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Problem solving in neurological physiotherapy – setting the scene

Margaret J. Mayston

HISTORY

A therapist using a problem-solving approach to the management of neurological patients prior to the 1940s may have asked: How can I train the person to use their unaffected body parts to compensate for the affected parts, and how can I prevent deformity? The result was a strong emphasis on orthopaedic intervention with various types of splints, strengthening exercises and surgical intervention. However, in the 1940s several other ideas emerged, the most popular being the Bobath approach. Bobath (1985) with others, such as Peto (Forrai 1999), Kabat & Knott (1954), Voss (1967) and Rood (1954), pioneered the neurological approach to these disorders, recognising that patients with neurological impairment, in particular stroke patients, had potential for functional recovery of their affected body parts. For the child with a neurodevelopmental disorder, the approach was based on the idea that each child's development could be guided by the therapist, to maximise their potential for functional independence and minimise contractures and deformities. While the Bobath approach is one of the most used and accepted in the UK, little has been written about it in recent years, and there is no robust evidence for its efficacy (Davidson & Waters 2000).

In the last few years there has been a further progression in the neurorehabilitation field, with increasing interest in different models of central nervous system (CNS) function, skill acquisition and training. For example, for some therapists, the emphasis for retraining of the neurologically

impaired person now is on the biomechanical requirements of a task (Carr & Shepherd 1998), accepting that the patient has to compensate for their damaged nervous system. Carr and Shepherd are to be applauded for their well-researched approach; however, it should be recognised that their actual ideas for management largely arose from the work of Bobath. The emphasis on patient participation and practice is helpful for the cognitively and physically able person, but it is unclear how the approach can be used with people who have significant neurological impairments.

It must be realised that the nervous and musculoskeletal systems cannot be separated; they interact with each other to meet the demands of both the internal and external environment. Thus it is important to approach the person with movement disorder with a balanced view of the neural control of movement, the biomechanical requirements for a task and the limitations of CNS damage on both of these systems.

In order to use a problem-solving approach for the treatment of people with neurological disability, it is necessary to have an understanding of the control of movement, the result of damage to different areas of the CNS, neuroplasticity and ways to promote skill learning.

CONTROL OF MOVEMENT

There are many models of motor control. Some examples are neurophysiological, systems/distributed model, neurobehavioural, engineering model, information processing and biomechanical. All have value, but individually do not provide the therapist with complete information on which to base their practice. Therefore an understanding of different approaches is helpful for the therapist working in the neuro-rehabilitation field. The most relevant of these are discussed below.

Neurophysiological/information processing

It is recognised that there is an interaction between central and peripheral components of

the CNS (see Dietz 1992 for a review). Dietz (1992) points out that neuronal mechanisms are a part of biomechanical strategies but are themselves constrained by biomechanics. This view is supported by Martenuik et al (1987) who make the following comment: 'While there are biomechanical factors which constrain movement control processes, there are also brain mechanisms which are potentially complementary to the biomechanical factors that take part in the planning and control processes. We cannot neglect one at the expense of the other ...'. What then do we need to know about the neurophysiological control of movement?

Early ideas suggested that the CNS controlled movement primarily by reacting to sensory input (Foster 1985, Sherrington 1906). Roland et al (1980) demonstrated the presence of brain activity when simply imagining a movement by studying changes in regional cerebral blood flow. This work alongside other studies of CNS activity during function (Deecke et al 1969, Shibasaki & Nagae 1984, Kristeva et al 1994) has demonstrated activity of the brain before a movement begins, and has shown that the nervous system is largely proactive and not simply reactive, in response to sensory feedback. Central (feedforward) mechanisms are based on innate and ongoing experiences of the individual and can take place in the absence of any kind of sensory feedback. Keele (1968) suggested that the CNS organises a general plan in advance of the task to be executed, referred to as the motor programme, on the basis of prior experience. Schmidt (1991) has taken up this idea of programme-based motor control, describing the comparative nature of how the brain organises the preparation and execution of movements. Much debate has taken place about the role of the motor programme and sensory feedback from the periphery in motor control (Morris et al 1994). However, it is clear that both central and peripheral factors are important in the efficient execution of motor tasks.

Central programming requires the integration of many neural structures, both supraspinal and in the periphery, to produce the required output to achieve the task goal. It is helpful to consider

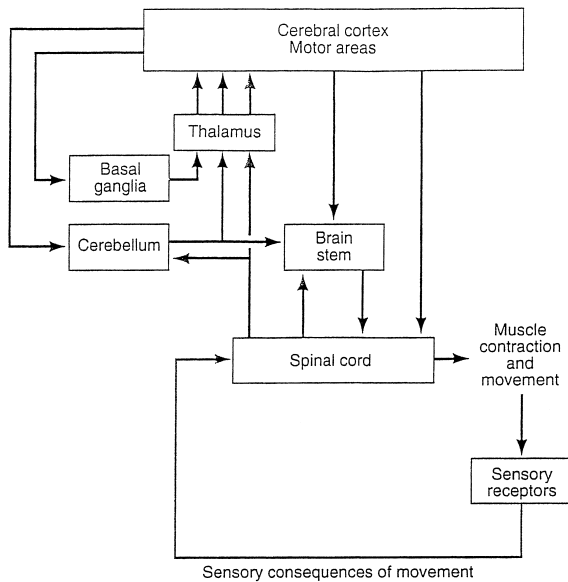


Figure 1.1 Knowledge of how different parts of the CNS connected to each other can be helpful in understanding the control of movement. (From Kandel et al 1991, p. 539.)

the wiring-type diagram which gives an idea of how different parts of the CNS interact (Fig. 1.1), but this gives little insight into the contribution of different systems to the control of movement. The advent of imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have enabled a window into the CNS to provide greater insight into how tasks are organised. For example, a recent PET study by Jueptner & Weiller (1998) shows that the cerebellum is mostly concerned with processing of sensory information during an ongoing task whereas the basal ganglia are more concerned with organisation of well-learned tasks. Neurophysiologists suggest that the CNS organises the required neural activity to perform a task on the basis of past experience, but, if prior knowledge is lacking, feedback systems will play a greater role. These of necessity take longer to effect a response. Information needs to be transmitted from the periphery to supraspinal structures for processing and the result sent via efferent pathways to the spinal cord and muscles acted on.

Feedback systems are therefore less efficient and inadequate to effect fast action.

For example, take the task of drinking from a cup. There are several stages in this process. First, there needs to be a stimulus generated, either internally or externally; for example, thirst or a social situation. On the basis of past experience, the CNS organises the required strategy to achieve the goal. Perceptual aspects such as the weight, shape and texture of the cup are essential in order for the correct grip and load forces to be computed by the CNS. Spatial concepts are important for the grading and timing of postural adjustments and the actual limb movements required to take the cup to the mouth. Oral and swallowing musculature need to be coordinated with breathing in order to have the drink without choking. A decision also needs to be made when sufficient liquid has been ingested.

Although sensory information is not necessary for tasks to occur, it is important for the fine-tuning and learning of any motor/postural task. Studies on the 'deafferented man' (neuropathy of the large-diameter pathways), have shown that tasks previously experienced by the individual can be performed in the same way, but the need for repetition results in a deterioration in the performance of the task, and an inability to learn new skills (Rothwell et al 1982). This is clearly demonstrated by the inability of the 'deafferented man' to drive a new car because the gears were organised differently from the car he had driven previously (Rothwell, personal communication). This highlights the importance of the perception and processing of sensory information not only for learning but also for the efficient execution of a required task. This is important when considering training neurologically impaired patients who may have difficulties of sensory perception or sensory processing.

