Another look at neurological rehabilitation

Delva Bethune

Advances in neurophysiological research over the past 20 years have led to dramatic changes in the understanding of the neural control of movement. These newer concepts have directed attention towards possibilities for motor recovery previously discounted. They also imply that changes in health care delivery may be necessary in order to take advantage of the recovery process and achieve maximum potential. If the neurologically disabled are to benefit from the advances of science, and if physical therapy practitioners are to reach and maintain scientific credibility, some fundamental changes in clinical thinking and practice seem indicated. This article discusses some implications for the practice of physical therapy in neurology.

[Bethune D: Another look at neurological rehabilitation. *Australian Journal of Physiotherapy* 40: 255-261]

Key words: Muscle Tonus; Neurology; Neurophysiology; Rehabilitation

D Bethune MHSc, DipPT, DipOT is Associate Professor in the Physical Therapy Division of the School of Rehabilitation Therapy at Queen's University, Kingston, Ontario.

Correspondence: Associate Professor D Bethune, Physical Therapy Division, School of Rehabilitation Therapy, Faculty of Medicine, Queen's University, Kingston, Ontario K7L 3N6, Canada.

"Recent information on the genesis and control of human movement calls for a new neurology with fundamental changes in our thinking about disorders of movement, their classification and how they are to be studied and treated."

Grimm, 1983

People with neurological disorders comprise one of the largest populations in rehabilitation hospitals. The vast majority of such disorders require more than crises intervention. The complexity of the problems, the persistence of the symptoms and the necessity for a longer rather than a shorter rehabilitation process are contributing factors to incomplete functional recovery of the victims. Professional burn-out of therapists and nurses working with the chronic neurologically impaired individuals and the increasing tendency in some regions to define the possible rehabilitation potential in terms of the duration of insurance company payouts are other contributing factors. Practical factors must be acknowledged, but in order for those affected to gain maximum recovery, and for health care professionals involved in the rehabilitation process to grow in professional competence, effectiveness and credibility, the procedures and practices of the profession need to be consistent with the scientific knowledge base relating to human physiology.

Another look at neurological rehabilitation is warranted, not only with reference to new information but also with reference to the older established facts. This discussion will primarily relate to some aspects of rehabilitation of motor function in the conditions defined as upper motor neuron syndrome.

Upper motor neuron syndrome

Hemiplegia resulting from cerebrovascular accident is the most common neurological impairment encountered in rehabilitation hospitals (Cozean et al 1988, Garrison et al 1988). Similar symptoms can occur, however, with upper motor neuron lesions resulting from other conditions, such as head trauma, multiple sclerosis and cerebral palsy. It is common practice to categorise these patients and to develop treatment protocols on the basis of their medical diagnosis (eg stroke units, head injury centres). While the differing disease categories may have some distinguishing clinical features varying with the extent and level of the lesion, the commonality of the symptoms is often overlooked. The release of proprioceptive reflexes (spasticity) has essentially the same functional result regardless of the pathophysiological mechanism (Burke 1988, Perry 1980).

The upper motor neuron syndrome (UMN) has both positive and negative features (Burke 1988, Ghez 1985, Landau 1978 and 1980). The negative symptoms are loss of dexterity and loss of strength. The positive symptoms are release phenomena (Brodal 1981, Burke 1988, Ghez 1985) and are all exaggerations of normal phasic proprioceptive reflexes, tonic proprioceptive reflexes and polysynaptic flexion reflexes (Landau 1980).

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Negative symptoms

As described by Burke (1988), the negative features of UMN syndrome comprise a specific pattern of pyramidal weakness and loss of dexterity. Interruption of the pyramidal pathway produces a greater deficit in those muscles that normally act as prime movers in volitional movement. In the upper extremity of man and primates, greatest weakness is found in the intrinsic muscles of the hand. Burke (1988) points out that finger extension is usually weaker than wrist extension and that both are weaker than flexion. Lance (1980) describes the pattern of upper extremity weakness as affecting abductor and extensor muscles. In the lower extremity, the pyramidal pattern is greater weakness of hip, knee and ankle flexors than of extensor muscles (Burke 1988, Lance 1980, Lance and McLeod 1981). No other single neurological or psychiatric illness will produce this pattern (Burke 1988). The patterns of pyramidal weakness have been noted consistently by other investigators (Brodal 1981, Denny-Brown 1966, Waxman 1988).

