The Bobath Concept in Contemporary Clinical Practice

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Background: Future development in neurorehabilitation depends upon bringing together the endeavors of basic science and clinical practice. The Bobath concept is widely utilized in rehabilitation following stroke and other neurological conditions. This concept was first developed in the 1950s, based on the neuroscience knowledge of those times. **Purpose:** The theoretical basis of the Bobath concept is redefined based on contemporary neuroscience and rehabilitation science. The framework utilized in the Bobath concept for the analysis of movement and movement dysfunction is described. This framework focuses on postural control for task performance, the ability to move selectively, the ability to produce coordinated sequences of movement and vary movement patterns to fit a task, and the role of sensory input in motor behaviour and learning. The article describes aspects of clinical practice that differentiate this approach from other models of practice. Contemporary practice in the Bobath concept utilizes a problem-solving approach to the individual's clinical presentation and personal goals. Treatment is focused toward remediation, where possible, and guiding the individual towards efficient movement strategies for task performance. The aim of this article is to provide a theoretical framework on which future research into the Bobath concept can be based. **Key words:** *neurofacilitation, physiotherapy, occupational therapy, stroke, rehabilitation*

It is more than 50 years since Berta and Karel Bobath proposed what was then a revolutionary new approach to the treatment of adults with lesions of the central nervous system (CNS) based upon recovery as opposed to compensation. This had far-reaching consequences for rehabilitation following stroke, as clinicians moved from simply teaching compensatory strategies with the unaffected side toward facilitating recovery of motor function on the affected side. Rehabilitation following stroke remains a predominant focus in the practice of the Bobath concept. Developments in the Bobath concept in recent years have been disseminated through the teaching of postgraduate courses in neurological rehabilitation, but little information has been published on the evolving theoretical framework and the subsequent influence on clinical practice. This has resulted in a lack of clarity regarding the theory underlying the concept[1] and, more seriously, in clinical studies in which the intervention procedures may not have reflected current practice in the Bobath concept.[2–4] Two recent systematic reviews have concluded that further investigation into the efficacy of the Bobath concept is required.[5,6] This, however, cannot be undertaken until the theoretical framework and clinical implementation have been defined.

The purpose of this article is the following:

- To define the Bobath concept in the treatment of adults with neurological conditions, including stroke, as currently taught by International Bobath Instructors Training Association (IBITA) instructors;

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- To highlight the developments in basic and rehabilitation science that have provided the theoretical framework for contemporary practice; and
- To describe current clinical practice based upon the Bobath concept and to discuss those aspects that differentiate this approach from other models of practice.

Defining the Bobath Concept

The Bobath concept was originally developed and defined in the 1950s. Berta and Karel Bobath acknowledged the need for the concept to remain dynamic and evolve as new neuroscientific evidence became available.[7] Berta Bobath stated that "the Bobath Concept is far-reaching and open, it enables us to go on learning and to follow continuous scientific development."[8]

In 1983, a small group of experienced Bobath instructors proposed the establishment of an international association to facilitate the development of the Bobath concept and the delivery of standardized Bobath clinical courses, consistent with the current neuroscience. With the approval of the Bobaths, the original association was formed in 1984. Since 1996, it has been known as the International Bobath Instructors Training Association (IBITA). Over the last decade, IBITA has updated the theoretical assumptions underlying the Bobath concept on an on-going basis and has published these assumptions on their website.[9]

At the annual general meeting and conference in Leeds in 2005, the IBITA membership commissioned us to prepare an article for publication to describe the contemporary theoretical basis for the Bobath concept. The article has been reviewed and endorsed by the three leading committees of IBITA: the Executive Committee, the Senior Instructors Group, and the Education Committee.

The Bobath concept is currently defined as 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.[9,10] The concept provides a way of observing, analyzing, and interpreting task performance.[11] The clinical implementation of the Bobath concept utilizes an individualized reasoning process rather than a series of standardized techniques.

Four key themes are discussed in this article with respect to the Bobath concept and rehabilitation:

- The Bobath concept and the International Classification of Functioning, Disability and Health (ICF)[12]
- Analysis of human movement with respect to contemporary neuroscience
- Movement dysfunction and recovery following neurological pathology
- Key aspects for clinical practice

The Bobath Concept and the International Classification of Functioning, Disability and Health

The Bobath concept is congruent with the ICF, acknowledging the entirety of human functioning in all spheres of life, as well as the individual nature of each person's problems. Activity limitations are regarded as the outcome of a complex relationship between the individual's health condition, personal factors, and the external factors of the environmental circumstances in which the individual lives.[12]

The structure provided by the ICF has moved the focus of clinicians beyond interventions that are only impairment directed toward enabling the individual to overcome activity and participation restrictions. Participation restrictions are identified in consultation with the individual, the family, and relevant caregivers. The functional goals that are set are those that are relevant and achievable for the individual. Underlying impairments are addressed if they are relevant to the achievement of the required skill. Addressing appropriate functional activities in daily life situations as necessary ensures that contextual factors are taken into consideration and allows the measurement of meaningful outcomes.

Analysis of Human Movement with Respect to Contemporary Neuroscience

The Bobath concept places particular emphasis on two interdependent aspects: the integration of postural control and task performance and the control of selective movement for the production

of coordinated sequences of movement.[9,13,14] These factors are regarded as critical to optimizing motor recovery and function following stroke. In addition, the contribution of sensory inputs to motor control and motor learning has always been and remains a key focus of the Bobath concept.[13]

The organization of human behaviour has been the subject of many publications and will not be repeated in detail here. Current theories of motor behaviour have developed from systems theory, first developed by Bernstein.[15] Mulder and Hochstenbach[16] describe the organization of motor behaviour as the activity of a largely nonhierarchical, self-organizing system driven by multisensory input. Motor processes interact with cognitive and perceptual processes. The interaction between the environmental context and the state of the organism shapes the output.[16]