It seems that the CNS operates in a task- or goal-directed way, an idea embraced by therapists using a motor learning approach (Carr & Shepherd 1998). Studies using transcranial magnetic stimulation (TMS) have shown that a muscle can be activated the same amount in two tasks, e.g. power and pincer grip, but that the task is organised in a different way by the cortex;

i.e. depending on the complexity of the task, or the prior experience of the task, the CNS will select only the necessary information for its execution (Datta et al 1989, Flament et al 1993, Harrison et al 1994). These experiments have shown that the cortex plays a lesser role in simple well-practised movements such as power grip.

Cross-correlation analysis is a useful technique to study the interactions between muscles and to learn more of the neural organisation of their activity. This computer-driven analysis programme analyses the times of occurrence of motor-unit spikes and determines the probability of two motoneurons firing at or around the same time more than expected by chance alone. This technique developed by Moore et al (1970) in their study of the simple CNS of the slug (aplysia), has been successfully applied to the study of respiratory muscles and the control of human muscle activity (Sears & Stagg 1976, Bremner et al 1991, Mayston et al 1997, Farmer et al 1998). Figure 1.2 indicates the three possible

probability histograms that can be computed. The histogram in Figure 1.2a has a short duration peak around time zero, indicating that the motoneurone pools which innervate this muscle pair receive shared synaptic input either due to branched synaptic inputs or from branched common presynaptic inputs. Figure 1.2c shows a flat histogram. From this it can be inferred that the probability of firing of motoneurone A & B is always the same and if the two motoneurons do fire simultaneously such activity occurs purely by chance alone. Figure 1.2b shows a histogram with a short duration central trough, indicating shared synaptic inputs which in this case are reciprocal, i.e. excitatory to one and inhibitory to the other. In this way the reciprocal innervation circuit described by Sherrington (1906) can easily be demonstrated using simple surface electromyographic (EMG) recordings and the appropriate computer-generated software. Using this simple technique applied to surface EMG recordings, changes in motor-unit synchronisation fol-

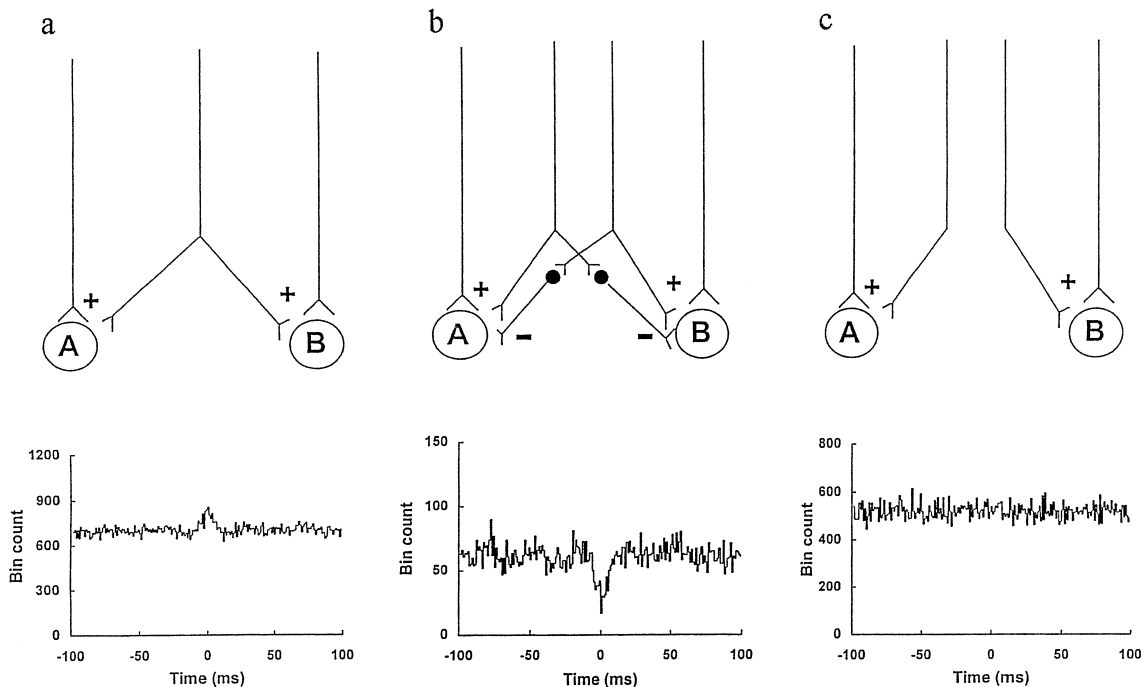


Figure 1.2 Cross-correlation analysis provides a way of examining the synaptic inputs to motoneurone pools which innervate muscle pairs.

lowing stroke have been demonstrated (Farmer et al 1993, Nadler et al 1999a). Similarly, a lack of reciprocal inhibition between antagonistic muscle pairs in healthy children younger than 5 years of age has been demonstrated and found to persist in children with spastic cerebral palsy (Mayston et al 1996, Gibbs et al 1999).

Early ideas underlying the Bobath concept emphasised the importance of reciprocal innervation circuits in the control of antagonistic muscle pairs and thus smooth coordination of movement (Bobath 1990). Bobath (1990) suggested that one of the problems for the patient with increased tone was excessive co-contraction which resulted in stiffness and slow, difficult movements for function. However, reports of abnormal co-contraction in adults with spasticity provide conflicting evidence for the presence of such co-contraction following stroke with muscle changes seemingly the primary problem in the inability to produce adequate force in the agonist, rather than antagonist restraint (Bourbonnais & Van den Noven 1989, Davies et al 1996). In contrast, for children with hypertonic

cerebral palsy, abnormal co-contraction is more common and is likely to contribute to the limb stiffness and associated difficulties in performing postural and voluntary tasks (Berger et al 1982, Leonard et al 1988, Woollacott & Burtner 1996). It is thought that for ataxia pure reciprocal inhibition without the usual overlap period of co-contraction at the reversal of movement direction results in jerky uncoordinated movement (Bobath (1997) course notes).

It is important to understand reciprocal innervation in order to appreciate how a disturbance of this mechanism may contribute to the movement problems encountered by the neurologically impaired client. Reciprocal inhibition is brought about by the reciprocal innervation circuit described by Sherrington (1906). This is shown in the simple diagrammatic representation in Figure 1.3a (Mayston 1996). It is important to note that the inhibitory interneurone which produces the inhibition of activity in the antagonistic muscle is facilitated by descending tracts, in particular the corticospinal tract. The efficiency of this reciprocal inhibition circuit

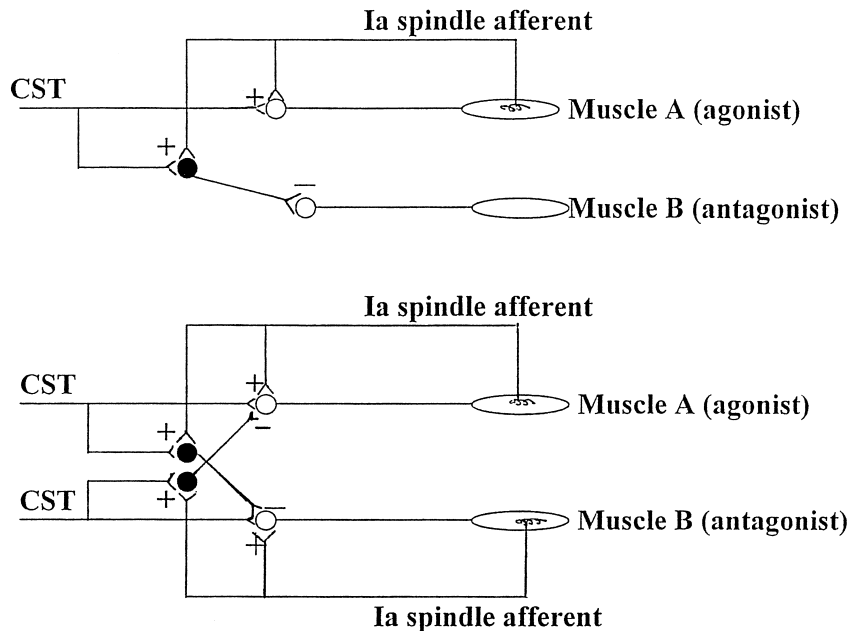


Figure 1.3 The Ia inhibitory interneurone receives input from spinal and supraspinal sources: muscle afferent (spinal) and the corticospinal tract (supraspinal).