Appreciation of the patterns of pyramidal weakness should stimulate reflection as to the appropriateness of activities common in many rehabilitation centres where more emphasis is often given to exercising upper extremity flexors, rather than extensors and lower extremity extensors, rather than flexors. Patients perform the movements that they can most readily initiate, often against weight resistance. Strengthening upper extremity flexors and lower extremity extensors in the absence of equal antagonistic muscle activity can only serve to exaggerate the existing patterns of muscle imbalance.

Positive symptoms

Lesions damaging corticofugal pathways (including the pyramidal tract) at any level of the neuraxis may result in release of proprioceptive reflexes that manifest the positive symptoms of UMN syndrome. The positive features include abnormal proprioceptive reflexes (spasticity), abnormal posture (dystonia) and abnormal cutaneous reflexes (Burke 1988, Denny-Brown 1980 and 1966, Lance 1980, Landau 1980). Spasticity is manifested clinically by a velocity dependent increase in tonic stretch reflexes and exaggerated tendon jerks, the phasic stretch reflexes (Burke 1988, Brodal 1981, Lance 1980, Landau 1980). Although some clinicians may still attribute spasticity to lesions of the pyramidal tract, the evidence is that lesions of pyramidal fibres alone produce little residual disability in man or in primates (Beneke and Conrad 1986, Bucy 1957, Burke 1988, Brodal 1981, Denny-Brown 1980 and 1966, Lance 1980, Landau 1980). Only 3 to 4 per cent of the pyramidal tract fibres arise from the Betz cells (Brodal 1981, Bucy 1957). Spastic hemiplegia appears to be the result of the loss of both pyramidal and parapyramidal motor pathways (Denny-Brown 1980, Brodal 1981). Despite evidence to the contrary, the term spastic hemiparesis implies a causal relationship to the dysfunction of UMN syndrome (Bucy 1957, Landau 1980). Although clinicians often attribute inability to move to spasticity, spasticity is the result, not the cause, of the loss of spontaneous movement. The distribution of spasticity in flexor and extensor muscles is controlled by labyrinthine and neck reflexes (Denny-Brown 1980).

Abnormal posture

Contrary to prevalent clinical perceptions, the classic hemiplegic posture that large numbers of patients with UMN syndrome are left with, even after completed rehabilitation periods, is not due to spasticity. The pyramidal tract facilitates extension and abduction in the upper extremities and flexion in the lower extremities of man (Lance and McLeod 1981). The absence of pyramidal control in disease states allows the decorticate posture to emerge: "A reversion to the 'antigravity' posture of human reflex standing" (Lance 1980). By definition, movement is the stimulus necessary to demonstrate a spastic increase in muscle tone. The muscle contractions that maintain the abnormal postures continue in the absence of movement. Overactivity in spinal reflex circuits is also not implicated in maintaining the hemiplegic posture, as interruption of the reflex arc by posterior root section does not abolish it (Burke 1988).

The decorticate posture (flexion of the upper limb/s and extension of the lower limb/s) appears to be due to excessive supraspinal drive determined by proprioceptive and labyrinthine inflow. As many clinicians have observed, the posture may be influenced by altering head position.

Labyrinthine input may be demonstrated by turning a hemiplegic (adult, child, monkey) head down and thereby eliciting extension of the upper extremity and flexion of the lower extremity (Burke 1988, Denny-Brown 1980 and 1966, Lance 1980). Electromyographic studies of tonic neck, tonic lumbar and tonic labyrinthine reflexes in normal persons by Tokizane et al (1951) underscore the labyrinthine influence in this posture. Muscles demonstrating facilitatory influences in the erect posture showed inhibitory influences in the inverted position (see Figure 1). The muscles that show facilitation in the erect posture (elbow and wrist flexors and tibialis anterior) are a frequent cause of much frustration during the rehabilitation process because of their unbalanced overactivity. Hemiplegic posture is best considered a form of dystonia (Burke 1988, Denny-Brown 1966), an important distinction when considering chemotherapeutic treatments as those medications designed to treat spasticity cannot be expected to be effective (Burke 1988). The distinction between the limitations imposed by dystonic posture and those imposed by spasticity should also be considered in the development of rehabilitation regimens. The performance of motor activities includes motor and postural components. Postural adjustments precede, accompany and follow execution of voluntary movements (Bouisse[illegible]l Zattara 1988, Martin 1967, R[illegible]ts 1978). Recognition of

Figure 1.
Reflex effects of the tonic labyrinthine reflex from Tokizane et al (1951). Electromyographic studies on tonic neck, lumbar, and labyrinthine reflexes in normal persons. Extracted from the *Japanese Journal of Physiology* 2: 130-146.

