posterior parietal cortex (spatial awareness) (Riise et al. 2005). Visual defects may therefore be a contributor to a patient's balance and functional problems.

Ascending and descending systems are closely linked both anatomically and functionally. It is therefore inappropriate to discuss these separately. Motor activity is the result of a complex interaction between sensory, motor, and cognitive systems.

■ Balance

The terms *balance* and *postural control* are often used interchangeably (Chapter 2 discusses these terms in greater depth). Reduced postural control is recognized as a major factor contributing to mobility problems in stroke patients. It is caused by dysfunction in the complex interaction of motor, sensory, and cognitive processes (de Haart et al. 2004). Balance is affected by afferent visual, vestibular, and proprioceptive information. Which system is weighted most depends on the need of the moment, and the relationship between the individual, the environment, and the function to be performed. Our CNS needs to be aware of where the body is, where the different body parts are in relation to each other and the environment, and the activity within the different regions of the body to be able to respond appropriately. Vestibular information seems to be dealt with in a distributed cortical network. The

integration of other signals, spatial information, body position, and movement occurs within the same network. This is connected to other cortical networks, for instance motor networks, and vestibular information probably contributes to our conscious awareness of body position and movement in relation to the environment (Brodal 2004). There is a continuous demand for adaptation to the environment, and therefore for variation in stability, balance, and movement. We move in relation to our base of support, and in standing or walking, our feet must adjust to the floor, the grass, stones, or sand.

Several studies demonstrate a close relationship between anticipatory adjustments (feedforward), associated adjustments (ongoing adjustments), and adaptations (feedback) in normal postural control (Horak et al. 1997, Wade and Jones 1997). "The classic concepts that the movement originates and commences in MI [primary motor cortex] and that the PT [pyramidal tract] represents the highest level of motor control are now thought to be incorrect. The modulation of PT neuron firing is determined largely by the 3 major types of information that MI receives and processes: *internal feedback* and *external sensory feedback*" (Davidoff 1990, p. 334).

- Anticipatory adjustment of muscles (feedforward) is an integral part of every movement, and is called *central motor programming*.
 - Anticipatory adjustment depends on *internal feedback* about the position of different body parts to each other, their activity and alignment, the relationship between the body and the environment, and the function to allow for error correction before errors occur. Anticipatory adjustment of alignment and muscular activity are therefore dependent on somatosensory feedback.
 - As movement is being initiated, the CNS receives continuous impulses from receptor organs (eyes, muscle spindles, tendon organs, joint receptors, pressure receptors, skin receptors) and can correct movement as required. This is a feedback function, *external sensory feedback*, whereby planned and performed movement may be compared and differences corrected. This is especially important for precise, small movements and postural stability.
- Information used for postural control is essential for our experience and image of own body.

Humans have an internal picture—body image or body schema—of how our body looks based on information from proprioceptors, vision, and equilibrium organs. *Body schema* is described as an internal postural picture informing about the position of the different body segments in relation to each other and their relationship to the environment. Body schema provides a basis for exploration of the environment for perceptual analysis and motor performance. Body schema information needs to be continuously distributed to networks that plan movement to update movements and make them appropriate to the moment. Networks that deal with different aspects of bodily perception and movement are closely linked. Most cortical areas activated by the vestibular apparatus also receive information from proprioceptors and probably contribute to the integration of these two sensory modalities (Brodal 2004).

Anticipatory adjustments of muscle activity (feedforward) are dependent on both external and internal feedback. That is, information about the internal relationship between body parts, the relationship between the body and the environment and from specific receptors from the eyes, muscle spindles, golgi tendon organs, and receptors in the skin is necessary for feedforward and therefore for balance.

Through feedforward the CNS may set an appropriate level of motor activity as a preparation for movement. Wade and Jones (1997) describe anticipatory and associated adjustments together as postural adjustments closely connected with the performance of voluntary activity. There is a belief that these types of adjustments reduce the postural displacements associated with a displacement of the centre of gravity caused by voluntary movement, as well as affecting voluntary movement directly. Massion (1992) states that motor activity may be recruited sequentially (after each other) or in parallel (simultaneously):

- Either first the anticipatory activity, until the postural adaptations eliminate the displacements caused by movement and thereby stabilize the body, and then movement of arms or legs in standing
- Or simultaneous (parallel) activation of stability and movement

The most important component of the human ability to stay upright is continuous information about the effect of gravity through specific receptors, for instance proprioceptive receptors registering weightbearing, the distribution of pressure, and the line of gravity in relation to the feet. Dietz (1992) stated that postural activity depends on the activity in specific receptors informing the CNS of deviations in the center of gravity from a certain *neutral position*. This is what Shumway-Cook and Woollacott (2006) call the *ideal alignment*. Pressure receptors are distributed throughout the body, in joints and the spinal column as well as the soles of the feet (Petersen et al. 1995, Brodal 2001). Studies indicate that there are specialized gravity-dependent receptors in our internal organs also (Karnath et al. 2000). Activity in postural muscles, such as the soleus (lower leg), also seems to be important in this respect. If specific receptors are to inform the CNS of displacement, they have to be fired by activation and variation in the sensory impulses through movement, alteration of pressure areas, changes to the stiffness-tension relationship within muscle, and alteration of joint alignment. Mobility of joints and soft tissue is important to fire the sensory receptors and therefore for balance. Mobility combined with good alignment and appropriate neuromuscular activation gives the patient the ability to respond to displacements.

Information (feedback) about muscle activity, movement, alignment, and weight distribution is important for balance, postural adaptation, and function.

Mobility is a prerequisite for the firing of specific receptors in relation to displacements and variations in sensory input, and therefore for balance.

We need information from the somatosensory system about the position of our body in the environment to plan and perform functional movement. An updated body schema is a prerequisite for appropriate and efficient feedforward. When standing on a stable base of support, somatosensory information is especially important to generate automatic postural adjustments and provides the most important, although not the only, input to postural control (Nashner 1982).

Dietz (1992) states that all motor activity in standing or walking both starts and finishes with postural adjustments. Mulder et al. (1996) says that the ability to react needs a higher level of sensorimotor (re)integration than anticipation.

An updated body schema is a prerequisite for appropriate and efficient feedforward control.

Stereognostic Sense

Stereognosis is described as "The faculty of perceiving and understanding the form and nature of objects by the sense of touch," that is, without the use of vision (Mosby's Medical, Nursing and Allied Health Dictionary 1994). Stereognostic sense relates mainly to hand function. The stability, mobility, sensitivity, and adaptive ability of the hands are crucial for exploration of the environment and object manipulation. Stereognostic sense is a combination of:

- The ability to move
- Somatosensory receptors informing the CNS of variations
- The ability to recognize variations (for manipulatory purposes)
- Joint position sense (the net information from joints, muscles, tendons, and skin)
- Perception

Stereognostic sense is based on somatosensory information, movement, the ability to recognize variations and perception.

There are several types of specialized receptor within the skin and epidermis, both free and encapsulated nerve endings that send different types of information to the CNS. Some of these receptors adapt quickly, others more slowly; some have a low threshold for firing, and others a high threshold. *Fast-adapting* means that the receptors quickly get used to the stimulus and stop firing even if the stimulus continues. This type of receptor informs the CNS of change, the start and finish of impulse firing, i.e., variations, and reacts to touch, pressure, and stretch. Other types of receptor are *slow-adapting* and continue to send information as long as stimuli are present. In this way, the CNS is updated about the

state of the body at all times. Examples include information from muscle spindles and tendon organs.

Nociceptors *sensitize*, i.e., their sensitivity increases with continuous stimulation. A quietly dripping tap becomes an unbearable noise after some time. If water drips on the head, the individual water droplets will feel like the blow of a hammer after a relatively short time. Some people react to wool directly on the skin with irritation or a rash. Discomfort or pain may be amplified over time until it occupies all thought.

The adaptive ability of the CNS allows us to wear clothes without our brain becoming over-bombarded with stimuli from all the specific receptors. Adaptation protects the CNS and modulates incoming information.

Some different receptor types are listed below (from Kandel et al. 2000, Brodal 2001):

- Free nerve endings—high-threshold mechanoreceptors (nociceptors)
- Meissner corpuscles—fast-adapting, low-threshold (light touch, e.g., fly crawling on the arm)
- Pacinian corpuscle—fast-adapting (vibration)
- Ruffini terminals—slow-adapting (friction, stretch of skin)
- Merkel disks—slow-adapting (continuous pressure, especially in distal areas of the extremities, lips, and the external genital organs)

Somatosensory information in the cortex is somatotopically arranged, representing different areas of the body (see Fig. 1.6). For somatotopic mapping of cortical function, it suffices to know which neurons respond to a stimulus at a particular site on the body. Each part of the body is represented in the brain in proportion to its relative importance to sensory perception. The map represents the sensory innervation density of the skin rather than its surface area. A *sensory unit* is a sensory nerve cell and all its branches, in the body and the CNS, and a *receptive field* is defined as the area of the body from which the sensory unit receives stimulation. Two-point discrimination is the result of two receptive fields being stimulated at the same time; each point must stimulate a sensory unit to enable the CNS to feel them both. The skin of the fingertips is the most densely innervated area of the body with approximately 300 mechanoreceptive nerve endings/cm²; on the proximal phalanges

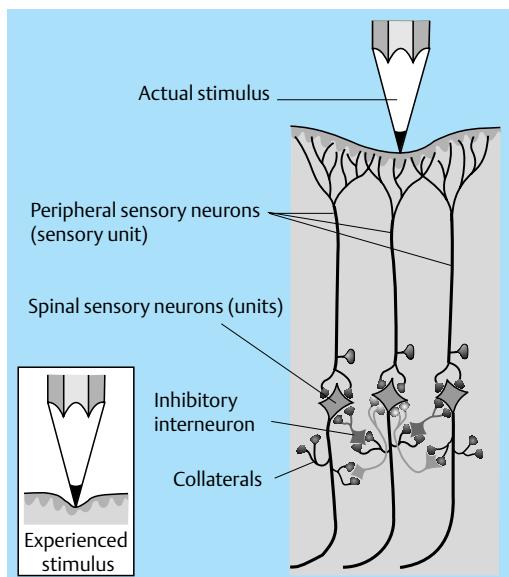


Fig. 1.9 Lateral Inhibition increases the contrast of sensory impulses.

there are $120/\text{cm}^2$, and in the palm of the hand $50/\text{cm}^2$. This means that two-point discrimination varies between the fingers and hand: it is approximately 2 mm on the finger tips, 10 mm in the palm of the hand, and 40 mm on the arm (Kandel et al. 2000).

Afferent information is modulated and localized through *lateral inhibition* (Rothwell 1994, Kandel et al. 2000, Brodal 2001), especially in relation to the fast-adapting receptors. When skin is stimulated, information is sent to the CNS. Where the stimulus is at its strongest, for instance at the edges of a book being held in the hand, the stimulated receptor organs transmit impulses to the spinal cord, where they give off collaterals that synapse with inhibitory interneurons in the dorsal horn (Fig. 1.9). The inhibitory interneurons inhibit sensory impulses from other sensory neurons arising from the periphery of the receptive field of the stimulated area. Therefore the sensory units in the center of the stimulated area are stimulated the most.

Thus the CNS is able to localize touch and is informed of changes (start/stop), edges, texture, and shapes, i.e., variations. If a person places his hand on another person's shoulder, he is informed of the shape and temperature of the area. If the hand is not moved, the receptors

quickly adapt and stop sending impulses. So without moving the hand, the size, shape, texture, and temperature of an object cannot be evaluated. Movement is necessary to activate receptors and obtain information through variations, and therefore for sensation. Motor activity and sensation are closely linked, and motor activity is a tool of sensation. Brodal (2001) states that most movements need continuous information from specific receptors within muscles, tendons, joints, and skin to know if the movement is progressing as planned.

The ability of the neuromuscular system to regulate tension and length (stiffness control) combined with information from joint and skin receptors provides the CNS with precise information about the position of joints and therefore of the position of body segments relative to each other. If the joint mechanoreceptors are anesthetized, the brain still receives precise information about joint position through muscle activity—muscle spindles and tendon organs—and skin receptors (Brodal 2001). The Golgi tendon organ is stimulated more through active contraction of muscle than through passive movement.

Brodal (2001) states that *joint position sense* is the conscious awareness of joint position, of movements of joints, and of the direction and speed of movement (without visual information). Joint position sense is the perceived result of sensory stimuli from proprioceptors and skin.

Muscle activity is important for joint position sense.

The brain stem contains many specialized neuronal groups (nuclei) that react to new sensory stimuli and increase the response of the motoneurons to specific stimuli. These nuclei receive information from the spinal cord, i.e., from peripheral receptors. Through their connection to the cortex, the hypothalamus, limbic structures, and hippocampus and other areas, they cause increased activation, arousal, and awareness to the stimuli. Shumway-Cook and Woollacott (2006) state that "Perception is essential to action, just as action is essential to perception." Human beings need to be able to receive and integrate information from stimuli, and recognize, compare, and perceive stimuli to interpret what the information means.

There is a close connection between somatosensory information, motor activity, and perception. All of these are important for stereognostic sense.

Variation in somatosensory information increases awareness of the stimulated body part.

Clinical Relevance

Somatosensory perception may change over time. The somatotopic maps are not fixed but can be altered by experience. The proportion of neurons in the cortex devoted to a specific area of the body will alter with use (Kandel et al. 2000). "If you don't use it, you lose it" (Kidd et al. 1992). Studies on stroke patients have shown that sensory and motor stimulation significantly improves both somatosensory information processing and motor control (Sunderland et al. 1992, Yekuitiel and Guttman 1993). Wade and Jones (1997) state that being active in relation to the environment improves perception.

The term *learned nonuse* refers to conditions in which reduced motor control leads to inactivity, e.g., reduced control of the affected arm in hemiplegia may lead to compensatory strategies whereby a patient uses his "good" arm and not the affected one. The affected arm is therefore not used, not stimulated and is inactivated. This predisposes to secondary muscular and soft tissue changes (Ada and Canning 1990), which may be important if the patient has reduced awareness or neglects the affected side.

If the patient is not able to move, he receives little or no information from the receptors within skin, muscle, and joints; the ability to recognize changes in joint position may be reduced as a result. Through formal testing of joint position sense, only the patient's *perception* and *cognitive awareness* are tested and not his possible potential for integration of joint position information through movement. Information from receptors is transmitted from the receptor areas in the body through to the spinal cord, modulated and passed on for further processing in the brain stem, the cerebellum, and the thalamus. Decreased sensory awareness will, for most patients with CNS lesions, be linked either to dysfunction in perceptual processing or to lesions in the ascending pathways within the brain itself, for in-

stance at the level of the internal capsule. Therefore, somatosensory information will be integrated at many levels, even if the patient is unable to feel and perceive it. Only through observation of the patient in functional activity will the clinician be able to get a true picture of his ability to receive and integrate sensory information.

Some patients with neurologic deficits have fixed stereotypical patterns of movement or may be unable to move a limb. Reduced movement and mobility reduce the stimulation and firing of receptors, and the CNS receives less information about variations in sensory information due to movement and the need for segmental adjustments. A consequence may be that the patient becomes more dependent on visual and vestibular information to maintain balance. In some patients the head and neck assume an asymmetric position due to imbalance between muscle activity and stability. Visual input may be distorted, and the firing of vestibular organs will be affected. This may cause further balance problems. Educating the patient about the position and control of head and neck posture is an important short-term goal in the rehabilitation of postural control. Other patients seem to visually override proprioceptive and vestibular information and the patient loses or reduces his ability to "listen" to his own body. The patient's response to the environment and to displacements becomes more cognitive and less automatic. As a result, movement strategies change and tempo will be reduced.

Interesting and varied sensory information through movement and stimulation may motivate and focus patients toward the stimulated body part or limb. It becomes important for the therapist to find which stimulus creates arousal and motivation of the patient's CNS.

The Brain and Spinal Cord

The Spinal Cord

The spinal cord receives information both from higher centers and from the periphery. It has a huge receptor area and both receives and modulates information from the whole body except the head before the information is transmitted to other systems or translated into muscle activity.

It has a gate-control function whereby information sent to the spinal cord is adapted to suit the needs of the organism and to protect the brain from overstimulation (Davidoff 1990, Kandel et al. 2000). Because of its large receptor area and gate-control function, the spinal cord influences activity in the higher centers.

■ Central Pattern Generators

"The existence of networks of nerve cells producing specific, rhythmic movements, without conscious effort and without the aid of peripheral, afferent feedback, is indisputable in a large number of vertebrates" (MacKay-Lyons 2002). Neural networks in the spinal cord, called **central pattern generators** (CPGs) are capable of producing rhythmical movements (Dietz 1992, Brodal 2001, MacKay-Lyons 2002). CPGs provide automatic, changing activity coordinating the two halves of the body and have been mostly studied in vertebrates. CPGs have been demonstrated for vital

functions such as breathing, chewing, and swallowing and are located in the brain stem, whereas those for locomotive functions are contained in the spinal cord. The probability of their existence in humans is high. Evidence is emerging from research on patients with spinal cord lesions and others. A baby's early stepping-reactions of rhythmical spinal activity may also be an expression of pattern generation.

In relation to reaching and grasping, Paillard (1990) states "Elaborate motor patterns are produced at the brainstem level by pre-wired circuitry. These circuits are triggered to operate at the required time and are automatically adjusted to postural requirements and environmental constraints through the servo-assistance of internal and external feedback loops at the spinal and midbrain level." In humans, findings are consistent with separate CPGs controlling each limb (Dietz et al. 1994 in MacKay-Lyons 2002). These are linked together through a complex interneuronal network. The dorsal root fibers from peripheral receptors terminate on interneurons that branch within the spinal cord. Interneurons that spread their branches over several segments are called *propriospinal fibers* and are found throughout the white matter of the spinal cord (Brodal 2001). The impulses from one dorsal root-fiber may therefore spread over several segments both up and down. Long propriospinal fibers (from the cervical to the lumbar areas) are necessary for coordination between the right and left side of the body during walking, and rhythmical interchange of activity between the two sides is caused by cells with pacemaker properties. Propriospinal fibers also have a role in the transmission of descending motor commands (Rothwell 1994).

The CPGs are, in their original form, independent of somatosensory information. Studies in humans and monkeys have shown that voluntary motor tasks, such as reaching and grasping, and more rhythmical movements, including swimming and walking, can be performed following deafferentation (Knapp et al. 1963, Rothwell et al. 1982 and Marsden et al. 1984 in MacKay-Lyons 2002). However, the interplay between central and sensory influences is critical in the production of adaptive behavior, i.e., for function. Somatosensory feedback is an integral part of the overall motor control system and is essential in modifying CPG-generated motor programs to fa-

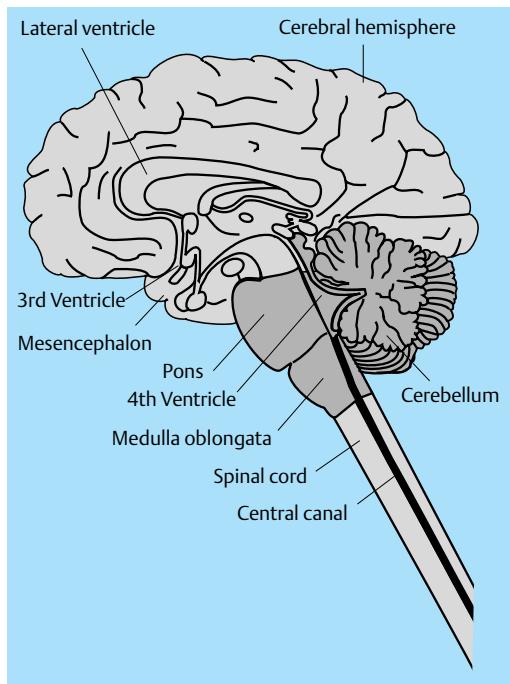


Fig. 1.10 A longitudinal view of the brain and spinal cord. The spinal cord and brain stem contain specialized groups of cells—neuronal pools called central pattern generators.

cilitate constant adaptations to the environment (MacKay-Lyons 2002). The CPGs use afferent information from several sources in the visual, vestibular, and somatosensory systems. Afferent information is essential for adaptation of CPG activity for locomotion to make walking appropriate to the environment and to the reason for walking, i.e., to the external and internal environment (Pearson 1993):

- Timing function: sensory feedback provides information ensuring that motor activity is adapted to the biomechanical state of the moving body parts for position, direction, and strength
- Facilitation of phase-changes in rhythmical movements to ensure that the next phase is not initiated before the biomechanical state of the body is ready

Dietz and DuySENS (2000) state that afferent information is necessary to strengthen CPG activity of antigravity muscles. Information about unloading, heel-strike, and weight transference are critical for the control of stepping (Maki and McIlroy 1997). Kavounoudias et al. (1998) researched the role of cutaneous receptors in the soles of the feet for balance. They anesthetized the feet and found that subjects were unable to balance on one leg after the loss of foot sensitivity. A specific distribution of mechanoreceptors in the feet codes the spatial origin, the amplitude and the rate of changes in the amplitude of pressure exerted on the skin. Therefore the CNS is continuously receiving information regarding the spatial and sequential distribution of pressure from the soles of the feet. There is co-processing of multiple tactile messages within the foot and between the feet.

The higher centers involved in the initiation, modulation, and control of locomotion are:

- The midbrain locomotor areas
- The vestibular system
- The reticular formation
- The cerebellum
- The cortex

From lampreys to primates, nuclei in the mesencephalon, referred to as the *mesencephalic locomotor region* (MLR) or the *mid-brain locomotor areas*, initiate locomotion through activation of the lower brain stem reticulospinal neurons (MacKay-Lyons 2002). Three different zonal areas have been identified, which all have a role in the initiation of locomotion for different rea-

sions: the lateral hypothalamus initiates gait in relation to hunger, thirst, or the need for the bathroom, the zona incerta for tourist-type walking, i.e., visually directed walking, and the periventricular zone for anger and fear.

The reticular formation and vestibular system in the brain stem are both involved in activating the antigravity musculature. Further modification is through the cerebellum, which is thought to coordinate the activity of the CPGs for the right and the left side of the body. The cerebellum is active in motor learning and error correction. The cortex exerts little influence on simple unobstructed walking, but has a role in visual scanning, perception, navigation, and in modifying the activity of the CPGs to make it appropriate to the moment: as walking becomes more complex, the cortex becomes more active.

When locomotion becomes complex, especially where footfall is narrowly specified and the need for visual input increased, the cortex fires more rhythmically to guide the placing of the foot. The CPGs cannot see the ground.

Walking on a flat, even surface is probably controlled by CPG in the spinal cord and brain stem and coordinated by the cerebellum, i.e., it is mainly an automatic activity.

Clinical Relevance

Pattern generation may have an important role in the early activation of postural activity and coordination in the acute/subacute stage in patients who have a CNS lesion. Facilitation of stepping in a simple noncomplex environment does not require cognitive problem solving by the patient:

- A pattern-generated step is a result of selective displacement and is different in its motor activity from a reactive step resulting from over-displacement.
- Pattern generation may be facilitated automatically, even in patients with severe motor, sensory, or perceptual dysfunctions.
- Pattern generation may enhance motor activity in the body as a whole by facilitating the interplay and coordination between the different segments and the two halves of the body, thereby promoting balance control.

- Early activation in the standing position and through stepping activates the CNS and neuromuscular system and motivates the patient.
- There are CPGs for each side of the body. A good stance phase resulting from heel-strike facilitates the swinging phase on the same side (release of kinetic energy).
- A good stance phase provides stability to the postural system, and therefore also frees the opposite leg for swing.
- Pattern generation depends on appropriate afferent information to adapt the activity to the environment.
- Tempo must be at an appropriate level for the individual to facilitate phase changes. Individuals seem to have different inherent speeds.
- Early facilitation of stepping facilitates the patient's awareness of the environment, and may improve perception.

■ Descending Systems

Descending pathways from the brain stem and higher centers have executive functions and a role in modifying and modulating incoming information. They regulate the transmission of sensory information from the spinal cord to the brain, and affect the excitability in spinal interneurons at the same time as being responsible for motor activity (Rothwell 1994). They are dependent on somatosensory information to be up to date on the state of the body at all times. Descending pathways may be grossly divided into the *lateral system* and the *medial system* (Kandel et al. 2000).

The *medial system* provides the basic postural control, based on which the cortical motor areas can organize more complex and differentiated movement. It includes the *vestibulospinal tracts (medial and lateral)*, the *reticulospinal tracts (medial and lateral)* and the *tectospinal tracts*. These pathways descend ipsilaterally in the anterior (ventral) part of the spinal cord and terminate predominantly on interneurons and long propriospinal neurons in the spinal cord. Thus they influence motoneurons that innervate proximal and axial musculature. They also terminate directly on some motoneurons that innervate axial muscles.

■ The Brain Stem

The Vestibular System and Balance

The vestibular system consists of several (four on each side) nuclei at the base of the brain stem (Fig. 1.11). These receive information from:

- The vestibular organs in the inner ear, signaling the position of the head and changes in movement, direction, and speed
- The spinal cord (proprioceptive information), reticular formation, the cerebellum and some nuclei in the mesencephalon (mid-brain, part of the brain stem), but not from the cortex directly
- The eyes

The vestibular system sends information to:

- The spinal cord, especially to motoneurons
- Brain stem nuclei for external eye muscles
- The cerebellum

The main function of the vestibular system is the control of balance in close cooperation with the proprioceptive and visual systems. The lateral and medial vestibular nuclei control posture. The *lateral vestibular nucleus* (Deiter's nucleus) is concerned with control of the position of the head and body in relation to gravity. The lateral vestibulospinal tract extends the total length of the spinal cord, and affects α - and γ -motoneurons monosynaptically, and antigravity musculature (Markham 1987, Brodal 2001). Due to its monosynaptic connection, it reacts quickly to restore balance. The activity of the vestibular system is greatest in standing and walking where the demands on postural control are greatest. Any displacement of position or posture, weight transference, or movements of the arm displaces the center of gravity in relation to the feet and the base of support. Irrespective of how small the displacements are (breathing causes small, almost invisible alterations in the intersegmental alignment of the trunk, which are perceived as small perturbations to the center of gravity), muscle tone and activity will need to adapt to maintain equilibrium.

The interplay between the body and the environment is crucial in standing, balancing, and walking. Somatosensory and vestibular information contribute to the perception of the body in space (Wade and Jones 1997).

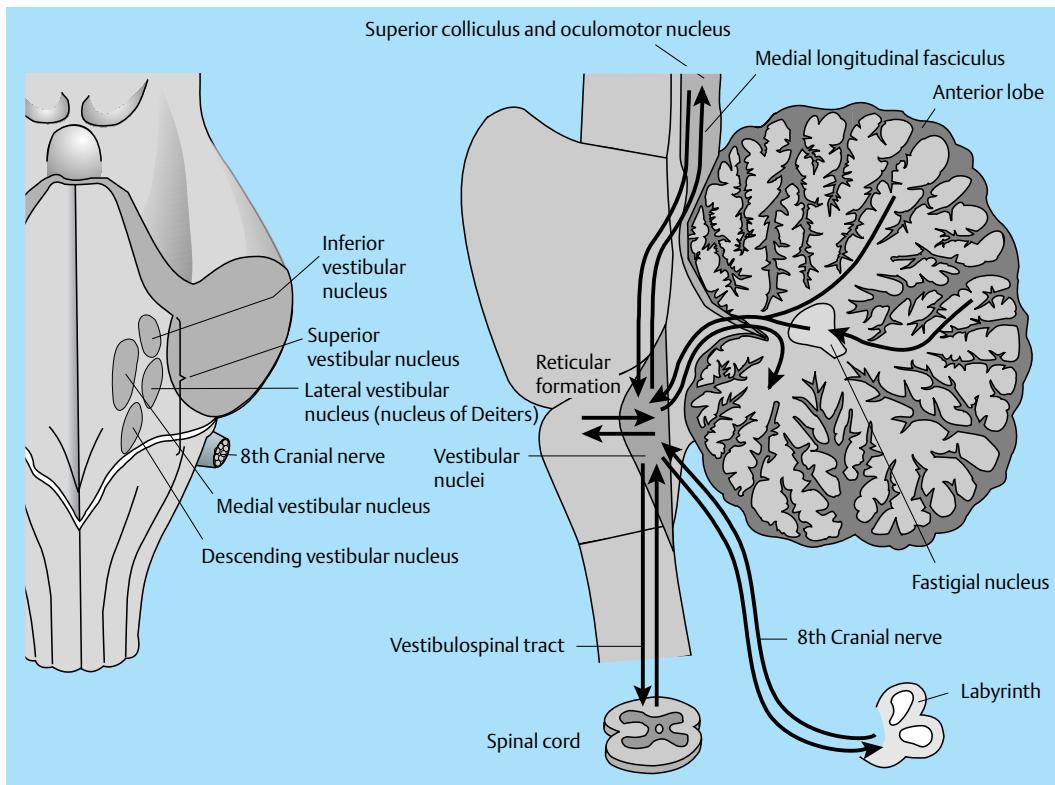


Fig. 1.11 The vestibular nuclei in the brain stem. The figure shows the most important afferent and efferent pathways.

The activity of the vestibular system is greatest when postural control is required.

The lateral vestibulospinal system supplies axial muscles (the deep postural muscles of the trunk and neck) and proximal extremity muscles (Markham 1987). Nashner (1982) and Dietz (1992) state that the vestibular system affects postural muscles directly and that its function is to maintain balance when the sensory environment is changing or moving, as during locomotion or when the support surface varies or moves (on an escalator or on a train or bus). The lateral vestibulospinal tract sends collaterals to both cervical and lumbar regions of the spinal cord. The same message is therefore conveyed to both levels to allow coordination of the body and the extremities with the head and neck muscula-

ture. Vestibular system function is geared toward the here-and-now situation.

The lateral vestibulospinal tract affects the same side of the body, i.e., it is ipsilateral to the trunk and extremities. It promotes extensor and inhibits flexor motoneuron activity ipsilaterally and facilitates flexor activity on the contralateral (opposite side) at the same moment in time (Dietz 1992). This view is supported by Kidd and colleagues (1992), who state that the lateral vestibulospinal tract is active during extension in standing and walking.

The *medial vestibular nucleus* innervates motoneurons in the cervical and the upper thoracic segments through the medial vestibulospinal tract. This pathway is smaller than the lateral and synapses with motoneurons ipsilaterally. Its function is to stabilize the head in relation to the body. The head is relatively free in standing

and walking to allow visual scanning of the surroundings.

Head control and postural control affect each other reciprocally.

The *vestibulo-ocular reflex* (VOR) compensates for head movements (Kandel et al. 2000). This reflex keeps the eyes still while the head moves, which allows for stabilization of images on the retina during movement. As the head rotates, the eyes rotate in the opposite direction. The vestibular apparatus signals how fast the head rotates. This information is used by the oculomotor system to stabilize the eyes to steady the visual images on the retina. Visual processing is much slower and less efficient than vestibular processing for image stabilization. This reflex needs to be adapted through motor learning as body proportions change as we grow.

Clinical Relevance

There are important functional consequences of the above discussion. Through its ipsilateral innervation of extensor musculature it may seem as if the activity in the vestibular nuclei is best facilitated when standing on one leg. Dietz (1992) discusses this in relation to a "true" standing posture or position. Clinically, it is perceived that the alignment of the body needs to be optimized to facilitate balance. Bussel et al. (1996) refer to studies on paraplegic patients that seem to indicate that the flexor reflex may interfere with CPG activity during attempts to step or walk. Combining these different authors' findings with clinical experience, it may seem as if balance on one leg in standing is a prerequisite for free swing of the opposite leg. At the same time, if the swing is initiated actively too early in swing, it may obstruct stability of the standing leg.

The ability to dynamically balance on one leg seems to be a prerequisite for a free swing.

Active swing that is initiated too early may interfere with the stability of the standing leg.

A stroke results in motor deficits on the opposite side of the lesion if the lesion is above the pyramids in the brain stem. The vestibular system is not under direct cortical control, and is therefore not affected by stroke directly (lesions in the vestibular nuclei are rare). Bringing the patient into a standing position to interact with gravity (placing or facilitating depending on the patient's level of motor activity) could stimulate the vestibular system to facilitate activity on the patient's affected side. Weight transference to the affected side through optimized/normalized alignment would best facilitate activation of the vestibular system. In locomotion, the time spent in stance phase is short, only 40% of the gait cycle (Smidt 1990, Whittle 1996). Standing on one leg allows freedom of movement of the other leg.

The Reticular Formation

The reticular formation (Fig. 1.12) is situated in the brain stem and extends from the lower part of the medulla oblongata to the upper part of the mesencephalon (Brodal 1995). It has many functions: it excites and inhibits activity, enhances flexion and extension, cooperates with

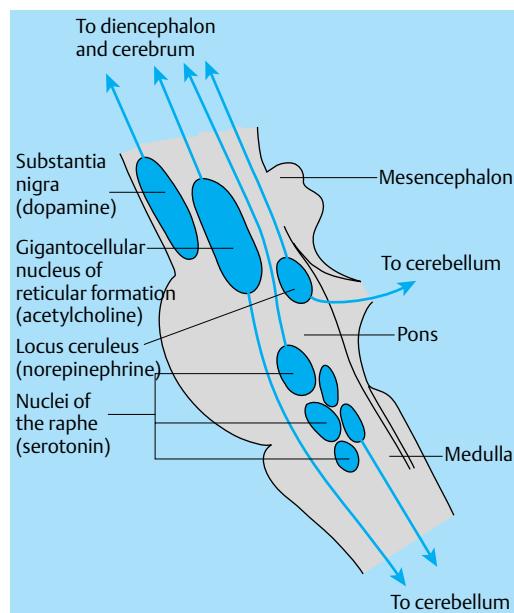


Fig. 1.12 The reticular formation in the brain stem has some important specialized groups of cells.

most systems within the CNS, and has a role in both proximal and distal movement. It activates both higher and lower levels and functions as an integration system. The reticular formation is highly organized and differentiated, consisting of distinct populations of neurons with specific functions (Kandel et al. 2000).

Some neurons from the reticular formation project to meet motoneurons of the spinal cord and influence functions such as cardiovascular and respiratory control. Generally, the reticular formation is broadly divided into a lateral and a medial part. The *medial part* is located in the pons and medulla and is mainly efferent—it sends long projections to the thalamus and cortex, cerebellum, and the spinal cord. The *lateral part* is smaller and receives many of the afferent connections to the reticular formation. Many secondary ascending fibers send off collaterals to the reticular formation, therefore the reticular formation receives information from:

- Most receptors, also for pain and hearing
- Cerebellum
- Cortex
- Basal ganglia
- Vestibular impulses
- Limbic structures

Some of the main inputs to the reticular formation are from the sensorimotor areas of the cerebral cortex. This input is bilateral, and therefore activates the reticular formation bilaterally. Information from the cortex through the *corticoreticular tract* is important for the control of more automatic movements. It affects motoneurons in the spinal cord both directly (monosynaptically) and indirectly via interneurons. The corticoreticular tract ends in the same regions of the reticular formation as the tract sending fibers to the spinal cord. This represents another pathway from the motor cortex to the spinal cord.

Reticulospinal fibers synapse indirectly via interneurons or directly on motoneurons (both α - and γ -motoneurons) and send collaterals to many levels within the spinal cord. In this way, reticulospinal pathways may affect many parts of the body at the same time, both inhibitory and excitatory. The sensitivity of the muscle spindle may be regulated from the reticular formation and muscle tone from the cortex, cerebellum, and other higher centers via the reticular formation. Psychological processes such as motivation and happiness may affect the reticular

formation and seem to increase a person's initiative and thereby muscle tone. Depression seems to have the opposite effect and downregulates tone.

Posture, locomotion, and muscle tone is regulated through the *medullary* and *pontine reticulospinal tracts* of the reticular formation, which run separately in the spinal cord. These tracts are both crossed and uncrossed, and give off several collaterals on many levels within the spinal cord. They therefore influence muscles in different parts of the body simultaneously (Brodal 2001). The pontine (or medial) reticulospinal tract arises from the upper pontine reticular formation. It facilitates spinal motoneurons that innervate axial musculature and extensors of the legs to maintain postural support (Kandel et al. 2000). According to Brodal (2001), this tract is probably more oriented toward axial musculature due to its location in the spinal cord (more laterally in the anterior horn). Stimulation of the midbrain locomotor region produces stepping movements or other stereotyped, patterned movements (Kandel et al. 2000). The medullary (or lateral) reticulospinal tract descends to the spinal cord and affects extremity muscles.

The reticular formation activates the body generally and specifically. The function of the reticular formation in motor activity is to maintain an upright position and orientate the body and head toward something in the environment, especially new stimuli, and to regulate some coarser movements of the extremities (reaching, making a fist). The medial part is both crossed and uncrossed and thereby supplies motoneurons on both sides, but mostly the same side, of the body.

The vestibulospinal and the ipsilateral reticulospinal pathways work together through interneurons and long propriospinal neurons to produce selective activation of many muscles at the same time, including the neck and axial muscles. The rubrospinal and the crossed reticulospinal pathways innervate motoneurons directly or indirectly through interneurons or short propriospinal neurons to distal musculature (Rothwell 1994). They probably also have a postural role with regard to the extremities, for instance providing the postural background for individual finger movements. Generally speaking, man can dig a ditch with the reticulospinal system, but not play the piano.

The bilateral and unilateral innervations to the musculature enable the body to stabilize contralaterally for arm reach or swing phase. The pontine reticular formation stabilizes the contralateral half of the body for the ipsilateral medullary component to initiate reaching an arm forward to grasp an object.

Stability and balance are prerequisites for movement.

The reticular formation in the pons contains the nucleus ceruleus, which projects to every major region of the brain and spinal cord, and maintains alertness to novel stimuli. It therefore has an important role in arousal as well as sensory perception and muscle tone. The raphe nuclei are located along the midline of the brain stem and project mainly to the forebrain where they help regulate sleep-wakefulness, affective behavior, temperature, and other functions. They also project to the spinal cord where they participate in the regulation of tone in motor systems and pain perception.

The midbrain contains neurons that are critical for the state of cortical arousal. These neurons project to the cerebral cortex, where they enhance cortical responses to incoming sensory stimuli. The ascending fibers of the reticular formation form a network—the *ascending reticular activating system (ARAS)*. This system influences wakefulness and the overall degree of arousal and consciousness.

There is a close connection between the regions that influence the spinal cord and the thalamus. Activity in the reticular formation is necessary for conscious perception and the specific reaction to sensory information. The reticular formation receives input from the cortex via the *corticoreticular pathways*. The *corticoreticular fibers* transmit information to both the excitatory and inhibitory areas of the reticular formation and synapse in these areas before information is passed on to the spinal cord, i.e., forming the cortico-reticulospinal system (Fig. 1.13). The cortico-reticulospinal system is also called the extra-pyramidal system.

Standing promotes awareness and arousal.

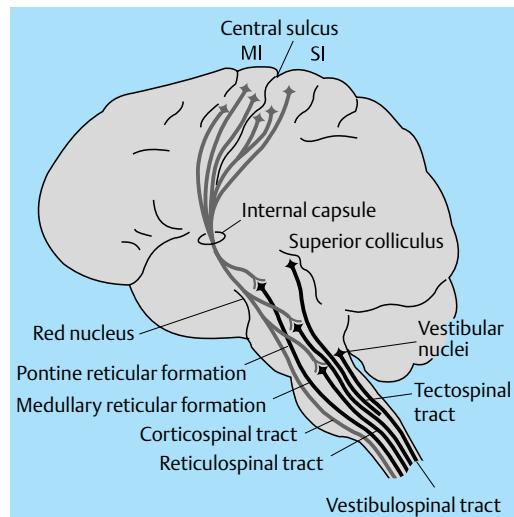


Fig. 1.13 Descending pathways to the spinal cord: the corticospinal tract and some pathways from the brain stem. Many of the pathways from the brain stem receive fibers from the cortex.

If the fibers innervating the excitatory area of the reticular formation are lesioned, there may be a loss of excitation to the spinal cord and the patient may experience hypotonia. If, however, the fibers synapsing in the inhibitory part are lesioned, there will be a loss of inhibitory influences to the spinal cord. The patient may develop a problem of severe hypertonia.

Clinical Relevance

Both the reticular and the vestibular systems innervate body musculature on the same and the opposite side of the body (ipsi- and contralaterally). A lesion affecting motor pathways on one side of the brain may result in reduced motor control on both sides, but predominantly on the contralateral side, and at the same time as motor function may be spared bilaterally. Therefore the term *hemiplegia* is misleading in a functional context. Brain stem lesions may result in reduced balance and mobility problems as well as dysphagia and/or dysarthria because the nuclei of the cranial nerves are located in this region. Muscle tone may be increased or severely decreased depending on which part—the inhibitory or the excitatory part—of the reticular formation is lesioned.

The cortico-reticulospinal system has an important role in proximal stability and in the regulation of postural tone. Lesions to the cortico-reticulospinal system seems to lead to decreased postural control, loss of selectivity of postural control even though the visual, vestibular, and somatosensory systems are still intact and not affected directly.

The lateral pathways are the rubrospinal tract, the corticospinal tract, and the corticobulbar tract.

The Cerebral Cortex

The cerebral cortex contains probably as many as 10–20 billion neurons. The different areas have network contacts with each other and with other parts of the CNS. Many of these networks are overlapping (Brodal 2004), i.e., they share some of the same structures and functions. There are individual differences in the size of different parts of the cortex (Brodal 2001). Approximately two-thirds of the cells are pyramidal cells. These function as the projection neurons of the brain because they send their axons to other parts of the cortex and the CNS. There are also large numbers of interneurons that transmit and exchange information. Movement depends on information from many areas of the CNS, areas that receive, integrate, modulate, and transmit impulses, areas that assist in the planning of movement, and areas linked to motivation. Somatosensory integration is discussed above. The following sections will take a closer look at the role of the cortex in movement production.

The Pyramidal Tract (Corticospinal Tract)

The pyramidal tract is a name given collectively to all fibers leaving the cerebral cortex that descend through the internal capsule and innervate interneurons and motoneurons in the spinal cord without synaptic interruption along the way. Most of these fibers cross in the lower part of the medulla oblongata (Fig. 1.14). The pyramidal tract is also called the corticospinal tract (Brodal 2001). It comprises of approximately 1 million fibers. More than half arise in the primary motor cortex (MI), the supplementary motor cortex (SMA) and the premotor areas (PMA), the rest arise in areas behind the central sulcus: the somatosensory areas (SI and SII) and part of the

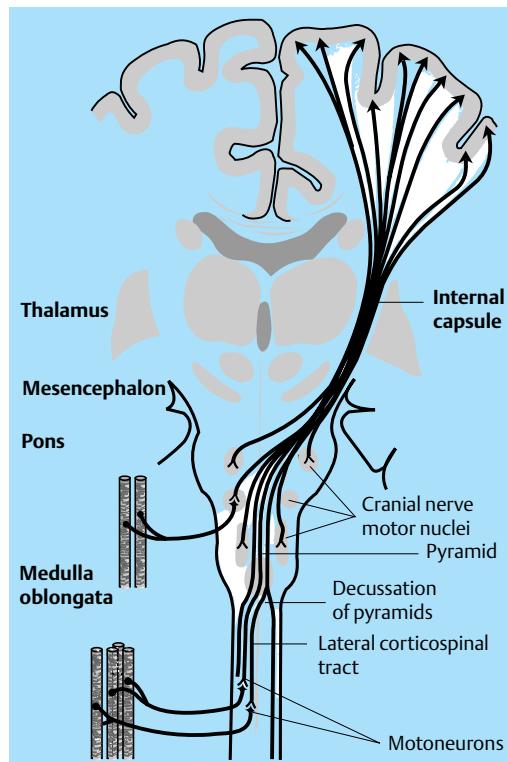


Fig. 1.14 The corticobulbar and corticospinal pathways.

posterior parietal cortex (Davidoff 1990, Brodal 2001). It is assumed that the pyramidal fibers arising in SI are as important for the signaling of impulses in sensory pathways as for the initiation of motor activity.

Fibers also descend to innervate cranial nerve nuclei, the red nucleus, nuclei in the pons and the reticular formation, sensory nuclei and others in the brain stem (Brodal 2001). This pathway is called the *corticobulbar tract*.

The axons from the primary motor cortex (MI) terminate in areas of the spinal cord where there are large numbers of interneurons (lamina VII–IX). The axons branch profusely and form synapses with other pyramidal cells within the cortex as well as excitatory and inhibitory interneurons in the spinal cord. Corticospinal axons supply most of the gray matter in the spinal cord, i.e., they synapse with most motoneurons, both α -motor to striped muscle fibers and γ -motor supplying the intrafusal fibers (the muscle spindles). When α - and γ -motoneurons are facilitated at

the same time, α - γ co-activation, the tension in the spindles changes so that they are sensitive to alterations in muscle length, speed, and tension across the whole range of muscle lengths. Irrespective of the muscle contraction, eccentric lengthening or concentric shortening, the CNS will be updated on the state of the neuromuscular system and able to change activation rapidly if needed.

The corticospinal system supplies mainly distal musculature, partly monosynaptically and fast, partly via interneurons, and has its most important role in the control and fractionation of movement. The cortex therefore is involved in voluntary movement (least automatic), i.e., hand and individual finger function, and the movement of the toes. Some fibers innervate proximal, axial, abdominal, and thoracic musculature. Muscles involved in facial expression, eating, speech, and movements of the mouth are supplied through the same system.

The corticospinal system innervates most motoneurons. Most movements may be voluntarily controlled to some degree. If needed, cognitive strategies may override more automatic movements.

The corticospinal system mainly supplies distal musculature. Distal motor control, i.e., dexterity of finger movements and movements of the toes, is an example of voluntary (least automatic) activity.

The fibers from the somatosensory areas of the cortex mostly terminate in the posterior horn of the spinal cord (laminae I–VI) where they synapse with interneurons receiving input from somatosensory receptors. Here, the corticospinal projections are involved in the regulation of afferent information from peripheral receptors to the spinal cord. This system influences the signal transmission from afferent somatosensory terminals in the muscle spindles, tendon organs, and certain skin receptors transmitting pressure and touch. Through presynaptic inhibition, the corticospinal system may modulate or even inhibit excitatory impulses transmitted from peripheral receptors that otherwise would influence the CNS. In this way, the corticospinal system func-

tions as a gate: it grades or filters incoming information, allows relevant and useful information to get through and inhibits disturbing or unnecessary information. The CNS is therefore able to decide and choose what information it needs in different situations.

The cortex is able to weight the relative importance of different sensory modalities via the corticospinal system.

The Cortico-rubrospinal System and the Red Nucleus

The red nucleus (Fig. 1.15) is found in the mesencephalon of the brain stem and consists of two parts: a small, caudal *magnocellular part* and a

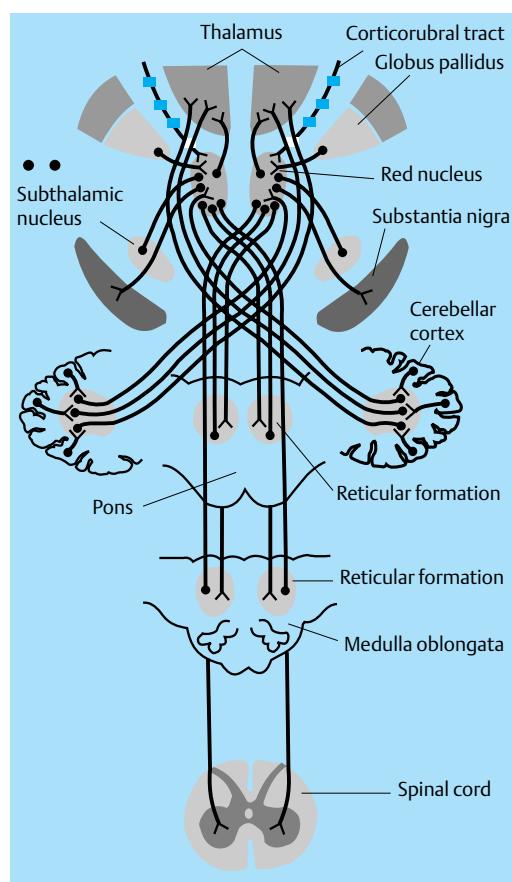


Fig. 1.15 The main connections of the red nucleus.

larger *parvocellular* part (Brodal 2001). It is smaller in humans than in animals, but its specific function in humans is not well known. It has been mostly studied in cats. In humans and apes, the magnocellular part is very small. The parvocellular part receives most of its inputs from the cerebellum, especially the dentate nucleus. Many fibers from the red nucleus terminate in the inferior olive, which in turn sends its fibers to the opposite side of the cerebellum. Efferents from the relevant areas of the cerebellum influence the motor cortex through the thalamus (Brodal 2001). It is therefore probable that the red nucleus has a role in motor learning and that it plays a more important part in influencing motor activity through integration and interplay with the cerebellum and cortex than influencing motoneurons directly.

The magnocellular part runs in the rubrospinal tract to the spinal cord, where it mingles with corticospinal fibers. The pathway crosses just below the red nucleus. The red nucleus receives collaterals from the corticospinal system and thereby it receives input from the cortex and a connection between the motor cortex, the red nucleus, and the spinal cord is established: the *cortico-rubrospinal pathway* (Davidoff 1990).

- The cortico-rubrospinal system acts on the spinal cord in a similar way to the corticospinal system. These two pathways may facilitate each other's function (Rothwell 1994, Davidoff 1990)
- Functionally, the rubrospinal system is facilitatory to flexion and inhibitory to extension during swing phase of locomotion (Kidd et al. 1992)
- Rothwell (1994) states that the rubrospinal system is facilitatory to flexion and inhibitory to extension in the cervical and lumbar areas of the spine and in the distal extremity musculature
- Brodal (1995) states that the red nucleus probably influences motor activity first and foremost through its interplay with the cerebellum more than directly affecting motoneurons in the spinal cord

Clinical Relevance

Damage to the corticospinal system mainly affects distal dexterity. Lesions may cause loss of, or disturbed, gating control for incoming information, and thereby hypersensitivity to peripher-

al stimuli (weight-bearing, stretch, touch, pressure) especially distally in the hands and feet. Hypersensitivity is often seen in patients with stroke, MS, or head injury (traumatic, severe stroke or hemorrhage or due to other conditions causing anoxia). Hypersensitivity causes a heightened response to stimuli. The patient may find the hand grasps objects but cannot let go, that the foot pushes off the floor or withdraws. The interplay between distal and proximal body segments is thereby interfered with and balance will be reduced. The cortex has an important role in deciding the relative importance of different peripheral stimuli through the corticospinal system. Clinical experience demonstrates that the patients may be able to use cognitive strategies to a certain degree—selective, focused attention—to control their own response to hypersensitivity (e.g., clonus), if the corticospinal system is not too severely damaged. A prerequisite seems to be that they experience more normalized movement through active and graded weightbearing in an optimized alignment at the same time. In the cat, the cortico-rubrospinal pathway is important for voluntary control of the distal body parts. The role of the cortico-rubrospinal system in humans is more uncertain because the magnocellular part is very small. Some authors state however, that it may have a similar function in humans.

The descending systems described in this chapter have both specialized and parallel functions. Lesions of the CNS rarely affect one system or pathway alone. What we see are the effects of the lesion on the patient as a whole. Fibers from the descending pathways from the cortex pass through the internal capsule with many others. A lesion in the internal capsule causes sensorimotor problems mostly on the opposite side of the body. Pathways from the cortex descend both ipsilaterally and contralaterally and will therefore result in some sensorimotor problems, also on the same side. Several studies have reported neurologic deficits on the so-called unaffected side in stroke patients (Thilmann et al. 1990, Marque et al. 1997) and even loss of strength (Patten et al. 2004). Therefore, the patient's total balance and sensorimotor abilities will be affected to some degree. Deviations in motor control and balance mainly in one half of the body always affect the patient's functional ability as a whole. Most stroke patients initially

have problems with standing on either leg. It is better to classify the two body halves as *least affected* and *most affected* than to describe the patient as having a *hemiplegia*.

Cortical lesions may result in more focal (local) problems, for instance affecting arm and/or hand movements or dexterity. Damage to the dominant hemisphere, most often the left, may result in speech and communication problems.

Functionally, the descending systems are broadly divided into lateral and medial systems. The main function of the medial pathway is maintenance of postural control, stability, and balance (most automatic). The main role of the lateral pathway is related to voluntary activity (least automatic).

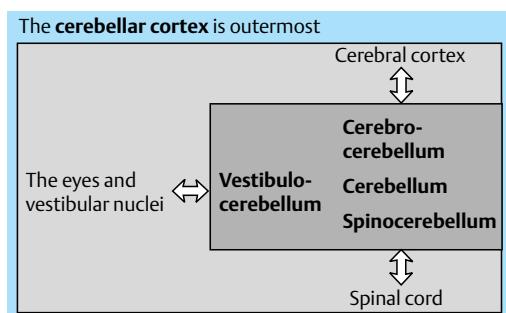


Fig. 1.16 The functional divisions of the cerebellum: the vestibulocerebellum, the cerebrocerebellum, and the spinocerebellum. These areas receive and pass on information to the vestibular nuclei, the cerebral cortex, and the spinal cord, respectively.

■ Perception and Cognition

Different types of cognitive and perceptual processes involve different regions and different combinations of regions within the brain. Lesions to either hemisphere and/or subcortical structures may have perceptual and cognitive consequences. More often, perceptual problems such as neglect, inattention, spatial problems, apraxias, etc. are associated with lesions affecting the nondominant hemisphere. Damage to the dominant hemisphere may cause decreased ability to remember, problem solve, and communicate. Perceptual and cognitive dysfunctions have a profound effect on motor behavior. The reader is advised to study these in more detail, e.g., Kandel (2000), Shumway-Cook and Woollacott (2006), Mulder (1991, 1996), and other relevant publications.

■ The Cerebellum

The cerebellum is a complex structure containing more neurons alone than the whole brain put together (Rothwell 1994, Brodal 2001). It has an important coordinating role in motor function based on the large amount of somatosensory information it receives—it has 40 times more input than output. The cerebellum consists of three functionally different parts that receive and pass information on to other parts of the CNS (Brodal 2001). The cerebellar cortex is outermost (Fig. 1.16).

The Vestibulocerebellum

The vestibulocerebellum (the flocculonodular lobe) is phylogenetically the oldest part of the cerebellum and works closely with the vestibular nuclei. Its main task is maintenance of balance, head control, and coordination of eye and head movements.

The Spinocerebellum

The spinocerebellum (Fig. 1.17) receives information mainly from the spinal cord. The *spinocerebellar tracts* (see The Somatosensory System) transmit impulses mainly from the spinal cord. They cross twice, and therefore influence the same side of the body, and transmit information from specific receptors in the skin, muscles, and joints. The spinocerebellum receives and transmits information about:

- The level of activity of the interneurons (especially inhibitory interneurons)
- Factors influencing the motoneurons at all times
- Movements resulting from the activity in the motoneurons
- The level of activity of descending pathways

The spinocerebellum is therefore kept up to date on the real-time activity in the body and compares intended and performed movements and corrects errors if needed. The spinocerebellum also receives input from nuclei in the mesencephalon through the *inferior olive*, thereby forming an indirect spinocerebellar tract, the *spino-olivo-cerebellar tract*.

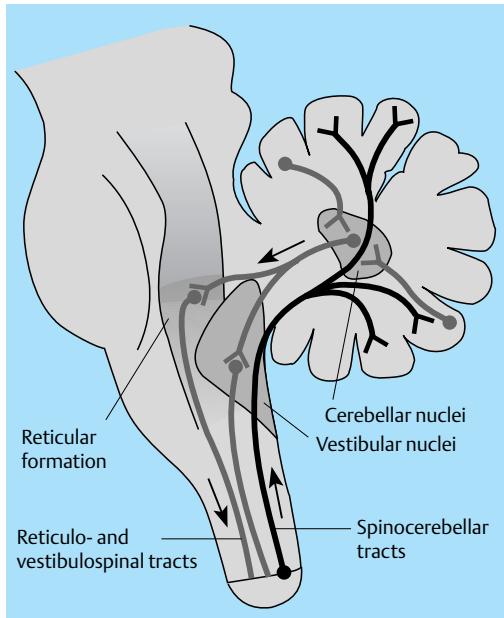


Fig. 1.17 The most important connections of the spinocerebellum.

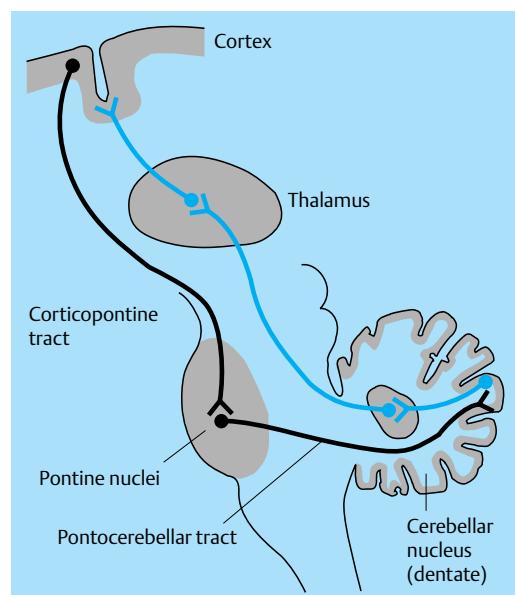


Fig. 1.18 The main connections of the cerebrocerebellum (pontocerebellum).

Function: The spinocerebellum has an essential role in relation to dynamic postural control because this area receives and integrates somatosensory information from the whole body. Information is passed on to the spinal cord via the red nucleus and the reticular formation (Horak and Diener 1994).

The Cerebrocerebellum

The cerebrocerebellum (pontocerebellum) (Fig. 1.18) has a contralateral influence on the motor systems. It receives most fibers from the cerebral cortex, a *corticopontine tract*. In the pons, the information from the cerebral cortex is integrated and modulated before crossing over and entering the cerebellum. The cerebrocerebellum receives information about which movements are planned and about which commands are sent from the cerebral cortex. Probably, there is a comparison between the intent to move and the actual movement at the same time as the cerebellum influences the activity of the motor cortex. Recent imaging data indicate that the cerebrocerebellum is intimately involved in planning and mental rehearsal of complex motor actions, and in the conscious assessment of movement errors. The cere-

bellum receives information from other peripheral and central influences (neck receptors, the red nucleus, the reticular formation, the level of activity in the interneurons from sources other than the spinocerebellar tracts).

The Cerebellar Nuclei

There are four nuclei situated deep in the cerebellum: the fastigial nucleus, the dentate nucleus, and the globose and emboliform nuclei (called the anterior and posterior interposed nuclei in animals). The *fastigial nucleus* has a role in posture, automatic movements, and locomotion. The *dentate nucleus* is involved in voluntary (least automatic) movement, as well as locomotion and initiation and termination of movement. The *globose* and *emboliform* nuclei compare the central motor command with the actual movement.

The Cerebellar Cortex

In the cerebellar cortex are four types of inhibitory neurons: the *stellate*, *basket*, *Purkinje* and *Golgi cells*, and one type of excitatory neuron, the *granule cell*. The outermost layer of the cerebellum, the cerebellar cortex, is organized into

three layers (Fig. 1.19). Afferent information to the cerebellum is via two main types of fibers, the *climbing* and the *mossy fibers*. These form excitatory synapses with cerebellar neurons, but terminate differently in the cerebellar cortex:

- The climbing fibers originate in the inferior olive, which conveys input from the spinal cord and the nuclei in the mesencephalon; this is somatosensory, visual, and cerebral cortical information (Kandel et al. 2000). The climbing fibers synapse with the Purkinje cells by twisting round the Purkinje cell and thereby forming many synapses with each cell. Each Purkinje cell receives input from only one climbing fiber, and each climbing fiber may contact between 1 and 10 Purkinje cells.
- The mossy fibers originate in nuclei of the spinal cord and brain stem, and carry information from the periphery as well as from the cerebral cortex to the vestibulo-, cerebro- and

spinocerebellum. They form excitatory synapses with the dendrites of the granule cells in the granular layer. The granule cells form the parallel fibers with their axons, which synapse with the dendrites of the Purkinje cells in the molecular layer.

The Purkinje cell is a specialized inhibitory neuron in the cerebellar cortex. It is the only cell that sends its axon out of the cerebellar cortex. There are about 30 million Purkinje cells in the human cerebellar cortex (Guyton 1976). Each Purkinje cell receives 150–200 synapses from *one* climbing fiber and approximately 80 000–200 000 synapses from many parallel fibers (granule cells) (Rothwell 1994). Enormous amounts of information therefore converge and are modulated and integrated before the Purkinje cell transmits the “result” of this communication to the deep cerebellar neurons. Information is modulated many times before it reaches its final target, the motoneurons of the spinal cord.

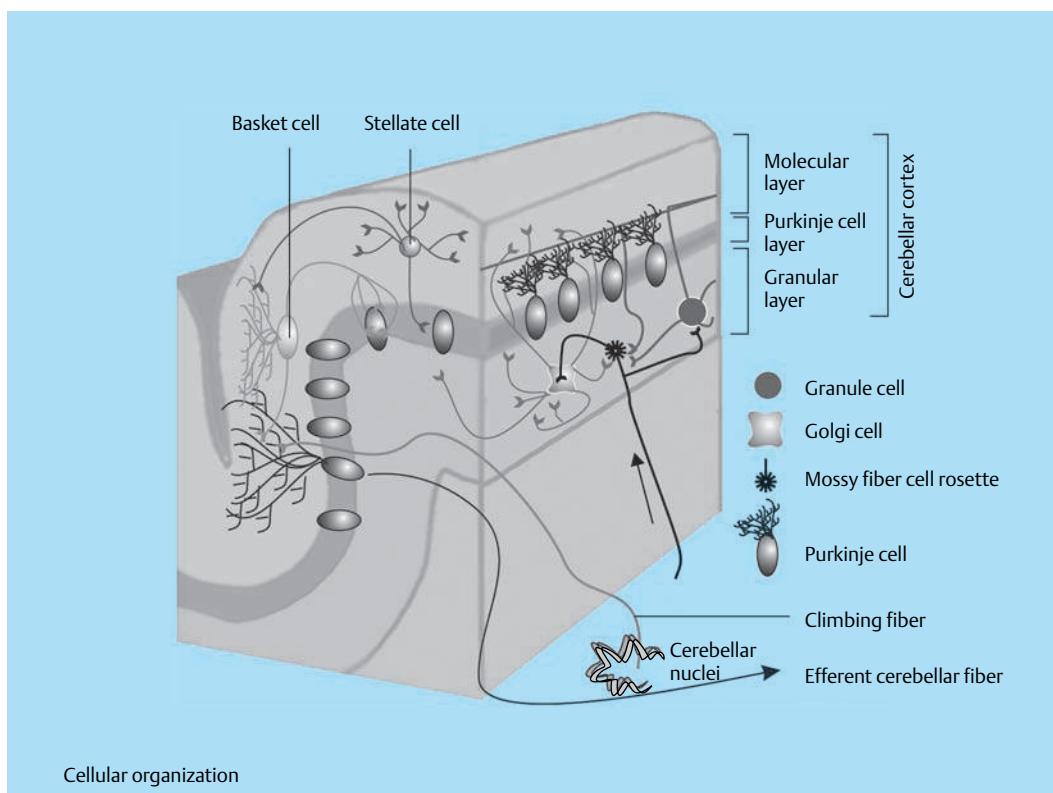


Fig. 1.19 The basic cerebellar circuit.

The Purkinje cells send their axons to the deep cerebellar nuclei as well as directly to the vestibular nuclei in the brain stem. The deep nuclei transmit information to the motor cortex and the reticular formation.

The Inferior Olive

The inferior olive is a nucleus in the medulla oblongata in the brain stem. It receives input from the spinal cord and the superior colliculus (which in turn receive information from the retina and visual cortex and somatosensory information from the primary sensory cortex SI). The largest part of the inferior olive is the *principal nucleus*, which sends its fibers to the cerebellar cortex via the climbing fibers, which cross over to the cerebellar nuclei and the opposite cerebellar hemisphere. The principal nucleus receives most of its inputs from nuclei in the mesencephalon (mostly the parvocellular part of the red nucleus) (Brodal 2001). The inferior olive has an important effect on the cerebellum and motor control. Damage to the inferior olive leads to severe disinhibition of the Purkinje cells. As a result, the inhibitory influences of the Purkinje cells on the cerebellar nuclei increase causing cessation of the cerebellum's influence on other parts of the CNS.

The climbing fibers ability to *error-signal* may have an important role in motor learning and certain forms of associative learning (conditioned blinking of the eye). The neurons in the inferior olive are rhythmically active (pacemaker function), and may therefore have a role in the ability of the cerebellum to rhythmically activate agonists and antagonists surrounding a joint in the correct sequence of recruitment at the right time.

The Role of the Cerebellum in Motor Control, Motor Learning, and Cognition

The climbing fibers from the inferior olive can induce selective *long-term depression* in the synaptic effect of the parallel fibers that are active concurrently. Long-term depression (LTD) is defined as reduced synaptic efficiency that may last minutes to hours (Brodal 2001). This long-term effect of the climbing fiber on the transmission of signals from the mossy fiber, the granule cell and parallel fiber through to the Purkinje cell may be important in motor learning (Kandel et al. 2000).

Clinical Hypothesis

The Purkinje cells of the cerebellum have a unique role in the control of movement and motor learning. Through their inhibitory activity they stop unwanted activity coming through. The Purkinje cells say "no" to unwanted movements and "not-no" to wanted movement (neurophysiologist Nigel Lawes, personal communication 1995). The hypothesis of how the cerebellum sculpts and shapes movement is especially interesting and relevant (Thach et al. 1992). The climbing fibers from the inferior olive transmit information from the spinal cord, nuclei in the mesencephalon, visual, and cerebral cortices. They seem to have a role in error-correction and motor learning. The mossy fibers are assumed to be involved in precise, graded movement. When people attempt to do something complicated, new, or strenuous, muscle activation radiates to muscles not primarily involved in the movement. For example, when a child first learns to use a pair of scissors, often his mouth opens and closes in rhythm with the movements of the hand. In adults, in the process of acquiring a new skill or performing something complex, more muscles are activated than is needed for the skill itself. When doing strenuous tasks such as sitting up from supine even the toes extend to help the activity although they are not primarily involved in the activity. The associated muscle activation is normal, and it is often termed associated movement. However, as the skill is perfected and becomes more automatic, these movements are gradually filtered away and they disappear. They will appear again during skill acquisition or the performance of heavy tasks.

The cerebellum seems to have a role in the refinement and sculpturing of movement by filtering unwanted muscle activation.

Brodal (2004) describes the storage of information necessary to perform certain activities, such as standing up from sitting, walking down stairs, or reaching out as *internal models* in the brain. This can be viewed together with the theory of *motor program storage* (Lalonde and Botez 1990) or *prototypes* (Mulder 1991) (Fig. 1.20). The (internal) knowledge of which components of movement are necessary for a certain activity

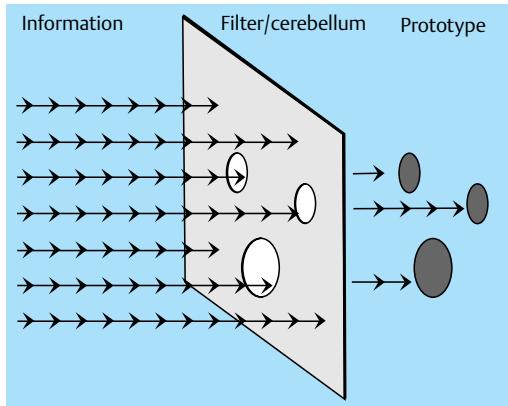


Fig. 1.20 The cerebellum as an information filter.

is called *prototypical representation* by Mulder (1991). Experience through position changes and variety of movement are prerequisites for the development of prototypes. Varied repetition is essential for all learning. When we stand up or sit down we do so to and from many different support bases: chairs, plinths, beds, stone walls, or fences; from different heights, textures, sizes, and postural alignments (postural sets), and with goals that involve different tempo, timing, and direction. Through varied repetition we learn which components are essential to evolve the wanted activity and are therefore essential for the function. Other, irrelevant, components are gradually filtered away for the sake of efficiency and ease of movement. There is evidence to support the hypothesis that heel contact is an essential component for various functions, for instance in sitting to standing—heel-down to stand up (Shepherd and Koh 1996, Khemlani et al. 1998, Chen et al. 1998)—and that heel-strike to load and heel-off to off-load is essential for stance and swing initiation in locomotion (Harkema et al. 1997, Trew and Everett 1998).

The prototype of movements and activities seem to be learned through varied repetition.

Large amounts of information converge on the Purkinje cells. The information is integrated and modulated before it is transferred to motor activity. This filter is an illustration of the continuous bombardment of information on the cerebellum.

Information deemed relevant to the movement is allowed through while irrelevant activity is stopped. What is allowed through represents a prototype that is adaptable to functional needs to make the movement appropriate to the here and now situation. The cerebellum seems to have a role in allowing relevant and relative activity through the filter by saying “not no.” In this way, the cerebellum also has a role in the control of selective movement.

