Critical Reviews in Physical and Rehabilitation Medicine, 5(3):227–246 (1993)

Kinesiological Issues in Motor Retraining Following Brain Trauma

Avinoam Nativ, Ph.D.

Department of Kinesiology, University of Waterloo, Waterloo, Ontario, Canada, N2L 3G1

ABSTRACT: Theories suggested by movement scientists are evaluated as to their potential contribution to neuromotor rehabilitation following brain trauma. Emphasis is placed upon theories with workable implications for professionals in the field. The rehabilitation of the motor system is viewed in light of its various components, from central control issues, to the role of lower level control mechanisms, to the integrity and strength of the effector mechanism. Within this framework, key issues concerning motor learning, such as the process of skill acquisition and the importance of feedback, in particular electromyographic biofeedback, are first discussed. This is followed by a discussion of neural networks underlying motor control, including the notions of the motor program, functional cell assemblies, and the movement versus action construct. A review of issues related to the effector mechanism, specifically resistance training and spasticity is included. A conceptual model of a multilevel approach to neurorehabilitation is provided in concluding this attempt to bridge between the science of movement and the art of physical therapy.

KEY WORDS: motor retraining, kinesiology, head trauma rehabilitation.

I. INTRODUCTION

The field of neurorehabilitation, particularly motor rehabilitation of individuals with acquired brain trauma, has reached a crossroads. Present approaches and techniques are no longer considered adequate, either practically or theoretically;[1] their scientific basis as well as practical application are in question.[2] The need for rejuvenation of the neurotherapies has led to renewed interest among clinicians in the basic sciences and other related disciplines, particularly those disciplines associated with movement, e.g., kinesiology, biomechanics, muscle biology, and psychology. The degree to which ideas from these fields are looked on as having direct applicability for the practitioner, however, remains under debate. Gordon,[3] for example, indicates that specific ideas for therapy must come from therapists, as science "provides us with general models; *we* must work out their practical implications."

Concern about the usefulness of a symbiosis between basic science and practical art is not new and has recently been debated seriously in other fields such as human-computer interaction, an interdisciplinary area of applied research and design practice. Opponents of close association between science and the practical arts doubt whether validated scientific theories have ever been a source of improvements in the practical arts.[4] It is the "real world" requirements of the practical setting that seem to present the greatest barrier to efficient transfer of theoretical ideas from the laboratory to the clinic.

There are means, however, to determine whether a science might have a constructive impact on a practical art. Again in the context of human-computer interaction, Carroll[5] suggests that specificity and applicability of the science be the evaluated requirements. While *specificity* implies that the content of the science must be appropriate to the target domain, *applicability* suggests a conformity of the science to the common procedures in that target domain. The requirement of applicability further complicates the relations between science and practical art: scientific ideas cannot be practical if they are not conceived with "a thorough understanding not only of the concerns of the target domain, but of the practice that has evolved to deal with these concerns."[5] The applicability requirement is almost diametrically opposed to the basic philosophy of sciences, namely

0896-2960/93/$.50

the search for generality. Moreover, any research area is going to be in a state of instability by definition, which may further confuse the interested therapist. Lack of applicability is also, as many therapists would attest, perhaps the main obstacle to successful integration of ideas offered by theoretical scientists.

Nevertheless, the importance of bridging the gap between scientific theory and clinical practice and developing an effective and productive relationship between the two has, perhaps, never been as vital as it is at present. Rapid advances in emergency medical treatment and technology over the last decade have resulted in the survival of an increasing number of individuals who have suffered traumatic injuries. This phenomenon is strikingly evident in the field of neurology/neurosurgery and as a result clinicians and therapists in neurorehabilitation are faced with an increasing number of patients with serious and significant rehabilitation needs. Thus, those in neurorehabilitation should review the state of their art and give serious consideration to the potential contribution of recent advances in the basic sciences and related disciplines.

One of the most relevant sciences related to rehabilitation is *kinesiology*, literally "the science of movement." The purpose of this article is to evaluate ideas and recommendations suggested by this "movement science" as to their potential contribution to neuromotor rehabilitation following brain trauma. For the sake of simplicity, this article collapses the sciences that contribute to the knowledge of movement under the kinesiological umbrella. However, this is not an in-depth review of theoretical notions associated with the field of kinesiology, inasmuch as there are many sources available for such a study.[6–9] Nor is it intended to review extensively present techniques of physical rehabilitation (the interested reader is directed to other sources).[10,11] The focus here is not merely the theoretical question of whether movement science can be applicable to practice, but rather, the examination of ideas from movement sciences as well as from other practical arts, e.g., physical education and coaching, as to their potential applicability to neuromotor rehabilitation.

The development of specific treatment paradigms has been influenced by kinesiological theories in the past.[12,13] and now, once again, when searching for new directions, the process may look toward the science of movement to advance the neurotherapies.[15,16] The relatively short history of neurological rehabilitation can best be divided into three periods. Prior to the 1950s, little attention was given to the role of the central nervous system (CNS) in recovery from neurological trauma. A more *orthopedic* approach focusing on surgery, bracing, and reeducation predominated.[17–19] The first major paradigm shift in neurorehabilitation occurred during the 1950s and 1960s, resulting in the neurotherapy *facilitation* approach. The facilitation model stressed control of movement rather than muscles and promoted sensory stimulation, especially from proprioceptive afferent pathways, as the critical component of the therapeutic intervention. It was hoped that such sensory stimulation would elicit the type of changes in the CNS required for improved neuromuscular function.

The facilitation approach, though significant in focusing the attention of therapists on the nervous system and its potential modulation, has led to a continued direct and rigid application of early scientific theories and principles. Since then, the theoretical framework for motor behavior has evolved considerably. For example, the idea of hierarchical organization of the CNS with higher centers commanding lower centers[20] has been long abandoned for the more realistic heterarchical model,[21] which suggests that different CNS structures exert redundant control over various behaviors and movements. This has been an important major conceptual change that should have relevance to how we view the residual motor mechanisms available to the survivor of brain trauma. Rather than subservient segmental structures left unattended by an injured executive, the residual motor apparatus may actually contain a number of intact controllers with complex interconnections.