Muscles	Erect	Inverted
Anterior Tibialis	+++	---
Soleus	---	+++
Gastrocnemius	--	++
Biceps Femoris L/H	--	++
Vasti Medial & Lateral	--	++
Rectus Femoris	--	++
Gluteus Maximus	-	+
Flexor Carpi Radialis & Ulnaris	+++	---
Biceps	++	--
Triceps	--	++
Deltoid Reflex	-	+

the limited movement repertoire imposed by the reflex nature of UMN postural disturbance and implementation of appropriate procedures to elicit automatic postural adjustments would enhance function by diminishing or eliminating the stereotypic features of this disabling syndrome.

Concepts of the role of reflex activity in the control and generation of movement and posture have been modified in recent years (Allum et al 1989, Back-y-Rita 1988, Carew 1985a and 1985b, Dietz 1986, Jankowska 1989, Paillard 1988, Peterson et al 1989) but not eliminated. Although more attention is now given to the influence of the context in which some movement occurs in shaping the resultant movement, basic reflexes underlie these interactions. It is an incontrovertible fact of contemporary neurophysiology that all animals have a consolidated repertoire (inherited or acquired) of motor abilities upon which they build. There is an evolutionary advantage for organisms to stabilise successful adaptation in their structural organisation (Paillard 1988).

In past years, much clinical attention was focused on the inhibition of primitive reflex patterns. Although it is a very credible neurophysiological concept, the term inhibition has now fallen out of favour in many clinical circles. Inhibition alone will not generate the desired motor behaviour (Landau 1980). More attention directed towards eliciting the automatic postural reactions underlying coordinate movement, as well as suppressing the limitations of the dystonic posture, is required.

Cutaneous reflexes

The release of cutaneous reflexes can affect both upper and lower extremity function. In the rehabilitation management of UMN lesions, little attention has been given to released cutaneous reflexes except for passing concern over the tonic palmar grasp reflex (grasp without release) and the tonic plantar grasp reflex (toes curling on shoe or floor contact with the sole of the foot). The exaggerated stretch and tendon reflexes of fingers and toes is not the cause of loss of movement but rather, is due to the loss of the tactile regulation of the grasp reflex (Denny-Brown 1980). Multiple factors, proprioceptive, labyrinthine, tactile and visual comprise the supporting reactions which make possible the ability to stand. Each of these components can become overactive in the absence of the appropriate level of influence from the others (Denny-Brown 1980). Release of the cutaneous reflexes contributes to the excitability of the flexor reflex afferents (FRA). These afferents (group II-IV muscle afferents, joint afferents and cutaneous afferents) share a polysynaptic reflex pathway. They may evoke flexion withdrawal responses of the lower extremity and are a factor in the flexor spasms frequently encountered in UMN syndrome (Burke 1988, Landau 1980).

Cutaneous receptors can be a factor in restricting normal movement by stimulating maintained contact with supporting surfaces. Conversely, appropriately applied cutaneous contact can be utilised to stimulate desired movement patterns (Rood 1973, 1975 and 1981). Rood considered that the increased extensor muscle tone often observed in patients with UMN syndrome when they are lying supine was partly due to contact with the supporting surface being maintained by cutaneous receptors. She suggested that diminishing the area of surface contact would enable easier initiation of movement (away from extension and into flexion). In neurological deficits, an example of the utilisation of cutaneous receptors to initiate movement can be seen with manual contact to the side of the face to facilitate rotation or side flexion of the head in a direction difficult to achieve as a voluntarily initiated or passive movement. With contact to the side of the face maintained, the face will gradually turn towards the contact and/or sufficient muscle relaxation can be obtained to position the head in the desired position without stretching neck muscles.

Some implications for rehabilitation

Perry (1980, p.87) has defined rehabilitation as "an organised therapeutic program directed toward recovering maximum function in patients with permanent or severely protracted physical disability". Despite

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the large numbers of patients suffering UMN syndrome, there is no universally accepted treatment regimen (Garrison et al 1988, Wade et al 1987). Rehabilitation procedures are often designed around variously stated treatment objectives and behavioural goals without adequate consideration being given to the pathophysiology of the neurological deficit. Consideration of the pathophysiology of the deficits might possibly result in more consensus as to how such deficits would best be managed. Such an orientation might also result in greater treatment effectiveness. Dombory and Back-y-Rita (1988) posed as a major question the degree to which the natural history of recovery from stroke might be modified by appropriate intervention.