Summary of the Many Possible Functions of the Cerebellum

Vestibulocerebellum:

- Balance during stance and gait, regulation of eye movements
- Fast acting in relation to equilibrium, postural mechanisms, and locomotion. The speed of signal transmission from the cerebellum is less than 100 ms. Therefore, this activity is classed as more automatic. Regarding a motor response to sensory information, more than 100 ms in the transmission of signals allows time for cognitive modulation

Spinocerebellum:

- Regulation of body and limb movements
- Feedforward control mechanisms in the regulation of movement
- Modulation of the descending motor systems in the brain stem and cerebral cortex, especially in relation to movements of the face, mouth and neck, and proximal parts of the limbs, and to postural control and balance during voluntary activity
- Accuracy of reaching through the timing of the components of movement and the control of direction and extent of movement
- Comparison between somatosensory information and motor commands
- Comparison between motor commands and actual movements through information from interneurons in the spinal cord. The cerebellum evaluates whether the planned movement gave the intended result
- Error correction
- Rhythm control—coordination of the CPGs in the spinal cord for locomotion

Cerebrocerebellum:

- Coordination of the timing of the various components of voluntary movements (e.g., prehension grip)

- Modulation of activity in the motor cortex improves accuracy and thereby shapes movement
- Motor learning and cognition:
 - new motor skills, thereby involved in selective movement
 - adaptation and modulation of movement by improving the performance of skills through repetition (trial and error)
 - acquisition and processing of sensory information for tasks requiring complex spatial and temporal evaluation, which are essential for programming complex motor actions and sequences of movement
 - simpler motor responses such as the vestibulo-ocular reflex (Brodal 2001)
 - adaptation of conditioned reflexes
 - mental rehearsal of movement and motor learning through the premotor-cerebellar loop
 - planning and programming of hand movements—eye-hand control
- Storage of basic prototypes for movements and activities

Clinical Relevance

Some patients with neurological deficits (e.g., stroke, MS, or head injury) seem to have a reduced ability to both initiate necessary activity and to correct the movement if it is not appropriate. These patients do not seem to recruit the prototype as a basis for varied activity. There may be a spread of activity to muscles that normally would not be primarily involved, which sometimes starts even before the wanted activity. These “extra” movements may not be filtered away as the activity is practiced, but sometimes increase and get stronger both in range of motion and force production, often involving a whole limb. These unwanted and inefficient movement patterns may be established—learned—if they are practiced. If the cerebellum learns these movements as part of an activity, it would seem possible that a new prototype is established, a coarser, less refined and less controlled form. The patient’s CNS might include them as a part of the prototype in day-to-day activity. This would lead to increased stress and effort, disturb the patient’s motor performance and—most important—negate the development of postural stability.

Lesions to the cerebellum may lead to hypotonia, *balance problems*, *ataxia* (meaning *loss of order*), *dysmetria* (meaning *abnormal measure*, characterized by impaired reaching, timing errors, and problems of direction and extent of movement), *terminal tremor* (irregular oscillation movements round a target), *visual problems*, and *incoordination*. These primary deficits often lead to secondary problems as the somatosensory, visual, or vestibular information is disturbed when the performance of movement is altered by pathology. The cerebellum receives misinformation and the primary deficits are enhanced.

Lesions to the vestibulocerebellum cause problems in the control of eye movements during rotation of the head (the vestibular-ocular reflex), and in the control of limb movements during stance and gait as well as severe balance problems. If the patient lies down, he can move his limbs accurately.

The Basal Ganglia

The basal ganglia consist of the caudate nucleus, the putamen and the globus pallidus (Fig. 1.21). Many authors also include the substantia nigra and the subthalamic nucleus in a functional con-

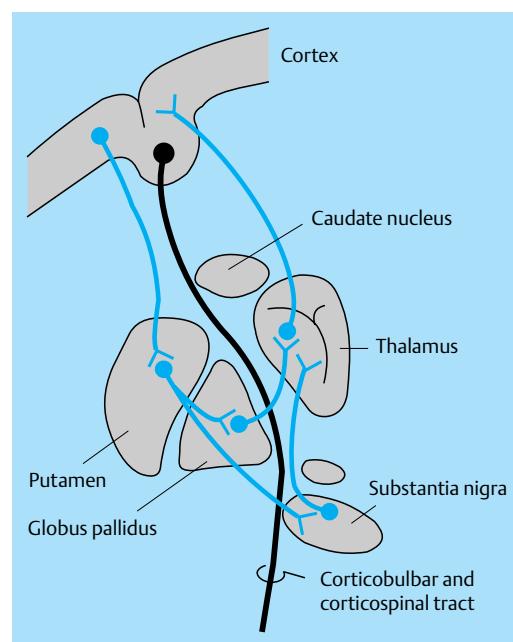


Fig. 1.21 The basal ganglia, an overview.

	Called the lentiform nucleus	
Caudate nucleus	Putamen	Globus pallidus (pallidum)
Called the (neo-)striatum		
Called the corpus striatum		

Fig. 1.22 An overview of the different nuclei, both individually and grouped together.

text (Brodal 2001). The different nuclei are often grouped in pairs and are then named together (see Fig. 1.22).

Afferent connections to the basal ganglia:

- The striatum is the main receiving nucleus—from almost all parts of the cortex, from the thalamus, and nuclei in the mesencephalon
- The caudate nucleus—from the association areas (higher mental functions)
- The putamen—from the somatosensory cortex (SI) and the primary motor cortex (MI)

Efferent connections from the basal ganglia:

- From the striatum to the globus pallidus and substantia nigra, which are the two output nuclei of the basal ganglia
- The globus pallidus and substantia nigra inhibit their target nuclei in the thalamus and brain stem nuclei through a direct and an indirect pathway
- Activation of the direct pathway facilitates movement
- Activation of the indirect pathway inhibits movement

The basal ganglia are the principal subcortical components of several parallel circuits or networks connecting the thalamus to the cerebral cortex. Brodal (2001) states that the basal ganglia first and foremost affect motor activity through connections with motor areas in the cerebral cortex and the brain stem and only to a small degree through their own descending fibers. According to Kandel et al. (2000) the nuclei of the basal ganglia receive their primary input from the cerebral cortex and send their output to the brain stem via the thalamus, back to the prefrontal, premotor, and motor cortices. The basal ganglia are also associated with complex cognitive and behavioral functions: through their interaction with the cerebral cortex, the basal ganglia also

contribute to musculoskeletal, oculomotor, cognitive, and emotional (limbic) functions.

The musculoskeletal circuit begins and ends in the precentral motor areas (Fig. 1.23). The precentral motor fields (the premotor, the supplementary motor, and the motor cortices) and the postcentral somatosensory send their information to the putamen. The putamen is an important site for integration of movement and sensory feedback information related to movement. It sends its information back through the two output nuclei via the thalamus to the supplementary motor area, the premotor cortex and to the precentral motor areas. Several authors have presented aspects of the basal ganglia's role in motor control (Rothwell 1994, Kandel et al. 2001):

- Disinhibition of some motor areas at the same time as facilitation of others, thereby "allowing" movement to happen
- Many aspects related to movement:
 - scaling the amplitude and velocity of movement
 - focusing the neural activity
 - identification of the position of the movement goal (feedforward)
 - identification of variables related to direction during the performance of movement
 - decision making: variables related to muscle activation necessary to move an extremity
 - performance of sequences of movement
 - tonic control and CPG initiation due to the links between the basal ganglia and the spinal cord

Movement is defined by the cerebral cortex, refined by the cerebellum and contextualized by the basal ganglia.

Clinical Relevance

Lesions to the basal ganglia produce three distinct types of motor disturbance: tremor and involuntary movements, changes in postural and muscle tone, poverty and slowness of movement without paralysis. Lesions do not cause paresis or paralysis, but affect motor performance for instance by tempo reduction. The basal ganglia send information to the thalamus via the internal capsule. This connection may be disrupted through a stroke affecting the internal capsule, and some stroke patients seem to develop parkinsonian symptoms.

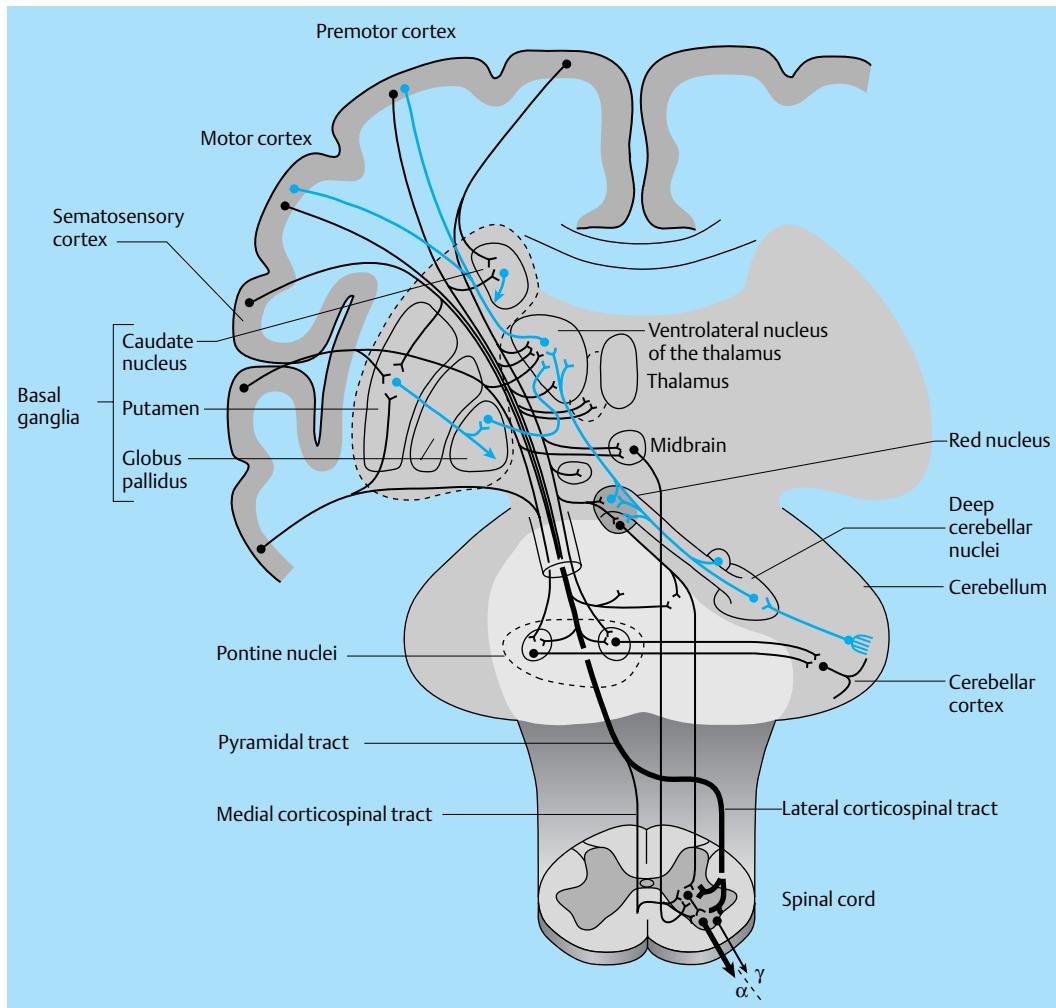


Fig. 1.23 Voluntary movement and the basal ganglia. (Redrawn with permission from Kandel et al., p. 347.)

The basal ganglia have an extensive network communication with different areas of the cortex, and are probably involved in motor planning. Clinically, it is necessary to use more cognitive strategies in the treatment of patients with parkinsonian features compared with patients who have lesions to other parts of the CNS. Lesions to the basal ganglia may lead to an increase of muscle tone (rigidity) and affect locomotion through its connections with the reticular formation in the brain stem. Lesions to the basal ganglia may give a variety of symptoms: *akinesia* (impaired initiation of movement), *bradykinesia* (reduced ampli-

tude and velocity of voluntary movement), *rigidity* (increased resistance to passive movement), or *tremor; dyskinesia, or dystonia* (involuntary movement), and *hypotonia* (reduced muscle tone).

Parkinson disease is characterized by akinesia or bradykinesia, rigidity, and resting tremor. The rigidity is probably caused by α -motoneuron excitability (Brodal 2001). Rothwell (1994) describes the consequences of loss of spontaneous movements as reduced balance, reduced anticipatory postural control, reduced righting reactions, reduced ability to step, and produce protective arm reactions. Both feedforward and feed-

back mechanisms seem to be affected. The basal ganglia receive extensive information from cortical areas. Lesions to the basal ganglia may cause loss of more automatic motor skills thereby causing the patient to compensate with cortical mechanisms. This may be one reason for the tempo reduction that patients with Parkinson disease experience.

Jobst et al. (1997) hypothesize that the movement problems that patients with Parkinson disease present with may in part be due to decreased proprioception. They refer to animal studies that show that the putamen responds to somatosensory stimuli and to passive movement, especially rotational movements. Rotation gives more proprioceptive feedback than, for instance, palpation of muscle or tendon tapping. They say that perhaps the basal ganglia can influence movement by modulating sensory information or function as a "sensory gate." Jobst et al. (1997) conclude by stating that the sensory aspects of kinesia are defective in patients with Parkinson disease because all the patients only had problems when they had to rely on their proprioceptive/kinesthetic sense alone. They say that the role of the basal ganglia is probably sensory modulation and integration of sensory input to motor tasks. This would lead to deficiency in the individual's ability to:

- Judge the position of the extremities in space, especially during movement
- Control sequence and timing due to reduced or defective feedback from the movements of the extremities

Jobst et al. (1997) suggest that physiotherapy should focus on improving the patient's kinesthetic sense as well as other interventions oriented toward the improvement of the patient's motor function.

Dystonia is a condition characterized by repeated muscular contractions that cause uncontrolled, slow, writhing, repetitive movements as in *Huntington chorea*, or abnormal stereotyped positions such as *spastic torticollis*. Dystonia is often related to the basal ganglia (putamen and globus pallidus; Rothwell 1994). Dystonia may present itself as:

- Focal dystonia that is localized to one body part, e.g., torticollis
- Segmental or multifocal dystonia that affects more body parts

- Generalized dystonia that affects the whole body, e.g., Huntington disease

One cause of dystonia may be *repetitive strain injury*. Movements that are performed repetitively may cause changes in the structure and function of the CNS (form–function). If a person pursues a profession or hobby requiring intensive repetition a movement of the hand, e.g., secretaries, musicians, he may develop *occupational hand cramp* (Byl et al. 1997), which is viewed as a type of focal dystonia of the hand. Dystonia may also develop secondary to a stroke or head injury. Approximately half of the generalized dystonias are assumed to be secondary. For the focal dystonias, it is only possible to diagnose a cause in approximately 10% of those affected (Gjerstad et al. 1991). If the disorder starts in childhood, it may be possible to find a cause in 40%; in 30% if developing during youth; and 13% in adult (Marsden and Quinn 1990, Borgmann 1997).

■ Inhibition—Regulation of Central Nervous Activity

The CNS regulates and modifies impulse transmission through *inhibition*. Special neurotransmitters in the CNS are responsible for the inhibition of impulses. GABA (γ -amino butyric acid) is the most common one. There are many forms of inhibition. The following seems to be especially important for movement:

- Presynaptic inhibition
- Postsynaptic inhibition
- Recurrent inhibition
- Lateral inhibition (see Stereognostic Sense earlier in this chapter)

One motoneuron may have 50 000 synapses. It is the *sum of inputs to the motoneuron that decides the output*. The nerve cells may receive many inhibitory inputs and still reach threshold for firing.

Presynaptic Inhibition

Presynaptic inhibition is important for precise, focused, and graded muscular activity. Presynaptic inhibition is transmitted through axoaxonic synapses. (When an axon synapses with the synaptic terminal bouton of another axon, an *axoaxonic synapse* is formed.) The release of excitatory neurotransmitter substances from the presynaptic bouton is inhibited by presynaptic inhibition, and the impulse stops (see Fig. 1.24). This

effect occurs in the presynaptic cell. The postsynaptic cell is unchanged (Fig 1.25).

Impulse transmission may be inhibited through presynaptic inhibition, and the excitatory effect on the postsynaptic cell moderated. In this way, somatosensory information may be modified and modulated, irrelevant information stopped and the contrast between sensory information and awareness increased (Rothwell 1994, Brodal 2001). In the clinical situation, somatosensory information may modify the activity of the CNS through presynaptic inhibition (Musa 1986, Kidd et al. 1992).

Changes in afferent information may modify the activity of the CNS.

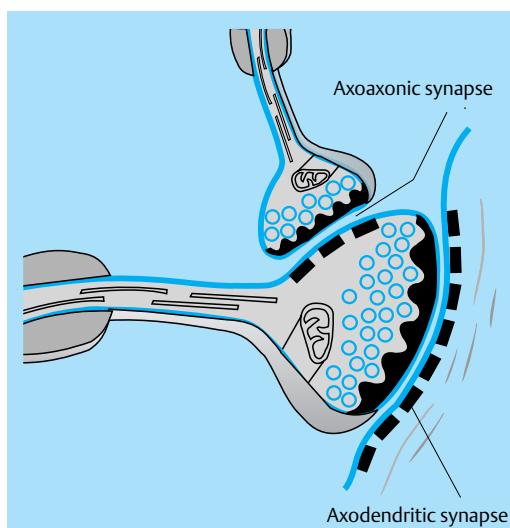


Fig. 1.24 Presynaptic inhibition

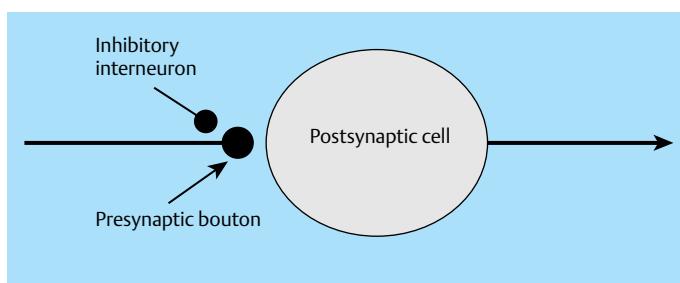


Fig. 1.25 Presynaptic inhibition. The inhibitory interneuron synapses on the presynaptic bouton of an axon, which in turn synapses with the postsynaptic cell.

Presynaptic inhibition is important for movement. It aids the recruitment of muscles with appropriate timing in the right sequence and it is a very specific mechanism that is precisely modulated to different types of movement: (Rothwell 1994).

- Presynaptic inhibition modulates the activity between different muscles over different joints in an extremity, for instance between gastrocnemius and soleus in standing and walking, and assists in the appropriate sequence and timing of muscles.
- There is reciprocal inhibition between flexors and extensors normally (one inhibits the other), for instance the tibialis anterior and soleus in the leg. Reciprocal inhibition means that a muscle is inhibited by activity in its antagonist. In some pathological conditions this mechanism does not work: there is no reciprocal inhibition from tibialis anterior on the soleus, while there is a strong reverse effect. That is, increased pathological activation of the soleus may inhibit tibialis anterior without the activity in the tibialis anterior being primarily affected by pathology.

Presynaptic inhibition may regulate the recruitment and modulation of motor units through *spatial* and *temporal distribution* (Fig. 1.26). There is a considerable axonal branching in the CNS, and thereby one axon may spread and supply many other neurons many times. *Temporal summation* of action potentials means that many action potentials travelling at a fast frequency in an axon build upon each other (Brodal 2001) and thereby increase the strength and duration of information. The repetition of impulses is regulated through presynaptic inhibition, and the repetition stopped if needed. Thereby, the

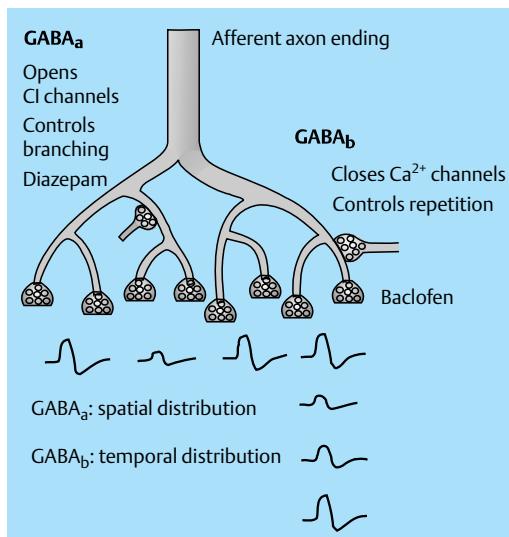


Fig. 1.26 Spatial and temporal distribution through presynaptic inhibition. Presynaptic inhibition may be activated or not at places where the axon branches (GABA_a) to select the spread of impulses (spatial distribution). The repetition of impulses (temporal distribution) is regulated through presynaptic inhibition (GABA_b) and repetition stopped if needed, i.e., the strength of impulse transmission is modulated. (Cl = calcium.)

duration of the action potentials is controlled through *temporal distribution*.

Spatial summation means that information from many different sources converge on one neuron (Brodal 2001).

Axonal branching from any one axon cause impulses to be spread over a large number of neurons, as well as repeatedly to one neuron (divergence). This leads to a *spatial distribution* of impulses. Presynaptic inhibition can be turned on or off where branching occurs (see Fig. 1.24, GABA_a) to focus the distribution. The action potentials are therefore transmitted to where they are needed. In this way, the number of motor units required for an activity may be regulated. The CNS has the ability to modify or control the direction and spread of impulses normally, to hinder a too diffuse activation of motor unit recruitment.

Postsynaptic Inhibition

Postsynaptic inhibition is when one neuron inhibits another by increasing the threshold for

postsynaptic depolarization. Release of inhibitory neurotransmitters from the presynaptic cell cause hyperpolarization of the post-synaptic cell membrane. More facilitatory/excitatory impulses will therefore be necessary to depolarize the postsynaptic cell, i.e., the threshold for depolarization has been increased.

Recurrent Inhibition

The Renshaw cell is an inhibitory interneuron (Rothwell 1994) that receives many collaterals from α-motoneurons locally, and is under supraspinal control by the corticospinal system. The collaterals from the α-motoneurons synapse with Renshaw cells (Fig. 1.27), which synapses back on to the same motoneuron as well as other motoneurons supplying motor units in the same muscles and muscles with similar functions. The Renshaw cell distributes inhibition to its:

- Own agonist α-motoneuron
- Synergic motoneurons
- Own agonist γ-motoneuron
- Synergic γ-motoneurons
- Other Renshaw cells and Ia inhibitory interneurons (from the muscle spindle)

At the same time, the antagonists are disinhibited, i.e., they are facilitated. The precise functional role of this system is still unclear, but some of the effects of Renshaw cell activation are:

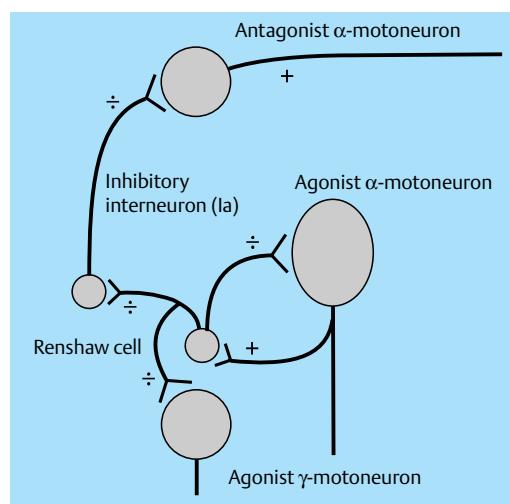


Fig. 1.27 The Renshaw cell loop

- Reduced firing frequency of the α -motoneuron
- The motoneuron is less sensitive to excitatory stimuli thereby increasing the contrast in motoneuron pools. This is best compared with lateral inhibition in the somatosensory system (see Stereognostic Sense)

Brodal (2001) states that the Renshaw cell may contribute to the rhythm in the CPGs by shortening the impulse trains from active motoneurons and at the same time increase the excitability of the antagonistic motoneurons. In this way, the rhythmic change may be facilitated. There seems to be reduced recurrent inhibition in distal body segments where the movements are faster and more voluntary, whereas in the proximal areas, recurrent inhibition seems to be important for slow or tonic muscle contraction.

Lateral Inhibition

Lateral inhibition is discussed under Somatosensory System, Stereognostic Sense.

Clinical Relevance

Selective control of movement depends on musculature being recruited in the appropriate sequence at the appropriate time, and that the duration and strength of muscular contraction (eccentric/concentric) is appropriate to the goal activity. Patients with CNS lesions have reduced control of movement for different reasons. There may be impairment of temporal and spatial distribution of activity. As the patient starts activity, the recruitment of motor units and muscles in relation to grading of the duration, spread of involvement, and repetition is sometimes disrupted. The reaction or response may therefore be greater than normal (see Cornall 1991 as an example).

When patients attempt to be functionally independent in a phase where the CNS is vulnerable and denervated, they may experience activation of muscles that normally would not be involved in the actual activity. Attempts at balancing in standing, transferring, or walking may lead to shortening of the patient's affected side, flexion of the arm or fingers, retraction of the pelvis or push through the foot. Clinically, if the patient learns to control his movements more selectively, these pathological mass patterns may be disrupted. Improved selective control is a sign that the patient is learning to control the distribution and spread of impulses, as well as focusing his activity.

Improved selective control seems to stop inappropriate spread of activity to other muscles. Pathologic mass patterns may therefore be disrupted by the improvement of selective control of movement.

Studies in stroke patients demonstrate that the lesion not only affects the affected side, but that there is pathologic activity also in the so-called unaffected side (Thilmann et al., 1990, Marque et al. 1997). This means that inappropriate activity may spread to both halves of the body, leading to an increase of effort.

Renshaw cell control may be reduced in CNS lesions. Disruption of this system may lead to clonus as the contrast within the motoneuron pools is reduced. The motor system may need more activation to depolarize the Renshaw cell to modulate the motoneuron activity. The grading and duration of a tonic contraction becomes less precise. Clonus is a sign of disturbed excitability of the spinal interneurons (Brodal 2001). Reduced recurrent inhibition may be a contributing factor to pathologically increased tone.

After a CNS lesion the motoneurons may be hypersensitive to excitatory stimuli.

Summary

Muscle fibers are able to alter their fiber type to some degree in relation to use. See page 7.

The length of a muscle is important for movement and function. See page 8.

Motor units in a muscle are recruited in a sequence, whereby the smaller motor units are activated before larger motor units. See page 9.

Postural stability is a basis for selective movement control and function. See page 9.

Compartmentalization describes the ability of a muscle that crosses more than one joint to perform different functions simultaneously. See page 9.

Muscle balance depends on muscular, neurologic, and biomechanical factors. Alterations in recruitment and the distribution of motor activity affect alignment. Altered alignment affects muscle function. See page 9.

Tone is related to both the state of the muscle fibers, the activity within the sense organs, muscle viscosity, and connective tissue. The most important cause of alteration of tone is muscular contraction. See page 11.

Heel-strike is important for initiation of stance and therefore locomotion. See page 17.

Heel-off is an important signal for the termination of stance, and therefore for swing phase. See page 17.

Ascending and descending systems are closely linked both anatomically and functionally. It is therefore inappropriate to discuss these separately. Motor activity is the result of a complex interaction between sensory, motor, and cognitive systems. See page 19.

Anticipatory adjustments of muscle activity (feed-forward) are dependent on both external and internal feedback. That is, information about the internal relationship between body parts, the relationship between the body and the environment, and from specific receptors from the eyes, muscle spindles, Golgi tendon organs, and receptors in the skin is necessary for feedforward and therefore for balance. See page 20.

Information (feedback) about muscle activity, movement, alignment, and weight distribution is important for balance, postural adaptation, and function. See page 20.

Mobility is a prerequisite for the firing of specific receptors in relation to displacements and variation in sensory information due to movement, and therefore for balance. See page 20.

An updated body schema is a prerequisite for appropriate and efficient feedforward control. See page 21.

Stereognostic sense is based on somatosensory information, movement, the ability to recognize contrasts and perception. See page 21.

Muscle activity is important for joint position sense. See page 22.

Variation in somatosensory information arouses awareness to the stimulated body part. See page 23.

Walking on a flat, even surface is probably controlled by CPG in the spinal cord and brain stem and coordinated by the cerebellum, i.e., it is mainly an automatic activity. See page 25.

The activity of the vestibular system is greatest when postural control is needed. See page 27.

Head control and postural control affect each other reciprocally. See page 28.

The ability to dynamically balance on one leg seems to be a prerequisite for a free swing. See page 28.

Active swing that is initiated too early may interfere with the stability of the standing leg. See page 28.

Stability and balance are prerequisites for movement. See page 30.

Standing promotes awareness and arousal. See page 30.

The corticospinal system innervates most motoneurons. Most movements may be voluntarily controlled to some degree. If needed, cognitive strategies may override more automatic movements. See page 32.

The corticospinal system mainly supplies distal musculature. See page 32.

Distal motor control, i.e., dexterity of finger movements and movements of the toes, are examples of voluntary (least automatic) activity. See page 32.

The cortex is able to weight the relative importance of different sensory modalities via the corticospinal system. See page 32.

Functionally, the descending systems are broadly divided into lateral and medial systems. The main function of the medial systems is maintenance of postural control, stability, and balance (most automatic). The main role of the lateral systems is related to voluntary activity (least automatic). See page 34.

The cerebellum seems to have a role in the refinement and sculpturing of movement by filtering unwanted muscle activation. See page 37.

The prototype of movements and activities seem to be learned through varied repetition. See page 38. Changes in afferent information may modify the activity of the CNS. See page 43.

Improved selective control seems to stop inappropriate spread of activity to other muscles. Pathological mass patterns may therefore be disrupted by the improvement of selective control of movement. See page 45.

After a CNS lesion the motoneurons may be hypersensitive to excitatory stimuli. See page 45.

1.2 Plasticity

In 2004, the Academy of Medical Science stated the importance of science for neurorehabilitation: "The last two decades have seen unprecedented advances in neuroscience that have transformed our understanding of the extent to which functional recovery is possible following neural damage, how this recovery takes place and how it may be promoted."

Hypotheses about the structure and function of the brain, and its ability for restructuring and repair, were originally based on studies on starfish and frogs. Just recently it was thought that there was no possibility for repair or change within the CNS after it had suffered a lesion. Clinically, however, therapists found that many patients improved and learned how to move again either as they had done before or by using other strategies (Bobath 1990). Scientific research has now shown that the structure of the brain changes and adapts to how it is being used by patients who have suffered a stroke (Ward and Cohen 2004). Ward and Cohen demonstrate that there is a connection between behavior and brain structure after lesions to the CNS. The human brain has a great ability to learn, and learning leads to structural and functional changes both in a healthy and in a lesioned CNS. Stein et al. (1995) viewed neuroplasticity in light of an organism's ability for survival and its ability to move around and interact with the world. Neuroplasticity is based on the property of nerve cells to store information about the

world and their ability to modify their own activity as a response to changes in the environment.

The neurophysiologist G. Kidd (Kidd et al. 1992) stated that "neuroplasticity is a concept based on the ability of the central nervous system to adapt, rebuild and reorganize itself in relation to its molecular form and function." He introduced the concept *form–function* to reinforce the interdependence between form (structure) and function. The interaction between form and function allows humans the opportunity to develop and meet functional needs. Plastic adaptation is use-dependent and the result of our interaction with the environment. Motivation and attention are necessary for learning. The role of the individual related to society, family, to social relations, possibilities, limitations, goals, demands, and needs are all important for how an individual develops and learns. How an individual uses his body and mind shapes the CNS. Movement, activities, strategies, and patterns of movement determine the connections in the CNS (Fig. 1.28).

Today, local brain function can be studied with noninvasive techniques such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), transcranial magnetic stimulation (TMS), electroencephalography (EEG), magnetoencephalography (MEG) and diffusion tensor imaging (tractography) (Fig. 1.29). These techniques make it possible to show different aspects of brain activity as specific images of the changes in the structure of the CNS can be obtained and these can be correlated with changes in the patient's functional ability after a CNS lesion (Academy of Medical Sciences 2004, Ward and Cohen 2004). This kind of research does have its limitations because the patients need to keep their head still while

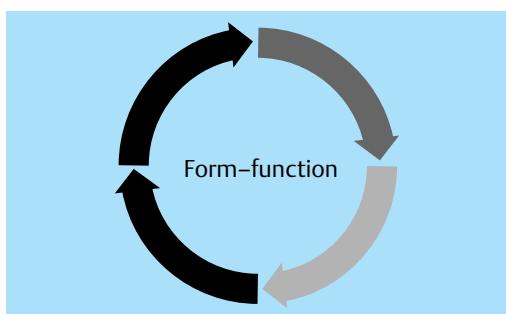


Fig. 1.28 Form and function

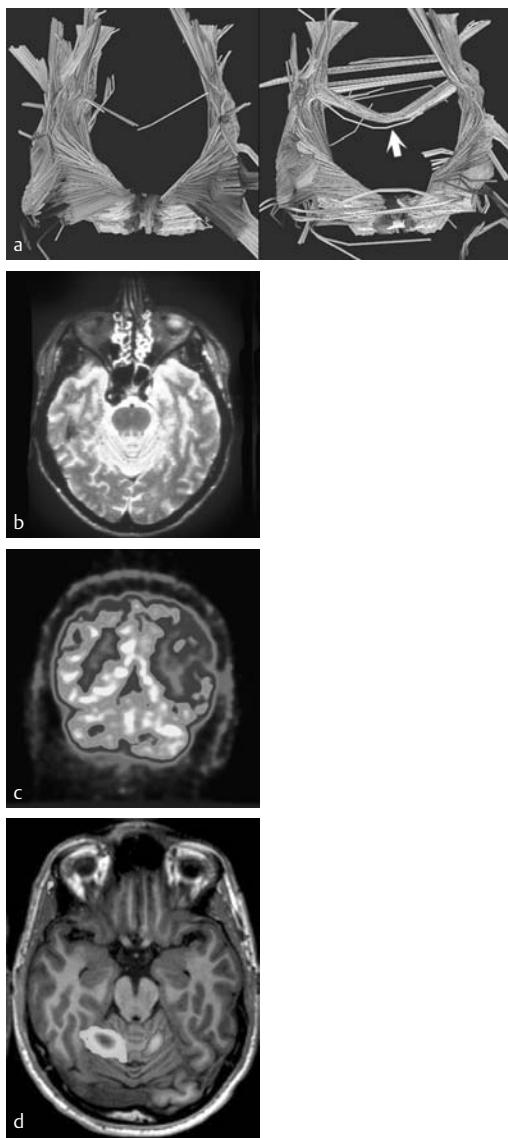


Fig. 1.29a-d Examples of noninvasive imaging techniques: (a) diffusion tensor imaging (tractography); (b) magnetoencephalography (MEG); (c) positron emission tomography (PET); (d) functional magnetic resonance imaging (fMRI).

the image is being taken, but new methods are developing all the time.

Brodal (2001) states that "The nervous system is plastic (adaptable), i.e., it has the capacity to alter its structure and function as a response to changed demands. The development and the per-

formance of the nervous system therefore depends on an interaction between genetics and environment, nature and nurture. The fact that we have an ability to learn demonstrates that the function of the nervous system at synaptic level somehow can be altered by external influences" (author's translation). Plasticity is present on all levels of the CNS, in the peripheral nervous system, and in musculature (See The Neuromuscular System).

Plasticity is a prerequisite for learning, both in the short term and in the long term (Brodal 2001). Plasticity implies that changes occur within the structure of the CNS, for example by the formation of new connections, and that these may weaken or increase in strength through use. Plastic adaptation occurs through life; on a local, cellular level this may cause substantial transformation of axons, dendrites, and the internal environment, synapses, and transmitters. Learning probably causes synaptic changes in many parts of the CNS, with a distribution that is specific to what is being learned (Brodal 2001). A prerequisite for change of the postsynaptic cell is that precise sensory information and modulating transmitters (for example, those transmitting information about motivation and awareness) hit the synapse at the same moment in time. This explains why motivation is important for structural changes to occur, i.e., for learning. Synaptic activity is based on several factors, and sculpturing of synaptic connections occurs throughout life (Benowitz and Routtenberg 1997). Plasticity also implies that inappropriate connections are inactivated or removed. Cellular plasticity may eventually cause system reorganization.

Functional plasticity refers to changes in the efficacy and strength of synaptic connections caused by modulation and release of transmitters (Agnati et al. 1992). These lead to:

- Changes in the size of the active zone of the synapse
 - Changes in the number of synaptic vesicles (small sac containing transmitters in the terminal boutons)
 - Pre- and postsynaptic structural modifications
- Brodal (2001) has described how experimental data suggest that synaptic plasticity is the basis for learning and memory. Synaptic plasticity implies that the presynaptic action potential leads to an increased release of neurotransmitters,

and that the postsynaptic cell changes its response to the same amount of transmitter, or both. *Structural plasticity* refers to changes in the organization and number of synapses by axonal collateral sprouting, increase in the size of the dendritic tree, and the formation and production of new synaptic connections.

Kandel et al. (2000) state that behavior is shaped by learning, and that memory, which is the outcome of learning, has at least two forms:

- *Implicit (nondeclarative) memory* is the unconscious memory for perceptual and motor skill.
- *Explicit (declarative) memory* is a memory for people, places and objects that require conscious recall. Explicit memory in mammals involves long-term potentiation in the hippocampus (for more detail, please see Kandel et al. 2000).

Implicit memory has many forms: habituation, sensitization, and classical conditioning. These have been mostly studied in vertebrates and invertebrates, but are much more complex in humans. *Habituation* involves presynaptic depression of synaptic transmission and is related to harmless stimuli. There is a decrease in synaptic strength as a result of decrease in the number of transmitter vesicles released from presynaptic terminals of sensory neurons. The plastic changes in the functional strength of synaptic connections constitute the cellular mechanisms mediating the short-term memory for habituation. Learning can lead to changes in the synaptic strength. There is both a short-term and a long-term form, and the duration of the short-term memory storage is determined by the duration of the synaptic change. *Sensitization* involves presynaptic enhancement of synaptic transmission and thereby of reflex responses caused by the application of harmful stimuli. It is more complex than habituation because stimuli to one pathway produce a change in another pathway. Repetition of the stimulus decides whether this is a short-term or a long-term change. *Classical conditioning* is learning through the association of ideas—associative learning. Classical conditioning involves the pairing of two stimuli and is a means by which an animal learns to predict events in the environment (Kandel et al. 2000). This is dependent on activity in both the pre- and the postsynaptic cell and involves presynaptic facilitation of synaptic transmission. The postsynaptic component is a retrograde signal from the sensory neuron. Three sig-

nals in a sensory neuron must converge to produce the large increase in neurotransmitter release that occurs with classical conditioning. Two of these are caused by action potentials to activate chemical processes for the conditioned and unconditioned stimuli, the third is a retrograde signal from the sensory neuron indicating that the postsynaptic cell has been adequately activated by the unconditioned stimulus. These different forms of implicit memory interact and may strengthen each other for longer lasting enhancement.

Squire et al. (1993) and Kandel et al. (2000) stated that short-term memory is associated with plastic changes lasting from seconds to minutes and caused by changes in the presynaptic membrane. Long-term memory, however, may last for as long as many weeks and is caused by changes in the postsynaptic membrane. Both these pre-and postsynaptic changes are called “short-term potentiation” (STP). Changes lasting long as months and years are associated with changes in the gene expression in the cell nucleus and called “long-term potentiation” (LTP).

All forms of learning cause structural and functional changes in the CNS.

■ Neuroplasticity

Neuroplasticity depends on gene expression, synaptic activity, axonal transport, neurotrophic factors, collateral sprouting, and probably more factors. Motor learning is assumed to take place on all levels of the CNS. The drive for change is caused by a need or desire to interact with the environment in an appropriate manner.

■ Gene Expression

All cells of the body have a complete set of genes. The different genes have different functions expressing skin, hair, nails, eyes, different types of muscle fibers, different types of nerve cells, and so on. The fact that nails become nails is a result of expression of the nail gene only—the other genes in the nail cells are silent. This is called gene expression (Martin and Magistretti 1998, Brodal 2001).

The term *genotype* refers to the complete genetic constitution of an organism as decided by the specific combination and localization of the genes on the chromosomes. Organisms with the same genetic make-up belong to the same genotype. The human race, *Homo sapiens*, constitutes a genotype (Mosby-Yearbook 1994). As humans we have a common inheritance that allows us to balance and walk on two legs and at the same time use our arms and hands for functional activities. Humans are the only genotype to have developed these fundamental abilities, which are the basis for the intellectual development of the human race (Eccles 1989). Each individual inherits a unique combination of genes from their parents, and develops through the interaction of nature and nurture to a unique phenotype.

The *phenotype* is defined as the complete observable characteristics of an organism, and includes anatomic, physiologic, biochemical, and behavioral aspects, formed by the interaction between the genetic make-up of the individual and the environment (Mosby-Yearbook 1994). Each individual has an inherent ability to develop and express genes in his own, unique way. Therefore, as individuals we move differently, behave differently, and have different talents that we develop in our own way—each person is unique. We do, however, have a common repertoire of movement that is expressed individually. The ability to learn is the basis for individual and specific characteristics of physical and intellectual abilities. Lasting plastic changes are a result of altered gene expression.

Several scientists describe changes in the CNS as activity-dependent (Seil 1997, Martin and Magistretti 1998, Kandel et al. 2000, Brodal 2001, Ward and Cohen 2004 and many more).

Environmental influences and stimuli direct plasticity and thereby learning.

■ Neurotrophic Factors

There are many proteins responsible for growth, development, and programmed cell death (apoptosis) in the CNS. Together these are called the *neurotrophic factors*. There are many different types of neurotrophic factors, for instance, nerve growth factor (NGF), growth associated protein (GAP-43/B-50), brain-derived neurotrophic factor

(BDNF), and more are being discovered all the time (Olson 1996). The production of these proteins is guided through gene expression; they are present at all times in the nervous system, but levels are higher during development and when the need for regeneration and reorganization is at its greatest.

Function

Neurotrophic factors influence and guide (Stein et al. 1995):

- Collateral sprouting and regeneration
- Survival of damaged neurons
- Neuronal death (apoptosis)
- New terminals and growth cones of axons
- Formation, maintenance, and transmission across new synapses
- Inhibition of the named processes

Neurotrophic factors are necessary for the process of learning. Physiologic activity in the form of training, exercise, and daily activity stimulates the release of neurotrophic substances; activity maintains the production and inactivity reduces production (Agnati et al. 1992, Bailey and Kandel 1993, Olson 1996). These factors stimulate metabolism of nerve cells, nerve fiber growth, and activity-driven changes in synaptic efficiency. They depend on retrograde signals from the postsynaptic to the presynaptic cell. Activity, the type of activity performed—how our body moves and is used—influence the CNS.

Activity and movement facilitate plastic changes in the CNS, both positively **and** negatively.

■ Axonal Transport

Nerve fibers, or axons, contain axoplasm. The axoplasm moves at various speeds in the axon by a process called *axoplasmic flow* in two directions and carries particles with it (Olson 1996, Benowitz and Routtenberg 1997):

- *Anterograde axonal transport*—from the cell body to the synapses
- *Retrograde axonal transport*—from the synapse and back to the cell body

These axonal transport mechanisms represent adaptations of mechanisms that facilitate the intracellular transport of organelles in all secretory cells (Kandel et al. 2000). Particles are actively

transported in a start-and-stop (salutatory) fashion along linear tracks aligned with the main axis of the axon. Axonal transport is a type of information transmission between neurons in addition to the *action potentials* (Kidd et al. 1992). Retrograde transport is also used to deliver signals to the cell body (Kandel et al. 2000). Action potentials may influence the speed of transport of the particles that the CNS needs for development, learning, and reorganization.

Motor activity may facilitate axonal transport.

Axonal transport is responsible for moving proteins and other particles to and from the cell bodies to the synapses. Activated growth factor receptors are thought to be carried along the axon by retrograde transport to their site of action in the nucleus and, for instance, cytoskeletal matrix is transported anterogradely. *Synthesis* is the production of complex substances from simple ones through chemical processes. Protein synthesis in the CNS is essential for gene expression and learning. Axonal transport has an important role in regeneration and reorganization of the CNS and the neuromuscular system.

Through retrograde transport the cell bodies are informed of activity at the synapses and the postsynaptic cells and about its—the presynaptic cell's—effect. The presynaptic cell may then, based on this form of feedback, alter its synaptic effect if necessary (Brodal 2001). The effector cells thereby have an important influence on the neurons innervating them, and may inform the CNS about what they want to know about and what information they need. The state of the muscles is therefore important for the function of the CNS, and the activity of motoneurons is essential for the maintenance of the structure of the motor end plate and the metabolism of the muscle. The motor end plate, the density and distribution of specific receptors within the muscle, and the functional characteristics of the muscle fiber types may be changed through direct stimulation (Troenner and Edgar 1982).

Muscular activity probably enhances the transport and production of neurotrophic substances. Stimulation may cause changes in the metabolism, structure, and function of muscle.

Collateral Sprouting

Axons may sprout as buds on a tree (Fig. 1.30). This is called axonal or collateral sprouting and is present in both the intact and lesioned nervous systems. In a CNS lesion if a nerve cell is damaged, its axon degenerates from distal to proximal and leaves behind empty synaptic sites where they previously made contact. This happens on all levels in the nervous system. Undamaged axons nearby are stimulated to sprout through reactive synaptogenesis or reactive reinnervation by neurotrophic substances that are released as a response to damage. Retrograde axonal transport carries information about activated growth factor receptors that may stimulate the formation of a growth cone on the axon. Growth-associated protein (GAP 43) is a protein released by the nerve cells with empty synaptic sites. The ensuing branches, or the collaterals, seek new contact sites to form new synapses where the old ones have been lost (Hallett

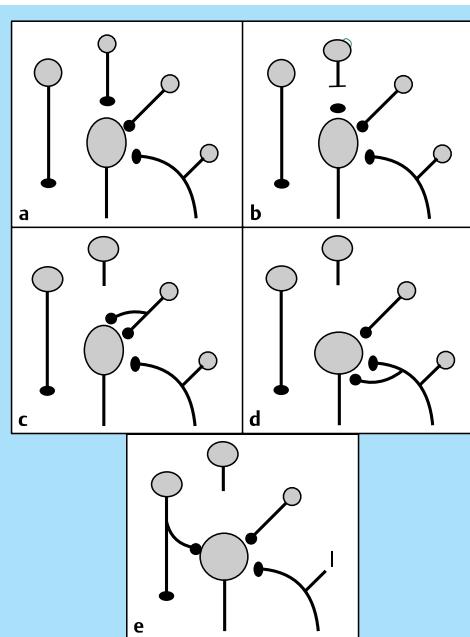


Fig. 1.30a–e Simplified illustration of the process of formation of new synapses through collateral sprouting: (a) normal situation; (b) damage leads to degeneration of axons from distal to proximal; (c) sprouting from an interneuron; (d) sprouting from an afferent (sensory) fiber; (e) sprouting from a descending fiber.

1995, Lee and van Donkelaar 1995). The new connections will not restore the original pattern of innervation (Goldberger and Murrey 1988, Brodal 2001). They may use other neurotransmitters, and may therefore not be able to reestablish lost function completely.

The effect of collateral sprouting may not always be positive; it may not lead to normalization of control of movement or other lost functions. If the collateral sprouts arise from sensory neurons, the patient may become hypersensitive to peripheral stimuli. Brodal (personal communication 1998) says that not all collateral sprouting is a result of learning; experimentally, collateral sprouts from nearby axons may form new synapses due to the release of local trophic factors. Whether they are retained depends on whether they are being used, i. e., stimulated, if the organism experiences them as appropriate. If the patient attempts to move appropriately, a secondary change of the first random contacts may result. These may become more permanent and contribute to more—or less—appropriate patterns of movement.

Both appropriate and inappropriate movement may be learned.

■ (Re)organization of Cortical Maps

There has been extensive research on cortical plasticity, and Nudo et al.'s (1996) study has received intense attention. It shows that the elbow and shoulder areas of the motor cortex learned to take over the control of hand movements after intensive training in monkeys who had been inflicted with a “stroke” in the hand area of the motor cortex. Many later studies have shown that the cortex has a significant ability for functional and structural plasticity. The cortical areas associated with the sensorimotor function of the body parts that are most active in the skills training have an increase in number of connections and/or size because of new learning (Nudo 2003, Ward and Cohen 2004). Learning after a CNS lesion is probably not so different from learning with an intact CNS (Brodal 2001). Through noninvasive imaging studies, we now know that the structure of the brain changes both after a lesion and as a result of training (Ward and Cohen 2004).

The motor and sensory cortices are able to undergo considerable reorganization after damage or other forms of peripheral or central influences causing changes in the neural patterns of activity (Stein et al. 1995, Nudo et al. 1996). Piano players and Braille readers have larger representations in the motor areas of the cortex representing manipulative skills (dexterity, fine motor activity) than the average population (see Fig. 1.6, p. 16). People who have had an amputation have larger representation of the body parts proximal to the amputation than the average population. Altered somatosensory perception may cause changes in the functional architecture in the brain. Activity is needed to sculpt the connections that form the neural representations (Bailey and Kandel 1993). Seil (1997) describes three main mechanisms for the reorganization of the cortex:

1. Unmasking of existing, but functionally inactive pathways
2. Collateral sprouting and the formation of new synapses
3. Redundancy: this hypothesis suggest that the CNS has many parallel systems with similar functions and that an alternative route may take over the damaged functions

After a CNS lesion, movement of a limb is associated with a bilateral pattern of activation within the brain. As a result, the patient activates larger areas for simpler movements than previously: an ipsilateral displacement of activity and an additional contralateral activation resulting in the recruitment of more motor areas. As movement improves, the activation becomes more focused (Cramer and Bastings 2000, Ward and Cohen 2004). Areas close to the lesion may take over, and/ or areas in the opposite hemisphere may be strengthened and latent connections developed.

Even areas far from the lesion may be affected through *diaschisis*. Diaschisis is described as the process whereby disturbance or injury to one part of the CNS may cause alteration in function of some distant part (Kwakkel et al. 2004a). Small et al. (2002) refer to neuroimaging studies whose results support a role for the cerebellum in mediating functional recovery from stroke. The data suggest that patients with good recovery have clear changes in the activation of the cerebellar hemisphere opposite the injured corticospinal tract. The work suggests a possible link between cerebellar activation and behavioral re-

covery from hand weakness following stroke. The underlying mechanism is not known, but it could be due to hemodynamic changes, such as diaschisis, or to the postulated role of the cerebellum in motor skill learning.

Studies on humans suggest that motor control areas are modifiable as a result of central or peripheral pathology or *motor skills training*. Studies support that motor and movement experiences post lesion have a major role in the following physiologic reorganization of the nearby, intact areas (Nudo et al. 1996). Nudo et al. (1996) state that if there is no rehabilitative training after lesion, a further loss of area related to the functional representation of the affected body part ensues, and they discuss whether this may be connected with *learned nonuse* or *disuse atrophy* (see Chapter 3, Physiotherapy). They claim that rehabilitative training may prevent further loss of representation in the nearby intact tissue, and that the intact tissue may be directed to take over the damaged function. Sensory information may alter the modification, distribution and thereby the motor activity of the CNS (Umphred 1991).

Motor training leads to reorganization of sensorimotor areas of the cortex.

There are two main modulators for cortical function: sensorimotor learning and cortical lesion. The two factors interact and cause remodeling of the structure and function of the brain, which are shaped by the sensorimotor experiences of the individual, in weeks and months post lesion. Skills training is associated with an increased area of representation, increased synaptic density, increased number of synapses, and increased thickness of the cortical motor areas, probably due also to angiogenesis (more blood vessels, increased blood flow) (Nudo 2003). The sensory and motor cortices have a significant ability for reorganization throughout life, both in an intact CNS and after damage (as long as degenerative disease does not disrupt the ability to change). Therefore there are considerable possibilities for functional plasticity in the human adult neuromuscular system.

■ Formation of New Nerve Cells

About 10–15 years ago, it was accepted that there was no regeneration of nerve cells in the adult brain. Since then, new regenerated nerve cells—stem cells—have been found in the hippocampus, which is the memory center of the brain (Eriksson et al. 1998, Kempermann et al. 1998, Sundar 1999). Neural progenitor stem cells give rise to neurons, astrocytes, and oligodendrocytes, and have an inherent plasticity providing self-renewal and differentiation. Indeed, studies of animal models have shown that neural stem cells can specifically replace populations of diseased or damaged cells, in some cases leading to behavioral recovery (Hori et al. 2003). Solheim (2005) refers to scientific research where embryonal stem cells were injected into the spinal cord of a 37-year-old woman who had become paraplegic almost 20 years ago. By 41 days after the injection, the woman had regained considerable sensory and motor functions, and noninvasive imaging techniques (MR and computed tomography) demonstrated regeneration of the damaged area and parts of the cauda equina in the spinal cord.

Recently the role of the glia in the CNS has been investigated. Nearly one half of the cells in a human brain are astrocytes. Astrocytes have an intimate association with synapses throughout the adult CNS, where they help regulate ion and neurotransmitter concentrations. Recent studies, however, have found that astrocytes also exert powerful control over the number of CNS synapses that form, are essential for postsynaptic function, and are required for synaptic stability and maintenance. Moreover, recent studies increasingly implicate astrocytes as participants in activity-dependent structural and functional synaptic changes throughout the nervous system (Ullian et al. 2004). It has been proposed that astrocytes should not be viewed primarily as support cells, but rather as cells that actively control the structural and functional plasticity of synapses in developing and mature organisms.

Dietrichs, cited by Sundar (1999) said that stem cell research reinforces and supports the existence of brain plasticity and demonstrates the importance of focusing on neurorehabilitation after lesions or disease to the CNS.

Denervation Supersensitivity

Normally in the presynaptic cell and surrounding glia, any surplus transmitter is absorbed after depolarization at a synapse. After a CNS lesion, patients may experience hypersensitivity to stimuli probably caused by *denervation supersensitivity* (Stephenson 1993). Denervation supersensitivity is when the re-uptake of neurotransmitters fails (Fig. 1.31): as a new wave of depolarization hit the synapse and causes depolarization, the effect on the postsynaptic cell is much stronger than intended due to the transmitter already present in the synaptic cleft. Over time, the number of receptor areas on the postsynaptic cells, as well as their sensitivity, increases. More transmitter substance is then let through by each impulse transmission (Stephenson 1993). These changes may happen at all levels of the CNS.

Clinical Relevance

The learning ability of patients who have suffered an acute CNS lesion is increased. Suddenly their life changes dramatically, and even the simplest of tasks such as sitting independently or more complex activities such as managing the bathroom themselves and dressing are difficult. The consequences are serious for the individual, both physically and psychologically, and the

CNS is therefore driven to learn as quickly as possible to meet the impending functional needs.

Knowledge of plasticity gives the health professionals caring for such people hope and at the same time considerable responsibility. We influence the patient through mutual interaction and thereby have an important role in the reorganization of the patient's CNS, and therefore the progress of their abilities. Learning may lead to positive or negative developments in both physical function and behavior. It may result in altered movements and function, which are compensated for by developing alternative motor strategies. How a patient uses his body, how he moves or is moved by others will further influence the restructuring of the nervous and muscular systems. If the patient twists and turns on one leg as he transfers from a chair to the bathroom while the arm flexes uncontrollably, this will be what the CNS learns. If repeated time and time again, learning is established through functional and structural plastic changes in the CNS. Several authors suggest a timeframe for the most acute plastic changes after a CNS lesion (Nudo et al. 1996, Seil 1997, and others). These studies were done mainly on animals (cat, rat, monkey), but some were functional imaging studies (PET, fMRI and others) on humans after a lesion to the nervous system. The following discussion refers to these timeframes, which are broad with ample room for individual interpretation depending on the patient, his premorbid status, general condition, and type, location, and size of the lesion.

Acutely post lesion the CNS is in shock. The spinal shock may last for 2–3 days and may be caused by direct neuronal damage and increased inhibitory activity to protect the CNS from further damage. Changes in cortical and spinal function begin after a few hours as the:

- Level of neurotrophic substances increase
- Latent synapses and connections are activated
- Synaptic strength increases (LTP)
- Denervation supersensitivity develops

Initial restitution starts early, probably due to regression of edema and degradation of necrotic tissue. This may last for days and possibly weeks, depending on the size of the lesion. There is a gradual transition from the acute to the subacute and later stages. The changes in activity and function observed in the patient after 3–4 weeks may be due to neuroplastic changes in the form of:

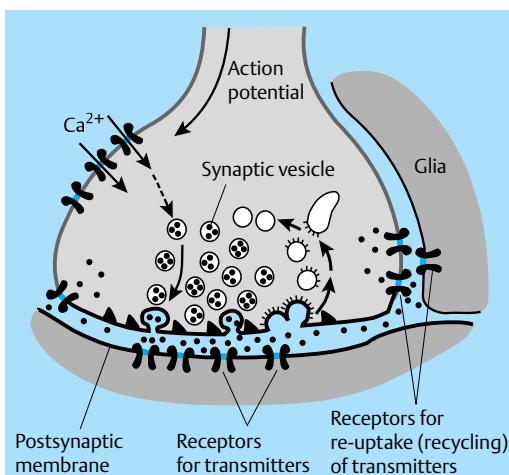


Fig. 1.31 Signal transmission at the synaptic cleft. Note the area of re-uptake of surplus transmitter.

- Synaptic changes
- Reorganization of cortical maps
 - further unmasking
 - redundancy
 - collateral sprouting (starts after a few days)
- The formation of new connections also at spinal level

Clinical experience demonstrates that many patients suffering from stroke, head injury, multiple sclerosis (MS), etc. may be hypersensitive to stimuli. Unexpected sounds, unrest, anxiety, fear of falling, and sudden or insensitive handling are examples of situations where the patient's tone may increase uncontrollably, and may constitute toward the development of spasticity (Craik 1991, Stephenson 1993). The requirement for activity that surpasses their balance and motor abilities and leads to malalignment during the recruitment of muscular activity is a major feature of this. "Functional recovery after CNS injury may depend, in part, upon reorganization of undamaged neural pathways. Spinal cord circuits are capable of significant reorganization, in the form of both activity-dependent and injury-induced plasticity." (Muir and Steeves 1997, p. 72).

Inappropriate handling of patients with CNS damage may cause inappropriate plastic adaptation.

Clinically, therapists encounter changes in the viscosity of muscle, contractures, altered alignment and pattern of recruitment, edema, and reduced circulation and metabolism, which have a negative effect on muscular activity. As well as the physical limitations to the repertoire of movement, axonal transport may be reduced through inactivity. Improvement of the above-mentioned factors should improve axonal transport and stimulate the production and transport of neurotrophic substances for the restitution and reorganization of the neuromuscular system, and thereby promote the patient's functional recovery. Muir and Steeves (1997, p. 75) state that "To train and modify spinal circuits for a particular motor task, it is important that the movements performed during training are executed as normally as possible" and "In several studies, enhanced peripheral stimulation has been shown to improve limb action after spinal cord injury." The

author understands *normally* to imply that alignment and muscle activation patterns are appropriate for the activity to be performed.

Improvement in motor control requires that the movements performed during training and exercising are executed as normally as possible, and that afferent information via skin, joints, and muscles is appropriate in temporal and spatial terms.

Early and probably intense rehabilitation are important for the patient's functional improvement. This is supported by studies on survival, psychosocial functioning, and the patient's home situation (Aboderin and Venables 1996, Stroke Unit Trialists' Collaboration 1997). Some authors claim that there is no use in continuing active rehabilitation after 6 months (Aboderin and Venables 1996). Ashburn (1997) stated that these studies had limitations because of the use of insensitive outcome measures, which do not detect qualitative changes in the patient's physical function.

Neuroplasticity gives rise to possibilities throughout life.

Shortly after a CNS lesion many patients experience paresis or paralysis to varying degrees. The CNS quickly compensates for the loss of function by new strategies for goal achievement. If an object cannot be reached by a patient's arm most affected after stroke, he quickly adapts to using the less affected arm. The most affected arm learns to be inactive, and the cortical areas representing this arm shrink and are taken over by intact areas. Learned nonuse presents a major limitation to the patient's sensorimotor improvement. Liepert et al. (2000) and Taub et al. (1999) have described changes in the brain after an intensive form of therapy called *constraint induced movement therapy*. This is based on theories of *learned nonuse* (learned inactivity). A certain level of intensity seems important to optimize the potential for improved function (Feys et al. 2004, Kwakkel et al. 2004b). If areas in the brain are not activated, transmitter production is lowered. Through intensive stimulation and requirement for use and activation, the brain is sti-

mulated to produce more neurotransmitter in the activated brain areas. This increase may last for approximately 36 hours. If not stimulated again within this timeframe, the production returns to its original low level (Lynch-Ellington, personal communication, 2005). This implies that patients who are in a restitution phase or have a long-term condition need daily focused treatment to improve sensorimotor function.

A certain intensity of treatment is necessary to improve the patient's sensorimotor function.

There is general consensus that training and rehabilitation need to start as early as possible after a CNS lesion. Studies performed at St. Olavs Hospital in Trondhjem, Norway, demonstrate that mortality and the need for nursing homes are halved and that the functional ability of patients improves following admittance to a stroke unit compared with admittance to non-specialized wards (Indredavik et al. 1991). The report from the Academy of Medical Sciences (2004) emphasizes the importance of early, correct, and appropriate rehabilitation. However, there is no widespread consensus on the intensity of stimulation and training during the first few days after lesions to the CNS. Turton and Pomeroy (2002) describe the biochemical cascades following brain infarction, especially in the penumbral zone surrounding the infarct. This area is very vulnerable, and if blood flow does not improve or is reduced even more, further cell death ensues (Fig. 1.32). As a result, the size of the infarct increases. The authors are of the opinion that caution must be shown the first few days. Activity causing increased blood flow and metabolism in areas of the brain not directly affected by the stroke may damage the penumbral zone even more.

Bishop stated in 1982 that the challenge for therapists in the future will be to learn how to promote and use neuroplasticity in neurorehabilitation.

Summary

All forms of learning cause structural and functional changes in the CNS. See page 49.

Environmental influences and stimuli direct plasticity and thereby learning. See page 50.

Activity and movement facilitate plastic changes in the CNS, both positively and negatively. See page 50.

Motor activity may facilitate axonal transport. See page 51.

Muscular activity enhances the transport and production of neurotrophic substances. Stimulation may cause changes in the metabolism, structure, and function of muscle. See page 51.

Both appropriate and inappropriate movement may be learned. See page 52.

Motor training leads to reorganisation of sensorimotor areas of the cortex. See page 53.

Inappropriate handling of patients with CNS damage may cause inappropriate plastic adaptation. See page 55.

Improvement in motor control requires that the movements performed during training and exercising are executed as normally as possible, and that afferent information via skin, joints, and muscles is appropriate in temporal and spatial terms. See page 55.

Neuroplasticity gives possibilities throughout life. See page 55.

A certain intensity of treatment is necessary to improve the patient's sensorimotor function. See page 56.

1.3 Consequences of and Reorganization after CNS Lesions

The consequences of a lesion to the CNS depend on the interaction of many factors:

- Diagnosis: a lesion, trauma, or a disease process
- Localization: one localized focus or many foci
- Extent of the lesion(s)
- Speed of development: acute or gradual onset

How the condition develops in an individual is related to the extent of plastic changes in his CNS, the individual characteristics of the lesion, and the patient himself: his premorbid physical status and whether complications arise, social si-

tuation and mental status (his resources and coping strategies), and his network of family, friends, and colleagues.

The initial paralysis or paresis after a stroke is due to the acute onset of the lesion (shock), neuron destruction and death, edema, decreased circulation, and possibly increased inhibitory activity that protects the brain against further damage (Fig. 1.32). As a result of the biochemical sequence of events, changes in the circulatory output and blood pressure occur in approximately 75% of stroke patients. In most patients

these levels return to normal in 7 days. Severe hyper- or hypotension is associated with poor prognosis.

Too high or too low blood pressure, hypo- or hyperglycemia, or increased temperature are factors that may cause further destruction of the penumbra. Turton and Pomeroy (2002) found that approximately 50% of the stroke patients showed an increase in the infarction area 2 weeks post stroke and 50% had a reduction in the infarction area. They advocate caution in stimuli or activity that may cause further cell death in the penum-

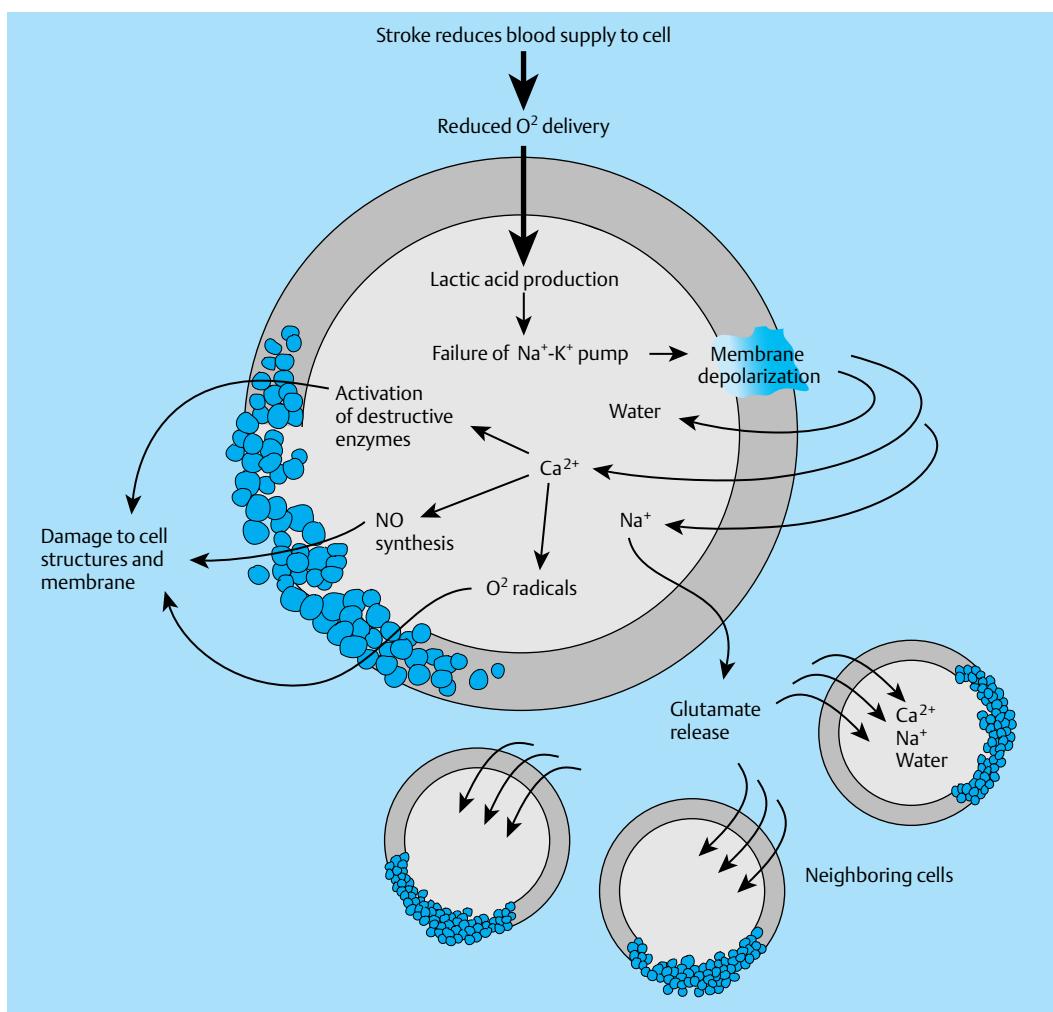


Fig. 1.32 The biochemical cascade. (Redrawn with kind permission from Turton A, Pomeroy V. When should upper limb function be trained after stroke? Evidence for and against early intervention. NeuroRehabilitation 2002; 17: 215–224.)

bra due to an increased blood supply in other, activated regions of the CNS. A gradual return of reflexes and motor activity is related to resolution of edema and necrotic tissue as well as initial reorganization of the CNS (see Plasticity). In stroke patients, Thilmann et al. (1990), Rothwell (1994) and Marque et al. (1997) report pathologic signs also on the least affected body half: weakness more proximally than distally, as well as altered stretch reflexes.

Over-activity (intense, specific stimulation) should be performed with great care the first week post stroke. Clinical decisions should be based on the patient's level of arousal, alertness, blood pressure, intracranial pressure, and temperature. Later patients probably suffer more from too little activity.

In MS patients there is a combination of lesions in more than one part of the CNS as well as an inflammatory disease process affecting the myelin sheaths and causing slowness in action potentials and some axonal degeneration. The lesions have a predilection for the optic nerves, periventricular white matter, cerebellum, and spinal cord white matter (Myhr 2001).

Stroke, MS, and other causes of brain lesions, damage, or disease, are together classified as upper motor neuron lesions. Upper motor neurons, or Betz cells, are pyramidal neurons located in the primary motor cortex. These neurons connect the brain to the spinal cord (Wikipedia 2006).

Upper Motor Neuron Lesions

Motor dysfunction after UMN lesions is classified into *positive and negative signs* (Canning et al. 2004). The *negative signs* are the direct results of the lesion itself, and the *positive sign* relate to secondary changes. This classification does not encompass cognitive or perceptual dysfunction or psychologic reactions, although these may be major reasons for patient's limitations toward learning or regaining of independence.