Intervention is directed at analyzing and optimizing all factors contributing to efficient motor control. Motor control has been defined by Shumway-Cook and Woollacott[17] as the ability to regulate or direct the mechanisms essential to movement. Movement arises from the interaction of perception and action systems. Cognition affects both systems at many different levels.[17] Movement must be understood in a task-orientated context, as goal-directed actions based on past experiences and the environment.[16] Motor output requires coordinated control of the enormous number of variables of the joints and muscles, referred to as the "degrees of freedom problem."[15] Integration of sensory information (visual, vestibular, and somatosensory) with motor output occurs at all levels of the CNS, shaping muscle activation patterns for task performance.[18,19]

Postural control and task performance

Postural control has been defined as the ability to control the body's position in space for the dual purposes of stability and orientation.[17] Although the exact mechanisms of postural control are unknown, recent research suggests that an internal representation of body posture, provided by sensory information, is important.[20,21] Massion[22] describes a postural body schema that provides an internal representation of body geometry, body dynamics including support conditions, and orientation of the body with respect to verticality. The vestibular and visual systems provide information about verticality and position in space.[23,24] The visual system also provides information regarding the environment. Cutaneous, joint, and muscle receptors mediate position sense regarding body segment orientation relative to each other and to the support surface.[25,26] Fingertip contact can modify postural control adjustments.[27,28] Investigations into the phenomena of contraversive pushing following stroke have led to theories of neural representations of trunk posture in relation to gravity, perhaps through sense organs in the trunk termed *graviceptors*.[29] Postural control is best viewed as a complex motor skill, derived from the interaction of multiple sensorimotor processes[30] (for review of the role of afferent information in postural control, see Massion and Woollacott[31]). The Bobath concept seeks to utilize appropriate sensory input to influence postural control and the internal representation of a postural body schema.[13]

The integration of posture and movement utilizes anticipatory and reactive postural control mechanisms, both of which are modulated by sensory inputs and influenced by learning and experience[32,33] (for review, see Massion & Woollacott[31]). The postural orientation of the individual relative to the base of support and gravity determines the movement strategies that will be accessible and effective.[34,35] The alignment of body segments both at the initiation of movement and throughout the evolvement of movement plays a critical role in the postural control strategies utilized.[36–39] The alignment of body segments in relation to each other and the base of support and the expression of postural control in relation to gravity and the environment are key areas of focus in the Bobath concept[13] in stroke rehabilitation and in the treatment of other neurological conditions.

Postural orientation for task performance requires interplay between stability and mobility. Muscle activation patterns are determined not only by postural alignment over the base of support and in respect to gravity but also by the interplay between closed- and open-chain movements.[40] Complex task-oriented movements, involving transitional movement sequences, necessitate controlled movement of the center of mass within and beyond the limits of stability. The Bobath concept differentiates between fixation (static muscle activation strategies)

and dynamic stability, defined as arrested mobility. Dynamic stability allows for the ongoing evolvement of selective movement and subsequent postural transitions.[13] This view is supported by the finding that, in quiet stance, stability of the proximal body segments during respiration is maintained by active neuromuscular control strategies, incorporating variable small amplitude movements of the trunk and lower limb.[41] The investigators in this study, Hodges et al., concluded that postural control of the multijoint kinetic chain of the trunk and lower limbs is organized as a combination of stability and mobility.

Selective movement and movement patterns

In human movement, selective movement of even a single joint is accompanied by activity that balances the unwanted forces at other joints.[42] The Bobath concept views selective movement as an essential component of coordinated movement sequences, or movement patterns, used for function.[13] Efficient movement is dependent upon the ability to limit and combine movements selectively into the desired functional activity under a wide range of environmental conditions. For accurate multijoint movements, the CNS must control for the effects of interaction torques arising from motions at other joints.[43] This activity provides appropriate postural stability throughout the multisegmental kinematic chain.[31] Lieber[44] describes the importance of synergistic muscle activity remote from the area of specific activation and the need to reconsider our association of movement dysfunction with strength at a single joint.

Appropriate postural control and the ability to move selectively facilitate the production of coordinated sequences of movement referred to as movement patterns. Patterns, which can be described in respect to spatial and temporal components, include (but are not limited to) walking, reach, grasp, and all postural transitions, such as sit to stand and moving between sitting and lying. Although similar between individuals, these sequences of movement are dynamic, changeable, and variable in relation to the individual, the environment, and the goal.

There has been considerable interest amongst neuroscientists in the neuronal circuitry supporting movement patterns.[45–50] Clinicians practicing the Bobath concept hypothesize that this circuitry can be accessed by facilitating task-specific patterns of muscle activation, using contextually appropriate sensory input, as well as by manipulating the environment and the task.[13]

The role of sensory input in motor behaviour

The role of the nervous system in receiving and interpreting sensory input, including proprioceptive input, is critical to achieving appropriate motor output. As stated by Mulder and Hostenbach,[16] "Without information, (sensory input) there is no control, no learning, no change, no improvement."(p146) Afferent information is important for enabling accurate feed forward commands for movement.[51–53] Sensory afferents have been shown to influence gait[54–58] (for review, see Rossignol et al.[48]) and postural control.[25,26,59] There is growing evidence that motor output utilizes internal models of sensorimotor integration, based in the parietal lobe, that are continuously refined by sensory input and efference copy of motor commands.[60–63] Improving performance and motor learning utilizes comparison of predicted and actual sensory feedback for error correction.[61,62] Movement dysfunction following stroke results in deprivation of movement experiences, minimizing both sensory input and motor output efference copy for updating internal models. Reduction of afferent information affects cortical representations of the body and the efficiency of motor output (for review, see Mulder and Hostenbach[64]). In recent studies using transcranial magnetic stimulation, it has been shown that sensory input to muscles can potentiate the responsiveness of the motor cortex.[65] In the Bobath concept, the therapist aims to utilize afferent input to re-educate the individual's internal reference systems to enable the person to have more movement choices and greater efficiency of movement.[13,14]

Movement Dysfunction and Recovery Following Neurological Pathology

CNS pathologies such as stroke can lead to movement dysfunction and to impaired function. The potential to reduce impairment and improve activity levels of the individual following

neurological damage is based upon the following factors: the ability of the neuromuscular systems to plastically adapt to the injury and the environment and experiences of the individual during the recovery period.[66,67]

Movement dysfunction

Movement dysfunction is the combined result of neurological dysfunction due to damage of the CNS, musculoskeletal changes, and learned movement strategies. It results in difficulties in initiating and controlling the appropriate postural and task-directed motor output required to perform functional activities in a safe, efficient, and timely manner. Due to the interactive nature of the nervous system, even neurons distant to the lesion may demonstrate altered function as a result of altered input and reduction of synaptic activity[68] (see review by Nudo[69]). The impact of the movement dysfunction is unique to each individual and is influenced by experiences prior to as well as post lesion.