Unfortunately, the failure of practitioners to adapt to evolving views of brain organization and recovery from trauma led to the further development and emphasis of reflex-oriented techniques. These techniques largely overlooked the more cognitive aspects of motor control and invariably involved sensory facilitation of *normal* muscle patterns. For example, the application of a neuro-

developmental model[22,23] to recovery from brain damage is based on a strict similarity between the stages of normal development and the recovery of function following such trauma. Contemporary knowledge regarding the development of hard-wired cell assemblies underlying mature gait, as well as the interaction between biomechanical factors such as joint limitation and muscle weakness and these cell assemblies, would seem to weaken the conceptual basis of this technique. As some practitioners have noted, one major drawback of the facilitation approach is in its promotion of a one-dimensional view of brain-injured patients, which results in the relative neglect of significant biomechanical and behavioral perspectives.[24]

Proponents of a more central, cognitively oriented approach to neurological recovery[25–27] have pointed out the potential significance of motor learning and control theories to the clinical field of rehabilitation. The process of learning a skill has become the cornerstone of the new paradigm. Some of the key issues that can have an immediate influence on the strategies and techniques employed in neurological rehabilitation are the motor program, closed- versus open-loop theories of motor control, parameters affecting feedback and practice, action theory and coordinative structures, as well as spinal generators.

If there is a healthy dissention among therapists towards the emergence of the new, learning-oriented paradigm, however, it may be because this quite different outlook on neurorehabilitation could prove as rigid and unidimensional as the existing facilitation approach. For the practicing therapist to suddenly view motor retraining as a sequence of problem-solving tasks[28] rather than a strictly orthopedic and neurological rehabilitation procedure is not an easy shift, particularly because many suggestions may come from scientists who are not familiar with clinical problems and concerns. During the last 30 to 40 years, the focus in neurorehabilitation shifted from the extremities (muscles and joints) to movements and reflexes, and now to more central processes. Understandably, it is not easy to abandon the familiar hands-on *clinical* techniques for theories that are not yet firmly established as effective and practicable in the clinical setting and that remain vague with respect to the clinician's role.

The view of this author is that optimal motor rehabilitation following head trauma must include all aspects of the neuromuscular system: biomechanical considerations, as well as the integrity of the neuropathways, and the higher integration of neural information that underlies skill acquisition. This discussion of kinesiological contributions to the recovering motor system, will, therefore, address the various needs of this system, from central control issues, to the role of lower level control mechanisms, to the integrity and strength of the effector mechanism. Issues associated with the motor learning process and their potential application to neurorehabilitation are discussed first. Second, principles and theories related to the development and function of neural networks underlying motor control are evaluated. Finally, the effector mechanism and related issues, including the role of spasticity in influencing the retraining process is addressed.

II. LEARNING OF MOTOR SKILLS

Neuromuscular rehabilitation can be likened to a process of motor learning under adverse circumstances. The general principles of motor learning when combined with an understanding of the mechanisms underlying pathological movement are equally applicable to the relearning situation. The capability of the posttrauma brain to recover and develop, even long after the so-called "spontaneous recovery stage," has been demonstrated in animals as well as in humans.[29–32] The mechanisms underlying brain plasticity, still the subject of much research, are beyond the scope of this article. The demonstrated plasticity of the posttrauma brain, however, suggests that techniques and principles of "normal" motor control and learning should also be applicable to the rehabilitation model. The potential usefulness of theories related to the acquisition and development of motor skills is central to our discussion.

A. The Process of Skill Acquisition

The gradual buildup, reshaping, and refinement of motor programs is part of the process of acquiring skills. This process has been exten-

sively studied by psychologists and educators[33–35] in an effort to better understand the conditions which may facilitate the normal acquisition of skills and contribute to the development of expert skills in various fields such as music, sports, or sciences. The knowledge and principles derived from studies associated with normal or elite skill acquisition, however, may also be valuable to the acquisition of skills following trauma to the central nervous system. Different stages have been identified in skill acquisition.[36,37] The first phase of learning has been variously termed the "cognitive" or "declarative" stage, wherein the learner determines the goal of the movement/action, comes to understand the requirements and the particular movements associated with the action, and attempts to execute early versions of the desired skill. It is during this cognitive stage that the therapist or coach provides verbal explanations and demonstrations, sets up the training environment to facilitate the desired learning process (e.g., walking surface, orthotics, harness, etc.), and provides the learner with relevant feedback and error correction.

Once production of the skill becomes more consistent, the role of the therapist gradually changes. At this stage, the clinician's main role is to enhance the progression and modification of an optimal learning environment and to ensure that maximal repetition of the whole skill or parts of that skill ensues. Intensive practice gradually leads to the automatization of the learned skill and finally to successful incorporation of that skill into daily living activities — a process that should now not be attention-demanding.[36] Some authors suggest that the latter phase in skill acquisition, the automatizing of skills, can be broken down into two substages: the fixation phase and the autonomous stage.[37–39] Other authors do not make this distinction.[36] It is important to note, however, that the learning process never stops, even after automatization. Observation of on-the-job performance of skilled workers,[40] for example, revealed that the performance (e.g., speed) was still improving after years of work.

The significance to motor rehabilitation of defining these distinct stages in skill acquisition lies in realizing the amount and intensity of practice needed to turn a specific behavior into an acquired skill. Turnbull and Wall[25] have illustrated this point: "If a patient receives treatment from the physiotherapist for 2 h daily, 5 d a week (this being a somewhat idealistic scenario), this amounts to less than 6% of the patient's total time in a given week. This seems less than adequate for optimal skills acquisition." In fact, when this brief practice time associated with relearning such functional skills as standing up or walking is compared to the much greater average daily practice time of an infant attempting to learn similar skills, the limitations inherent in many current neurorehabilitation training procedures become obvious. How can one expect the true acquisition of skill with once per week (or once per 2 weeks) 30-min sessions of training, even if the efficacy of the technique is well established? Similarly, it is very unlikely that even 3 half-hour sessions per week of gait retraining, for example, with the therapist demonstrating and explaining the correct determinants of ambulation, will be sufficient to ensure real acquisition of the desired skill, i.e., progression from the cognitive to the automatic stage of motor learning. Further stressing the significance of practice intensity, Sivenius and co-workers[41] showed that an intensive rehabilitation program for stroke patients resulted in significantly better gains in activities of daily living and motor function compared with a normal treatment control group, thus reinforcing the value of intensified physiotherapy treatment.