The Negative Signs

- Weakness
- Loss of dexterity
- Fatigue

Weakness

Weakness constitutes a major cause of the patient's physical disability. Stroke patients may experience weakness not only on their affected side but also on their so-called "good side," or more correctly, their least affected side (Canning et al. 2004). The weakness is first and foremost due to reduced or changed neural activation, i.e., weakness in systems and pathways in the CNS. Secondarily, inactivity and reduced muscular activation may cause atrophy and changes in the muscle fiber population (Patten et al. 2004). Toft (1995) states that reduced force production is also caused by fewer normally functioning motor units and the inability to activate as many units as before.

Reduced neural excitation may cause muscular weakness. Muscular weakness may then cause further secondary weakness of neural transmission. The main factor of weak neural transmission may be weak or vague body schema (especially the parietal cortex). This may affect the ability to create a postural image or body schema to move an extremity, i.e., the anticipatory postural adjustments (APA; feedforward postural control) to activate the corticopontine pathways as a background for extremity function.

Loss of Dexterity

Dexterity is the ability to adapt to the needs of the moment and depends on postural control for background stability. Canning et al. (2004) define dexterity as the ability to solve any motor task precisely, quickly, rationally, and deftly, where flexibility with respect to the changing environment is an important feature, and the ability to coordinate muscle activity to meet environmental demands.

Fatigue

Many patients report fatigue after a CNS lesion. In MS, fatigue is one of the major symptoms (Myhr 2001), and it is seen after other diseases or infections in the body (Soderlund and Mälterud 2005). Many stroke patients suffer from fatigue (Canning et al. 2004). Even in patients who

recover well, noninvasive studies of brain function demonstrate considerable changes in activation (Ward and Cohen 2004, Cramer and Bastings 2000, Cramer et al. 1997). The altered pattern of brain activation may constitute one reason for fatigue. Considerable energy and brain activation is recruited just to move an index finger – even after good restitution (Cramer and Bastings 2002).

Toft (1995) states that reduced force production causes an increase in the effort required to move. Therefore, weakness may be another reason for fatigue. Current evidence suggests that, generally after stroke, it is the negative impairments that limit recovery of function, rather than the positive impairments (Canning et al. 2004). Clinical experience suggests that these factors have also a major role in the recovery of function in other diseases affecting the CNS.

The negative impairments—weakness, loss of dexterity, and fatigue—seem to be the major factors that limit recovery of function in stroke patients.

The Positive Signs

The positive signs following an upper motor neuron lesion are (Pandyan et al. 2005, Canning et al. 2004):

- Increased tendon reflexes with radiation
- Mass reflex
- Clonus
- Dyssynergic patterns of co-contraction during movement
- Spasticity
- Associated reactions and dyssynergic stereotypical spastic dystonias
- Flexor spasm
- Extensor spasm

These symptoms are mainly related to the reorganization of the CNS, which implies that learning may have a major role in the development and establishment of secondary changes (positive signs). In the clinical situation, the terms spasticity and associated reactions are often used. Spasticity is used in a variety of contexts with different meanings. The two terms are discussed below.

Spasticity—Clinical Reflections

The interpretation of the term spasticity is wide, from Lance's (1980) definition of hyperreflexia and velocity-dependent resistance to passive stretch to complex problem consisting of both neural and non-neural changes in tissues. Spasticity is described as a syndrome (Burke 1988, Brown 1994), a condition (Toft 1995), as a result of development (Carr et al. 1995, Brodal 1998), and is related to functional plasticity in the CNS (Burke 1988, Brown 1994, Brodal 1998). These authors describe hyperreflexia as the first clinical sign and the main feature of spasticity.

It has been difficult to compare studies of spasticity as the outcome measures in clinical trials or tests have varied due to a lack of precise definition of spasticity. Recently the Support Network for the Assembly and Database for Spasticity Measurement—the EU-SPASM group was established (Johnson 2005) and charged by the European commission with the task of reviewing and evaluating methods for the measurement of spasticity and building a framework of expertise in Europe. This group has reviewed the literature and highlighted different aspects of the use of the term spasticity. The results of the group have been published in the journal *Disability and Rehabilitation* in 2005 (Johnson 2005, Pandyan et al. 2005, Platz et al. 2005, Wood et al. 2005, Voerman et al. 2005, Burridge et al. 2005).

Spasticity is defined by Pandyan et al. (2005) as: “Disordered sensorimotor control, resulting from an upper motor neurone (UMN) lesion, presenting as intermittent or sustained involuntary activation of muscles.”

This definition includes changes in the structure and function of the CNS and excludes both the negative signs of an upper motor neuron lesions and the biomechanical changes in soft tissues and joints.

Disordered motor control may involve:

- Loss of modulation from higher centers cause reduced inhibition of α - motoneurons; therefore they react with an abnormal firing frequency and duration to an excitatory stimulus. The loss of descending fibers to the spinal cord cause reduced activity in many different types of inhibitory interneuron

- Activity in other afferent pathways (e.g., cutaneous, proprioceptive). Cutaneous pathways seem to have a role in spasticity
- Disordered feedforward modulation of reflex activity
- Nonclassical behavior of motoneurons/internurons described as *plateau potentials* (a stable membrane potential that is more depolarized than the normal resting potential cause a cell to fire action potentials in the absence of continuous synaptic excitation); the threshold for firing is maintained at a lower level, which cause the neuron to depolarize even without continuous synaptic excitation

Activity in cutaneous and proprioceptive pathways seems to have an important role in spasticity.

Examples of changes in the structure and function of the CNS are:

- Increased gain (amplification) in the stretch reflex networks, i.e., for a given afferent input (Ia and II) the response from the respective α-motoneuron is greater (for revision, see The Neuromuscular System, Sense Organs in Muscle). This may be due to a number of different mechanisms:
 - increased excitability of motoneurons
 - changes in the characteristics of the α-motoneuron
 - reduced Ia presynaptic inhibition
 - changes in inhibition from the efferent pathway
 - altered reciprocal inhibition
 - reduced recurrent inhibition
 - increased excitability in the flexor reflex pathways (withdrawal)
 - altered force feedback (feedback about the actual force production in muscle)
- Decreased threshold of the stretch receptors, i.e., the stretch reflex is more easily elicited in people with spasticity. This may be caused by increased receptor sensitivity and an increased drive to the muscle spindle efferents. Current evidence suggests that spindle afferents activity is not necessarily abnormal in stroke patients (Pandyan et al. 2005)

The Complex Problem of the Upper Motoneuron Syndrome

The complex problems that health professionals experience in patients with upper motor neuron lesions may therefore be a combination of negative signs, spasticity as defined above, and other factors. Pandyan et al. (2005) state: "There is insufficient evidence in the literature to support the hypothesis that the abnormal muscle activity in spasticity results exclusively from stretch reflex hyperexcitability. It would appear that activity in other afferent pathways (e.g., cutaneous), supraspinal control pathways (or systems) and even changes in the α-motor neurone may also contribute to signs and symptoms associated with spasticity and other features of the UMN syndrome." The other features associated with the UMN syndrome may be:

- Components of inertia from extremity segments
- Changes in viscoelastic properties of soft tissues and joints
- Abnormal voluntary activation of muscle
- Abnormal involuntary activation from phenomena other than the stretch reflex hyperexcitability
- The patient's cognitive and/or perceptual abilities (the ability to understand instruction, etc.)

Clinically, altered sensory feedback and altered sensory perception also seem to have an important role. What therapists see in patients are the *consequences* of the lesion for the patient as a whole. Therefore the patient's resources and problems must be analyzed and interventions planned on an individual basis.

Pandyan et al. (2005) and many other authors (Burke 1988, Brown 1994, Brodal 2001, etc.) associate spasticity with lesions affecting corticoreticulospinal pathways; lesions to the corticoreticular pathways at the level of the cortex or internal capsule, and the reticulospinal and vestibulospinal tracts at the level of the spinal cord. The reticulospinal system plays an important part in the stability of proximal body segments; therefore lesions that affect the cortico-reticulospinal system are often associated with dysfunction in postural control and balance, as well as spasticity. Studies on the corticospinal system suggest that lesions probably do not cause spasti-

city, but produce a loss of distal dexterity (Brodal 2001). Indirectly, the functions of the cerebellum and the basal ganglia may be disturbed causing clumsiness.

Lesions to the cortico-reticular, reticulospinal and vestibulospinal pathways are associated with spasticity.

■ Associated Reactions— Clinical Reflections

The term associated reactions is both understood and used differently by different clinicians. This section presents clinically relevant reflections about spasticity and associated reactions and the formation of some hypotheses of possible causal relations, factors that initiate these responses, and consequences for the patient's motor control.

The term associated movement refers to natural activity that either requires considerable effort or is complex or new and is a normal feature of movement. Associated movements have many features in common with associated reactions, but some aspects are different. As a new skill is being learned, associated movements gradually disappear. This is not the case with associated reactions. Associated reactions are characterized by an activation of motor units or muscles not normally involved in the performed movement, and they get stronger through practice of the movement. They may also be called *dyssynergic patterns of movement*. The following general discussion is related to the development of motor disorders post stroke. Many patients with different CNS lesions experience similar symptoms irrespective of the exact diagnosis, e.g., cerebral infarctions, intracerebral hemorrhage, subdural hemorrhage, head injury, MS, incomplete spinal cord injury and other upper motor neuron lesions.

A few hours or days after a stroke the patient starts to show some initial motor activity, some controlled and often some uncontrolled movement in the most affected parts of the body (the so-called hemiplegic side). Uncontrolled activity is usually first observed in situations that require motor control that the patient has not yet developed, i.e., balance. The uncontrolled, or involuntary motor activity is usually first

observed in the arm or leg, but is also often present in the trunk, and seems like a compensatory involuntary motor response when the task is too difficult or associated with effort (too much effort for the needs of the actual situation), or in situations where the patient feels insecure, unstable, or unhappy—often connected with the fear of falling or the feeling of having too little control to cope. As time goes on, this involuntary recruitment of muscle activity may increase, it presents itself in more situations, is more easily triggered, and may develop into—for the individual patient—a stereotypical involuntary pattern of activity. The patterns are characterized by little variety, and may be activated progressively more easily in more situations to different degrees. In the literature they are often described as:

- Abnormal synergic muscle activity (Carr and Shepherd 1983)
- Abnormal movement or mobility (Shumway-Cook and Woollacott 1995)
- Abnormal movement synergies (Tyldesley and Grieve 1996)
- Spastic patterns of movement (Stokes 1998)
- Associated reactions (Bobath 1990, Dvir and Panturin 1993, Edwards 1996, Stephenson et al. 1998, Pandyan et al. 2005)

The author perceives these motor disorders to refer to the same aspect of motor activity, and the following text uses the term *associated reactions*. Previously, from a hierarchical model of the CNS, associated reactions were thought to be the release of primitive reflexes due to a CNS lesion (Bobath 1978). Research related to the structure and function of the CNS, plasticity and different aspects of movement science has shown that the CNS is not governed by reflexes as such in context-based movement and is not hierarchically organized, but it is a multidirectional, integrated system that is developed through the interaction of the individual with his environment.

Associated reactions are related to CNS reorganization post lesion, and viewed as an activity-dependent process of learning as the patient attempts to interact with the environment without the necessary prerequisites for motor control, i.e., as a result of altered behavior. If motor control is reduced generally or locally, balance is affected. Several studies support the view that a certain level of postural control is necessary for

independent movement, transfers and ADL (Masić et al. 1996, Shumway-Cook and Woollacott 2007, Ching-Lin et al. 2002, Verheyden et al. 2004). The ability to move to and from different positions (transfers and locomotion) and stabilize for extremity function is reduced.

Spasticity and associated reactions are linked to lesions of the cortico-reticulospinal and vestibular systems, which have an important role in postural control. Therefore one might assume that a patient's postural control will be reduced to some degree in an upper motor neuron lesion if they present with positive features. The patient therefore has to compensate for both reduced postural control and motor function. The (re)organization of the CNS is activity dependent, i.e., dependent on how the body is being used (form-function). On the other hand, motor control is dependent on the structure and function of the CNS and the integrated systems control, as well as biomechanical factors (form-function). Probably there is no *one* cause of the development of associated reactions. It is multifactorial and the exact mechanism varies between individuals. Possible mechanisms are:

- An upper motor neuron lesion causes a loss of descending commands to the spinal cord. Axons from damaged and dead neurons degenerate from their distal end and leave behind empty synapses at many levels in the spinal cord and brain; inhibitory mechanisms are disrupted.
- Neurotrophic substances stimulate collateral sprouting.
- Reduced or altered modulation of somatosensory input and denervation supersensitivity render the spinal cord more hypersensitive to peripheral stimuli.
- Networks in the spinal cord may be affected by spasticity caused by altered presynaptic inhibition, reciprocal Ia inhibition, recurrent inhibition and other factors (see Spasticity) due to altered supraspinal influences and plasticity.
- Changes in presynaptic inhibition may cause increased spread (spatial distribution) and repetition (temporal distribution) of action potentials causing more motor units and muscles to be recruited than normal for the intended activity.
- The Golgi tendon organ may lose its inhibitory effect on the motoneuron causing an altered

length-tension relationship, and as a result an increase in tension in the activated muscle/the activated motor units.

- Changed reciprocal Ia inhibition may cause tonic activity in a muscle to inhibit its antagonist and increase response to muscle stretch.
- Altered recurrent inhibition may render the motoneurons hypersensitive to stimuli and lead to repeated reflexive muscle contractions (clonus).
- Deviation from the Henneman recruitment principle may cause increasing divergence in the recruitment of motor units (disturbed temporal and spatial recruitment) leading to both a stronger activation and increased number of motor units recruited in the activity. The motor response is therefore more widespread and stronger.
- Reduced or altered sensation and perception of cutaneous and proprioceptive input may affect the patient's motor output.
- Altered neurophysiologic state cause altered behavior as a response to environmental demands. Altered muscle activation and function change postural alignment. Together these factors alter the input to the CNS, which secondarily alters the recruitment of motor activity.
- How the individual perceives his abilities and problems and interacts with the environment affects his motor behavior.

Plasticity, which in most cases is viewed as entirely positive because it causes learning, may be a negative contributor to the development of associated reactions.

Craik (1991) describes the following characteristics of normal motor behavior:

- Appropriate sequence of recruitment and tension development in muscles
- Adequate range of joint motion
- Adequate development of torque
- Efficiency
- Precision
- Success

The ability to adapt to different situations, tasks, and environments is crucial to the individual's appropriate interaction with the environment. If the above characteristics describe basic proper-

ties of normal motor activity, then clearly, associated reactions are viewed as pathologic. Associated reactions are characterized by:

- Gradual increase in stereotypical patterning
- Altered sequence of recruitment
- Increased tone
- Inadequate ranges of joint motion
- Altered production of torque
- Reduced precision
- Reduced efficiency
- Increased effort

The patient may still be able to attain his goal through other strategies available to him.

Associated reactions are a sign of deviant motor behavior as well as a positive sign of reinnervation and reorganization of the CNS. The patient may, however, still achieve his goal through alternative or compensatory motor strategies.

Associated reactions have been described as flexion or extension patterns, which might lead the reader to understand associated reactions as stereotypical patterning for a population of patients with upper motor neuron lesions. Clinically, associated reactions have individual expressions, but may have some common features:

- Triggering factors—antigravity activity, weightbearing, balance and transfers, fear of falling, requirement of selective control, coughing, sneezing, emotional factors
- The strength of the triggering factor—how much effort the patient has to put in
- Motivation and understanding—perceptual and cognitive factors
- General condition (fatigue, nourishment, infections)

There is no general consensus as to what degree it is possible to grade associated reactions or how useful this may be (see Stephenson et al. 1998 for discussion, Dvir and Panturin 1993).

In my clinical experience, grading of associated reactions may give information of the development of associated reactions and motor control over time. Important factors are:

- The patient's own control over the associated reactions. Although they are defined as involuntary there may still be some degree of volitional control.

- Triggering mechanisms: are they elicited and what are they elicited by?
- As a reaction to destabilizing forces to the center of gravity (interaction with gravity, postural control, speech, or breathing), i.e., reduced ability to adapt
- In anticipation to move (feedforward—loss of anticipatory contrast) or in an attempt to voluntarily move without the inherent prerequisites to do so (compensatory)
- How early they are elicited—sometimes the thought of moving (feedforward) is enough, or whether they develop during ongoing movement
- How long the reaction lasts
- How widespread the reaction is—how many joints, muscles, body segments are involved
- How strong the reaction is, i.e., force production
- How quickly the pattern develops or subsides (how long it lasts)
- How large is the excursion of movement

Clinically, associated reactions may be broadly graded into mild, moderate, and severe.

Mild Associated Reactions

- The patient may have some volitional control over the associated reactions.
- The associated reactions only present in stressful situations physically or emotionally.
- The associated reactions may be localized and only involve an increased muscle activity over one joint (e.g., wrist flexors).
- The associated reactions are transient and demonstrate small ranges of movement.

Moderate Associated Reactions

- The patient may have some volitional control over the associated reactions.
- They are often elicited at the thought of activity (feedforward), and may increase during ongoing activity.
- They may affect more than one body segment.

Severe Associated Reactions

- The patient rarely has voluntary control in the presence of severe associated reactions (in the same body segment).
- Associated reactions may be present even if the patient is inactive.
- Many body segments may be involved.

Clinical experience suggests that repetition of associated reactions cause a faster, stronger, and more uncontrolled response over time, and may become established as a part of the patient's movement repertoire.

The cause of this motor disorder is neurophysiologic: all the mechanisms ascribed to spasticity caused by a lesion to the higher centers. Associated reactions are what the clinician observe or perceive as involuntary, disorganized activity, an expression of the activity in the CNS and the interaction with external and internal demands. Associated reactions may lead to secondary changes in the muscles, connective tissue, skin, and alignment, i.e., *non-neural changes*.

■ Non-neural Changes

Many authors describe secondary muscular and connective tissue changes as part of a patient's motor disorder (Goldspink and Williams 1990, Given et al. 1995, Voerman et al. 2005, Wood et al. 2005). Some studies show that there may be a gradual transition of muscle fibers after an upper motor neuron lesion. Goldspink and Williams (1990) and Hufschmidt and Mauritz (1985) found that the type I fibers atrophy quickly through immobilization or altered use due to changes in the activation pattern. They also demonstrated that muscles that were originally more phasic partially transformed to musculature with more tonic characteristics. Dietz et al. (1997) state that spastic stroke patients demonstrate increased muscle atrophy especially of type II fibers. The researchers found an increased population of type I fibres in the gastrocnemius, which normally contains more type IIa fibres. Different studies have shown different results, depending on the patients and muscles studied, but they all do demonstrate that changes in muscle fibers occur as a response to an upper motor neuron lesion.

The unique way in which the skeleton and the musculature come together allows humans to effectively interact with the environment in a variety of ways. The several types of joint and grades of movements in different directions give an enormous variety to movement. The structure of the different muscles, their organization and ana-

tomic attachments allow for an optimal exploration of rotation. Postural control, balance, transfers, locomotion, and the use of the extremities demand interaction between body segments, and rotation is an essential component of this. Coordination of different body segments allows crossing midline while moving, balancing, and interacting with gravity. Changes in the working relations of the musculature due to changes in structure and function cause altered alignment, which negatively affects the ability of the muscles to work efficiently.

Changes in muscle and muscle fiber composition associated with spasticity are a result of neural and behavioral factors, i.e., plasticity and learning.

Altered use may cause changes in connective tissue, atrophy or hypertrophy of motor units, and altered muscle fiber composition. The patient therefore has a different anatomic configuration to move with: If a muscle is kept in a shortened position over time, sarcomeres may be lost; conversely a muscle kept in a lengthened position may stretch with increase in its number of sarcomeres. Both these conditions affect the ability of the muscles to be recruited and used appropriately.

Some muscles with more postural functions, e.g., the soleus, contain more connective tissue, especially collagen. If kept immobilized and shortened, the concentration of connective tissue may increase. Goldspink and Williams (1990) refer to trials where changes occurred after only 2 days of immobilization. These authors state that only half an hour of "stretching" every day may be sufficient to stop loss of sarcomeres and increased connective tissue. Goldspink and Williams (1990) suggest that intermittent stretching, which aims at reproducing normal activity, is probably better than just stretching. Muscles that are kept in a stretched position do not develop increased concentrations of collagen.

Yarkoni and Sahgal (1987) followed up patients with head injuries to assess factors contributing to the development of contractures. They found that patients who were comatose for more than 3 weeks had a significant increase in contractures, probably due to the period of immobilization. They also found that hemiplegic

patients have contractures on their less affected side. Hufschmidt and Mauritz (1985) state that it takes a year to develop changes related to muscle fibers and connective tissue after clinical manifestation of hyperreflexia.

When muscles change in structure and function and connective tissue loses its supporting function, changes in alignment ensue. Altered muscle structure and function combined with altered alignment further lead to changes in the use of muscles. There are fewer possibilities for variations and combinations, and the muscles adapt to their new area of use. This is in addition to the direct affects and consequences of the lesion itself.

A poor physical outcome is related to secondary musculoskeletal complications (Ada and Canning 1990).

It is therefore of utmost importance that the treating team analyzes which factors trigger the individual's associated reactions, his motor resources, and deviations. The combination of impaired balance and motor control together with demands for independent function may predispose the patient to develop associated reactions and secondary non-neural complications (Ashburn and Lynch 1988, Cornall 1991).

After injury it may be possible to direct or reinforce synaptic plasticity by appropriate manipulation of the system from the periphery to aid recovery of motor skills or cognitive function (Stokes 1998, p.70).

The CNS and neuromuscular system have a capacity for reorganization throughout life.

Clinically, it is important to differentiate between neural and non-neural components in this motor disorder. The interventions for gradual development of contractures differ from the treatment of the motor disorder as such, and may involve splinting, surgery, more rigorous positioning and a comprehensive management program.

Summary

Over-activity (*intense, specific stimulation*) should be performed with great care the first week post stroke. Clinical decisions should be based on the patient's level of arousal, alertness, blood pressure, intracranial pressure, and temperature. Later patients probably suffer more from too little activity. See page 58.

The negative impairments—weakness, loss of dexterity, and fatigue—seem to be the major factors that limit recovery of function in stroke patients. See page 59.

Spasticity is defined by Pandyan et al. (2005) as: "Disordered sensorimotor control, resulting from an upper motor neurone (UMN) lesion, presenting as intermittent or sustained involuntary activation of muscles." See page 59.

This definition includes changes in the structure and function of the CNS and excludes both the negative signs of an upper motor neuron lesions and the biomechanical changes in soft tissues and joints. See page 59.

Activity in cutaneous and proprioceptive pathways seems to have an important role in spasticity. See page 60.

Lesions to the cortico-reticular, reticulospinal, and vestibulospinal pathways are associated with spasticity. See page 61.

Plasticity, which in most cases is viewed as entirely positive because it is involved in learning, may be a negative contributor to the development of associated reactions. See page 62.

Associated reactions are a sign of deviant motor behavior as well as a positive sign of reinnervation and reorganization of the CNS. The patient may, however, still achieve his goal through alternative or compensatory motor strategies. See page 63.

Clinical experience suggests that repetition of associated reactions cause a faster, stronger, and more uncontrolled response over time, and may become established as a part of the patient's movement repertoire. See page 64.

Changes in muscle and muscle fiber composition associated with spasticity are a result of neural and behavioral factors, i.e., plasticity and learning. See page 64.

A poor physical outcome is related to secondary musculoskeletal complications. See page 65.

After injury it may be possible to direct or reinforce synaptic plasticity by appropriate manipulation of the system from the periphery to aid recovery of motor skills or cognitive function. See page 65.

The CNS and neuromuscular system have a capacity for reorganization throughout life. See page 65.

2

Physiotherapy

2.1 Balance and Movement	68
2.2 Intervention—Considerations and Choices	90
2.3 Other Interventions: Some Key Points	132

Introduction

Normal movement is complex. Many books have been written on the subject, and more recently there has been a lot of research in different branches of movement science. Movement science is the study of movement from different perspectives: for instance physiotherapy, psychology, pedagogy, physics, neurophysiology, biomechanics, and biologists studying human movement. It provides the basic knowledge of movement important for all therapists. Most research on movement, however, is still in its early days in that most of it is conducted in research laboratories with healthy, younger people, and therefore not always directly applicable to patients in a clinical setting. There is no international consensus on how this research could—or should—be applied clinically, and no consensus on intervention.

In this book, I aim to present physiotherapy from my own experience, knowledge base, and point of view, and as an advanced course instructor of the International Bobath Instructors Training Association (IBITA). The word *concept* means “something understood, an idea” (Taber’s Cyclopedic Medical Dictionary 1997) or it is “an element used in the development of a theory” (Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing and Allied Health 1992). Here, *concept* is applied to the knowledge base that clinical reasoning is based on. A conceptual understanding is therefore not learning a method, but learning to analyze and understand connections in an individual’s movement problems—*Why do they move*

in this way? The aim of this chapter is to establish an understanding of balance and movement, and to highlight some of the reasoning and choices therapists make in the treatment of individuals with central nervous system (CNS) lesions.

We now know that the CNS undergoes changes depending on what input it receives and the response to this input. There is a constant interaction between the individual and his environment that shapes the body and brain—plasticity is the bridge between brain and behavior. As a consequence, human movement is adaptable and humans have a capacity for learning, both with a healthy CNS and after CNS lesions. Therapists form an important part of the patient’s environment and training; treatment induces changes in cortical activation patterns over time (Nelles 2004). Therefore, theoretically, health professionals should be able to help the patient shape and adapt, to learn what seems appropriate and “unlearn” what seems inappropriate (associated reactions, inappropriate and inefficient motor strategies). Our aim should be to help the patient to develop and optimize their potential. But “We are currently being forced into interventions to ‘get the patient out’ that are supposedly cost effective. However, are we training compensatory strategies that will prevent true recovery, thus lengthening the time and increasing the level of care that the person will need in the long run?” (Held 1987, p. 174). This statement seems as true today as when it was written 20 years ago.

Health professionals face this dilemma; often we know what is possible but rarely have the re-

sources to see it through. We may limit the patient's potential; perhaps we lack optimism and vision and do not believe that the patient can improve? The goals are set at a level we can reach based on our resources (time, economy, competence) and beliefs, not necessarily at the patient's real potential for regaining function based on the localization and extent of the lesion, the patient's premorbid status, his general condition, and his plastic ability—his capacity to learn.

"First of all, we as therapists need to change our operating assumptions. We should expect recovery and work for that by preventing rather than encouraging compensation to occur. We should carefully analyse each person's problems, and we must intervene early" (Held 1987, p.173).

In rehabilitation, goals are often related to the following. The patient should, as much as possible be able to:

- Participate in his life, fulfil his roles
- Master activities
 - daily functions
 - function in his own environment
- Control balance, movement, and function:
 - improve postural control and selective movement
 - regain the ability to interact with the environment
 - control the recruitment of tone. Learning through early and gradual interaction with gravity with good alignment to optimize muscle activation and with support or facilitation where and when it is needed
 - develop appropriate strategies and patterns of movement
 - develop appropriate strength to interact with gravity
 - maintain muscle length and range of movement

Physiotherapy-related goals are set by the patient and therapist together. The patient's needs and goals and therapy goals must be in harmony. The therapist's challenge is to increase the patient's competency, insight, and understanding for a process leading to greater participation and independence, and this takes time. Treatment for many patients will first and foremost be aimed at improving selective postural control and balance in functional situations to strengthen carry-over to daily activities in the patient's own environment. If the patient's postural control is regained to a level where he is able to take more part in his environ-

ment, be more oriented toward and integrated with the world around him, he will, in my understanding, have greater opportunities of participating in a variety of social settings.

People are different: interests, wishes, goals, needs, previous experiences, what we like, who we like ... To be able to create a constructive patient-therapist relationship, the therapist has to adapt to many different individuals and meet each one with a positive attitude, empathy, professionalism, and respect for the person's integrity. It is important to create a good learning environment to motivate and inspire the patient, and at the same time give realistic information without taking hope away. Evidence supports the theory that the patient should be exposed to an enriched environment to develop his potential for learning (Virji-Babul 1991). Motivation and focused attention are important factors for learning (see *Plasticity*). Assessment and treatment build on:

- Analysis of "normal" movement
 - Analysis of deviations from "normal" movement
 - Clinical reasoning
 - Appropriate treatment techniques to facilitate the patient's regaining of motor control
- The following sections will focus on:
- Balance and movement
 - Conceptual understanding for treatment rationale and choice of individually adapted treatment techniques
 - Other forms of treatment/interventions

2.1 Balance and Movement

Human Movement Control

When we study humans in movement we are able to recognize whether they are sitting, standing or walking, standing up or sitting down, or turning around "normally," because we all perform these activities in a basically similar way. We recognize when people have good balance, and observing their movement strategies enables us to recognize common features in the average population, which define our genotype. There are many common aspects to how individuals problem-solve a movement task:

- Appropriate sequence of recruitment and tension development of muscles
- Necessary range of movement in different single joints
- Necessary ranges of movement between the extremities and the torso
- Appropriate development of torque
- Appropriate alignment, which is essential for the sequencing and selective activation of muscles
- Appropriate effort—no more than needed for efficiency and successful goal achievement
- Rhythm, tempo, variation

Basic components of movement that are coordinated in time and space have to be activated for us to interact with gravity and use our arms functionally at the same time. Our daily activities demand both postural and movement control. Which components are most important depends on the person, the goal, the environment, and the actual situation. Each individual has his own, personal way of moving, the individual expression—the phenotype. We recognize a person by the way he moves. It may be enough to hear the steps in the hall: rhythm, step, tempo, firmness, light, hard or shuffling steps—all characteristics of the individual. The movement expressions may even reflect the person's state of mind: the extended self-assured type or the flexed, modest insecure or anxious person are two extremes.

Normal movement is varied, without inappropriate effort, efficient, effective, precise, and successful, developed through the interaction between the person, the task, and the environment.

The expression *normal movement* reflects both the common features of humankind and the individual expression. A person's build and posture inform the therapist of his movement experience and how he has used his body before. Through analysis of movement, the individual expression is evaluated, as well as how movement is performed in relation to the goal activity, the environment, and the ability to vary during different activities in different situations. Analysis of movement involves both observation and handling (hands-on) during activity, with special focus on:

- Balance
- Postural control
 - postural activity, build, and posture—postural tone
 - the relationship to gravity and to the environment, the base of support
 - coordination, reciprocal innervation
 - the relationship between body segments and their function (stability-movement)
- Coordination of the individual components in sequence, time, and space, i.e., the recruitment of movement patterns
- The selective movement of single components

Balance

Balance is the result of the interaction between motor, sensory, and cognitive processes. Many routine activities, for instance balance, walking, reaching for something, or dressing demand none or very little effort or cognitive awareness. Balance is the foundation for our daily activities. It allows us to be stable and active in relation to gravity and the base of support whilst at the same time using our arms—which are free—for functional activities. Balance is movement as well as a prerequisite for movement. Through movement we interact with the environment and learn to perceive ourselves in relation to the world around us. Balance is a holistic sensorimotor and perceptual interplay between our surroundings and us, and requires graded and coordinated neuromuscular activity of the whole body at the same time.

Balance may be expressed as a *reaction* when displacement occurs suddenly and unexpectedly, or a *strategy* when we right ourselves, transfer weight, turn around, or step. *Strategy* refers to cognitive processing because it involves the organization and sequential planning of a goal-directed movement. *Strategy* should imply the existence of choice. That is, the goal should be attainable via different ways (Windhurst et al. cited in Mission 1992).

- *Strategy* involves a level of processing, problem solving, and planning (feedforward).
- *Reaction* defines the response of an organism, or part of it, to a stimulus (Taber's Cyclopedic Medical Dictionary 1997). Reactions are the result of external influences (feedback).
- *Balance* contains both these elements: It is a reaction to displacement at the same time that

movement is planned in relation to the task and the environment.

Examples

Many studies have been carried out to explore where muscular activity is recruited first in standing and walking. Humans are normally able to use different strategies when displacement occurs. The strategies depend on the situation; the sequence of muscle activation varies in relation to needs and possibilities. The response varies depending on the feet; if they are free to move or kept stationary when displacement occurs, whether the base of support is smaller or larger than the feet, how displacement is given and if the study participants are instructed to stay still or are allowed to move.

In a free-standing position on a stable base of support, research suggests that motor activity is first recruited in relation to the base of support, i.e., the foot and ankle, lower leg, then more proximally, the arms coming last (Nashner 1982). Several authors who call this the *ankle strategy* support this concept. The ankle strategy opposes body sway in standing, and is based on a distal-to-proximal activation (Horak and Nashner 1986, Rosenbaum 1991, Rothwell 1994, Shumway-Cook and Woollacott 2006).

If the base of support is smaller than the feet, for instance in standing on a narrow ledge, the displacement is fast or large, or the base of support is pliable; the ankle strategy may not be sufficient to maintain balance, and the *hip strategy* is used. The hip strategy activates lower trunk-, pelvic- and hip-related musculature first, i.e., a more cranial-caudal (proximal-distal) sequence of recruitment. More recent research has found that in many conditions, stepping occurs even if the line of gravity is within the base of support, when balance is not threatened (Maki and McIlroy 1997, Shumway-Cook and Woollacott 2006).

Prince et al. (1994) studied balance control in the upper body during locomotion. Their results indicate that hip extensors and the erector spinae muscles are activated immediately after heel-strike, which is the initiation of stance phase. Hip extension stabilizes the pelvis shortly after heel-contact, while the main role of the paraspinal muscles is to oppose trunk flexion (Fig. 2.1). The balance of the upper body in relation to the pelvis and hips shows a cranial-caudal activation to stabilize the head first, then segmentally to the pelvis. When the arms swing during normal walking, the musculature in the region of T2-T6 is especially active.



Fig. 2.1 Rotation between the body segments during walking. The rotation occurs throughout the spinal column, but mostly in the mid-thoracic region. The pelvis and shoulder girdles rotate, and the extremities rotate in relation to their proximal attachments and the base of support. The line of gravity seems to be falling between the model's feet.

The trapezius muscle assists in stabilizing the shoulder at the end of each arm swing forward.

Normally, most people can adapt to the actual situation. Balance provides the body harmony and safety in relation to the environment, and is the foundation of our motor system. With reduced or no balance we have to use other strategies to prevent falls. Patients with neurologic conditions have lost some of their movement repertoire and are unable to adapt to the same degree as before. Therefore they have fewer choices open to them if balance is threatened.

In my understanding, *balance* is a holistic term encompassing:

- Postural control
- Righting
- Protective reactions

Postural control, righting, and protective reactions represent three lines of defence and are closely integrated in normal balance and movement.

■ Postural Control

The consequence of this specific behavior (humans' ability to stand and walk) is that the nervous system automatically has to balance the body's center of mass over the feet during all motor activities performed in a bipedal posture: every movement necessarily begins and ends with a postural adjustment (Dietz 1992).

Postural control develops through the interaction of sensory, perceptual, cognitive, and motor systems as well as the musculoskeletal apparatus of the body. The desire to move combined with information received and modulated from all receptor organs and senses of the body forms the basis for execution of movement. Movement is constantly adapting to variations in the environment, goals, and situations. Postural control is recognized as a key component in the acquisition of independence in activities of daily living (ADLs) and IADLs (instrumental ADLs, for instance the ability to go shopping) (Hsieh 2002).

Postural control depends on a complex interaction of afferent information and motor activity. Humans use multiple sensory references, gravity (the vestibular system), contact with the environment (somatosensory systems), and the relationship between the body and objects in the environment (vision), to maintain an appropriate relationship between body segments for balance. It appears that under normal conditions, the nervous system may weight the importance of somatosensory information for postural control more heavily than vision and vestibular inputs (Shumway-Cook and Woollacott 2006). There is a continuous adjustment of postural tone before, during, and after every movement. Anticipatory postural control is the pre-tuning of sensory and motor systems in expectation of postural demands based on experience and learning (Shumway-Cook and Woollacott 2006). Anticipatory postural adjustments are active before movement starts. Postural control in sitting and standing allows us to maintain activity against gravity. This provides us with a vertical orientation, an antigravity activity with alignment of body parts as a reference frame for balance, movement, and orientation. Muscular activity on all

sides of the trunk is balanced simultaneously to allow selective, segmental extension of the spinal column and to free the arms for functional use. Abdominal muscle activity is important as a stabilizing factor for the segmental extension. Postural control has individual expressions.

The nervous system seems to weight the importance of somatosensory information for postural control more heavily than vision and vestibular inputs under normal conditions.

Equilibrium reactions are defined as automatic reactions that serve to maintain and restore balance through all activities (Bobath 1990). Edwards (1996) states that equilibrium reactions are synonymous with postural adaptations present in all daily activity. She uses the term *equilibrium control* to describe the ongoing segmental movements that compensate for displacement of the center of gravity caused by the actual movement or activity (Edwards 1996, refers to Massion 1992).

The author understands *equilibrium reactions* and *equilibrium control* together as synonymous with *postural control*. The postural activity seems to be a result of changes of tone—a functional adaptation, i.e., altered distribution of activity in different motor units for the maintenance of stability, like ripples in water. We see the movement, but we don't see the postural control. We see the activity, but we don't see the stability. Postural control is like the part of the iceberg under the water, movements are like the small part above the water. We can see postural responses only in the feet, especially when balancing on one leg.

Skilled movement has both postural and voluntary components: the postural component establishes a stabilizing framework for the second component the primary movement. Postural control is central to all human activity and adjusts continuously during activity. The aim is to maintain equilibrium during functional activity. It gives us stability together with an orientation to the environment. Postural muscles of the trunk, pelvis, and shoulder girdles contract before the arm is lifted in sitting or standing in order to stabilize the body by minimizing displacement of the center of gravity (Morris et al. 1994). Trunk control is only a small part of postural control;

when the hand is used the wrist and palm are positioned to stabilize for individual finger movements. Postural control allows us a dynamic adaptation of background activity and is a prerequisite for dynamic alteration of activity in the extremities and therefore for selective movement.

Clinically, reduced postural control seems to be one of the main problems for many patients with CNS lesions. A loss of a stable reference frame as a basis for voluntary movement causes balance problems, reduced coordination and interaction between muscle groups, as well as a reduced tempo and ability to react. Postural control counter-stabilizes movements of the extremities; if the body is not kept stable as a person reaches out in space, his body moves with the arm and he is destabilized. As a consequence, the person's choice of how to solve different motor tasks becomes limited, which again may reduce his ability to be independent and master daily activities. Postural control and precise, goal-oriented movement are not separate phenomena in the CNS; they are coordinated together to allow successful achievement of motor tasks in a context-based environment.

■ The Function of Postural Control

- Maintain the alignment of posture—different alignments for postural variations in different situations
- Adopt a vertical relationship between body segments to oppose gravity
- Create the posture of being upright—where grading of postural (muscle) tone is fundamental to this function
- Control the position of the body in space for orientation and stability
- Create a stable reference frame for the extremities and the head

Postural control represents the most automatic activity in human movement (Maison 1992 and 1994, Mulder 1991 and Mulder et al. 1996), although it is probably less automatic than previously thought. To be able to maintain postural stability demands a certain level of attention, and studies have demonstrated that attention demands increase with increasing balance demands (Hunter and Hoffman 2001). Simultaneous demands for cognition and balance increase the tendency to fall in elderly people (Brown et al.

1999); dual tasks demonstrate the same relationship (Mulder et al. 1995). The combination of a cognitive task and a balance task result in a poorer solution of the cognitive task if it contains spatial elements (Brodal 2004).

■ Postural Tone

In the following section the term "postural tone" or "tone" will be used instead of "muscle tone" to emphasize that the CNS activates many muscle groups for the maintenance of postural activity. Shumway-Cook and Woollacott (2007) describe postural tone as an increased level of activity in antigravity muscles.

The most important factor in altering the level of tone is muscular contraction (Brodal 2001). Tone gives an automatic background for activity and may vary from moment to moment. It needs to be high enough to withstand the effect of gravity and at the same time low enough to allow dynamic adaptation through small movements. Tone changes and adapts in relation to movements and the activities we perform. Postural tone has a wide spectrum in normal movement: it is normally at its lowest in relaxed supine, when the base of support is large and the center of gravity low. It may change in a millisecond as when we jump out of bed to run to the bathroom. In normal daily activity postural tone is at its highest in toe-off in stance phase, just before weight transference to the opposite heel. Humans may grade activity in the postural system as needed and through experience and exercise. The ballet dancer who dances on tiptoes or the tight-rope walker are examples of postural activity on a much higher level than needed for ADLs.

Tone is highest in those areas of the body where the need for maintaining and sustaining activity against gravity is highest, and varies in relation to the base of support and the effect of gravity. There are individual differences in postural tone, some people have a naturally higher level of tone, and others have lower tone. Psychologic factors may play a role; people who are anxious often have a higher tone than those who are laid-back.

Gravity acts on the body at all times, whether at rest or during activity. The level of activity in different muscles depends on the orientation of the body and the extremities to gravity and base of support, the relative relationship between body

segments, the activity to be performed, and the environment and experience.

All ADLs demand that we relate to and oppose gravity, from turning over in bed, shifting our position, turning over pages in a book, and reaching out to turn off the light in bed, to initiating head movement for sitting up, getting dressed, and managing the bathroom, lifting a fork to eat, writing a letter, running after the bus, or walking in the mountains. The musculoskeletal system is constantly adapting to modify activity during changing relationships. Outdoors, even the weather and wind act on our bodies and demand postural adjustments.

Descriptions that may seem artificial in relation to movement science on healthy people may seem logical in clinical situations. *Against gravity* is a term used in the treatment of neurologically damaged persons. Acutely after stroke, a spinal cord injury, or relapse of multiple sclerosis

(MS), many patients suffer from severely low postural tone (Fig. 2.2) and are unable to activate or sustain activity against gravity and therefore also have problems moving.

The *base of support* is the contact area between the body and the environment: the feet in standing; the thighs, buttocks, or back (if there is a backrest on the chair) in sitting; and the contact area between the hand and an object. The quality of the base, its size, material, softness, temperature, and the distance between the base of support to the center of gravity, are all factors of importance in setting the level of postural tone and our motor response: we lie down to rest and sit, stand, or walk when we want to be active.

These factors are not all-important, however; postural tone is not always low in supine. Most people will recognize the situation in the dentist's chair: if you are uneasy or nervous, your postural tone is higher. In this situation, adapta-



Fig. 2.2a



Fig. 2.2b

Fig. 2.2a, b The patient has had an acute stroke.
a It seems as if he is either falling or pushing himself up with his right arm. He has very low postural tone and balance. He is unable to stay upright and interact with gravity.

b He compensates by increasing his flexor activity in the right side of head, neck, and trunk in an attempt to pull himself up. Notice his right foot in both a and b, which seems to push against the floor and footrest. There is very little activity in his left side.

tion to the contact area is not appropriate, and you may have a feeling of being suspended in the air above the chair through muscular tension instead of resting *in* the chair. If a person sits uncomfortably, too still, unable to adjust or change position, postural tone may increase.

Because recovery depends, to a large extent, on the ability of the CNS to adapt to (peripheral) changes, the study of recovery is largely the study of adaptation (Mulder et al. 1996).

Recovery largely depends on the ability of the CNS to adapt to (peripheral) changes.

The interaction with the environment demands a constant adjustment of tone, alignment, balance, and movement. This interaction depends largely on the ability to receive and perceive information from peripheral receptors and to adapt and adjust in response to this information. Our initial evaluation of conditions of the base of support is based on tactile information from base of support (BOS): feet (soles), bottom, and hands. We evaluate the support conditions by tactile contact. Our next calculation is of inertia; a smaller support means that inertia is more easily overcome and more quickly evaluated and assessed. The larger the estimation of the tactile area, the more difficult it is to overcome inertia. The relationship between the body and the base of support is the foundation for adjustment of tone to varying environments and activities. Tone needs to be optimal for the actual activity, whether it is to relax, to initiate movement, balance, or perform functional activities.

In a functional context, the sensorimotor and perceptual interaction between the body and the base of support is more important for the level of postural tone than the size of the base of support.

In standing and walking the feet need to be mobile and continuously adaptable to the environment. The feet transmit information directly between the body and the environment and must *adapt* and *respond to* the base of support and not *react to* the base with inappropriate tension—pushing or clawing—for balance to be optimal.

The feet are our base of support, our foundation, in standing. They are sense organs that communicate the information they pick up about the base of support—unevenness, texture, hardness, direction, incline, and the distribution of body weight—to the CNS. The structure of the foot determines which rotatory components are available in the foot and leg during walking and running (Nawoczenski et al. 1998), i.e., patterns of movement. Alignment and neuromuscular activity in the feet have a significant influence on the alignment and neuromuscular activity in the rest of the body (Ljunggren 1984, Thornquist 1984). Eline Thornquist (1984) states that the load and tension condition of the feet influence our way of moving and determine to a great extent the movements available to us. She uses the words interplay, balance, and interdependence to describe the relationship between our foundation, physical and psychologic factors, and the environment. Changes in alignment and neuromuscular activity proximally will influence the ability of the feet to function as balance organs.

In the CNS there is widespread integration of somatosensory information and motor activity. Afferent input via both the somatosensory systems and vision informs us of where we are in relation to the environment and is therefore vital to our ability to adapt to the surroundings and to a variable base of support. When we move through the environment, vision is important in setting the level of neuromuscular activity through feedforward planning (Wade and Jones 1997). If we are able to see, we scan the environment to pick up information about our surroundings, and alter direction or speed if appropriate. Vision does not direct the placement of the foot in detail under normal circumstances. If the terrain is uneven or difficult, vision becomes more involved but has no role in the actual adaptation and adjustment of the foot to the base. The feet respond immediately to unevenness and changes. When standing on a mobile base—in a bus, on a train or boat, or on an unsteady rock—there is a flood of information from the feet to the CNS about the properties of the surface and the line of gravity in relation to the feet. Postural tone is automatically adjusted to the situation and the goal. This adaptation is in response to information received through the somatosensory systems, i.e., feedback. Activity—the interaction with

gravity—demands continuous interplay between feedforward and feedback.

When the hands are in contact with objects or directly with the environment (surfaces), either to handle and manipulate objects or to seek support or reference, they need to interact, adapt, reach, and let go, and vary their activity in relation to the task. Neuromuscular activity, sensory information and alignment distally (i.e., in hands and feet) influence the activity and alignment in more proximal areas and reversely.

Reciprocal innervation is the coordination of muscle activity for efficient, harmonious, rhythmic, and smooth movement without involving more effort than is appropriate for the actual activity (Bobath 1990, Edwards 1996). All complex movements are a result of finely graded interaction between external forces (gravity, inertia, and passive, biomechanical characteristics in involved structures) and variations in the tension–length relationships in the different motor units in agonist, antagonist, and synergic muscle groups. Reciprocal innervation tunes and balances muscle action, and I understand this to be the same as that which Sahrmann (1992, 2002) calls muscle balance (see Chapter 1.1 The Neuromuscular System). Reciprocal innervation is described as the harmonious interplay in and between muscles, i.e., coordination between eccentric and concentric muscle activity leading to selective control of movement. Reciprocal innervation involves:

- Differential activation of motor units in a muscle
- Coordination of different muscles surrounding a joint—agonists, antagonists, and synergists—i.e., eccentric and concentric interplay
- Coordination of different body parts, right and left, proximal and distal, through neuromuscular activity

Neurophysiologically, the recruitment principle is an important element of reciprocal innervation; the motor units involved in an activity are sequentially recruited and modified through pre-synaptic inhibition (see Chapter 1.1, The Neuromuscular System and The Spinal Cord and Brain). Reciprocal innervation is the foundation for stability, selectivity, and coordination in normal movement.

Postural stability is not related to the trunk alone, even if the trunk is essential for postural control. Postural control is a basis for all compo-

nents of movement. For instance, when changing a light bulb above your head, the whole body needs to be stabilized and oriented toward the manipulation of the bulb into the socket while the hands, wrists, and forearms are the main components of the function of rotating the bulb into place. Inherent in the term *postural control* are its *dynamic characteristics*. The body is multi-segmental with lots of rotatory, translatory, and multidirectional joint movements and muscles that need to be controlled. Bernstein called this the *degrees of freedom problem* (Bernstein 1967 in Shumway-Cook and Woollacott 2007).

Some activities may push stability to the limit and beyond, and necessitate a change of base of support, e.g., taking a step. Some tasks will focus on attaining the goal rather than maintaining stability limits, e.g. the keeper who throws himself to catch the ball, while the fall itself is as controlled as possible. Stability is a result of forces being balanced in relation to each other or equal to each other. In normal movement, stability is always dynamic, i.e., movement also occurs in stabilizing segments. Even as we stand still, there is a segmental adjustment through the body. The ability to hold a position demands continuous adjustment of neuromuscular activity. Stability allows movement between body parts. To reach out for an object, the body must stabilize and stay, then gradually move with the arm to reach further if necessary. As we reach, the hand initiates the actual movement of the arm while feedforward mechanisms (i.e., anticipatory postural adjustments) stabilize the body. During forward locomotion, the neuromuscular activation of the hip and pelvis has to maintain the stability of these segments at the same time as we are moving forward in space, and the hip joint rotates and changes from flexion to extension. As we reach swing phase, the activity changes; tone is reduced to allow a free swing. Stability areas vary, depending not only on the function, but also on all phase changes of movement, i.e., continuously. Therefore, mobility is essential for stability, as is stability for movement. The trunk may stabilize for movements of the extremities, the extremities may stabilize for trunk movement, the upper trunk may stabilize for pelvic mobility, pelvic area for trunk movements, the right side may stabilize for movements of the left half of the body, and so on. Even distally, the lower arm must be stabilized for movements

of the hand and the wrist for the movements of the fingers. The same is true for the lower leg and foot. Stable areas of reference adapt and may change through the evolution of movement.

Mobility is essential for stability, as is stability for movement.

Selective movement is understood as the controlled, specific, and coordinated movement of one joint or body part in relation to other segments. Selective movement is the result of precisely graded neuromuscular activity based on reciprocal innervation.

Stability and selectivity are both dependent on adequate ranges of motion, muscle length, alignment, and the coordination of agonists, antagonists, and synergists in concentric and eccentric work. Eccentric muscle activity is the result of active, neurophysiologic processes. Reciprocal innervation leads to the ability to:

- Automatically adapt muscle activity for postural adjustments
- Stabilize for selective movement
- Grade the activation and interaction of agonists, synergists, and antagonists for precision in timing and direction

Postural orientation involves:

- The ability to maintain an appropriate alignment between body segments
- The ability to maintain an appropriate relationship with the environment
- The need to establish a vertical orientation to oppose gravity
- Creating a reference frame for perception and action

Dexterity, i.e., flexibility and skill in movement (not only distal, manipulatory skills; see Chapter 1.3, Upper Motor Neuron Lesion) enables adaptation to the here and now situation, and depends on postural control. Postural control is the basis for all voluntary motor skills (Massion and Woollacott 1996).

Information that is needed to maintain postural control is crucial for conscious awareness of our own bodies, spatial orientation, and internal models (Brodal 2004). *Internal models* are the stored information needed for specific activities such as walking down stairs or reaching for something in space (see Chapter 1.1, Cerebellum, Clinical Hypotheses). Neural networks in the cor-

tex coordinate the different sensory modalities and give them meaning. Postural control therefore creates a reference frame for perception and action in relation to the world around us (Brodal 2004).

Body schema may be described as an internal postural image that informs us of the position of the body segments in relation to each other. It therefore provides a foundation for the exploration of space—the environment—for perceptual analysis and motor action. It is monitored by multiple sensory inputs. We need detailed information from all our sensory receptors to create and continuously update a body schema in order to balance and move:

- Information from proprioceptors:
 - The muscle spindles—la afferent input is necessary for the anatomic relationships of the different body parts, i.e., activity. Studies imply that information from the hips and the trunk itself is important to trigger a person's balance corrections. Proprioceptive information from the lower legs aids in the final adjustment and intramuscular coordination of postural programs and locomotion patterns (Allum and Honegger 1998).
 - The Golgi tendon organ measures the number of active motor units at a given time in each muscle used in postural control (Massion and Woollacott 1996).
 - Joint receptors
 - Skin receptors
- The ability to evaluate the base of support:
 - Low threshold mechanoreceptors (pressure receptors) in the soles of the feet
 - Plantar cutaneous information (Kavounoudias et al. 1998)
- The probable presence of graviceptors in internal organs:
 - *Graviceptors* are specialized sensory receptors detecting displacement of weight in relation to gravity, for instance the weight of shifting fluid in the intestines and kidneys. Recent research has argued for a separate pathway in humans for sensing body orientation in relation to gravity (Karnath et al. 2000a).
 - There is preliminary evidence to suggest that somatic gravity receptors, originating in the trunk, exist in monkeys and humans (Di Fabio et al. 1997).

- The graviceptors may monitor the force vector exerted at each joint to oppose gravity, and this information contributes to an internal representation of the vertical axis.
- Information from the vestibular apparatus in the inner ear that senses the orientation, movement, and acceleration of the head in space in relation to gravity
- Vision detects movement and monitors the displacement of the head and body in relation to the environment

Information reaching the central networks for postural control must be modulated, evaluated,

and acted on. Postural control requires good alignment (biomechanics), appropriate muscle strength, and all the above-mentioned information to be appropriate to the activity of the moment.

Patterns of movement are sequences of selective movements that vary from person to person, the task at hand, and the situation (Figs. 2.3 and 2.4). Movements are the output of a dynamic interaction between muscular forces and peripheral field effects (e.g., gravity, friction, joint reactive forces) and can be described in terms of their pattern, displacement, or topology (Mulder et al. in Harrison 1995). The motor system has



Fig. 2.3

Figs. 2.3 and 2.4 Compare the patterns of movement in the two pictures.

In Figure 2.3, the patient's center of gravity is to the right of his right leg, and he presses actively (down on his walking stick). Therefore, the flexor activity in his right side is increased at the same time as he seems to lift his left side forward with his right. There is decreased mobility and interplay between body segments generally, but especially proximally. The patterns of movement in his arms and legs are stereotypical. Rotation in his left leg does not change to swing the leg forward. There is reduced dorsiflexion



Fig. 2.4

in the foot. This could be caused either by stiffness of his calf muscles or weak dorsiflexors, or malalignment of the whole leg negating the activation of dorsiflexion. Figure 2.4 shows a healthy young woman who is taking a step forward with her left leg. Notice the extension over the standing leg and the interplay between body segments. On the swinging side the pattern of movement changes from stance phase activity (more extensor/abductor/external rotation activity) to let go of the same activity through rotation to swing the leg forward. The left dorsiflexion ensures clearance of the floor.

to contract the agonist with the right force at the right time as well as time and organize the contraction patterns of the antagonists, synergists, and postural muscles necessary for the agonist in the actual function. The number of muscle fibers and motor units recruited in a muscle varies depending on the function and other muscles that are recruited in relation to the task. The anatomic construction of the skeleton enhances coordination and rotation between the different body parts. The muscles cause rotation to varying degrees depending on the anatomic form and arrangement of the muscle fibers, their function, and the organization of different parts of the muscle to each other (alignment) and their attachments. Examples are the tendo-Achilles that is rotated 90° before its attachment to the calcaneus, and the pectoralis major that is rotated 180° before its attachment to the humerus.

Rotation is an essential component of normal movement, and it gives enhanced proprioceptive feedback to the CNS compared with, for instance muscle palpation or tendon tapping. Humans move along three planes: sagittal, frontal, and transverse (or horizontal), and this is combined through rotation. All weight transference and movement demand rotational components. We vary between symmetry and asymmetry caused by interplay between body segments through rotation. Rotation is based on the ability to grade and combine flexor and extensor components in infinite variations of movement patterns, and gives flexibility and resilience. Rotation, for instance between the upper and lower arm and between the thigh and lower leg, in relation to their proximal segments, and through trunk rotation cause a functional diversity that allows anything from fine motor activity and distal dexterity to coarse grasping and locomotion.

Basic patterns of movement are *reach* and *grasp* and *stance* and *swing*. These patterns vary infinitely depending on the task and the environment. *Reach* and *stance* are both patterns that are mainly dominated by components of extension, outward rotation, and abduction. *Grasp* and *swing* have more flexor components to their patterns, often with components of outward rotation and adduction. When the arm is by the side, the hand is brought to the mouth through flexion/adduction/external rotation initially changing to graded internal rotation of the shoulder, flexion of the elbow, and supination of the forearm.

The patterns interchange and vary in all our activities: we position ourselves in more extension if we need to reach out for something above shoulder height, when performing activities that demand fine eye-hand coordination we often chose to sit in a more flexed position—based on eccentric trunk extensor activity. Normal postural tone and normal reciprocal innervation allow individuals to choose their own background neuromuscular activity as is most appropriate for the situation. Normal postural control allows us to:

- Oppose gravity
- Move
- Develop functional skills

Righting

Righting refers to observable movement between body segments in relation to each other and to the environment, and happens when the line of gravity moves toward the limit of the support surface. The movements that represent the ability for righting are part of our most automatic balance control as well as being voluntary. Activities such as turning over in bed, changing direction in walking, sitting up, or standing from sitting are examples of righting. A person rights himself in relation to the task, environment, and the effect of gravity (Figs. 2.5–2.7). There is a continuous adjustment of muscular activity to maintain equilibrium at the same time as righting occurs.

There are two main forms for righting:

- Head righting—the head rights itself on the trunk as a response to displacement and aims to maintain the vertical position of the head.
- Trunk righting:
 - When the trunk moves in relation to the base of support. All weight transference and positional transfers require changes and adjustments in activity between the shoulder girdles, the thorax, and the pelvis.
 - When we turn our head to look about or change direction, the body follows and rights itself in relation to the movement of the head.
 - When we sit up the head leads the sequence of movement. The abdominals stabilize the thorax and allow the neck flexors to take the weight of the head. The trunk moves and follows the head until the



Fig. 2.5a



Fig. 2.5b

Figs. 2.5a, b The two figures show the interplay between righting and postural control as the arms reach up. Thoracic extension enhances the full range of movement at the shoulders and allows a further reach.



Fig. 2.6 Postural control, righting, rotation, and weight transference interacting in the function of fetching a book. Thoracic extension, based on core stability allows postural control and rotation.

center of gravity is within the new base of support in sitting. The first part of this movement is trunk righting, as the new base has been acquired head righting follows.

Righting is an essential and basic component of the ability to move from one position to another: weight transference, transfers, changes in the direction of movement, and for the development of protective reactions and movement strategies. Righting is therefore essential to all our functional activities.

■ Protective Reactions and Strategies

If righting is inappropriate or inadequate to maintain balance, a person takes a step or uses his arms to protect himself from falling. When we step to avoid falling, and if the displacement is very sudden and the feet are free to move, anticipatory postural adjustments may not always be present (Maki and McIlroy 1997). Steps in this situation are more of a reaction, but elements of planning and strategy are present in the background at most times: we place the foot in the direction in which we are likely to re-



Fig. 2.7a



Fig. 2.7b

Figs. 2.7a, b If the trunk is flexed, the center of gravity falls to the back of the base of support. As the arms reach above the head without the trunk righting itself, the reach is more limited.

gain balance. However, when we step to initiate locomotion, we plan ahead and initiate feedforward strategies. Stronger cognitive elements are now present, and this is therefore not a protective reaction.

The arms are recruited in a protective reaction if stepping is not possible or inadequate. Often some elements of planning will be present: we place the arms where they may most appropriately save us or lessen or grade the fall.

Balance is expressed in both reactions and strategies. Postural control, righting, and protective reactions are elements of balance.

The *midline* is a term that is frequently used clinically. It is a broad term that is difficult to define precisely. *Midline* refers to the interplay of body segments and alignment, and involves both physical and perceptual factors, like body schema. We explore and adapt to our environment, and action requires perception, balance, and movement. We have to perceive our body in space to adapt to the environment. The term *midline control* refers to both an ability and an experience of being balanced.

Clinical Relevance

It seems appropriate to distinguish between postural control and righting because they seem to be functions of different systems, and may therefore guide particular therapeutic interventions. Postural control is not under direct cortical control. There are indirect connections through the reticular formation and the cerebellum. Postural control can therefore not be regained through verbal instruction. Postural control requires segmental displacement and interaction with gravity. Therefore, the therapist should ensure that the patient has segmental mobility, for instance of the spinal column. Righting seems to be a result of activity in many interacting systems: cortical, cerebellar, vestibular, reticular, and spinal. Therefore it can be influenced both through verbal instruction and handling (hands-on). Postural control and righting are the foundations for head control.

Deviations from Normal Movement and Balance Control

Patients with CNS lesions are heterogeneous and show individual characteristics; lesions in a similar location and of a similar size in two patients will manifest differently. Patients have different personalities, talents, and experiences that have shaped their body and mind. Some patients may present with other medical diagnoses as well, such as diabetes and cardiac problems. Some patients may be in poor general condition for different reasons. The aim of treatment is to enhance the patient's potential as much as possible, keeping this heterogeneity in mind. Many patients will not reach the same level of function as before the lesion and will have to compensate to be functional within their environment.

Shumway-Cook and Woollacott (2006) define compensation as behavioral substitution, that is, alternative behavioral strategies that are adopted to complete a task. The two terms *compensation* and *compensatory strategies* are perceived to mean the same.

In the acute phase there is often seemingly severe paresis or paralysis after an upper motor neuron lesion. The CNS is vulnerable in this phase (see Consequences and Reorganization after CNS Lesions); the concentration of neurotrophic substances increases and facilitates the formation of new connections. We learn by doing, and the patient's CNS quickly learns new strategies that may seem appropriate for the moment. What the patient is stimulated to do, or required to manage do to by people, himself or the environment, drive the formation of new connections. Many patients are required to master independence in activities of daily living without having the postural or movement control needed to do so. When the patient sits alone on the side of the bed in the first few days, he is driven to compensate if he lacks the postural control to do so safely.

If compensation leads to goal achievement, the drive toward improvement may stop. The brain is oriented toward immediate success and rewards, not to the process involved in reaching the goal. The compensatory strategies will be learned as relevant and appropriate for this stage. Held (1987) states that: "In other words, if compensation is allowed to occur, there is apparently no stimulus to the partially damaged

system to recover and behavioral substitution will occur." This may be explained by reactive synaptogenesis (the formation of new synapses and collateral sprouting) forming abnormal connections that compete with more appropriate connections. Compensation may therefore limit the neural functions that are spared after lesions.

Clinical experience supports the theory that therapy may influence the restructuring processes in the lesioned CNS (see Chapter 1.2 Plasticity, Reorganization of Cortical Maps and Neurotrophic Factors). The CNS recovers very quickly after the initial shock, and some functions may be spared: as edema and the penumbral zone shrinks and circulation improves, neurons start working again and some functions return; this is called spontaneous recovery. If the patient learned to use compensatory strategies during the acute phase, these may not be necessary any longer. But if the CNS experiences these strategies as appropriate in the acute phase, they may be established and difficult to change. The strategies will often have developed based on the need to balance; as balance and movement control is reduced or absent to prevent falls or the feeling of insecurity, therefore they often involve fixing with the arms, grasping, and holding on, weight transference to the least affected side in stroke, fixing through flexion of trunk or flexion/adduction of hips, or pushing off the floor.

This development may be illustrated as follows (Bryce 1989 in Edwards 1996, p.19; Fig. 2.8). Reduced postural tone and loss of reciprocal innervation as the basis for coordination as well as weakness influence the patient's balance and quality of movement negatively; postural stability and orientation are reduced, and therefore also the control of movement. The main aim of the CNS is to ensure the person's safety, and therefore available alternative strategies are recruited and enhanced. The sequence of recruitment (Henneman's recruitment principle) and the sensorimotor organization of activity are altered as a response to balance impairment. The feedback to the CNS of the execution of movement through peripheral receptors will be different due to the movement being different and to the altered integration and modulation of sensorimotor activity: previous responses, feedback from a healthy system, and normal

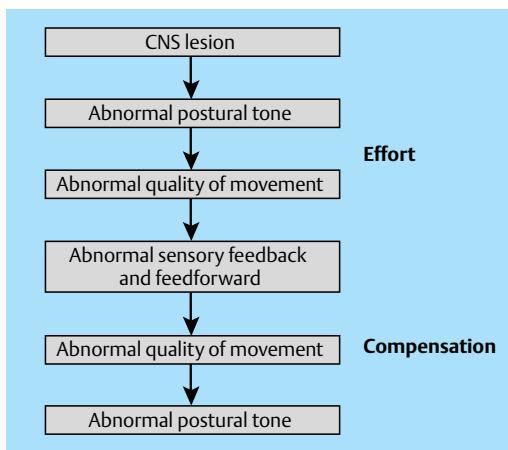


Fig. 2.8 Development of an abnormal postural tone.

movement repertoire. The basis for the execution (i.e., anticipatory postural adjustments) of the next movement and movements in the future will therefore be altered.

After a CNS lesion patients alter their strategies to compensate for loss of or reduced balance control. Many patients develop cognitive and visual strategies that seem to override the integration of information from somatosensory receptors—they do not listen to their bodies any more. Movement becomes slow and patients poke their head forward and look down to enhance visual information (this change in alignment of the head and neck will influence the vestibular system). With this increased dependence on cognitive strategies and vision, patients are increasingly prone to falls if they are distracted or people catch their attention. Mulder et al. (1996) propose three measures for the improvement of balance: reduced cognitive regulation, decreased dependency on vision, and increased sensorimotor adaptation. When we have the ability to balance, we rarely think of it; if we don't have it, we think of it all the time. CNS lesions result in a move away from automatically regulated background control of balance and postural control to increased awareness and cognitive regulation of balance. Which balance strategy the patient adopts depend on his build, experience, personality, and the consequences of the lesion.

Inappropriate compensatory strategies—alternative behavioral strategies—may delay or hinder the development of balance and selective motor control in patients with CNS lesions.

Clinical examples

Balance develops through interaction with gravity. We have to be exposed to gravity to develop postural control in standing, sitting, step standing, and walking. Åsberg (1989) found that stroke patients had improved orthostatic blood pressure, improved ADLs, and had fewer severe functional impairments and limitations after being placed in the standing position early and on a regular basis (every day for 12–days after admission) compared with patients in the control group. The differences between the groups were significant, and no other interventions could explain these differences. At the same time, it is not proved that early standing alone caused this, but Åsberg recommends early standing as a treatment intervention.

Jacobs et al. (1985) found that a group of trial participants significantly improved their perception of trunk orientation (trunk positional sense) in relaxed standing compared to lying.

These studies support the importance of early standing both for functional improvement and the patient's perception of his body.

Standing seems to improve both trunk positional sense and overall function.

Normally the hip and pelvis are stabilized in stance phase for the swing of the opposite leg. This stability is dynamic, the pelvis and hip move in relation to each other, the trunk, and the base of support to allow the translation of the center of gravity in the direction of movement. Stability is maintained over the standing leg throughout this movement. Neuromuscular stability is a prerequisite for selective movement control.

In clinical practice the therapist will meet patients who have reduced or altered recruitment of appropriate stability and movement. Many patients have instability both truncally (core stability) and over the pelvis and hips. Reduced stability or increased trunk flexion seem to prevent activation of hip extension, necessary for stabili-

lity. At the same time, reduced stability over the pelvis and hip may influence trunk stability negatively. Weight transference in standing may increase compression of the ankle and thereby deform the foot. As a result, alignment is changed distally compromising the recruitment of neuromuscular activity proximally.

Patients compensate in different ways: shifting their weight onto the least affected side or body parts (Fig. 2.9), using the environment for support, enhancing the support and protective reaction of the arms, fixing within their own body, recruiting activity more proximally—a shift from ankle to hip strategy—there is an infinite variety of possibilities. Many patients are not able to step due to reduced stability of the standing leg. They move their strategies more proximally to their arms. Patients use arm support in different ways: fixing through adduction, inward rotation of the shoulder, and through increasing their flexion activity in the trunk, fixing through increasing flexion/adduction, compression of the hip, etc. The muscular interplay and muscle balance for selective stability necessary for maintaining a free standing position, weight transference, and walking are disturbed. Clinically, it may be necessary for two or three therapists to work together to facilitate trunk, pelvis, and hip activity for stance phase to allow for swing phase. If appropriate and available, a treadmill with body weight support may be of some help to ease the facilitation and rhythmic interplay between stance and swing as well as finding the right speed to facilitate central pattern generation in the individual (see Chapter 1.1, The Spinal Cord, Central Pattern Generators). Speed is very individual.

Sometimes an orthosis or splint may be appropriate to stabilize the ankle. Heel-strike and heel-off seem to be important to signal phase changes to the patient's CNS, therefore the heel should, if possible, be free to receive and transmit somatosensory and weight information. Therefore, an orthosis that stabilizes the ankle medially and laterally or taping may be of use in a period of transition (Fig. 2.10).