increases with maturation of the nervous system and can be altered as a result of a cortical lesion. Reciprocal inhibition allows for the reciprocal activity of agonist and antagonist as required. For example, biceps activity is required to bend the elbow, usually occurring with the triceps relaxed (i.e. reciprocal inhibition). However, in order to produce smooth changes in the direction of the movement, the triceps co-contracts for a short time and then becomes the prime mover and the biceps is reciprocally inhibited. To explain the interaction between the agonist and antagonist when both are actively contracting, Sherrington introduced the term double reciprocal innervation, to explain how the circuits from each muscle will act simultaneously (Sherrington 1906). This is probably why Bobath (1990) emphasised the need for the healthy individual to have all degrees of reciprocal innervation in order to have well coordinated muscle activity for function. In retrospect, it is clear that Bobath placed too much emphasis on abnormalities of co-contraction in his explanation of adult neurological dysfunction, although it seems to be significant in children with spastic cerebral palsy. This is most likely because these children retain characteristics of the immature CNS, including co-contraction of the limb muscles (Forssberg 1985, Woollacott & Burtner 1996).

In summary, the neurophysiological model helps us to understand the interactions between various neural mechanisms, both central and peripheral, and indicates in particular the importance of supraspinal mechanisms for the modulation of spinal systems to produce the required control of movement.

Systems/distributed model

A therapist using a systems-based model on which to base therapy intervention helps the neurologically impaired person to problem-solve the achievement of a task goal, rather than to learn movement patterns (Shumway-Cook & Woollacott 1995). The systems approach has its origin in the work of Bernstein (1967), who suggested that an understanding of the characteristics of the system being moved (in this case the

human body), and the internal and external forces acting on it, were necessary in order to understand the neural control of movement. He suggested that the control of movements was most likely distributed throughout several cooperative and interactive systems. This has been described as the distributed model of motor control. Bernstein suggested that we have many degrees of freedom: that is, we have many joints which make several types of movement such as flexion, extension and rotation. In order for co-ordinated movement to occur, muscles are activated together in synergies such as locomotor, postural and respiratory synergies.

To use this approach as a basis for therapy, several assumptions are made (Horak 1992). The major assumption is that movements are organised around a functional goal and achieved by the interaction of multiple systems such as the sensorimotor and the musculoskeletal systems. In addition, this organisation is also determined by environmental aspects, and emphasises the importance of the interaction between the individual and the environment. The model further hypothesises that the role of sensation is important not only for adaptive control of movement but also to the predictive control of movement. Accordingly, for the neurologically impaired person, abnormal motor control results from impairments in one or more of the systems controlling movement and their resultant attempts at achieving functional goals are produced by activity of the remaining systems, which are doing the best they can. It is the therapist's task to improve the efficiency of the person's compensatory strategies to effectively perform functional tasks. While this model may be useful, some difficulty is encountered when the contribution of each system needs to be identified and evaluated.

Engineering model

This is well described by Miall (1995), who explains that the motor system has to solve problems in response to changing sensory inputs, internal goals or errors in performance. He suggests that the motor system needs to select an appropriate action, transform control signals

from sensory to motor frameworks, coordinate the selected movement with other ongoing behaviours and postural activity, and then monitor the movement to ensure its accuracy and efficacy.

In this model, the motor command is sent out to the controlled object (Fig. 1.4). In this example, the arm is the controlled object and the intended position of the arm is the reference. If the controller bases its actions on signals which are not affected by plant output (that is the sensory consequences of the action) it is said to be a feedforward controller; however, if comparisons are required – for example, between a reference signal or changing signal due to interactions with the environment – then it is a feedback controller.

This is useful for understanding how the nervous system can be both proactive and reactive, as already described in the neurophysiological model: proactive to produce activity on the basis of past performance and knowledge of outcome; and reactive to ensure that the task is executed as required in the context of the changing internal and external environments. However, there is usually a need for error correction before the command is executed and during the task performance. As Miall (1995) suggests, there are many examples of feedback control in physi-

ology, such as changes in muscle length which are detected by muscle spindles relayed to both spinal and supraspinal neural structures.

Motor systems also use this information in a feedforward way. For example, the motor command is sent to both alpha and gamma systems to ensure co-contraction of the extrafusal and intrafusal muscle fibres to enable the sensitivity of the muscle spindle to respond to unexpected load.

It must be recognised that feedback systems, although necessary for skill learning and updating of motor performance, are slow. It takes a minimum of approximately 50–100 ms for sensory information to be processed by the CNS, which for efficient postural adjustment and fine motor control is a long time.

While this is a useful model, because it assumes that the CNS acts in a linear way, there are some limitations when it is applied to brain lesions or neurophysiological recordings (Miall 1995, Loeb et al 1999).

Biomechanical model

It is possible that an overemphasis on the neural control of movement has led to a neglect of the importance of muscle strength, force production

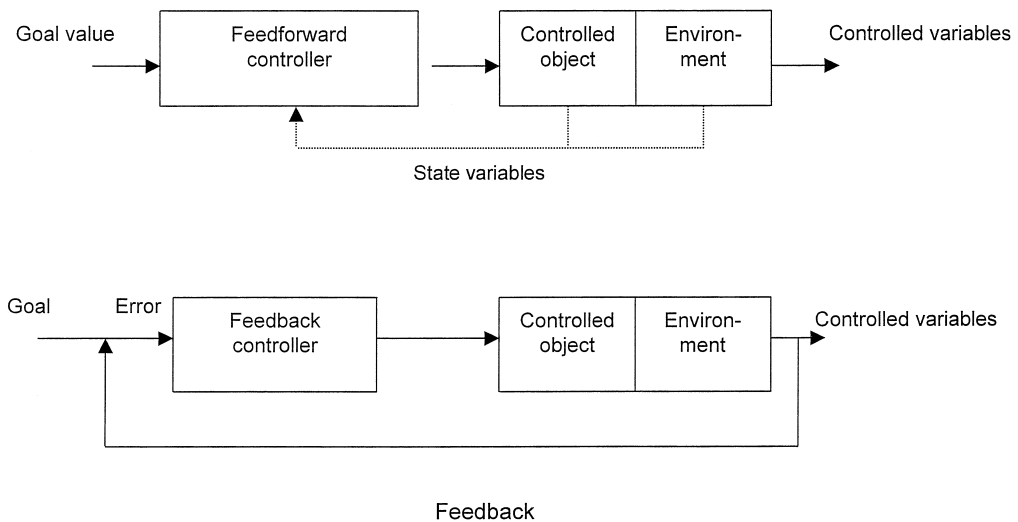


Figure 1.4 Feedforward control in the ideal situation can give perfect performance: i.e. there is no error between the reference signal and the output of the system (upper panel). A feedback system can correct performance by comparing the expected and actual outcome of a movement strategy. (Adapted from Miall 1995.)

and movement velocity. Carr & Shepherd (1998) primarily base their therapy of neurological movement disorder on principles of motor learning and biomechanics, stressing the importance of muscle length, muscle strength and activation of appropriate muscle synergies in a task-specific context. There is good evidence to support this view. Davies et al (1996) showed that a lack of force generation by paretic agonists was the major cause of reduced torque generation in a group of ambulant stroke patients. Biomechanical properties of muscle are also an important aspect of force production and changes in the distribution of muscle fibre types may also contribute to problems of force generation (Edstrom 1970, Dietz et al 1986, Ito et al 1996). It is well known that a muscle will produce optimal force at mid-range where maximal overlap of cross-bridges can occur (Rothwell 1994). Most people with neurological movement disorder demonstrate changes in muscle length which no doubt affects their ability to produce adequate force to achieve an efficient movement strategy. These changes in muscle length also alter joint alignment, which affects the ability to generate sufficient torque and efficient muscle activation patterns. It is possible that the inappropriate co-contraction of agonist and antagonist muscles results from altered biomechanical alignment *in addition* to abnormal neural control of the reciprocal inhibitory circuits between the muscle pair (Woollacott & Burtner 1996). Neurophysiotherapists must therefore consider biomechanical principles in the assessment and management of the neurologically impaired individual.