Effective solutions for the optimal practice intensity problem are not easy to find. Some of the more practical suggestions by current workers in the field[25,42,43] include assignment of home programs by therapists, proper supervision of learning progress, and the incorporation of computer technology with clinical techniques to improve the motivation and hence the efficacy of practice.

A successful initial (cognitive) stage of learning does not ensure the successful acquisition of a desired skill. The therapist must bear in mind that, following the apparent understanding and successful execution of the action to be learned, sufficient time must be allotted for repetition of the skill until the point when it is automatized and can be incorporated into more complex sequences of behavior with little attentional effort. While the significance of consolidating the early learning with further practice is obvious, there will be certain situations in which the learner may not

follow the standard "declarative" first then "procedural" or "automated" phase order in skill acquisition.[44] It has been argued that children may learn skills differently than most adults.[35] Children appear to hop into the procedural stage without having to consciously understand the rules of the skill to be learned. They appear to do so by experimentation and motor exploration until they successfully carry out the desired activity. At that point, their execution may be smoother or more automatic than that of their adult counterparts, even in the absence of verbal understanding/cognition of the rules underlying action. Along the same line, it has been demonstrated that amnesiac patients can improve on motor skills with no declarative learning having taken place.[45]

It would make sense to assume that, following trauma to the CNS, reacquisition of skills may not in all cases follow the cognitive to procedural phase order. The deviation from this rule, noted in the manner in which children and amnesiac patients acquire skills, suggests that, though typical to motor learning, the declarative, rule-guided phase may not be essential to the acquisition of skills but, rather, a convenient strategy adopted by most adults. Following CNS trauma, it may become difficult or unnecessary to explain to the learner the rules and principles associated with the skill. Yet, as long as clever structuring of the learning environment allows for motor exploration on the part of the learner, there is good reason to believe that he/she will be able to acquire certain motor skills despite the cognitive limitations that may hinder the successful attainment of the so-called declarative stage.

B. The Role of Feedback

The role feedback plays in motor learning and skill acquisition has long been acknowledged.[9,46–48] Sensory information associated with the movement itself (intrinsic feedback) may include visual, acoustic, haptic, vestibular, cutaneous, and kinesthetic information produced by the motor response. Information associated with the result of that response is termed "extrinsic" and may appear in the form of verbal or nonverbal responses and be provided during the action, immediately following it, or following the action with a certain delay. The extrinsic or augmented information may relate to the movement itself (knowledge of performance) or to the environmental consequences of the action (knowledge of results). That feedback of intrinsic and extrinsic information associated with the motor response is essential for the development of motor skills has never been debated (see Schmidt's schema notion,[49] for example). However, issues pertaining to the nature, frequency, intensity, and delay period of the extrinsic information have been systematically studied in the laboratory (e.g., review by Salmoni et al.[48]) in an effort to find the most effective form of teaching and perfecting motor skills.

The focus on extrinsic feedback, i.e., knowledge of results (KR), rather than on intrinsic feedback, which is the sensory feedback associated with the movement, appears to fit well within the rehabilitation setting. Neurologic impairments very often are associated with reduced intrinsic information, leaving the learner more dependent on extrinsic forms of feedback such as knowledge of performance and knowledge of results. Still, caution must be taken before adopting principles and rules that are the result of laboratory experimentation for motor rehabilitation.[50] For example, KR literature shows that in many cases, less intensive feedback during the acquisition stage translates into more successful learning.[51] That may arguably happen because internal processes have to be allowed to take their course during the acquisition period; limited feedback may actually force the learner to develop independent problem-solving strategies.[52,53]

When problem-solving capabilities are impaired, however, as is the case with various forms of neurologic damage, learning may be facilitated by more, rather than less, intensive feedback. Moreover, if it is the combination of intrinsic and modified extrinsic feedback that optimizes learning, then, in the case of the neurologic population with limited intrinsic information, that optimal combination may require a greater rather than normal amount of KR. This caution is aimed at the rigid application of rules regarding the frequency and intensity of feedback to motor relearning, but not at the value of augmented sensory information, as well as KR, to motor training following CNS trauma.

The significance of augmented feedback information in neuromotor rehabilitation has been stressed by several workers.[27,29,50,54] Because the restoration of limb function is an important focus of neuromuscular rehabilitation, the sensory information from the ongoing movement is especially important for the relearning process. One promising technique of augmenting the sensory information from muscle activity is electromyographic (EMG) biofeedback: a visual or auditory display of the electromyographic activity in the targeted muscles. This form of augmented sensory feedback is both faster and more accurate than verbal feedback and therefore can be more effective at modifying the impaired motor skills. The following section will examine the efficacy and potential benefit of EMG biofeedback in neuromuscular reeducation.

1. EMG Biofeedback

EMG biofeedback was employed as a therapeutic model as early as the 1950s.[55] However, it has become a viable option to the more traditional rehabilitation methods only in more recent years when modern technology, particularly the extensive development in the computer industry, increased the complexity and flexibility of the EMG display. The simple increase or decrease in muscle activity that had been the objective of practice in early EMG biofeedback applications is gradually giving way to more sophisticated and specific practice, for example, matching the EMG output of a muscle to a reference waveform on a computer monitor and gradually adjusting the reference as muscle output/control improves.[42,43] Modern technology also allows for matching the EMG output in the involved side to that of the uninvolved extremity, thus providing the learner with an additional effective reference. Also, motor learning and functional improvement can be enhanced by focusing on agonist-antagonist co-activation rather than on the agonist output alone. Typically, in such practice, the patient/learner attempts to increase the agonist activity to reach a (high) reference target, while at the same time trying to reduce the antagonist activity to reach another (low) reference target.