In patients with Parkinson disease, one of the early symptoms is a flexed posture and lack of rotation. One of the early motor symptoms is reduced muscle function in the trunk (Bridgewater and Sharpe 1998), which causes reduced postural control. The patient's reduced ability to right



Fig. 2.9 The patient's left shoulder girdle and pelvis is retracted, the left leg is pulled back with the pelvis and therefore seemingly outward rotated and adducted. The arm is pulled back with the shoulder girdle but is observably inwardly rotated and adducted with flexion/pronation of the elbow and lower arm. The stability and movement control is reduced on both sides (see also Fig. 2.3) but more on the left, most affected side. His body weight shifts toward the right in both standing and walking.

himself increases the flexor tendency and inhibits rotation. Flexion causes a forward lean of the trunk and displacement of the center of gravity, and the patient develops a tendency to fall forward. Patients have a shuffling and tripping gait with short steps and seem to be running after their own center of gravity. Some patients develop compensatory strategies in an attempt to bring the center of gravity back by pressing their feet into the floor through plantar flexion. Bridgewater and Sharpe (1998) refer to other studies in which dorsiflexion was found to be reduced. Trunk flexion and rigidity combined with pushing back from the feet cause the patients to lose mobility and to seem stiffened in their



Fig. 2.10a



Fig. 2.10c



Fig. 2.10d



Fig. 2.10b



Fig. 2.10e



Fig. 2.10f

Figs. 2.10a-f This stroke patient has congenital hip dysplasia and 7 cm difference in leg length, the shorter leg being his left. He has always walked on his toes on his left side, and perceived no balance or movement problems and no restrictions in function or participation. Among other things, he and his wife used to dance a great deal. He then suffered a stroke, resulting in left side affection and motor apraxia. He had a lot of instability on his left side, no hip, knee, or ankle stability or voluntary movement. To help increase the stability of the ankle, the ankle was taped to recruit more proximal stability. Notice the difference in alignment and therefore the ability to recruit more appropriate neuromuscular stability in Figures 2.10a-f.

Note: Taping is not a long-term solution; it may cause skin problems and is difficult for the patient and his carers to put on. After some time this patient received a sport bandage, which gave adequate stability distally.

total expression of movement. Plantar flexion contractures may develop as a result.

By improving mobility of the spinal column, shoulder girdles, and neck and facilitating selective extension, rotation is often spontaneously regained. The feet and muscles of the lower leg should be maintained at an appropriate length for mobility. Through improved distal mobility

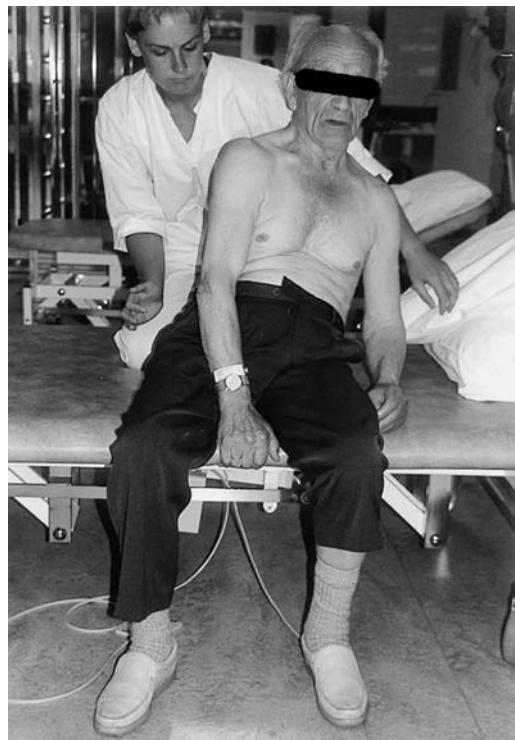


Fig. 2.11 The patient shows reduced balance as a result of an acute stroke. He has reduced tone on his left side and lacks the interplay of movement between body segments in all three planes.

the patient will be able to receive and perceive somatosensory information and orientation of his body in relation to space and the support surface. These interventions may improve the patient's ability to adjust his posture and thereby balance.

Some stroke patients seem to develop a tendency to push away through their least affected extremities. This is a complex combination of symptoms, and mostly seen in patients with left side affliction (right hemispheric stroke). Clinically, the patients are often totally paralyzed on their affected side for a relatively short time initially (Fig. 2.11). Many names have been given to this syndrome; postural hemineglect (Schäger and Kool 2001), the pusher syndrome (Davies 2001), contraversive pushing (Karnath et al. 2000a), etc. Vestibular information is probably integrated in a widely distributed cortical network (Brodal 2001). At least two centers in the brain seem to be involved in the regulation of



Fig. 2.12a

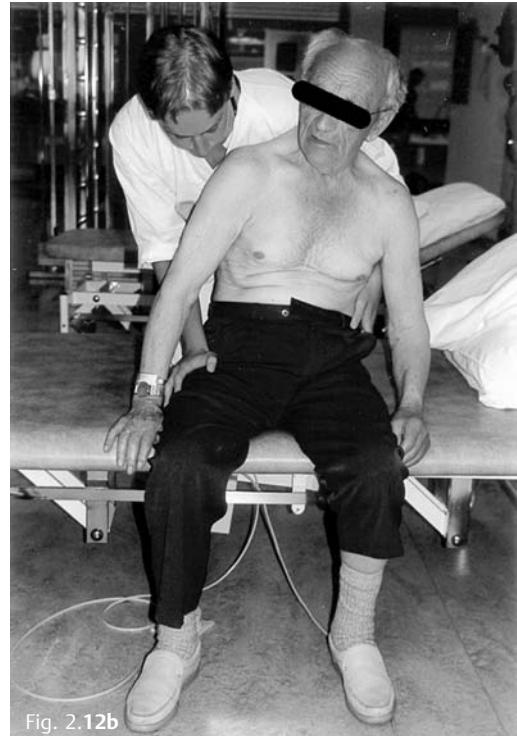


Fig. 2.12b

Figs. 2.12a, b The patient pushes with his right arm and leg.

a There is severe side flexion throughout the right side and the pelvis is elevated on the right side. His interaction with the base of support and between body segments is disturbed. He is malaligned and out of midline.

b The therapist handles the patient to improve the alignment of his pelvis to the base of support. The patient pushes less, but there is still observable malalignment of his upper trunk and head.

vertical perception and summate to achieve postural orientation of the vertical axis:

- Subjective postural vertical (SPV) is a sensitive direction-specific orientation for vestibular function which receives input from sense organs in the trunk—a truncal gravitation dependent system
- Visual subjective vertical (VSV) is a visually perceived sense of being vertical that depends on visual, proprioceptive, and vestibular inputs If perceptual systems are conflicting and mismatch, the patient may develop a “pusher syndrome” (Karnath et al. 2000a and b, 2001, Lösslein and Kolster 2001, Schäger and Kool 2001). They fall to the left side (usually) and their eyes perceive the world on a slant to the right. The cortex listens to the system it trusts most. In some patients, therefore, the input from the eyes will

dominate and override positional information. The patient’s subjective vertical is $101\text{--}7^\circ$ to the left, but they think that they are falling to the right (Fig. 2.12). There is increased flexor activity throughout the right side of the trunk and neck whilst pushing away from the right using the right arm and leg, and little or no activity throughout the left side. This is not a primary perceptual dysfunction, but as the patients receive conflicting information from the PSV and VSV of where they are in space, combined with flaccid paralysis and no information from the paralyzed side, they may develop perceptual problems.

Perceptual problems that are often observed in patients with “pusher syndrome” are:

- Visual neglect: Some patients do not seem to perceive visual information from the most affected side.

- Auditive neglect: Some patients do not seem to perceive what they hear from the most affected side.
 - Neglect of the most affected side of the body: The patient has reduced perception of the most affected side and does not integrate information from this side. But, as previously stated, this syndrome often involves flaccid paralysis initially, therefore there is very little somatosensory information to receive or integrate.
 - Spatial problems, both in relation to one's own body and to the relationship between the body and the environment
 - Reduced sensory perception, but the patient may have normal sensation if the two body halves are tested separately. Often the therapist finds that the patient has severely affected bilateral simultaneous perception of sensation.
 - Altered perception of the midline: The patient is afraid of falling to his least affected side.
 - Other perceptual and cognitive problems
- Physical problems that could occur are:**

- Hemianopia: The patient has reduced visual field to the paretic side.
- Initial paralysis, which seems to recover quickly in many patients. They may have relatively good selective movement in the most affected side, but cannot use it because of severe midline disorientation.
- Reduced postural control
- Reduced weight transference, reduced interplay between left and right, reduced righting of head and trunk
- Use of the least affected extremities to push with. If the patient has a right hemispheric stroke, his head, neck, and trunk are sideflexed to the right, and the head often rotated to the right. An increased use of protective reactions and strategies (the push from the right to avoid the perceived falling to the right) seem to inhibit the interaction of right and left in all three planes.

If this syndrome is not treated successfully, patients develop severe flexor/retractor activity throughout their most affected side and are totally dependent on carers for all functions. They demonstrate inappropriate use of their least affected side, and their ability to vary patterns of movement is reduced. Often, carers and therapists experience lots of resistance when attempt-

ing to correct the patient's alignment and relationship to the base of support. There may be bilateral problems, and problems arise in all practical situations that require balance and transfers as well as positioning in a chair and in bed.

Treatment is goal-oriented toward regaining midline control. The following factors are important to evaluate:

- The patient's awareness of and activity in his paretic side
- Regaining interaction of movement between body segments in all three planes, especially trunk, shoulder girdles, and pelvis
- Improvement and facilitation of distal sensorimotor integration
- Interaction between the right and left side to regain trunk and head righting and postural control

In a treatment situation perceptions need to be matched. Vertical orientation requires alignment of the pelvis and intra-abdominal pressure. Therefore vision may be taken away (the patient blinded or vision blocked to the right) combined with using a broad bandage or belt to assemble the trunk and abdominal contents in the midline to increase abdominal pressure. Lying on the right side or with the right side supported in sitting and standing may make the left side more available for increasing the somatosensory information through the left side.

Some patients develop a hypersensitive foot that pushes off the floor through plantarflexion and inversion (Figs. 2.13–2.14). They may experience a primary hypersensitivity toward stretch, touch, or weightbearing to the forefoot or this reaction may develop as a response to gravity in order to stiffen the leg to be able to stand on it to walk when the proximal areas are too weak or unstable to carry weight selectively. The severity of the reaction will vary in different patients and in different situations. Pressure, touch, or stretch to the foot in weightbearing cause plantarflexion with a backward translation of the tibia in relation to the foot. The pattern causes plantar flexion with varying degrees of inversion, a mechanical hyperextension of the knee as a result of the plantarflexion and is usually combined with flexor components at the hip, i.e., varying degrees of flexion/adduction/inward rotation. The mechanical hyperextension of the knee is a result of the knee being caught in the middle be-



Fig. 2.13a



Fig. 2.13b

Figs. 2.13a, b When this patient stands up from sitting he uses a lot of effort. Note the facial expression and the associated reactions in the arm. Compare with Figure 2.9, where the associated reactions are less when the patient has been standing for a short time. The transfer to standing demands interplay between body segments and the base of support. The patient has very little mobility generally, and his left foot

is not adapting to the base of support, therefore weight transference to the left is not possible. He has to compensate by pushing up through his right side. This patient had two strokes approximately 30 years ago. He manages most activities and is active in participation. But it is costing him a lot of effort, and he becomes tired, more so as he is getting older.

tween a plantarflexed foot and a flexed hip during weightbearing. Quadriceps is rarely active in this reaction. The incoordination of foot, ankle, knee, and hip negatively affect balance and reduce interplay and variation of movement.

Flexor withdrawal may be caused either by a hypersensitive foot or sensitive (short) hip flexors (flexor reflex afferents), therefore either from a distal or a proximal initiation:

- Weight transference through the foot causes stretch of the soft tissues within the foot. In some cases initial stretch in attempted weightbearing produces withdrawal of the

foot from the floor. The pattern varies in severity and rotational components, but usually causes inversion of the foot and flexion of the hip and knee.

- Withdrawal reaction may also be caused by stretch to the hip flexors, often initiated as a reaction when the patient starts to raise himself to standing from sitting. The stretch of the hip flexors causes hip and knee flexion combined with dorsiflexion of the foot with varying degrees of inversion or eversion depending on the neuromuscular rotational components of the hip.



Fig. 2.14a



Fig. 2.14b



Fig. 2.14c

Figs. 2.14a–c Reduced adaptability of the foot in standing and walking. The left knee is hyperextended, the hip flexed. Stability is reduced over his left side and effort increases as balance requirements increase during gait judging by the level of associated reactions (compared with Fig. 2.9).

- Both these reactions may retract the pelvis and therefore seemingly abduct and outwardly rotate the flexed hip. The apparent abduction is a result of the backward pull of the pelvis in retraction taking the hip with it, without the hip necessarily being truly abducted or externally rotated.

All of the above-mentioned deviations prevent normal weight transference, balance, and transfers. Treatment is targeted toward the impairments: the hypersensitivity, the immobility of the foot, and improving muscle length and flexibility. Improving postural control, especially the stability component in different situations, involves bringing all the following into activity through graded weightbearing and varied active movement over the foot in different weightbearing activities: sitting to standing over one or two feet, controlled standing to sitting, standing and step standing, stepping in different directions, one-legged standing, stepping down from a height, sitting on one or two legs, stairs, etc. The restoration of dynamic interplay between the foot, knee, and hip is essential to the patient's ability to balance and move.

2.2 Intervention—Considerations and Choices

On the basis of continuous assessment and clinical reasoning the therapist chooses interventions that seem appropriate to the individual patient. The Bobath concept does not give a solution or method for treatment of patients.

The current definition of the Bobath Concept is: It is a problem-solving approach to the assessment and treatment of individuals with disturbances of function, movement, and postural control due to a lesion of the central nervous system (IBITA, Theoretical Assumptions 2007; www.ibita.org).

Treatment is tailored to the individual patient, and is response-based. Therefore interventions depend on the individual patient, his sensorimotor dysfunctions, his perceptual and cognitive resources and problems, the adaptive compensatory strategies developed, the environment, and the goal or task. All interventions, even if impairment-oriented, need to integrate activities to make treatment as functional as possible and the carry-over effect as strong as possible. Motivation is a crucial factor to learning. Treatment incorporates:

- Regaining movement control
- Motor learning
- Interdisciplinary approach to enhance learning and carry over
- The use of compensatory strategies when further motor learning does not seem possible (may incorporate the use of aids and orthoses)
- Management strategies to prevent or minimize complications

Postural Sets

Berta Bobath described postural sets as “adaptations of posture” which “change with the intended movement—in fact, they may precede it” (Bobath 1990, p. 68). Body segments have a biomechanical and a neuromuscular relationship, both of which are the basis and consequence of the build of the individual, movement, and the

actual relationship with the environment. This relationship continuously changes during activity. Neuromuscular activity and biomechanical factors influence and are affected by each other. Changes in the relationship with the environment and alterations in the biomechanical relationships through changes in rotational components or direction of joint movement require adaptation of the neuromuscular activity, even if the goal of movement or task stays the same. Neuromuscular activity depends on where the person started the movement from, i.e., standing up from sitting on a low, soft cushion or stepping down from a high seat. When a person flexes his elbow in sitting or standing, the biceps is the prime mover. The same movement performed in supine, with the arm stretched up in the air or the arm being held over shoulder level in sitting or standing, requires more eccentric control from the triceps as the prime mover and agonist.

The neuromuscular activity required to perform pelvic tilt varies in different positions because the biomechanical relations alter with the changing relationship of gravity to the base of support. Therefore, the activity is different in sitting, in the transition from sitting to standing, in standing, through sitting to supine, and in supine. Analysis of movement is the detailed analysis of movement during every phase of an activity to form hypotheses for the patient’s recruitment of neuromuscular activity in function. This analysis is the foundation for clinical reasoning, together with an analysis of the patient’s performance that includes an assessment and evaluation of perceptual and cognitive function.

Postural sets describe the interrelationship between body segments at a given moment. Movement may be described as a continuous change of postural sets.

If serial photographs of a movement or activity are taken, each photograph represents a postural set. Analysis of postural sets gives information about:

- The effect of gravity
- The relationship to the base of support
- Alignment
- Patterns of movement
- Neuromuscular activity

There is a tendency to analyze basic postures, but humans move within and between postures. *Basic postures* are sitting, standing, step standing, and supine or prone, with symmetry or asymmetry if including step standing and even weight distribution. Postural sets are all different variations within a basic posture and the transitions between postures.

There is an interrelationship between postural control and postural sets. The analysis of postural sets in a functional activity allows the clinician to observe actual and gradual changes in alignment and to form hypotheses regarding the neuromuscular activation creating the movement. The chosen interventions may or may not support the hypotheses. If the patient's motor control is not improving, either the interventions or the hypotheses may be wrong and must be reconsidered. The clinician must continuously adapt the interventions to the patient's response and required movement.

Postural sets are also used in treatment to adapt and adjust requirements to suit the patient's ability. The choice of postural sets depend on the patient's balance control and relationship to the base of support, and therefore the choice of goal activity or task. If the patient has low postural control, or is flexed with asymmetry, malalignment, and deviant tone distribution, he will not be able to recruit a more appropriate activity to interact or respond to the environment. Shumway-Cook and Woollacott (2007) describe *ideal alignment* in relation to the standing position as follows: "muscles throughout the body, not just those of the trunk, are tonically active to maintain the body in a narrowly confined vertical position during quiet stance. Once the centre of gravity moves outside the narrow range defined by the ideal alignment, more muscular effort is required to recover a stable position." Optimal or ideal alignments in any postural set allow us to use no more effort than necessary to maintain stability. Inappropriate alignment or malalignment may maintain an inappropriate neuromuscular recruitment pattern and thereby prevent the patient adapting his response to the environment. Sahrmann (1992) states that a normal neuromuscular interplay, muscle balance, or adaptive muscular activity facilitates good alignment, and that good alignment facilitates normal, adaptive neuromuscular activity.

A selective movement in one postural set requires a different neuromuscular activity in a different postural set. As the biomechanical alignment changes, so does the neuromuscular activity.

Analysis of Basic Postures and Postural Sets

The postural sets chosen as intervention have to be adapted to the patient's specific problems to enhance success and motivation. Advantages and disadvantages, possibilities for variation, how easy or difficult it will be for the patient to move into and out of and between the postural sets must be considered in the light of the patient's movement control at the time. Balance and movement result from the interaction of many muscle groups and their eccentric/concentric work as agonists, antagonists, and synergists. It is not possible to analyze the activity in all muscles in all phases of different activities, and not possible to describe this variation in words. In the following sections, the main features or qualities of standing, sitting, supine, and side lying will be analyzed. Any other position may be analyzed in the same way.

Standing

The basic posture is characterized by extension: trunk, head and neck, and legs. This selective extension is based on the interplay of trunk musculature, core stability, and the balanced muscular activity of the legs. The postural tone is relatively high if the person stands actively and the base of support is relatively small. The shoulders are slightly protracted but relatively relaxed and the arms hang by the side (Fig. 2.15). The rotation of the arms depends on the biomechanical alignment and neuromuscular activation of the individual, especially of the trunk and shoulder girdles. Increase of thoracic extension and of the shoulder girdles may give more outward rotation of the arm, whereas active protraction and flexion enhance inward rotation. Standing is generally facilitatory to the development of extension. The patient is exposed to gravity, which enhances postural tone and postural control if alignment is good.



Fig. 2.15a



Fig. 2.15b

Figs. 2.15a, b Basic standing posture.

Advantages

There is wide variety standing postural sets, changing foot position, using mobile or stable supports behind, at the side, at the front to allow the patient to explore his motor control safely. All changes cause neuromuscular adaptations. The placing of the arms influences tonic activity in the body: if the arms are placed above 90°, trunk extension is facilitated to a larger degree. The arms may be placed in different positions and at different heights (Figs. 2.16–2.19).

An active arm facilitates postural activity, while the use of arm support may alter or negate postural control depending on how it is used (Jeka and Lackner 1994, Jeka 1997, Slijper and Latash 2000). Standing postural sets may vary from parallel to step standing and thereby enhance weight transference and access alterations of stance to swing in different directions. Changes of hip rotation require altered neuromuscular activity; physiologic outward rotation in the standing leg may facilitate abduction and extension and thereby stability. The “let go” or eccentric activation of hip extension/abduction/outward rotation facilitates initiation of the swing phase.

There are good opportunities for therapeutic handling and correction of alignment, both distally and proximally, if the patient has a certain level of postural control, feels safe in this situation, and is willing to explore his possibilities. It is important to give adequate and appropriate support to knees for instance to allow the patient to experience and develop trunk control or pelvic movement with or without facilitation. The use of a plinth placed at different heights, at different positions in relation to the patient (at the side, at the back, diagonally, at the front) allows variation and active exploration between sitting and standing (Fig. 2.20). The patient is facilitated to experience and explore variations in eccentric control and grade movement in different directions. Eccentric muscle activity seems to improve strength and generalization (carry-over) to more varied muscle work and functional activities (Patten et al. 2004). Strength training facilitates significant synaptogenesis on the motor neurons in the spinal cord and does not seem to have a negative impact on spasticity (spasticity as defined by Pandyan et al. 2005. See Chapter 1.3, Consequences of and Reorganization after CNS



Fig. 2.16a



Fig. 2.16b

Figs. 2.16a, b Lateral weight transference with the arm abducted at shoulder level as the model rolls a ball. The demands for extension and stability increase on the weightbearing side if the model does not press down on the ball.



Fig. 2.17a



Fig. 2.17b

Figs. 2.17a, b The use of the wall may facilitate placing of the arms. The model has to stabilize both body and arms to maintain his postural set and move at the same time. **a** More outward rotation of the arm and hand combined with extension at the elbow and protraction of the shoulder facilitates abdominal activation as part of postural control. Improved postural control facilitates stability through the shoulder and arm. **b** Improved postural control and stability of the left arm facilitates freedom of movement for functions in the right arm.



Figs. 2.18a–c The demands for postural stability and orientation increase even more when the arms are active.



Maintenance of postural control is necessary for daily activities.



Fig. 2.20a

Figs. 2.20a-d As the model sits on a high plinth she needs to stabilize and move over the standing leg. In this situation there are specific needs for abduction



Fig. 2.20b

and outward rotation of the weightbearing hip to lift the pelvis onto the plinth, as well as for righting body segments to each other.



Fig. 2.20c

Fig. 2.20c Components of rotation may be varied to alter the demands for postural control, balance and movement both in standing to sitting and sitting to standing.



Fig. 2.20d

Fig. 2.20d A postural set of high sitting. This posture demands good pelvic mobility (i.e., movement of the pelvis in relation to the hips and the lumbar spine) as well as facilitating the transition from standing to sitting and sitting to standing—stand down.



Fig. 2.21a Dressing in a standing position is normal. The model balances over her right leg at the same time as putting her left leg into the trousers. This is a complex perceptual, cognitive, and sensorimotor activity demanding problem solving and a continuous adaptation to displacement over a small base of support.



Fig. 2.21b The model hangs her jumper on a hook above shoulder level. She has to locate the hook and problem solve the activity, weight transfer, and at the same time sustain her postural stability to free the arm to lift the jumper onto the hook.



Fig. 2.21c When the model puts on her jumper, the hand and arm actively lead the movement through extension into the sleeve.



Fig. 2.21d As the jumper goes over the head, vision is obscured, and the integration of somatosensory inputs is weighted more for postural control. The need for sensorimotor adaptation to maintain postural control increases. Righting occurs between body segments.

Lesions). Patten et al. (2004) also state that skill training combined with task-specific training improves activity-dependent cortical reorganization. Therefore, treatment needs to be targeted, involves specific aspects of strength training (eccentric), and is context-based as this combination seems to improve the patient's function.

Use of the standing postural set often motivates the patient. The patient is oriented to the relationship between body and space, which improves perception. Standing also improves orthostatic control of blood pressure, circulation, lung function, and bowel and urinary functions (Fig. 2.21).

Disadvantages

Some patients have very low postural tone and are unable to interact with gravity to right themselves. If the patient supports himself, leans onto, or presses down on an external support, he may recruit inappropriate active flexion components, which further negates interaction with gravity and the acquisition of postural control. Flexor strategies may increase if he feels insecure. Standing postural sets may require two thera-

pists or the help of an assistant to make the situation facilitatory for the patient's postural activation. The use of a standing frame or similar device may be appropriate in some cases. Note that the patient needs to be placed completely upright in a vertical alignment at 90° to facilitate postural tone and activity over his base of support, the feet.

Sitting

The basic sitting position is characterized by trunk extension, core stability, and head and neck alignment in extension and balanced to abdominal activity. The hips are in a position of flexion biomechanically, but stability in sitting depends on the interplay of neuromuscular activation of extension/abduction/outward rotation balanced with flexion. The thighs rest on the plinth and are a reference for the trunk. Components of hip rotation may vary, but the optimal neuromuscular activity as a foundation for stability favors outward rather than inward rotation (Fig. 2.22).



Fig. 2.22a



Fig. 2.22b

Figs 2.22a, b The basic upright sitting position.

The arms rest in adduction when they are not active, and rotation depends on the neuromuscular and biomechanical relationship of the thorax, head, neck, and shoulder girdles. Sitting is a functional position for many activities and allows the therapist good handling opportunities and variation.

Advantages

Sitting postural sets (Fig. 2.23) may be varied infinitely depending on the neuromuscular activity the therapist wants to facilitate: sitting straight, diagonally, more or less rotated, leaning backward or forward, sitting high or low, far into the seat or on the edge, different bases (bed, plinth, a variety of chairs, stools), different textures and hardness, or using the floor to sit on. Sitting postural sets are varied in relation to the task; where and in which direction the patient's

next movement is going, i.e., the direction of the evolving activity, and therefore which neuromuscular activity is needed (Figs. 2.24–2.25).

The position, posture, and activity of the arms influence the neuromuscular activity in the trunk. The use of arm support may reduce the patient's postural control by reorientating the references for equilibrium responses to the support and the supporting arm instead of the feet in standing and the buttocks and thighs in sitting—depending on how the arm support is being used (Jeka and Lackner 1994; Jeka 1997; see Handling). Arms above 90° facilitate trunk extension and postural activation. If the patient can start to take over the weight of the arms, postural stability and strength are enhanced throughout the body and arms, which is needed, for example, when placing plates in a high cupboard or hanging a coat on a hook (Fig. 2.26). Movement of the



Fig. 2.23a Increased adduction of the hips is a frequent problem in recruitment of stability and movement of the hips and pelvis. Enhanced abduction/outward rotation of the hips facilitated a more upright posture and postural control, and thereby interplay between body segments. A bigger and more open base allows for more movement of proximal segments.



Fig. 2.23b Variation with rotation alters the weight-bearing areas and therefore stability requirements.



Fig. 2.24a



Fig. 2.24b

Figs. 2.24a, b Selective movement of the pelvis in relation to the hips and lumbar spine requires righting of the trunk, head, and neck. Note the position of the head as the model flexes thoracically. When the arms

are resting on a table or a plinth, as shown in the picture, there is a need for more movement of the shoulders as she moves the body away from the plinth.



Fig. 2.25a

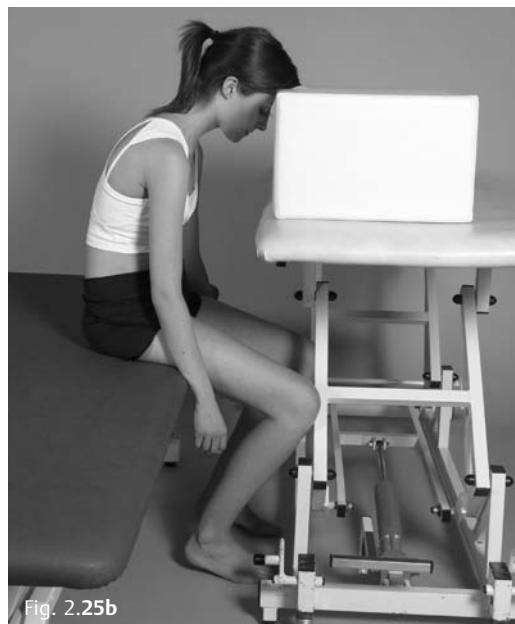


Fig. 2.25b

Figs. 2.25a, b Forward-lean sitting is a good postural set for freeing the head and neck, and facilitating interplay between body segments for righting and postural activation. In a clinical situation it is important to

facilitate active sitting in the patient and not allow the patient to lower his tone and lean on the support as this might negate the aim of using this postural set.



Fig. 2.26 The model stabilizes through her left arm and hand as she transfers weight and rotates to fetch a book on her left side with her right hand. The left arm becomes part of her base of support and allows her to move further to the left, out of her original stability limits. She rights herself in relation to her arm and trunk, stabilizes over her left side and has to move in relation to her left arm. The arm gives her dynamic support; she has to move to and from the arm and needs to have good mobility, stability, and coordination in the shoulder girdle and between the body segments.

body in relation to stable arms or moving the arms in relation to a stable trunk facilitates the interplay between stability and movement, which are facilitatory for postural control.

Sitting postural sets are easy to vary through different phases of treatment: from a more mobilizing intervention to facilitation of activity to the patient moving himself without hands-on in the same treatment. Sitting postural sets are adapted to optimize the neuromuscular activity for the actual function the patient will perform; for instance, using high sitting, which is more extensor dominated and more facilitatory for postural control, to stand up from or early activation/stimula-



Fig. 2.27 Backward-lean sitting. Note the importance of obtaining good alignment to the base of support. Depending on the adaptability of the patient's hip flexors, the position will have to be adapted with more or less support at the back to allow eccentric lengthening of the lumbar extensors. Good contact between the lumbar area and the support facilitates activation of abdominals to adjust the position or sit up.

tion/facilitation of arm and hand function. Sitting postural sets are used for:

- Mobilizing the pelvis in relation to the trunk and the base of support
- Facilitating segmental lumbar extension to facilitate components of hip stability and abdominal activity, which helps to achieve postural control and free arms
- Variety and movement from sitting to supine or sitting to standing
- Stimulating and facilitating fine motor activity of the hand if postural activation is maintained, or using the arm and hand to facilitate postural control
- Backward-lean sitting to access a stiff or hypersensitive foot or shortened hip flexors and adductors as preparation for improved stability in transfers and standing (Fig. 2.27)



Fig. 2.28a



Fig. 2.28b



Fig. 2.28c



Fig. 2.28d



Fig. 2.28e



Fig. 2.28f



Fig. 2.28g

Figs. 2.28a–g Undressing in a sitting position. Balance, movement, weight transfer, rotation, righting, and postural control are all necessary elements for dressing or undressing in a sitting position. Note the extension of the arms as the jumper and T-shirt are taken off (Figs. c, e, and f). The arms are free due to the stable trunk and may therefore move easily in and out of the garments.

Disadvantages

Because of the bigger base in sitting, patients with low postural tone easily sink into flexion and may start to fix in flexion because this may be the only strategy available to them. Patients who are already fixing in flexion may be stimulated to do so even more: short muscles enhance short muscles.

Use of supports influence the patient's postural tone, depending on how the support is used by the patient and the therapist. A support such as a table, plinth, stick, pillow, or a wall increase the supporting base. If the patient leans on the support, the requirement for postural control is reduced. If the patient presses into the support, supports himself heavily or fixes to the base of support, flexor activity is increased. However, the patient and therapist may use the support as a reference for movement or to reduce the weight of heavy arms and thereby facilitate postural control.

The material that the support base is made from is important for the neuromuscular activity that is promoted. A soft seat may stimulate more flexor/adductor/inward rotator activity, especially over the hips, pelvis, and lower trunk, than a firm support. A high support—a wall, high stick, high table, high cupboard—facilitates extensor activity more than a low support, depending on how it is being used.

■ Supine

Supine postural sets are characterized by extension if the person is able to eccentrically lengthen his hips, lumbar spine, neck, and shoulder girdles. The base of support is large and the center of gravity low, and if the person is able to relate to the support and let go of muscular tension, the postural tone will be low. There is a tendency toward anterior tilt of the pelvis which may be due to the sustained tone in the hip flexors, but if the person is able to eccentrically lengthen both hip flexors and lumbar extensors, he will have a better contact to the base of support and a more appropriate alignment, both for rest and activity. With this alignment, the extremities will tend to be slightly abducted, outwardly rotated, and extended. The forearms are often pronated and the elbows slightly flexed, which is normal. We tend to relate to the environment through the palms of our hands for orientation (Fig. 2.29).

Advantages

Supine postural sets may be varied in different ways: both legs may be flexed and the feet placed in different distances from the hips. The degree of hip and knee flexion will determine how difficult or easy it is to move the pelvis. If the feet are



Fig. 2.29 Supine postural set.



Fig. 2.30a



Fig. 2.30b

Figs. 2.30a, b Crook lying and pelvic tilt to facilitate postural activation. Note the position of the knees in relation to the feet in the two pictures, and consequent hip extension and abdominal activation. **a** The knees move forward over the feet to facilitate selective pelvic tilt. **b** Selective pelvic tilt involves stability of ankles and knees together with activation of proximal hamstrings, gluteal muscles, and abdominal muscles.

placed closely to the hips, weight transference onto the feet through pelvic tilt is facilitated due to biomechanical relationships (Fig. 2.30a). Hip extension and postural activation are enhanced if the knees move distally to be in line with the feet (Fig. 2.30b).

If the feet are placed further down and away from the hips, the biomechanical alignment changes, and the recruitment of pelvic and postural activity made more difficult. The upper body may be supported with pillows to enhance eccentric lengthening of trunk extensors and thereby facilitate abdominal activity. The interplay between abdominal and extensor neuromuscular activation is essential for selective pelvic (in relation to hips and lumbar spine) activity, mobility, and stability. Supine postural sets may be suitable for specific mobilization of muscles that are short or inactive if the patient is able to adjust to the position.

Different phases of the transfer from sitting to supine or supine to sitting are used clinically to facilitate a graded coordination and interplay between flexor, extensor, abductor, adductor, and rotatory components within and between body segments for the control of stability and movement.

Disadvantages

Postural tone is basically low in supine. Therefore the initiation of activity to oppose gravity may be difficult. The large contact area means that there are many frictional and inertial components to be overcome. Therefore, to be active in supine and to transfer from supine to sitting requires a reasonable postural activity or this activity to be facilitated. The transfer from supine to sitting is complex and requires a certain level of postural control combined with graded alterations of flexor, extensor, abductor, adductor, and rotatory components to be performed independently. At the same time, the patient is often required to perform this with no or little help or positional adaptations even in the early stages after a CNS lesion, and this may drive the choice of compensatory strategies for other functions also. Supine may therefore not be the first position of choice in the treatment of the very low toned patient, but may be used more appropriately for the training of specific components of stability, movement, and strength with a patient who has more background activity.

Some patients have increased tone in supine. They may have a reduced ability to adapt to the base of support and feel uncomfortable, insecure, or vulnerable. Others may have reduced segmental mobility and interaction between body segments, and therefore reduced ability to actively move and transfer weight to change position. Weight and friction make movement less accessible, also for therapeutic handling.

■ Side-Lying Postural Sets

These are characterized by extension through the weightbearing side and a more flexor bias on the upper side (Fig. 2.31b). The stability requirements are greatest on the weightbearing side because of the interaction with the base of support.

Advantages

Side-lying postural sets may be varied by changing rotatory components within the trunk or extremities or by using more or fewer pillows to influence stability (Fig. 2.31). Side-lying postural sets allow for great mobility between proximal body segments and facilitate placing of the upper arm or leg to enhance postural stability and strength. Side lying on the most affected side in stroke may stimulate this through weightbearing and tactile input, and facilitate postural activity in both central and proximal segments. Side lying on the least affected side in stroke may improve the stability of this side as a background for movement of the more affected limbs in space.

Disadvantages

Side-lying postural sets may be very unstable as the base is long and narrow with a slightly higher center of gravity than supine. Therefore it may be a difficult position to use in treatment. If the adaptation to the base of support is inadequate, for instance, if the patient has reduced ability to eccentrically lengthen the supporting side, then the uppermost shoulder girdle will be very unstable. The stable position of the scapula depends on the activation of intercostal muscles and the muscles of the trunk. If these are stretched or inactive, the scapula will glide upward on the trunk, with inappropriate alignment for selective placing of arm. The stability of side lying may be enhanced by placing tightly-rolled towels closely into and very slightly under the patient's trunk posteriorly



Fig. 2.31a



Fig. 2.31b

Figs. 2.31a–e Side lying with different supports.



Fig. 2.31c



Fig. 2.31d



Fig. 2.31e

and anteriorly as well as by using pillows to stabilize the upper leg in neutral (Fig. 2.31c).

During treatment, the therapist needs to be specific, critical, selective, and needs to use postural sets that are appropriate in relation to the patient's problem. Essential factors in the choice of postural sets are:

- How easy or difficult it is to vary the patient's posture for the appropriate recruitment of neuromuscular activity
- How easy or difficult it is to vary postural sets in the gradual transition from one position to another
- How much effort is required
- What motor strategies are facilitated
- Whether the patient loses or strengthens the control he regained in one position as he moves into another

We move from one postural set to another continuously in our daily activities. We rarely stay in one position to do anything. Therapy has to reflect this: facilitating the patient to regain control of movement, not static activity. The functional importance of the treatment is essential for learning.

■ Key Areas

Movement causes continuous displacement of body segments in relation to each other, and also intra-segmental alignment and distribution of tone through neuromuscular activity. This may be analyzed through observation and handling (Taylor et al. 1995). Bobath (1990), Bader-Johansson (1991), Kidd et al. (1992), and Edwards (1996) call some of the body segments *key points (of control)*. This term is easy to misunderstand as it refers to segments, regions, or areas, and not to points. *Key areas* or *functional units* are more accurate names, also because these areas have their own activity at the same time as they interact with the rest of the body. The key areas are: central, proximal, and distal.

■ The Central Key Area

The central key area is the thorax with its joints and muscular attachments to head and neck, the shoulder girdles, and the pelvis. Especially important is the middle thoracic area and the ribs with their costal attachments to the sternum

and the musculature in this area. The functions of the central key area are first and foremost balance, postural control, and a stable reference for extremity function. Movement in three planes, frontal, transverse, and sagittal, allows for weight transference, interplay between right and left, and the ability to cross midline with the extremities.

Some therapists classify the head and neck as an extension of the central key area, or as a proximal key area in itself. The function of the head and neck in movement (excluding communication or eating) is many fold. Important aspects are orientation to the world around us, being a stable reference for the eyes, keeping the eyes horizontal to receive and therefore perceive information as accurately as possible, and enhancing balance as far as possible.

■ Proximal Key Areas: Shoulder and Pelvic Girdles

- **The shoulder girdle:** Caillet (1980) describes the shoulder girdle as consisting of seven components: the glenohumeral joint, the supraventricular connection (the coracoacromial ligament; the coracoid process and the acromion process together form the coracoacromial arch which supports the head of the humerus above), the acromioclavicular joint, the sternoclavicular joint, sternocostal joints, costovertebral joints, and the scapulocostal attachment. The shoulder girdle cannot be viewed separately from other body segments.

Through its attachment to the spine and the pelvis via the trunk musculature, the shoulder girdle influences and is influenced by alignment and neuromuscular activity of the pelvic girdle, the trunk, head and neck, and extremities. The function of the shoulder girdle is to be a mobile yet stable reference (mobile stability) for arm and hand function and at the same time a functional part of balance.

- **The pelvic girdle:** The pelvis moves both in relation to the lumbar spine and therefore the trunk, and to the hips. The pelvis comprises the two iliac bones and the sacrum, the mobile areas being the sacroiliac joints and the symphysis pubis. These joints are immovable, although they do allow for small rotational components to transfer stresses and strains. The pelvis moves between the lumbar spine

above and the hip joints below. The pelvis as a key area is, therefore, not the pelvis alone but the pelvis together with its proximal and more distal relationships. Functionally, the pelvis, with the lumbar spine and hip joints, is mainly responsible for translation of those forces that act down on to the base of support and up through the body, stability and mobility (mobile stability), and weight transference.

■ Distal Key Areas

The hands and feet are mobile and adaptable entities with lots of specific sensory receptors to allow interaction of the body with the environment.

- **Hands:** The human hands are unique in their ability to oppose the thumb to the other fingers, thereby allowing the whole range of movement from finely-tuned and graded movement to strength and power grip. Many layers of small muscles within the hand make it possible to alter rotational components to change and adjust movement. Some areas are more stable, others more mobile, depending on the function. The lumbrical grip is based on extension of the wrist and is a foundation for reach and grasp and thus for both pinch grip and power grip. The function of the hands is to explore the environment to touch and feel and receive information, to underline expression and meaning (gesticulation), to manipulate objects and perform fine motor skills as well as carrying, lifting, and moving things, and being an extension of the body in pushing, for instance, a wheel barrow. They have a role in balance, seeking environmental support when needed, together with the arms.
- **The feet:** The feet transmit forces between the environment and the body and between the body and the environment. They are resilient and provide springiness when walking in hilly environments or up the stairs, running, and when changing direction. They improve the reach of the arm by their power in tiptoeing. The toes are important to turn around and change direction. The function of the feet is to seek information from and about the environment, to adapt to the support base to balance and weight transfer.

Elbows and knees allow the change of patterns of movement through the change of rotation be-

tween the upper and lower limb segments in conjunction with the proximal and distal key areas. The sum of individual components within these areas enables vast variations to meet the requirements of a diversity of tasks. The interplay between the key areas allows balance, weight transfer, and movement at the same time. No body segment functions in isolation.

Many muscles and joints converge in the key areas, for instance, in the hand alone there are 19 bones, combined with the wrist and lower arm there are 29; 20 muscles are intrinsic to the hand and approximately 19 are in the lower arm and act on the hand in some way, not counting the upper arm. The central key area comprises the ribs, sternum, vertebrae, and deep and superficial musculature. Specific receptors in muscles, tendons, joints, and skin pick up any change in activity and report to the CNS. This allows for an infinite variety of movement, stability, and adjustment. Clinical experience suggests that handling of one key area influences tone and activity of other body segments and key areas in two ways: (i) through skin, joint, and muscular attachments directly and indirectly, and (ii) probably because handling influences many specific receptors and the transmission of information to the CNS. All information from the periphery converges in the spinal cord, and an abundance of interneurons transmit this information over many levels within the spinal cord and therefore spread information over a relatively large area, as well as to the brain.

Many muscles and joints converge at the key areas. Therefore the proprioceptive influence, as well as those from the skin, on the CNS is substantial.

Control of key areas and the interplay between them seem especially important for balance, selectivity of movement, adaptation to the environment and tasks, and therefore for function.

Treatment aimed at improving the muscular interplay, alignment, and mobility in and between key areas may improve coordination and the relationship between stability and movement. As a result, the patient may experience improved balance and selectivity and generally more control over his body.

In a treatment situation the therapist has to assess which key area (or areas) is most dysfunctional, whether this has to be treated in isolation first, or whether the patient will regain more control if there is interplay between more key areas in activity. This focus often varies during treatment. The choices must be directly related to the individual patient's movement problem and the functions that need to be regained first.

Selective Movement and Functional Activity

A functional task may be divided into short-term goals or components, which is composed of the motor activity needed to reach the goal (the process), movement strategies and patterns, selective movement, and neuromuscular activity, and must relate to the environment in which it is being performed.

Functional goal	For example, dressing, personal care, fetching a book, making a cup of coffee, going to the bathroom, walking to answer the telephone, or opening the door. Extended goals are instrumental ADLs, such as shopping.
↑ Motor activity	For example, turning around, weight transference for transfers, stepping, sitting down, lying down.
↑ Patterns of movement	Movement over more than one segment or joint, the sequencing of selective movements for instance in righting, reaching and grasping, and stance and swing.
↑ Selective control	Isolated movement over one joint or key area based on stability in other parts.
↑ Neuromuscular activity	Depends on the postural set(s) chosen for the task. Relates to the recruitment of neuromuscular activity necessary to move to reach the goal.

Few studies have focused on the relationship between body functions and structures, activity, and participation (WHO 2006). Normann (2004) showed that even if the therapist spent most of the treatment time improving the patient's control of neuromuscular recruitment in relevant activities, the treatment showed observable changes in activity and the patients spontaneously reported improvement in participation. Smedal et al. (2006) in her study of two multiple sclerosis patients, demonstrates that it is possible to regain activity through training that focuses on body function and structures, and that the effect lasts.

Clinical example

The movement of the pelvis (pelvic tilt) in lateral and anteroposterior direction is essential to all weight transference and transfers and is therefore integrated in all functional activities. Pelvic tilt requires different neuromuscular activity in different postural sets and through movement from one position to another, for instance in sitting down, in supine to change position in bed, moving from one chair to another, or to wiping one's bottom in the bathroom (Fig. 2.32).

Control of pelvic tilt in supine does not necessarily carry over automatically to the transfer from sitting to standing or walking. If, based on clinical reasoning, it is necessary to work on pelvic tilt in supine, for instance, to improve the actual range of available movement, to enhance proprioceptive information for the awareness of the body part and body image, then pelvic tilt needs to be worked on and facilitated throughout the transfer from supine to sitting as well, moving through and controlling the different postural alignments in this function to gain carry over into sitting. The same applies from standing to sitting and vice versa. The relationship between stability and movement changes through the transfer and needs to be facilitated, controlled, and regained throughout the movement. Carry-over to different situations may be enhanced if the treatment is varied, incorporating different alignments and rotational components and specific learning to control eccentric activity.

The hip abductors are important for the stability of the pelvis during locomotion (Whittle 1996, Shumway-Cook and Woollacott 2006).



Figs. 2.32a–c Different alignments of the pelvis in relation to gravity and the base of support require different neuromuscular activation to move. **a** Pelvic tilt in supine.



Fig. 2.32b

b Pelvic tilt in the movement from sitting to standing.



Fig. 2.32c

c Pelvic alignment in standing.

The hip abductors on the stance leg prevent a contralateral drop of the pelvis during swing phase of the opposite leg, i.e., a functional (or physiologic) lateral pelvic tilt. Many patients have reduced recruitment of this activity during stepping and are therefore unstable during transfers or walking. Hip abductors may be recruited and facilitated in different ways: stepping sideways, sitting asymmetrically on a high support (Fig. 2.20), walking from step standing, improving specific strength in side lying. Stepping down from a high support requires recruitment of hip abductors on the standing leg and may facilitate the first step to walk, if the alignment is appropriate. Locomotion is initiated from many different postural sets, not just parallel standing or step standing, and we walk forward, backward, sideways, and turn around. All these variations need to be incorporated in treatment.

The Relationship between Automatic and Voluntary Movement

Information, perception, and cognition are all important for action. Umphred (1991) states that motivation, challenge, and success are cognitive elements, even if we are not consciously aware of such feelings during everyday movement. She also states that visual and vestibular systems play an essential part in cognitive analysis and recall of previous experiences, and that these lead to learning and semi-automated movement. Whiting and Vereijken (1993) are of the opinion that the CNS is self-organizing and solves its motor tasks here and now as a response to environmental requirements, without cognitive attention. Luria (1989) states that cognition is the same as thinking, and refers to several stages in this process:

- Confronting the task
- Assessing and evaluating the terms and components of the problem

- Choosing a solution from many possible solutions
- Choosing an appropriate way to solve the problem
- Using the preferred choice
- Operative stage (action)
- Comparison between the result and the original terms of the task

The role of *cognition* in movement is undisputable, but on which level cognition is most involved will vary depending on the requirements of the task. Luria and Umphred emphasize conscious thought in the planning of motor strategies when attention to the movement is important, whereas Whiting believes that cognition is an awareness of movement within our CNS without reaching a conscious level.

Motor learning seems to require the same CNS processes irrespective of the CNS being healthy or damaged (see Chapter 1.2, Plasticity). The difference lies in the patient's CNS ability to receive information, to process the information, and to recruit the appropriate activity. Brodal (2001) talks of *least automatic* or *most automatic*, which seem to be more appropriate terms than voluntary or automatic; there are elements of cognition in all activities. The differentiation between conscious thought (least automatic) and awareness (more automatic) is clinically relevant, even if both these aspects have elements of cognition. We are more conscious when we learn a new complex task that demands precision than of the background activity of postural control and balance that allows improving precision. When learning how to play tennis, we are consciously aware of the handgrip on the racket, the direction and rotational components of the strokes, and the visual feedback from the flight of the ball in time and space. Our conscious thought is not involved in all the background activity of the body; the actual and immediate adaptation and adjustments that need to be in balance to perform the skilled movement at the same time. We are more concerned with reaching the goal than in the processes involved to be successful. When basic skills are learned, we develop the memory to recall the experience, i.e., how the movement *feels*. This feeling seems to be based on the comparison of expected performance (previous experience) and actual performance, i.e., perception, and seems to be closely linked to the feeling of success.

The CNS has a capacity for dual or simultaneous activity, i.e., to do two or more things at the same time. Walking on a busy street, jumping from one stone to another in the mountains, or shopping require that we can receive and process many different types of information at the same time as we move and act appropriately. We are aware of the goal, of people around us, the complex environment or things that happen around us, as long as we are in balance. We are able to perform two functions at the same time normally; both walk and talk, shop and move around to pick things down from shelves at the same time as reading the shopping list, dress and undress while standing, shower and soap our bodies, and do mathematical calculations while walking. Our attention is not focussed on walking but on the simultaneous functions. This is called *simultaneous activity* or *dual task* (Mulder et al. 1996). Many routine and more automatic activities may, however, be brought to conscious awareness and controlled through focused attention, concentration, and will power if the person deems it necessary. The CNS problem-solves tasks in different ways depending on the situation.

Following neurologic damage or, for instance, lower limb amputation, the ability for simultaneous activity diminishes. When balance is threatened, the focus of our attention is pulled away from the task to how balance can be preserved to avoid falls. Many patients use conscious effort in an attempt to preserve balance. If their attention is disturbed—a ringing phone, a kettle boiling, other people moving in the vicinity, they are in danger of falling and may hurt themselves.

Postural control is one of the most automated functions of a healthy CNS (Mulder 1991, Dietz 1992, Massion 1992, 1994, Mulder et al. 1996, Horak et al. 1997, Shumway-Cook and Woollacott 2007, Brodal 2004). Postural control is the foundation for selective activity of the extremities, and postural stability is a prerequisite for movement control and the ability to vary movement. Movement and postural control are closely integrated. Movement of the extremities requires adjustments of the postural mechanisms, both before (anticipatory postural adjustments), during (ongoing), and in response to (feedback) the movement. Trunk adjustments are more automatic and are learned during childhood, as move-

ment and the position of the center of gravity have to be controlled simultaneously (Massion 1994). The hands and feet interact more directly with the environment, and are classified as least automatic elements of normal movement control. The movements of individual fingers for precision of movement is least automatic and mostly voluntary, while the posturing of the hand and wrist along with the postural control of the arm and body are mostly automatic and less voluntary. Movements range from least to most automatic.

Examples of skills are writing, throwing and catching a ball, cycling, and driving a car. All these are based on an appropriate postural background. Skills become more automatic as they are learned, and require progressively less attention. Many (over)learned routine-based basic tasks such as balancing, ADLs, walking, reaching, need little or no conscious thought or attention. The term *overlearned* was used by Mulder et al. (1996).

Everyday activities such as balancing, walking, reaching, and eating are mostly automatic functions that normally require little attention and effort.

(Over)learned activities and balance seem to have a structural correlation (form-function; see Chapter 1.2, Plasticity) in the CNS, developed through activity-dependent interaction with the environment, i.e., experience. *Structural* is **not** the same as unchangeable or fixed. The CNS is not stereotyped or rigid. The ability to vary is great and depends on the situation in which the motor activity is being performed. Possibly, overlearned actions are structured like prototypical representations (Mulder 1991; see Chapter 1.1, The Spinal Cord and Brain, The Cerebellum). New skills, from the first attempt (willed, attention demanding) to initial control (semi-automated) to acquired skill (automated) exist in a landscape between functional and structural plasticity.

Everyday activities have a structural correlation in the CNS, based on experience.

The expression of activity varies depending on the individual, the goal, and the situation.

In situations where there are increasing demands for adjustment, for instance when the base of support changes or is moving, when objects are in the way, when the shirt buttons are small or the button holes are too small, or when the sock on the foot is twisted, our attention is increasingly focussed on the task until the problem is solved. Once solved, the progression of the activity is more automatic again. Cognitive regulation, visual information (eye-hand contact), and sensorimotor adaptation are all important to skill acquisition, especially to hand function. More and less automatic movements are closely integrated, and humans switch between these controls depending on how easy or difficult, known or new the task is.

Automatic and voluntary control of movement are closely integrated and form the basis for functional skills and balance.

Walking has both cognitive and more automatic elements. The initiation of gait, alteration of tempo, speed, and direction, attention to obstacles, people, and unevenness are the more cognitive elements. The cognitive elements are not focused on the actual motor strategies that are used, but related to problem-solving the initiation of gait, the goal, and the environment. Walking is at its most automatic in unchallenging environments if there is no need to change (see Chapter 1.1, The Spinal Cord, Central Pattern Generators). After the initial step, the steps follow more automatically as the line of gravity falls in a controlled way outside the base of support and the individual steps to regain balance. The trunk moves forward and up, the legs follow, i.e., a cranial to caudal recruitment of activity.

Walking may be consciously controlled if we so wish. Please follow the instructions below closely before you read on:

- Stand up
- Place your feet parallel with each other
- Flex the right hip and knee, lift the leg and stretch out the leg
- Place the heel down on the floor
- Transfer weight on to the right leg and straighten the right knee
- Flex the left hip and knee, lift the leg and stretch out the leg

- Place the heel down on the floor
- Transfer weight on to the left leg and straighten the left knee
- Flex the right hip and knee, and swing the right leg through
- Place the heel down on the floor
- Then please go back to your seat

The question is, did you use the same movement strategies when walking under instruction as when you went back to your seat. Usually people experience a big difference. Experience suggests that other motor strategies are used during verbal instruction than used during normal effortless gait. The first step is mostly voluntary, and attention is focused on the actual movement as well as the goal. Detailed instruction either internally generated or externally generated by a therapist increases conscious attention toward controlling components of movement that are not voluntarily controlled or directed in a normal situation. When movement is instructed, the sequence of components seems to be reversed (after the initial step); the leg moves in relation to the body, and the line of gravity lies behind the moving leg (normally the body moves in relation to a standing leg). The patterns of movement in the swinging leg are characterized by greater degrees and earlier initiated flexion over the swinging hip than during normal walking. In this example, the trunk moves after the legs, i.e., a caudal to cranial recruitment. The recruitment sequence is reorganized and the flexor activity increases, efficiency is reduced, it takes more time, and the physical and cognitive effort increases. The use of verbal instruction to recruit the activity of individual muscles, muscle groups, or isolated components may override automaticity and alter the recruitment sequence compared to normal function.

Postural control is based on vestibular, somatosensory, and visual information. The weighting of the relative importance of these sources of information depends on the actual situation. Patients who have a CNS lesion often have reduced, inappropriate, or limited anticipatory postural adjustments (feedforward) (Baykousheva-Mateva and Mandaliev 1994, Dickstein et al. 2004). Mulder et al. (1996) studied improvement after CNS lesions and stated that: "From the work performed in Nijmegen during the past 5 years, three principles of recovery can be distilled: (a) a decrease of cognitive regulation; (b) a decrease

in visual dependency; (c) an improvement in sensorimotor adaptability."

People with reduced balance become more dependent on vision and attention, even during more automatic functions such as unchallenged walking. If visual information dominates, information via other channels equally important for balance—somatosensory and vestibular systems—is in danger of becoming neglected by the CNS. Vision is closely related to cognitive control through regulation and focusing of attention (see Chapter 1.1, The Somatosensory System, Vision, and Balance, Vision and the exercise instructions at the bottom of page 114). The patient's CNS may stop listening to signals from the body, tempo and balance reactions are reduced, and the sequence of recruitment of neuromuscular activity reorganizes.

Clinically, the factors mentioned by Mulder et al. (1996) may be used as treatment interventions if the patient has some balance control but is too cognitively regulated:

- Distract the patient by giving him a cognitive task, progressing to mental tasks containing spatial elements, e.g., describing the interior of his house or flat in detail
- "Blinding" the patient by asking him to close his eyes or use nonsee-through glasses and sense where he is to improve perception
- Improve the patient's sensorimotor adaptation by, for instance, specifically mobilizing the structures of his feet, improving flexibility and muscle length, improving alignment, and introducing gradual weightbearing combined with functional tasks working for dual task interaction

During assessment, the therapist collects information through observation and handling, and forms hypotheses about why the patient moves as he does. The reasoning process for the therapist involves deciding which are the patient's main problems; reduced stability or postural control, or more mobility problems. The focus may change as treatment progresses. If postural control is most affected, it might seem logical to facilitate regaining this through more automatic processes, i.e., not use specific verbal instructions to maintain balance. Appropriate interventions are specific choices of postural sets, nonverbal demands on the patient's postural control by introducing a dual task (throw a balloon, roll a ball, move a glass of water), free arms, especially

facilitating or supporting the arms above shoulder level, in standing, standing to sitting, and in sitting, at the same time as alignment and muscle function are optimized (for individual examples, see Chapter 4).

If the patient has some postural control and balance mechanism combined with normal cognition and therefore the ability to problem solve, but has problems in the recruitment and initiation of selective movement, other interventions may be more appropriate, for instance, the use of verbal instruction combined with facilitation of more optimal alignment during a relevant functional task. In some situations, the specific focused attention toward detail, stimulation, and facilitation of muscles may improve the patient's awareness and body image, and result in better control of movement as a preparation for task acquisition.

Many patients with CNS lesions have reduced cognitive ability and/or perception. The interventions have to be made appropriate to the patient's ability—to what he responds best. For instance, if a patient suffering from inattention or neglect makes eye contact with a body part that is being moved, or stimulated, it implies that this intervention raises the patient's awareness, and the possibility for integration of information from that body part is strengthened, improving the patient's body image.

It is important for the clinician to determine what level of cognition should be focused on during treatment of the individual patient. Verbal instruction lifts the problem solving to a conscious level, which is not always appropriate. How much conscious thought should the patient put into movements and activities that in a healthy person would be more automatic? Should the therapist instruct or not? When should the therapist use verbal commands? What about mental imagery and practice? These questions are relevant for the clinical reasoning process.

The clinical challenge is to decide whether balance can be regained through conscious voluntary planning, or be facilitated on a more automatic level in functional situations. Tone, muscle dynamics, alignment, and sequence of recruitment must be optimized in both scenarios.

Handling

Handling refers to the physical contact between patient and therapist in a treatment situation, and is not limited to the therapist's use of hands. Therapists have been interested in the influence handling may have on the patient's development of independence in balance and movement. Some professionals claim that handling may hinder the patient's own development of movement strategies because handling may act as an external support. It is claimed that an external support such as a brace, splint, or walking aids or another person, may stop the patient in exploring his relationship with gravity. According to Gentile (1987) the therapist should be an instructor and adapt the environment to the patient to allow him to explore (see Postural Sets and The Relationship between Automatic and Voluntary Movement).

Clinical experience underlines the importance of *appropriate* handling. The important question is *how* and *why* handling is used in the process of the patient's regaining and relearning of independence. Jeka (1997) and Jeka and Lackner (1994) studied the effect of external supports on patients' postural control. Jeka (1997) states that it is difficult to draw clinical conclusions from this research. However, it is interesting to discuss these findings in relation to handling. The researchers found that when the trial participants had fingertip contact with the environment, their postural activity changed. In these trials two different ways of using fingertip contact with a metal stick that stood firm, was examined: (a) weightbearing or leaning on the stick and (b) light fingertip touch. Weightbearing or leaning on the support reduced postural activity in the participants. Use of such an external support causes sensorimotor reorganization of activity by, for instance, changing the sequence of muscle activation. If the participants only touch lightly, they receive information through their fingertips and increase their postural activity. Light fingertip touch gives additional information to the CNS, more than vision alone. Touching the environment may orient the body and improve perception of the relationship between the body and space. Postural activity was greatest when participants did not use any external support (Jeka and Lackner 1994).

The influence of peripheral stimulation on movement has been studied both in animals and in people with spinal cord injuries (SCI) (Muir and Steeves 1997). During training it seemed to be important that movements were performed as normally as possible to train and modify spinal circuits for specific motor tasks. This article demonstrated that increased peripheral stimulation through manual and electro-stimulation techniques improved movements of the extremities after SCI. Schultz (1997) referred to research demonstrating that the excitability in the spinal reflex arc is reduced through massage in healthy and traumatic SCI (the article does not specify if this refers to complete or incomplete SCI). As well as these neurophysiologic effects, massage has a positive effect on muscles, tendons, and connective tissue. Leivseth, Tornstensson and Reikerås (1989) examined people suffering from atrophy and loss of range of movement due to osteoarthritis. The patients received specific stretching of muscles without affecting the joints or ligaments during treatment. The study found that specific stretching may cause changes such as increased muscle fiber diameter, fiber length, and improved range of movement.

Many other studies stress the importance of somatosensory information for standing control (Mayer et al. 2004), locomotion, reach, and grasp (MacKay-Lyons 2002) and postural control (Magnusson et al. 1994, Morningstar et al. 2005). MacKay-Lyons (2002) state that there are potentially three different roles for afferent feedback and that all involve adapting movement to the internal and external environments: (a) reinforcing the activities of the central pattern generator (CPG), especially those involving load bearing muscles; (b) timing function, whereby the sensory feedback provides information to ensure that the motor output is appropriate for the biomechanical state of the moving body part in terms of position, direction of movement, and force; and (c) facilitating phase changes in rhythmic movements to ensure that a certain phase of the movement is not initiated until the appropriate biomechanical state of the moving body part has been achieved. Handling provides somatosensory information to the patient, and may therefore enhance, facilitate, or hinder the patient's development of postural and movement control, depending on how it is used.

The skin is our largest sensory organ. Skin, musculature, tendons, and connective tissue have abundant specific receptors that continuously inform the CNS of the state of the body. During handling, either through the therapist's hands or through other body parts (shoulder, knee, hip, etc.), a stream of information flows between the patient and the therapist. Physical contact through skin and musculature leads to intimate and intense communication between the two, which should not be misinterpreted. The therapist both receives and gives information through handling. When the patient moves or is facilitated to move, the therapist receives information about the patient's ability to respond, initiate, and move, and how he moves, i.e., recruitment of activity both locally and generally. If the therapist optimizes the patient's alignment locally to enhance muscle function, for instance by aligning the patient's pelvis more appropriately to the base of support in sitting, the therapist can assess the patient's response to handling in general.

Two of the therapist's most important assessment tools are her eyes and her hands. The most important part of handling is "listening" to the response. Figuratively, our hands may see around corners. Stereognostic sense means that we can identify objects through touch alone by picking up information about texture, temperature, and firmness and comparing this with previous experience to identify what it is (see Chapter 1.1, The Somatosensory System, Vision, and Balance, Stereognostic Sense). Therefore, the hands have the ability to both listen and see. Therapists need to improve and extend this skill for their interaction with patients. The therapist receives information through her hands (and eyes) about:

- Local aspects:
 - weight distribution
 - alignment
 - muscle qualities, which may give rise to hypotheses about tone, flexibility, elasticity, activity, and adaptability or become the foundation for ideas about activity
 - quality of other soft tissues in the area
 - skin quality and temperature
- This information is received through the direct, local contact.
- General aspects:
 - tone distribution
 - reciprocal innervation—interplay
 - patterns of movement

The therapist's hands form part of the patient's base of support. If the patient is sitting, the therapist may adapt her hands to the patient's musculature in the pelvic area. By using her hands, the therapist may gently move the patient in different directions, sideways, forward, and backward, and introduce rotatory components and assess the patient's righting ability in response to changes in the support base (the hands) and movement of body segments in relation to each other. The therapist observes, listens to the response, evaluates, and forms hypotheses about the characteristics of the key area and the interplay between key areas.

Touch may be one of the strongest direct influences on the patient, physically and psychologically/emotionally. The therapist therefore has to take great care in how she introduces handling to the patient, and what information she gives to the patient. The patient has to accept handling for it to be effective. Through her hands and body language the therapist must impart empathy, respect, and care. The use of handling is based on clinical reasoning, problem analysis, hypothesis formulation, goals, and which tools to use to help the patient to achieve his goals.

Many patients suffering from CNS lesions have paresis, weakness, altered or reduced somatosensory input and perception, reduced coordination, and dexterity, and are unable themselves to recruit appropriate activity in good alignment for task achievement. Malalignment may be in relation to the base of support, a body part in relation to other body segments, within a body segment, or between distal and proximal. If the patient is unable to create alignments that promote appropriate muscular activity or activate appropriate muscular strength, handling may be used to facilitate this. Specific mobilization of muscle and other soft tissues combined with enhanced somatosensory input in better alignment may improve the performance of the motor task. Handling is used to give the patient information, perception of movement, and movement experience, aiming to mimic how the patient used to move before the lesion, and thereby reawaken memory of experiences and the feeling of "how it felt." Handling should cause a feeling of something recognizable by the patient and relate to familiar movements, activities, and functions.

Therapeutic handling is dynamic, specific, and varied; it may be mobilizing (musculature, joints),

stabilizing, and/or facilitatory. In treatment, *handling* should never be static or stereotyped and it is not the same as massage or stretching, but may have elements of both. Occupational therapist Christine Nelson says the following about Berta Bobath, "I observed in her hands all the tissue mobilizing skills that have now become specialties" (cited in Schleichkorn 1992).

Handling may be corrective, supportive, informative, leading, stimulating, or demand movement. The hands are the most mobile parts of the body. The hand depends on inherent mobile stability, and the stable reference areas for movement vary depending on the task, for example, the neuromuscular activity in the thenar eminence and the metacarpal of the thumb, over the wrist and metacarpophalangeal joints as in the lumbrical grip, the hypothenar eminence and the index finger for the precision of the thumb and index in a precision grip, or a combination. The fingers are the mobile parts of the hand, the palm the more postural part. The postural activity and adjustment of the palm allows for variations in the use of the fingers. The therapist needs to explore and exploit these characteristics to invite the patient's neuromuscular system to be more appropriately active. The hands must shape themselves to the contact area and give comfortable, yet stimulating information.

Handling is achieved not only through the therapist's hands. The therapist may use her other body parts in contact with the patient to facilitate stability in one key area and movement in another to promote the relationship between stability and movement, and postural control and movement. The hand may function as a dynamic support and recruit stability in postural sets requiring postural activation. The hands should mimic the function in the area to be facilitated; if the patient has reduced hip stability, handling should impart activity to the abductors and extensors.

The therapist's hands may touch, create friction, stretch, compress, and give information about muscle length and tension, direction, speed, and range. They may produce traction, compress or rotate, demand stability, and/or mobility depending on the problem and the functional goal. Information is specific to the desired activity.

The aim of using handling as a treatment tool is to recruit neuromuscular activity in a functional context. Clinical experience supports the theory that postural activity and control as well as the control of movement may be improved through handling.

Some patients do not accept handling. Sometimes, due to perceptual problems, they do not understand the information given and cannot relate to it, or they dislike the physical contact and may feel that it invades their personal space. Handling must then be on a minimal level and the patient clearly informed of why—for safety reasons, for instance. If the tone increases or there is tension as a response to what the therapist would deem as appropriate and relevant handling, it works contrary to its aim. This is quite rare, but needs to be respected. If the therapist is professional, empathetic, explains everything, and is careful, most patients are receptive to handling both as an assessment and a treatment tool.

Facilitation

Berta Bobath stated that facilitation means “making easy” but in treatment it also means “making possible” and, in fact, making it necessary for a movement to happen (unpublished material from 1988, cited in Schleichkorn 1992).

Facilitation means “making easy.” The aim of the therapist is to handle the patient in such a way that movement feels easier for the patient because the patient’s own activity is recruited. In this context, facilitation must not be interpreted to mean passive movements or passive techniques such as the use of tapping over muscle or stimulation using ice.

Handling is founded on the following principles:

- *Make possible* (realignment, information)
↓
- *Make necessary* (demands) }
↓
- *Let it happen* (activity) } Facilitation

To Make Movement and Activity Possible

To achieve this, the neuromuscular activity and biomechanical relationships have to be as optimal for the actual movement as possible. In this process the therapist works to improve the patient’s underlying impairments. This phase of treatment prepares for facilitation, and contains all previous elements mentioned earlier in this chapter: choice of postural sets, key areas, selective components to functional activity, the relationship between automatic and voluntary activity, and handling.

An individual with a CNS lesion may develop secondary muscular and biomechanical changes that hinder balance, the ability to transfer, locomotion, and functionally active and free arms. The musculature is plastic and adapts to how it is being used (see Chapter 1.1, The Neuromuscular System). Many factors influence muscle function, e.g.:

- Tone
- Positioning
- Information to the muscle from the CNS and peripheral systems
- Altered use or nonuse
- Altered alignment
- Circulation
- Connective tissue adaptation (contractures or increased compliance/hypermobility)

“Shortened muscles are recruited more readily than their elongated synergists and as a result are stronger” (Sahrmann 1992). Shortened muscles, either because of viscoelastic properties, inertia, tonus, activation, positioning, contractures, or reduced ability to eccentrically lengthen, seem to be recruited first during activity. Handling seeks to improve these factors.

Inhibition of spasticity/associated reactions/increased tone are terms used by many physiotherapists. *Inhibition* refers to neurophysiologic processes (see Chapter 1.1, Systems Control) and should not be used with regard to movement or the control of movement. The aim of treatment is to regain a more balanced interplay between excitatory and inhibitory processes in the CNS to improve coordination of muscle activation. Inhibition is an active neurophysiologic process that requires excitation to release inhibitory neurotransmitters. A hypotonic muscle has decreased ability to eccentrically lengthen. Clinical experience suggests that specific handling of

muscle may influence and enhance mobility, flexibility, contraction, and eccentric activity of muscle function. Handling is not classic stretching, whereby a muscle is stretched to its full length, but a treatment aimed at the intrinsic function of the muscle itself combined with facilitation of increased neural activation. Eccentric control is an essential element of this, the patient actively giving length. This form of mobilization is always combined with movement and is called *specific mobilization of muscle*. Treatment is combined with correction of alignment and leads into functional movement.