Hierarchical model

Although this model is considered outdated, it has some value when one considers the effect of the cortex on the control of movement (Lemon 1993). While it is not thought to be useful to think of higher centres controlling lower centres, the cortex is known to exert considerable control over the spinal cord and acts with subcortical areas such as the cerebellum and basal ganglia in the selection, planning and execution of motor commands (Shibasaki et al 1993, Winstein et al 1997). The

cortex though traditionally associated with the control of skilled voluntary movements, has been shown to be active during more automatic activities such as swallowing (Hamdy et al 1998) and locomotion (Schubert et al 1997, Capaday et al 1999). Another departure from the traditional view of motor control is that the spinal cord is capable of producing motor activity without any input from supraspinal centres, just as the cortex can generate commands without feedback from the periphery. This has been well described in the work on central pattern generators (Grillner 1985, Rossignol et al 1988). The central pattern generator is defined as a 'network of neurones ... able to produce a repetitive, rhythmic output ... that is independent of necessary sensory feedback' (Delcomyn 1980). In this way, the spinal cord via its networks of interneurons and motoneurons, can produce rhythmical, alternating lower limb movements which are the basis of locomotor activity (walking). On the other hand, fractionated finger movements necessary for fine hand control rely on the integrity of the corticospinal tract for their efficiency and are largely under cortical control (Lemon 1993, Olivier et al 1997). It is well known that a lesion affecting the corticospinal tract results in deficits of independent finger movements (Kuypers 1978, Galea & Darian-Smith 1997, Farmer et al 1993).

The view that the cortex has an important influence on control of the spinal cord's organisation of movement is also reflected in reflex studies. Matthews (1991) presents a comprehensive review of the human stretch reflex which consists of a short latency component (M1) and a long latency component (M2). This paper reviews the evidence from studies of latencies of reflex components, lesions and stimulation techniques which show that the simple stretch reflex is more complex than originally proposed by Liddell & Sherrington (1924, as cited in Matthews 1991). Matthews (1991) presents robust evidence for a transcortical route for the transmission of the long latency M2 component.

IMPLICATIONS FOR THE THERAPIST

Successful performance of a sensorimotor task requires the integrated action of the CNS.

Descending commands from the brain interact with spinal neuronal circuits, and incorporate the dynamic properties of muscles and activity of somatosensory receptors (Loeb et al 1999). From the previous discussion it can be concluded that no one model is sufficient for the therapist to apply a problem-solving approach to the management of the neurologically impaired person. The musculoskeletal system is critical to the execution of the motor command, in addition to the various cortical and subcortical areas involved in the organisation of the task. Therapists must understand the nature of the movement disorder to employ effective treatment strategies and to set appropriate goals for those individuals to maximise the potential for functional independence.

Which therapy approach?

It is thought that approximately 88% of therapists in the UK base their intervention on the Bobath concept (Sackley & Lincoln 1996, Davidson & Waters 2000). Although there have been changes to the underlying basis of the concept (Mayston 1992), the lack of relevant literature has resulted in many misconceptions and continuation of outdated ideas, such as an emphasis on reflex activity as a basis of tone and postural activity, a correspondingly misplaced emphasis on the inhibition of spasticity and an overemphasis on the significance of righting and equilibrium reactions. The following discussion attempts to clarify some of the basic ideas underlying the Bobath approach to the management of people with neurological movement disorder.

Normal and abnormal tone

It is clear from the neurophysiological and biomechanical models of motor control that the muscles themselves are important contributors to the concept of tone. The original idea proposed by Sherrington (1906) and adopted by Bobath (1990) that tone is the result of tonic reflex activity is now outdated. Tone comprises both neural and non-neural components (Basmajian et al 1985). Various definitions lead the therapist to

realise that this is the case. Basmajian et al (1985) states 'at rest a muscle has not lost its tone although there is no electrical activity in it'. Ghez (1991) describes tone as 'a slight constant tension of healthy muscles'. The definition by Bernstein (1967) that describes tone as a state of readiness seems a useful explanation. Different individuals can have differing states of readiness, as do patients with movement disorder: for example, the person with hypotonia has a reduced state of readiness, whereas the person with spasticity/hypertonia may be said to have an increased state of readiness. If tone is an important aspect of the control of movement, all factors contributing to it must be taken into account. Tone is not simply produced by tonic reflex activity – viscoelastic properties of muscle are equally important. This has significance for the movement problems of the patient with abnormal tone. It is now known that muscles which are thought to be hypertonic are in fact not usually overactive but cannot generate sufficient electrical activity to exert a force about a joint or to produce a movement (Davies et al 1996).

The controversy regarding the use of the terms 'spasticity' and 'hypertonia' is discussed in Chapter 5, but the therapist must ask this question: Am I managing spasticity, hypertonia or both?

An example of clinical practice may help to clarify the dilemma. Recently a 12-year-old child was referred for physiotherapy because of increasing 'spasticity' for which baclofen (an antispastic agent; see Chapter 7) had not been helpful. This girl presented with increasing stiffness shown by an increased flexion posture of the lower limbs and resistance to extension. Is the increased stiffness due to:

- a lack of power in anti-gravity extensor muscles and associated changes in viscoelastic muscle properties which has resulted in contractures and apparently increased tone over time, or
- is the increased stiffness due to a velocity-dependent increase in hyperreflexia?

After assessment it was clear that the major factor causing increased stiffness was muscle

weakness and contracture. Therefore it was not surprising that baclofen had no effect in this case. Careful assessment of what is true spasticity as opposed to weakness, loss of dexterity and contracture (stiffness) is thus essential and may require specific testing, for example using EMG recordings, in order to be accurately determined.

Are inhibitory techniques relevant?

This altered view of tone must influence the way the therapist manages the person with abnormally increased tone. The EMG traces in Figure 1.5a show the activity recorded from the quadriceps and hamstrings of a 10-year-old child during free standing, only possible with some flexion of the hips and knees. When the child is aligned so that the hips and knees are extended, the hamstrings are no longer active and the quadriceps generate larger spike EMG, thus activating larger motor units which results in more dynamic postural activity (Fig. 1.5b). Has this child's hypertonia been 'inhibited', or rather does he now have more appropriate alignment to allow more efficient activation of the quadriceps muscle and hip extensors?

The word 'inhibition' poses many problems. Tone may be influenced (reduced) by elongating and mobilising stiff, tight joints and muscles to enable optimal activation from the required

muscles, but this is not inhibition as understood by physiologists. Inhibition in neurophysiological terms means that synapses are weakened due to reduced transmitter release or that activity in a synapse is dampened down. There are many examples of inhibition in the CNS: for example reciprocal 1a inhibition, lateral inhibition, Renshaw cell inhibition, pre- and post-synaptic inhibition. The term 'inhibition' was introduced by Bobath to explain tone reduction commensurate with the idea that hypertonia was produced by abnormal tonic reflex activity (Bobath 1990). This view can no longer be supported. Bobath therapists achieve tone reduction in various ways: mobilisation of tight joints and muscles, muscle stretch, practice of more normal movements (whole or part practice) and functional tasks.