Training with EMG biofeedback has been reported to successfully decrease the tone in spastic muscles,[56] to strengthen weak muscle groups,[43] and, coupled with transcutaneous electrical nerve stimulation (TENS), to improve the timing and control in gait reeducation.[57] However, despite the sound theoretical ground for employing this technique, and the positive reports from both the field and the laboratory, it is still difficult to argue that artificial sensory feedback (EMG biofeedback) is a more efficient procedure than standard methods in neurorehabilitation.[58] Mulder,[27] in an elegant critical review of EMG biofeedback practice in neuromuscular retraining, argues that with regard to "the transfer of the treatment effects to the activities of daily living ... the reviewed studies provided no unambiguous evidence for the superiority of artificial sensory feedback methods, compared to conventional procedures."

Mulder[27] further cautions researchers and therapists that additional factors may determine the efficacy of EMG biofeedback in neurorehabilitation. First, the type of disorder, whether central or peripheral, can affect the potential of the biofeedback technique. Central disorders have been characterized by inability or reduced ability to carry on the cognitive processes essential for functional processing of incoming signals/feedback.[59] Second, it is not clear whether the benefit from EMG feedback originates with its quality as a source of information or simply because it is an effective motivator that improves the learner's attentiveness to the target information. Several studies have been unable to show consistent improvement with EMG biofeedback alone when uncoupled from its motivational element.[60,61] Finally, the nature of damaged brain structure appears to influence the efficacy of EMG biofeedback. Damage to areas with topical arrangement (e.g., sensory or motor cortex) is harder to rehabilitate than damage to a nontopical structure such as the reticulo-spinal tract.[62]

Still, augmented sensory feedback is a promising technique that needs further exploration to achieve optimal clinical potential. The combination of EMG feedback with an exercise program appears to be especially effective. For example, such combination resulted in approximately double the improvement (reduction of foot-drop was in-

vestigated and the measures were range of motion and strength) compared with conventional exercise treatment.[63,64] The intensity of the standard EMG biofeedback application also requires special attention. As suggested in this discussion, true acquisition of a skill requires many hours of daily practice. Learning a skill through the process of EMG biofeedback should not be an exception to this rule. Standard EMG training, which, even optimally, involves two to three practice sessions per week with net training time of 15 to 20 min per session, could not be considered sufficient to carry the learner from the initial cognitive stage to the automatic stage of skill acquisition. Worse still, the majority of postacute neurologic individuals are unable to attend rehabilitation centers with such frequency.

Some changes are required in order to solve the two major problems that reduce the efficacy of EMG biofeedback, namely limited accessibility and lack of sufficient practice. Such changes will (1) enhance the learner's motivation and thus increase the practice time with the tool, and (2) enable users to practice away from the clinic/laboratory through the development of portable EMG biofeedback devices that can hold the attention of the learner for relatively long intervals. The recent technological advances have, in fact, nurtured innovative ideas regarding the enhancement of EMG biofeedback.[42,43]

Games are, by nature, an effective motivator and a means by which a newly acquired skill can be perfected. A more powerful EMG biofeedback device would allow for the sensory feedback from an agonist-antagonist muscle pair to be incorporated into a computer game, in a setting simple enough to be considered portable. Figure 1 demonstrates such a unit. The potential of portable EMG biofeedback units, even less advanced than the one described in Figure 1, was demonstrated nearly 2 decades ago by Johnson and Garton,[65] who were able to improve strength and range of motion (foot dorsiflexion) in postacute hemiplegic patients. There are encouraging results from the laboratory, as well as the clinic.[27,55] In light of this, and in view of the further potential development of the EMG biofeedback technique, there is good reason to expect that, when integrated into a comprehensive exercise program, this will be an important tool for the practitioner to facilitate neuromuscular rehabilitation in people who have sustained brain trauma.

III. THEORIES OF MOTOR CONTROL

A. The Motor Program

The concept of motor program, i.e., the central control of movement, is an old one in psychology[66-68] and has been the cornerstone of open-loop theories. The open-loop approach maintains that the necessary specifications for the motor act, e.g., spatial and temporal parameters, are stored centrally in motor programs and that these programs can activate functional movements without the need for immediate updating, i.e., feedback. There has been convincing evidence for central programs that can direct human and animal behavior in the absence of kinesthetic feedback.[66,69,70] However, information regarding the movement, as well as the outcome of the movement, is of

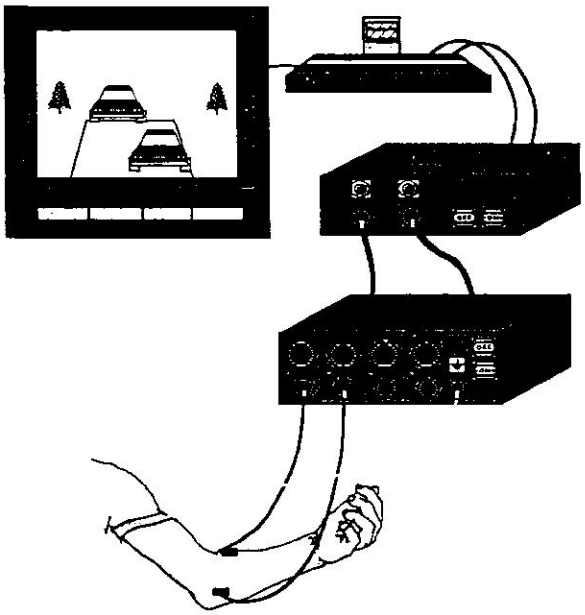

FIGURE 1. EMG biofeedback training unit. Amplified, rectified, and smoothed EMG signals from antagonistic muscles on the affected forearm are led to an interface box, replaces the standard joystick of a commercial video game. Attempted flexion of the wrist turns the car on the TV sceeen to the left, whereas attempted extension turns the car to the right (Unitech Research Inc., 702 North Blackhawk Ave., Madison, WI 53705).

utmost importance for the execution of fine motor skills and the continuous development and adjustment of the central set.[71] (See also Sachs,[72] for a vivid demonstration of function in the absence of afferent information.)