Clinical example

Many patients suffering from neurologic dysfunction spend a lot of time sitting.

Sitting may cause the thighs to roll into adduction and inward rotation. There may be a danger of developing shortening of adductors, inward rotators and flexors of the hips (Fig. 2.33). Recruitment of abductors and extensors to stabilize the pelvis during transfers, for standing and walking, may therefore be limited.

During treatment it may be necessary to mobilize shortened structures to improve alignment and facilitate muscular activity for muscle balance and stability.

As the range of movement, alignment, and muscle length improve, *in the same treatment session*, the transition to the next phase is made.

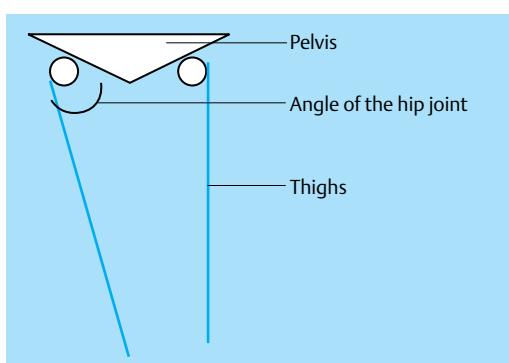


Fig. 2.33 Schematic drawing of the hip in sitting posture.

To Make Movement and Activity Necessary

The treatment moves into the activity phase, which implies that activity exercises are given to the patient: he may be placed in a postural set that necessitates control of the muscles that have been mobilized and facilitates their activation. The CNS is challenged into activity. The therapist's hands may facilitate key areas and stimulate activity to get a response. The situation is structured to allow the patient to move and respond without being afraid of falling. The aim is to gradually withdraw as the patient starts to take over, but the therapist may need to repeat input to increase the response to an appropriate level for the activity by facilitating neurophysiologic processes of temporal and spatial summation.

When movement is possible, the patient is challenged to explore the possibilities during activity, for instance by being displaced through weight transference while standing to necessitate the initiation of a step. The therapist uses her hands to invite the patient's muscles to be active in stabilizing the hip to free the opposite leg for swing phase. Muscle function and alignment are facilitated, as well as interplay between key areas. Handling needs to be specific in placing, timing, and transmission of information, to enhance the patient's own activation to make movement easier. Therapeutic handling may involve facilitation of components of stability, mobility, rotation through compression, traction, touch, or stimulation to move. The therapist's two hands have to give different inputs.

Facilitation is the bridge between assisting and stimulating the patient to recruit activity and his ability to take over and make the activity his own. *Placing* is a response to facilitation. It is the ability to automatically adapt to imposed movement and to support any movement by one's own activity. Placing may be stimulated through compression, distraction, rotation, movement, and touch, and it improves the proprioceptive knowledge of the position of the limb in the body schema. As a result the patient may move more actively. If the therapist asks the patient to hold a limb in space, she asks for a response that is the result of cortical feedforward, i.e., a voluntary movement. Placing is the automatic and active control in any phase of the movement (Bobath 1990).

Let It Happen

This is when the therapist allows the patient to respond to the challenge through his own activity by, for instance, taking a step. If the patient is unable to move his leg due to inactivity, another therapist may assist or facilitate the distal activity. Important factors are rhythm, tempo, and sequence of activation.

Facilitation has happened when the therapist removes her hands or reduces input significantly (there may still be weakness in the relevant muscles). It is important to recognize the right moment for taking off the hands. This is a very challenging part of the treatment. The aim is *hands-off*; to enable the patient to take over control. The patient has to be able to initiate movement without intervention to regain independence. By using her hands to give intermittent information, for instance intermittent light compression to facilitate stability, the therapist may feel and observe when the patient is ready to take over. There is continuous interplay between *hands-on* and *hands-off* as long as it is needed. When the patient takes over control, the therapist removes her hands or the input that the hands have given. The patient is encouraged to experiment with his regained control; the therapist may give intermittent stimulation as a reminder of where the patient is supposed to control, or to enhance and strengthen the response. Too much hands-on or static use of hands may make the patient passive.

Treatment requires a continuous interplay between working on impairments and facilitating activity, making movement possible, demanding control, and encouraging action: Make possible → make necessary → let it happen.

The three stages *make possible* → *make necessary* → *let it happen* are closely integrated in treatment. The therapist does not wait for—or expect—alignment and muscle function to have normalized before the patient is required to activate and control. As soon as the patient acquires any level of control, the new possibilities have to be used in a functional context to facilitate the patient's own experience through more appropriate recruitment of motor activity in function.

The aim of handling is to enable the patient to be more active so that the therapist's hands can be taken away.

The therapist may use a number of different tools to aid the patient toward regaining control. Among the different tools and environments Berta Bobath used to facilitate patients were large balls. These soon became known as the Bobath balls. Berta Bobath did not like this, and said "Once a Japanese doctor asked for permission to use the 'Bobath ball' in a publication. It is a beach ball, not a Bobath ball. What makes it Bobath is what you do on it" (Schleichkorn 1992). Later she expressed concern as to how the ball was used. "She still, however, has very valid concerns that people misuse and overuse the ball and associate it too closely with being her therapy, rather than just another tool to achieve specific aims" (Schleichkorn 1992).

Clinical example: facilitating stepping

During locomotion there is a constant change of activity during phase changes; the standing leg becomes the swinging leg, the swinging leg becomes the standing leg. The neuromuscular activity especially related to the pelvis, hips, and legs continuously changes from more stability during stance, to letting this activity go to allow swinging of the same leg (Fig. 2.34).

In walking forward on even ground, initiation of swing depends on several factors, for example:

- Postural control, which depends largely on the stability of the contralateral leg
- Quality of stance prior to the swing on the same side
- Speed, propulsion, direction of movement-momentum
- Ability to overcome inertia
- Ability to eccentrically lengthen the extensors and other muscles involved in creating stance
- Trunk stability to counteract the movement of the swinging leg
- Selective movement, acceleration, and deceleration

Gravity and forward momentum assists the swinging leg, and the main neuromuscular activity in the leg during this part of swing phase is normally eccentric. The leg is not consciously



Fig. 2.34a



Fig. 2.34b

Figs. 2.34a–e **a** During facilitation of stepping the therapist has to ensure that she is not in the model's or patient's way. The movements of the proximal and the central key areas are small and continuously changing and adapting.

b Facilitation of the hips and pelvis. The therapist's hands have to mimic the activity of the muscles that normally stabilize the pelvis over the standing leg in this situation. In stance phase, the pelvis moves in relation to the hip joint through extension of the hip. The therapist facilitates stability through light compression of the musculature together with a small

input to lift, to gain height, over the standing leg to facilitate the movement of the hip in relation to the pelvis at the moment when weight transference is supposed to happen. The therapist's focus is on the stance leg to enhance stability and postural control to enable the patient to free the swinging leg. The therapist's hands are constantly changing. The swinging leg may be facilitated to eccentrically lengthen hip extensors. At the same time, the patient's center of gravity is slightly displaced in the direction of movement to necessitate the stepping through.

and actively lifted through concentric flexion when walking. The most cognitive part is the initial step and the intention of walking; the steps that follow are more automatic. Bussel et al. (1996) studied paraplegic patients attempting to step and walk, and found that the flexor reflex seemingly inhibited central pattern generation. This fits well with clinical experience with patients with other CNS lesions (MS, stroke), where initiation of swing too early seems to destabilize the standing leg. This could be caused by several factors:

- As many patients with CNS lesions sit for hours every day, their hip flexors may become short, hypersensitive to stretch, or lose the

ability to eccentrically lengthen in the last part of stance as a phase transition is about to occur. The early swing would be initiated more as a reflex reaction in this case

- Patients who have difficulties in walking often seem to be more preoccupied with lifting the leg to initiate swing. This strategy increases the cognitive aspect of walking, as well as reversing the recruitment order. Normally, the CNS is more concerned with sustaining stance than with initiating swing. We have to have a leg to stand on to be able to walk

Treatment will be very different in these two examples. In the first scenario the therapist needs to mobilize the shortened (stiff and contracted)

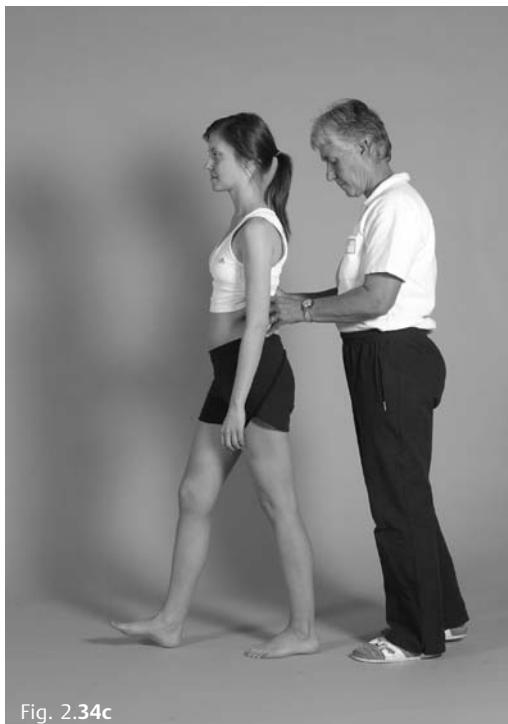


Fig. 2.34c



Fig. 2.34d

Figs. 2.34c–e

c, d Facilitation of stepping through the central key area. The therapist facilitates extension on the weight-bearing side at the same time as the thorax is stabilized bilaterally to allow the freeing of the swinging leg. The centre of gravity is displaced in the direction of movement to facilitate pattern generation. The facilitation needs to mimic the model's internal rhythm and tempo.

e Facilitating a change of direction via the pelvis and hips. Extension through slight compression into the musculature and a small input to lift and gain height on the standing leg together with some rotation of the swinging side of the pelvis facilitated a change of direction of movement. As soon as the swinging leg has passed the stance leg and is approaching heel-strike, the therapist stops facilitating the stance leg to allow the heel-strike, getting the foot to the floor. The swinging leg is thereby ready to become a standing leg again.



Fig. 2.34e

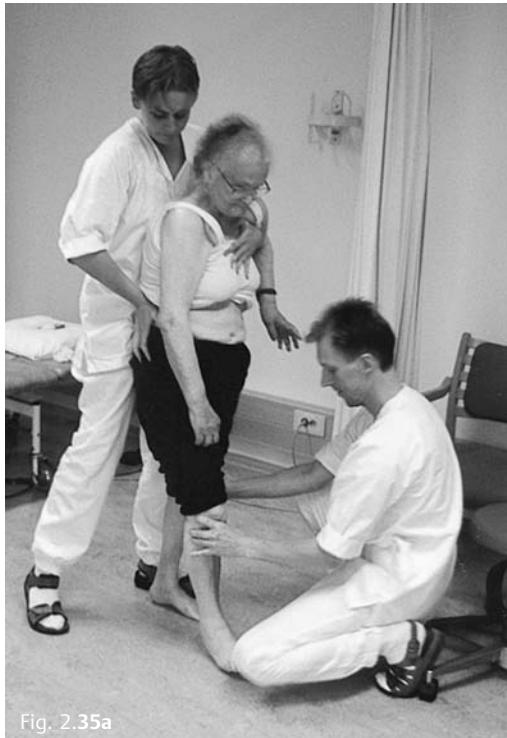


Fig. 2.35a



Fig. 2.35b

Fig. 2.35a, b

a The patient is being helped to achieve postural activation and to stay upright through the therapist's handling of her central key area. She has at this moment no stability or selectivity of her most affected side, and the assistant helps her stance phase first, then the transition to swing. The challenge is to coordinate the timing of the three people for rhythmical activation and phase transitions to facilitate central pattern generator activity.

b The patient has regained some stability and selectivity of her right leg, but her postural tone and core stability is reduced. The therapist brings both arms into outward rotation from the shoulders to free thoracic extension in order to enhance coordination of the trunk. She is helped to right herself and to achieve postural activity through the central and proximal key areas. Note the inappropriate gripping of both hands, which may be a compensation for proximal instability and reduced postural control.

tissues, to desensitize the flexors, and facilitate the patient's control of eccentric activity, followed by relearning components of locomotion and the locomotor patterning. In the second example the patient has to unlearn lifting the leg too early; to learn to focus on acquiring a good stance on both legs (one at a time) to prepare for swing, and to learn the distal activation of the foot-toes-off to lifting the toes to evolve swing—and then be facilitated through his individual rhythm to gain activation of the CPG. Treadmill training at a relatively fast speed may be useful for this patient to take away his increased cognitive regulation.

Experience suggests that it is possible to facilitate stepping even in patients who have severe neurologic deficits and little or no volitional control of the most affected side, such as in the early stages after a stroke (Fig. 2.35). The facilitation of early stepping is strongly indicated in neurologic patients to maintain the memory of walking and CPG activity, to facilitate postural control and the patterns of movement. The patient has to be facilitated through good alignment and with focus on stance initially. Bringing the patient early into a stepping rhythm may facilitate a responsive swinging of the opposite leg, which provides the foundation for stronger perception and body

image of the most affected side and further volitional control of the leg in different situations.

The prerequisites for this to happen seem to be:

- The patient requires help and facilitation to remain upright to oppose gravity (make possible)
- Mobile feet to allow weight transference onto and through the foot (make possible)
- Optimization of alignment to enhance appropriate muscular activity (make possible)
- Facilitation of selective hip stability on the stance leg immediately prior to destabilization
- Destabilization of the patient's center of gravity in the direction of movement (make necessary → let it happen)
- Allowing stepping
- Facilitation of rhythmical interchange without cognitive override (let it happen), as close as possible to the patient's own individual rhythm. If the stepping is too slow or too fast, the patient's CPG activity may not be facilitated

Reduced interplay between the two halves of the body after a stroke generally has a strongly negative influence on postural control and also on the stance activity on the *least* affected side. The patient has problems in weight transferring, and stabilizing and balancing over the least affected side, which seems to influence the acquisition of swing on the most affected side. If stance activity is reduced, the patient may have to weight-bear on both sides, and the release of swing may not be possible. Patients need to work on stance phase on both sides to regain postural control and stability to acquire CPG activity independently.

Active Movement; Learned Nonuse; Neglect; Passive Movement

"Motor activity is the tool of sensation" (Brodal, personal communication, 1998). Two aspects seem especially important for motor control: the memory of (i) how it felt to perform a specific movement or action and (ii) the result. "The task is carried out and modified by adjustments, so that the sensations from the evolving movement match the memory of 'how it felt'" (Brooks 1986). Berta Bobath (1990) also said that, "The hemiplegic patient, just like a normal person, does not

learn movements, but the 'sensation' of how the movement felt."

The peripheral nervous system (PNS) is still intact after a lesion to the brain or a spinal cord injury. The CNS receives all somatosensory information, which is integrated to a certain degree in the spinal cord. The CNS therefore still "feels" although the patient may have severely reduced perception of sensation. Sensory deficits may be due to lesions to ascending systems, perceptual deficits, or learned nonuse.

Active Movement

Movement generates feedback from the body to the CNS from specific receptors and vision as well as information of the result. Active movement causes a diversity of information within the CNS. The length-tension relationship changes, receptors in skin and soft tissues and joints are stimulated to transmit information to the CNS (see Chapter 1.1, The Somatosensory System, Vision, and Balance). This information gives us something to feel and therefore perceive. Exploratory behavior, for instance using the hands to touch our close environment and our own bodies, facilitates the perception of the body in relation to the environment and itself. Shumway-Cook and Woollacott (2007) state that "Perception is essential to action, just as action is essential to perception." These authors define perception as the integration of sensory impressions into psychologically meaningful information. If a patient is unable to move he receives little or no information from his own body and is unable to explore his environment through his own body, and perception may be disturbed as a result. Improved movement may improve perception, and improved perception may have a positive effect on movement control.

Yekuitiel and Guttman (1993) describe a trial in which stroke patients with reduced sensation of the affected hand received a systematic training program more than 2 years post stroke. The patients were treated in their own homes—45 minutes each session, three times a week for 6 weeks. The patients had to identify touch to their own arm, find their affected thumb, discriminate between different objects placed in their affected hand and draw with help from an assistant (document assisted). Compared with a control group, they showed significant improvement

on all sensory tests. Some patients experienced functional improvement even if they were not encouraged to use it more in daily activities. This study demonstrates the close relationship between experiencing sensory stimulation, perception of sensation and motor function.

■ Learned Nonuse

Learned nonuse or disuse is associated with conditions where reduced motor control, for instance of a hand, results in the patient not using his hand. Patients use what they are able to recruit, therefore body parts that function may compensate for loss of sensation or movement in non-functioning body parts. Stroke patients quickly learn to compensate by increasing the use of the less affected hand. If the affected hand is not used, it will not be stimulated, little or no impulses are transmitted to the CNS, the arm is therefore not stimulated and is progressively more passive. This development predisposes the patient to secondary soft tissue changes and learned nonuse because the arm does not experience stimuli (Ada and Canning 1990). Learned nonuse may contribute to the changes in cortical maps after a CNS lesion (see Chapter 1.2, Plasticity). Nudo et al. (1996) state that if rehabilitative training is not present after a lesion, this may cause progressive area loss in relation to the functional representation of the affected body part. "In addition to the primary motor deficit, mechanisms of sensory inhibition are involved to varying degrees in the stroke patient's non-use of the affected hand, whether this is due to central neglect or learned non-use" (Yekuitiel and Guttman 1993).

Active movement provides a diversity of information to the CNS.

Active movement is essential to perception.

■ Neglect

Neglect is described as inattention to a body part or space. Patients suffering from unilateral neglect do not respond to incidents on the most affected side. Neglect may be observed in patients with CNS lesions, and most frequently in those with right hemispheric stroke. In the following therefore, *left* will be used to describe the most

affected side. Robertson et al. (1993) state that the most important aspect of treatment of *neglect* seem to be regaining of active movement in the affected extremities in the field of neglect. Several studies of neglect, (Lin et al. 1996), sensation (Yekuitiel and Guttman 1993), and motor function (Sunderland et al. 1992, Feys et al. 1998) demonstrate that active movement improves neglect. Sensory stimulation and intensive treatment improve neglect, sensation, and motor function. Increased motor activity and increased awareness are strengthened through positive feedback. It seems as if activation of the left arm in the left part of the room (in the field of neglect) changes lateral awareness and spatial representation (Robertson et al. 1998).

Experience supports this; unilateral focus and stimulation of the left side in treatment of patients suffering from neglect enhance the patient's awareness and concentration of the neglect field, i.e., perception. Through specific mobilization and stimulation of muscles and soft tissues, the correction of alignment, stimulation of the hand by contact with the patient's face and body or objects that he finds interesting, as well as facilitating the patient's postural control the patient's concentration and perception of the neglected side may be strengthened. Clinically, intensive proprioceptive and tactile stimuli seem to have a positive effect.

Progression during treatment is indicated by increased interaction and interplay between right and left. Bilateral simultaneous activity, such as carrying a tray or using one hand to support the activity of the other (e.g., opening a water bottle, cutting a piece of bread, holding a bunch of grapes while picking one off to eat), may be introduced once the patient is able to shift his focus between left and right with little prompting. If the patient "forgets" his left arm during bilateral activities, the focus has to go back to the left again. The therapist must emphasize that the patient keeps his focus in order to promote bilateral simultaneous processing of stimuli. Rhythmic interaction between right and left may have a positive effect. Therefore, in some patients, the facilitation of locomotion may promote interaction between the two body halves and thereby perception.

The therapist needs to continuously evaluate the patient's response and change strategy if awareness fails.

Facilitation of active movement through intense sensory stimuli seems to improve neglect. The patient's awareness increases during active movement.

In a clinical situation it is not possible to test and evaluate the degree and relative presence of somatosensory and perceptual deficits if the patient has severe motor problems as well. Only through treatment that focuses on sensation (feeling contrasts in different objects), perception, and the facilitation of movement and observation of the patient in different situations over some time, is it possible to formulate a more accurate hypothesis.

Passive Movement

If the patient is unable to initiate any activity, it is important to give the patient the sensation and experience of movement through being moved to:

- Avoid the development of nonuse
- Create awareness to the body part—stimulating a body part by moving it does cause some transmission of impulses to the CNS
- Give to the patient a feeling of movement and interplay
- Maintain motion, range, and circulation

The therapist aims to facilitate and stimulate activity even if the body part has to be moved passively, by drawing the patient's attention and focus if this does not improve spontaneously during treatment. The patient may be encouraged to create a mental image of the movement, or how the movement or activity was solved or used to feel. Mental imagery may strengthen or maintain connections in the CNS. If the patient is comatose, verbal information may strengthen the input, although most information needs to be transmitted through the proprioceptive and tactile systems. The PNS and the spinal cord are intact in patients with CNS lesions. Handling aims to make the patient's CNS listen and respond as far as possible, and is therefore not really passive.

Passive movement is important also for circulation, muscle length, and range of movement, which may allow development of the patient's own activity.

Passive movement is important if the patient is unable to initiate movement of his own.

Passive movement seeks to stimulate activity and requires the patient's attention.

Handling through passive movement aims to make the patient's CNS listen to the input and respond as far as possible, and is therefore not really passive.

Clinical example

A patient once said to me that *movement is the expression of the soul*. She had amyotrophic lateral sclerosis and was completely paralyzed. When a patient is unable to move by himself, his perception of his own body changes and diminishes. Patients who have a swollen, stiff, and inactive hand have a reduced feeling of contrasts in sensory input. The information they do receive about their hand is of an inactive weight, sometimes combined with pain. Some feel numbness, whereas others are unable to feel the hand at all even if the patient is able to respond positively to classic sensory testing. The therapist needs to re-create the feeling of mobility and movement, to move the hand as it would have been moved when active. Specific mobilization of muscles and joints, tactile stimulation, shaping of the hand to one's own body parts and different objects may enhance the perception of the hand as a part of the body. This way some of the prerequisites for more active movement are achieved (make possible).

Control of Associated Reactions

Associated reactions and associated movements were discussed in Chapter 1.3, Consequences of and Reorganization after CNS Lesions. Associated reactions are well-known phenomena in CNS pathology. They are viewed as a result of an activity-dependent process of learning whereby the CNS makes new connections and strengthens or weakens connections depending on their use. The interplay between the individual and his environment informs behavior and CNS processes and function. Associated reactions may develop as a response to impaired stability or movement or to hypersensitivity to stimuli. Over time, there is often a causal combination. Assessment and clinical reasoning allows the therapist to for-

mulate hypotheses both for the main problem and the trigger for associated reactions. Therapy aims to treat the patient's main problem(s)—the negative signs—and should not focus primarily on the associated reactions. A one-sided intervention aimed toward the so-called positive signs will not improve the patient's underlying movement problem, and therefore not facilitate his regaining of function. If the main problems are targeted, and the patient improves his motor control, the associated reactions may gradually diminish by themselves because they are no longer required. Sometimes, associated reactions may be so disturbing or destabilizing that the main problem is not accessible, and they need to be treated more directly. In these situations the associated reactions need to be changed or influenced to change, to access the patient's primary movement problem of, for instance, instability.

It is essential to analyze and treat the cause of the associated reactions and not only attempt to dampen down the reactions.

■ The Role of the Therapist

The therapist should:

- Form hypotheses of the causal relationship between the patient's main problem (weakness, instability, perception, and others) and the presence of associated reactions through observation and handling
- Choose a relevant and appropriate task for goal achievement
- Recognize which movement components are missing, then make it possible to achieve the goal through as optimal control as possible (note the importance of input, alignment, and muscle function)
- Create an environment that is conducive to the patient's control
- Make it possible for the patient to control his own associated reactions and deviant motor behavior by finding the right level of challenge
- Handle the patient to correct alignment and facilitate muscle activity to make the control of movement possible and necessary
- Inform the patient: increase his awareness and knowledge of himself in relation to possible causal relationships and the consequences of destabilizing associated reactions

■ The Role of the Patient

The patient should:

- Learn to control the triggering of associated reactions by focused attention if possible, and stop them from happening. The patient needs to be made aware that they are present and why. The patient's own control over his associated reactions is the first step in acquiring a wider movement repertoire and more selective movement control. The ability to adapt to different contexts is enhanced
- Learn to control the force, the timing, and the total involvement of the reaction

Some patients move relatively efficiently with mild associated reactions. Clinically these reactions seem totally automated and stable, and may be an expression of an established and relatively appropriate behavior modification (sensorimotor). They may be a nuisance to the patient, or they may cosmetically embarrass him. If the patient really wants to minimize them, he will have to be very motivated and focused, not only in treatment, but even more when he moves about himself. During a learning phase he will have to slow down and be more aware of how he moves, which requires a reduction in tempo and efficiency. The therapist has to assess whether there is a potential for change and if this change would enhance the patient's efficiency.

■ Feedback

Feedback may give the patient information of and about his movement, and may take many different forms:

- *Intrinsic feedback* is the information the patient receives through his own systems as a result of moving, vision, and somatosensory impulses:
 - Through the experience of movement and level of control
 - Through one's own feeling and observation of the level of success—was the goal reached or not?
 - Through handling from the therapist. This is both intrinsic and extrinsic, as handling in itself is imposing movement and information to the patient from the outside at the same time as response and adaptation to the handling transmits intrinsic feedback to the patient

- Extrinsic feedback is a supplement to the intrinsic form. It is verbal and visual (e.g., the expression and body language of the therapist) and has different aims:
 - motivating and encouraging
 - knowledge about the process of movement, i.e., about the performance itself (KP)
 - knowledge about the result (KR) of the movement

There is no consensus on type and timing of feedback, and several authors have debated this (Gentile 1987, Schmidt 1991, Shumway-Cook and Woollacott 2007). Most studies have been performed on healthy individuals. It is therefore difficult to extrapolate the findings to clinical settings and neurologic patients.

■ Intrinsic Feedback

Individuals suffering from CNS lesions have different physical and neuropsychologic deficits. Altered movement and perception of somatosensory information influence the patient's intrinsic feedback. There may be deficits in ascending systems, altered muscle tone, altered sequence of recruitment and sensorimotor organization, altered alignment, or perceptual problems. Perceptual and cognitive problems affect the patient's planning, feeling of movement, and movement experience. Their ability to integrate feedback and use this constructively to find appropriate and optimal solutions for motor problems will therefore also be affected. In many cases the patient is able to evaluate himself how successful the movement was, and may not need verbal reinforcement. The feeling that the patient gets when he feels that "this was right," or "this is what used to feel like," may be stronger than any verbal information.

Therapeutic handling provides the patient with feedback through touch and facilitation to move. Specific mobilization of musculature and correction of alignment allows the patient a better starting point for motor control. Through therapeutic handling the patient receives normalized information of the interrelationship between body segments and between the body and the environment. This may provide the patient with a better basis for successful goal achievement which both reminds him of what it used to feel like and uses memory to strengthen the patient's own performance.

■ Extrinsic Feedback

The therapist frequently uses a combination of intrinsic and extrinsic feedback. The type of feedback depends on the patient's problems, motivation, and cognitive ability. Knowledge of performance ([KP] Schmidt 1991) and knowledge of results ([KR] Schmidt 1991, Shumway-Cook and Woollacott 2007) both require the patient to perceive and integrate information and develop new strategies based on the feedback. Many patients do not have this ability because of perceptual or cognitive problems. These patients therefore cannot use KP or KR. Verbal feedback assumes that the patient is able to perform the activity differently through verbal information or commands. Many patients with a neurologic deficit do not have sufficient or appropriate motor function to alter or adjust their movement and are unable to change strategies. Internal feedback provides the CNS with information that is different from before and therefore gives a different basis for movement production compared with before. Their ability to recruit movement is changed; their ability to solve motor problems will be difficult if their previous movement experience is very different from their movement ability now or if their body image or perception is altered. Long or too detailed explanations are often confusing for patients and may hamper their feeling of the activity.

Through extrinsic feedback, movement planning and production may be lifted to a level of cognitive attention that should not normally be there for the activity. Motivating feedback through short words or sentences that are to the point may be more appropriate: Yes, stop, no, good, excellent, or similar expressions, may be enough to strengthen the patient's perception or provide the feedback he needs. Positive feedback is motivating and must be objective and honest. KP is provided about the actual performance, the process of movement. A patient with sensory problems or perception may find KP useful if he is able to understand and integrate the information and problem-solve from it. This requires that the patient has few or no cognitive problems. KR is knowledge of the end result of the movement. Many patients are able to evaluate their own achievements; they observe or feel the result of their own actions and do not need verbal reinforcement. Some neuropsycholo-

gic deficits may disrupt the patient's ability to analyze or understand the level of achievement; and such patients will need information about the results.

The feedback varies and depends on the patient's movement ability, his perceptual or cognitive function and the type of goal activity (more automatic or more voluntary).

■ Carry-over

Transfer of task and *carry-over* are terms for the effect of training into practical situations and tasks at home or on the ward. In the literature effective learning is related to *context*; that the activity is goal-oriented and used in different situations in the patient's daily life. Schmidt (1992) discusses the importance of varied training; the ability to perform the same skill in different contexts or situations. Carry-over may be analyzed on different levels:

- *Generalization*: transfer of movement components to different movements and functional activities
- *Performance*: the ability to maintain improved control from the beginning to the end of a treatment session
- *Learning or retention*: the maintenance of improved control between treatment sessions
- *Transfer or carry-over*: from treatment to ADLs at home or in the ward

■ Generalization

Movement is organized in relation to the individual, the task, and the environment (Shumway-Cook and Woollacott 2007). Action is organized in time and space to suit the task in the environment. Therefore, training needs to focus on variation, i.e., varying the components of movement in different contexts, environments, and requirement for control through the evolution of movement to ensure carry-over.

Training may be performed through *drill* or *varied repetition*. *Drill* is when the same component, movement, or activity is repeated many times in the same way.

Example

The patient may practice standing up repeatedly in the same way from the same chair. Although a person never performs the same activity in exactly the same way, there is very little variation in the combination of different movement components. If the person has very little problem-solving capacity, and therefore a reduced ability to carry over, drill may be necessary. One patient suffered a severe stroke, which caused complete expressive and receptive problems, severe apraxia, memory and problem-solving deficits, as well as severe motor problems. He was able to stand up from sitting in his wheelchair and to walk with personal assistance after several months of training. He was not able to transfer this ability to standing up from his bathroom at home, and had to be drilled in his own bathroom to succeed. Too much variation even within the same task may be too confusing for some patients. Generalization is not possible through drilling. The patient improves his performance only in what he practices in, the context he practices in.

Varied repetition is when the movement, postural set, and activity are varied for the neuromuscular component trained.

Example

Hip stability may be trained in supine, in different ways in supine using equipment and in different supine postural sets, through the activity of sitting to standing combining different alignments, different supports, and different heights, in standing, step standing, single leg standing, in stepping down from a high seat, in different transfers and ADLs, and during personal care. The focus is on facilitating hip stability, but the contexts are varied to create a wide repertoire of movement experiences, which may strengthen the patient's ability to carry over. Through variation, the prototype of the activity or component may be learned, thereby strengthening the transfer between tasks (see Chapter 1.1, Cerebellum).

Varied repetition in treatment allows the patient to develop a wide repertoire of movement and movement experience, which he may use in different functional contexts.

■ Performance: Transfer of Control from the Beginning to the End of a Treatment Session

Treatment is goal-oriented toward improvement of the patient's control of movement. The therapist evaluates her clinical reasoning and treatment in the same session. If the patient shows improvement in a component or activity level from the beginning to the end of the session, i.e., improvement in performance, he has achieved this level of carry-over. If carry-over has not occurred, the therapist has to reevaluate her clinical reasoning, her hypotheses, and the intervention.

■ Learning or Retention: Maintaining Improved Control between Treatment Sessions

If the patient's movement control remains the same from one treatment session to the next, he has not retained what he learned. The therapist has to consider the following aspects:

- Her clinical reasoning and choice of intervention
- Has the patient been allowed and encouraged to practice enough? A certain level of repetition is necessary
- Whether the patient is being met with conflicting demands: is he doing one thing in treatment while his daily life requires something different?
- Whether health professionals and carers are emphasizing conflicting components
- Whether the patient is compliant, i.e., is he able to understand and integrate information and follow advice

Bobath is reported to have stated:

- If the patient is unchanged, change the treatment. It has had no effect
- If the patient is worse, change the treatment. It may be inappropriate
- If the patient has improved, change the treatment. The patient is no longer the same

The change needs to be appropriate and not just for change's sake.

The patient should always feel that specific treatment leads to functional improvement within the same treatment session. The patient should not feel that the training does not meet his needs.

■ Transfer or Carry Over from Treatment to ADLs at Home or in the Ward

Berta Bobath said in 1988: "Our treatment does not consist of a number of exercises. We are preparing the child for daily life and this in functional situations. For instance, we treat him while he is being fed or when he feeds himself, while he dresses or undresses himself, or is being dressed or undressed. We treat the child while he plays, while he stands or walks, and so on. This is necessary to obtain direct carryover of the treatment into daily life" (cited in Schleichkorn 1992). The same principles apply to adult rehabilitation.

In our society it is a reality that most people suffering an acute CNS lesion are admitted to hospital. Many will be transferred to rehabilitation wards or centers after a short time. This environment is dramatically different from the home situation: lay-out, furniture, objects, size, or people whom the patient may have to share rooms with. If variation and generalization is attended to, it may be possible to achieve carry-over to the patient's own home. It is a challenge to use the available facilities for the benefit of the patient: training in a gym, on a ward, in the patient's room on the ward, on the stairs, and other environments inside and outside. Some patients may be motivated by staying in an institution for some time, because they meet other people in a similar situation to their own and may motivate, advise, and help each other. Treatment aims at improving the patient's control of movement in functional activities: during dressing and undressing, during transfers, walking, and using the arms, as well as directly improving missing components. Variation is ensured during:

- Use of different supports: chairs, plinths, stools, mats, walls, objects, tables
- Exploration of movement through different postural sets
- Different activities and different environments inside and outside

As well as specific therapy, the training needs to be made clear to helpers and carers with whom the patient interacts throughout the day. Multidisciplinary communication and cooperation between different health professionals is therefore necessary.

It takes time to learn, to change synaptic connections, to reorganize established and damaged systems, to learn new and to unlearn inappropriate things. Clinically, significant changes may be achieved in treatment (functional plasticity), but these are not always transferred into the patient's daily activities (structural plasticity). If this happens repeatedly, the therapist has to re-evaluate her analysis and approach. Treatment should have reached a certain level of intensity and carried out for a certain time (12 weeks) before it is possible to state that treatment is not working. In the acute stage following a stroke, new functions are learned very quickly, more so than at later stages due to the increase in neurotrophic factors (see Chapter 1.2, Plasticity). If the patient has learned strategies that might have seemed appropriate at the time, but not any more, these may take time to unlearn to allow the learning of new ones. Neurophysiologically, learning and unlearning is the same process because it involves synaptic changes—both are learning. An important factor is how much and how often the synapses are stimulated in one specific direction.

Learning and carry-over takes time to be established.

2.3 Other Interventions: Some Key Points

Strength Training

As we grow older, most of us experience loss of strength. The quadriceps of a 70-year-old person has only 60% of the strength of that of a person in his twenties. This is the same for men and women (Macaluso and De Vito 2003). These authors describe the process of losing strength, which is attributed to a quantitative loss of muscle mass (sarcopenia), a selective atrophy of type 2 fibers due to a progressive loss of motor neurons in the spinal cord that cause an initial denervation of fast twitch fibers. These fibers are re-innervated by type 1 motor units through collateral sprouting. Strength training in elderly people improves their muscle strength and function.

An upper motor neuron lesion causes weakness (See Chapter 1.1, Consequences of and Reorganization after CNS Lesions, Upper Motor Neurone Lesion, Weakness). Many people suffering from a CNS lesion are elderly and may have experienced considerable weakness even before their lesion. Current evidence suggests that, generally after stroke, the negative impairments, weakness, loss of dexterity, and fatigue limit recovery of function more than the positive impairments (Canning et al. 2004). In their longitudinal study of 22 patients suffering a first stroke, Canning et al. found that strength and dexterity in total contributed significantly to function throughout, strength made a significant separate contribution to function at all test times, and that the combined contribution of strength and dexterity was greater than either alone.

Historically, strength was not thought to be relevant in patients with upper motor neuron lesions. Bobath (1990) stated that "Weakness of muscle may not be real, but relative to the opposition of spastic antagonists." She also stated that "Weakness of muscle may be due to sensory deficit, either tactile or proprioceptive or both." This last statement still holds as one reason for weakness. Berta Bobath did not use resisted strength training in her treatment, but she did increase the strength of the patients, for instance, by using their body weight in opposition to gravity, stepping down, sitting down, and one-legged standing.

Research has demonstrated, however, that weakness is a significant problem in CNS lesions, and that it may be due to weakness of neural transmission and a reduced ability to recruit the appropriate number of motor units for the actual function. Therefore, the problem of weakness has to be addressed in treatment.

Many patients may be able to recruit strong muscle activation in total patterns, but are unable to recruit muscle activation selectively to enhance functional stability, for instance in moving from sitting to standing, in stance and for locomotion. Strength training therefore needs to be selective and in functional patterns. Heel-strike is recognized as being one of the most important signals to the CNS to activate a selective stance phase. Information about unloading, heel-strike, and weight transference are critical for the control of stepping (Maki and McIlroy 1997). Some important components of heel-strike are postural

control and core stability, selective activation of the proximal hamstrings to bring the heel in contact with the floor, selective extension of the knee, eccentric lengthening of the distal hamstrings and of the posterior compartment of the lower limb, active dorsiflexion and extension of the toes. As the body moves forward over the ankle, which acts as a pivot, the muscular activation patterns change, but knee and hip extension is maintained throughout stance to varying degrees and with different combinations of muscle coordination. Strength training requires focused attention on the part of the patient and may enhance his awareness and perception of the part.

Strength training needs to be selective and in functional patterns.

Strength training requires focused attention on the part of the patient and may enhance his awareness and perception of the part.

Clinical examples

Clinically, some muscles seem specifically important to strengthen:

- Abduction of arm for deltoid to facilitate separation of trunk and arm: core stability and functionally free arm movement
- Triceps as a selective antagonist to biceps for coordination of arm and hand function
- Thumb abduction for wrist extension
- Toe extensors for heel-strike
- Ankle evertors for heel-strike
- Soleus and gastrocnemius for toe-off
- Proximal hamstrings and distal quadriceps for the different stages of stance phase
- Proximal hamstrings that stabilize for the selective extension of the knee in swing, as well as acting with the quadriceps to maintain and evolve extension through stance
- Hip extensors, abductors, and external rotators for hip stability through stance
- Strength needs to be based on core stability.

In the lower limb, muscle weakness may be seen as caused by the loss of excitation of the CPG due to disruption of excitatory commands to reticular pathways. About 18 million fibers run from the brain to the reticular pathways, which are the biggest pathways in the brain. These provide strength and control all CPG activity. A

lesion to these pathways causes weakness of CPG activity.

Vestibular augmentation seems to be mostly lacking, therefore to strengthen the lower limb for stance and swing phase, the therapist needs to focus on strengthening from the foot upward so that the exercise is based on the context of the vestibular system (Mary Lynch-Ellerington, personal communication, 2005).

Treadmill Training

Treadmill training is used for many different neurologic conditions and it is based on two basic principles: (i) facilitating CPG activity and (ii) repetition to consolidate new learning. Treadmill training has been extensively researched, especially in stroke and spinal cord injury as well as in healthy subjects (Mudge and Rochester 2001, Moseley et al. 2005), and the results regarding its efficacy is debated. Some essential factors need to be taken into consideration when a therapist thinks that treadmill training may be indicated for an individual patient.

Aaslund (2006) in her study of 28 healthy people walking on the ground and on a treadmill with and without body weight support found that gait is significantly influenced when walking on a treadmill, when using a harness and when using approximately 30% body weight support.

- Treadmill alone:
 - Increased cadence
 - Increased forward tilt of the trunk
 - Increased vertical acceleration
 - Increased variability of anteroposterior trunk acceleration
- Treadmill with harness and body weight support
 - restricted the mean acceleration in all directions
 - increased variability in trunk acceleration in anteroposterior and vertical directions
 - caused stereotypical trunk acceleration in the mediolateral direction

Aaslund concluded that based on these results, task-specificity of treadmill therapy is questionable.

Several studies suggest that to regain the ability for independent walking, a patient needs to be able to rise independently from sitting

(Lee et al. 1997, Chen et al. 1998, Cheng et al. 2004), and that getting the heel down to stand up is an essential factor for this function. Heel-strike is also important for facilitating the phase shifts during locomotion.

Clinically, treadmill training seems to be effective for some patients. If patients have used treadmill training in a sports studio before, they seem to adapt more easily after a CNS lesion. Patients who already have an independent walking ability seem to have improved speed and rhythm overground, but they need time to get used to the treadmill before finding it of use. Some patients who have problems with mild dyssynergic movement patterns seem to normalize their movement patterns as speed increases to a level probably more like their own internal speed (CPG rhythm). However, some patients suffering from severe neglect and low tone do not seem to benefit from the use of body weight supported treadmill training; some seem to be facilitated into inactivity and use the harness as a swing, but respond more positively to facilitation of walking overground. Further research is needed in treadmill training to clarify specific indications for individual patients related to their problems.

Constrained-Induced Movement Therapy and Robot Training

Constrained-induced movement therapy (CIMT or CIT) is an intensive treatment program aimed at overcoming learned nonuse in stroke patients. This treatment approach was first introduced in long-term stroke patients by Taub (Taub et al. 1999) following the research by Nudo et al. (1996). It is hypothesized that the neural mechanisms underlying the adaptive changes seen in the patient's cerebral cortex unmask existing but previously inactive connections: "The short time course of 12 days makes the formation of new anatomic connections by means of sprouting as a major mechanism unlikely because clear evidence of axonal growth has not been found until months after a lesion occurred. A more likely mechanism is a reduction in activity of local inhibitory interneurons, thus unmasking pre-existing excitatory connections. An alternate and possibly complementary mechanism would be the enhancement of the synaptic strength of existing synaptic connections" (Liepert et al. 2000).

There are strict inclusion criteria for this treatment program. The patients must have (Kim et al. 2004):

- A certain level of balance which is not dependent on their less affected arm
- Display 20° active wrist extension and at least 10° active extension at the metacarpophalangeal joints of two fingers and the thumb
- No severe spasticity or pain
- Good cognition
- A high level of motivation

It is worth noting that these criteria make this program relevant to only 4–6% of all stroke patients, so 95% will not benefit from this treatment.

The program itself consists of inactivation of the patient's less affected hand by fitting a specialized glove, which has a hard plastic plate extending beyond the palm. This prevents the patient from using the hand in any manual dexterity tasks, but at the same time allows it to act as a support in two-hand activities. The patient has to wear the glove at least 6 hours a day and up to 90% of their wakening time, shorter if used early after stroke. It may be used also to promote weight transference on to the most affected lower limb by the use of an inflatable splint or back splint to the less affected leg. Caution should be exercised if the constraints are used in the early rehabilitation phase. Patients should not be enrolled on this program until at least 1–2 weeks post stroke due to the vulnerable penumbral zone surrounding the infarcted area (see Chapter 1.2, Plasticity). Training consists of a structured programme with coarse, fine motor, and general ADLs for 6–7 hours a day as well as using it outside the treatment area. The patients are allowed 10 minutes off every hour, as well as during hygiene activities (going to the bathroom, showering).

This degree of intensity of training is in sharp contrast to the amount of therapy time given to most patients in a hospital or rehabilitation setting. Comparing conventional therapy time of at the most 1–1.5 hours (physiotherapy and occupational therapy together) a day (7.5 hours per week) to CIT of at least 6 hours per day (30 hours per week) demonstrates the importance of the intensity of treatment to enhance the patient's potential. In their meta-analysis of augmented exercise time, Kwakkel et al. (2004) found that increased intensity in the form of

more treatment improved outcomes related to ADLs and walking.

There is also increasing interest in robot training for both upper limb (Lum et al. 2002, Casadio et al. 2006) and gait (Bharadwaj et al. 2005) which aims to promote motor performance and function through repetition, rhythm, and facilitation of relevant muscles and to enhance or maintain range of motion at relevant joints. This needs to be further investigated to define the potential role of robotics in neurorehabilitation.

Multidisciplinary Teamwork

As long as the patient is an inpatient, he probably needs some physical assistance to achieve ADLs. All these activities are relatively complex physically, requiring balance, weight transference, alterations in rotational components, alterations of stability and movement components and areas of references, variations in movements strategies, and problem solving. The most complex activity is supine to sitting at the same time, as this transfer is the one transfer many carers expect the patient to perform independently early on.

If the patient is to regain control of movement, all personnel should have the same understanding of the treatment plan. This is to ensure that the patient is not given conflicting messages. The corner stones of teamwork are each professional's general and specific knowledge, expertise, and role. Multidisciplinary teamwork challenges all parties and is difficult to achieve. The individual professionals need to understand and respect each other, be loyal to the patient, to the goals and the interventions, strengthen each other's role, and follow each profession's interventions as closely as possible. At the same time, the different professions have their specific role that cannot be replaced by others. The patient is exposed to the specific interventions from different professionals, and receives the sum of the multidisciplinary teamwork. This is the environment in which he learns anew. The rehabilitation process is discussed in Chapter 3.

Example

The *24-hour concept* is multidisciplinary teamwork put into practice and relates specifically to the carrying through of treatment principles in daily activities. The multidisciplinary team agrees on which movement components are most important to facilitate, repeat, and consolidate during different activities in the patient's daily life. The 24-hour concept strengthens the patient's learning process because:

- The activity is varied and repeated during the day and possibly the night (bathroom).
- Varied repetition
- The activity aims at a known performance for the patient.
- The activity is adapted to suit the needs of the individual and of his movement problems.
- The effect of carry-over increases when treatment interventions are transferred to the everyday context.
- Teamwork requires good multidisciplinary communication, both formally and informally, a follow through, and loyalty toward the patient and goals, as well as a common basic understanding and competency.

It is the responsibility of the involved professionals to ensure the treatment is carried through. Often, this is made easier if they see the patient together in practical situations: nurse, occupational therapist, and physiotherapist may see the patient together in the morning during personal care, during transfers in and out of bed, during dressing or at meal times and agree on a strategy to help the patient improve and regain his independence if possible. The use of photos from practical situations may aid this process. Cooperation between the patient, his carers, and health professionals needs to be stimulating for learning. Good multidisciplinary teamwork gives motivation and learning to the whole team.

Assistive Devices

Many patients do not recover fully after a lesion to the CNS. The sensorimotor problems may vary from slightly reduced balance and dexterity to severe loss of function causing a need for assistance to all activities of daily life. It is therefore

not possible to put forward general guidelines on the use of compensatory aids. Several aspects need to be evaluated:

- Timing for giving an assistive device
- Positive and negative aspects of different aids within the same group; i.e., walking aids and wheelchairs
- Evaluating how the aid is used in relation to its effect on the patient's function over time and adapting or changing the type of aid as needed as the patient progresses

■ Timing

The therapist may need to decide on the use of aids. Important things to consider are whether the aid:

- Reduces the patient's effort at the same time as allowing him to explore his environment
- Enhances or improves the patient's motor problems over time

How the patient moves and the degree of associated reactions express his movement ability at that time but do not necessarily reflect the extent of the lesion. The patient's motor problems are a result of the lesion itself, cognitive, perceptual, and sensorimotor deficits, the immediate restitution process, and use-dependent plastic changes in the CNS, together with muscular changes and compensatory motor strategies. The aim is for the patient to compensate as little as possible yet at the same time be able to be active and participating. The patient's movement abilities change with time, therefore any assistive device needs to be adapted and altered in different phases of recovery. In the early stages after stroke, in some patients suffering from MS, head injury, or incomplete spinal cord injury, the use of a wheelchair may be appropriate and necessary to allow the patient to explore his environment and be more independent. A walking aid may be appropriate for some patients during different stages and in different situations, but it may be disadvantageous for other patients. The different types of assistive devices are discussed in the next section.

■ Positive and Negative Aspects of Different Aids within the Same Group

- Wheelchairs
- Walking aids
- Orthotics
- Other
- Wheelchairs

Some patients may need a wheelchair temporarily or for different purposes such as transport over longer distances or shopping trips, whereas others may need a wheelchair for daily use. There are some important aspects that need to be considered when fitting a wheelchair:

- Sitting posture and comfort. The patient needs to be seated in proper alignment to enhance postural activity.
- Use. The chair needs to be appropriate for the patient's and the carer's needs:
 - Ease of transfers into and out of the wheelchair for the patient and carers
 - Temporary, periodical or constant use
 - Active or supporting (comfort), or a combination
 - Environment; inside, outside. Terrain?
 - Manual or electric? Does the patient need more than one type of chair, for instance an active manual chair, a chair with standing function, electric chair for inside or outside use?
 - Carer-driven or self-driven? Some patients are unable to drive their own wheelchair, either due to the extent of motor problems or due to cognitive or perceptual dysfunctions. Generally, the neuropsychologic problems of severe neglect, inattention, some apraxias, decreased problem-solving abilities, uncritical behavior may prevent the use of a self-maneuvered wheelchair. Clinically, however, some patients with neglect or inattention learned to use the wheelchair independently. Sometimes, the confrontation with the problem seems to cause an intellectual compensation in otherwise cognitively able patients. Therefore, if appropriate, these patients should be allowed to explore independent wheelchair maneuvering in controlled circumstances
 - Transportation generally (in and out of cars and so on)

Manual Wheelchairs

If the patient is driving the wheelchair manually, it needs to be as light as possible. Depending on the patient's need for stability and his balance ability, the position of the center/line of gravity in relation to the drive shaft is important. The closer the center of gravity of the patient is to the drive shaft, the easier it is to maneuver the wheelchair; however, it is more unstable.

Patients suffering from stroke may need to drive the wheelchair using the less affected arm and leg. Ashburn and Lynch (1988) and Cornall (1991) have pointed out the disadvantages of asymmetric and static use of one side of the body, which may enhance the development of associated reactions and work against recovery and treatment. One-hand drive wheelchairs may strengthen use-dependent plastic changes toward the patient's less affected side and induce learned nonuse in the more affected body half. Therefore, it is of primary importance to evaluate the patient's use of a wheelchair and what it may do to the patient in the longer term. Therapists should experience themselves the effect of manually driven wheelchair both inside, outside, and up an incline, using one-hand and leg, and two arms.

Power Chairs

An electric wheelchair may be an appropriate tool in a rehabilitation phase or for permanent use for some patients. The patient is able to participate to a greater degree socially and in different activities such as bringing himself to appointments or to the shop without undue effort or a feeling of being dependent on carers.

Walking aids may be sticks, crutches, rollators, high walking frames, or other devices. Any walking aid alters the person's relationship with the base of support, increases the base and changes the line of gravity. The patient needs to learn new postural and motor strategies if he has not used a walking aid before. The type, the height, and the way in which it is being used will influence the patient's postural activity (see Postural Sets). If a walking aid is chosen for the patient, the therapist needs to teach the patient how to use it appropriately.

Treatment should focus on the patient regaining as much independent control as possible. The use of a walking aid may be appropriate to allow the patient to move around safely and be exposed

to gravity to enhance postural activity if it is used as a reference more than a support. Many patients receiving a walking aid early in their rehabilitation as a compensation for balance problems, weightbear on the aid and so increase flexor activity in their trunk, pelvis/hips, and arms. Increased flexor activity may hamper development of stability as a basis for balance. Therefore, treatment should aim at improving the patient's postural control and movement and the patient should not be given a walking aid before this is explored.

Motivation is important to recovery. For some patients the ability to ambulate as soon as possible is a major driving force, even if the balance and movement prerequisites are not sufficiently developed to allow them to walk safely. The challenge is to inform, motivate, and, at the same time, teach the patient to improve his postural control and movement patterns, and teach him to use the walking aid as well as possible. The patient needs to experience taking steps through therapeutic facilitation as early as possible. Stepping may enhance balance and rhythm through CPG activation, not allowing the CNS to "forget" walking by increasing cognitive control. Seeing that the aim is to walk again may motivate the patient. The timing of when the patient may walk in the department with or without assistance or supervision is a decision that depends on the patient's safety and motor control as well as on the competency of the carers.

Bilateral Aids

Bilateral aids (sticks, crutches, rollator, Zimmer frame, and high walking frame) may invite the patient to lean or pressure bear on the aid, especially if they are too low. A rollator, Zimmer frame, or high walking frame allow little variation and flexibility because rotation is not needed. With severely disturbed stability, for instance in ataxia, a bilateral walking aid may be appropriate.

One-Handed Aids

One-handed aids (stick or crutch) displace the patient's line of gravity laterally toward the aid depending on how it is being used. If the patient is able to use it more as a reference for balance and movement than a support to weightbear upon, the patient's postural control may in some cases be facilitated (Jeka and Lackner

1994, Jeka 1997). A walking stick may hamper development of the patient's balance control if he leans on it, and increase asymmetry. A high stick may be used to enhance extension and interplay between body segments if it is used as a balance aid without pressure. Sometimes it may be appropriate to use a stick outdoors, during shopping or in other circumstances when balance is especially challenged, but the patient might not use it in his own home.

■ Orthoses

Studies by Mulder et al. (1996) and Geurts et al. (1992) demonstrate that orthopedic footwear influences the size of the base of support and sensory feedback.

Shoes

Shoes that support the ankle reduce the proprioceptive feedback from the ankle and lower leg, influence postural control, and may reduce the degree of automatism in balance (Geurts et al.

1992). Their study included patients with neuropathies and amputations, but some of the results may extend to CNS disorders. Heel-strike is an important phenomenon to transfers and walking. A shoe with a good heel support, a firm sole, and firm heel may promote feedback about loading and unloading of the heel.

Ankle/Foot Orthoses

Ankle/foot orthoses are used to stabilize this area during transfers, in standing and walking, and to facilitate the lift off of the foot during swing phase. The cause of the instability must be assessed and treated. Orthoses that enclose the ankle and lower leg may give distal support to allow patients to explore their postural control. At the same time, external fixation may provoke loss of range of movement, flexibility, and movement. Pressure on muscles and joints may cause a sensorimotor reorganization, which leads to the development of new strategies for balance and movement. Instability of the foot and ankle is rarely an isolated problem in CNS lesions, and



Fig. 2.36a



Fig. 2.36b

Figs. 2.36a–d Two different orthoses.

a, b The model is wearing a splint of polypropylene. Notice the change of alignment in the two pictures;

in Figure **b** she is more flexor dominant and her left hip seems to be more inactive as compared with Figure **a**.



Fig. 2.36c

c, d The model with a different type of splint, called an air cast. This stabilizes the ankle mediolaterally without compromising dorsal and plantar flexion. It takes up a lot of room in the shoe and is not good



Fig. 2.36d

for continuous use, but may allow facilitation of postural activity in better alignment when standing early after a CNS lesion.

needs to be viewed together with alignment, tone distribution, recruitment pattern, and sequence of muscle activation in the body as a whole.

Most orthoses aim to maintain the ankle slightly dorsiflexed. This may cause increased flexion of the hip and knee and alter alignment throughout (Fig. 2.36). Hip stability may be negatively influenced by this increased flexor activity, which affects the patient's ability to transfer and walk. A general disadvantage of the use of splints is the immobilization of the foot, which loses its adaptability, flexibility, and varied feedback. The ability to use the foot and ankle as a mobile and stable base for the body may be compromised.

A lower limb support may be indicated in some cases. There are several different types available, and more are being developed. The patient needs to try different types and evaluate them over time. There are different plastic splints for covering the posterior aspect of the lower leg and the sole of the foot (Figs. 2.36a and b), toe-off types of different materials, ankle supports of soft

materials with Velcro fastenings (mostly used in orthopedics), ankle orthoses that support the ankle medially/laterally (Figs. 2.36c and d) and arrangements fastened to the patient's shoe (for instance Klenzak splint) with or without a T-strap. All of these influence alignment and support the ankle and foot to varying degrees. Only evaluation over time may determine if this influence is positive or negative.

It is important to decide whether a potential splint should be used all the time a patient is on his feet, or whether it should be used for specific situations. The foot has an increased tendency to twist in situations in which balance is perceived to be threatened; outside, in a throng of people, uneven ground, traffic. If the splint is used only at certain times the adaptability of the foot and ankle may be maintained thereby allowing the patient to experience weightbearing through extension. The patient should stand and move in his bare feet regularly and receive varied input.



Fig. 2.37a



Fig. 2.37b



Fig. 2.37c

Figs. 2.37a–c Single-strap arm sling: a view of the hand (a). The arm sling is rolled round the hand a few times proximal to the metacarpophalangeal joints to maintain wrist extension. Figure b demonstrates the way it is wound up the arm to maintain neutral rotation of the arm and crossing the shoulder. It is important to place the sling precisely as it crosses the shoulder to influence the alignment of the shoulder joint. If there is a tendency for anterior or inferior subluxation, the strap should be placed to maintain the head of the humerus within the glenoid fossa. The sling crosses the shoulder at the back to the opposite shoulder. Here it goes under the shoulder, wraps round the thorax to give some postural facilitation, crosses behind the affected shoulder diagonally across the back and over the less affected shoulder. It fastens on itself. At no time should the strap be tightened or stretched as it is fitted to the patient as this may impede circulation. If placed properly, patients may experience some improved postural stability and facilitation of extension of the thorax as well as neutral to external rotation of the arm. Figure c shows the sling from the back.

Knee Orthoses

Some patients experience hyperextension of the knee which destabilizes them. During normal walking, the hip and knee move forward over the foot in stance phase. The knee is rarely fully extended, but is at its peak of extension at mid-stance and just before toe-off. Reduced mobility of the foot and ankle in dorsiflexion prevents this forward movement of the lower leg and brings the knee into hyperextension as the body continues its forward direction. This may be caused by increased tone to the posterior crural muscles, reduced eccentric control of gastrocnemius, soleus, and the deep posterior muscles of the lower leg, the medial hamstrings group or hip adductors as well as malalignment of the rotational components between the different segments of the leg, pelvis, and trunk. If the hip flexes in stance, hyperextension of the knee ensues. Mobility and stability of hip/pelvis and ankle/foot as well as of the trunk are essential for dynamic knee function. Reduced coordination of hip stabilizing activity and ankle/foot mobility catches the knee in the middle. Therefore, a splint to reduce hyperextension of the knee is rarely necessary if the underlying cause is treated.

Shoulder Orthoses

Shoulder subluxation is a frequent problem after stroke, and may be a risk factor for the development of pain and additional functional impairment. The use of arm slings is controversial, but has been shown to reduce subluxation in some cases. There are many different types which influence alignment to varying degrees. Some of the disadvantages are that some of these maintain the arm in a fixed flexed pattern, hamper movements at the shoulder, and patients need help with putting them on. Most slings give proximal supports, and some patients feel that their balance improves during the use of an arm sling and that the arm is supported in a position of less pain. Yavuzer and Ergin (2002) studied the effect of a single-strap sling on postural control and gait parameters in 31 patients and found that this type of sling improved gait as measured by three-dimensional gait analysis and video recordings (Fig. 2.37).

Patients who are unable to take care of their arm due to neglect, inattention, or cognitive deficits, and who have a severe subluxation of the shoulder or are starting to experience pain may

benefit from using a shoulder orthosis. There are many different types, which need to be tried in conjunction with the patient and his carers to ensure proper use. None of these seem to be very accurate in reducing subluxation, as subluxation of the shoulder joint is caused by a combination of different factors: reduced postural control, reduced stability of the shoulder girdle complex, altered alignment of the scapula to the thorax, as well as paresis. The orthosis may, however, protect against trauma due to an arm falling heavily by the patient's side and signal to carers that handling of the shoulder and arm must be performed with care.

■ Other Aids

Aids to help with daily activities at home or at work need to be evaluated on an individual basis. A home visit or workplace visit together with the patient and his carers may expose problem areas where assistive devices may be of help; specially fitted working chairs, kitchen appliances, for example. The physiotherapist and occupational therapist may work well together to ensure that the patient's functional needs are met as well as optimization of balance and movement.

■ Evaluation and Adaptation

All aids should be evaluated regarding how they are used and their effect on the patient's function, postural control, and movement, positively and negatively. They will need to be modified as the patient's condition changes over time; either improvement in function or some deterioration. The patient needs to be followed up for some time to assess, adapt, and evaluate the type of aid used and to determine whether he is benefiting from its use any more or if the type of aid needs to be changed.

■ Medical Intervention for the Treatment of Spasticity

Medical treatment for spasticity seems to be increasing, however, spasticity is rarely defined or discussed in this literature. Therefore it is uncertain what some authors mean and treat. Most refer to functional and movement problems asso-

ciated with spasticity, and therefore seem to also include secondary soft tissue problems (see Chapter 1.3 Consequences of and Reorganization after CNS Lesions).

There are several forms of treatment for what is termed spasticity, both surgical and medical. In this section only two interventions will be discussed:

- Botulinum toxin A
- Baclofen

■ **Botulinum Toxin A**

This is a toxin derived from the bacteria *Clostridium botulinum*. It is injected directly into musculature that is most affected, and it binds to the presynaptic terminal at the motor end plate. The exact mechanism of denervation is discussed, but is associated with inhibiting the release of acetyl choline and thereby muscle activation of the motor units affected by the toxin (Gjerstad et al. 1991). With repeated injections the patient may experience a reduction in effect due to a production of antibodies (Bakheit et al. 2004). Botulinum toxin thereby acts as a neuromuscular blockage. The effect starts between 24 and 72 hours post injection and causes a functional denervation of the affected muscle fibers. Kerty and Stien (1997) state that probable denervation stimulate axonal branching from undamaged nerve fibers so that the effect of the injection is reversed within 3–4 months, and injections need to be repeated in most cases. Spasticity may then return.

Botulinum toxin is used for focal dystonias, for instance torticollis (Gjerstad et al. 1991, Borgmann 1997), and spasticity (Childers et al. 1996, Cromwell and Paquette 1996, Kerty and Stien 1997, O'Brian 1997, Smedal et al. 2001, Bakheit et al. 2004). Improvement in function is most likely if the patient has (O'Brian 1997):

- Some agonistic/antagonistic muscle function
- Acute or subacute spasticity
- Easily identifiable areas of focal spasticity that hamper the patient's function
- Specific short-term and long-term goals for treatment

Cromwell and Paquette (1996) describe a case history detailing the effect of botulinum toxin combined with intensive physiotherapy and occupational therapy in a 2-week period after injection. The patient had tetraplegia after suffering a

brain stem infarct, and severe problems in transfers and gait. The authors followed the patient for 6 weeks after treatment, and found that the patient improved in both transfers and gait following this treatment combination. There was no long-term follow-up.

Smedal et al. (2001) studied 10 patients with different neurologic diagnoses and functional problems that received a combination of botulinum toxin and physiotherapy. Nine patients experienced improvement of function and/or pain relief. One patient had improved perception in his hand, and one patient needed only two injections to stop a vicious cycle. The effect varied from patient to patient. It seemed that patients with some preserved motor control in the affected limbs could use it to learn more appropriate movement strategies. The authors recommend that doctors and physiotherapists work together to identify muscle groups that need to be targeted for injection and evaluate consequences of the treatment, and that injections with botulinum toxin should be combined with physiotherapy.

The effect of botulinum toxin is to lower muscle tone and recruitment in the injected muscles and may lead to changes in function. If botulinum toxin is injected into muscles that the patient uses for transfers or maintenance of some form of stability, a reduction in activation may cause deterioration in function. Specific analysis of movement is therefore required to determine where botulinum toxin should or could be injected (Albany 1995). Before injection, electromyographic analysis of muscle activation of relevant muscles is recommended.

Albany (1995) states that the aim of physiotherapy is to facilitate more normal patterns of movement to:

- Facilitate activity in antagonistic and synergistic muscle groups
- Improve muscle length and flexibility
- Facilitate and maintain optimal alignment when the patient is positioned in different positions
- Stimulate proprioception
- Improve balance and stability

Botulinum toxin seems to have a positive effect on pain in some patients. Clinical experience supports that clinical problems associated with spasticity may be reduced circulation, fixation of the involved extremity in a posture that may reduce

personal hygiene and cause sores, and compression of joints and soft tissues as well as other problems that may be associated with pain. An improvement in any of these factors may influence the patient's pain positively. A long-term negative effect of botulinum toxin alone on injected muscles has not been documented.

Baclofen

Baclofen may be infused into the spinal cord through an implanted spinal catheter connected to a programmable infusion pump which supplies the spinal cord with baclofen continuously. Baclofen binds to inhibitory interneurons in the posterior horn of the spinal cord ($GABA_b$ -receptors) and causes a presynaptic inhibitory effect (Berg-Johnsen et al. 1998). Baclofen has a general effect on tone and may reduce problems with tone in different CNS disorders. It is most widely used in spinal cord injury, multiple sclerosis, and cerebral palsy.

Berg-Johnsen et al. (1998) refer to a study on a group of patients suffering from multiple sclerosis, spinal cord injury, and cerebral palsy who experienced severe problems with activity. They received intrathecal infusion with baclofen, and 60–70% of the patients improved their ability to transfer and their independence. Some of the patients experienced a reduction in strength and deterioration of gait. Intrathecal infusion with baclofen seem to have a positive effect on pain in some patients, however, there may be complications such as infections, catheters problems, overdose, and pump failure.

Discussion

Intrathecal infusion with baclofen provides a generalized reduction in tone distally and partially proximal to the area of infusion. All muscles affected will have a reduction in tone, including the musculature that the patient may need to use to be functionally active within their limits. Some patients therefore experience reduced function as a result of treatment. Hypothetically, facilitation to more optimal movement strategies may be influenced negatively if plasticity is affected through medically induced presynaptic inhibition at spinal level. These factors need to be considered along with the need for pain relief, hygiene, and other factors important to the patient and his carers.

Other Forms of Medication

Baclofen (Lioresal), Valium and other drugs may be given to the patient in the form of pills to reduce the level of involuntary muscle activity in some patients (Cromwell and Paquette 1996). These exert a general effect on the CNS and may cause tiredness, cognitive changes, and general muscle weakness of the extremities. Cromwell and Paquette do not mention any effect on postural muscles, but it is hypothesized that it may be similar. Berg-Johnsen et al. (1998) also describes problems of nausea, dizziness, sleeplessness, confusion, and gastrointestinal dysfunction. These authors therefore recommend botulinum toxin (Cromwell and Paquette 1996) and intrathecal infusion of baclofen (Berg-Johnsen et al. 1998) as alternatives in the treatment of spasticity associated problems. Clinically, it may seem as if anti-inflammatory drugs, antibiotics, and anti-depressive types of medication influence the patient negatively in some cases.

Summary

Normal movement is varied, without inappropriate effort, efficient, effective, precise, and successful, developed through the interaction between the person, the task, and the environment. See page 69.

The nervous system seems to weight the importance of somatosensory information for postural control more heavily than vision and vestibular inputs under normal conditions. See page 71.

Recovery largely depends on the ability of the CNS to adapt to (peripheral) changes. See page 74.

In a functional context, the sensorimotor and perceptual interaction between the body and the base of support is more important for the level of postural tone than the size of the base of support. See page 74.

Mobility is essential for stability, as is stability for movement. See page 76.

Balance is expressed in both reactions and strategies. Postural control, righting, and protective reactions are elements of balance. See page 80.

Inappropriate compensatory strategies—alternative behavioral strategies—may delay or hinder the development of balance and selective motor control in patients with CNS lesions. See page 82.

Standing seems to improve both trunk positional sense and overall function. See page 82.

The Bobath concept is a problem-solving approach to the assessment and treatment of individuals with disturbances of function, movement, and postural control due to a lesion of the central nervous system. See page 90.

Postural sets describe the interrelationship between body segments at a given moment. See page 90. Movement may be described as continuous change of postural sets. See page 90.

A selective movement in one postural set requires a different neuromuscular activity in a different postural set. As the biomechanical alignment changes, so does the neuromuscular activity. See page 91.

Many muscles and joints converge at the key areas. Therefore the proprioceptive influence, as well as those from the skin, on the CNS is substantial. See page 110.

Control of key areas and the interplay between them seem especially important for balance, selectivity of movement, adaptation to the environment and tasks, and therefore for function. See page 110. Everyday activities such as balancing, walking, reaching, and eating are mostly automatic functions that normally require little attention and effort. See page 114.

Everyday activities have a structural correlation in the CNS, based on experience. See page 114.

The expression of activity varies depending on the individual, the goal, and the situation. See page 114.

Automatic and voluntary controls of movement are closely integrated and form the basis for functional skills and balance. See page 114.

The clinical challenge is to decide whether balance can be regained through conscious voluntary planning, or be facilitated on a more automatic level in functional situations. Tone, muscle dynamics, alignment, and sequence of recruitment must be optimized in both scenarios. See page 116.

The therapist's hands may touch, create friction, stretch, compress, approximate, and give information about muscle length and tension, direction, speed, and range. They may produce traction, compress or rotate, demand stability and/or mobility depending on the problem and the functional goal. Information is specific to the desired activity. See page 118.