To answer such a question, serious consideration must be given to the re-evaluation of rehabilitation treatment practices. Despite the long established recognition of the function of the muscle spindle as a stretch receptor which, when activated, facilitates its homonymous muscle to contract (Carpenter 1990, Carew 1985, Ghez 1985, Granit 1970, Matthews 1972), manual muscle stretching to maintain range of motion remains a standard procedure in many clinical centres. The physiological response often elicited is diametrically opposed to the stated goal.

The progressive increase in muscle tone seen in some patients with static lesions who are receiving regular stretching, and which is interpreted as increased spasticity, often is a facilitated response to a regularly occurring stimulus. In a discussion of the difference between the reflex response of the quadriceps muscle to stretch and the reflex response of the hamstring muscles to stretch, Burke (1988) noted that while the spastic quadriceps muscle could be inhibited, depending on the velocity of stretch and muscle length (clasp knife phenomenon), the reflex activity in the hamstring muscles builds up commensurately with the extent to which the muscles are stretched. Thus the question arises of whether regular stretching of spastic hamstring muscles contributes to the difficulty often encountered in some centres in achieving sufficient knee extension for tilt table activities (and subsequently for standing) in patients who have sustained traumatic head injuries.

Rood (1973 and 1975) believed that passive stretching of fingers contributed to upper extremity dysfunction by activating the traction response (passive stretching of the fingers increases finger flexor activity and activates proximal flexors in the upper limb – Denny-Brown 1980). Lance and McLeod (1981) describe the stretch reflex of spastic man as a dynamic reflex and have stated that slow stretching may evoke a reflex response. Perry (1980) believes manual stretching techniques to be futile as a means of reducing contracture, as the stretch period is too brief and the force too uneven.

There are alternatives to passively stretching spastic muscle. If there is any ability to generate voluntary muscle contraction, autogenic inhibition can be used to lengthen muscle and facilitate a greater range of motion. Golgi tendon organs (GTO) inhibit their homonymous muscle. Once thought to function as a protective mechanism to prevent excessive muscle stretch, GTOs are now known to be contraction-sensitive receptors measuring tension changes (Brodal 1981, Brooks 1986, Carpenter 1990). The inhibitory action of the GTO will dampen the stretch reflex (Back-y-Rita et al 1988). Three quick contractions of the muscle restricting the range of motion will make that muscle less responsive to stretch and will allow an increased range of movement in the opposite direction. For example, if finger flexors are tight, restricting the range of finger extension, three quick contractions of finger flexors will permit an increased active (or passive) range of finger extension. Ideally, the active range of finger extension should be resisted to strengthen these muscles in their new range. This principle can be utilised almost anywhere and is very useful in achieving and maintaining range, especially in those instances where voluntary movement is present. This mechanism can be demonstrated readily in neurologically normal persons. The author utilises this procedure as a classroom demonstration with the hamstring muscles of students. The subjects take a crude measure of hamstring length by bending forward and noting the distance between their fingers and the floor. They then stand up, perform three quick hip extensions with knees extended (without allowing lumbar extension), and then bend over again. Their fingers can then be seen to be closer to the floor. The hamstring muscles are inhibited at the hip. Using the new range ensures an effective carry-over. Patients can maintain their own hamstring (and other muscle) length with this procedure, if necessary.

In teaching her neurophysiological facilitation approach, Rood emphasised the interactions between sensory and motor systems. She taught some procedures to reduce muscle tone and thus eliminate the need to passively move the spastic muscle against resistance (stretching) that have demonstrated clinical effectiveness, but which have as yet no reported scientific investigation.

One procedure taught by Rood (1973, 1975 and 1981) is perioral pressure. This is firm (not heavy) pressure maintained with the therapist's finger on the patient's top lip. This procedure causes relaxation of the excessive muscle tone and was taught by Rood as a pre-requisite for initiating active movement. Perioral pressure is maintained for a few seconds until the desired relaxation response is observed or felt. Then the limbs may be moved into the desired range against little or no detectable resistance. Patients who are able, can apply perioral pressure to themselves. Perioral pressure also evokes many other beneficial responses (Bethune 1975 and 1991).