Proponents of the modern program notion view the motor program as the open-loop part of a conceptual model of human performance (Figure 2).[7] Feedback processes, the closed-loop part of the model, can modify and update the program. Despite some major limitations of the motor program concept (see Schmidt[6,7] for in-depth discussion), this model can be useful to neurotherapists. It demonstrates that learning/relearning of motor acts involves adjustments and reshaping of a central neural network. Furthermore, the model shows the significance of an active movement component — an initial attempt that starts the process of program updating through practice and feedback. Arguably, it is this active component in the development of a motor skill that may not have received proper consideration by proponents of the facilitation approach. The practical applications of a simple motor program model for the development of new skills in the able as well as the disabled person are straightforward: in order for a central representation of a movement to develop, the person needs to initiate an attempt that will be realistically successful to allow the information provided by movement outcome and the sensory system to update and adjust the existing program. Frequent repetition of this process with a focus on active initiation will, according to this notion, improve and refine the existing skill. This may appear quite trivial to the therapist, yet the approach based on motor control and learning theories requires a change in perspective and in the methods prevalent in neurorehabilitation.

The implications of this new perspective are clear. No longer can one allow an individual with an acquired brain injury to sit and wait until he/she is "ready" to execute a new movement, be it an attempt to move a limb, to stand up, or to walk. According to the motor program notion, if the person is to successfully reacquire such skills, active initiation of the required skill must be attempted with reasonable success. How can this be done? How can a nonambulatory patient attempt to take a step, or how can a person who is unable to hop attempt a reasonable version of that act?

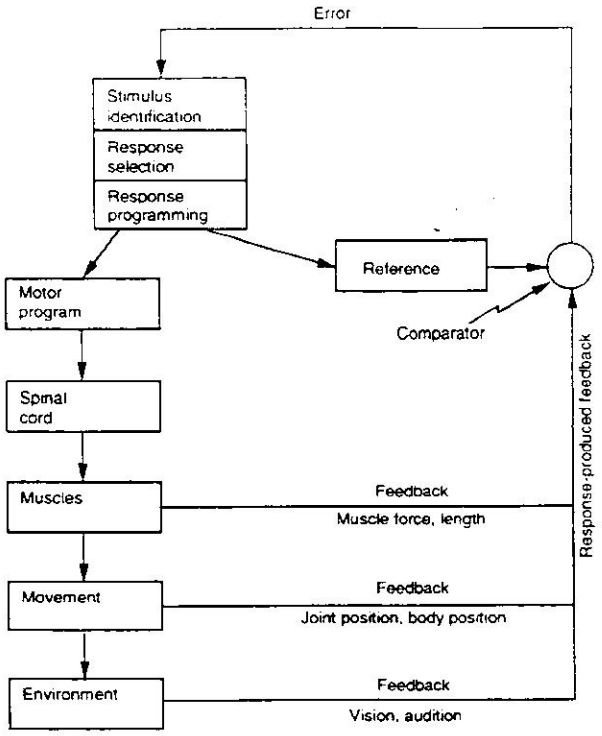

FIGURE 2. An expanded conceptual model of human performance. Note the significance of a central initiation of movement to the learning process (i.e., to the modulation of the motor program). (From Schmidt, R. A., *Motor Learning & Performance: From Principles to Practice*, Human Kinetics Publishers, Champaign, IL, 1991, 50. With permission.)

To answer these questions the therapist must either use his/her imagination or apply some of the methods adapted in high level competitive sports. For example, a gymnast attempting to learn a complicated movement pattern or a descent from a high bar will often be supported by a suspension harness mechanism that enables him/her to create the first rough models of the desired movement. Once the movement is perfected and the correct timing learned, practice can proceed in real time and space without the assistive device. A platform diver in the early stages of skill acquisition will rely on similar techniques to learn a complex dive. The assistive devices in both examples not only provide a safety mechanism for a dangerous act but, more importantly, allow the motor system to attempt reasonable first efforts of the desired motor act and, hence, create a working program that can be further refined with practice. Figure 3 illustrates a harness mechanism that enables the person

with brain trauma to initiate ambulatory activity as well as various mobility exercises.

A wide variety of assistive devices, from the functional walker or cane to less typical tools such as balls, trampoline, ergometer bicycle, treadmill, or stairclimber may help create and reshape the program or central representation of a new skill. The role of the therapist in creating the optimal learning situation, i.e., enhancing the formation of new or modified programs, is crucial and requires considerable creativity. In the following sections, some other perspectives of human motor control are discussed and their implications for neurorehabilitation considered.

B. Functional Cell Assemblies

The motor program concept assumes central organization of motor activity, i.e., sets of stored commands for individual movements complete with specification of the muscles involved in the planned action, the order in which these muscles are to be activated, and the relative force and timing of muscle contraction.[7] The weakness of this concept lies in its difficulty to explain how the motor system can store the enormous number of programs needed for daily operation or to explain novel movements for which there are no stored programs. It has become evident to neurophysiologists and motor control researchers that functional assemblies of cells at different levels of the CNS are responsible for initiating and maintaining complex coordinative movements.[73] These neural assemblies reduce the number of details (degrees of freedom) attended to by the executive mechanism (the motor program) and thereby simplify its task. Furthermore, the actual control process may not rely on a single "executive" (hierarchical organization), but, rather, on a coordination between various neural assemblies. This notion, which has been termed "distributed control," better explains the enormous flexibility of the system.[74]

This view of human motor control is consistent with Easton's[75] earlier notion of "coordinative structures," a celebrated term for reflexes that form the basic language of the motor program. The notion of pattern generators is also consistent with this philosophy. Pattern generators have been described as neural assemblies capable of maintaining complex rhythmical activities (e.g., locomotion) and thus they simplify the process of motor control.[76,77] In fact, it is a similar concept to that of the motor program[7] in that it implies the existence of a hard-wired executive, except in this case it is at the spinal level. Despite the existence of hard-wired pattern generators or oscillators at the segmental level or even in supraspinal structures such

FIGURE 3. A harness mechanism, allowing the degree of weight bearing to be controlled by the therapist, assists the learner in attempting novel motor activities (e.g., gait, jumps, kicking a ball).

as the cerebellum,[78] there is still considerable variability in the movement patterns. The sensitivity of gait to changes in peripheral mechanical conditions (e.g., in amputees[79] or under ischemic conditions[80]) suggests that the segmental pattern generators are attuned to the mechanical limitations and may not, therefore, exhibit their *normal* patterns in the face of such limitations. The successful rehabilitation of coordinated movement may be determined by the therapist's ability to access or affect these lower levels of control, rather than by his/her ability to start rebuilding the motor skill element by element.