Facilitation means "making easy." The aim of the therapist is to handle the patient in such a way that movement feels easier for the patient because the patient's own activity is recruited.

In this context, facilitation must not be interpreted to mean passive movements or passive techniques such as the use of tapping over muscle or stimulation using ice. See page 119.

Treatment requires a continuous interplay between working on impairments and facilitating activity, making movement possible, demanding control, and encouraging action: Make possible → make necessary → let it happen. See page 121.

The aim of handling is to enable the patient to be more active so that the hands can be taken away. See page 121.

Active movement provides a diversity of information to the CNS. See page 126.

Active movement is essential to perception. See page 126.

Facilitation of active movement through intense sensory stimuli seems to improve neglect. The patient's awareness increases during active movement. See page 127.

Passive movement is important if the patient is unable to initiate movement of his own. See page 127.

Passive movement seeks to stimulate activity and requires the patient's attention. See page 127.

Handling through passive movement aims to make the patient's CNS listen to the input and respond as far as possible, and is therefore not really passive. See page 127.

It is essential to analyze and treat the cause of the associated reactions and not only attempt to dampen down the reactions. See page 128.

The feedback varies and depends on the patient's movement ability, his perceptual or cognitive function and the type of goal activity (more automatic or more voluntary). See page 130.

Varied repetition in treatment allows the patient to develop a wide repertoire of movement and movement experience that he may use in different functional contexts. See page 130.

The patient should always feel that specific treatment leads to functional improvement within the same treatment session. The patient should not feel that the training does not meet his needs. See page 131.

Learning and carry-over take time to be established. See page 132.

Strength training needs to be selective and in functional patterns. See page 133.

Strength training requires focused attention on the part of the patient and may enhance his awareness and perception of the part. See page 133.

3 Assessment

3.1 The International Classification of Functioning, Disability, and Health	145
3.2 Physiotherapy Assessment	147
3.3 Outcome Measures	163

In different phases and stages of a patient's rehabilitation, healthcare professionals may take on different roles according to the patient's needs at that moment: fellow human being, supervisor, guide, informant, professional, helper, or carer. These roles depend on where the patient is in his rehabilitation process and his current needs. Healthcare professionals diagnose, treat and inform, adapt, plan, and structure the rehabilitation together with the patient, taking into perspective the patient's potential and limitations as a person, as a whole.

Multidisciplinary teamwork brings together each profession's general and specific competencies and role, and the patient has his own competencies. Together, these give insight into the challenges in the rehabilitation process of the individual patient. Possibly, the healthcare professionals' most important task is to find possibilities and potential—the positive building blocks within the patient and in his network of carers—that may enhance his progress. The patient's needs are central to the choice of interventions. This chapter discusses

- The International Classification of Functioning, Disability, and Health (ICF)
- Physiotherapy assessment
- Outcome measures

3.1 The International Classification of Functioning, Disability, and Health

The ICF is a tool used to classify different aspects and factors which influence a person's life and describe how people live with their health condition. ICF is a classification of health and health-related domains that describe body functions and structures, activities, and participation. The domains are classified from the body, the individual, and societal perspectives. As an individual's functioning and disability occurs in a particular context, the ICF also includes a list of environmental factors.

The ICF is useful to understand and measure health outcomes. It can be used in clinical settings, health services, or surveys at the individual or population level. Thus it complements the *International Statistical Classification of Diseases and Related Health Problems, 10th edition* (ICD-10) and looks beyond mortality and disease. By using the ICF, there is also the hope that healthcare professionals will communicate in the same language.

The last version of the ICF was published in 2001, and it moved away from being a "consequences of disease" classification (1980 version) to become a "components of health" classification (WHO 2001, Introduction p. 4). The ICF is divided into different sections for body functions, body structures, activities and participation, and environmental factors (ICF 2006, online version) (Table 3.1).

- *Body function* is a classification of the physiologic and psychologic systems of the body
- *Body structure* classifies anatomic parts of the body: organs, extremities, and their components
- *Impairments* are problems of body functions and structures
- *Activities and participation* covers all activities performed by individuals
- *Limitations* are problems the individual may experience in the performance of activities
- *Participation* classifies the involvement in life situations by the individual in relation to health, body functions and structures, activities, and relations
- *Restrictions* are problems that an individual may encounter in the way or level of participation in life situations
- *Environmental factors* make up the physical, social, and attitudinal environment in which people live and conduct their life

As well as these sections, the ICF involves another area that is not classified:

- *Personal factors* are the particular background of an individual's life and living, and comprises features of the individual that are not part of a health condition or health states, which may be age, sex, experiences, personal beliefs, religion, lifestyle, etc.

The interaction between these factors is illustrated in Figure 3.1 (from ICIDH-2 1999). The interaction among these factors is dynamic: interven-

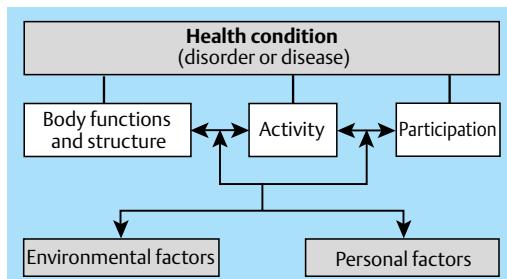


Fig. 3.1 The interrelationship between the different factors of the ICF (adapted from: ICF, WHO 2006).

Table 3.1 ICF sections

Body functions	Activities and participation
1. Mental functions	1. Learning and applying knowledge
2. Sensory functions and pain	2. General tasks and demands
3. Voice and speech functions	3. Communication
4. Functions of the cardiovascular, hematologic, immunologic, and respiratory systems	4. Mobility
5. Functions of the digestive, metabolic, and endocrine systems	5. Self-care
6. Genitourinary and reproductive functions	6. Domestic life
7. Neuromusculoskeletal and movement-related function	7. Interpersonal interactions and relationships
8. Functions of the skin and related structures	8. Major life areas
9. Community, social, and civic life	
Body structures	Environmental factors
1. Structures of the nervous system	1. Products and technology
2. The eye, ear, and related structures	2. Natural environment and man-made changes to environment
3. Structures involved in voice and speech	3. Support and relationships
4. Structures of the cardiovascular, immunologic, and respiratory systems	4. Attitudes
5. Structures related to the digestive, metabolic, and endocrine systems	5. Services, systems, and policies
6. Structures related to the genitourinary and reproductive systems	
7. Structures related to movement	
8. Skin and related structures	

tions at one level have the potential to modify other related elements. ICF may be used to ensure that all aspects of a person's situation have been evaluated as a basis for his rehabilitation process.

3.2 Physiotherapy Assessment

All aspects of motor, sensory, cognitive, and perceptual functions are important for action as illustrated by a model first presented by Shumway-Cook and Woollacott (2001) (Fig. 3.2). The physiotherapist has a specific and important role in the regaining, learning, and maintenance of physical function.

The aim of assessment is to understand the patient's situation. The therapist has to come to know who he is, how he lives, his networks and family relations, work situation, and his resources, and at the same time analyze his movement function. Assessment is therefore both resource and problem orientated. The aim of physiotherapy is the improvement of physical function to the patient's full potential to enable him to participate as actively as possible in his life again. The assessment should indicate what functions have been spared in relation to the regaining and learning of activities, postural control and movement, which functions have been damaged or are dysfunctional, and the consequences for the patient. Assessment leads to in-

formation which allows the therapist to formulate hypotheses as to cause and effect of the patient's problems, and evaluating which systems within the central nervous system (CNS) seem to be functional or dysfunctional and then using this as a foundation for treatment interventions. Knowledge of components of movement which are important for balance and extremity function is a basis for both assessment and treatment. The goal of assessment and treatment is to define the patient's potential and how he can reach optimal function within the limits of available resources.

Evaluating the patient's potential is an important goal of assessment

Neuropsychologic dysfunction and sensory loss have in many cases been used to decide on the patient's rehabilitation potential in the acute phase after a stroke. These early symptoms are not a sign of the patient's real prognosis: perception and cognitive dysfunctions in most cases do improve, as the patient is more oriented to his environment. Thus focus on sensory, perceptual, and cognitive problems alone may lead to a more negative view of the patient's potential. Things take time; the patient must be allowed time for reflection before reaching conclusions about the prognosis and thereby the level of rehabilitation effort the patient needs/is offered. The role of the physiotherapist is to assess *what* activities the patient is able to do, *how* he solves his movement tasks and reaches his goals, and *why* he moves as he does. This assessment forms the basis for physiotherapy interventions.

Occupational therapist Christine Nilson said the following about Berta Bobath in 1991: "She encouraged my creativity and taught me to see each patient as an individual. She offered problem-solving skills that led so logically to treatment intervention" (Schleichkorn 1992, p. 98). Observational analysis of movement during activity is the most important tool of the assessment, and leads on to appropriate handling of the patient followed by clinical reasoning and treatment interventions.

Handling is both an assessment tool and an intervention, and leads to a response from the patient. During handling the aim is to influence the patient's ability to move and his response to being moved. His response is important to deter-

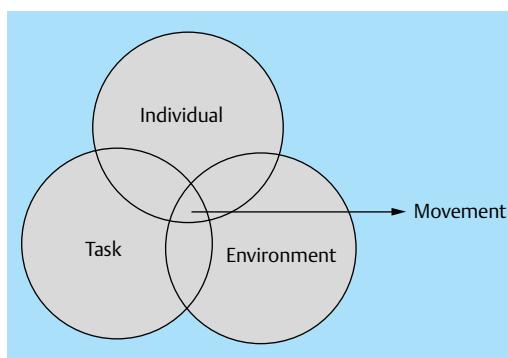


Fig. 3.2 Illustration of how movement is influenced by factors within the individual himself, his environment, and the goal of his actions.

mine the level of his response and his ability to learn, and evaluation of the response is therefore important for the assessment. In this way, assessment and treatment are interlinked processes and cannot be separated. During assessment, the therapist collects information and starts a process of clinical reasoning to form hypotheses about *why* the patient moves as he does—hypotheses about the patient's *main problem* regarding activity and function. Treatment interventions are started, the results continuously evaluated, and hypotheses are discarded if treatment does not improve the patient's motor control, and new hypotheses are formulated as the treatment progresses. In general, the assessment follows the following process:

- History
- Functional activity
- Body functions and structures
- Clinical reasoning
- Outcome measures
- Evaluation and documentation

History

This part of the assessment aims at getting to know the patient and view him as a whole in the knowledge that rehabilitation is a process that often takes many years. The patient is assessed in relation to the ICF dimensions—participation, activity, and body functions and structures—both as he previously functioned and how he presently functions. The therapist has in mind the patient's likely potential, resources, and possible problem areas. It is of essential importance to form a relationship founded on respect and trust, and gain information on social aspects, his roles, medical history, present situation, needs, and wishes. In a team setting, the different members of the team can decide whether they wish to interview the patient and his carer together or separately. Preferably, they should decide on dividing the interview in a way that the patient does not have to tell the same story again and again to the whole team.

Social Aspects

- Marital status, family, and close social network
- Social roles, his needs and wishes
- Housing

- Hobbies and leisure activities
- Work situation
 - Profession, type of position/work, tasks
 - Unemployed or pensioner? Reasons

Medical History

- Previous level of function
 - Physically: vision, hearing, problem areas, use of aids or assistive devices (walking aids, wheelchair, orthopedic shoes, orthotics, and similar), level of activity
 - Mental function
 - Other diseases
 - Medication
- Previous treatments
 - Physiotherapy. For what reason? Effect of treatment?
 - Other
- Previous contact with the health service
- Present illness or disorder
- Results from medical examinations and tests, e.g., computed tomography (CT) scans, magnetic resonance imaging (MRI), X-rays, neurophysiologic tests
- Any contraindications or any aspects of treatment that may need special attention
- Factors that may exacerbate or improve the patient's motor control and why—as the patient sees it. The patient is often able to verbalize or express his experience of how different things may influence each other, i.e., stress and tone
- The patient's perception of his own situation: frustrations, hopes, needs, goals

If the patient is unable to communicate, this information needs to be collected from his carers, who are often able to give good information and insight.

A person who suffers from acute illness or trauma undergoes catastrophic changes in his life, which may last for a short or long time or may even be permanent. The therapist needs to find out where he is in his own rehabilitation process: Is he in shock or have he and his carers started a process of reorientation. Time and information is of the essence and may be the most important intervention besides specific treatment and empathy. It does take time to realize and understand what has happened, and it is not possible to get an overview of the extent or consequences of the lesion for the patient at once.

Functional Activity

This part of the assessment builds on interview, observational analysis, and handling together. The aim is to clarify *what* the patient is able to do, his degree of independence, and his ability to cooperate and interact.

During the interview the patient is asked what activities of daily living (ADLs), personal hygiene, instrumental ADLs (IADL, e.g., going to the shops), and leisure activities he is able to do which are relevant to the situation now. His ability for activities informs the therapist of:

- General condition and general level and ability of movement
- Communication function
- Functional activity:
 - Quantity: *What* the patient is able to do
 - Quality: *How* the patient moves
 - Clinical reasoning process: Why does he move in this way
- Use of aids

If the patient is in an acute phase after a CNS lesion, both his level of activity and his movement control will alter quickly due to spontaneous recovery and increased learning potential. The therapist needs to evaluate the patient's condition and abilities continuously to adapt and change her interventions appropriately to enhance the patient's recovery.

General Condition

Observation of the patient's general condition provides the initial impression of the patient's status and how he feels:

- General condition and respiration
- Stamina
- Comfort and feeling of security
- Effort
- Ability to relax
- General autonomic function (see p. 156)

Communication

During the interview and observation of the patient's general condition the therapist forms an impression of the patient's verbal and symbolic understanding. Does he understand words? Does he understand nonverbal instructions given through the use of gestures?

Functional Activity: What? How? Why?

Berta Bobath said, "See what you see, and not what you think you see" (cited in Schleichkorn 1992, p.48). Observation of the patient's activity starts when the therapist sees the patient for the first time, and before any form of intervention, such as transferring to a plinth, and before he is asked to change his clothing if appropriate. The patients' movement repertoire is analyzed through functional activity (Table 3.2). If he has the ability to stand or walk, transfer, and dress and undress in sitting and standing, these functions are analyzed first, as appropriate. The patient is met at the level he functions; if he is unable to do any of the above-mentioned activities, his ability to accept the base of support, to maintain a position and move within it, his ability to be placed are assessed. A general observation informs the therapist of:

- Feeling of security
- Effort
- Time, efficiency, appropriateness
- Posture
- Balance (postural control, righting, and protection)
- Patterns of movement, sequence of activation and alignment
- Selective function of the extremities. Ability to vary and change
- Tone
- Compensatory strategies
- Associated reactions
- Sensation
- Perception: attention to and experience of one's own body in relation to the environment
- Cognition: attentiveness, understanding, focus, problem-solving abilities, memory, concentration, and insight
- *What* functional activities is the patient able to perform? Is he sitting passively in a chair or lying in a bed without the ability to move? How does he respond when facilitated or helped? Can he transfer, stand, or walk and how safe is he in those situations?

The analysis is both resource and problem oriented. It is supposed to give answers to what the patient can realistically perform independently, when he needs support, and how he solves his tasks through movement. It is important to discuss with the multidisciplinary team

Table 3.2 ICF sections

• Interaction with the environment	The patient's ability to interact with the environment: – The patient moves in relation to the environment. – The environment, people, and objects move in relation to the patient, gives information on the patient's perceptual and dual task capacity and how automatic his balance is.
• Transfers	<p>For example in a wheelchair or walking depending on his functional level, weight transference in different postural sets of sitting and standing, in the transfers standing-sitting/chair-bed or other chair/in and out of bed, i.e., his ability to control and vary movement. The key words are postural stability and orientation, eccentric and concentric control.</p> <p>What is he able to do by himself, what does he need help for, and why?</p> <p>The transfers from sitting to supine and vice versa are possibly the most complex and demanding tasks a person performs in his daily life.</p> <p>This transfer requires that we are able to eccentrically grade the movement from sitting to supine through a continuous changing relationship with the base of support, rotational components to align the body to the new base, eccentric work combined with aspects of specific concentric activity to lower the body down. Sitting up from supine requires a selective, graded, varied recruitment of motor activity through rotation to align the body in sitting on the edge of the bed. Both these transfers need the combination of flexion, extension, and rotational components, and a controlled recruitment of motor units for selective eccentric/concentric activation based on postural control from one position to another. The relationships with gravity and the base of support are very different in the postural sets of sitting and supine, and therefore require different muscle activation to achieve and maintain.</p> <p>There is a conflict between the complexity of this task and the expectation from health personnel that the patient should be able to achieve this function as soon as possible for the sake of independence.</p> <p>For most patients, the transfer from sitting to standing, standing and walking is easier than getting in and out of bed.</p>
• Dressing and undressing	Dressing and undressing require both postural control and righting to enable the patient to weight transfer in sitting or standing and free their arms for function (see Chapter 2, Figs. 2.40–2.43 and 2.54–2.60). For most patients dressing and undressing will also require learning, as many must find new strategies or even wear different clothes to master this task.
• Personal hygiene	Is the patient able to manage visits to the bathroom by himself? Is he continent, does he participate in washing himself in the morning? Is he used to taking a shower or bath, and can he manage this? Is he able to get out of bed, to sit, or stand for any of these functions? If not, what help does he need? Why?
• Eating/drinking	Does the patient eat or drink by himself? Does he spill food/drink, why? Sensation in his face may be poor, his motor control around his mouth may be decreased, or there may be perceptual/cognitive dysfunctions. If the patient coughs when he drinks or eats, he may have dysphagia. This is often a problem that is overlooked if the problems are small, but may cause complications for nutrition or for the patient's lung function and be an important social factor.

Table 3.2 Continued

• Perception and cognition (see also Body Function and Structures)	<p>How does the patient interact with his own body and with the environment? Is he able to avoid obstacles; is he attentive to people, furniture, objects? Is he able to vary his movement repertoire in relation to the room and what is in it? If in a wheelchair, how does he relate to it? Can he maneuver it himself, how? How does he problem solve footplates, brakes, and table.</p> <p>If he is able to dress and undress or participate in this activity, is he able to cross his body to undress sleeves, does he find his arms and legs? How does he solve the task? Is he focused, attentive—to what degree? Does he finish what he has started? If the patient has suffered a stroke, how does he take care of his affected arm during these tasks?</p> <p>If the patient has problems understanding or responding, either to verbal information or problem solving a “new” situation, he may have cognitive deficits. The therapist needs to find out if he has organic deficits (vision or hearing) or cognitive problems. His ability to problem solve may be assessed in all practical situations: use of wheelchair or walking aids, during transfers or any other relevant activity.</p> <p>If the patient seems to have perceptual or cognitive deficits, a multidisciplinary assessment is of special importance. Nurses, assistants, carers may inform the team about how the patient problem solves and masters different situations through the day, and about his concentration, attention, moods, insight, self interest and interest in his environment. Neuropsychologists and occupational therapists may assess the patient more specifically, and give advice on how the patient should be helped in daily activities to enhance his perception and cognition. The patient's perceptual and cognitive function should be assessed and reevaluated over time for appropriate treatment and to evaluate the consequences for the patient's function and life.</p>
--	---

and the patient's carers to form as full a picture as possible, as well as observing the patient when he is not aware of being observed.

Use of Aids

Does the patient need aids: a wheelchair, walking aids, other technical or orthopedic aids? Why?

Information from other health professionals working with the patient gives a more complete picture of the patient's strengths and weaknesses.

■ Observation

This informs on overall functions, both visible and invisible aspects; spatial relationships, perception of one's own body and its relationship with the environment, other perceptual and problem-solving abilities, concentration, attention, motivation, mood, and orientation as well as sensation and movement ability. Therapists tend to assess the patient in different positions, and because we move *between* different postures and positions normally, the patient is assessed in relation to dynamic activity. Assessment in supine may give additional information about tone, alignment, and range of movement, if appropriate. If the patient experiences increased problems with hypertonia during the night or in the morning before or as he is getting out of bed, his sleeping patterns and positions need to be assessed specifically.

■ Body Functions and Structures

This part of the assessment involves observation, handling, and analysis. Important factors are:

- Quality of movement, movement patterns, stability, and mobility
- Sensation, perception, and learned nonuse
- Pain
- Autonomic function

■ Handling

During handling the therapist assesses how movement is performed—initiation, recruitment, sequence, alignment—and gains information about muscle activity, stability, and movement within and between key areas. The therapists then forms hypotheses about which key areas seem to be most affected or fixed. Through an invitation to move through handling and facilitation, quality of movement and muscle activity as well as range of movement is assessed further. Assessment is not passive; it is the patient's ability to recruit activity and move which is important. Through correction of components that seem deviant or malaligned, the therapist again invites the patient to move through handling. Has anything changed, positively or negatively? In this way, the therapist gets an impression of tone distribution, stability, postural control and balance, tempo and selectivity, and the ability to vary and adapt.

■ Analysis

Movement is analyzed through muscle activity, interplay, alignment, patterns of movement, which may have consequences for the patient's ability to regain and learn movement. Observation and handling is not performed satisfactorily without the patient being adequately undressed, preferably wearing short trousers (with a bra or sun-top for women). If the patient is unwilling to undress to this level, even if other people are not around and after being given information on the importance of being undressed to allow the therapist to analyze his movement properly, his wishes must be respected.

Assessment of body functions and structures require the therapist to have a good knowledge of normal movement and to be competent to analyze interplay of movement specifically. This part of the assessment is qualitative and resource and problem-oriented. The therapist needs to gain insight into *how* the patient moves, *what* he is able to perform, and *how* movement is being performed. This requires an analysis of body functions and structures in the activity dimension (ICF). Handling allows the formation of hypotheses of which *neuromuscular interplay* brings the patient to where he is. The musculature changes alignment and moves joints under

the influence of gravity. Component analysis is the analysis of neuromuscular activity.

Knowledge of normal movement and prerequisites for balance and movement in normal situations allows the analysis of deviations from normal movement. The patient's way of moving is analyzed in relation to hypotheses of how he moved before the CNS lesion.

■ Quality of Movement

The therapist needs to have a picture in mind of how the patient might have previously moved, and view the deviations in this light. It is complicated to analyze simultaneously both how the patient is moving and the underlying neuromuscular connections and the consequences this may have for the patient's function. It may be easier to divide this part into phases: What neuromuscular activity does the patient recruit to maintain a posture or to move (Fig. 3.3)?

The following qualities are evaluated in relevant postural sets and activities:

- Midline orientation: Does the patient move in all planes and seem to have a perceptual relationship to his own body and the environment?
- Ability to move to and from the physical base of support is evaluated through observation and handling. Is the patient able to adapt his tone and neuromuscular activity to be where he is, to maintain the postural set, and move through weight transference and righting to another postural set? For example, if the patient is sitting, the therapist may place her hands over his greater trochanters, sense the hips, the position of the pelvis, his ischial tuberosities, and the muscle activity in the area. The patient is facilitated to move in different directions and the therapist observes senses and analyzes the adaptive movements of the related key areas during weight transference in different directions. The therapist observes and handles the feet and the hands to assess their adaptive abilities: to weight transfer on to the feet or the shaping of the hand to different objects; reach, grasp, and let go.
- Interplay and interrelationship between different key areas: The key areas are assessed both individually and to see how they adapt to each other through movement. Neuromuscular activity is analyzed in the specific postural

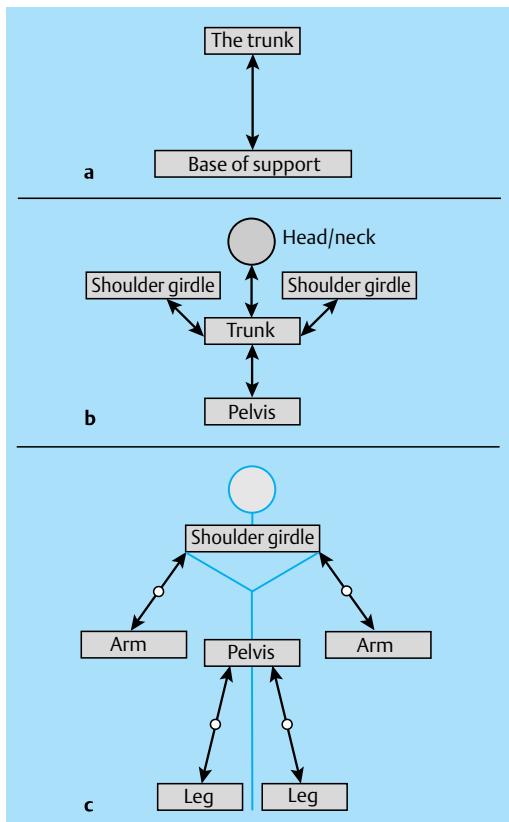


Fig. 3.3a–c

a Observation and analysis of the patient's line of gravity in relation to the base of support. Where does it fall, and which neuromuscular activity must the patient recruit to be where he is and to move from there? The trunk, head, and neck are central to balance and require coordinated movement in three planes. Analysis of this relationship gives information on:

- Holistic function and balance
- Midline: symmetry/asymmetry
- Weight distribution: active/passive (weight)

b Analysis of the connection and interplay between the central and proximal key areas

- Interplay
- Selectivity
- Variation and change
- Mutual influence

c Analysis of patterns of movement within the extremities and the distal–proximal relationships

- Adaptation to the environment
- Patterns
- Selectivity
- Mutual influence

sets the patient adopts and moves within, i.e., the patient's relationship to gravity and the base of support.

- The interplay between the trunk, head and neck, the pelvic girdle, and the shoulder girdles gives information on the patient's postural control, especially core stability and his righting ability.
- Is the patient able to maintain postural stability as he moves his extremities (or they are moved for him), or is the patient displaced or dependent on fixation through the arms/legs to maintain balance?
- The interplay between the hands/feet and the proximal key areas informs of postural control, selectivity, patterns of movement, and the ability to vary in relation to the goal (task).
- Is the patient able to support himself using arms and legs and move his trunk in relation to his extremities?
- Patterns of movement, sequence of activation, and biomechanical factors: Joint position and range, rotatory components, alignment inform of which neuromuscular activity the patient has used to get where he is and is currently using. Does the patient recruit anticipatory postural activity to maintain stability for movement? Does this background activity vary with different tasks? Is selectivity present in all parts or are some movement components missing or inadequate?
- Selective control of movement: The individual movement and neuromuscular activity within the key areas. Is there freedom of movement and activity in all planes? *Selectivity* is controlled activity of one body part based on stability in another. Does the trunk rotate too early or too little during arm movement; is the head free to scan the environment? Is the scapulae free and stable or does it glide too early when the other shoulder girdle is moved during tasks?
- Muscle quality: flexibility, length, and elasticity. Does the musculature exhibit qualities needed for interplay of eccentric and concentric activity? Is compartmentalization maintained (see Chapter 1.1 Systems Control, The Neuromuscular System).

- Tone:
 - Normal, adaptable tone that varies in relation to different postural sets and activity
 - Hypotonia: tone that is less than that which would seem appropriate or expected for the activity. Is there an increased sense of *weight* (limpness or flaccidity, inactivity, weakness) during movement or when moving and handling the patient?
 - Hypertonia: more tone than would seem appropriate or expected for the activity. Is there resistance or increased assistance to movement in one direction? Where? Which qualities does this express?
 - The presence of associated reactions: when are they expressed? In which situations (see Chapter 1.3, p. 61)? Is it possible to facilitate the patient's postural control and movement so that the associated reactions are diminished?
 - Spasticity (see Chapter 1.3, p. 59) or secondary problems of soft tissue changes?
- Compensatory strategies. Which strategies does the patient employ to solve motor tasks? Appropriate or inappropriate? Is it possible to facilitate the patient to make such strategies to a certain degree superfluous?
- Does the patient respond to facilitation? Is it possible to facilitate the patient to take over and make the movements his own (placing)? If not, why not? Is there resistance to movement, where? Is there little or no muscular activation, is the stimulation strong enough? What are the consequences? If the patient does respond, where is the initiation, how is the pattern?

The patient's response to being handled gives important information about how the patient may be facilitated, if he tolerates the closeness of the therapist and if he interprets the information and demands that she transmits through handling. This requires that the therapist is precise in her handling and facilitation.

Sensation, Perception, and Learned Nonuse

Observational analysis gives the therapist an impression of whether information is integrated in the patient's CNS or not, and if this information is being used in relation to the patient's ability to move. Sensory testing may be performed to assess the patient's *conscious* awareness of sensory impulses, which is important especially for stereognosis and dexterity of the hand (see Chapter 1.1 Systems Control, Stereognostic Sense). Testing shows how sensory information is transmitted and processed within the CNS. Poor sensation may be due to lesions of the ascending systems directly or due to sensory perception, i.e., association areas. If sensory perception is affected, the CNS does not interpret the information that it receives, which is a problem with sensory integration and not reception. Therefore, results from sensory testing should not be used to decide on a patient's rehabilitation potential. If appropriate, sensory testing should be performed both before and after a period of intensified sensory stimulation to the most affected body part. Sensory stimulation aims at improving both transmission and the patient's focus toward the body part that is being treated. An improved focus often implies that the patient's CNS starts to interpret and integrate sensory information. The therapist should attempt to discriminate whether the sensory problems are organic (sensory pathways) or perceptual, as this is important for therapeutic intervention.

Lesions of the Ascending Systems

It is necessary to have a good sensory perception for discriminative tasks, such as threading a needle. In this case sensory testing may be appropriate. Discrimination requires localization of sensory inputs.

Starting Position

In sitting, the patient should place—or get help in placing—both his hands behind his body with the palms up. In this way he is not able to see what the tester is doing, and tonic influences are often neutralized as the arms are in flexion, abduction, and internal rotation with flexion at the wrists. First the therapist needs to gain a gen-

eral impression of whether there are differences in superficial sensation between the hands, using her own hand to touch.

Finger Discrimination

Finger gnosis is tested by the therapist touching one finger at a time and asking the patient to name the finger. Recognition of the individual fingers is important for discrimination, and informs the therapist if the fingers have maintained their cortical representation.

- If the patient is aphasic, he may move the same finger on the opposite hand to indicate which one he thinks it is.
- If recognition is weak, but mostly correct, there is some connection to the cortex.
- If there is no recognition, the patient has *finger agnosia*: the cortical representation of the individual fingers is not aroused.
- The patient may have receptive problems and not understand these instructions, in which case testing like this is inappropriate.

Sensory stimulation may improve this to some level, but the patient's prognosis for discriminative hand and finger function is poor.

Localization of Touch

Two-point discrimination is performed to test the ability to precisely locate stimuli. The therapist uses two equal and sharp objects (needles or similar) and starts by testing the patient's index finger, because this finger is most densely packed with sensory receptors. The therapist pricks the patient simultaneously with the two objects. The therapist needs to test for different distances between the two points at different locations on the finger to find where the patient is able to discriminate the two points. The smallest distance for two-point localization is measured for future reference.

Joint Position Sense

The therapist moves the joints of the index finger or the thumb and asks whether the patient can describe the position or copy with the other hand. If he is not able to do so, the therapist may test his wrist and gradually more proximal joints of the arm. It should be noted that this form of testing is very limited:

- Joint position sense depends more on input from active muscles and compression/stretch of skin than joint receptors alone

- Only the patient's conscious awareness is tested, not how the CNS receives, interprets and integrates the information that it actually receives (see Chapter 1.1 Systems Control, The Somatosensory System), therefore firm conclusions should not be drawn from these results if they are deviant

Conscious awareness of sensory information is more important for hand function than for walking. In people with CNS lesions there is no primary damage to ascending systems at the level of the spinal cord. Sensory impulses are received and integrated to some degree in the spinal cord, and transmitted to the cerebellum and other higher centers. This information may therefore be used for pattern generation and inter-limb coordination through the cerebellum. The patient's fine tuning of balance will be impaired to some degree if sensory information from the soles of the feet is diminished (Kavounoudias et al. 1998, Meyer et al. 2004).

Perceptual Function

Patients with CNS disorders may exhibit perceptual dysfunctions that cause decreased attention or neglect toward the most affected side. Neglect is obvious in patients who do not turn to the affected side, do not take care of, or dress their most affected extremities, walk or wheel into objects, people, door frames, furniture on the most affected side.

Some patients do not integrate information from the most affected side when they receive information from their less affected side at the same time. The therapist may suspect this form of inattention if the patient has some movement in their most affected arm, but do not attempt to use it. This may be assessed through *simultaneous bilateral touch*. The prerequisite for performing this test is that the patient does have sensation when the most affected side is tested alone.

Simultaneous Integration

The test is performed in the same testing position as above if possible. The therapist stands behind the patient and touches one arm at a time, asking the patient to say which arm/hand is touched (right or left). At intervals she touches both arms or hands at the same place at the same time. If the patient still only says the less affected side, this implies that information from the most affected side is suppressed, i.e., not integrated.

This means that when the patient is in situations that require integration of stimuli from both sides at the same time—in traffic, among people, in many daily situations—he may be in danger of causing damage to himself.

Learned Nonuse

Patients may exhibit sensory problems as a result of inactivity or nonuse. Usually this applies more to the distal body parts, hands, and feet, more than the rest. Learned nonuse may be overcome by stimulating, mobilizing, and facilitating the patient's activity. If the patient expresses that he can feel his hand or foot better after treatment, this implies that there is a degree of learned nonuse (see Chapter 2, Physiotherapy).

Pain

Pain may limit the patient's recovery and learning processes and lead to depression, loss of motivation, and social isolation. The patient may experience pain when the arm is being moved for instance during dressing and washing and this may lead to withdrawal from daily activities and treatment and deterioration of functional ability.

Possible Causes

- *Increased tone:* malalignment and possible fixation of joints in unnatural postures, static activation of muscles and decreased circulation, or sudden pulls (cramps, spasms)
- *Trauma:* due to poor handling, falls, or instability of joints
- Altered sensory awareness/perception
- Other causes (e.g., disuse over time, swelling, inflammation, degenerative conditions)

The physiotherapist must assess the cause of pain; where the pain is, which situation exacerbates or improves it, and when the patient experiences pain (day, night, during activity, at rest), and thereby get an impression of the causal factors and severity. History and movement analysis (observation and handling) as well as information from other examinations (X-rays, ultrasound, other) and from carers as well as using a *visual analogue scale* (VAS) may be useful. Pain is always a priority of treatment.

Autonomic Function

A CNS lesion may cause altered autonomic function both locally and more generally. Local changes are present in many patients with CNS dysfunction, and may be caused by dysfunctions in central regulation or as a result of inactivity and immobility. It often manifests itself as a more distal problem, in the hands or feet.

- Altered circulation: the skin color is more bluish, reddish, or pale.
- Changes in temperature follow changes in circulation: the extremity is cold to touch. If the patient has an infection such as vasculitis the area will be warmer and redder.
- Swelling is observed and palpated, and is fairly common in the hand or foot of a patient who has had stroke or has MS. If chronic, it may cause further circulatory-, movement- or pain-related problems. If there is a generalized stiffness and/or swelling in a patient's leg and/or thigh combined with pain or tenderness, or the pain increases when the foot is dorsiflexed and the big toe extended (Homans sign), the patient has to be examined for possible deep vein thrombosis.

Skin Quality

There may be changes in the skin due to inactivity, immobilization, and decreased circulation. Inactivity may lead to thick and hard skin, and may cause further immobility of the affected area. A hand that is not used becomes drier with increased skin thickness because the dead skin does not rub off during use.

General Symptoms

These are more common in spinal cord injury (SCI), especially complete SCIs. The symptoms may vary in intensity and character:

- Sweating above trauma level
- Increased heart rate
- Headaches
- Increased blood pressure
- Reddened skin

Clinical Relevance

Stroke patients frequently suffer from shoulder or wrist pain, or a combination. In a systematic review by Geurts et al. (2000), both shoulder-

hand syndrome (SHS) and *post-stroke hand edema* are described. Geurts et al. concluded that:

- The shoulder is involved in only half of the cases with painful swelling of the wrist and hand, suggesting a *wrist-hand syndrome*.
- Hand edema is not lymphedema.
- SHS usually coincides with increased arterial blood flow.
- Trauma causes aseptic joint inflammation in SHS.
- No specific treatment has proved advantageous over any other physical methods for reducing hand edema.
- Oral corticosteroids are the most effective treatment for SHS.

In the author's experience, careful but persistent mobilization combined with correction of alignment and sensory stimulation may improve this problem.

WHAT? HOW? WHY?

These are the three most important questions in the ongoing assessment of the patient.

Clinical Reasoning

History, observation, and handling form the foundation for the process of clinical reasoning as well as the therapist's special and general competency and information from others. Activity and participation are assessed in combination with the patient's problem-solving ability and motor behavior in relation to the tasks and environment:

- Resources and restrictions in participation
- Resources and limitations of activity
- Deviations or loss of functions and structures of the body as a direct result of neurologic lesions or consequent to the lesions. Quickly learned compensatory strategies may be difficult to separate from the direct results. Compensatory strategies may lead to further deviations. In this context, deviations from body functions and structures relate to those problems that change the patient's prerequisites for normal movement control.
- Changes in tone will influence the patient's ability to remain upright, interact with gravity, and may cause malalignment, alterations in muscle length, muscle flexibility and elas-

ticity, range of movement, changes in non-contractile tissues, and the ability to vary eccentric and concentric activity.

- Changes in reciprocal innervation may disrupt the muscular interplay between agonists, antagonists, and synergists, alter the recruitment sequence of motor units, and the interplay between stability and mobility in movement.
- Changes in patterns of movement and their ability to vary according to the goal disrupts the sequence of muscular activation and causes altered alignment and thereby altered working relationships for muscles.

Problems with body structures and functions cause reduced balance and movement, and will influence the patient's ability to transfer and to perform daily activities (Fig. 3.4). Therefore, the therapist's specific knowledge of movement and analysis are important tools in assessment and treatment, and for the process of reasoning to improve the patient's ability to act and interact.

Clinical reasoning is based on the therapist's general and specific knowledge and experience, both professional and personal. The therapist needs to evaluate all findings from interview, observation, and handling to gain a picture of the individual patient. Clinical reasoning is a process of problem solving whereby the therapist formulates a *main problem(s)* or *hypotheses* based on the collected data, causal relationships, and what the patient expresses as problems. This leads to goal formulation, interventions, and evaluation of interventions (Fig. 3.5). Clinical reasoning requires an ability to analyze the interaction between the various ICF dimensions.

The therapist needs to:

- Understand the patient's needs and expectations
- Gain an impression of the patient's resources and limitations in all three dimensions
- Formulate hypotheses about the factors that seem to be most important and limiting for the patient's level of activity, his movement ability, and the way he moves
- Choose goals, both short and long term, preferably in cooperation with the patient
- Choose treatment interventions: tools
- Evaluate treatment interventions and develop further hypotheses. Is the hypothesis appropriate? Judged on the results here and now and over a longer period of time

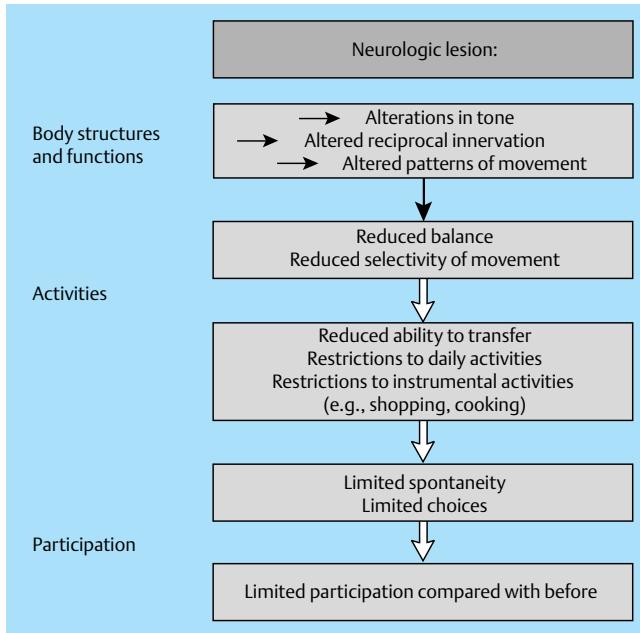
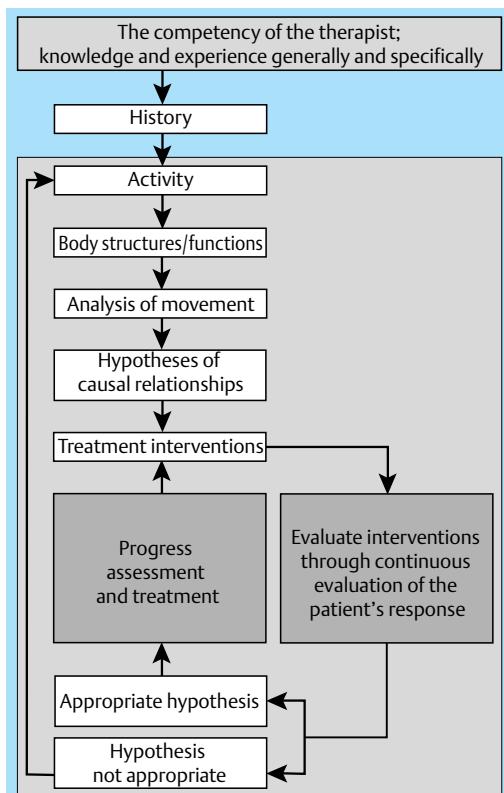


Fig. 3.4 Consequences of a lesion to the CNS.



The Aim of Assessment

The aim of assessment is two-fold:

- To create a hypothesis about the patient's potential
- To create hypotheses of *why* the patient moves as he does

What is the patient able to do? Which resources does he have? If function fails or if there is lack of function, the question arises: *Why* or *Why not?*

- Is it due to balance or movement problems?
- Is it due to somatosensory and/or perceptual dysfunction?
- Are there cognitive problems?

Causes Based on Balance or Movement Problems

- Which neuromuscular activity is recruited and is not recruited in different situations?
- Is activity missing or not changing, which should change between different activities normally?
- Is tone low because the patient fixes using compensatory strategies somewhere else and thereby does not allow efficient recruitment of muscle activity?

Fig. 3.5 Assessment and clinical reasoning.

- Are there primary sensorimotor problems or a combination of these and inappropriate compensatory strategies?
- Does the patient display reduced postural control and balance?

Generally the somatosensory, vestibular, and visual systems have important roles in postural control and orientation.

Are there dysfunctions in any of these systems, direct or indirect? In stroke, the vestibular nuclei are seldom affected directly, infarction or bleeding are rare at this low level in the brainstem; also the vestibular nuclei are not under direct cortical control. If the patient's ability to move is reduced, this may lead to altered somatosensory information to the vestibular system, and the vestibular nuclei may become dysfunctional. Visual problems are frequent in many different neurologic conditions, e.g., nystagmus, hemianopia, and other visual field deficits, visual neglect or inattention. All these will influence the patient's postural control.

- Does the patient display reduced postural tone or areas of associated reactions that cause reduced postural control and balance? If so, the cortico-reticulospinal and the cortico-rubrospinal systems may be dysfunctional. These systems are partly responsible for postural tone and proximal stability.

Clinical experience suggests that balance dysfunctions may be caused by changes in tone (more or less than normal), reduced or altered movement control and interplay between segments and/or perceptual dysfunction. Balance is one of the more automatic functions in human movement. If balance is reduced, the therapist needs to choose interventions (functions, activities, postural sets, handling, etc.) which enhance balance and movement on a more automatic level.

- Does the patient have any selective control in his arms or legs?

Selective control is understood as controlled and coordinated movement of one body part or one joint based on postural stability. Is there any sign of movement in the fingers or toes? If not, why not? Could it be that the postural components are not activated, thereby not allowing stability and selective interplay between key areas? Is there true paresis and therefore severely reduced neural activation due to the lesion itself? With true paresis especially distally, the corticospinal system would be partly damaged to

varying degrees. The cortical systems are the most voluntary activated (least automatic) systems in human movement control. Enhanced demands for selective focus combined with voluntary activation may be appropriate when selective movement is primarily affected.

Examples

Some patients have a problem with hyperextension of their most affected knee during loading of their leg. This may be caused by several factors:

1. Incoordination between agonists and antagonists around the knee or reduced interplay between the hip/pelvis and the ankle/foot as a direct result of the lesion (body structure and function)
2. Over-reaction due to hypersensitivity to sensory/proprioceptive input (stretch or cutaneous hypersensitivity) as the foot meets the floor to weight-bear (body structure and function) (see Figs. 2.10, 2.11a, 2.13–2.14)
3. As a consequence of the patient "stiffening" his knee to be able to stand and walk (activity dimension), i.e., a compensatory strategy

The neuromuscular activity in the hip region should vary in different phases of stance and swing. Does the patient's neuromuscular activity vary as it should? Is extensor activity recruited appropriately at heel-strike and through stance? Is there eccentric lengthening of hip flexors and adductors to allow stability?

- If hip extension is not recruited appropriately, the therapist needs to facilitate this activity. Lennon (2001) demonstrated through two case histories that recovery of more normal movement patterns for walking and functional ability can be achieved through physiotherapy post-stroke. Hesse et al. (1998) demonstrated a more balanced walking pattern with facilitation according to the Bobath Concept during therapeutic intervention. Facilitation enhances a more optimal recruitment (body structure and function) during functional activity (activity dimension).
- If the flexor musculature does not eccentrically lengthen to allow for hip extension, the therapist needs to mobilize and facilitate the eccentric control (body structure and function) and place demands on the hip extensors (make necessary—let it happen) during activity (see Chapter 2, Physiotherapy, Handling)

The activity around the shoulder girdle is an important component in all transfers (activity dimension). The neuromuscular activity in the interplay between the shoulder girdle, the arm, and trunk should vary depending on the activity to be performed (body structure and function). If the shoulder girdle is fixed in, for instance, elevation, the trunk on the same side is kept elongated and cannot shorten for stability or weight transference. The therapist needs to hypothesize why the shoulder girdle is kept elevated:

- Is there reduced stability in the thorax or between the thorax and the scapula? Why?
- Is the instability due to reduced intercostal activity (body structure and function)?
- Is tone too low/too high, and causing malalignments and poor coordination (body structure and function)?
- Is distal activity reduced (body structure and function) and causing the patient to attempt moving the arm from the shoulder to be active and task oriented (activity dimension)?

Hypotheses on cause and effect leads to goals for treatment and possible interventions. The treatment will differ depending on which hypothesis is most probable, and the continuous evaluation related to this.

- In which way does the patient compensate? Why?
- What components of movement is he missing that drive him to compensate in the way he does?
- Does he seem afraid or insecure? The patient needs to feel safe to explore his own movement abilities with less compensatory strategies.

The patient's compensatory strategies change according to the different activities he attempts to perform, while the main problem will be a dysfunctional component (or more than one) throughout. If the patient moves more appropriately with less compensation after intervention, the hypothesis is strengthened.

- Where and why are there associated reactions?

In which situations do they occur? As the patient is attempting to balance or move his arms or legs? How is the patient's postural control? How much effort does he use just to stay where he is?

Two aspects seem especially important when assessing and treating patients displaying associated reactions:

1. Analyze and formulate hypotheses as to the reason why:
 - Do the associated reactions seem to be a response to balance demands? The reason may then be reduced stability and equilibrium (postural) control. (Reduced interplay between stability and mobility between key areas; reduced stability more specifically related to the hip/pelvic area or the trunk; reduced stability and equilibrium control distally and therefore inefficient ankle strategy).
 - Do the associated reactions seem to be related to movement of the extremities? What is the quality of the patient's movement ability, sequence of recruitment, selectivity, variation? Is tempo, initiation, or strength reduced?
 - Do the associated reactions increase with effort? Why does the patient increase his effort in situations that normally are not strenuous?

2. Does the patient display any control over his associated reactions? Is he aware of them and does he himself hypothesize on why they happen?

Many patients have good body awareness and may sense and verbalize causal relationships. These hypotheses are frequently correct, and the therapist needs to explore this in assessment and treatment.

■ Causes Based on Somatosensory or Perceptual Dysfunctions

Sensory information reaches the spinal cord and is modified and integrated to a certain degree already at this level. In stroke, there is small probability of a patient experiencing sensory deficits at this level. The spinal cord "senses," even if the patient cognitively does not. Sensory loss in stroke is caused by lesions to the ascending pathways or structures (e.g., internal capsule, thalamus, or cortex). Decreased sensation may be caused by learned nonuse or lesions to the perceptual systems causing inattention or neglect to the more affected side. In multiple sclerosis, information may be disrupted at spinal cord level as well as in other areas of the CNS.

It may be difficult to differentiate between sensory loss and reduced sensory perception. Treatment aimed at drawing the patient's attention and ability to feel through improved mobility may reveal some answers; if the patient's sensation of the body part improves during treatment, it is possible that the problem is due more to a reduced sensory perception than true sensory loss. Learned nonuse may be improved through specific therapy as well as activating the extremity in all functional contexts. Perceptual dysfunction is seen in most daily life activities; the patient may display reduced attention in relation to his own body and to his environment. It is important to develop a good working relationship with other health professionals to assess these dysfunctions and make a plan for interdisciplinary intervention. The patient's attention must be stimulated and demanded in all activities.

Balance and movement require the ability to perceive where the different body parts are in relation to each other and the environment. If the patient has perceptual dysfunction, this may influence his perception of midline, and thereby interplay and balance. Treatment aimed at orienting the patient to his own body improves the interplay and coordination between body segments, which may lead the patient to be more oriented to his own body in the environment.

■ Causes Based on Cognitive Deficits

Does the patient understand what he is doing or is being asked to do? What about his hearing? Is he aphasic? Is he depressed or uncritical? Does he display insight and can his problem solve new situations? Is he concentrated and focused? Which consequences may this have for his functional ability?

Answers to all these questions are primarily hypotheses as they guide treatment and the choice of interventions related to the patient's movement problem.

Choice of treatment follows a process of clinical reasoning. Observation and movement analysis in activities form the basis for hypotheses of which systems seem to be more affected and which ones seem more intact.

Clinical Example 1

The patient, Sissel, is asked to remove the foot-plates of the wheelchair (Fig. 3.6).

Observation

- Sissel looks to her right (more affected side), weight transfers to her right and reaches down to find the release mechanism to the foot plate. She takes hold of it and adjusts her grip. She lifts her right leg off the foot plate, taking a little help from her left arm. She then removes the right foot plate.

Clinical Reasoning

- Cognitive function:** Sissel remembers the instruction. She is attentive to her right, realizes where the release mechanism for the foot plate is, integrates the information, and problem solves how to remove the foot plate. She is focused and concentrated on the task. In this way, Sissel demonstrates that she understands, remembers, and solves the task. Her cognitive ability is good in this context.
- Perceptual function:** She displays that she no perceptual dysfunction through her attention to her more affected side. She receives, perceives, and integrates information from her more affected body part adequately for this situation.
- Sensation:** Probably her sensation is good, because she is able to adjust her grip on the release mechanism even if she does not lean over far enough to make direct eye-contact with it.
- Balance:** Sissel's balance may be reduced because she uses her left arm to seek support on her right thigh and does not rotate her body far enough to gain eye-contact with the release mechanism.



Fig. 3.6a



Fig. 3.6b

Fig. 3.6a, b Clinical example 1.



Fig. 3.7a



Fig. 3.7b

Fig. 3.7a, b Clinical example 2.

Clinical Example 2

Sissel reaches for a tissue (Fig. 3.7).

Observation

- Sissel does not right herself spontaneously as she reaches for a tissue. Her posture is flexed, both trunk and head/neck, and it seems as if her trunk is pulled down with the movement of her arm. Sissel demonstrates that she is able to right herself through facilitation to her pelvic area.

Clinical Reasoning

- Why does she not right herself as she reaches forward (activity dimension)? The anticipatory interplay between trunk stability and arm function seems to be reduced (body structure and function).
- She is dominated by flexion. Are the pectoral muscles recruited too much and too early in this movement? Do compensatory strategies prevent her from recruiting more appropriate activity?
- The therapist needs to sense and understand the difference in quality of movement and evaluate why these differences occur to form hypotheses about causal relationships. Clinical reasoning follows through into treatment, which is continuously evaluated by the therapist.
- The therapist needs to assess physical and neuropsychologic problems and decide on how these affect each other. Frequently there are combinations of dysfunctions. The therapist needs to analyze and interpret the data to inform or request supplementary examinations and treatment by the doctor, occupational therapist, speech therapist, neuropsychologist, or others as needed.

Assessment and treatment are integrated in a continuous process.

3.3 Outcome Measures

There is an increasing focus on the use of formal, validated, and reliable outcome measures to document change in neurologic rehabilitation. Most of these measures assess the patient's activity, and few focuses on the assessment of impairments and how these may affect the patient's function. Most rehabilitation centers and hospitals have chosen some outcome measures that they prefer in their working context. This book will not describe outcome measures in detail; only a few specific ones are mentioned.

Body Structure and Function Measures

- The Trunk Impairment Scale ([TIS] Verheyden et al. 2004, 2005). This scale aims at measuring quantity and quality of trunk stability and movement in sitting. It has been validated for stroke patients (Verheyden et al. 2004). It evaluates the patient in three main domains: (1) static sitting balance with three items; (2) dynamic sitting balance with 10 items; and (3) coordination with four items. A total score of 23 indicates normal trunk control. Verheyden et al. (2005) refer to studies that state that stroke patients have impaired trunk muscle activity and demonstrate the correlation between paretic trunk muscles and limitations in everyday activities. Studies indicate that postural control is a significant predictor of motor and functional recovery after stroke (Hsieh 2002). Trunk function—or core stability—is an essential part of postural control. TIS discriminates between stroke patients and healthy individuals. Verheyden et al. (2005) found a sub-maximal score in 45 % of healthy subjects (mostly elderly men, mean age 69 years) suggesting that the lower score on this scale still indicates a trunk function within normal limits and a full participation in daily life.
- The Rivermead Visual Gait Assessment ([RVGA] Lord et al. 1998). This test consists of two observations of the arms at swing and stance phases of gait, and 18 observations of the trunk and lower limb: 11 observations during stance phase and seven during swing phase of gait. A four-point scale is used to

- quantify the degree of abnormality for each of the component items. A global score is calculated by summing the total numbers of scores, range from 0 (normal gait) to 59 (grossly abnormal gait). RVGA can be applicable to measure change over time for patients with neurologic disease, and is sensitive to gait impairment (Lord et al. 1998). There is indication of reasonable reliability and validity (Lord et al. 1998). Our clinical experience suggests that the therapist needs time to learn this test, and that it is valuable to test inter-tester reliability between colleagues to ensure that they agree on how to evaluate the different items for scoring. In a test situation, the patient needs to be able to walk for 10 minutes, but may take shorter breaks.
- GAITRite is a 5-m-long mat connected to a portable software tool for automated measurement of different gait parameters that are registered when people walk on the mat, e.g., maximum gait velocity, step-length, single and double stance phases. It is recommended that the person walk at many different speeds (as slow as possible, a little faster, normal preferred speed, faster than normal, and as fast as possible) to gain a reliable picture of the patient's walking ability and to compare with changes over time. By interpolation, a point estimate can be calculated for each of the variables at a normalized speed representative for that subject. Thus comparisons between test occasions can be done without the confounding effect of walking speed (Moe-Nilssen 1998) Results from the GAITRite have shown strong test retest reliability and concurrent validity for healthy adults (Bilney et al. 2003).

Activity Measures

- The Postural Assessment Scale for Stroke Patients ([PASS] Benaim et al. 1999). PASS has been validated for stroke patients. It consists of two main domains: (1) maintaining a posture in sitting without support, sitting with support, standing without support, standing on paretic leg, and standing on nonparetic leg; and (2) changing posture through seven transfers including turning over in supine, supine to sitting, transfer between sitting, and standing and mobility in standing. These dif-
- ferent items are measured on a four-point scale (0–3). Nearly 40% of the assessed stroke patients in Benaim et al.'s study scored 36/36 on day 90 post stroke. It is therefore recommended that more difficult items are added after this date.
- Berg Balance Scale ([BBS] Berg et al. 1992, Finch 2002) consists of 14 standardized subtests scored on five-point scales (0–4), with maximum (best) score of 56 (Berg et al. 1992). Reliability and validity have been demonstrated in elderly people (Berg et al. 1989, 1992, 1995), and scores below 45 may indicate increased risk of falling for elderly people (Thorban and Newton 1996).
- Single Leg Stance ([SLS] Berglund 1999a) measures the patient's ability to stand on one leg without falling. It is measured in seconds. Normal values have not been decided, but the test seems to be relevant to demonstrate problems in activities where one-legged stance is necessary (stairs, turning around in standing). Many different ways of doing this test are described in the literature—it is not standardized (with or without shoes, eyes open or closed). Therefore, it is necessary to standardize it for the individual or the clinical setting in which it is being used.
- Functional Reach ([FR] Berglund 1999b). This is a balance test which seems to have strong correlation to ADLs. The person stands and reaches one arm forward at 90° forward flexion at the shoulder without changing his base of support. A mark is set on the wall at the tip of the patient's finger. He then reaches forward without falling. A new mark is placed and the difference between the two marks is measured in inches. The test should be repeated three times, and the median measure calculated. The measured distance demonstrates a relative risk of falling.
- Not willing to try: 28 times greater risk of falling
- 1–6 inches: four times greater risk of falling
- 6–10 inches: two times greater risk of falling
- >10 inches: The probability of falling is very low
- Timed Up and Go ([TUG] Podsiadlo and Richardson 1991, Finch 2002) Time is measured as the patient stands up from a standard arm chair, walks a distance of 3 m, turns, walks back to the chair and sits down again. Patients

who perform the test in less than 20 seconds are supposed to be independently mobile. The test has been found to be reliable and valid and responsive to change over time in elderly people.

- Six-minute walking test ([6MWT] Enright 2003) The test measures the distance covered when subjects are instructed to walk as quickly as they can for 6 minutes (Lord and Menz 2002). The patients should walk alone, not with other patients. While speaking to the patient, standardized phrases should be used to avoid the effect of encouragement and enthusiasm which can make a difference of up to 30% in the 6MWT (Enright 2003). In older people, the 6MWT appears to provide a measure of overall mobility and physical functioning rather than a specific measure of cardiovascular fitness (Lord and Menz 2002). The 6MWT is believed to better reflect ADL performance than other walk tests of shorter duration (Solway et al. 2001). The minimum clinically important difference (MCID) before compared with after for 6MWT was defined by Lacasse et al. (1996) to be 50 m and by Guyatt et al. (1987) as 30 m.

Self-Report Measures

- Borg's Rating Scale of Perceived Exertion ([RPE] Borg 1970, Finch 2002). Patients are asked to estimate the experience of exertion on a 15-point graded scale from 6 (no exertion) to 20 (maximal exertion) after exertion (e.g., 6MWT). According to Borg's range principle, a judgment of 50% of maximal exertion would have the same perceptual meaning for two people, even if it represented different absolute exercise intensity for each person (Buckworth and Dishman 2001). The scale values correlate well with exercise variables such as heart rate, ventilation, %VO₂ max and workload (ACRM 1988).
- The Visual Analogue Scale ([VAS] Kaasa and Lode 2002) may be used to measure, e.g., the patient's experienced gait problem or experienced ADL problems as well as experienced pain. The patients are asked to estimate their perceived amount of problem on a scale from 0 to 100 mm, where 0 is no problem and 100 is the worst imaginable problem.

Objective Goal Setting

- Objective activity goals that are specific, measurable, achievable, realistic, and time framed may be used in the clinical setting ([SMART] Monaghan et al. 2005). SMART is a valuable multidisciplinary tool that involves the patient and his carer actively. Used in a physiotherapy setting, the therapist chooses a short term goal together with the patient, following assessment and clinical reasoning. This goal should be task related and relevant to the patient's problems, resources and needs, and achievable in a few days. The therapist decides the prerequisites for the goal achievement: quality, environmental factors, relevance for daily activities, what kind of assistance, if needed. Preferably the patient should achieve the goal independently, but this may not always be possible.

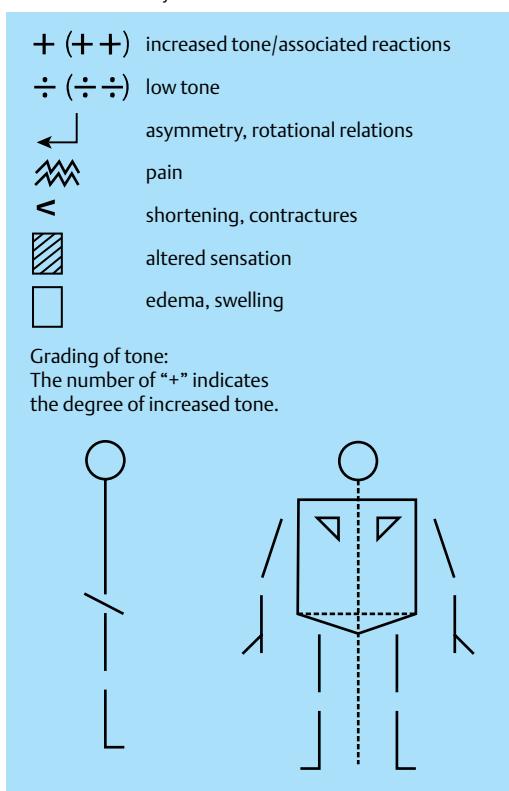
Assessment Diagram

In the clinical reasoning process, it might be helpful to draw a body diagram to illustrate the interaction between body segments, the distribution of tone, selectivity, and specific problems (pain, altered sensation, edema, muscular shortening). The diagram can help give a quick overview of the sensorimotor problems and assist the clinical reasoning process.

The diagram would **not** inform of causal relationships or about the patient's total situation, but sum up the findings of the assessment. It might be useful to make two or three illustrations: from the back, the front, and the side (Fig. 3.8). Associated reactions may be marked down on the diagram on the patient's more affected side in stroke. The symbols may demonstrate degree of compensatory increased activity or fixation. The patient compensates in areas of the body that he can voluntary control, therefore these may be have to be used bilaterally. It may be appropriate to illustrate the difference between voluntary activation and pathology with different colors.

There may be a gradual transition between associated reactions and compensatory evoked strategies, and therefore difficult to decide which is which. The number of "+" indicates the degree of increased tone in affected body parts:

Fig. 3.8 Example of a drawing which can be illustrated with symbols as follows:



- + Mild increased activity, therefore variable in their expression depending on the activity requirements—what the patient attempts or is asked to perform. At rest they are not present.
- ++ Moderate increased activity. Quickly apparent when balance or movement is required, beyond the patient's control. Beginning stereotypical patterning in associated reactions. The compensatory strategies vary with the activity to be performed.
- +++ Strong increased activity. The associated reactions and/or compensatory strategies are fairly stereotyped even if the patient is not very active.

The number of " \div " indicates the degree of reduced tone in affected body parts:

- \div Mild reduced tonus or stability
- $\div \div$ Moderate paresis
- $\div \div \div$ Paralysis, i.e., **no** tone or activity

The diagram gives a visual impression of the patient's sensorimotor problems, and does not reflect one position or situation. Free text may be added to highlight the patient's:

- Main problem (primary neurologic problem/negative signs)
- Compensatory strategies (secondary feature)
- Associated reactions/spasticity (secondary features/positive signs)
- Clinical reasoning

Evaluation and Documentation

The assessment must be documented and serves many purposes: to document the assessment and treatment given, for communication between professionals, to evaluate own practice, and information to the patient. Documentation needs to include:

- Assessment
- Clinical reasoning
- Goal setting
 - the patient's own goals (short term and long term)
 - multidisciplinary goals (short term and long term)
 - physiotherapy-specific goals
- Outcome measures, which should encompass measures in the different domains of the ICF (body structure and function and activity). It may not be possible to set more than very general goals for participation if the patient is in the hospital or rehabilitation unit.
- Treatment intervention, both physiotherapy specific and multidisciplinary
- Progression of treatment
- Evaluation, including test results of the chosen outcome measures
- Recommended further treatment or controls

Conclusion

The physiotherapist is the one professional in the multidisciplinary team who knows most about movement, and therefore is able to analyze specific movement in activities. Therefore physiotherapists have a special responsibility to focus not only on the patient's activity performance, but also on *how* the patient performs these activities, and *why* in this way. The hypo-

theses formed as a result of the assessment process are based on clinical reasoning, and view the patient in all ICF domains, although physiotherapy specifically aims at improving the patient's control of function, postural control, and movement. Clinical reasoning is the link between assessment, goal setting, and interventions and is a continuous process.

Summary

Evaluating the patient's potential is an important goal of assessment. See page 147.

WHAT? HOW? WHY? These are the three most important questions in the ongoing assessment of the patient. See page 157.

Choice of treatment follows a process of clinical reasoning. Observation and movement analysis in activities form the basis for hypotheses of which systems seem to be more affected and which ones seem more intact. See page 161.

Assessment and treatment are integrated in a continuous process. See page 163.

4 Case Histories

4.1 Case History: Sissel	169
4.2 Case History: Lisa	195

This chapter presents the case histories of two women, Sissel and Lisa, who represent different diagnostic and age groups. Sissel and Lisa are pseudonyms, but both women gave consent for their pictures to be published in this book. They were treated by colleagues under supervision of the author, later referred to as "I." The case studies focus on specific issues related to assessment and treatment guided by clinical reasoning. The histories aim to tell two stories with the use of text and pictures and will not make sense unless they are read as a whole. The case studies do not follow a specific treatment method, but illustrate the use of techniques based on conceptual thinking and clinical reasoning according to the Bobath concept.

In this chapter we follow Sissel over a period of 3 months. First we meet her in a stroke unit in a hospital, then in a short-term rehabilitation program in a nursing home before she is discharged and goes home. Chapter 4.2 follows Lisa over two treatment sessions during a 3-week treatment period in a hospital rehabilitation unit.

4.1 Case History: Sissel

Past Medical History, Social History, Activities, and Participation

Sissel is an 81-year-old widow. Before she was admitted to hospital with an acute stroke she

was fit, healthy, and active. She lived alone in a basement flat prior to her illness. She was self-sufficient and did not need assistance from the local community services.

Sissel's flat is all on one level, but she has to climb a flight of concrete steps to access her flat. Her children are grown up, and a daughter lives in the same neighborhood. Sissel is socially active, has friends, and is a member of a club for the elderly. Most of her friends, however, do not dare to visit her at her home because of the steep flight of stairs. Instead, she is a regular visitor in their homes. Her hobbies are knitting and crocheting—she can knit socks with proper heels and crochet the most delicate tablecloths.

History of Present Illness

One day Sissel was found in her home in a chair unable to move or make herself understood. She was taken to the local emergency department and immediately admitted to the stroke unit. She cannot recall anything about the first few days in hospital. On admission she was aphasic and there was no hyperreflexia and no voluntary activity on the right side except for slight movement in the fingers. A computed tomography scan showed infarction in the frontal lobe on the left side.

Sissel was referred for physiotherapy, occupational therapy, and speech therapy. Sissel's aim was to return home, preferably to a flat without stairs, and regain her independence. She also wished to be able to continue with her hobbies.



Fig. 4.1 Sissel 3 weeks after admission to the hospital.

Physiotherapy was started 5 days after she was admitted.

For ethical reasons, I did not want Sissel to feature in the book until she was medically stable, which was 3 weeks after she was admitted (Fig. 4.1). She understood that this would involve being photographed during therapy for future publication in a book. She was very positive.

Assessment

Functional Activity

Sissel was seated in a wheelchair and was not able to move about herself. She was nursed and needed help in transfers and dressing. She was able to feed herself when food was provided and she was continent, but she needed assistance in the bathroom.

She could sit unsupported on a plinth but could not move actively in sitting, nor was she

able to right herself. She showed some activity in her right arm; she could move it forward and had some selective finger movements, although none was useful functionally.

Language and Cognitive Function

Sissel could say "yes" and show, by facial expression, whether she meant "yes" or "no." She had a good understanding of words, but could not find the right ones to use. There did not seem to be any cognitive or perceptual deficits.

Body Functions and Structures

Observation and handling (Figs. 4.2–4.11).



Fig. 4.2 Sissel seated in a wheelchair. Her posture is slightly flexed and displays some overactivity or fixation in her left side, especially in her hand and foot. Her right arm rests in inward rotation, adduction, and flexion on a cushion in her lap. She does not move it actively in this position. The hand is swollen, and the wrist alignment is not clearly defined. On examination the hand is subluxed in a palmar direction in relation to the radius and ulna. Both legs seem slightly internally rotated.



Fig. 4.3 The footrests are being removed. Sissel is not able to transfer weight to the left leg when the right leg is lifted. She presses her left arm down on the armrest and clenches her fist. The right leg feels heavy and inactive except for some hold in inward rotation and adduction at the hip. She is not able to take part in the movement.



Fig. 4.4 Sissel cannot move forward in the chair. The physiotherapist facilitates her moving forward by a rhythmical weight transfer from hip to hip. There is no spontaneous righting over the pelvis or in the trunk, and she stays flexed in the head, neck, and trunk. She does not respond easily to facilitation and is difficult to move; there is a distinct resistance against weight transfer to the left when her right pelvis and hip is being moved forward in the chair. When the left hip is moved forward she seems to fall to the right. The alignment around the hips and pelvis is then adjusted to prepare for more activity during the next phase of the movement.