Disruption of postural control can result in delayed anticipatory postural adjustments,[70,71] disturbed temporal synchronization,[70] and decreased amplitude of postural responses.[71,72] Motor control deficits present as impaired motor unit recruitment[73–76] (for review, see Gracies[77]), weakness,[78–80] and changes in the spatial and temporal patterns of muscle activation,[43,75,81–84] including deficits of interjoint coordination[85–87] and coactivation of agonists and antagonists[81,88–90] (for review, see Gracies[91]). Changes within the muscle itself present as changes to the properties of muscle fibers,[92–94] atrophy,[92,93] and increased mechanical stiffness[94,95] (for review, see Lieber[96] and Gracies[77]).

Observed weakness of muscles following stroke is recognized as being due to multiple causes. Levin et al.[90] describe the following factors as contributors to observed muscle weakness:

- Lack of excitation in descending pathways responsible for voluntary movement,
- Muscle fiber atrophy and contracture,
- Changes in the spatial and temporal patterns of muscle activation, resulting in an inefficient EMG-torque relationship, and
- Loss of functioning motor units and changes in the properties of the remaining ones.

Posture and movement can be impeded by increased muscle stiffness (tone), including intermittent or sustained involuntary activation of muscle.[97] The neural elements of increased tone include inability to modulate reflex activity over the contraction range and inability to reduce background levels.[98] Increased tone also has nonneural elements, involving intrinsic changes in the passive mechanical properties of muscle.[99] These changes occur both in the muscle cell and in the extracellular matrix (for review, see Lieber[96]). Spasticity, defined as a velocity-dependent increase in tonic stretch reflexes,[100] is no longer regarded as the primary cause of movement dysfunction.[87,101] In a recent review, Gracies[91] argues that, while spastic hypertonia does not contribute to disability, both spastic dystonia and spastic co-contraction are disabling. Clinicians practicing the Bobath concept address both neural and nonneural elements of tone to potentiate improved muscle activation patterns, minimize unnecessary compensatory movement strategies, and identify potential secondary impairments.[13]

Deficits of motor control can lead to the use of compensatory mechanisms.[85,102] At a functional level, compensatory mechanisms may achieve the task. If they do, they will reinforce the motor strategies used and may prevent the reacquisition of other strategies available to the person.[86,103] At a neural level, the compensatory activity may limit the recovery of spared neural mechanisms. Compensatory activities of the unaffected upper limb have been implicated in "learned nonuse" of the affected limb following stroke, leading to reduced recovery of function.[104]

The neurological dysfunction results in neuropsychological disturbances as well as deficits of motor control and sensation. These may present as perceptual, behavioural, emotional, and cognitive changes. In planning intervention, all aspects of motor behaviour are taken into consideration, including neuropsychological factors, psychosocial factors, and environmental issues.[13]

Neuromuscular plasticity

Neurological rehabilitation is the management of recovery.[105] Recovery is an active time-dependent process that includes plasticity and reorganization

of brain structures, as well as adaptive changes in musculoskeletal, cardiovascular, and respiratory systems.[105] Neuromuscular plasticity is a key element of functional recovery. Plastic adaptation of the neural and musculoskeletal systems occurs in response to trauma or to changes in the internal and external environment or as a result of sensorimotor learning and experience.

Neural plasticity is the adaptive capacity of the nervous system and its ability to modify its own structural organization and function.[106,107] Neural plasticity enables strengthening or weakening of synapses and alteration of functional connections in response to specific inputs, including training leading to motor skill acquisition (see review by Nudo[108]). These changes include reorganization in the cortex[109,110] and dendritic sprouting and synaptogenesis[111,112] (for review, see Nudo[108] and Duffau[113]). Remodeling occurs at the molecular and cellular level, involving changes to presynaptic efficiency (for review, see Leenders and Sheng[114]), the receptivity of the postsynaptic membrane (for review, see Luscher et al.[115]), and structural changes to neurons (for review, see Xu-Freidman and Regehr[116]).

The interaction between form (the anatomy of the neuromuscular system) and function (the behavioural strategy utilized to perform a task) influences the remodelling.[108] Motor recovery and plasticity are dependent on the nature of motor rehabilitation. In animal models, enhanced synaptogenic responses have been observed following brain lesions with complex motor skills training compared to simple repetitive exercises[112] (for review, see Kleim[117]).

Plastic changes in muscle occur readily in response to neural pathology, alteration in muscle length, or muscle use. These include alteration in muscle fiber size and distribution,[92,93] increased stiffness,[95] and alterations in the extracellular elements[94] (for review, see Lieber et al.[96]).

Motor learning

Motor learning refers to the acquisition and modification of movement.[17] Motor learning requires the intention to perform a task, practice, and feedback (for review, see Shumway, Cook, and Woollacott[17]). Certain types of practice and feedback are more beneficial for motor task acquisition, retention, and transference.[118–120] Explicit and implicit learning are involved in motor learning.[121] Brain injury can have differential affects on these two types of learning.[122–123]

As the goal of intervention is optimal participation in daily life situations, the Bobath concept demands training in different real-life situations as appropriate and not only in the therapy department. Task-specific muscle activation patterns and sensory input are used to enable successful completion of the task in different contexts and environments, taking perceptual and cognitive demands into consideration. Improvement of task performance is not only limited to practicing the task.