Another procedure to inhibit muscle tone derived from Rood is light moving touch. Rood taught that a light

moving tactile stimulus would facilitate movement. Perioral pressure was the prerequisite to reduce muscle tone, then, when tone was reduced, she advocated a light moving tactile stimulus to initiate movement. Light moving touch is listed as a facilitatory procedure in several rehabilitation texts (O'Sullivan 1994, Sullivan et al 1982). This stimulus was said to have a refractory period such that the same area of skin could not be used in succession (Rood 1973, 1975 and 1981). Rood was of the opinion that patients could not activate their own movement by using this stimulus.

The author has observed that a light moving tactile stimulus alone will reduce excessive muscle tone and has for a number of years taught this procedure to patients and students as an inhibitory, as well as a facilitatory, technique. The stimulus is a light short stroke applied to the skin, usually with a finger (the therapist's finger or the patient's finger). A light moving tactile stimulus around the central thorax will decrease excessive muscle tone in the upper extremities.

A light moving tactile stimulus on the central abdomen around the umbilicus (and below this level) will decrease excessive muscle tone in the lower extremities. It usually is necessary to repeat the stimulus four or five times or more. Inhibitory light moving touch can be applied by the patient (especially the abdominal touch) and provides those affected with spasticity a measure of symptom relief. Some paraplegic patients find this procedure, self applied, particularly useful. Where patients are taught to do this procedure themselves, it should be emphasised that the appropriate stimulus is light and brief, because many patients are of the mistaken opinion that if a little is good a lot is better.

There are other procedures besides those discussed here which will allow the goal of maintaining range of motion to be achieved in a manner that produces physiological responses supportive of the stated objective. For example, vibration to the antagonistic muscle will often reduce spasticity (Brodal 1981). Clinically, joint approximation through the long axis of the extremity bones has also been demonstrated to reduce spastic muscle tone.

The influence of sensory information

Therapeutic programmes have traditionally directed concern towards the motor output side of function. Generally, sensory deficits are viewed as a limiting factor to recovery of motor function and little attention is directed towards utilising sensory input in movement production. Brodal (1981) declared that it was a theoretical abstraction to deal with a motor function without considering its concomitant sensory function. In view of the present day concepts that the descending fibre systems, including the pyramidal tract, act on afferent mechanisms in the spinal cord to regulate sensory impulses from the periphery to the brain (Brodal 1981, Carpenter 1990), Brodal stated that it is not even permissible to speak of the descending pathways collectively as motor fibre systems. It currently is recognised that afferent activity influences several levels of the nervous system to trigger, tune, guide and assist local mechanisms (Paillard 1988). Certainly in rehabilitation settings, more attention to the sensory side of movement production is indicated.

Emerging concepts of motor control perhaps acknowledge more directly the role of afferent input in initiating and modulating movement patterns. For nearly a century, neural pattern generators have been studied in a variety of species. A neural pattern generator (also called a central pattern generator) is a neuronal network within the central nervous system that autonomously produces a basic motor programme. Studies in cats, rats and other vertebrates have demonstrated neural pattern generators for locomotion in the lumbar spinal cord based upon the fact that these animals can perform walking movements after a spinal transection (Grillner 1991, Grillner and Shick 1973, Grillner and Zangger 1975, Grillner et al 1988, Smith 1980). Recently, more direct evidence of locomotor pattern generators in man has been reported in studies of paraplegic man (Bussel et al 1988a and 1988b, Zomlefer et al 1983). Supraspinal control and peripheral input influence the spinal generator (Jordan 1991, Shick et al 1966). Although spinal generators can produce the fundamental motor pattern without afferent input, such input may trigger the generator and is important in modifying the motor output so that it is adapted to the particular needs of the moment (Atwood and McKay 1989, Carew 1985, Grillner 1991, Stein 1978). Stein (1978), in a review of locomotor control research, suggested some principles applying to central pattern generators. One principle states that neurons within local centres co-ordinate muscular synergies and generate timing signals. Another states that both centrally derived signals and sensory derived signals can co-ordinate activity among the centres. Sensory signals can measure and modify motor performance. Grillner (1991) has suggested that present knowledge of the organisation of locomotor control implies a possible role for physical therapy directed towards spinal cord function in paraplegia patients. Other central pattern generators exist for respiration and mastication (Atwood and McKay 1989).