Fig. 4.5 Sissel cannot stand up unaided. There is a definite push down from her left leg, which forces the pelvis backward and stops her from being able to bring her center of mass over her feet. She therefore needs facilitation and handling for her upper trunk to move forward, achieve a slight anterior pelvic tilt, and thereby some thoracolumbar extension. At this moment she grabs hold of her right hand with the left, probably to include the right arm and shoulder girdle in the movement. This further increases the trunk flexion. Sissel is given some input and support to her right hip and knee because there is no spontaneous recruitment of motor activity in the leg. This preparation allows Sissel to be helped to a standing position.



Fig. 4.6a



Fig. 4.6b



Fig. 4.6c

Fig. 4.6a–c There is no activity in Sissel's right hip and knee in standing, and she needs full support. She pushes with her left leg. She holds on to the therapist with her left shoulder and arm. After a short time in standing she gradually adjusts and becomes more active in extension. The tendency to push with her left leg decreases and she is able to right her trunk more. Weight transference to the left was not possible at this time.



Fig. 4.7 Sitting on a plinth. Sissel holds the position through flexor activity of her head and neck, left trunk sideflexion, and flexion of the arms and hips. Hip adductor and inward rotator activity is more easily observed in this position. Sissel needs help to take off her jacket. She is not able to free her arms and falls backward and to the right when she attempts the activity by herself.



Fig. 4.8 Sissel is rotated forward and flexed on her left side (observe the clavicle which is more prominent on the left side). Her left arm and leg are active and fix on to her left thigh and push into the floor, respectively. The internal rotation in the right hip is more clearly visible: notice the alignment of the thigh/knee in relation to the floor.

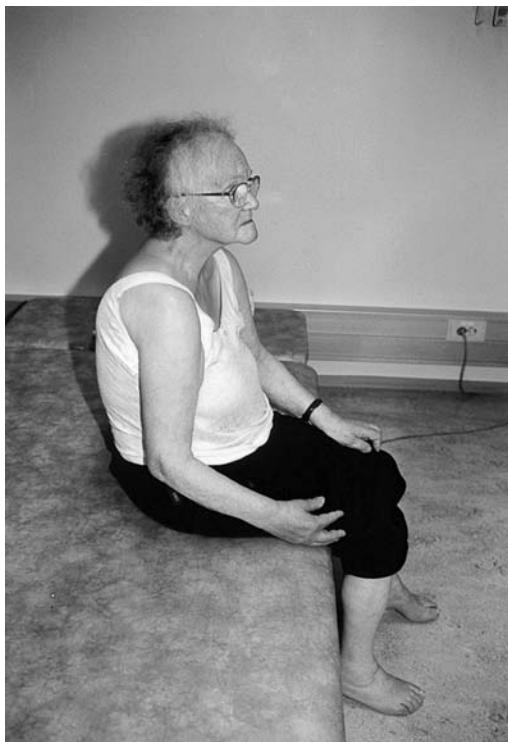


Fig. 4.9 Sissel seen from the right side: the tendency to fall to the right and backward is apparent in this figure. Her left foot pushes against the floor and brings her even further back. She attempts to compensate through forward rotation and flexion of her left side together with protraction of her right shoulder girdle and poking her head forward. Sissel's right arm is actively internally rotating, adducting, and displays increased flexor activity of her shoulder girdle, elbow, wrist, and hand. The right hand seems to press into her thigh.



Fig. 4.10 Sissel reaches for her handkerchief with her right hand. She opens her hand and moves her arm forward. Her forward reach is limited by extensor activation at the shoulder joint (possibly the action of proximal triceps, teres major, latissimus dorsi) combined with increased recruitment into elevation, protraction, flexion, and internal rotation of the right shoulder girdle. She does not right herself prior to the movement of the arm—she does not seem to recruit anticipatory postural adjustments. Without this adjustment, she would fall forward if she reached further, which may explain the extensor activation in her right shoulder. Even if Sissel moves her arm forward to some degree, her left trunk and shoulder girdle are still rotated forward in relation to the right side, fixing her position.

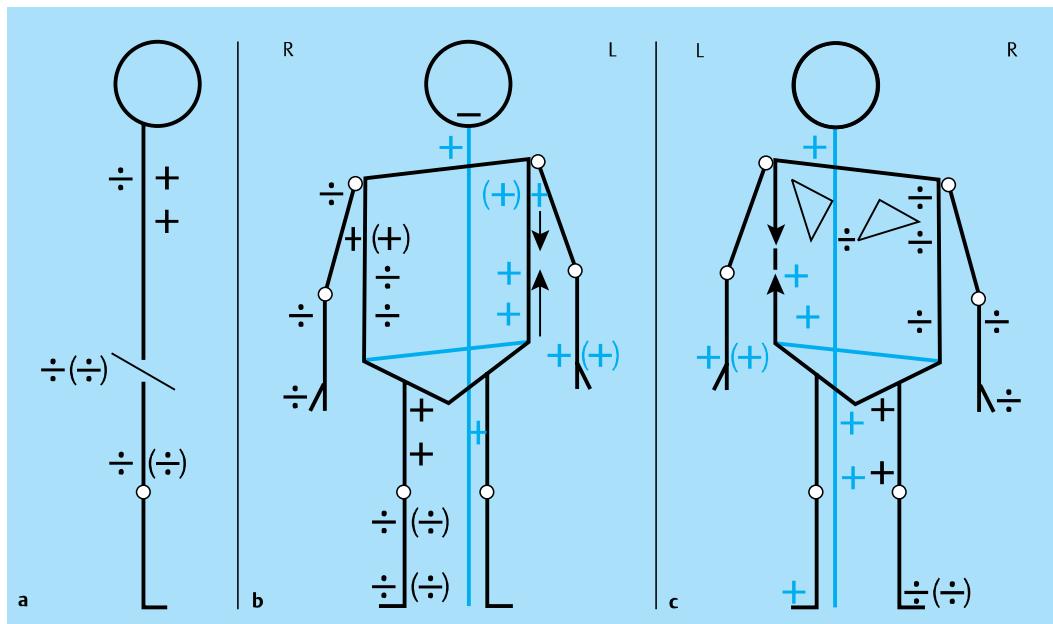


Fig. 4.11a–c Diagrammatic representation of the assessment. The right side (R) is her most affected side, the left side (L) her less affected side. As the illustration shows, on both sides the tonic relationships and movement strategies are different from what would be expected in a healthy person of the same age and sex.

(a) Sissel seen from her right side; “÷” demonstrates reduced extensor activity and “+” increased flexor activity in her head and neck and upper trunk. She has mild to moderate paresis of her hip and right leg.

(b) Sissel seen from the front. There is increased activity on her right side in adduction, internal rotation and flexion of both shoulder and hip combined with mild to

moderate paresis of extensors and abductors. Her left side displays a level of increased activity in general; a mild to moderate pressure from the shoulder in depression (arrow) at the same time as she pulls her weight on to her left side resulting in what would look like an elevated shoulder. The arrow above her left pelvis illustrates elevation of the pelvis, demonstrating an active shortening of her left side.

(c) Sissel seen from behind. The illustration shows the rotatory components of her shoulder girdles, her active left side, and the paresis on her right side as well as increased activation of both hip adductors and medial hamstrings.

Clinical Reasoning and Hypothesis

Systems Control

1. Sissel displays some voluntary activity in the fingers of her right hand and arm. This indicates that the corticospinal system is partly intact, and the potential for further recovery of selectivity of her right arm and leg should be good.
2. Sissel's balance is severely affected, but she seems to have intact sensation and vision.

She can perceive that she will fall and attempts to regain her balance. This suggests that her basic balance mechanism which is based on visual, vestibular, and somatosensory information, is intact. However, the specific receptors in her skin, muscles, tendons, and joints are not being optimally stimulated due to low tone and reduced motor control on her right side. This will influence the function of the vestibular system indirectly, and may contribute to Sissel's balance problem.

3. The musculature of her right side is generally hypotonic, mostly in her trunk, pelvis, and right leg. Her proximal stability is therefore reduced. This indicates a lesion affecting her cortico-reticulospinal and cortico-rubrospinal pathways.
4. The timing of recruitment for postural control seems to be disturbed: she does not recruit anticipatory postural adjustments as a background for voluntary activation of her arm; she does not right herself for forward reach.
5. The problems in points 2–4 severely affect her postural control, especially her postural stability as a foundation for recruiting activity in relation to opposing gravity, for weight transference and selective function. The interplay between body segments is severely reduced, and therefore also her balance and ability to transfer and move actively and independently.
6. Sissel does not seem to have perceptual or cognitive dysfunction apart from aphasia; she is attentive to herself and her environment, and is motivated for training.

Compensatory Strategies

When Sissel is assisted to lift her right foot off the footrest, she tries to help (Fig. 4.3); she presses her left arm down on the armrest of her wheelchair, possibly in an attempt to recruit her left side for contralateral stability in order to free her right leg for movement. This strategy is inappropriate as the fixation through her left side limits weight transference to the left. She is therefore unable to move her right leg freely.

Sissel has reduced activation and stability in her right hip and pelvic area. As she attempts to move forward in the chair and transfer her weight to the right to move her left pelvis forward, she almost falls to the right and backward. She compensates by pulling herself further to the left through increasing flexion of her trunk and left arm (Fig. 4.4).

As Sissel stands up, she does not initiate righting activity between the body segments (Fig. 4.5). She keeps her hands together, pulls herself up using the therapist and thereby increases her flexor activity generally. Her balance is threatened as the interplay between the proximal and central body segments is reduced together with an inactive right leg. Sissel's strategy is therefore to fix

on to the therapist, pull herself up through flexor activity, and push with her left leg in an attempt to recruit antigravity activity. To stop herself from falling in sitting, Sissel increases the muscular activation throughout her left side to compensate for the instability of her right side (Fig. 4.7–4.10). She displays shortening through flexion on her left side and elongation of her right side, which makes it more difficult for Sissel to activate her right side (biomechanical disadvantage).

Lack of anticipatory postural adjustments and sustained flexor activity between the central and the proximal body segments in all planes result in a decreased ability to reach forward for the handkerchief (Fig. 4.11).

Hypothesis

Sissel's main problem is reduced postural control through inappropriately timed activation of postural activity, decreased proximal stability, decreased tone and reduced activation of her right side. She therefore increases her activation of her less affected left side to oppose her tendency to fall: she flexes her trunk and sideflexes to the left, fixes with her left arm and pushes with her left leg.

This increases the imbalance between her right and left side and causes malalignment in all three planes. As a result, the chances of recruiting activity on her right is increasingly disturbed and causes a further loss of postural control. This causes reduced postural control and trunk interplay. Therefore, the postural background for stability, weight transference, balance, and movement is reduced.

Physiotherapy and Clinical Reasoning

On the basis of the above hypothesis, it should not be necessary to specifically train selective motor control of the extremities. Postural control is the basis for voluntary activity and improved postural control in Sissel's case should allow recovery of the selectivity Sissel already displays (active movement of her right hand and fingers). However, it is necessary to maintain muscle length, alignment and range of movement in both shoulders and hips. As Sissel compensates by activating flexors in these areas, her pectora-

lis, hip flexors, and adductors are kept in an active, shortened position. If the assumption that the cortico-reticulospinal and cortico-rubrospinal systems are the ones most affected, she may be in danger of developing spasticity and secondary non-neural problems. The shortened muscles will be exposed to these changes the most. If flexors shorten and lose their eccentric ability, other muscle groups (e.g., the extensors) will be biomechanically disadvantaged. As a result, stability, interplay, and weight transference are further compromised.

Aims of Therapy

Main Aims

- Participation: Sissel is to return to her own flat or a flat with adaptations. An application for a council flat has been made.
- Participation and activity: Sissel shall recover independence; possibly with some help for shopping.

Activity and Body Functions and Structures: Improved Balance

- Activity: independent walking ability
- Activity: independently perform activities of daily living (ADLs)

Short-term Goals

- Improved postural control (body functions and structures)
- Selective and functional extremities (body functions and structures)

Interventions

Body Functions and Structures—Activity

- Improve alignment in and between body segments, especially head, neck, and trunk (make possible, see Chapter 2):
 - improve the relationship and adaptation to the base of support
 - improve thoracic extension and extension, abduction, and outward rotation in shoulder girdles and hips
- Facilitate the interplay between body segments based on improved extensor/rotatory control
- Facilitate weight transference and stability over left side to free the right side for balance, movement, and transfers

- Weight transference in all planes in sitting, standing, and standing to sitting
- Explore the recovery of arm function based on improved postural control

Physiotherapy: Assessment and Treatment as a Continuous Process

Sissel received physiotherapy daily, and pictures are taken at intervals of 4–7 days. Therefore, the pictures only give an overview of the physiotherapy Sissel is receiving. Unfortunately the photographs were not standardized, which makes comparison between pictures difficult.

First Photo Session (Figs. 4.12–4.17)

However, Sissel's shoulder girdles are still active in flexion and therefore need to be brought down and back to facilitate stability of the scapulae against the thorax and thereby the interplay between the posterior and anterior trunk musculature for postural activity. By doing this, the interplay between trunk and arm function is facilitated.

In this situation, antigravity activity with an emphasis on extension and weight transference through rotation is enhanced. This should facilitate contralateral stability on the left side for activity in the right leg in preparation for stepping.

Weight transference is different from weight shift.

Weight transference is a dynamic activity whereby the center of gravity is moved to the weightbearing side through stability and interplay. Weight shift is a more passive shift of weight without moving the center of gravity. It may cause strain on joints due to low muscle activation. Weight shift does not activate postural stability, and therefore Sissel would be unable to take over movement control.

Walking is an activity, which in its simplest form seems to be driven from the pattern generators at spinal cord level. Independent walking requires postural control, stability, and interaction between body segments. Control of extension and rotation are core elements of this inter-

action. If pattern generation can be facilitated, it may improve Sissel's control of balance and movement. The prerequisites for this to happen are optimal alignment, facilitation of the sequence of recruitment of muscle activity, heel-strike, and hip extension. Sissel therefore needs to be facilitated simultaneously in two areas:



Fig. 4.12a

her trunk to enhance postural stability (rotation and alignment of the trunk in relation to the lower extremities) and her leg for heel-strike, hip extension, and phase shifts from stance to swing. The timing of facilitation between the two facilitating people is a challenge.



Fig. 4.12b

Fig. 4.12a, b The alignment of the hips in relation to the pelvis is corrected by giving eccentric length to the internal rotators and adductors. Sissel is helped to right her trunk through the therapist's handling of the pelvis in sitting. Sissel's hands are in her lap.

There are some compensatory activities of the arms into adduction, flexion, and internal rotation of shoulders and arms. This limits her ability to recruit trunk righting. Therefore, another postural set is chosen to develop her extensor activity.



Fig. 4.13a



Fig. 4.13b

Fig. 4.13a, b Standing is a postural set that may enhance postural tone, extension, and postural control, if the alignment allows. Sissel is helped to stand through handling of the pelvis and hips combined with knee support. The shoulder girdles and the arms, the lower arm, and the hand are mobilized and facilitated to allow extension. The alignment improves, and Sissel is able to stabilize her arms on a high support behind. The arms are placed in external rotation to enhance extension of the arms and upper trunk.



Fig. 4.14

Fig. 4.14 Sissel attempts to lift her right leg forward by herself. The weight transference to the left is not good enough. There is too much flexion on her left side, which negates the trunk interplay and contralateral stability required for freeing her right leg through selective movement.



Fig. 4.15 The alignment is corrected: more extension in her trunk and over the left side to facilitate weight transference through rotation. Sissel still has some flexion in her left side; note the flexion of her left arm in a compensatory attempt to fix. Sissel is not able to recruit appropriate amounts of extension over her right hip and knee, and requires some support.



Fig. 4.16a, b Her right leg is facilitated in swing to stance. Sissel's trunk, pelvis, and hip are brought over her right leg. The hip/pelvis is stabilized and the knee supported without hyper-extension. After a few steps Sissel stands better and more actively, and the activation of her right leg is stronger.





Fig. 4.17a



Fig. 4.17b



Fig. 4.17c

Fig. 4.17a–c Sissel sitting after treatment. She rights herself more easily and demonstrates less flexor tendency. The alignment of her legs to the bases of support (plinth/pillow in chair and floor/ foot rests) show less adductor and internal rotator components (compare with Figs. 4.7–4.9), which provide an improved basis for core stability and righting. Sissel has been provided with a table on her wheelchair instead of pillows in her lap. This may help her develop her selectivity and function of her arms and hands based on improved righting.

**■ Second Photo Session, 4 Days Later
(Figs. 4.18–4.24)**

Fig. 4.18 Sissel sits asymmetrically and rotated in her chair. Her left pelvis/hip area seem to be rotated back and tilted down, with flexion, internal rotation and abduction, of her left hip. Her left shoulder seems to be elevated and rotated forward. Sissel's right pelvic area seems elevated and her right shoulder depressed. The right hip seems to be more internally rotated than her left. Sissel's right arm is in a position of depression, internal rotation, and flexion. The general impression is that Sissel has enhanced flexor and internal rotator activity in her right side. Therefore, she may find it difficult to initiate and recruit trunk interplay, and activate her legs to come forward and stand up.

Sissel's position and neuromuscular activity need to be corrected to improved alignment to facilitate standing up: her pelvic alignment is corrected to gain an even weight distribution and adaptation to the base of support. The flexor, adductor, and internal rotator components of Sissel's hips are mobilized to enable her to recruit extensor activity and stability when rising. Selective trunk extension is facilitated to enhance interaction within her trunk based on improved stability of pelvis and hips.



Fig. 4.19 Sissel needs less facilitation than before. She still recruits flexor activity of her upper trunk, especially through her arms, head, and neck. She needs some support to her right knee. In standing, she is more stable and the therapist can take her hands off as Sissel takes over her own control. Sissel's hip extension is much improved.





Fig. 4.20 Sissel is more stable in sitting due to improved activation of extension and abduction of pelvis and hips. She adapts more appropriately to the base of support, and is able to move without losing her balance. Sissel is therefore able to use and develop the selective control of her arms and hands and undress her upper body unaided; compare with Figures 4.7 and 4.8 and note the alignment of the thighs on the plinth. Her hand function is very much improved, as is her postural control over wrist and hand to allow selectivity of the fingers.



Fig. 4.21 Compare with Figures 4.7 and 4.8. Sissel's trunk, pelvis, and lower extremities are more optimally aligned, enabling her to recruit a neuromuscular activity that gives her stability.

However, her right shoulder is flexed, internally rotated, abducted, and elevated, probably due to activation of the pectoralis major and upper trapezius. Note how her top is pulled in under her right arm and the backward rotation of her right ribs.

During therapy, the eccentric length of the musculature of her right arm needs to be maintained to enable stability and selective movement for function. Increased activation of trapezius, pectoralis and biceps destabilize the scapula against the thorax, and Sissel will not be able to free her arm for a variety of movement. Short pectoralis and trapezius negate full thoracic extension and a free arm, which will have consequences for stability and movement. (There are no pictures of this intervention.)



Fig. 4.22a



Fig. 4.22b



Fig. 4.22c



Fig. 4.22d

Fig. 4.22a-d Postural control in standing depends on, among other components, the adaptation of the feet to the base of support. The feet need to be both mobile and stable to vary their activation appropriately in relation to changes in the projection of the center of gravity.

Sissel's right foot is swollen and inverted more than the left. Her lower leg seems to be outwardly rotated, her heel is drawn medially and her right big toe is more adducted than the left. The foot is stiffer and less mobile than needed to provide a dynamic foundation for balance in standing and walking. The musculature of the lower leg and foot needs to be handled to improve alignment and adaptation. The forefoot is stabilized at the same time as the soleus and gastrocnemius are mobilized and facilitated to give eccentric length. The heel is moved in different directions in relation to the forefoot.



Fig. 4.23a



Fig. 4.23b

Fig. 4.23a, b Improved adaptation of the foot to the floor enhances mobility and stability of hips and pelvis and Sissel is helped to stand. She needs less support to hip and knee. The plinth is raised to hip level, and Sissel stands with her back to it. This gives Sissel a more secure feeling, which allows her to explore, experiment, and “play” with her own movement control.

Sissel transfers weight in all directions. The weight transference and interplay between key areas are not optimal; there is too much flexion of Sissel’s upper trunk and left side. The alignment of the right leg to her pelvis is improved, which enhances the activation of her left quadriceps.



Fig. 4.24a



Fig. 4.24b



Fig. 4.24c

Fig. 4.24a–c Facilitation of steps between two people. The therapist on Sissel's left side facilitates extension for stance phase, especially hip extension. Sissel compensates using her left arm in flexion, although less than before. It is easier to facilitate more thoracic extension and extension at the pelvis and hip, which therefore frees the right leg for swing. The therapist on Sissel's right gives length to her pectoralis major to facilitate trunk extension at the same time as she provides stability to Sissel's thorax through the back of her hand and facilitates the foot forward on the floor (**a**).

In stance over the right side, the therapist facilitates extension of the hip and pelvis (**b**). Sissel's foot adapts well to the floor, and the alignment is appropriate. She therefore automatically recruits knee extension and stabilize(s) herself during the swing phase of the left leg. The compensation of her left arm and hand reduces as Sissel gains rhythmical steps and she is able to look up (**c**). As the stepping progresses, Sissel starts to initiate dorsiflexion and toe extension in the swing phase on her right side. Alignment is optimized through the movement, and the timing of input from the therapists needs to be precise.

■ Third Photo Session, 4 Days Later, Approximately 1 Month Post-stroke (Figs. 4.25–4.33)

Fig. 2.25a–c Sissel sits more actively and symmetrically in her chair. She removes the table from her chair and brings it toward her right side to enhance eccentric control of pectoralis, biceps, and trapezius and activates extension of her thorax, shoulder girdle, and arm at the same time. Her thighs fall into internal rotation due to the softness of the seating cushion more than being drawn together by active adductors and internal rotators; Sissel stabilizes her right leg as she moves forward to remove the foot rest (**b**). Sissel is not completely stable over her right side. As she leans forward to remove the foot rest she stabilizes her right hip initially. As she leans further forward, she feels the need to compensate using her left arm on her right thigh, and the flexor, internal rotator, adductor activity increases to some degree. She would benefit from facilitation to her hip and thigh to ensure the maintenance of hip stability as the movement progresses forward. Sissel problem-solves how to remove the wheelchair table, finds the release mechanism for the foot plates, adjusts her grip, swings it to one side and removes it. Through this, Sissel demonstrates the ability for awareness, concentration and problem solving, and somatosensory perception and integration.



Fig. 4.25a



Fig. 4.25b



Fig. 4.25c



Fig. 4.26a



Fig. 4.26b

Fig. 4.26a, b Sissel is now able to undress the top half of her body independently and unsupported on a plinth. She maintains her balance as she pulls the jumper over her head, indicating that her balance is becoming more automatic. This activity requires stability and interplay. She sits slightly rotated but demonstrates increasing trunk control. The selectivity of finger movements is gradually improving.



Fig. 4.27 There is increased flexor tendency through her left side, which negates lateral pelvic tilt and makes weight transference to the left problematic. Weight transference in sitting requires eccentric lengthening of the adductor and internal rotators in the hip opposite the weightbearing side normally. Sissel's right hip is pulled into internal rotation and adduction due to reduced selectivity and stability. The flexor tendency is therefore maintained. By placing Sissel's left arm in abduction on a high plinth, trunk extension and weight transference to the left are facilitated.



Fig. 4.28



Fig. 4.29



Fig. 4.30

Fig. 4.28 Sissel's right leg is crossed over the left. This facilitates weight transference to the left and Sissel has to stabilize her right leg at the same time. Both arms and hands move across the plinth. This rotation improves the interplay and activity of the trunk and pelvis, and Sissel transfers her weight further over her left pelvis and hip as a preparation for dynamic sitting.

Fig. 4.29 Sissel lifts her left arm up into the air. The weight transference and extension through her left side is facilitated, which cause reciprocal activation of her right side. The right arm is incorporated to demand more stabilizing action as she uses it as a reference for movement and not support.

Fig. 4.30 Sissel is facilitated through her trunk over her left side into standing to progress trunk extensor activation on the left. She recruits enough stability in her right hip, knee, and foot to allow this activity. She no longer needs support of her right knee. The left arm is moved into abduction to enhance trunk extension. She displays some flexor activity of her left shoulder girdle and tends to fix in this area. However, she does not press down on the plinth.



Fig. 4.31



Fig. 4.32



Fig. 4.33

Fig. 4.31 Sissel maintains her weight transference to the left and waves with both arms. This demonstrates improved trunk interplay and postural control.

Fig. 4.32 Sissel's right arm is placed on the plinth to her right. This frees trunk movement through both arms being in abduction without downward pressure. She is therefore able to maintain her stability over her left side as swing phase is initiated through eccentric activity of extensors, abductors, and external rotators in the right hip and pelvic area.

Fig. 4.33 Sissel has problems in the next stage of swing phase because the selectivity of hip flexion is reduced. To ensure that she does not lose her stability through compensation, her right foot is facilitated forward.

■ **Fourth Photo Session, 1 Week Later
and the Last Treatment Session
before Sissel is Discharged
(Figs. 4.34–4.41)**

Fig. 4.34 Sissel sits more symmetrically and seems to adapt appropriately to the base of support—the plinth. The right side of her thorax is slightly rotated backward, her left shoulder girdle is rotated forward with a slightly flexed trunk, although less than before. Compare with Figures 4.8 and 4.9.



Fig. 4.35 Sissel's right shoulder is treated to gain more eccentric lengthening in the pectoralis major, trapezius, and biceps before the arms are placed behind Sissel on the plinth (compare with Fig. 4.13b). In this way, the arms are placed in a natural position and situation, and trunk extension is facilitated while Sissel's right foot is prepared for better interaction with the floor.





Fig. 4.36



Fig. 4.37



Fig. 4.38

Fig. 4.36 Sissel has reduced dynamic control over her left hip and pelvis due to her compensatory flexor strategies. These strategies prevent her from regaining optimal stability over her left side during transfers and walking. A stable left side would free her right side for movement. Sissel is helped to stand on one leg (left) to enhance dynamic activation of trunk, hip, and pelvis on her left side.

Fig. 4.37 Sissel lifts her left arm up and out and moves the therapist's left leg with her right foot at the same time. She then has to stabilize over her left hip as she moves the right leg without fixing through her arms. This is very demanding, and she develops an associated reaction in flexion of her right elbow.

Fig. 4.38 Sissel is asked to maintain the stability of her left hip as she lowers her right foot in a controlled manner down to the floor. The associated reactions ease off because she is able to recruit dynamic activity in optimal alignment in her left side.



Fig. 4.39



Fig. 4.40



Fig. 4.41

Fig. 4.39 Facilitation of stepping. Sissel is now able to take more steps together for walking. She is facilitated through external rotation of both shoulders and slight timed compression of her rib cage. She controls the rest herself; stability, weight transference, and movement of the legs in stance and swing. She no longer needs help to swing the right leg forward into heel strike, unless the tempo is too low to facilitate her pattern generators.

Fig. 4.40 Sissel stands on her own. She is still unable to walk unaided, but she has the potential for independent walking within a relatively short time span.

Fig. 4.41 Sissel is happy with her treatment and looks forward to continuing training closer to home; she will attend the local rehabilitation unit.

Evaluation at Discharge

Sissel developed a deep vein thrombosis of her right lower leg the day before her planned discharge. She therefore stayed a few more days in the department. There were no restrictions for physiotherapy, and Sissel started using a compression stocking.

Functional Activity

Sissel is independent in all ADLs. She uses a manual wheelchair for transport, transfers herself, and manages the bathroom herself. She walks with slight support and is safe in this situation. A walking aid has been discussed, and deemed as not relevant at this time; Sissel's postural control is still not optimal, and a walking aid might increase her flexor tendency and fixation strategies and thereby negate interplay between body segments. This interplay needs to be strengthened to allow Sissel to develop good balance. If

this happens, then Sissel will not need a walking aid in the near future either.

Sissel has started knitting using rather large diameter knitting needles and coarse thread. Her finger and hand dexterity is slightly reduced. Sissel's aphasia is much reduced; she has only slight problems in finding the correct words sometimes. She is discharged to a local rehabilitation unit.

With good communication of Sissel's needs to the local physiotherapist, she continues the interventions that have been started in the hospital. The local council is looking for a more appropriate flat for Sissel without steep, concrete stairs. Sissel stays at the unit for approximately 2 months, before she moves in with her daughter while waiting for her new flat.

Approximately a year after Sissel had her stroke, she is living in her own new flat. Her goals have been attained. It was a privilege to be involved in Sissel's training and to include her case study in this book.

Thank you, Sissel.



Fig. 4.42



Fig. 4.43

Figs. 4.42 and 4.43 We visited Sissel several times in the rehabilitation unit and in her daughter's flat. Our last visit was 6 months after Sissel had had her stroke. She is now totally independent, and she has started cro-

chetting the most beautiful and fine tablecloths again. Although she walks without walking aids, she feels that her balance is not as good as before. She therefore uses her wheelchair for longer distances outside the home.

4.2 Case History: Lisa

Social History, Activity, and Participation

Lisa is a 32-year-old woman with cerebral palsy (Fig. 4.44). She is a qualified social worker and works part-time from her own office at home. She is unmarried, and lives in the garden flat of her parents' house. The flat has been adapted for wheelchair use. Lisa has a large social network and is active in organisational work. She is a member of the public transport services for wheelchair users, but the capacity of these services does not allow her to be independent. Her parents assist her when required, providing transport for example. She has her own car, which her parents drive. Lisa has a home help twice a week, and a community nurse once a day for personal assistance. She prepares her own food on most days.

Until 7 years ago, Lisa could walk short distances using two elbow crutches. Now Lisa can walk a few steps with a rollator, but is functionally wheelchair dependent in daily life. Generally, Lisa is independent in most activities.

Medical History

As Lisa has cerebral palsy, she spent a lot of her time in contact with the health services until the age of 16. She has had several operations: lengthening the Achilles tendon at the age of 4–5, and finally arthrodesis of both ankles. All in all, she has been through eight operations between the age of 4 and 16. She has contractures of her hips, knees, and ankles as well as deformed feet. She probably has growth retardation of the lower half of her body.

Lisa has a lot of pain in her lower back. She has had chronic tendonitis and pain in both shoulders since 1992. Lisa has visual coordination problems.



Fig. 4.44 Lisa

Previous Training Experience and Treatment

- Lisa has been to a training center in the Norwegian mountains at Beitostølen a few times; the last time was in 1992. She did swimming training and riding, and enjoyed this very much.
- Riding from the age of 8 to 16
- Treatment by a chiropractor for her back pain: stretching of her back and hips. Short-lasting effect
- Physiotherapy: training and ultrasound for her shoulder pain. Short-lasting effect
- Acupuncture. Short-lasting effect

Current Problems

Lisa feels that she is gradually deteriorating; she feels weaker, her balance and coordination are reduced, and she does not trust her legs anymore. She has therefore applied for a period of inpatient training.

Lisa's Goals

Lisa wishes to regain her previous level of function and control of movement. She has great expectations and is very motivated. She is admitted for 3 weeks' training and receives physiotherapy daily for 5 days a week. She was asked if she would like her case study to be included in this book and she gave written consent. The following photographs were taken during a few of Lisa's treatment sessions.



Fig. 4.45a

Assessment

Functional Activity

Lisa is independent in most daily activities except for putting on socks and shoes. She prefers to dress and undress sitting in a chair, because her balance is threatened if she sits freely on the bed or plinth. During undressing, Lisa displays some involuntary movement, generalized reduced coordination, intention tremor, and reduced dexterity and distal movement control.

Body Functions and Structures

Observation and handling (Figs. 4.45–4.53).



Fig. 4.45b

Fig. 4.45a, b Lisa has well-developed musculature in her upper body. She has a functional strategy for taking off her trousers in sitting: she pushes away through her feet, tilts her pelvis, and pushes into and away

from the backrest of her wheelchair. In this way, her pelvis is lifted off the seat and she is able to take off her trousers.



Fig. 4.46

Fig. 4.46 Lisa has moderate involuntary movements, and dressing in free sitting is therefore complicated. She has reduced ability to adapt to the base of support. Even on sitting on the corner of the plinth to increase the size and width of the support surface, she is not able to recruit enough stability to undress her upper body. Sudden involuntary movements destabilize her and seem to increase the tone in her extremities. There may be some degree of spasticity present, as well as severe non-neural changes.



Fig. 4.47a

Fig. 4.47a, b Lisa attempts to use her crutches for the first time in a very long time. She shifts forward in her chair. She is very asymmetric: her head is drawn toward the right side, her right side flexes and rotates forward while her left side retracts. She seems to pull herself forward over her right side by her right arm and push away through her left arm. Lisa's knees press together and her hips adduct as she draws herself into standing. The movement of her pelvis is restricted due to severe hip adductor and flexor activity. She has very limited ability to grade knee movement, and therefore stiffens her legs to ensure that they will carry her weight. Lisa is unable to get to standing unaided, and the physiotherapist helps her bring her weight over her feet and to balance.



Fig. 4.47b



Fig. 4.48a



Fig. 4.48b



Fig. 4.48c

Fig. 4.48a–c Lisa's knees are pressed hard against each other causing a white pressure mark on the inside of her right knee. Lisa manages only two steps. She has all her weight on her arms, and her legs do not carry her. The pressure of her arms on her crutches activates her latissimus dorsi and thereby increases her lumbar lordosis. Her pelvis tilts anteriorly, and increases the flexor, adductor and inward rotator activity of her hips, left more than right. She rotates her pelvis back during swing phase on the same side. This backward rotation causes increased activation of adductor activity of the swinging leg, which she uses to bring the leg past the standing leg. She has no stability over her hips and pelvis. The activity in her neck and back muscles is strong. She looks down to see where she places her feet and keeps an eye on the near environment. She is not able to follow what goes on in the rest of the room.



Fig. 4.49 Lisa's lower legs seem outwardly rotated in relation to her thighs and her feet outwardly rotated in relation to her lower legs.



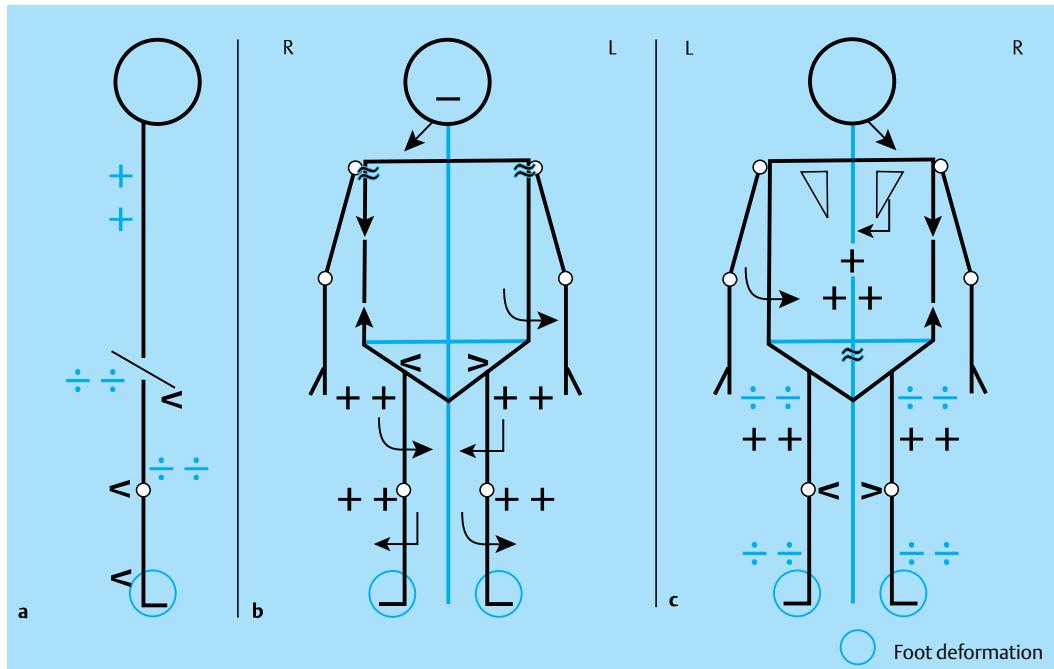
Fig. 4.50 Lisa sits asymmetrically with sideflexion on her right and the pelvis tilted down on her left. The left side of her pelvis seem rotated backward, and the left hip more adducted, internally rotated, and flexed as compared with her right hip. Both arms are heavy as the therapist helps to move them upward.



Fig. 4.51 Lisa's pelvis is tilted anteriorly. Her back is straight and there is significant activity in her neck and throat musculature. She moves her head very little.



Fig. 4.52 Lisa's right shoulder is depressed. She is sideflexed on her right compared to her left side. The middle part of her trunk seems to be rotated back on her right while her right pelvis is rotated forward. The left side of her pelvis, trunk, and shoulder are rotated backward.



4.53a–c Diagrammatic illustration of the assessment. (a) Lisa has increased activation of her back extensors, possibly as a compensation for flexor contractures ($<$) and increased tone of hip flexors. There is significantly reduced activation of the gluteal and quadriceps musculature, and contractures of her knees, ankles, and feet. (b) Lisa has pain (\approx) in both shoulders, her right shoulder is depressed and her head side flexed to her right. She is rotated back (\rightarrow) on the left side of her lower trunk. The legs show rotation as described in

the text, as well as increased tone and contractures of hip adduction and inward rotation, with reduced activation of quadriceps.

(c) Lisa seen from behind. Her head is pulled to the right, and the right side flexed. The right scapula is rotated down, and the left lower trunk rotated back. There is increased lumbar lordosis, and Lisa reports pain in this area. The pelvic musculature is inactive and atrophied. Both knees are contracted and there is increased activation of the adductor musculature.

Clinical Reasoning and Hypotheses

Systems Control

Lisa seems dependent on visual input to maintain some balance, both in sitting and standing/stepping. When Lisa takes off her T-shirt in sitting, she almost falls. The imbalance occurs as she pulls her T-shirt over her head, thereby obliterating visual information. As visual references are lost she loses her orientation to her environment. The observation of imbalance during unsupported undressing combined with her fixed head position (probably to stabilize vision) leads to the hypothesis that her balance largely

depends on visual more than somatosensory inputs. She seems unable to anticipate the displacements and adjust her postural control in preparation for the activity. She has reduced control and activity of the muscles surrounding her pelvis, hips, thighs, and legs. This musculature is relatively inflexible and less sensitive and adaptable to changes. Even if her superficial sensation is good, there will be reduced information from muscle and tendon organs due to muscular inactivity. She may therefore have reduced ability to perceive small changes and displacements and to adjust her activity in relation to the base of support, and to the ongoing demands for stability.

Lisa's central nervous system (CNS) weights the information through her visual systems more than somatosensory information, thereby shifting to a more cognitive control of balance. This causes reduction of anticipatory postural adjustments. She has reduced distal dexterity; the corticospinal system may be affected.

Lisa has significantly reduced stability and movement control of her pelvis, hips, and legs, also some trunk involvement. She displays sudden involuntary movements and reduced coordination. Possibly, these involuntary movements are reinforced by the reduction of stability and balance. Balance and movement is the result of interaction of many systems in relation to the goal (task) and the environment. Lisa was born with cerebral palsy and her CNS has therefore developed differently from a person with an undamaged CNS. Through plasticity, she has developed sensorimotor control based on her available resources and the interaction with the environment—to enable her to function to the best level possible. The sensorimotor (dys)functions she displays are a result of this interaction (form–function). It is therefore not possible to state specifically which systems are more or less affected than others.

Lisa's pelvis and legs seem growth retarded. She has never walked to any degree, and has reduced muscular activity and control. There are obvious non-neural changes in relation to musculature and connective tissues with muscular atrophy, contractures, malalignments, and reduced recruitment. She may have some spasticity in her legs (hyperreflexia). Recruitment of the muscular activity necessary to sit, rise to standing, stand, and walk is reduced (extensors and abductors among others).

Compensatory Strategies

Lisa compensates for reduced walking ability and the pain in her shoulders and back by using a wheelchair (activity dimension). She compensates for reduced balance and stability by using her arms and upper body in all transfers. Reduced activity and control of her legs increases the adductor, flexor and inward rotator activity in both hips to 'stiffen' the legs during transfer to and from the wheelchair (body structures and functions).

Clinical Reasoning Related to Development Over Time: A Reduction in Functional Ability (Figs. 4.54 and 4.55)

Lisa was previously able to move better than she does today. She does not have a progressive disorder, although her physical ability is progressively worsening. Through life we adapt to what we do and what we do not do, physically and mentally. Lisa's shoulder problems may be her main problem. The pain in her shoulders causes her to sit more and use a rollator instead of crutches. The use of a rollator and increased time spent in sitting may contribute to increased compensatory flexion through her arms and her pelvis and hips, and increasingly deactivate the control of her legs that she did have at one time. This may have resulted in a vicious circle of events. Lisa has good abdominal musculature and control, but as her pelvis is tilted anteriorly it prevents her from using these in an interplay with trunk extensors for core stability.

Improvement in hip and pelvic movement based on more eccentric length in her adductor, flexor, and inward rotator musculature will improve the range of movement at the hips, may improve the flexibility of the musculature and soft tissues and thereby enable Lisa to gain a better interplay between body segments and between the body and the environment/base of support.

Improved length and flexibility of the hips may enable Lisa to improve her control of extensors and abductors and thereby balance. She may then experience a reduction in low back pain because of improved alignment and muscular support. Improved stability and balance might improve her shoulder pain because she would not need to support herself as heavily during transfers.

Main Subjective Problems

Lisa's subjective main problems (with a reference to her previous functional ability):

- Progressively reduced balance
- Reduced ability to walk
- Pain in lower back and both shoulders

Goals

- Improved balance during functional activity
- Ease of pain in back and shoulders

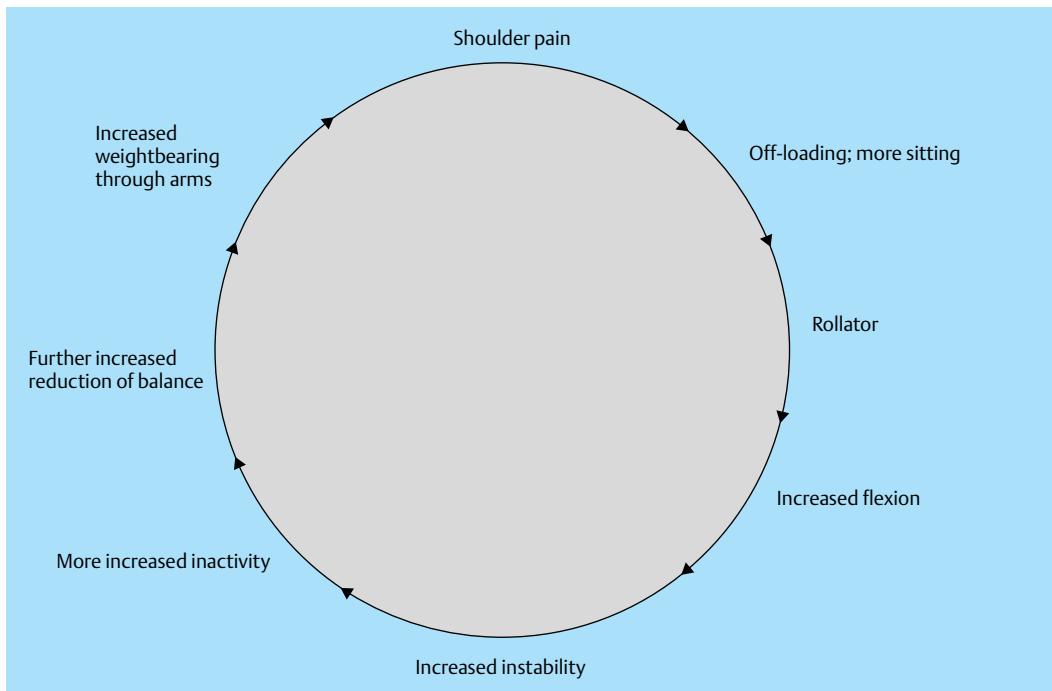


Fig. 4.54 Hypothesis of cause and effect in the activity dimension.

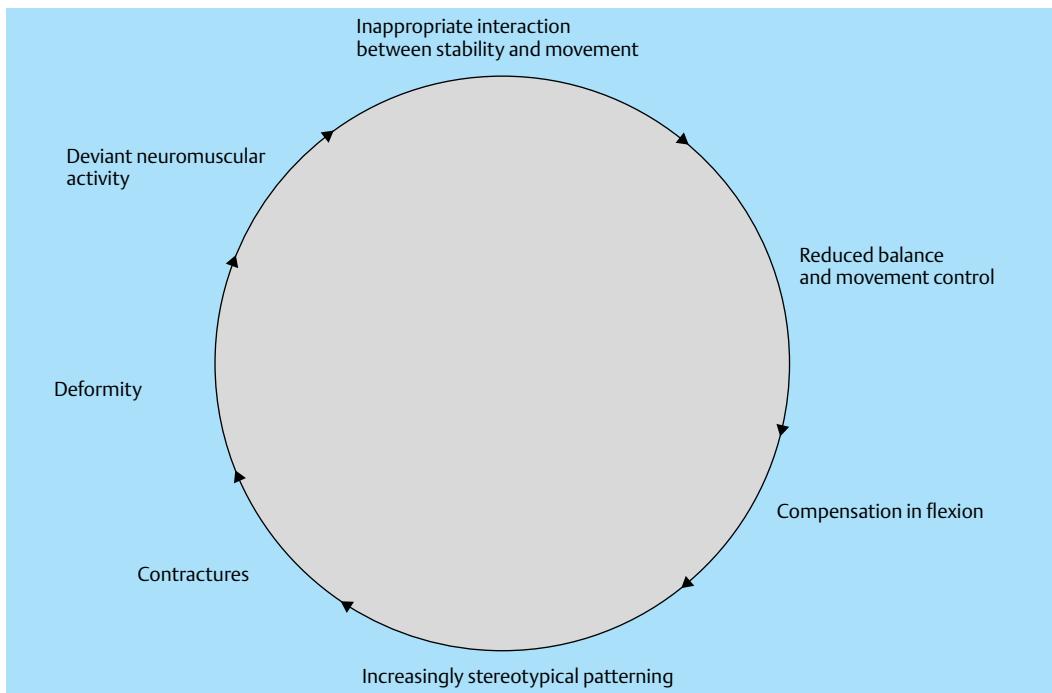


Fig. 4.55 Hypothesis of causal relationships in body structures and functions.

■ Intervention

- Improve muscle length and flexibility, especially the adductors, flexors, and internal rotators of the hips
- Facilitate an improved recruitment of appropriate musculature and alignment in the lower body during sitting, transfers, in standing, and stepping
- Facilitate the interplay between the central and proximal key areas
- Facilitate adaptation to the base of support

■ Physiotherapy (Figs. 4.56–4.80)

Lisa transfers to a plinth. She is positioned on the corner of the plinth with one leg at either side of the corner. This postural set is chosen because the plinth allows a wider base of support and a feeling of more security, at the same time as the position of Lisa's legs facilitates abduction without forcing her legs apart. A firmer base of support may also enable her to activate better.



Fig. 4.56 Lisa's pelvis is tilted anteriorly due to short hip flexors and a relatively fixed lumbar lordosis, therefore she needs to have her upper body supported. The position is adapted by use of a firm wedged support and pillows. A position of flat supine would force the range of both her hips and lumbar spine and might increase tone and pain.



Fig. 4.57 Lisa's back is straightened by flexing both hips and tilting her pelvis more posteriorly. Her seat is lifted toward the corner of the plinth to eccentrically lengthen her back extensors and provide a better opportunity for mobilizing her hip musculature.



Fig. 4.58 Lisa's legs are moved carefully from her hips to start freeing her movement. One leg is moved at a time to maintain the proximal stability of her trunk and pelvis as much as possible.



Fig. 4.59 Lisa's hip and pelvis, contralateral to the leg that is moved, are stabilized manually. The therapist facilitates the pelvis into a more posterior alignment on her right. The facilitation to recruit stabilizing activity is intermittent (not fixed or stereotyped), milliseconds prior to the initiation of movement in her left leg.



Fig. 4.60 The flexor muscles in Lisa's left hip, especially the rectus femoris, are lengthened caudally at the same time as the pelvis is stabilized posteriorly. Possibly Lisa's iliopsoas are short, and these may only be influenced indirectly. The adductors and inward rotators are mobilized and facilitated to lengthen eccentrically.



Fig. 4.61 Lisa is helped to sit by the therapist stabilizing Lisa's pelvis in a posterior position at the same time as she moves her upper body forward through abdominal activity.

The rectus femoris is mobilized and moved laterally over the hip at the same time as the therapist uses her hands in contact with Lisa's trochanters to facilitate hip abduction and hip extension. Alignment is corrected in this way to make it possible to activate the stabilizing musculature surrounding Lisa's hips.

The anterior superior iliac spine is moved backward to allow length to the rectus femoris as the therapist gives length and stabilizes Lisa's thighs with her forearms. This activates Lisa's abdominal muscles. Lisa tries to grade the movement of the pelvis back actively through eccentric activation of the hip flexors and the stabilizing activity of the abdominals. In the adapted supine/half-sitting Lisa's pelvis is stabilized in as much posterior tilt as possible to gain eccentric lengthening of lumbar extensors and hip flexors. This facilitation to improve stability continues as Lisa initiates the movement back into sitting. She is encouraged to reach for the therapist's shoulders to enhance the activation of her abdominals.

The interchange between eccentric and concentric work around the pelvis, hips, and trunk at the same time as the alignment improves and the musculature gains more length, facilitates the interplay between these body segments. The aim is to improve alignment, stability, and balance.

Fig. 4.62a, b Lisa is facilitated to recruit stability around her pelvis and trunk through demands for active and free arms. Compare with Figures 4.46, 4.50, and 4.62. Nearly 3 weeks of physiotherapy where improved alignment and muscle length, facilitation to active control and interplay between the body segments trunk, pelvis, hips/thighs, and arms have enabled improved balance when sitting. Lisa wears socks when her feet are not treated directly because she gets cold easily.

Fig. 4.63a, b When Lisa transfers into standing she usually recruits her adductors and medial hamstrings. The therapist facilitates improved alignment of her hips and activation of abductors, extensors, and external rotators. The range of movement and activity round her hips is still too limited for optimal interplay round Lisa's pelvis and hips, therefore Lisa needs to compensate and does this by fixing her head and neck. Lisa gets some support of her knees to reduce her effort. If Lisa uses too much effort, she loses selectivity and increases her tone. She needs primarily selectivity, reciprocal innervation, and then more strength. Through working in standing and in the transfer from and to standing with improved alignment, Lisa's activation and control of antigravity activity is facilitated.



Fig. 4.62a



Fig. 4.62b



Fig. 4.63a



Fig. 4.63b



Fig. 4.64a



Fig. 4.64b



Fig. 4.64c

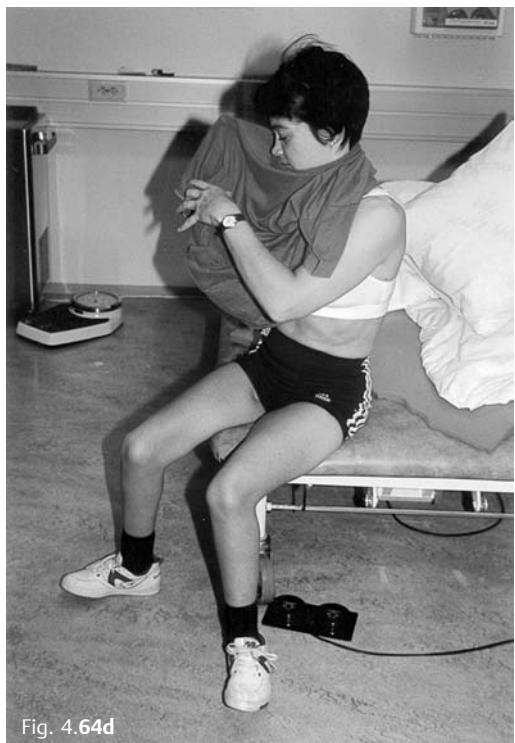


Fig. 4.64d



Fig. 4.64e



Fig. 4.64f

Fig. 4.64a-f Lisa dressing in free sitting. Her feet are in contact with the floor throughout the activity. Compare with Figures 4.45 and 4.46.



Fig. 4.65a



Fig. 4.65b

Fig. 4.65a, b Lisa has never been able to dress whilst standing, but it is important to develop her motor control by demanding activation. Through dressing in a standing position, Lisa uses and develops further the interplay between body segments and her balance.

She obtains the necessary help and support to enable her to perform the activity as optimally as possible. (b) Lisa starts experimenting with her new control and lets go of her arm support. She still needs support of both knees, but they are much more stable.



Fig. 4.66a

Fig. 4.66a, b Lisa's quadriceps are stiff and shortened in all their length. The distal elements toward the knees are mobilized and freed. Through improved muscular flexibility, Lisa's pelvis may be tilted further back. The pelvis is stabilized in this position to enable Lisa to recruit more abdominal flexion to sit up. The therapist gradually lets go of the pelvis as Lisa moves further forward. This position is not good for Lisa: she lies too flat and uses too much effort to sit up and her tone increases and her legs extend.



Fig. 4.66b



Fig. 4.67 Lisa receives some assistance from the therapist in moving her shoulders forward at the same time as the lower parts of the abdominals are facilitated to form a more stable background for the concentric activation in the upper part of the abdominal musculature.



Fig. 4.68



Fig. 4.69



Fig. 4.70

Fig. 4.68 The form of activation demonstrated in the last few photographs influences the hip flexors, adductors, and internal rotators to gradually lengthen. Lisa's thighs seem to rest better on the plinth, which may enhance adaptation to the base of support and thereby both anticipatory and ongoing adjustments of postural control.

Fig. 4.69 Lisa lifts both arms and maintains her balance.

Fig. 4.70 Compare with Figure 4.52. As these photographs were taken with Lisa sitting on the side of the plinth, they are not standardized. They do, however, demonstrate the importance of adapting the postural sets to the patient's main problems. The choice of the corner of the plinth as a seat, combined with mobilizing the hips and facilitating the interaction of stability and movement enables Lisa to recruit improved postural control. Lisa's symmetry is improved through treatment, even if the therapist has not worked on Lisa's trunk directly, which demonstrates Lisa's potential for further improvement.



Fig. 4.71a



Fig. 4.71b



Fig. 4.72

Fig. 4.71a, b Lisa sits with more stability and her alignment is improved. Compare with Figure 4.50. Lisa is able to transfer weight and reach to the side due to improved stability in her pelvis, hips, trunk, and arms. She may influence the flexion of her right trunk herself by being more active.

Fig. 4.72 Lisa is helped to stand. Her hip extensors, abductors, and lateral rotators are activated at the same time as the hip flexors are eccentrically lengthened. Compare with Figure 4.63.



Fig. 4.73



Fig. 4.74

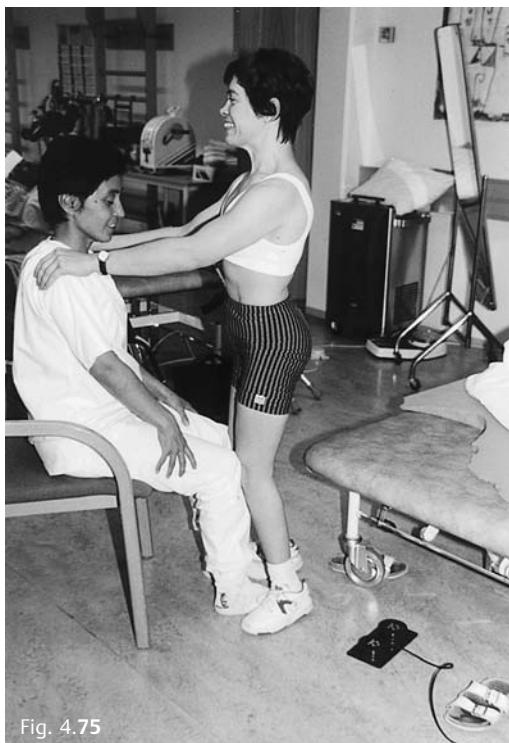


Fig. 4.75

Fig. 4.73 Lisa's stability, movement, and range have improved. She does not need to compensate as much through arm support and flexion of the upper trunk and head. She stands up more easily with better alignment (see Fig. 4.63).

Fig. 4.74 Note the thighs and knees: they are not pressed together and knee extension is more active. She still needs some support of both knees to facilitate the truncal interplay.

Fig. 4.75 Lisa stands with less effort and more upright. The hands are more relaxed.



Fig. 4.76



Fig. 4.77



Fig. 4.78

Fig. 4.76 Hip extension is facilitated as Lisa experiments, challenges, and “plays” with her postural control. She lifts both hands into the air.

Fig. 4.77 Lisa practices knee flexion and extension to enhance her dynamic activity and strength of hips and knees.

Fig. 4.78 Lisa has a tendency for trunk flexion and falling back as she sits down due to reduced coordination of pelvis and trunk. She fixes and locks her trunk through agonist/antagonist co-contraction. Lisa is helped to eccentrically lengthen her back extensors by the therapist stabilizing her upper trunk toward slight flexion to free the movement of the pelvis and improve the interaction and grade the activity down with gravity.



Fig. 4.79a



Fig. 4.79b



Fig. 4.79c

Fig. 4.79a–c Compare with Figures 4.47 and 4.49. This is only the second time in 7 years that Lisa has attempted walking with crutches. It still requires a lot of effort! Lisa takes a few steps as the therapist facilitates trunk interplay. The legs are freer and the thighs and knees not pressed as tightly together (no white marks). Lisa is able to walk several steps. Improved stability demands less effort and better endurance.



Fig. 4.80 Two happy people after 3 weeks of training.

Evaluation

- Lisa has significantly less pain in her shoulders and lower back; both intensity and frequency are reduced.
- She sits more relaxed and secure, her thighs rest better on the base of support, her arms are freer, and she turns her head more easily.
- She has improved coordination and stability of her trunk and pelvis. She dresses more easily and seems less dependent on vision.
- She transfers from sitting to standing more efficiently when she has slight support to both knees and hands. She is able to stand with knees more separated and her patellae pointing more forward. Her hips are more extended. The flexion is most observable in her knees. She is able to stand for short periods with only slight knee support.
- She has not regained her walking function of 7 years ago.
- She feels generally better. She is more stable in sitting, feels safer and uses less effort in dressing. If destabilized, she recovers her balance quicker. She is able to dress in free sitting and fetch objects down from shelves and cabinets. She feels that she has better endurance and that she performs activities more easily. She also says that she recognizes her body and feels better in herself.

Follow-up

- Lisa will be followed up by a therapist who has attended Bobath courses and who has experience in working with adults with neurologic disorders and children with cerebral palsy.
- Riding was not possible, as no qualified hippotherapists were available at this time. Lisa was placed on a waiting list.
- Lisa was offered a further training period in the department some months later in order to build on the current improvement.

The author would like to thank Lisa for her positive attitude, motivation, and good cooperation.

Thank you Lisa.

Bibliography

Introduction

- Bhakta BB, Cozens JA, Chamberlain MA. Use of botulinum toxin in stroke patients with severe upper limb spasticity. *J Neurol Neurosurg Psychiatry* 1996; 61: 30–35.
- Gelber DA, Jozefczyk PB. Therapeutics in the management of spasticity. *Neurorehabil Neural Repair* 1999; 13: 5–14.
- Lagalla G, Danni M, Reiter F, Ceravolo MG, Provinciali L. Post-stroke spasticity management with repeated botulinum toxin injections in the upper limb. *Am J Phys Med Rehabil* 2000; 79: 377–384.
- Malterud K. Qualitative research: standards, challenges, and guidelines. *Lancet* 2001a; 358: 483–488.
- Malterud K. The art and science of clinical knowledge: evidence beyond measures and numbers. *Lancet* 2001b; 358: 397–400.
- Mant D. Can randomised controlled trials inform clinical decisions about individual patients? *Lancet* 1999; 353: 753–757.
- Rose DJ. A multilevel approach to the study of motor control and learning. Boston: Allyn and Bacon, 1997.
- Sampaio C, Ferreira JJ, Pinto AA, Crespo M, Ferro JM, Castro-Caldas A. Botulinum toxin A for the treatment of arm and hand spasticity after stroke. *Clin Rehabil* 1997; 11: 3–7.
- Schleichkorn J. The Bobaths. A biography of Berta and Karel Bobath. USA: Therapy Skill Builders, 1992.

Chapter 1 Applied Neurophysiology

1.1 Systems Control

The Neuromuscular System

- Academy of Medical Sciences. Report: Restoring Neurological Function: Putting the Neurosciences to work in neurorehabilitation, 2004. Available at: www.acmedsci.ac.uk (accessed 2005).
- Ada L, Canning C. Anticipating and Avoiding Muscle Shortening. In: Ada L, Canning C, editors. Key Issues in Neurological Physiotherapy. Physiotherapy: Foundations for Practise 1990: 219–224.
- Brodal P. The Central Nervous System. Structure and Function. Oxford: Oxford University Press, 1998.
- Brodal P. Sentralnervesystemet. 3rd ed. Universitetsforlaget, 2001.
- Brodal P. Det nevrobiologiske grunnlaget for balanse. *Fysioterapeuten* 2004; 8: 25–30.
- Burke RE, Levine DN, Salcman M, Tsairis P. Motor units in cat soleus muscle: physiological, histochemical and morphological characteristics. *J Physiol* 1973; 234: 723–748.
- Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 1992; 1: 33–69.
- Goldspink G, Williams P. Muscle fibre and connective tissue changes associated with use and disuse. In: Ada L and Canning C, editors. Key Issues in Neurological Physiotherapy. Physiotherapy: Foundations for Practise 1990: 197–215.
- Henneman E, Mendell LM. Functional organisation of motor neuron pool and its inputs. In: Brooks VB,

- editor. *Handbook of Physiology—the Nervous System*. Baltimore: Williams and Wilkins, 1981: 423–505.
- Hufschmidt A, Mauritz K-H. Chronic Transformation of muscle in spasticity: a peripheral contribution to increased tone. *J Neurol Neurosurg Psychiatry* 1985; 48:676–685.
- Kandel ER, Schwartz JH, Jessel TM. *Principles of neural science*. 4th ed. New York: McGraw-Hill, 2000.
- Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. *Proc Natl Acad Sci USA* 2000; 25:13031–13036.
- Kerty E. Synsrehabilitering etter hjerneskade. *Tidsskr Nor Lægeforen* 2005; 125:146–147.
- Kidd G. The myotatic reflex. In: Downie P, editor. *Cash's Textbook of Neurology for Physiotherapists*. London: Faber & Faber, 1986: 85–103.
- Kidd G, Lawes N, Musa I. *Understanding Neuromuscular Plasticity*. London: Edward Arnold, 1992.
- Langton P. What determines muscle fibre type? www.bris.ac.uk/Depts/Physiology/ugteach/ugindex/m1_index/nm_tut5/page4.htm, 1998 (accessed 2005).
- MacKay-Lyons M. Central pattern generation of locomotion: a review of the evidence. *Phys Ther* 2002; 82:69–83.
- Massion J. Movement, posture and equilibrium. *Prog Neurobiol* 1992; 38:35–56.
- Massion J. Postural control system. *Curr Opin Neurobiol* 1994; 4:877–887.
- Mosby's Medical, Nursing and Allied Health Dictionary. 4th ed, 1994.
- Mulder T, Nienhuis B, Pauwels J. The assessment of motor recovery: a new look at an old problem. *J Electromyogr Kinesiol* 1996; 2:137–145.
- Riise R, Gundersen B, Brodal S, Bjerke P. Synsproblemer ved hjerneslag. *Tidsskr Nor Lægeforen* 2005; 125:176–177.
- Rothwell J. *Control of Human Voluntary Movement*. London: Chapman & Hall, 1994.
- Sahrmann SA. Posture and Muscle Imbalance. *Physiotherapy* 1992; 78(1) Postgraduate Advances in Physical Therapy—APTA 1987: 1–19.
- Sahrmann SA. Diagnosis and Treatment of Movement Impairment Syndromes. St. Louis, MO: Mosby; 2002.
- Shumway-Cook A, Woollacott M. *Motor Control. Translating Research into Clinical Practice*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Sieck GC. Plasticity in skeletal, cardiac, and smooth muscle [editorial]. *Highlighted Topics Series. J Appl Physiol* 2001; 90(1).
- Simons DG, Mense S. Understanding and measurement of muscle tone as related to clinical muscle pain. *Pain* 1998; 1:1–17.
- Stokes M. *Neurological Physiotherapy*. London: Mosby; 1998.
- Taber's Cyclopedic Medical Dictionary. Thomas CL: FA Davies Company, 1997.
- Tyldesley B, Grieve JL. *Muscles, Nerves and Movement. Kinesiology in Daily Living*. Oxford: Blackwell Science, 1996.
- van Ingen Schenau GJ, Bobbert MF, van Soest AJ. The unique action of bi-articular muscles in leg extensions. In: Winters JM, Woo SLY, editors. *Multiple Muscle Systems: Biomechanics and Movement Organization*. Berlin: Springer-Verlag 1990: 639–652.
- Ward NS, Cohen LG. Mechanisms underlying recovery of motor function after stroke. *Arch Neurol* 2004; 61:1844–1848.
- Wikipedia online encyclopedia. Wikimedia Foundation, Inc. <http://en.wikipedia.org/wiki/> (accessed 2006).
- Zackowski KM, Dromerick AW, Sahrmann SA, Thach WT, Beatian AJ. How do strength, spasticity and joint inividuation relate to the reaching deficits of people with chronic hemiparesis? *Brain* 2004; 127:1035–1046.

The Somatosensory System, Vision, and Balance

- Ada L, Canning C. Anticipating and avoiding muscle shortening. In: Ada L, Canning C, editors. *Key Issues in Neurological Physiotherapy. Physiotherapy: Foundations for Practise* 1990: 219–224.
- Brodal P. *Sentralnervesystemet*. 2nd ed. Oslo: Tano, 1995.
- Davidoff RA. The pyramidal tract. *Neurology* 1990; 40: 332–339.
- Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 1992; 72: 33–69.
- Harkema SJ, Hurley SL, Patel UK, Requejo PS, Dobkin BH, Edgerton VR. Human lumbbosacral spinal cord interprets loading during stepping. *J Neurophysiol* 1997; 77: 797–911.
- Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: new insights for treatment of balance disorders. *Phys Ther* 1997; 77: 517–533.
- Kandel ER, Schwartz JH, Jessel TM. *Principles of Neural Science*. 4th ed. Columbus, OH: McGraw-Hill; 2000.
- Kidd G, Lawes N, Musa I. *Understanding Neuromuscular Plasticity*. London: Edward Arnold, 1992.
- Massion J. Movement, posture and equilibrium. *Prog Neurobiol* 1992; 38: 35–56.
- Mudge S, Rochester L. Neurophysiological rationale of treadmill training: evaluating evidence for practice. *NZ J Physiother* 2001; 2: 6–15.

- Mulder T, Nienhuis B, Pauwels J. The assessment of motor recovery: a new look at an old problem. *J Electromyogr Kinesiol* 1996; 6: 137–145.
- Nashner LM. Adaptation of human movement to altered environments. *TINS* 1982; 358–361.
- Petersen H, Magnusson M, Johansson R, Åkesson M, Fransson PA. Acoustic cues and postural control. *Scand J Rehabil Med* 1995; 27: 99–104.
- Rothwell J. Control of Human Voluntary Movement. London: Chapman & Hall, 1994.
- Shumway-Cook A, Woollacott M. Motor Control. Translating Research into Clinical Practice. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Sunderland A, Tinson DJ, Bradley EL, et al. Enhanced physical therapy improves recovery of arm function after stroke. *J Neurol Neurosurg Psychiatry* 1992; 55: 530–535.
- Taber's Cyclopedic Medical Dictionary. 18th ed. Thomas CL, editor. Philadelphia: FA Davies, 1997.
- Trew M, Everett T. Human Movement: An Introductory Text. 3rd ed. London: Churchill Livingstone, 1998.
- Wade M, Jones G. The role of vision and spatial orientation in the maintenance of posture. *Phys Ther* 1997; 77: 619–628.
- Yekuitieli M, Guttman E. A controlled trial of the re-training of the sensory function of the hand in stroke patients. *J Neurol Neurosurg Psychiatry* 1993; 56: 241–244.
- Chen PT, Liaw MY, Wong MK, Tang FT, Lee MY, Lin PS. The sit-to-stand movement in stroke patients and its correlation with falling. *Arch Phys Med Rehabil* 1998; 79: 1043–1046.
- Cornall C. Self propelling wheelchairs: the effect on spasticity in hemiplegic patients. *Physiother Theory Pract* 1991; 7: 13–21.
- Davidoff RA. The pyramidal tract. *Neurology* 1990; 40: 332–339.
- Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 1992; 72: 33–69.
- Dietz V, Duysens J. Modulation of reflex mechanisms by load receptors. *Gait Posture* 2000; 11: 102–110.
- Dietz V, Zijlstra W, Duysens J. Human interlimb coordination during split-belt locomotion. *Exp Brain Res* 1994; 101: 513–520.
- Gjerstad L, Kerty E, Nyberg-Hansen R. Behandling av focale dystonier med botulinumtoxin. *Tidsskr Nor Lægeforen* 1991; 21: 2637–2639.
- Guyton AC. Textbook of Medical Physiology. Physiotherapy Theory and Practice. 5th ed. Philadelphia: WB Saunders company, 1976.
- de Haart M, Guerts AC, Huidekoper SC, Fasotti L, Van Limbeek J. Recovery of standing balance in post-acute stroke patients: A rehabilitation cohort study. *Arch phys med rehabil* 2004; 85(6): 886–895.
- Harkema SJ, Hurley SL, Patel UK, Requejo PS, Dobkin BH, Edgerton VR. Human lumbosacral spinal cord interprets loading during stepping. *J Neurophysiol* 1997; 77: 797–911.
- Horak FB, Diener HC. Cerebellar control of postural scaling and central set in stance. *J Neurophysiol* 1994; 72: 2: 479–493.
- Jobst EE, Melnick ME, Byl NN, Dowling GA, Aminoff MJ. Sensory perception in Parkinson disease. *Arch Neurol* 1997; 54: 450–454.
- Karnath H-O, Ferber S, Dichgans J. The origin of contraversive pushing—Evidence for a second graviceptive system in humans. *Neurology* 2000; 55: 1298–1304.
- Kavounoudias A, Roll R, Roll JP. The plantar sole is a “dynamometric map” for human balance control. *Neuroreport* 1998; 9: 3247–52.
- Kerty E. Vision rehabilitation after brain injury [in Norwegian]. *Tidsskr Nor Laegeforen* 2005; 125: 146.
- Khemlani MN, Carr JH, Crosbie WJ. Muscle synergies and joint linkages in sit-to-stand under two initial foot positions. *Clin Biomech* 1998; 14: 236–238.
- Kidd G, Lawes N, Musa I. Understanding neuromuscular plasticity. London: Edward Arnold, 1992.
- Knapp HD, Taub E, Berman J. Movements in monkeys with deafferented forelimbs. *Exp Neurol* 1963; 7: 305–315.
- Lalonde R, Botez MI. The cerebellum and learning processes in animals. *Res Rev* 1990; 15: 325–332.