Key Aspects of Clinical Practice

The Bobath concept is an interactive problem-solving approach. Reassessment is ongoing with attention to individual goals, development of working hypotheses, treatment plans, and relevant objective measures to evaluate interventions. Intervention strategies are unique to the individual.[13,14]

The Bobath concept is inclusive; it is used with individuals of any age who have suffered damage to their CNS, regardless of the degree of severity.[10] In this respect, it differs from the motor relearning approach[124] and constraint-induced movement therapy,[104] both of which can only be used with relatively high-functioning individuals.

The ICF provides a framework for describing problems of functioning, disability, and health. The identification of participation restrictions requires effective communication with the individual, his or her family, and any other caregivers. Analysis of movement and task performance enables the therapist to identify activity limitations as well as underlying problems of impairment.[125] These impairments may involve the CNS or the target tissues. Treatment strategies address underlying impairments, task-specific components of posture and movement, the functional activity, and its integration into participation in relevant situations in daily life.[13] The ultimate goal of intervention is to optimize activity and participation thereby improving quality of life.

The process of assessment, goal-setting, and intervention requires clinicians practicing the Bobath concept to utilize present-day knowledge of motor control, the nature of movement dysfunction, neuromuscular plasticity, biomechanics, and motor learning. Client needs and expectations are also taken into account together with the experience of expert clinicians.

Treatment is focused toward remediation, exploring the individual's potential to regain abilities through neuromuscular plastic adaptation. Treatment objectives endeavor to allow performance of variable tasks in various environments. Function in diverse environments is dependant upon effective postural control and selective movement patterns; these are interdependent and enable movement efficiency.[13,14]

The emphasis in the Bobath concept on the integration of postural control and task performance is integral to the choice of intervention strategies. A misinterpretation of the Bobath concept is the assumption that perfect alignment of body segments and postural control are required before engaging in task performance. The use of task-directed movement during treatment does not presuppose independent postural control. By changing the environment and providing an appropriate external support, the individual can perform complex motor tasks that in turn can improve postural control and selective movement. Alternatively, directly addressing alignment of body segments (macro) or tissue (micro) and postural control may improve efficiency of complex motor tasks.[14]

Facilitation: Manipulation of sensory inputs

In the Bobath concept, the use of afferent information to effect improvements in motor performance is described as facilitation. Facilitation is used to enable successful movement and task performance with regard to aspects such as postural orientation, components of movement, functional sequences of movement, recognition of the task, and motivation to complete the task.[13] Facilitation is specifically regulated, including timing, modality, intensity, and withdrawal as critical elements. The facilitation utilized should not be contradictory to the task. The objective is to provide appropriate afferent information approximating that usually experienced during performance of the motor task.[13]

The use of facilitation during intervention has been a key feature of the Bobath concept since its inception. Facilitation is part of an active learning process in which the individual is enabled to actively overcome inertia and initiate, continue, or complete a functional task. It assists the patient in problem solving and enables him or her to experience the patterns of movement and success in achieving the task. Success in motor performance is required for motor learning.

Facilitation may be directed primarily toward the postural control needed for task-directed movement, toward the task-directed movement itself, or toward both. Facilitation involves specific manipulation of afferent inputs inclusive of somatosensation, vision, vestibular, and auditory in order to bring motor systems to threshold. Through facilitation, the Bobath clinician specifies the sequences of movement and specific muscle activity that will produce efficient task performance. Facilitation via handling skills is intended to provide key components of the spatial and temporal aspects of a specific movement/task to enable the individual to have an experience of movement that is not passive but one that they cannot yet do alone. Facilitation is designed to make the activity possible, to demand a response, and to allow the response to happen.[13]

Two studies have examined the effects of manual facilitation while it was occurring. Hesse[126] demonstrated improvement in spatial and temporal parameters and patterns of muscle activation during facilitation of gait. Miyai et al.[127] showed similar spatial and temporal changes, accompanied by changes in cortical activation in the affected cerebral hemisphere.

Facilitation can be utilized for specific muscle activation as preparation for volitional activity. For example, working for grasp may include activation of the muscles required using specific compression or distraction with manual guidance. Similarly, preparation for walking may include activation of hip extensors and abductors to control the pelvic tilt. One of the most difficult aspects of motor control for the patient is to produce sufficient muscle activation to overcome inertia and initiate

an effective movement.[74,80] At the same time, the clinician seeks to maximize interjoint coordination and minimize abnormal coupling of muscle activity or excessive co-activation (as discussed in the previous section). Facilitation is thus used both to enhance activation and body part stabilization and to reduce muscle activation that is not relevant to the task.

In the facilitation of functional movement sequences, the clinician is goal orientated to enable the movements to be performed in familiar patterns with familiar timing and speed, thereby using neural substrates that do not demand excessive cortical attention. With CNS damage, the individual often uses cognitive problem-solving abilities in an attempt to find a solution to the current dilemma. This may lead to novel movements that are less efficient. The facilitation of familiar movement sequences by the therapist may enable the patient to access existing, undamaged neuronal circuits rather than having to learn a new skill. Activation and/or modification of central pattern generators for walking may potentially be achieved by facilitating appropriate loading and unloading of the limb, hip alignment in the stance phase, and cutaneous inputs through the feet (see review by Rossignol et al.[48] for evidence of relevance of these components to gait).

Facilitation is a clinical skill that is developed over time and requires both problem-solving skills and motor learning on the part of the clinician. If facilitation is to be successful, it must lead to a change in motor behaviour. To ensure that this occurs, the degree of facilitation is reduced within a treatment session and is withdrawn over a period of treatment until the individual can initiate and complete the task independently. During the period in which facilitation is still being used as part of the intervention process, repetition and variability of patterns of movement and behavioural strategies are incorporated. In this way, the individual gains experience and insight into their movement strategies and learns how to adapt their problem solving to different tasks and environments.