Motor impairments that are the result of trauma to the CNS may, at times, leave some of the lower level CNS synergies intact. The movement impairments in such cases may result from a central deficit, i.e., from an inability to properly access and activate those systems or from mechanical reasons such as muscle contractures or muscle weakness. In either case, modifying the learners' environment, changing some movement parameters, or strengthening muscles central to the coordinative structure could retrieve a coordinated set of behavior otherwise unavailable to the brain-injured person. The strengthening issue is discussed later in the article. As for the manipulation of the movement parameters or the environment, let us examine the contribution of tools such as the treadmill and stationary bicycle.

The treadmill (Figure 4) provides the brain-injured individual with artificial initiation as well as with dynamic gait maintenance. Given that the complex coordination of gait is likely organized in low level (spinal) generators, the input provided by the moving surface may, in some cases, activate those generators and initiate a more coordinated form of walking than is initially possible over ground. Therefore, further training directed at improving strength, balance, and functional range of motion around the involved joints will complement and ease initiation and maintenance of this behavior. Cycling, a familiar reciprocal motor skill may, in a similar manner, help elicit movement in an otherwise impaired extremity through the activation of a well-learned coordinative structure in which the motion of the affected extremity is an integral part. Evidence of the potential benefit to neuromuscular rehabilitation of such reciprocal training tools has only recently been emerging from the laboratory and clinic.[14,81,82]

FIGURE 4. Gait practice on a treadmill.

1. The Dynamic Pattern Theory

An extension of the pattern generator thesis is the dynamic pattern theory.[83] The theory maintains that there are intrinsic patterns of behavior that can be spontaneously elicited by changing the control parameters under which the specific patterns are available to the system. For example, speed of movement, a possible control parameter, would have to increase in order to change walking into running in bipeds (or trotting into galloping in quadrupeds). The significance of this theory lies in the notion that one does not need to change or relearn specific components of movement in order to reacquire a movement pattern (e.g., reaching or locomotion). Often, reestablishing weight shift or, alternatively, increasing the speed of motion will bring about the required gait pattern.

This view of motor control directs the focus of motor rehabilitation away from the restoration of technically correct individual movements and towards the restitution of function: inherent synergies or coordinative structures that, when allowed to act, can result in goal-directed motor

behavior. It is the therapist's responsibility to find optimal parameters and to identify and try to eliminate obstacles, such as muscle weakness or limited joint range of motion, that prevent such synergies from being activated.

2. The Dual Strategy Hypothesis

In a recent attempt to apply the essentials of motor control principles to measurement and treatment procedures in movement rehabilitation, Corcos[84] has suggested the *dual strategy hypothesis*. According to this control theory, two basically different sets of rules for motor commands exist for "speed-sensitive" and "speed-insensitive" movements. Examples of speed-sensitive movements would be executing a tennis stroke or pushing the car accelerator while driving; both movements have strict timing requirements and call for varying intensities of force production.

Corcos[84] argues that in many movement impairments (e.g., CVA, parkinsonism, spasticity), the motor system is unable, or shows reduced capability, to adequately vary the intensity of central command to the muscles and, therefore, the temporal requirements of the movements cannot be met. The actual mechanism impeding the speed-sensitive movements may vary; it could stem from coactivation of agonist-antagonist muscle pair or from a central deficiency in varying the agonist burst. However, because speed-sensitive control processes are essential in normal movement as well as in motor rehabilitation, Corcos strongly suggests that strength training be applied where speed-sensitive movements are impaired. He points out supportive evidence from the laboratory, which together with the above theoretical considerations make a strong case for the use of strength training in neurorehabilitation.

C. Movement versus Action

What is the critical aspect of motor control? Is it the specific movements chosen to serve a given function, or the overall efficiency of the act and its impact on the environment? In fact, motor output may be viewed and measured in a variety of ways. The electrical events measured as EMG at the muscle level, or electroencaphalography (EEG) measured at the brain level, represent one dimension of motor response. The various joint movements can be studied with regard to their kinematics and kinetics to offer a second dimension. Finally, the action (i.e., walking, throwing a ball, balancing on a tilt board) may be assessed as to its effectiveness in reaching the performer's goals. Any of these dimensions of motor control and behavior may vary independently. For example, the act of hitting a target with a ball may be accomplished using different trajectories, different movements, or even different limbs. Furthermore, when the same motions are employed, there may still be considerable variation in neuronal firing and muscle coactivation that may not show in kinematics associated with the throwing motion.

According to one school of movement scientists, the ecologists, the emphasis in the study of motor control should be placed on the action rather than the particular movements composing it.[86–89] The early ecologists promoted a view of animal-environment synergy rather than animal-environment dualism. They perceived humans (and animals in general) and the environment as an inseparable and compatible subsystem of the ecosystem and, hence, movement could not be separated from the environment in which it took place. This early ecological approach suggested *direct perception*, i.e., a direct interaction between the perception and the action without the need for cognitive intervention. Such a conceptual mechanism of action must be flexible in order to accommodate for the ever-changing environment. This flexibility, in turn, requires an equally flexible neural basis, which holds further implications for the acquisition of motor skills.

The neural control system that would better fit an action model is not a centrally organized one, but, rather, a distributed network with various elements or neural assemblies that can be modified by experience. As a result of the interaction between those elements, the motor act in such systems is a novel creation, despite its apparent regularities.[21,90,91] This dynamic perspective better accounts for the flexibility of motor behavior and better explains issues such as the complexity of control (degrees of freedom), storage size, or the initiation of novel movements. It also underlies the significance of a large repertoire of

well-established neural assemblies at different levels of the CNS. Such assemblies, which are covered in more detail in the previous section, may be interconnected, when necessary, to solve a particular environmental problem.