The changes in explanations of tone and techniques of handling as viewed by paediatric Bobath therapists are summarised in Table 1.1 which shows how the understanding of abnormally increased tone has changed over several decades. Accordingly, the explanation underlying the treatment technique has also changed. It has been suggested that therapists do not so much need to change what they do, but to rethink the explanations for what they do (Gentile, personal communication). Another misleading term related to 'inhibition' is the tech-

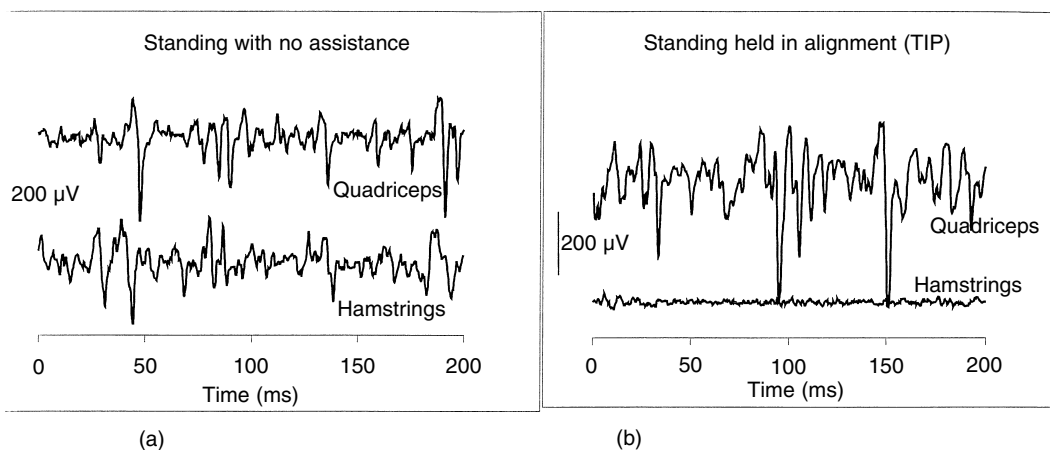


Figure 1.5 The electromyographic (EMG) activity recorded from a 10-year-old child with spastic diplegia standing without support in a typically flexed posture (a) and when held with the hips and knees extended (b). TIP = tone influencing pattern.

Table 1.1 Tone and techniques of handling

<i>Abnormal postural tone</i>	<i>Handling technique</i>	<i>Aim of use of technique</i>	<i>Comment</i>
Released tonic reflexes (1940s)	Reflex inhibiting postures (RIPs)	Inhibition of released tonic reflexes	Static – little or no movements; often opposite to pattern of spasticity
Abnormal tonic (postural) reflex activity (1960s)	Reflex inhibiting patterns (RIPs)	Simultaneous inhibition, facilitation & stimulation	Emphasis on facilitation of postural reactions
Abnormal neural and non-neural aspects of tone (1990 – present)	Tone influencing patterns (TIPs)	'Inhibition', facilitation, stimulation and biomechanical influences	Influence both the control of posture and task performance

nique of specific inhibitory mobilisations (SIMs) introduced by adult Bobath therapists. SIMs apparently stretch tight ligaments and tendons, and are therefore not inhibitory in the physiological sense. The activity of every motoneurone pool depends on the sum of the inhibitory and excitatory inputs at any moment in time. It is by altering sensory feedback due to altered task performance that the CNS, if it has the capacity to adapt, will then provide the neurologically impaired person with the possibility to move more efficiently or to regain lost skills. This neural approach to client management needs to be integrated with a biomechanical approach which takes into account the importance of muscle length, strength and joint alignment.

Postural control

The early work of the Bobaths placed a great emphasis on postural reactions, namely the righting, equilibrium and protective reactions (Bobath & Bobath 1964, 1975). They proposed that postural adjustment took place before, during and after an action, an idea shown by researchers in the postural control field (Massion 1994, Gatev et al 1999).

Unfortunately some users of the Bobath approach are still dominated by an overemphasis on these reactions, and even these are not always clearly understood. Bobath therapy is not facilitation of balance reactions, although this is the perception of some workers (Palmer et al 1988). It is perhaps important to review these reactions before a broader discussion of balance. The righting reactions are a discrete group of reactions which are only seen in the developing infant and in specific animal preparations. In the mature adult these righting reactions cannot be separated from the more complex equilibrium reactions (Bryce 1972). It is therefore incorrect to look for head righting or trunk righting in the mature adult, but rather one should determine whether an individual has the appropriate activity of the head and trunk within the equilibrium response. The equilibrium reactions are either:

- invisible changes in muscle tone which enable the maintenance of the desired posture,
- or, when greater perturbation necessitates visible activity, the response of the body being to extend/elongate the weight-bearing side with flexion of the non-weight-bearing side with some rotation within the body axis. The degree of rotation depends on the direction of the perturbation. When the perturbation is too large or too fast then the protective reactions come in to protect the individual from injury and to assist in restoration of the centre of gravity to lie within the base of support.

In summary, balance in the mature adult is achieved by equilibrium and protective reactions; righting reactions cannot be observed. In the developing infant the various righting reactions can be observed, but early in development become a part of the equilibrium reactions which commence in prone at approximately 3 months of age when the infant can maintain the prone position with head lifting and weight on elbows.

For several years, balance has been viewed in a functional way by the Bobath Centre, London, recognising that the central command for an action includes both the postural and task-related components (Rothwell 1994). Balance reactions are complex responses based on prior

experience in addition to the CNS response to unexpected perturbations occurring during task execution (Horak & Nashner 1986). There seems to be much controversy about how balance should be trained in people with neurological movement disorder. Should balance be trained separately or as part of the task goal? For the developing infant the experience of a posture precedes the attainment of postural control in that posture. For example, an infant is propped in sitting to practise using their hands before independent sitting is possible. Thus, for some patients, it could be reasonable to assume that it is necessary to give them the idea of the postural activity required for a task and then to add in the task component. Both components need to be practised simultaneously for the training to be effective. Similarly, testing of sitting balance is not achieved by testing righting and equilibrium reactions, but rather by assessing the person's possibilities to reach in all directions for objects or to carry out activities as in the performance of tasks of daily life. This ability relies not only on sensorimotor activity but also on the perceptual ability of the individual (Massion 1994), which should be considered as part of the postural mechanism to be taken into account during therapy and the goals adjusted accordingly. If a person's instability is primarily caused by a perceptual deficit, simply training balance reactions will not address the main problem.

It would be preferable to view balance as an adaptable background to skill performance and to train it in the appropriate functional context, rather than emphasising the different groups of reactions (righting, equilibrium and protective) as being responsible for postural control.

Compensation

Compensation is another term which has different meaning for therapists, neurologists and movement scientists. If the nervous system is damaged in some way, then there will necessarily be compensation by the system for the damage sustained. This can take many forms, which may include plastic changes such as muscle adaptation and cortical reorganisation.

How the patient moves in response to their neurological reorganisation is another question. Shepherd & Carr (1991) suggest that the way in which the neurologically impaired person attempts to achieve a goal represents the best that can be done given the state of the neural and musculoskeletal systems. The questions we might ask are: How much does the person need to compensate? Can they function more efficiently and compensate less? For example, a stroke patient will prefer to use the unaffected side, only using the stroke-affected side when absolutely necessary and only if physically possible. However, the work of Bobath (1990) and evidence provided by Taub & Wolf (1997) has shown that by training of the stroke-affected side it is possible that, for some patients, fewer compensatory movements will be required because more effective movement is possible on the stroke side. No therapist should try and stop a patient moving in a certain way unless they can replace it with an alternative strategy which achieves the same goal. Concern for quality of movement needs to be realistic.