Management of compensatory motor behaviors

Compensation is inevitable post CNS lesion. The challenge is to minimize compensations that will limit ongoing recovery while enabling ongoing goal achievement. Clinicians practicing the Bobath concept differentiate between appropriate and inappropriate compensation strategies. Appropriate compensations are those that are necessary for the performance of a specific task in a given environment at a certain time but that do not persist once the task has been accomplished. With effective intervention strategies directed at underlying impairments or specific components of movement and motor control, these compensatory strategies should diminish over time. Inappropriate compensations are those that persist beyond the completion of a task, limit other functions, or mask potential for further recovery.[13]

Within the Bobath concept, active participation in functional tasks is not prevented in an attempt to avoid compensatory strategies.[14] The objective is to adapt the task to enable active participation, without impacting on the potential for future task performance.

The Bobath concept as an overall management strategy

The Bobath concept strives toward a 24-hour interdisciplinary management approach.[14] When the individual, family, all professionals, and other caregivers have insight into the problems and work together for the same goals, these goals are usually accomplished. Motivation and the therapeutic relationship between the clinician, the patient, and their family and/or caregivers are recognized as essential aspects for successful rehabilitation. The holistic approach to intervention is integral to the Bobath concept. An overall management strategy implies that all aspects of functioning are addressed, consistent with the ICF model.

Conclusion

This article endeavours to update the reader and explain the current theoretical framework underlying the Bobath concept in the rehabilitation of individuals who have sustained a stroke or other lesion of the CNS. This framework forms the basis for contemporary clinical practice. This article has emphasized aspects of clinical implementation that differentiate this approach from other approaches to

rehabilitation, but it has not attempted to cover all intervention strategies that might be incorporated within the overall approach depending upon the needs of the individual.

Intervention should bring about change at all three levels: participation, activity, and impairment. The measurement of clinical change requires tools that are sensitive to the types and degrees of change that are clinically important.[128] There is a need to provide evidence that goes beyond reduction of impairment or achievement of activity and includes a real, meaningful, and sustainable change in the lives of individuals and their families.[120]

It is hoped that this updated framework will be used as a basis for discussion in future research publications and that protocols will be designed to reflect the reality of contemporary clinical practice. There is no recipe for treatment. Assessment, goal setting, treatment planning, and implementation of treatment are highly individualized, and ways should be sought in which outcomes can be studied in an appropriate and meaningful manner.

REFERENCES

1. Lennon S. The Bobath concept: A critical review of the theoretical assumptions that guide physiotherapy practice in stroke rehabilitation. *Phys Ther Rev.* 1996;1:35–45.
2. Barret J, Evans L, Chappell J, Fraser C, Clayton L. Bobath or motor relearning programme: A continuing debate. *Clin Rehabil.* 2001;15:445–446.
3. Brock K, Jennings K, Stevens J, Picard S. The Bobath concept has changed. *Aust J Physiother.* 2002; 48:156–157.
4. Panturin E. The Bobath concept. *Clin Rehabil.* 2001;15: 111.
5. Luke C, Dodds K, Brock K. Outcomes of the Bobath concept on upper limb recovery following stroke. *Clin Rehabil.* 2004;18:888–898.
6. Paci M. Physiotherapy based on the Bobath concept for adults with post-stroke hemiplegia: A review of effectiveness studies. *J Rehabil Med.* 2003; 35: 2–7.
7. Bobath B, Bobath K. Interview for Neuro-developmental Therapy Association. Baltimore, MD: 1981.
8. Bobath B. Die Entwicklung und Veränderung des Neuro-Developmental-Treatment. Lecture for the Vereinigung der Bobath-Therapeuten Deutschlands, Bremerhaven; 1984.
9. IBITA. Theoretical assumptions and clinical practice. Available at: www.ibita.org. Accessed January 8, 2008.
10. Raine S. Defining the Bobath concept using the Delphi technique. *Physio Res Int.* 2006;11:4–13.
11. Mayston M. The Bobath Concept today. *Synapse.* 2001;Spring:32–35.
12. World Health Organization. *International Classification of Functioning, Disability and Health (ICF).* Geneva; World Health Organization: 2001.
13. Gjelsvik BE. *The Bobath Concept in Adult Neurology.* Stuttgart, Germany: Thieme; 2008.
14. Raine S. The current theoretical assumptions of the Bobath concept as determined by the members of BBTA. *Physiother Theory Practice.* 2007;23: 137–152.
15. Bernstein N. *The Coordination and Regulation of Movement.* London: Pergamon; 1967.
16. Mulder T, Hochstenbach J. Motor control and learning: Implications for neurological rehabilitation. In: Greenwood RJ, ed. *Handbook of Neurological Rehabilitation.* New York: Psychology Press; 2003: 143–157.
17. Shumway-Cook A, Woollacott M. *Motor Control: Translating Research into Clinical Practice.* 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2007.
18. Guillery RW, Sherman SM. The thalamus as a monitor of motor outputs. *Phil Trans R Soc Lond B.* 2002;357:1809–1821.
19. Bosco G, Poppele R. Proprioception from a spinocerebellar perspective. *Physiol Rev.* 2001;81: 539–568.
20. Mergner T, Maurer C, Peterka R. A multisensory posture control model of human upright stance. *Prog Brain Res.* 2003;142:189–201.
21. Spinazolla L, Cubelli R, Della Sala S. Impairments of trunk movements following left or right hemisphere lesions: Dissociation between apraxic errors and postural instability. *Brain.* 2003;126:2656–2666.
22. Massion J. Postural control system. *Curr Opin Neurobiol.* 1994;4:877–887.
23. Keshner EA, Peterson B. Mechanisms controlling human head stabilization. I. Head-neck dynamics during random rotations in the horizontal plane. *J Neurophysiol.* 1995;6:2293–2301.
24. Keshner EA, Peterson B. Mechanisms controlling human head stabilization. II. Head-neck dynamics during random rotations in the vertical plane. *J Neurophysiol.* 1995;6:2302–2311.
25. Kavounoudias A, Roll R, Roll J. Foot sole and ankle muscle inputs contribute jointly to human erect posture regulation. *J Physiol.* 2001;532(3):869–878.
26. Roll R, Kavounoudias A, Roll J. Cutaneous afferents from human plantar sole contribute to body posture awareness. *Neuroreport.* 2002;13:1957–1961.
27. Lackner JR, DiZio P, Jeka J, Horak F, Krebs R, Rabin E. Precision contact of the fingertip reduces postural sway of individuals with bilateral vestibular loss. *Exp Brain Res.* 1999;126: 459–466.
28. Krishnamoorthy V, Slijper H, Latash M. Effects of different types of light touch on postural sway. *Exp Brain Res.* 2002;147:71–79.