In the rehabilitation domain, the emphasis has typically been placed on the movement to be retrieved.[18,92] The previous discussion, however, suggests that the particular movement that has been lost or impaired may not be as important to recovery of function as the ability to successfully interact with the environment and solve the problems presented by that environment. The significance of the action approach to motor learning in general and to neurologic rehabilitation in particular is twofold. First, it may shift the emphasis of motor rehabilitation from the neuromotor approach to a more functional, goal-oriented retraining. Rehabilitation of the post head trauma person may need to be viewed in a different light than that of the nonneurologic patient. The available repertoire of functional movements that can be incorporated into a meaningful action is smaller in the traumatically brain-injured. Hence, the most reasonable action chosen by this individual in a given circumstance may differ. Just as the person on crutches is not expected to ambulate using a "normal" walking technique, the brain-injured person may need to develop and adopt a different set of movements in order to interact with his/her environment in such a way that will produce ambulation. An approach stressing the strict reestablishment of "normal" gait patterns[13,22,92] may not be capable of accomplishing the same results as the action approach.

A second important contribution of the action approach is its view of the flexibility of the motor system. The choice of the response depends on person-environment compatibility, and the actual motor output is a function of the established synergies available to the individual. Therefore, the emphasis in motor reeducation may be shifted from learning a complete skill to learning the essential components that can then be reassembled to form the appropriate environmentally compatible response. Thus, gait-training, for example, may focus on basic skills such as balancing on one leg, lateral and fore-aft weight shifting, and hopping. Because walking can be viewed as a continuous process of recovery from perturbation,[79] the specific components of response will depend on such factors as dynamic weight shift, floor texture, obstacles, etc. to form the complete motor behavior of gait.

This approach to gait reeducation requires extensive manipulation of the environment to elicit behavior as well as a good understanding of biomechanics on the part of the therapist in order to improve strength and flexibility in key joints and increase the "library" of basic maneuvers. This, in turn, will increase the number of options the environment affords the learner. In essence, the action approach to gait reeducation promotes (1) manipulation of the learner's action storage (i.e., increase the number of coordinative structures or synergies), and (2) manipulation of the environment to elicit a specific behavior (e.g., use of a treadmill to initiate gait).

The therapist, thus, becomes a designer of learning situations and must surpass the narrow clinical perspective of healing.[93] In fact, teaching, directing, or coaching would better describe the role of a therapist in rehabilitation of the brain-injured. Along the same line, *patient* may not be an appropriate term to use for a person with acquired brain trauma (especially in the postacute stage of recovery) who attempts to enrich his/her repertoire of motor behavior.

Moving, in fact, is now viewed by some as a process of problem solving and a means by which the individual expresses skill.[28] The process of skill acquisition becomes a dynamic organization of motor expression that is specific to the individual and expresses the individual's solutions to coping with his/her environment. For the traumatically brain-injured (TBI) to solve motor problems, however, a strong set of motor options has to be available. It is the therapists' responsibility to target specific needs of the individual with regard to basic skills, design the relevant exercise program for the individual to acquire these skills and, finally, create an environment conducive to expression of the skills. In addition, movement difficulties following brain injury very often originate in a cognitive inability to solve problems. The practitioner must recognize and understand these difficulties and try to circumvent them by either solving some of these problems for the disabled learner or by improving his/her problem-solving capability.

IV. THE EFFECTOR MECHANISM: THE SIGNIFICANCE OF RESISTANCE TRAINING

Our discussion of motor learning following head trauma has highlighted the significance of muscle strength in the rehabilitation process. Muscle strength, though an important constituent of motor ability, traditionally has been surrendered by the therapist in favor of reshaping the neural control mechanisms[92,13] and, as is demonstrated in a recent recommendation of stroke patient management, still remains so. "Strengthening is not an appropriate goal during this stage of training owing to the presence of spasticity."[94] The reason for neglecting an admittedly powerful tool in combating physical handicap[95] is the association between spasticity, a common characteristic of head trauma, and resistance training.

The strong discouragement of applying resistance training in neurorehabilitation explains the scarcity of available literature on this issue. There have been, however, some recent efforts to demonstrate the efficacy of more aggressive exercise programs, including resistance training, in improving the motor function of various neurological groups. Because strength training appears critical to a complete neuromuscular rehabilitation, the remainder of this section is dedicated to these recent efforts, as well as to the theoretical basis for the strength training-spasticity link.

Bohannon[96–98] points out the significance of muscle strength to upper and lower extremity function following head trauma. He lists studies in which a significant and, at times, high correlation was observed between objective measures of muscle strength and functional measures of motor performance, such as gait velocity and cadence.[98] Bohannon's primary intention was to provide support for muscle strength testing. Although his work drew cautious response from therapists and educators,[99] the obvious linkage between strength and function in acquired CNS trauma can no longer be denied.

The next logical step in examining the strength issue is a demonstration of trainability, i.e., the proven ability of neurological patients to improve their strength with practice in a manner similar to their healthy counterparts. Milner-Brown and colleagues[100–102] have examined the effects of strength training on several muscle groups in neuromuscular disorders. They initially observed considerable improvement in muscle strength and endurance as well as a reduction in fatigability in subjects with progressive neurological diseases. They noted, however, that muscles were trainable only when disease progression was slow and the targeted muscles were at least 20% of "normal" strength. Under these conditions, they postulated that with weight training, weak muscles could sustain maximal workloads without being abnormally fatigued.[100] They proceeded to more extensive testing of various forms of strength training, including a program of low resistance weight training combined with electric stimulation[102] and a program of high resistance weight training.[101] In both instances, trained muscles became significantly stronger (108% ± 56% in the former investigation and 80% ± 48% in the latter) with the exception of severely weak muscles (<10% normal strength), which did not respond well to either method.

Subjects with selected neuromuscular disorders have made considerable gains in strength as measured in the movements of elbow flexion and knee extension.[103] Nine weeks of training (three sessions per week) resulted in 19 to 34% increase in arm strength and 11 to 50% increase in leg strength. Dependent measurements consisted of maximum isometric, dynamic, and isokinetic strength as well as assessment of the contractile properties of the elbow flexors, computerized tomography of the upper arm and thigh, and muscle biopsies. Careful examination of these measures indicated that most gains in strength were the result of neural adaptation during the 9-week training period rather than of muscle hypertrophy.