Associated reactions

Associated reactions (see Chapter 5) are another example of confusion in neurophysiotherapy and represent one of the greatest controversies and possibly mysteries in the neurological therapeutic world in the UK (Stephenson et al 1998).

Early positioning and the avoidance of effort were advocated by Bobath (1990) to reduce the effect of associated reactions which in the long-term might lead to contracture and reduce the potential for functional recovery. The main features of the management of these reactions in the more able client were:

- The client should be taught strategies to reduce them when they occurred. For example, using the sound arm to stretch out the affected side.
- To train more normal activity of the affected side to reduce effort and therefore the severity of the associated reactions. It was suggested that improving balance on the stroke-affected

side could lead to less effort in the maintenance of balance and less increase of tone in the upper limb associated with the need to balance.

However, there is no evidence to suggest that preventing a person who has had a stroke from moving in the early stages of recovery will influence spasticity and associated reactions; in fact, it may be detrimental to the client's potential for CNS recovery and thus functional recovery.

THE NATURE OF THE MOVEMENT DISORDER

It would seem that therapists have become so enthusiastic in the control of tone that other factors, such as weakness and dexterity, have assumed less importance. But a purely biomechanical view cannot be supported either. Neural damage that results in dysfunction of cortical and subcortical areas, particularly the descending tracts, reduces neural drive onto the motoneurone pool and results in reduced force generation which will not necessarily be regained. Thus there will always be a degree of weakness and loss of power. Muscle imbalance will be accompanied by muscle shortening, another contributor to lost ability to generate force – for example in walking (Ada et al 1998).

It has been shown in children with cerebral palsy (CP) that the inability selectively to activate muscles is in part due to a lack of synchronisation of muscles (Gibbs et al 1999). Axons usually branch to innervate several motoneurone pools to bring about the cooperative action of the muscles for a required task (Bremner et al 1991), or are activated synchronously if flexible strategies are required by the task (Gibbs et al 1995). This is one aspect of function of the corticospinal tract known to be disrupted when there is brain injury. Abnormal synchronisation of motor unit activity has been demonstrated in people with dystonia (Farmer et al 1998) and those with hemiplegic stroke (Farmer et al 1993, Nadler et al 1999), although the functional significance of this is unclear. Another aspect of the movement disorder associated with spasticity in children is

co-contraction of antagonistic muscle pairs (Leonard et al 1991, Woollacott & Burtner 1996, Gibbs et al 1999). There are varying reports of co-contraction of antagonistic muscle pairs in adults with hypertonia, but the phenomenon seems less common. It is likely that weakness and altered viscoelastic properties of muscle are a more likely explanation of the stiffness experienced and felt in adult patients with increased tone (Gowland et al 1992, Davies et al 1996).

THE WAY FORWARD

Neuroplasticity

Plasticity underlies all skill learning and is a part of CNS function in healthy and brain-damaged individuals at any age (Leonard 1998).

The advent of imaging techniques such as PET and fMRI in conjunction with neurophysiological recordings in primates and humans has provided evidence of the plasticity of the CNS. In a study of monkeys following amputation of digit 3, it was shown that adjacent areas of the sensory cortex expanded to take over the representation of the lost digit (Merzenich et al 1984). Plasticity of the sensory cortex has also been induced through behavioural training. The tips of the second and third fingers were stimulated with a rotating disk, which resulted in an expansion of the sensory representation of those digits (Jenkins et al 1990). This suggests that sensory stimulation, if given effectively and often enough, can expand sensory areas of the cortex and may have implications for therapy.

Plastic changes have also been demonstrated in the motor system as a result of motor training. Recent work by Nudo and his group (Nudo et al 1992) has shown that training a hand expanded the cortical areas represented by the muscles executing that task. A later study by his group has shown that lesioning the motor cortex of a monkey and then training motor activity during recovery resulted in greater recovery of skill than the untrained group, and reduced loss of cortical tissue in the area surrounding the infarct (Nudo et al 1996). The effect of training of a novel motor skill in healthy human adults has also

demonstrated changes in sensorimotor function (Nadler et al 1998). In this study subjects were trained to simultaneously flex the index finger (first dorsal interosseous; 1DI) and abduct the fifth finger (abductor digiti minimi; ADM). Before, during and after the training period, cutaneous reflexes in response to stimulation of the digital nerves of the index finger were recorded. After a short period of training (2–3 days), the long-latency components of the reflex were significantly larger. This indicates that the sensory fields of the two muscles had expanded and come to lie closer together in the sensory cortex, so that the sensory input now reached the two muscles rather than just the 1DI. This correlate of Nudo's work in training motor skill in monkeys (Nudo et al 1992) suggests that motor training and skill learning can be detected using simple reflex testing and may be useful as a means of monitoring the effects of therapy in clients with neurological disability.

Skill learning

Practice is fundamental for motor learning and improving skill in both healthy and movement-impaired individuals (Taub et al 1993, Winstein et al 1997). Two other principles of equal significance are active participation and working to achieve meaningful goals. Therapy programmes should be based on these three principles and can be enhanced by 'preparation'.

The Bobath approach has been much criticised for its use of preparation for function (Shepherd 1995, Carr & Shepherd 1998), but this has been misunderstood. Preparation given as a treatment is of no value on its own, and must be incorporated into useful activity (Bobath 1965, unpublished notes). It includes the following:

- mobilisation of tight connective tissue and/or joints
- elongating muscles to enable activity from a better biomechanical advantage, to achieve better body alignment for more efficient balance and muscle activation
- practice of task-component parts to enable the patient to get the idea of the movement required

- practising in a functional task which the patient wants to achieve. To do this requires realistic goal setting.

Bobath (personal communication) suggested that it is what the neurologically impaired person can do with some assistance that is their potential. However, it is of little use to the person if these potentially achievable skills can only be practised with the therapist's help. When required, it is appropriate and important to enlist the help of others to enable the person to practise activities which are possible, with a little help, to achieve independently. Equally it is of no use to the person to be prevented from trying to practise activities because there is a danger of increasing spasticity through the occurrence of associated reactions. Indications are that early training will enable less secondary loss of cortical tissue and thus enable greater possibilities for recovery (Nudo et al 1996).

Can we predict outcomes?

Part of the realistic setting of goals (see Chapter 2) depends on having realistic expectations of the individual's optimal potential based on the therapist's expertise. However, neurophysiological tests such as TMS and reflex testing may also be used to predict recovery. Turton et al (1996) in their study were able to identify two patient groups (A = rapid recovery; B = slow and incomplete recovery) and further categorised them on the basis of EMG recordings and responses to TMS. While responses to TMS could be elicited from all muscles in group A from the outset, in the slow recovery group, the ability to elicit TMS responses was commensurate with the subsequent activation of hand muscles. In this way TMS provided a prognostic test for the return of muscle activity.

Nadler et al (1999b) studied the cutaneous reflexes (see Jenner & Stephens 1982) of a small cohort of people who had a stroke. Their results suggest that those subjects in whom a large short-latency reflex response is recorded are unlikely to make a good recovery. Similarly, stroke patients who present with transient mirror

movements early in recovery usually regain good function of that side (clinical observation).

However, not all therapy departments have access to these diagnostic and prognostic tools; therefore, for the moment, the clinical experience

and the problem-solving ability of the therapist in the context of a knowledge and understanding of current research literature remains the main way to determine realistic goals for each client's management.