29. Karnath H, Ferber S, Dichgans J. The neural representation of postural control in humans. *Proc Natl Acad Sci USA.* 2000;97:13931–13936.
30. Horak F. Postural orientation and equilibrium: What do we need to know about neural control of balance to prevent falls? *Age Ageing.* 2006;35-S2:7–11.
31. Massion J, Woollacott M. Posture and equilibrium. In: Bronstein A, Brandt T, Woollacott M, Nutt J. *Clinical Disorders of Balance, Posture and Gait.* 2nd ed. London, UK: Edward Arnold; 2004:1–19.
32. Cordo P, Nashner L. Properties of postural adjustments associated with rapid arm movements. *J Neurophysiol.* 1982;47:287–302.
33. Horak F, Nashner L. Central programming of postural movement: Adaptation to altered support surface configurations. *J Neurophysiol.* 1986;55: 1369–1381.
34. van der Fitz I, Klip A, van Eykern L, Hadders-Algra M. Postural adjustments accompanying fast pointing movements in standing, sitting and lying adults. *Exp Brain Res.* 1998;120:202–216.
35. Forssberg H, Hirschfeld H. Postural adjustments in sitting humans following external perturbations. *Exp Brain Res.* 1994;97:515–527.
36. Aruin A. The effect of asymmetry of posture on anticipatory postural adjustments. *Neuroscience Lett.* 2006;401:150–153.
37. Krishnamoorthy V, Latash M. Reversals of anticipatory postural adjustments during voluntary sway in humans. *J Physiol.* 2005;565:675–684.
38. Mille M, Mouchino L. Are human anticipatory postural adjustments affected by a modification of the initial position of the center of gravity? *Neuroscience Lett.* 1998;242:61–64.
39. Mouchnino L, Aurenty R, Massion J, Pedotti A. Coordination between equilibrium and head-trunk orientation during leg movement: A new strategy built up by training. *J Neurophysiol.* 1992;67: 1587–1598.
40. Butler P, Major R. The missing link? Therapy issues of open and closed chains. *Physiother.* 2003;89: 465–470.
41. Hodges PW, Gurfinkel VS, Brumagne S, Smith TC, Cordo PC. Coexistence of stability and mobility in postural control: evidence from postural compensation for respiration. *Exp Brain Res.* 2002;144: 293–302.
42. Wiess E, Flanders M. Muscular and postural synergies of the human hand. *J Neurophysiol.* 2004;92:523–535.
43. Beer R, Dewald J, Rymer W. Deficits in the coordination of multijoint arm movements in patients with hemiparesis: Evidence for disturbed control of limb dynamics. *Exp Brain Res.* 2000;131:305–319.
44. Lieber RL. *Skeletal Muscle Structure, Function, and Plasticity: The Physiological Basis of Rehabilitation.* Baltimore: Lippincott Williams & Wilkins; 2005.
45. Balter J, Zehr E. Neural coupling between the arms and legs during rhythmic locomotor-like cycling movement. *J Neurophysiol.* 2007:97;1809–1818.
46. Graziano MS, Taylor CS, Moore T. Complex movements evoked by microstimulation of precentral cortex. *Neuron.* 2002;34:841–851.
47. Mason C, Gomez J, Ebner T. Hand synergies during reach to grasp. *J Neurophysiol.* 2001;86:2896–2910.
48. Rossignol S, Dubuc R, Gossard J. Dynamic sensorimotor interactions in locomotion. *Physiol Rev.* 2006;86:89–154.
49. Schieber MH. Motor control: Basic units of cortical output? *Curr Biol.* 2004;14:R353–R354.
50. Van Kan PL, McCurdy ML. Role of primate magnocellular red nucleus neurons in controlling hand preshaping during reaching to grasp. *J Neurophysiol.* 2001;85:1461–1478.
51. Ghez C, Gordon J, Ghilardi MF Impairments of reaching movements in patients without proprioception. II. Effects of visual information on accuracy. *J. Neurophysiol.* 1995:73;361–372.
52. Knox, J, Coppieters, M, Hodges P. Do you know where your arm is if you think your head has moved? *Exp Brain Res.* 2006;173:94–101.
53. Sainburg RL, Ghilardi F, Poizner H, Ghez C. Control of limb dynamics in normal subjects and patients without proprioception. *J Neurophysiol.* 1995;73:820–835.
54. Deshpande N, Patla A. Dynamic visual-vestibular integration during goal directed human locomotion. *Exp Brain Res.* 2005;166:237–247.
55. Deshpande N, Patla A. Postural responses and spatial orientation to neck proprioceptive and vestibular inputs during locomotion in young and older adults. *Exp Brain Res.* 2005;167:468–474.
56. Earhart GM, Melvill Jones G, Horak FB, Block EW, Weber KD, Fletcher WA. Transfer of podokinetic adaptation from stepping to hopping. *J Neurophysiol.* 2002;87:1142–1144.
57. Earhart GM, Melvill Jones G, Horak FB, Block EW, Weber KD, Fletcher WA. Forward versus backward walking: Transfer of podokinetic adaptation. *J Neurophysiol.* 2001;86:1666–1670.
58. Sorensen K, Hollands M, Patla A. The effects of human ankle muscle vibration on posture and balance during adaptive locomotion. *Exp Brain Res.* 2002;143:24–34.
59. Kavounoudias A, Roll R, Roll J. The plantar sole is a "dynamometric map" for human balance control. *Neuroreport.* 1998;9:3247–3252.
60. Pellijeff A, Bonilha L, Morgan PS, McKenzie K, Jackson SR. Parietal updating of limb posture: An event related fMRI study. *Neuropsychologia.* 2006; 44:2685–2690.
61. Sainburg RL, Ghez C, Kalakanis D. Intersegmental dynamics are controlled by sequential anticipatory, error correction, and postural mechanisms. *J Neurophysiol.* 1999;81:1045–1056.
62. Wolpert D, Ghahramani Z, Jordan M. An internal model for sensorimotor integration. *Science.* 1995;269:1880–1882.
63. Wolpert D, Goodbody S, Husain M. Maintaining internal representations: The role of the human superior parietal lobe. *Nature Neurosci.* 1998;1: 529–533.
64. Mulder T, Hochstenbach J. Adaptability and flexibility of the human motor system: Implications for neurological rehabilitation. *Neural Plasticity.* 2001;8:131–140.