The Brain and Spinal Cord

- Borgmann R. Behandling av spastisk torticollis med botulinumtoxin A. *Tidsskr Nor Lægeforen* 1997; 13: 1889–1891.
- Bridgewater KJ, Sharpe MH. Trunk muscle performance in early Parkinson's disease. *Phys Ther* 1998; 78: 566–576.
- Brodal P. Sentralnervesystemet. 2nd ed. Oslo: Tano, 1995.
- Brodal P. The central nervous system [in Norwegian]. 3rd ed. Oslo: Universitetsforlaget; 2001.
- Brodal P. The Central Nervous System. Structure and Function. Oxford: Oxford University Press, 1998.
- Brodal P. Det nevrobiologiske grunnlaget for balanse. *Fysioterapeuten* 2004; 8: 25–30.
- Bussel B, Roby-Brami A, Neris OR, Yakoleff A. Evidence for a spinal stepping generator in man. Electrophysiological study. *Acta Neurobiol Exp* 1996; 56: 465–468.
- Byl NN, Merzenich MM, Cheung S, Bedenbaugh, Nagarajan SS, Jenkins WM. A primate model for studying focal dystonia and repetitive strain injury: effects on the primary somatosensory cortex. *Phys Ther* 1997; 77: 269–284.

- MacKay-Lyons M. Central pattern generation of locomotion: a review of the evidence. *Phys Ther* 2002; 82: 69–83.
- Maki EB, McIlroy WE. The role of limb movements in maintaining upright stance: the 'change-in-support' strategy. *Phys Ther* 1997; 77: 488–507.
- Markham C. Vestibular control of muscle tone and posture. *Can J Neurol Sci* 1987; 14: 493–496.
- Marque Ph, Felez A, Puel M, et al. Impairment and recovery of left motor function in patients with right hemiplegia. *J Neurol Neurosurg Psychiatry* 1997; 62: 77–81.
- Marsden CD, Quinn NP. The dystonias. *BMJ* 1990; 300: 139–144.
- Marsden CD, Rothwell JC, Day BL. The use of proprioceptive feedback in the control of movement. *Trends Neurosci* 1984; 7: 253–258.
- Mulder T. A process-oriented model of human behavior. *Phys Ther* 1991; 71: 157–164.
- Mulder T, Nienhuis B, Pauwels J. The assessment of motor recovery: A new look, at an old problem. *Journal of Electromyography and Kinesiology* 1996; 6(2): 137–145.
- Musa I. The role of afferent input in the reduction of spasticity; an hypothesis. *Physiotherapy* 1986; 72: 179–182.
- Nashner LM. Adaptation of human movement to altered environments. *TINS* 1982; 358–361.
- Paillard J. Basic neurophysiological structures of eye-hand coordination. In: Williams HG, editor. *Growth, Motor Development and Physical Activity Across The Life-Span*. Columbia: University of South Carolina Press, 1990: 26–74.
- Patten C, Lexell J, Brown HE. Weakness and strength training in persons with poststroke hemiplegia. Rationale, method and efficacy. *J Rehabil Res Dev* 2004; 41: 293–312.
- Pearson KG. Common principles of motor control in vertebrates and invertebrates. *Annu Rev Neurosci* 1993; 16: 256–297.
- Riise R, Gundersen B, Brodal S, Bjerke P. Visual problems in Cerebral stroke [in Norwegian]. *Tidsskr Nor Laegeforen* 2005; 125(2): 176–177.
- Rothwell J. Control of Human Voluntary Movement. London: Chapman & Hall, 1994.
- Rothwell JC, Taub MM, Day BL, et al. Manual motor performance in deafferented man. *Brain* 1982; 105: 515–542.
- Shepherd RB, Koh HP. Some biomechanical consequences of varying foot placements in sit-to-stand in young women. *Scand J Rehabil Med* 1996; 28: 79–88.
- Shumway-Cook A, Woollacott M. *Motor Control. Translating Research into Clinical Practice*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Smidt GL. *Gait in Rehabilitation. Clinics in Physical Therapy*. New York: Churchill Livingstone, 1990.
- Thach WT, Goodkin HP, Keating JG. The cerebellum and the adaptive coordination of movement. *Annu Rev Neurosci* 1992; 15: 403–442.
- Thilmann AF, Fellows SJ, Garms E. Pathological stretch reflexes on the 'good' side of hemiparetic patients. *J Neurol Neurosurg Psychiatry* 1990; 53: 208–214.
- Trew M, Everett T. *Human Movement: An Introductory Text*. 3rd ed. New York: Churchill Livingstone, 1998.
- Wade M, Jones G. The role of vision and spatial orientation in the maintenance of posture. *Phys Ther* 1997; 77: 619–628.
- Whittle MW. *Gait analysis, an introduction*. 2nd ed. Oxford: Butterworth-Heinemann, 1996.
- ## 1.2 Plasticity
- Aboderin I, Venables G, for the Pan European Consensus Meeting on Stroke Management. *Stroke Management in Europe*. *J Intern Med* 1996; 240: 173–170.
- Academy of Medical Sciences. Report: Restoring Neurological Function: Putting the Neurosciences to work in neurorehabilitation, 2004 (available at: www.acmedsci.ac.uk/) (accessed 2005).
- Agnati LF, Zoli M, Biagini G, Fuxe K. Neuronal plasticity and the ageing processes in the frame of the 'Red Queen Theory'. *Acta Physiol Scand* 1992; 145: 301–309.
- Ashburn A. Physical recovery following stroke. *Physiotherapy* 1997; 83: 480–490.
- Bailey CH, Kandel ER. Structural changes accompanying memory storage. *Annu Rev Physiol* 1993; 55: 397–426.
- Benowitz LI, Routtenberg A. GAP-43: an intrinsic determinant of neuronal development and plasticity. *Trends Neurosci* 1997; 20: 84–98.
- Bishop B. Neural plasticity IV. *Phys Ther* 1982; 62: 1442–1451.
- Bobath B. *Adult Hemiplegia: Evaluation and Treatment*. 2nd ed. London: William Heinemann, 1978.
- Bobath B. *Hemiplegia, Evaluation and Treatment*. 3rd ed. Oxford: Heinemann, 1990.
- Brodal P. *Sentralnervesystemet*. 2nd ed. Oslo: Tano, 1995.
- Brodal P. *The Central Nervous System. Structure and Function*. Oxford: Oxford University Press, 1998.
- Brodal P. *Sentralnervesystemet*. Oslo: Universitetsforlaget, 2001.
- Craik RL. Recovery processes: maximizing function. In: *contemporary management of motor control problems*. Proceedings of the II Step Conference 1991: 165–173.

- Cramer SC, Bastings EP. Mapping clinically relevant plasticity after stroke. *Neuropharmacology* 2000; 39: 842–851.
- Dietrichs E, Gjerstad L. *Vår fantastiske hjerne*. Oslo: Universitetsforlaget, 1995.
- Dietz V, Wirz M, Jensen L. Locomotion in patients with spinal cord injuries. *Phys Ther* 1997; 5: 508–516.
- Eccles JC. *Evolution of the brain: creation of self*. London: Routledge, 1989.
- Eriksson PS, Perfilieva E, Björk-Eriksson T, et al. Neurogenesis in the adult human hippocampus. *Nat Med* 1998; 4: 1313–1317.
- Feys H, De Weerdt W, Verbeke G, et al. Early and repetitive stimulation of the arm can substantially improve the long-term outcome after stroke. A 5-year follow-up study of a randomized trial. *Stroke* 2004; 35: 924–929.
- Goldberger ME, Murray M. Patterns of sprouting and implications for recovery of function. In: Waxman SG, editor. *Advances in Neurology: Functional recovery in neurological disease*. New York: Raven Press, 1988; 361–385.
- Hallett M. The plastic brain. *Ann Neurol* 1995; 38: 4–5.
- Hori J, Ng TF, Shatos M, Klassen H, Streilein JW, Young MJ. Neural progenitor wells lack immunogenicity and resist destruction of allografts. *Stem Cells* 2003; 21: 405–416.
- Indredavik B, Bakke F, Solberg R, Rokseth R, Lund Haheim L, Holme I. Benefit of a stroke unit. A randomized controlled trial. *Stroke* 1991; 22: 1026–1031.
- Kempermann G, Kuhn HG, Winkler J, Gage FH. Neue Nervenzellen für das erwachsene Gehirn. *Der Nervenarzt* 1998; 10: 851–857.
- Kidd G, Lawes N, Musa I. *Understanding Neuromuscular Plasticity*. London: Edward Arnold, 1992.
- Kwakkel G, Kollen B, Lindeman E. Understanding the pattern of functional recovery after stroke: facts and theories. *Restor Neurol Neurosci* 2004a; 22: 281–299.
- Kwakkel G, van Peppen R, Wagenaar RC, et al. Effects of augmented exercise therapy time after stroke. A meta-analysis. *Stroke* 2004b; 35: 2529–2536.
- Lee RG, van Donkelaar P. Mechanisms underlying functional recovery following stroke. *Can J Neurol Sci* 1995; 22: 257–263.
- Liepert J, Bauder H, Miltner WHR, Taub E, Weiller C. Treatment-induced cortical reorganization after stroke in humans. *Stroke* 2000; 31: 1210–1216.
- Martin J-L, Magistretti PJ. Regulation of gene expression by neurotransmitters in the central nervous system. *Eur Neurol* 1998; 39: 129–134.
- Mosby's Medical, Nursing and Allied Health Dictionary. 4th ed. New York: Mosby-Year Book Inc., 1994.
- Muir GD, Steeves JD. Sensorimotor stimulation to improve locomotor recovery after spinal cord injury. *Trends Neurosci* 1997; 20: 72–77.
- Nudo RJ. Adaptive plasticity in motor cortex: implications for rehabilitation after brain injury. *J Rehabil Med* 2003; Suppl. 41: 7–10.
- Nudo RJ, Wise BM, SiFuentes F, Milliken GW. Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. *Science* 1996; 272: 1791–1794.
- Olson L. Neurotrofa faktorer i CNS. Allt fler proteiner med klinisk potensial. *Nordisk Medicin* 1996; 111: 3–6.
- Seil FJ. Recovery and repair issues after stroke from the scientific perspective. *Curr Opin Neurol* 1997; 10: 49–51.
- Shumway-Cook A, Woollacott M. *Motor Control. Translating Research into Clinical Practice*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Small SL, Hlustik P, Noll DC, Genovese C, Solodkin A. Cerebellar hemispheric activation ipsilateral to the paretic hand correlates with functional recovery after stroke. *Brain* 2002; 125: 1544–1557.
- Solheim BG, Stamceller fra navlestrøm gav bedring hos paraplegiker. *Tidsskr Nor Lægeforen* 2005; 125: 32–38.
- Squire LR, Knowlton B, Musen G. The structure and organization of memory. *Annu Rev Psychol* 1993; 44: 453–495.
- Stein DG, Brailowsky S, Will B. *Brain Repair*. Oxford: Oxford University Press, 1995.
- Stephenson RA. Review of neuroplasticity: some implications for physiotherapy in the treatment of lesions of the brain. *Physiotherapy* 1993; 79: 699–704.
- Stroke Units Trialists' Collaboration. Collaborative systematic review of the readmission trials of organised inpatient (stroke unit) care after stroke. *BMJ* 1997; 314: 1151–1159.
- Sundar T. Nytt liv for neuroner. *Tidsskrift Norska Lægeforening* 1999; 1: 100.
- Taub E, Uswatte G, Pidikiti R. Constraint-Induced Movement Therapy: A new family of techniques with broad application to physical rehabilitation—a clinical review. *J Rehabil Res Devel* 1999; 36: 237–251.
- Troenen H, Edgar H. The regulation of neuronal gene expression. *TINS* 1982; 7: 311–313.
- Turton A, Pomeroy V. When should upper limb function be trained after stroke? Evidence for and against early intervention. *Neurorehabilitation* 2002; 17: 215–224.
- Ullian EM, Christopherson KS, Barres BH. Role for glia in synaptogenesis. *Glia* 2004; 47: 209–216.
- Umphred D. Merging neurophysiologic approaches with contemporary theories. I: Contemporary Management of Motor Control Problems. Proceedings of the II Step Conference 1991: 127–130.
- Ward NS, Cohen LG. Mechanisms underlying recovery of motor function after stroke. *Arch Neurol* 2004; 61: 1844–1848.

1.3 Consequences of and Reorganization after CNS Lesions

- Ada L, Canning C. Anticipating and Avoiding Muscle Shortening. In: Ada L, Canning C, editors. Key Issues in Neurological Physiotherapy. Physiotherapy: Foundations for Practise 1990; 219–224.
- Ashburn A, Lynch M. Disadvantages of the early use of wheelchairs in the treatment of hemiplegia. *Clin Rehabil* 1988; 2: 327–331.
- Bobath B. Hemiplegia, Evaluation and Treatment. 3rd ed. Oxford: Heinemann, 1990.
- Brodal P. The Central Nervous System. Structure and Function. Oxford: Oxford University Press, 1998.
- Brodal P. Sentralnervesystemet. Oslo: Universitetsforlaget, 2001.
- Brodal P. Det nevrobiologiske grunnlaget for balanse. *Fysioterapeuten* 2004; 8: 25–30.
- Brown P. Pathophysiology of spasticity. *J Neurol Neurosurg Psychiatry* 1994; 57: 773–777.
- Burke D. Spasticity as an adaptation to pyramidal tract injury. In: Waxman SG, editor. Advances in Neurology: Functional Recovery in Neurological Disease. New York: Raven Press 1988: 401–423.
- Burridge JH, Wood DE, Hermens HJ, et al. Theoretical and methodological considerations in the measurement of spasticity. *Disabil Rehabil* 2005; 27: 69–80.
- Canning CG, Ada L, Adams R, O'Dwyer NJ. Loss of strength contributes more to physical disability than loss of dexterity. *Clin Rehabil* 2004; 18: 300–308.
- Carr JH, Shepherd RB. A Motor Relearning Programme for Stroke. Aspen, 1983.
- Carr JH, Shepherd RB, Ada L. Spasticity: Research findings and implications for intervention. *Physiotherapy* 1995; 81: 421–427.
- Ching-Lin H, Ching-Fan S, I-Ping H, Chun-Hou W. Trunk control as an early predictor of comprehensive activities of daily living function in stroke patients. *Stroke* 2002;33: 2626–2630.
- Cornall C. Self-propelling wheelchairs: the effect on spasticity in hemiplegic patients. *Physiother Theory Pract* 1991; 7: 13–21.
- Craik RL. Recovery processes: maximizing function. In: Contemporary Management of Motor Control Problems. Proceedings of the II Step Conference 1991: 165–173.
- Cramer SC, Bastings EP. Mapping clinically relevant plasticity after stroke. *Neuropharmacology* 2000; 39: 842–851.
- Cramer SC, Nelles G, Benson RR, et al. A functional MRI study of subjects recovered from hemiparetic stroke. *Stroke* 1997; 28: 2518–2527.
- Dietz V, Colombo G, Jensen L, Baumgartner L. Locomotor capacity of spinal cord in paraplegic patients. *Ann Neurol* 1995; 37: 574–582.
- Dietz V, Wirz M, Jensen L. Locomotion in patients with spinal cord injuries. *Phys Ther* 1997; 77(5): 508–516. Review.
- Dvir Z, Panturin E. Measurement of spasticity and associated reactions in stroke patients before and after physiotherapeutic intervention. *Clin Rehabil* 1993; 7: 15–21.
- Edwards S. Neurological Physiotherapy, a Problem Based Approach. 1st ed. Edinburgh: Churchill Livingstone, 1996.
- Given JD, Dewald JPA, Rymer WZ. Joint dependent passive stiffness in paretic and contralateral limbs of patients with hemiparetic stroke. *J Neurol Neurosurg Psychiatry* 1995; 59: 271–279.
- Goldspink G, Williams P. Muscle fibre and connective tissue changes associated with use and disuse. In: Ada L, Canning C, editors. Key Issues in Neurological Physiotherapy. Stoneham: Butterworth-Heinemann, 1990: 197–215.
- Hufschmidt A, Mauritz K-H. Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. *J Neurol Neurosurg Psychiatry* 1985; 48: 676–685.
- Johnson GR. Editorial. *Disabil Rehabil* 2005; 27(1/2): 1.
- Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, editors. Spasticity: Disordered Motor Control. Chicago, IL: Year Book Medical Publishers, 1980: 485–94.
- Marque PH, Felez A, Puel M, et al. Impairment and recovery of left motor function in patients with right hemiplegia. *J Neurol Neurosurg Psychiatry* 1997; 62: 77–81.
- Mission J, Woollacott MH. Posture and equilibrium. In: Bronstein AM, Brandt T, Woollacott M, editors. Clinical Disorders of Balance, Posture and Gait. London: Arnold, 1996: 1–18.
- Myhr KM. Multiple sclerosis: etiology, interferon treatment and prognosis. Doctoral thesis. The National Multiple Sclerosis Centre, Department of Neurology, Haukelan University Hospital, University of Bergen, Norway 2001.
- O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996; 119: 1737–1749.
- Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005, 27: 2–6.
- Patten C, Lexell J, Brown HE. Weakness and strength training in persons with poststroke hemiplegia. Rationale, method and efficacy. *J Rehabil Res Dev* 2004; 41: 293–312.
- Platz T, Eickhof C, Nuyens G, Vuadens P. Clinical scales for the assessment of spasticity, associated phe-

- nomena, and function: a systematic review of the literature. *Disabil Rehabil* 2005; 27: 7–18.
- Rothwell J. *Control of Human Voluntary Movement*. London: Chapman & Hall, 1994.
- Shumway-Cook A, Woollacott M. *Motor Control. Translating Research into Clinical Practice*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Soderlund A, Malterud K. Why did I get chronic fatigue syndrome? *Scan J Primary Health Care* 2005; 23: 242–247.
- Stephenson R, Edwards S, Freeman J. Associated reactions: their value in clinical practise? *Physiother Res Int* 1998; 3: 69–81 (plus discussion).
- Stokes M. *Neurological Physiotherapy*. London: Mosby, 1998.
- Thilmann AF, Fellows SJ, Garms E. Pathological stretch reflexes on the “good” side of hemiparetic patients. *J Neurol Neurosurg Psychiatry* 1990; 53: 208–214.
- Toft E. Mechanical and electromyographic stretch responses in spastic and healthy subjects. *Acta Neurol Scand* 1995; Suppl 163; 92: 1–24.
- Turton A, Pomeroy V. When should upper limb function be trained after stroke? Evidence for and against early intervention. *Neurorehabilitation* 2002; 17: 215–224.
- Tyldesley B, Grieve JL. *Muscles, nerves and movement. Kinesiology in daily living*. Oxford: Blackwell Science Ltd, 1996.
- Verheyden G, Nieuwboer A, Mertin J, Kiekens C, De Weerd W. The trunk impairment scale: a new tool to measure motor impairment of the trunk after stroke. *Clin Rehabil* 2004; 18: 326–334.
- Voerman GE, Gregoric M, Hermens HJ. *Neurophysiological methods for the assessment of spasticity: the Hoffmann reflex, the tendon reflex, and the stretch reflex*. *Disabil Rehabil* 2005; 27: 33–68.
- Ward NS, Cohen LG. Mechanisms underlying recovery of motor function after stroke. *Arch Neurol* 2004; 61: 1844–1848.
- Wikipedia, the free encyclopedia, last modified 2 January 2006 (available at: <http://en.wikipedia.org/wik>).
- Wood DE, Burridge JH, van Wijck FM, et al. Biomechanical approaches applied to the lower and upper limb for the measurement of spasticity: a systematic review of the literature. *Disabil Rehabil* 2005; 27: 19–32.
- Yarkony GM, Sahgal V. Contractures. *Clin Orthop Relat Res* 1987; 219: 93–96.
- Aaslund M. Treadmill training—is it different from overground walking? Master Degree dissertation, Section for Physiotherapy Science, Department of Public Health and Primary Health Care, Faculty of Medicine, Bergen, Norway 2006.
- Ada L, Canning C. Anticipating and avoiding muscle shortening. In: Ada L, Canning C, eds. *Key Issues in Neurological Physiotherapy*. Stoneham: Butterworth-Heinemann, 1990: 219–224.
- Albany K. Rehabilitation for patients receiving Botox for spasticity. Considerations for function and efficacy. *We Move, Worldwide Education and Awareness for Movement Disorders*. Mt Sinai Medical Center, New York, 1995.
- Allum JH, Honegger F. Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. *Exp Brain Res* 1998; 121: 478–494.
- Allum J, Bloem B, Carpenter M, Verschueren J, Honegger F. Triggering of balance corrections and compensatory strategies in a patient with total leg proprioceptive loss. *Exp Brain Res* 2002; 142: 91–107.
- Åsberg KH. Orthostatic tolerance training of stroke patients in general medical wards. *Scan J Rehabil Med* 1989; 21: 179–185.
- Ashburn A, Lynch M. Disadvantages og the early use of wheelchairs in the treatment of hemiplegia. *Clin Rehabil* 1988; 2: 327–331.
- Bader-Johansson C. *Grundmotorik Studentlitteratur*, Lund, 1991.
- Bakheit AMO, Fedorova NV, Skoromets AA, Timerbaeva SL, Bhakta BB, Coxon L. The beneficial anti-spasticity effect of botulinum toxin type A is maintained after repeated treatment cycles. *J Neurol Neurosurg Psychiatry* 2004; 75: 1558–1561.
- Baykousheva-Mateva V, Mandaliev A. Artificial feed-forward as preparatory motor control in postictal hemiparesis. *Electromyogr Clin Neurophysiol* 1994; 34: 445–448.
- Berg-Johnsen J, Røste GK, Solgaard T, Lundar T. Kontinuerlig intratekal infusjon med baklofen. *Tidsskr Nor Lægeforen* 1998; 21: 3256–3260.
- Bergland A. Postural kontroll. Bevegelsens skygge. *Fysioterapeuten* 1994; 15: 18–24.
- Bharadwaj K, Sugar TG, Koeneman EJ. Design of a robotic gait trainer using spring over muscle actuators for ankle stroke rehabilitation. *J Biomech Eng* 2005; 127: 1009–13.
- Bobath B. *Adult Hemiplegia: Evaluation and Treatment*. 2nd ed. Oxford: Heinemann, 1978.
- Bobath B. *Hemiplegia, Evaluation and Treatment*. 3rd ed. Oxford: Heinemann, 1990.

- Borgmann R. Behandling av spastisk torticollis med botulinumtoxin A. *Tidsskr Nor Lægeforen* 1997; 13: 1889–91.
- Bridgewater KJ, Sharpe MH. Trunk muscle performance in early Parkinson's disease. *Phys Ther* 1998; 78: 566–576.
- Brodal P. *Sentralnervesystemet*. 2nd ed. Oslo: Tano, 1995.
- Brodal P. *Sentralnervesystemet*. 3rd ed. Oslo: Universitetsforlaget; 2001.
- Brodal P. Det neurobiologiske grunnlaget for balanse. *Fysioterapeuten* 2004; 8: 25–30.
- Brooks VB. *The Neural Basis for Motor Control*. Oxford: Oxford University Press, 1986.
- Brown LA, Shumway-Cook A, Woollacott MH. Attentional demands and postural recovery. *J Gerontol* 1999; 54A: M165–171.
- Bussel B, Roby-Brami A, Neris OR, Yakoleff A. Evidence for a spinal stepping generator in man. Electrophysiological study. *Acta Neurobiol Exp* 1996; 56: 465–468.
- Caillet R. *The Shoulder in Hemiplegia*. New York: FA Davies, 1980.
- Canning CG, Ada L, Adams R, O'Dwyer NJ. Loss of strength contributes more to physical disability than loss of dexterity. *Clin Rehabil* 2004; 18: 300–308.
- Casadio M, Morasso P, Sanguineti V, Giannoni P. Impedance-controlled, minimally-assistive robotic training of severely impaired hemiparetic patients. Proceedings of the 1st IEEE/RAS-EMBS International Conference "Biomedical Robotics and Biomechatronics", February 20–22, 2006, Pisa (Italy), art. 227.
- Chen PT, Liaw MY, Wong MK, Tang FT, Lee MY, Lin PS. The sit-to-stand movement in stroke patients and its correlation with falling. *Arch Phys Med Rehabil* 1998; 79: 1043–1046.
- Cheng PT, Chen CL, Wang CM, Hong WH. Leg muscle activation patterns of sit-to-stand movement in stroke patients. *Am J Phys Med Rehabil* 2004; 83: 10–16.
- Childers MK, Stacy M, Cooke DL, Stonnington HH. Comparison of two injection techniques using botulinum toxin in spastic hemiplegia. *Am J Phys Med Rehabil* 1996; 17: 462–469.
- Cornall C. Self propelling wheelchairs: the effect on spasticity in hemiplegic patients. *Physiother Theory Pract* 1991; 7: 13–21.
- Cromwell SJ, Paquette ML. The effect of botulinum toxin A on the function of a person with poststroke quadriplegia. *Phys Ther* 1996; 74: 395–402.
- Davies PM. *Steps to Follow*. Berlin: Springer-Verlag, 1985.
- Davies PM. Skridt for skridt. 2nd ed. FADL, 2001
- Di Fabio RP, Emasithi A, Paul S. Validity of visual stabilization conditions used with computerized dynamic platform posturography. *Acta Otolaryngol* 1998; 118: 449–454.
- Dickstein R, Shefi S, Marcovitz E, Villa Y. Anticipatory postural adjustments in selected trunk muscles in poststroke hemiparetic patients. *Arch Phys Med Rehabil* 2004; 85: 261–267.
- Dietz V. Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiol Rev* 1992; 72: 33–69.
- Edwards S. *Neurological Physiotherapy, a problem based approach*. 1st ed. Edinburgh: Churchill Livingstone, 1996.
- Encyclopedia and Dictionary of Medicine, Nursing and Allied Health. 5th ed. Philadelphia: WB Saunders, 1992.
- Feys HM, De Weerd WJ, Selz BE, et al. Effect of a therapeutic intervention for the hemiplegic upper limb in the acute phase after stroke. *Stroke* 1998; 29: 785–792.
- Gentile AM. Skill acquisition: action, movement and neuromotor processes. In: Carr JH, Shephard RB, Gordon J, Gentile AM, Held JM, eds. *Theoretical implications for therapeutic intervention. I: Movement science—Foundations for physical therapy and rehabilitation*. Rockville, MD: Aspen Publishers, 1987: 93–154.
- Geurts ACH, Mulder TW, Nienhuis B, Rijken RAJ. Influence of orthopedic footwear on postural control in patients with hereditary motor and sensory neuropathy. *J Rehabil Sci* 1992; 5: 3–9.
- Geurts ACH, Visschers BAJT, van Limbeek J, Ribbers GM. Systematic review of aetiology and treatment of post-stroke hand oedema and shoulder-hand syndrome. *Scand J Rehabil Med* 2000; 32: 4–10.
- Gjerstad L, Kerty E, Nyberg-Hansen R. Behandling av focale dystonier med botulinumtoxin. *Tidsskr Nor Lægeforen* 1991; 111: 2637–2639.
- Harrison MA. *Physiotherapy in stroke management*, Edinburgh: Churchill Livingstone, 1995.
- Held JM. Recovery of function after brain damage: Theoretical implications for recovery of function. In: Carr JH, Shephard RB, Gordon J, Gentile AM, Held JM, eds. *Theoretical implications for therapeutic intervention. I: Movement science—Foundations for physical therapy and rehabilitation*. Rockville, MD: Aspen Publishers, 1987: 155–177.
- Horak F, Henry S, Shumway-Cook A. Postural perturbations: new insight for treatment of balance disorders. *Phys Ther* 1997; 5: 517–533.
- Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. *J Neurophysiol* 1986; 55: 1369–1381.
- Hsieh CL, Sheu CF, Hsueh IP, Wang CH. Trunk control as an early predictor of comprehensive activities of daily living function in stroke patients. *Stroke* 2002; 33: 2626–2630.

- Hunter M, Hoffman M. Postural control, visual and cognitive manipulations. *Gait Posture* 2001; 13: 41–48.
- ICF. Internasjonal klassifikasjon av funksjon, funksjonshemming og helse. Sosial og helsedirektoratet. Trondheim: Aktietrykkeriet 2001.
- Jacobs T, Muller JAA, Schultz AB. Trunk position sense in the frontal plane. *Exp Neurol* 1985; 90: 129–138.
- Jeka JJ. Light touch as a balance aid. *Phys Ther* 1997; 77: 476–487.
- Jeka JJ, Lackner JR. Fingertip contact influences human postural control. *Exp Brain Res* 1994; 100: 495–502.
- Karnath HO, Ferber S, Dichgans J. The origin of contraversive pushing. Evidence of a second graviceptive system in humans. *Neurology* 2000a; 55: 1298–1304.
- Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. *Proc Natl Acad Sci USA* 2000b; 97: 13031–13036.
- Karnath HO, Brötz D, Götz A. Klinik, ursache und therapie der pusher-symptomatik. *Der Nervenarzt* 2001; 2: 86–92.
- Kavounoudias A, Roll R, Roll JP. The plantar sole is a "dynamometric map" for human balance control. *Neuroreport* 1998; 9: 3247–3252.
- Kerty E, Stien R. Behandling av spastisitet med botulinum toxin. *Tidsskr Nor Lægeforen* 1997; 14: 2022–2024.
- Kidd G, Lawes N, Musa I. Understanding Neuromuscular Plasticity London: Edward Arnold, 1992.
- Kim Y-H, Park J-W, Ko M-H, Jang S-H, Lee PKW. Plastic changes of motor network after constraint-induced movement therapy. *Yonsei Med J* 2004; 2: 241–246.
- Kwakkel G, van Peppen R, Wagenaar RC, et al. Effects of augmented exercise therapy time after stroke: a meta-analysis. *Stroke* 2004; 35: 2529–2539.
- Lee MY, Wong MK, Tang FT, Cheng PT, Lin PS. Comparison of balance responses and motor patterns during sit-to-stand task with functional mobility in stroke patients. *Am J Phys Med Rehabil* 1997; 76: 401–410.
- Leivseth G, Torstensson J, Reikerås O. Effect of passive muscle stretching in osteoarthritis of the hip. *Clin Sci* 1989; 76: 113–117.
- Liepert J, Bauder H, Miltner WHR, Taub E, Weiller, C. (2000). Treatment-induced massive cortical reorganization after stroke in humans. *Stroke* 2000; 31: 1210–1216.
- Lin K-C, Cermak SA, Kinsbourne M, Trombly CA. Effect of left-sided movements on line bisection in unilateral neglect. *J Int Neuropsychol Soc* 1996; 2: 404–411.
- Ljunggren AE Fötter. Enkle tanker omkring fotens biomekanikk. *Fysioterapeuten* 1984; 51: 644–650.
- Lösslein H, Kolster F. Posturaler hemineglect—Neubewertung des pushersyndroms und vorschläge der therapie. *Krankengymnastik* 2001, 1.
- Lum PS, Burgar CG, Shor PC, Majmundar M, Van der LM. Robot-assisted movement training compared with conventional therapy techniques for the rehabilitation of upper-limb motor function after stroke. *Arch Phys Med Rehabil* 2002; 83: 952–959.
- Luria ARH. En introduksjon til nevropsykologien. Nytt Nordisk Forlag: Arnold Busck, 1989.
- Macaluso A, Vito G De. Muscle strength, power and adaptations to resistance in older people. *Eur J Appl Physiol* 2004; 91(4): 450–472. Epub 2003.
- MacKay-Lyons M. Central pattern generation of locomotion: a review of the evidence. *Phys Ther* 2002; 82: 69–83.
- Magnusson M, Johansson K, Johansson BB. Sensory stimulation promotes normalisation of postural control after stroke. *Stroke* 1994; 25: 1176–1180.
- Maki EB, McIlroy WE, The role of limb movements in maintaining upright stance: the 'change-in-support' strategy. *Phys Ther* 1997; 77: 488–507.
- Massion J. Movement, posture and equilibrium. *Prog Neurobiol* 1992; 38: 35–56.
- Massion J. Postural control system. *Curr Opin Neurobiol* 1994; 4: 877–887.
- Massion J, Woollacott MH. Posture and equilibrium. In: Bronstein AM, Brandt T, Woollacott M, editors. *Clinical Disorders of Balance, Posture and Gait*. London: Arnold, 1996.
- Mayer PF, Oddson LIE, De Luca SJ. The role of plantar cutaneous sensation in unperturbed stance. *Exp Brain Res* 2004; 156: 505–512.
- Mayston M. Problem solving in neurological physiotherapy. In: Edwards S, editor. *Neurological Physiotherapy. A problem Solving Approach*. Edinburgh: Churchill Livingstone, 2001.
- Morningstar MW, Pettibon BR, Schlappi H, Schlappi M, Ireland TV. Reflex control of the spine and posture: a review of the literature from a chiropractic perspective. *Chiropr Osteopathy* 2005; 13: 16–33.
- Morris ME, Summers JJ, Matyas TA, Iansek R. Current status of the motor program. *Phys Ther* 1994; 74: 738–752.
- Moseley A, Stark A, Cameron I, Pollock A. Treadmill training and body weight support for walking after stroke. *Cochrane Database Syst Rev* 2003; (3): CD002840. Update in: *Cochrane Database Syst Rev* 2005; (4): CD002840.
- Mudge S, Rochester L. Neurophysiological rationale of treadmill training: evaluating evidence for practice. *NZ J Physiother* 2001; 2: 6–15.
- Muir GD, Steeves JD. Sensorimotor stimulation to improve locomotor recovery after spinal cord injury. *Trends Neurosci* 1997; 20: 72–77.
- Mulder T. A process-oriented model of human behavior. *Phys Ther* 1991; 71: 157–164.
- Mulder T. Current ideas on motor control and learning: implications for therapy. In: Illis LS, editor. *Spinal Cord Dysfunction. Intervention and Treatment*.

- ment, Vol. II. Oxford: Oxford University Press, 1992: 187.
- Mulder T, Pauwells J, Nienhuis B. Motor recovery following stroke: towards a disability-oriented assessment of motor dysfunctions. In: Harrison M, editor. *Physiotherapy in Stroke Management*. Edinburgh: Churchill Livingstone, 1995: 275–282.
- Mulder T, Nienhuis B, Pauwels J. The assessment of motor recovery: a new look at an old problem. *J Electromyogr Kinesiol* 1996; 6: 137–145.
- Musa I. The role of afferent input in the reduction of spasticity; an hypothesis. *Physiotherapy* 1986; 72: 179–182.
- Nashner LM. Adaptation of human movement to altered environments. *TINS* 1982; 358–361.
- Nawoczenski DA, Satzman CL, Cook TM. The effect of foot structure on the three-dimensional kinematic coupling behavior of the leg and rear foot. *Phys Ther* 1998; 78: 404–416.
- Nelles G. Cortical reorganisation—effects of intensive therapy. *Restorative Neurol Neurosc* 2004; 22: 239–244.
- Normann B. Individualisering i nevrologisk fysioterapi. Bobathkonseptet. Hjerneslagpasienter—behandling og kunnskapsgrunnlag. Hovedfagsoppgave i helsefag, flerfaglig studieretning. Avdeling for sykepleie og helsefag, IKM, Med.fak. Universitetet i Tromsø 2004.
- Nudo RJ, Wise BM, SiFuentes F, Milliken GW. Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. *Science* 1996; 272: 1791–1794.
- O'Brian CF. Overview of clinical trials and published reports of botulinum toxin for spasticity. *Eur J Neurol* 1997; 4(Suppl. 2): S11–S13.
- Paillard J. Basic neurophysiological structures of eye-hand coordination. In: Williams HG, editor. *Growth, Motor Development and Physical Activity Across the Life-span*. Columbia: University of South Carolina Press, 1990: 26–74.
- Paillard J. Body schema and body image—a double dissociation in deafferented patients. In: Gantchev GN, Mori S, Massion J, editors. *Motor control, today and to-morrow*. Sofia, Bulgaria: Drinov Academic Publishing House, 1999: 197–214.
- Pandyan AD, Gregoric M, Barnes MP, et al. Spasticity: Clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005; 27: 2–6.
- Patten C, Lexell J, Brown HE. Weakness and strength training in persons with poststroke hemiplegia. Rationale, method and efficacy. *J Rehabil Res Dev* 2004; 41; 3A: 293–312.
- Perennou DA, Leblond C, Amblard B, Micallet JP, Rouget E, Pelissier JY. The polymodal sensory cortex is crucial for controlling lateral stability. Evidence from stroke. *Brain Res Bull* 2000; 53: 359–365.
- Prince F, Winter DA, Stergiou P, Walt SE. Anticipatory control of upper body balance during human locomotion. *Gait Posture* 1994; 2: 19–25.
- Robertson IH, Halligan PW, Marshall JC. Prospects for the rehabilitation of unilateral neglect. In: Robertson IH, Marshall JC, editors. *Unilateral neglect: clinical and experimental studies*. Hove, UK: Lawrence Erlbaum Ltd., 1993: 279–292.
- Robertson IH, Hogg K, McMillan TM. Rehabilitation of unilateral neglect: improving function by contralateral limb activation. *Neuropsychol Rehabil* 1998; 8: 19–29.
- Rosenbaum DA. *Human Motor Control*. New York: Academic Press Inc., 1991.
- Rothwell J. *Control of Human Voluntary Movement*. London: Chapman & Hall, 1994.
- Sahrmann SA. Posture and Muscle Imbalance. *Physiotherapy* 1992; 78: 1.
- Sahrmann SA. *Diagnosis and Treatment of Movement Impairment Syndromes*. Edinburgh: Mosby, 2002.
- Schäger ST, Kool JP. Pushen: Syndrom oder Symptom?—Eine Literaturübersicht. *Krankengymnastik* 2001: 1.
- Schleichkorn J. *The Bobaths. A Biography of Berta and Karel Bobath*. USA: Therapy Skill Builders, 1992.
- Schmidt RA. Motor learning principles for physical therapy. In: Listen MJ, editor. *Contemporary Management of Motor Control Problems. Proceedings of the II-Step Conference*, 1991: 49–63. Alexandria, VA: Foundation for Physical Therapy.
- Schultz C. Massasjens fysiologiske virkninger. *Fysioterapeuten* 1997; 4: 12–15.
- Shumway-Cook A, Woollacott M. *Motor Control. Translating Research into Clinical Practice*. 3rd ed. Philadelphia: Lippincott Williams and Wilkins, 2006.
- Slijper H, Latash M. The effects of instability and additional hand support on anticipatory postural adjustments in leg, trunk, and arm muscles during standing. *Exp Brain Res* 2000; 135: 81–93.
- Smedal T, Gjelsvik B, Lygren H, Borgmann R, Waje-Andreasen U, Grønning M. Botulinum toxin A and effect on spasticity. *Tidsskr Nor Lægeforen* 2001; 121: 3277–3280.
- Smedal T, Lygren H, Myhr K-M, et al. Balance and gait improved in patients with MS after physiotherapy based on the Bobath concept. *Physiother Res Int* 2006; 11(2): 104–116.
- Smidt GL. *Gait in Rehabilitation. Clinics in Physical Therapy*. Edinburgh: Churchill Livingstone, 1990.
- Sunderland A, Tinson DJ, Bradley EL, Fletcher D, Langton Hewer R, Wade DT. Enhanced physical therapy improves recovery of arm function after stroke. *J Neurol Neurosurg Psychiatry* 1992; 55: 530–535.
- Taber's Cyclopedic Medical Dictionary. 18th ed. Thomas CL: FA Davies Company, 1997.
- Taub E, Uswatte G, Pidikiti R. *Constraint-Induced Movement Therapy: A new family of techniques with broad application to physical rehabilitation—*

- a clinical review. *J Rehabil Res Devel* 1999; 36: 237–251.
- Taylor BA, Ellis E, Haran H. The reliability of measurement of postural alignment to assess muscle tone change. *Physiotherapy* 1995; 81: 485–490.
- Thornquist FE. Kroppens spennende samspill Fysioterapeuten 1984; 51: 636–643.
- Umphred D. Merging neurophysiologic approaches with contemporary theories. In: Listen MJ, editor. *Contemporary Management of Motor Control Problems. Proceedings of the II-Step Conference 1991:* 127–130. Alexandria, VA: Foundation for Physical Therapy.
- Virji-Babul N. Effects on post-operative environment on recovery of function following brain damage: a brief literature review. *Physiotherapy* 1991; 77: 587–590.
- Wade M, Jones G. The role of vision and spatial orientation in the maintenance of posture. *Phys Ther* 1997; 77: 619–628.
- Whiting J, Vereijken B. The acquisition of coordination in skill learning *International J Sport Psychol* 1993; 343–357.
- Whittle MW. Gait analysis, an introduction. 2nd ed. Oxford: Butterworth-Heinemann, 1996.
- WHO. International Classification of Functioning, Disability and Health (ICF). Geneva. 2006 (<http://www.who.int/classifications/icf/en/>).
- Winstein C, Wing AM, Whitall J. Motor control and learning principles for rehabilitation of upper limb movements after brain injury. In: Grafmann J and Robertson LH, editors. *Handbook of Neuropsychology*, 2nd ed. Vol. 9. Edinburgh: Elsevier Science 2003: 77–137.
- Yavuzer G, Ergin S. Effect of an arm sling on gait pattern in patients with hemiplegia. *Am Phys Med Rehabil* 2002; 83: 960–963.
- Yekutiel M, Guttman E. A controlled trial of the retraining of the sensory function of the hand in stroke patients *J Neurol Neurosurg Psychiatry* 1993; 56: 241–244.
- Zoltan B, Siev E, Freishtat B. *The Adult Stroke Patient: A Manual for Evaluation and Treatment of Perceptual and Cognitive Dysfunction*, 2nd ed. New Jersey: Slack Inc., 1991.

Chapter 3 Assessment

- American College of Sports Medicine (ARCM). Resource manual for guidelines for exercise testing and prescription. Philadelphia: Lea and Febiger, 1988.
- Benaim C, Perennou DA, Villy J, Rousseaux M, Pelissier JY. Validation of a postural assessment scale for stroke (PASS). *Stroke* 1999; 30: 1862–1868.
- Berg K, Wood-Dauphinee S, Williams JI, Gayton D. Measuring balance in the elderly: preliminary development of an instrument. *Physiother Can* 1989; 41: 304–11.
- Berg K, Wood-Dauphinee S, Williams JI, Maki B. Measuring balance in the elderly: validation of an instrument. *Can J Public Health* 1992; 83: 7–11.
- Berg K, Wood-Dauphinee S, Williams JI. The Balance scale: reliability assessment with elderly residents and patients with an acute stroke. *Scand J Rehabil Med* 1995; 27: 27–36.
- Bergland A. Bergs balanseskala. Postural kontroll–balanse. Kompendium Undersøkelser–skalaer. MSc Thesis 1999a: 76–80 (e-mail address: astrid.bergland@hf.hio.no).
- Bergland A. Functional reach. Postural kontroll–balanse. Kompendium Undersøkelser–skalaer. MSc Thesis 1999b: 35–38 (e-mail address: astrid.bergland@hf.hio.no).
- Bergland A. Et-bens stående. Postural kontroll–balanse. Kompendium Undersøkelser–skalaer. MSc Thesis 1999c: 27–30 (e-mail address: astrid.bergland@hf.hio.no).
- Bergland A. Timed up and go. Postural kontroll–balanse. Kompendium Undersøkelser–skalaer MSc Thesis. 1999d: 57–60 (e-mail address: astrid.bergland@hf.hio.no).
- Bilney B, Morris M, Webster K. Concurrent related validity of the GAITRite walkway system for Quantification of the spatial and temporal parameters of gait. *Gait Posture* 2003; 17: 68–74.
- Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehabil Med* 1970; 2–3: 92–98.
- Buckworth J, Dishman RK. Perceived exertion. In: Biddle SJH, Mutrie N. *Exercise psychology*. Champaign, IL: Human Kinetics, 2002: 256–84.
- Enright PL. The six-minute walk test. *Respir Care* 2003; 48: 783–785.
- Finch E. *Physical rehabilitation outcome measures: a guide to enhanced clinical decision-making*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2002.
- Geurts ACH, Visschers BAJT, Limbeek J van, Ribbers GM. Systematic review of aetiology and treatment of post-stroke hand oedema shoulder–hand syndrome. *Scand J. Rehabil Med* 2000; 32: 4–10.

- Guyatt GH, Townsend M, Pugsley O. Bronchodilators in chronic air-flow limitation. *Am Rev Respir Dis* 1987; 135: 1069–1074.
- Hesse S, Jahnke MT, Schaffrin A, Lucke D, Reiter F, Konrad M. Immediate effects of therapeutic facilitation on the gait of hemiparetic patients as compared with walking with and without a cane. *Electroencephal Clin Neurophysiol* 1998; 109: 515–522.
- Hsieh CL, Sheu CH, Hsueh IP, Wang CH. Trunk control as an early predictor of comprehensive activities of daily living function in stroke patients. *Stroke* 2002; 33: 2626–2630.
- ICF. The International Classification of Functioning, Disability and Health. Geneva: WHO 2006 (<http://www.who.int/classifications/icf/en/>).
- ICF. International Classification of Functioning, Disability and Health by the World Health Organization. Endorsed by the Fifty-fourth World Health Assembly for international use on May 22, 2001 (resolution WHA54.21).
- ICIDH-2. International Classification of Functioning and Disability. Beta-2 Draft, Short Version. Geneva: WHO, 1999.
- Kaasa S, Lode JH. Patient evaluation and outcome measures; quality of life in palliative medicine—principles and practice. In: Doyle D, Hanks GWC, MacDonald N, editors. *Oxford Textbook of Palliative Medicine*. 3rd ed. Oxford: Oxford University Press, 2004: 196–210.
- Kavounoudias A, Roll R, Roll JP. The plantar sole is a 'dynamometric map' for human balance control. *Neuroreport* 1998; 9: 3247–3252.
- Lacasse Y, Wong E, Guyatt GH, King D, Cokk DJ, Goldstein RS. Meta-analysis of respiratory rehabilitation in chronic obstructive pulmonary disease. *Lancet* 1996; 348: 1115–1119.
- Lennon S. Gait re-education based on the Bobath Concept in two patients with hemiplegia following stroke. *Phys Ther* 2001; 81: 924–935.
- Lie I. Rehabilitering og habilitering. Ad Notam Gyldendal, 1998.
- Lord SE, Halligan PW, Wade DT. Visual gait analysis: the development of a clinical assessment and scale. *Clin Rehabil* 1998; 12: 107–119.
- Lord SR, Menz HB. Physiologic, psychologic, and health predictors of 6-minute walk performance in older people. *Arch Phys Med Rehabil* 2002; 83: 907–911.
- Malterud K. Qualitative research: standards, challenges, and guidelines. *Lancet* 2001a; 358: 483–488.
- Malterud K. The art and science of clinical knowledge: evidence beyond measures and numbers. *Lancet* 2001b; 358: 397–400.
- Mayer PF, Oddson LI, De Luca CJ. The role of plantar cutaneous sensation in unperturbed stance. *Exp Brain Res* 2004; 156: 505–512.
- Moe-Nilssen R. A new method for evaluating motor control in gait under real-life environmental conditions. Part 2: Gait analysis. *Clin Biomech* 1998; 13: 328–335.
- Monaghan J, Channell K, McDowell D, Sharma AK. Improving patient and carer communication, multidisciplinary team working and goal-setting in stroke rehabilitation. *Clin Rehabil* 2005; 19: 194–199.
- Podsiadlo D, Richardson S. The timed "Up&Go": a test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc* 1991; 39: 142–48.
- Rehabilitering. Et liv i verdighet. Sosial-og helsedepartementet (I-0863 B og N).
- Rehabiliteringsmagasinet Bris. Rikstrygdeverket, 1 (1998).
- Schleichkorn J. The Bobaths. A biography of Berta and Karel Bobath. USA: Therapy Skill Builders, 1992.
- Shumway-cook A, Woollacott MH. Motor Control: Theory and Practical Applications. Philadelphia: Lippincott Williams and Wilkins; 2001.
- Solway S, Brooks D, Lacasse Y. A qualitative systematic overview of the measurement properties of functional walk tests used in the cardiorespiratory domain. *Chest* 2001; 119: 256–270.
- Thorban LDB, Newton RA. Use of Berg Balance Scale to predict falls in elderly persons. *Phys Ther* 1996; 76: 576–82.
- Troll i Ord. Brukermedvirkning på alvor. Kommuneforlaget, 1995.
- Verheyden G, Nieuwboer A, Mertin J, Preger R, Kiekens C, De Weerd W. The trunk impairment scale: a new tool to measure motor impairment of the trunk after stroke. *Clin Rehabil* 2004; 18: 326–334.
- Verheyden G, Nieuwboer A, Feys H, Thijs V, Vaes K, De Weerd W. Discriminant ability of the trunk impairment scale: a comparison between stroke patients and healthy individuals. *Disabil Rehabil* 2005; 27: 1023–1028.

Index

Page references in *italics*
refer to illustrations only.

A

- active movement 124–125
 - facilitation 126
- activities of daily living (ADLs) 70, 71–72
 - assessment 148
 - transfer of treatment to 130–131
 - *see also* assistive devices
- activity measures 163–164
- adaptation 73
 - *see also* plasticity
- agnosia, finger 154
- air cast 138
- akinesia 40, 41
- alignment, ideal 10, 19, 90
- amyotrophic lateral sclerosis 126
- ankle
 - orthoses 137–138, 138
 - taping 82, 83–84
- anterograde axonal transport 50
- anterolateral system 13
- anticipatory adjustment of muscles (feedforward) 18, 19, 73
- arm sling 139, 140
- ascending reticular activating system (ARAS) 29
- assessment 146–162
 - aim of 157–160
 - assessment diagram 164–165, 174, 200
 - balance or movement-based problems 157–159
 - body functions and structures 150–153, 156
 - case histories 169–174, 195–200
 - clinical reasoning 156–157, 174–176, 200–201
 - cognitive deficits 160, 169
 - documentation 165
 - functional activity 148–150, 169
 - history 147
 - learned nonuse 155
 - pain 155–156
 - perception 154–155
 - sensation 153–154
 - somatosensory/perceptual dysfunctions 159–160
- assistive devices 134–140
 - adaptation 140
 - evaluation 140
 - orthoses 137–140
 - timing 135

- walking aids 136–137, 150
- wheelchairs 135–136
- associated reactions 60–63, 64, 118, 126, 164
 - assessment 153, 159
 - control of 126–127
 - grading of 62–63
 - inhibition 118
 - mechanisms 61–62
- astrocytes 52–53
- ataxia 38
- autonomic function changes 155
- axonal transport 50

B

- baclofen 142
- balance 18–20, 68–69, 81
 - ankle strategy 69
 - deviations from normal balance control 80–88
 - hip strategy 69
 - improvement 81
 - problems 38, 114, 158
 - assessment 157–159
 - protective reactions and strategies 78–79
 - vestibular system and 25–27
- see also* postural control
- basal ganglia 38–41, 39, 40
- base of support 72, 73
 - evaluation 75
- basic postures 90
 - sitting 94, 96–97, 96
 - standing 90, 91
- see also* postural sets
- basket cells 35
- Berg Balance Scale (BBS) 163
- Bobath balls 120
- Bobath, Berta 1
- Bobath Concept 1, 89
- Bobath, Karel 1
- body schema 19, 75
- body structure and function measures 162–163
- botulinum toxin A 141–142
- bradykinesia 40–41
- brain stem 21, 25–27
- brain-derived neurotrophic factor (BDNF) 49
- Busse, Berta 1

C

- carry-over 129–131
- generalization 129
- learning or retention 130
- performance 130
- transfer from treatment to activities of daily living 130–131
- caudate nucleus 38
- central key area 107–108
- central motor programming 18
- central pattern generators (CPGs) 23–25
- cerebellar circuit 35
- cerebellar cortex 35–36
- cerebellar nuclei 35
- cerebellum 33–38, 33
 - functions 36–38
- cerebral cortex 30
 - plasticity 51–52
 - somatosensory areas 16, 16
 - somatotopic organization 15, 20
- cerebral palsy case history 194–216
- cerebrocerebellum 35, 35, 38
- circulatory changes 155
- classical conditioning 48
- climbing fibers 35, 36
- clinical reasoning 156–157
 - assessment diagram 164–165, 174, 200
 - case histories 174–176, 200–201
- cognition 33, 111–112
 - assessment 22, 150, 160
 - case history 169
 - reduced 115, 160
 - walking control 113–114
 - collateral sprouting 50–51, 50
 - communication assessment 148
 - compartmentalization 8, 12
 - compensatory strategies 80–84, 164–165
 - assessment 153, 159
 - case histories 175, 201
 - constraint induced movement therapy (CIMT) 55, 133–134
 - contractures 64
 - contraversive pushing 84
 - see also pusher syndrome
 - cortex *see* cerebellar cortex; cerebral cortex
 - cortico-rubrospinal system 31–33
 - corticobulbar tract 30, 30
 - corticopontine tract 34
 - corticoreticular tract 28, 29
 - lesions 60
 - corticospinal tract 29, 30–31, 30
 - crutches 136

D

- degrees of freedom problem 74
- deltoid muscle 7
- denervation supersensitivity 53
- dentate nucleus 34–35
- descending pathways 25, 29
- dexterity 75
 - loss of 57–58
- diaschisis 52
- distal key areas 108–109
- disuse atrophy 52
 - *see also* learned nonuse
- documentation 165
- dorsal column medial lemniscus system 13, 14
- dressing 95
 - assessment 149
- drill 129
- dual task 112
- dyskinesia 41
- dysmetria 38
- dystonia 41

E

- eating/drinking assessment 149
- elbow 108–109
- emboliform nucleus 35
- equilibrium reactions/control 70
- exploratory behavior 124
- eyesight *see* vision

F

- facilitation 118–124
 - active movement 126
 - assessment 153
 - stepping 120–124, 121–122, 123
 - fastigial nucleus 34
 - fatigue 58
 - feedback 19, 24, 73, 81, 127–129
 - external sensory 18, 128–129
 - handling and 127, 128
 - internal 18, 127, 128
 - feedforward (anticipatory adjustment of muscles) 18, 19, 73
 - finger discrimination 154
 - foot 73, 108
 - heel-off 16, 37, 82
 - heel-strike 16, 24, 37, 82, 131–132
 - hypersensitivity 86–88
 - orthoses 137–138
 - reduced adaptability 88
 - form–function concept 46
 - functional activity
 - assessment 148–150, 169, 195
 - case histories 169, 193, 195
 - Functional Reach (FR) 163

G

- GAITRite 163
- gastrocnemius muscle 6
- gene expression, plasticity and 48–49
- genotype 49
- glia 52
- globose nucleus 35
- globus pallidus 38
- gnosis, finger 154
- goals
 - objective goal setting 164
 - of physiotherapy 67, 146
- Golgi cells 35
- Golgi tendon organ 9, 10, 61, 75
- granule cell 35
- grasp 77
- graviceptors 75
- gravity 19, 71–72, 81
 - line of 152
- growth associated protein (GAP) 49, 51

H

- habituation 48
- handling 115–118
 - active movement 124–125
 - appropriate 115
 - assessment 146–147, 151
 - facilitation 118–124
 - feedback and 127, 128
 - learned nonuse 125
 - neglect 125–126
 - passive movement 126
- hands 108
 - cramp, occupational 41
- hands-off stage 120
- head 26–27, 108
- heel-off 16, 37, 82
- heel-strike 16, 24, 37, 82, 131–132
- hemianopia 18, 86
- hemiplegia 29, 33
- Hennemans recruitment principle 7, 81
- hip
 - abductors 110–111
 - extension 158
 - stability training 129
- hippocampus 52
- history taking 147
 - case histories 168–169, 194
- Huntington chorea 41
- hyperextension, knee 86, 140, 158
- hypersensitivity 32, 54, 86–88
- hypertonia 11–12, 153
- hypotonia 11, 38, 41, 118–119, 153

I

- ideal alignment 10, 19, 90
- impaired joint individuation 8
- incoordination 38
- inferior olive 34, 36
- inhibition 41–44, 118
 - lateral 21, 21
 - postsynaptic 43
 - presynaptic 42–43, 42, 43, 61
 - recurrent 43–44, 61
- instrumental activities of daily living (IADLs) 70
 - assessment 148
- International Bobath Instructors Training Association (IBITA) 1–2
 - mission 2
- International Classification of Functioning, Disability, and Health (ICF) 144–146
 - sections 145, 149–150
- interosseous muscles 6, 7

J

- joint position sense 21
 - assessment 154

K

- key areas 107–111
 - assessment 151–152
 - central 107–108
 - distal 108–109
 - proximal 108
- knee 108–109
 - hyperextension 86, 140, 158
 - orthoses 140
- knowledge of performance (KP) 128
- knowledge of results (KR) 128–129

L

- lateral inhibition 21, 21
- latissimus dorsi muscle 12, 12
- learned nonuse 22, 52, 55, 125, 159
 - assessment 155, 160
- learning 47, 52, 53–55, 66–67, 80
 - motor learning 36, 38, 112–113
 - retention 130
- long-term depression (LTD) 36
- long-term potentiation (LTP) 48
- lumbricals 6
- lying *see* side-lying postural sets; supine

M

- medical history 147
- medical treatment, spasticity 140–142
- Meissner corpuscles 20
- memory 48
 - explicit (declarative) 48
 - implicit (nondeclarative) 48
- Merkel disks 20
- mesencephalic locomotor region (MLR) 24
- midline 79
 - control 79
 - orientation 151
- mossy fibers 35, 36
- motivation 67, 136
- motor cortex 18
- motor program storage 37
- motor skills training 52
- motor units 7–8, 7
 - recruitment 7–8, 81
 - active 124–125
 - assessment 148, 150–153, 157–159
 - control 67–68
 - automatic and voluntary 111–115
 - selective 44, 152, 158
 - deviations from normal movement 80–88
 - dyssynergic patterns 60
 - facilitation 118–124
 - functional activity 109–111
 - passive 126
 - patterns of 76–77, 76, 152
 - changes in 156
 - peripheral stimulation influence 116
 - quality of 151–153
 - righting 77–78, 78–79, 80
 - vision role 17–18
 - *see also* postural control; stepping; walking
- multidisciplinary teamwork 134, 144
- muscle *see* skeletal muscle
- muscle balance 8
- muscle fibers 5–8
 - contraction 6–8
 - intrafusal 9
 - pathological changes 12–13, 63–64
 - type 1 (ST) 5–6
 - type 2 (FT) 6
 - type 2B (FG) 6
 - use-dependent plastic adaptation 6
- *see also* skeletal muscle
- muscle imbalance 8
- muscle spindles 9–10, 9, 75
- muscle tone 10–13
 - assessment 153
 - changes in 156
 - stiffness 10
- *see also* postural control; skeletal muscle

N

- neck 108
- negative signs 57–58
- neglect 125–126
- nerve cell regeneration 52–53
- nerve growth factor (NGF) 49
- neuromuscular system 5–13
- neuroplasticity *see* plasticity
- neurotrophic factors 49
- non-neural changes 63–64
- noninvasive imaging techniques 46, 47

O

- objective goal setting 164
- observation 148, 150
 - *see also* assessment
- occupational hand cramp 41
- opisthotone 12
- orthoses 137–140, 137
 - ankle/foot 137–138, 138
 - knee 140
 - shoes 137
 - shoulder 140
- outcome measures 162–165
 - activity measures 163–164
 - assessment diagram 164–165, 174, 200
 - body structure and function measures 162–163
 - self-report measures 164
- over-activity 57
- overlearning 113
- overstretch weakness 7, 13

P

- Pacinian corpuscle 20
- pain 155
 - autonomic function 155
 - clinical relevance 155–156
 - possible causes 155
- paralysis 55, 56, 80, 86, 126
- paresis 55, 56, 80
- Parkinson disease 41, 84
- passive movement 126
- patterns of movement 76–77, 76
- pelvic girdle 108
- pelvic tilt 103, 110, 110, 111
- perception 33, 124
 - assessment 22, 150, 154–155, 159–160
 - reduced 86, 115
- personal hygiene assessment 149
- phenotype 49
- physiotherapy
 - assessment 146–162
 - case histories 175–193, 203–215
 - goals 67, 146
- placing 119

plasticity 46–55
 – associated reactions and 62
 – clinical relevance 53–55
 – collateral sprouting 50–51, 50
 – cortical maps 51–52, 80
 – functional 47–48
 – gene expression and 48–49
 – muscle fibers 6
 – nerve cell regeneration 52–53
 – neurotrophic factors 49
 – structural 48
 plateau potentials 59
 pontocerebellum 34, 34
 positive signs 58–59
 post-stroke hand edema 156
 postsynaptic inhibition 43
 Postural Assessment Scale for Stroke Patients (PASS) 163
 postural control 18–20, 70–71, 80, 112–115
 – assessment 158
 – dynamic characteristics 74
 – function 71
 – righting and 78–79
 – stability 74–75
 – tone 10, 71–77, 158
 – *see also* balance
 postural hemineglect 84
 postural sets 89–107
 – side-lying 104–107, 105–107
 – sitting 96–102, 97–99
 – standing 90–96, 92–93
 – supine 102–104, 102–103
 postures *see* basic postures; postural sets
 presynaptic inhibition 42–43, 42, 43, 61
 principal nucleus 36
 proprioceptors 9–10, 75
 propriospinal fibers 23
 protective reactions and strategies 78–79
 prototypical representation 37
 proximal key areas 108
 Purkinje cells 35, 36, 37
 pusher syndrome 84, 85–86, 85
 putamen 38, 39
 pyramidal tract 18, 30–31

■ Q

quadriceps muscle 8

■ R

Rating Scale of Perceived Exertion (RPE) 164
 reach 77
 receptive field 20
 reciprocal innervation 74, 75
 – changes 156
 recovery 73
 – spontaneous 80

recurrent inhibition 43–44, 61
 red nucleus 31–33, 31
 regeneration, nerve cells 52–53
 rehabilitation 54–55, 67, 144
 Renshaw cell 43–44, 43
 repetitive strain injury 41
 resting tone 10
 retention 130
 reticular formation 27–30, 27
 reticulospinal tracts 25, 28
 – lesions 60
 retrograde axonal transport 50, 51
 righting 77–78, 78–79, 80
 – head 77
 – postural control and 78–79
 – trunk 77
 rigidity 41
 Rivermead Visual Gait Assessment (RVGA) 162–163
 robot training 134
 rotation 69, 77
 rubrospinal tracts 28, 32
 Ruffini terminals 20

■ S

sarcomeres 6–7
 selective control 44, 152, 158
 self-report measures 164
 sensation assessment 153–154, 159–160
 sensory cortex 16, 16
 sensory unit 20
 shoes 137
 short-term potentiation (STP) 48
 shoulder
 – orthoses 140
 – subluxation 140
 shoulder girdle 108
 – assessment 159
 shoulder-hand syndrome 155–156
 side-lying postural sets 104–107, 105–107
 simultaneous activity 112
 simultaneous bilateral touch 154
 simultaneous integration 154–155
 Single Leg Stance (SLS) 163
 sitting 94, 96–97, 96, 119
 – postural sets 97–102, 97–99
 six-minute walking test (6MWT) 164
 skeletal muscle 5–10
 – assessment 152
 – biased recruitment 7
 – compartmentalization 8, 12
 – contraction 6–8, 71
 – motor unit 7–8
 – muscle tone 10–13
 – noncontractile elements 9
 – pathological changes 12–13, 63–64, 118
 – sense organs in 9–10
 – shortened 118

- specific mobilization of 119
- stretch weakness 7, 13
- *see also* muscle fibers; muscle tone; *specific muscles*
- skills 113
 - motor skills training 52
- skin quality 155
- sling, arm 139, 140
- SMART goals 164
- social aspects 147
- soleus muscle 6
- somatosensory system 13–16
 - assessment of dysfunctions 159–160
 - handling and 116
 - sensorimotor integration 16–17
- spastic torticollis 41
- spasticity 58–59, 60, 140–141
 - definition 58–59
 - inhibition 118
 - medical treatment 140–142
 - adverse effects 142
 - baclofen 142
 - botulinum toxin A 141–142
- spatial distribution 43
- spatial summation 43
- specific mobilization of muscle 119
- spinal cord 22–23
 - symptoms of injury 155
- spino-olivo-cerebellar tract 34
- spinocerebellar tracts 13, 15, 33
- spinocerebellum 33–34, 34, 37–38
- spinoreticular tract 13
- spinothalamic tract 13
- splints 138, 138
- stability 74–75, 82
 - hip stability training 129
- stance 77
- standing 19–20, 69, 73, 81, 90, 91
 - generalization 129
 - postural sets 90–96, 92–93
 - therapeutic significance 81–82
 - *see also* balance; postural control
- stellate cells 35
- stepping
 - facilitation of 24–25, 120–124, 121–122, 123
 - *see also* movement; walking
- stereognostic sense 20–22, 116
- strength training 131–132
- stretch weakness 7, 13
- stretching 64
- subjective postural vertical (SPV) 84, 85
- supine
 - hip stability training 129
 - pelvic tilt 103, 110, 110
 - postural sets 102–104, 102–103
- Support Network for the Assembly and Database for Spasticity Measurement (EU-SPASM) 58
- swelling 155

- synaptic connections 10, 11
 - axoaxonic 42, 42
 - formation 50

■ T

- taping 82, 83–84
- tectospinal tracts 25
- temporal distribution 43
- temporal summation 43
- terminal tremor 38
- thalamus 14–15
- therapeutic handling *see* handling
- tibialis anterior muscle 6
- Timed Up and Go (TUG) 163–164
- touch 115–117
 - localization assessment 154
- training 54–55
 - constraint induced movement therapy (CIMT) 55, 133–134
 - hip stability 129
 - motor skills 52
 - strength 131–132
 - treadmill 132–133
- transfer *see* weight transference
- transfer of task 129
- treadmill training 123, 132–133
 - with harness and support 132
- tremor 40, 41
 - terminal 38
- Trunk Impairment Scale (TIS) 162
- 24-hour concept 134
- two-point discrimination 154

■ U

- undressing 100–101
 - assessment 149
- upper motoneuron syndrome 59–60
- upper motor neurons 57
 - lesions 57–59, 131
 - negative signs 57–58
 - positive signs 58–59

■ V

- valium 142
- varied repetition 129
- vestibular nucleus
 - lateral 25–26
 - medial 26–27
- vestibular system 25–27, 75
- vestibulo-ocular reflex (VOR) 27
- vestibulocerebellum 33, 37
- vestibulospinal tracts 25, 26, 28
 - lesions 60
- vision 17–18, 17, 73, 76, 114
 - problems 158

Visual Analogue Scale (VAS) 164
visual problems 38
visual subjective vertical (VSV) 84, 85

■ W

walking 69, 69, 113–114, 120–121
– pattern generation 24
– step cycle 16
– *see also* balance; movement;
postural control; stepping
walking aids 136–137
– assessment 150
– bilateral 136
– one-handed 136–137

weakness 57, 131
– overstretch 7, 13
– strength training 131–132
weight transference 149
– abnormalities 87–88
– assessment 149–151
– *see also* movement; walking
wheelchairs 135–136
– manual 136
– power 136
wrist-hand syndrome 156

■ Z

Zimmer frame 136