The concept of motor synergies in motor control also underscores the importance of directing more attention towards afferent influence on motor output in rehabilitation practices. Such motor synergies are task dependent, co-activated sets of motor units which have no physical connection beyond the performance of a specific task. Task dependent motor synergies may involve the use of specific sensory cells in recruiting phases of movement required for the task in a precise time sequence. Atwood and McKay (1989) provide the example of the task of maintaining a regular typing rhythm. Contact with the keys is required. Cutaneous and proprioceptive stimuli help to trigger the succeeding key stroke. Concepts such as neural pattern

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generators and task dependent motor synergies require those concerned with rehabilitation and motor recovery to pay more attention to the afferent control of movement and to the theoretical bases for motor re-education. Current motor learning theories, as useful as they might be, are based on the motor skill training of normal persons or on cognitive learning theories. Few, if any, provide strategies for motor relearning which address motor deficits, such as an inability to initiate movement. They depend, to a large degree, on maximising motivation and place the responsibility for increased functional gains on the patient,rather than the therapist (Back-y-Rita 1988). Some of the procedures advocated by the late Margaret Rood that produced automatic motor responses to specific sensory stimuli warrant re-examination. Many aspects of her approach were difficult to explain in terms of traditional constructs but correlate well with the newer theories of motor control.

Utilisation of sensory stimuli that produce or trigger an automatic movement response can be a means of more effective carry-over of functions achieved during treatment to situations outside the treatment sessions. Rood (1973,1975 and 1981) taught that once a movement pattern could be consistently triggered (upper extremities), the patient should be able to voluntarily reproduce it. Whyte (1990) emphasised that automaticity is the characteristic of normal human performance. He stated that tasks should come to require a minimum of concentration and that recovered motor patterns become truly functional only when they become relatively effortless. For this reason, he suggested that treatment procedures with the most potential for automaticity be strongly considered in treatment planning.

Perspectives for the future

Another look at neurological rehabilitation also requires a consideration of outcome expectations. There has been an evolution in thinking, away from the accepted ideas that neurological disease is either relentlessly progressive or that it results in a fixed deficit with permanent functional sequelae (Waxman 1988). This change in thinking reflects advances made in technology and in cellular and molecular neuroscience (Back-y-Rita 1985, Waxman 1988). Patients who do not have spontaneous recovery of UMN syndrome should not be expected to acquire new motor control within a few months. Such unreal expectations for the achievement of comprehensive long term goals in a short span of time leads to disappointment, frustration and early termination of therapy programmes (Back-y-Rita 1988). As early as 1936, it was reported that a patient with complete degeneration of the medullary pyramid regained the ability to hold a pen in the affected hand and to write with it as a result of a year long programme of intensive physical therapy (Denny-Brown 1981). This represents a higher expectation than many clinicians hold today, although the author has had similar experiences with hand classes for stroke patients.

The design of the central nervous system provides alternative methods for the recovery of function and does not require recapitulation of structure (Waxman 1988). Back-y-Rita (1989) suggests that recovery of comprehensive long term goals may be obtained as late as five years post insult. Recovery of function may be a discontinuous process. Recovery of function may occur, as does learning, in periods of acquisition alternated with periods of consolidation. If recovery from brain damage is characterised by intermittent progress, a treatment plateau may not necessarily be the final level of function but may be a period of consolidation. Such a view would imply a change in the pattern of service delivery. It suggests that intensive rehabilitation services should be provided to optimise the periods of acquisition and that periods of consolidation may require only home management (Back-y-Rita 1985, Back-y-Rita et al 1988).

Conclusions

Resistance to change in health care practices often masquerades behind the spectre of increasing health delivery costs or the notion that somehow the proposed changes will prolong treatment time more than current practices. A re-evaluation of current practices with respect to both older and newer concepts of neural control may permit a sounder theoretical basis for practice to evolve. The implementation of therapeutic practices consistent with more recent neurological findings may produce greater functional recovery over time and thus reduce the long term costs of caring for those whose functional disability does not allow them to care for themselves. With respect to rehabilitation following stroke, Wade et al (1987) observed that the reports of apparent success (50-80 per cent of stroke survivors will be walking independently) hide the fact that many so-called independent patients walk slowly and rarely venture outdoors. However, better outcomes are possible. Fundamental changes in thinking about disorders of movement, their classification and how they are to be studied and treated are required (Grimm, 1983) and physiotherapists need to put those changes into practice.

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