65. Rothwell JC, Rosenkranz K. Role of afferent input in motor organisation in health and disease. *IEEE Engineer Med Biol.* 2005;24:40–44.
66. Nudo RJ Wise BM, SI Fuentes F, Milliken GW. Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. *Science.* 1996;272:1791–1794.
67. Nudo RJ. Postinfarct cortical plasticity and behavioral recovery. *Stroke.* 2007;38:840–845.
68. Seitz R, Azari N, Knorr U, Binkofski F, Herzog H, Freund H. The role of diaschisis in stroke recovery. *Stroke.* 1999;30:1844–1850.
69. Nudo R, Plautz E, Frost S. Role of adaptive plasticity in recovery of function after damage to motor cortex. *Muscle Nerve.* 2001;24:1000–1019.
70. Dickstein R, Sheffi S, Markovici E. Anticipatory postural adjustment in selected trunk muscles in poststroke hemiparetic patients. *Arch Phys Med Rehabil.* 2004;85: 261–267.
71. Kirker S, Jenner J, Simpson D, Wing A. Stepping before standing: Hip muscle function in stepping and standing balance after stroke. *J Neurol Neurosurg Psych.* 2000;68:458–464.
72. Slijper H, Latash M, Rao N, Aruin A. Task-specific modulation of anticipatory postural adjustments in individuals with hemiparesis. *Clin Neurophysiol.* 2002;113:642–655.
73. Gemperline J, Allen S, Walk D. Characteristics of motor unit discharge in subjects with hemiparesis. *Muscle Nerve.* 1995;18:1101–1114.
74. Landau WM, Sahrmann SA. Preservation of directly stimulated muscle strength in hemiplegia due to stroke. *Arch Neurol.* 2002;59:1453–1457.
75. Lum P, Burgar C, Shor P. Evidence for strength imbalances as a significant contributor to abnormal synergies in hemiparetic subjects. *Muscle Nerve.* 2003;27:211–221.
76. Newham DJ, Hsiao SF. Knee muscle isometric strength, voluntary activation and antagonist co-contraction in the first six months after stroke. *Disabil Rehabil.* 2001;23:379–386.
77. Gracies J. Pathophysiology of spastic paresis: 1. Paresis and soft tissue changes. *Muscle Nerve.* 2005;31:535–551.
78. Andrews A, Bohannon R. Distribution of muscle strength impairments following stroke. *Clin Rehabil.* 2000;14:79–87.
79. Chae J, Yang G, Park BK, Labatia I. Muscle weakness and co-contraction in upper limb hemiparesis: Relationship to motor impairment and physical disability. *Neurorehabil Neural Repair.* 2002;16:241–248.
80. Davies JM, Mayston MJ, Newham DJ. Electrical and mechanical output of the knee muscles during isometric and isokinetic activity in stroke and healthy adults. *Disabil Rehabil.* 1996;18:83–90.
81. Chae J, Yang G, Park BK, Labatia I. Delay in initiation and termination of muscle contraction, motor impairment, and physical disability in upper limb hemiparesis. *Muscle Nerve.* 2002;25: 568–575.
82. Dewald J, Pope P, Given J, Buchanan T. Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. *Brain.* 1995;118: 495–510.
83. Dewald J, Beer RF. Abnormal joint torque patterns in the paretic upper limb of subjects with hemiparesis. *Muscle Nerve.* 2001;24:273–283.
84. Kautz SA, Brown DA. Relationships between timing of muscle excitation and impaired motor performance during cyclical lower extremity movement in post stroke hemiplegia. *Brain.* 1998; 121:515–526.
85. Cirstea CM, Levin MF. Compensatory strategies for reaching in stroke. *Brain.* 2000;123: 940–953.
86. Michaelsen SM, Levin MF. Short-term effects of practice with trunk restraint on reaching movements in patients with chronic stroke: a controlled trial. *Stroke.* 2004;35:1914–1919.
87. Zackowski K, Dromerick A, Sahrmann SA, Thach W, Bastian A. How do strength, sensation, spasticity, and joint individuation relate to the reaching deficits of people with chronic hemiplegia? *Brain.* 2004;127:1035–1046.
88. Gracies J, Wilson L, Gandevia S. Stretched position of spastic muscles aggravates their co-contraction in hemiplegic patients. *Ann Neurol.* 1997;42:439.
89. Kamper DG, Rymer WZ. Impairment of voluntary control of finger motion following stroke: Role of inappropriate muscle coactivation. *Muscle Nerve.* 2001;24:673–681.
90. Levin MF, Selles RW, Verhuel MH, Meijer OG. Deficits in the co-ordination of agonist and antagonist muscles in stroke patients: Implications for normal motor control. *Brain Res.* 2000;853:352–369.
91. Gracies J. Pathophysiology of spastic paresis: 2. Emergence of muscle overactivity. *Muscle Nerve.* 2005;31:552–571.
92. Dattola R, Girlanda P, Vita G et al. Muscle rearrangements in patients with hemiparesis after stroke: an electophysiological and morphological study. *Eur Neurol.* 1993;33:109–114.
93. Dietz V, Ketelsen W, Quintern J. Motor unit involvement in spastic paresis: Relationship between leg muscle activation and histochemistry. *J Neuro Sci.* 1986;75:89–103.
94. Lieber RL, Runesson E, Einarsson F, Friden J. Inferior mechanical properties of spastic muscle bundles due to hypertrophic but compromised extracellular matrix material. *Muscle Nerve.* 2003;28:464–471.
95. Friden J, Lieber R. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve.* 2003; 27:157–164.
96. Lieber RL, Steinman S, Barash A, Chambers H. Structural and functional changes in spastic skeletal muscle. *Muscle Nerve.* 2004;29:615–627.
97. Burridge JH, Wood DE, Hermans HJ, et al. Theoretical and methodological considerations in the measurement of spasticity. *Disabil Rehabil.* 2005;27: 69–80.
98. Burne J, Carleton V, O'Dwyer N. The spasticity paradox: Movement disorder or disorder of resting limbs? *J Neurol Neurosurg Psych.* 2005;76:47–54.
99. Dietz V, Berger W. Normal and impaired regulation of muscle stiffness in gait: A new hypothesis about muscle hypertonia. *Exp Neurol.* 1983;79:680–687.