REFERENCES

- Ada L, Vattanaslip W, O'Dwyer N J, Crosbie J 1998 Does spasticity contribute to walking dysfunction after stroke. *Journal of Neurology, Neurosurgery and Psychiatry* 64: 628–635
- Basmajian J V, De Luca C J 1985 *Muscles alive. Their functions revealed by electromyography*. Williams and Wilkins, Baltimore, ch 10 and 11
- Berger W, Quintern J, Dietz V 1982 Pathophysiology of gait in children with cerebral palsy. *Electroencephalography and Clinical Neurophysiology* 53: 538–548
- Bernstein N 1967 *The co-ordination and regulation of movements*. Pergamon, Oxford
- Bobath B 1985 *Abnormal postural reflex activity caused by brain lesions*, 3rd edn. William Heinemann Medical Books, London
- Bobath B 1990 *Adult hemiplegia: evaluation and treatment*, 3rd edn. Heinemann Medical Books, London
- Bobath B 1997 *Bobath course notes (8-week paediatric course)*. The Bobath Centre, London
- Bobath B, Bobath K 1964 The facilitation of normal postural reactions and movements in the treatment of cerebral palsy. *Physiotherapy* 50: 246–262
- Bobath B, Bobath K 1975 *Motor development in the different types of cerebral palsy*. Heinemann Medical Books, London
- Bourbonnais D, Van den Noyen S 1989 Weakness in patients with hemiparesis. *American Journal of Occupational Therapy* 43: 676–685
- Bremner F D, Baker J R, Stephens J A 1991 Correlation between the discharges of motor units recorded from the same and from different finger muscles in man. *Journal of Physiology (London)* 432: 355–380
- Bryce J 1972 Facilitation of movement – the Bobath approach. *Physiotherapy* 58: 403–408
- Capaday C, Lavoie B A, Barbeau H, Schneider C, Bonnard M 1999 Studies on the corticospinal control of human walking. 1. Responses to focal transcranial magnetic stimulation. *Journal of Neurophysiology* 81: 129–139
- Carr J, Shepherd R 1998 *Neurological rehabilitation – optimizing motor performance*. Butterworth-Heinemann, Oxford
- Datta A K, Harrison L M, Stephens J A 1989 Task-dependent changes in the size of response to magnetic brain stimulation in human first dorsal interosseous muscle. *Journal of Physiology (London)* 418: 13–23
- Davidson I, Waters K 2000 Physiotherapists working with stroke patients: a national survey. *Physiotherapy* 86: 69–80
- Davies J M, Mayston M J, Newham D J 1996 Electrical and mechanical output of the knee muscles during isometric and isokinetic activity in stroke and healthy adults. *Disability and Rehabilitation* 18: 83–90
- Deecke L, Scheid P, Kornhuber H H 1969 Distribution of readiness potential, pre-motion positivity, and motor potential of the human cerebral cortex preceding voluntary finger movements. *Experimental Brain Research* 7: 158–168
- Delcomyn F 1980 Neural basis of rhythmic behaviour in animals. *Science* 210: 492–498
- Dietz V 1992 Human neuronal control of automatic functional movements: interaction between central programs and afferent input. *Physiological Review* 72: 33–69
- Dietz V, Ketelsen U P, Berger W, Quintern J 1986 Motor unit involvement in spastic paresis. Relationship between leg muscle activation and histochemistry. *Journal of Neurological Science* 75: 89–103
- Edstrom L 1970 Selective changes in the sizes of red and white muscle fibres in upper motor lesions and parkinsonism. *Journal of Neuroscience* 11: 537–550
- Farmer S F, Swash M, Ingram D A, Stephens J A 1993 Changes in motor unit synchronization following central nervous lesions in man. *Journal of Physiology (London)* 463: 83–105
- Farmer S F, Sheehan G L, Mayston M J et al 1998 Abnormal motor unit synchronization of antagonist muscles underlies pathological co-contraction in upper limb dystonia. *Brain* 121: 801–814
- Flament D, Goldsmith P, Buckley C J, Lemon R N 1993 Task dependence of responses in first dorsal interosseous muscles to magnetic brain stimulation in man. *Journal of Physiology (London)* 464: 361–378
- Forrai 1999 *Memoirs of the beginnings of conductive pedagogy and Andras Peto*. Uj Aranyhid Budapest and the Foundation of Conductive Education, Birmingham
- Forssberg H 1985 Ontogeny of human locomotor control. I. Infant stepping, supported locomotion and transition to independent locomotion. *Experimental Brain Research* 57: 480–493
- Foster M 1985 *A textbook of physiology*. MacMillan, New York
- Galea M G, Darian-Smith I 1997 Manual dexterity and corticospinal connectivity following unilateral lesion of the cervical spinal cord in the macaque monkey. *Journal of Comparative Neurology* 381: 307–319
- Gatev P, Thomas S, Kepple T, Hallett M 1999 Feedforward ankle strategy of balance during quiet stance in adults. *Journal of Physiology (London)* 514: 915–928
- Ghez C 1991 *Muscles: effectors of the motor systems*. In: Kandel E R, Schwartz J H, Jessell T M (eds) *Principles of neuroscience*, 3rd edn. Elsevier, Amsterdam.