100. Lance JW. Symposium synopsis. In: Feldman R, Young R, Koella W, eds. *Spasticity: Disordered Motor Control.* Chicago: Year Book; 1980.
101. Ada L, Vattanasilp W, O'Dwyer N, Crosbie J. Does spasticity contribute to walking dysfunction following stroke? *J Neurol Neurosurg Psych.* 1998;64: 628–635.
102. Roby-Brami A, Feydy A, Combeaud M, Biryukova E, Bussel B, Levin M. Motor compensation and recovery for reaching in stroke patients. *Acta Neurol Scand.* 2003;107:369–381.
103. Michaelsen SM, Dannenbaum R, Levin M. Task-specific training with trunk restraint on arm recovery in stroke. *Stroke.* 2006;37:186–192.
104. Taub E, Miller N, Novack T, et al. Technique to improve chronic motor deficit after stroke. *Arch Phys Med Rehabil.* 1993;74:347–354.
105. Gordon, J. A top-down model for neurologic rehabilitation. In: *Linking Movement Science and Intervention, Proceedings of the III Step Conference.* Salt Lake City, Utah: American Physical Therapy Association; 2005:28–33.
106. Nudo RJ. Recovery after damage to motor cortical areas. *Curr Opin Neurobiol.* 1999;6:740–747.
107. Bach-y-Rita P. Theoretical and practical considerations in the restoration of function after stroke. *Top Stroke Rehabil.* 2001;4:51–58.
108. Nudo R. Adaptive plasticity in motor cortex: Implications for rehabilitation after brain injury. *J Rehabil Med.* 2003;41:7–10.
109. Dancause N, Barbay S, Frost S, et al. Extensive cortical rewiring after brain injury. *J Neurosci.* 2005;25: 10167–10179.
110. Liepert J, Bauder H, Miltner W, Taub E, Weiller C. Treatment induced cortical reorganization after stroke in humans. *Stroke.* 2000;31:1210–1121.
111. JonesTA, Shallert T. Use-dependent growth of pyramidal neurons after neocortical damage. *J Neurosci.* 1994;14:2140–2152.
112. Jones T, Chu D, Grande L, Gregory A. Motor skills training enhances lesion-induced structural plasticity in the motor cortex of adult rats. *J Neurosci.* 1999;19:10153–10163.
113. Daffau H. Brain plasticity: From pathophysiological mechanisms to therapeutic applications. *J Clin Neurosci.* 2006;13:885–897.
114. Leenders AGM, Sheng ZH. Modulation of neurotransmitter release by the second messenger-activated protein kinases: Implications for presynaptic plasticity. *Pharmacol Ther.* 2005; 105:69–84.
115. Luscher C, Nicoll R, Malenka R. Synaptic plasticity and dynamic modulation of the postsynaptic membrane. *Nat Neurosci.* 2000:3:545–550.
116. Xu-Friedman M, Regehr W. Structural contributions to short-term synaptic plasticity. *Physiol Rev.* 2004;84:69–85.
117. Kleim JA. Neural mechanisms of motor recovery after stroke: Plasticity within residual cortical tissue. In: *Linking Movement Science and Intervention. Proceedings of the III Step Conference.* Salt Lake City, UT: American Physical Therapy Association; 2005:73.
118. Schmidt RA. Motor learning principles for physical therapy. In: *Contemporary Management of Motor Control Problems. Proceedings of the II Step Conference.* Alexandria VA: American Physical Therapy Association; 1992:49–62.
119. Schmidt RA, Lee TD. *Motor Control and Learning: A Behavioural Emphasis.* Champaign, IL: Human Kinetics; 2005.
120. Winstein CJ. Motor learning. In: *Linking Movement Science and Intervention, Proceedings of the III Step Conference.* Salt Lake City, UT: American Physical Therapy Association; 2005:158–160.
121. Orrell AJ, Eves FF, Masters RSW. Implicit motor learning of a balancing task. *Gait Posture.* 2006;23:9–16.
122. Boyd L, Winstein C. Impact of explicit information on implicit motor learning following middle cerebral artery stroke. *Phys Ther.* 2003;83: 976–989.
123. Boyd L, Winstein C Providing explicit information disrupts implicit motor learning after basal ganglia stroke. *Learning Memory.* 2004;1:388–396.
124. Carr JH, Shepherd RB. *Stroke Rehabilitation. Guidelines for Exercise and Training to Optimise Motor Skill.* Oxford, UK: Butterworth Heinemann; 2003.
125. Lazaro R, Roller M, Umphred D. Differential diagnosis phase 2: Examination and evaluation of disabilities and impairments. In: Umphred D, ed. *Neurological Rehabilitation.* St. Louis, MO: Mosby; 2001.
126. Hesse S, Jahnke M, Schaffrin A. Immediate effects of therapeutic facilitation on the gait of hemiparetic patients as compared with walking with and without a cane. *Electroenceph Clin Neurophysiol.* 1998;105:149–155.
127. Miyai I, Yagura H, Oda I. Premotor cortex is involved in restoration of gait in stroke. *Ann Neurol.* 2002;52:188–194.
128. Whyte J. Measuring (treatment-induced) change: conceptual issues. In: *Linking Movement Science and Intervention, Proceedings of the III Step Conference.* Salt Lake City, UT: American Physical Therapy Association; 2005:100–104.