- Gibbs J, Harrison L M, Stephens J A 1995 Organization of inputs to motoneurone pools in man. *Journal of Physiology* (London) 485: 245–256
- Gibbs J, Harrison L M, Stephens J A 1999 Does abnormal branching of inputs to motor neurones explain abnormal muscle cocontraction in cerebral palsy? *Developmental Medicine and Child Neurology* 41: 465–472
- Gowland C, de Bruin H, Basmajian J V, Plews N, Burcea I 1992 Agonist and antagonist activity during voluntary upper limb movement in patients with stroke. *Physical Therapy* 43: 624–633
- Grillner S 1985 Neurobiological bases of rhythmic motor acts in vertebrates. *Science* 228: 143–149
- Hamdy S, Rothwell J C, Quasim A, Singh K D, Thompson D G 1998 Long-term reorganisation of human motor cortex driven by short-term sensory stimulation. *Nature Neuroscience* 1: 64–68
- Harrison L M, Mayston M J, Gibbs J, Stephens J A 1994 Central mechanisms underlying task dependence of cutaneous reflexes in man. *Journal of Physiology* 476: 18P
- Horak F B 1992 Motor control models underlying neurologic rehabilitation of posture in children. In: Forsberg H, Hirschfeld H (eds) *Movement disorders in children*. Medicine and sport science series. Karger, Basel, Vol. 36, 21–30
- Horak F B, Nashner L M 1986 Central programming of postural movements: adaptation to altered support-surface configurations. *Journal of Neurophysiology* 55: 1369–1381
- Ito J, Araki A, Tanaka H, Tasaki T, Cho K, Yamazaki R 1996 Muscle histopathology in spastic cerebral palsy. *Brain Development* 18: 299–303
- Jenkins W M, Merzenich M M, Ochs M T, Allard T, Guic-Robles E 1990 Functional reorganisation of primary somatosensory cortex in adult owl monkeys after behaviourally controlled tactile stimulation. *Journal of Neurophysiology* 63: 82–104
- Jenner J R, Stephens J A 1982 Cutaneous reflex responses and their central nervous pathways studied in man. *Journal of Physiology* (London) 333: 405–419
- Jueptner M, Weiller C 1998 A review of differences between basal ganglia and cerebellar control of movements as revealed by functional imaging studies. *Brain* 121: 1437–1449
- Kabat H, Knott M 1954 Proprioceptive facilitation therapy for paralysis. *Physiotherapy* 40: 171–176
- Kandel E R, Schwartz J H, Jessell 1991 *Principles of neural science*, chs 36–40, 3rd edn. Elsevier, Amsterdam
- Keele S W 1968 Movement control in skilled motor performance. *Psychology Bulletin* 70: 387–403
- Kristeva Feige R, Walter H, Lutkenhoner B et al 1994 A neuromagnetic study of the functional organization of the sensorimotor cortex. *European Journal of Neuroscience* 6: 632–639
- Kuypers H G 1978 The motor system and the capacity to execute highly fractionated distal extremity movements. *Electroencephalography and Clinical Neurophysiology Supplement* 429–431
- Lemon R N 1993 The G. L. Brown Prize Lecture. Cortical control of the primate hand. *Experimental Physiology* 78: 263–301
- Leonard C T 1998 *The neuroscience of human movement*. Mosby, St Louis
- Leonard C T, Hirschfeld H, Forssberg H 1988 Gait acquisition and reflex abnormalities in normal children and children with cerebral palsy. In: Amblard B, Berthoz A, Clarac F (eds) *Posture and gait: development, adaptation and modulation*. Elsevier Science (Biomedical Division), Amsterdam
- Leonard C T, Hirschfeld H, Forssberg H 1991 The development of independent walking in children with cerebral palsy. *Developmental Medicine and Child Neurology* 33: 567–577
- Loeb G E, Brown I E, Cheng E J 1999 A hierarchical foundation for models of sensorimotor control. *Experimental Brain Research* 126: 1–18
- Martenuik R G, MacKenzie C L, Jennerod M, Athenes S, Dugas C 1987 Constraints on human arm movement trajectories. *Canadian Journal of Psychology* 41: 365–378
- Massion J 1994 Postural control system. *Current Opinions in Neurobiology* 4: 877–887
- Mathews P B 1991 The human stretch reflex and the motor cortex. *Trends in Neuroscience* 14(3): 87–89
- Mayston M J 1992 The Bobath concept – evolution and application. In: Forsberg H, Hirschfeld H (eds) *Movement disorders in children*. Karger, Basel
- Mayston M J 1996 *Mechanisms underlying cocontraction during development and in pathology in man*. PhD thesis, University of London
- Mayston M J, Harrison L M, Stephens J A 1996 Co-contraction of antagonistic muscles during development and in children with cerebral palsy. *Journal of Physiology* 494: 67P
- Mayston M J, Harrison L M, Quinton R, Krams M, Bouloux P-M G, Stephens J A 1997 Mirror movements in X-linked Kallmann's syndrome. I. A neurophysiological study. *Brain* 120: 1199–1216
- Merzenich M, Nelson R J, Stryker M P, Cynader M S, Schoppmann A, Zook J M 1984 Somatosensory map changes following digit amputation in adult monkeys. *Journal of Comparative Neurology* 224: 591–605
- Miall R 1995 In: Arbib M (ed) *The handbook of brain theory and neural networks*. MIT Press, Cambridge, Massachusetts, pp 597–600
- Moore G P, Segundo J P, Perkel D H, Levitan H 1970 Statistical signs of synaptic interaction in neurons. *Biophysics Journal* 10: 876–900
- Morris M E, Summers J J, Matyas T A, Ianssek R 1994 Current status of the motor program. *Physical Therapy* 74: 738–748
- Nadler M A, Harrison L M, Moore M, Townend E, Stephens J A 1998 Acquisition of a new motor skill is accompanied by changes in cutaneous reflexes recorded from finger muscles in man. *Journal of Physiology* 511 P
- Nadler M A, Harrison L M, Stephens J A 1999a Motor-unit synchronization between pairs of hand muscles is latered following stroke in man. *Journal of Physiology* 518P: 63P
- Nadler M A, Harrison L M, Stephens J A 1999b Changes in cutaneous reflexes following stroke in man: a two year longitudinal study. *Society for Neuroscience Abstracts* 52.14
- Nudo R J, Jenkins W M, Merzenich M M 1992 Neurophysiological correlates of hand preference in primary motor cortex of adult squirrel monkeys. *Journal of Neuroscience* 12: 2918–2947
- Nudo R J, Wise B M, SiFuentes F, Milliken G W 1996 Neural substrates for the effects of rehabilitative training on

- motor recovery after ischaemic infarct. *Science* 272: 1791–1794
- Olivier E, Edgley S A, Armand J, Lemon R N 1997 An electrophysiological study of the postnatal development of the corticospinal system in the macaque monkey. *Journal of Neuroscience* 17: 267–276
- Palmer F B, Shapiro B K, Wachtel R C et al 1988 The effects of physical therapy on cerebral palsy: a controlled trial in infants with spastic diplegia. *New England Journal of Medicine* 318: 803–808
- Roland P E, Larsen B, Lassen N A, Skinhoj E 1980 Supplementary motor area and other cortical areas in organization of voluntary movements in man. *Journal of Neurophysiology* 43: 118–136
- Rood M S 1954 Neurophysiologic reactions: a basis for physical therapy. *Physical Therapy Review* 34: 444–449
- Rossignol S, Lund J P, Drew T 1988 The role of sensory inputs in regulating patterns of rhythmical movements in higher vertebrates. A comparison between locomotion, respiration and mastication. In: Cohen A H, Rossignol S, Grillner S (eds) *Neural control of rhythmic movements in vertebrates*. Wiley, New York
- Rothwell J C 1994 *Control of human voluntary movement*, 2nd edn. Chapman Hall, London
- Rothwell J C, Traub M M, Day B L, Obeso J A, Thomas P K, Marsden C D 1982 Manual motor performance in a deafferented man. *Brain* 105: 525–542
- Sackley C M, Lincoln N B 1996 *Physiotherapy for stroke patients: a survey of current practice*. *Physiotherapy Theory and Practice* 12: 87–96
- Schmidt R A 1991 *Motor learning and performance: from principles to practice*. Human Kinetics Publishers, Leeds
- Schubert M, Curt A, Jensen L, Dietz V 1997 Corticospinal input in human gait: modulation of magnetically evoked responses. *Experimental Brain Research* 115: 234–246
- Sears T A, Stagg D 1976 Short-term synchronization of intercostal motoneurone activity. *Journal of Physiology (London)* 263: 357–381
- Shepherd R B 1995 *Physiotherapy in paediatrics*. Butterworth-Heinemann, Oxford
- Shepherd R B, Carr J H 1991 An emergent or dynamical systems view of movement dysfunction. *Australian Journal of Physiotherapy* 37: 5–17
- Sherrington C S 1906 *The integrative action of the nervous system*. Yale University Press, New Haven
- Shibasaki H, Sadato N, Lyshkow H et al 1993 Both primary motor cortex and supplementary motor area play an important role in complex finger movement. *Brain* 116: 1387–1398
- Shibasaki H, Nagae K 1984 Mirror movements: application of movement-related cortical potentials. *Annals of Neurology* 15: 299–302
- Shumway-Cook A, Woollacott M 1995 *Motor control – new models for rehabilitation* Williams and Williams, Baltimore
- Stephenson R, Edwards S, Freeman J 1998 Associated reactions: their value in clinical practice? *Physiotherapy Research International* 3: 69–78
- Taub E, Wolf S L 1997 Constraint induced techniques to facilitate upper extremity use in stroke patients. *Topics in Stroke Rehabilitation* 3: 38–61
- Taub E, Miller N E, Novack T A 1993 A technique for improving chronic motor deficit after stroke. *Archives of Physical Medicine and Rehabilitation* 74: 347–354
- Turton A, Wroe S, Trepte N, Fraser C, Lemon R N 1996 Contralateral and ipsilateral EMG responses to transcranial magnetic stimulation during recovery of arm and hand function after stroke. *Electroencephalography and Clinical Neurophysiology* 101: 316–328
- Voss D E 1967 Proprioceptive neuromuscular facilitation. *American Journal of Physical Medicine* 46: 838–898
- Winstein C J, Merians A, Sullivan K 1997 Motor learning after unilateral brain damage. *Neuropsychologia* 37: 975–987
- Woollacott M H, Burtner P 1996 Neural and musculoskeletal contributions to the development of stance control in typical children and children with cerebral palsy. *Acta Paediatrica Scandinavica Supplement* 416: 58–62