Training programs based on progressive resistance training have demonstrated good results with regard to improvement both in strength and functional measures in individuals with cerebral palsy.[104,105] Horvat[104] observed an improvement in strength, endurance, and range of movement in an individual with spastic cerebral palsy who undertook 8 weeks (3 sessions per week) of progressive resistance training. McCubbin and Shasby[105] compared a group of adolescents with

cerebral palsy who received 6 weeks of isokinetic resistance training to a control group and to a similar group undergoing repetitive practice with no resistance. Results showed that the isokinetically trained group clearly was superior to the two control groups with regard to both torque development and movement time. This led investigators to conclude that "isokinetic resistance exercise affected neuromuscular performance in these youths with cerebral palsy similar to the nonhandicapped population."[105]

Brain injury does not appear to be an exception to the potential application of strength training. Cardenas and Clausen have suggested that classic rehabilitation techniques be modified such that they suit each patient's unique requirements.[106] The combination of paralysis, spasticity, contractures, in many cases, and disuse atrophy in others often leaves the person who has suffered brain trauma with weakened and less functional muscles. For the patient to regain motor skills, considerable strengthening of these muscles has to take place. Such strengthening requires a gradual and well-planned exercise program that includes resistance training.

Cohandon has developed a comprehensive model of rehabilitation for traumatically brain-injured persons, a model centered around physical activity.[107] The model is a combination of a personalized program of physical therapy activities and group sessions, including flexibility, strengthening, coordination, and relaxation activities. Sullivan and co-workers employed this physical rehabilitation program and evaluated its effects on the locomotory status of 51 TBI patients, most of whom had been just released from an acute care hospital.[108] Of the patients in the study, 88.2% completed the rehabilitation program (4.03 ± 4.72 months) with the status of walker or jogger compared to only 31.3% who had had that classification at the outset of the program. These remarkable results, obtained through an aggressive physical activity program based mainly on locomotion, conditioning, and strengthening, demonstrate the significant role physical conditioning can play in rehabilitation from head trauma. The determinants of physical conditioning, i.e., strength, endurance, flexibility, and coordination, should be as central to motor recovery following traumatic brain injury as they are central to motor skill acquisition in able-bodied individuals.

A. Supplemental Factors in Strength Training

Resistance training may contribute to the rehabilitation process in more than one way. The act of contracting and then gradually relaxing the muscle against external weight can, in fact, be regarded as a learned skill. For such a skill to be improved, motor unit recruitment has to become more efficient (an observation noted by Sale et al.[109]) and neural coordination between agonist and antagonist needs to improve, especially in spastic muscles where the antagonist muscle activity often interferes with the agonist function. With the repetition inherent in resistance training, one would expect efficiency of the movement to improve. This would be achieved, at least in part, by reducing muscle coactivation and hence reducing spasticity. The synchronization of motor unit activity, which underlies muscle strength, as well as the timely execution of skilled movements, has been shown to improve with the regular execution of large and brief forces such as those produced during strength training regimes.[110] The investigators demonstrated that experienced weight lifters showed more synchronization of motor unit activity than control subjects. Furthermore, the level of motor unit synchronization was elevated in control subjects following 6 weeks of resistance training (dorsal interosseus muscle). The authors suggested that supraspinal influence from motor cortex on spinal motorneurons may have been enhanced as the result of strength training, leading to a significant synchronization of motor units. Rather than produce spasticity, then, strength training may produce the very neural retraining more traditional methodologies were seeking.

Some of the traditional methods of muscle stretching, "contract relax contract"[111,112] and "agonist contraction,"[113] use the same physiological processes active in dynamic weight training. The former technique uses autogenic inhibition (via Golgi tendon organs) as well as reciprocal inhibition to lengthen a tight muscle, whereas reciprocal inhibition to the antagonist motor pool ex-

plains the effectiveness of the latter technique. Similarly, dynamic or isokinetic procedures of resistance training invoke autogenic as well as reciprocal inhibition and thus should help stretch the tight/spastic muscle. The process of repeatedly contracting a muscle against resistance, then stretching it, (precisely what is involved in a dynamic/isokinetic resistance training routine) should by way of reciprocal inhibition to the antagonist muscle followed by autogenic inhibition to the eccentrically working agonist, help increase, rather than decrease, the range of motion around the targeted joint.

B. Spasticity and Resistance Training

The term *spasticity* is widely used by clinicians to describe various abnormalities in patients with CNS lesions. The term, however, may not be very accurate.[114] The global term that is more appropriate for the different symptoms associated with CNS trauma is *upper motoneuron syndrome*,[115] a term that includes symptoms and signs such as hyperactive flexor reflexes, poor dexterity, weakness, and fatigability. The more exclusive term, *spasticity*, is more accurately presented as "a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome."[116] From a functional point of view, the symptoms associated with upper motoneuron syndrome are more critical than the more dramatic ones associated with spasticity. Moreover, as will be discussed, it has become apparent that the elimination of spasticity, even if it were possible, would not be expected to restore function in most patients who have suffered CNS trauma.[3,117,118]

The phenomenon of spasticity has received considerable attention.[114,119,120–131] Researchers as well as therapists have been trying to better understand the mechanisms underlying the clinical symptoms, to find efficient techniques of quantifying spasticity,[132,133] and to search for more successful therapy. Further elucidation of the preceding issues is outside the scope of this discussion. However, association, or lack of association between spasticity and motor control in general and between spasticity and resistance training in particular will be further examined.

As already mentioned, the avoidance of effortful activities has been one of the hallmarks in the treatment of neurological patients.[13] It is believed that, in spastic individuals, effort increases muscle tone throughout the body by way of associated reactions. Since tone is detrimental to function, "any situation or activity known to lead to an increase in spasticity should be avoided."[134] Typically, however, the long-term relationship between training requiring effort and valid measures of spasticity has not been appropriately established nor has the tradeoff between potentially improved function and the proposed increase in tone been seriously evaluated. For example, it has been suggested that self-propulsion of wheelchairs by hemiplegic patients increases the tone in some of the patients and thus should not be recommended in certain cases.[134] This conclusion was based on the analysis of photographs of spastic arm position in the self-propelled compared to the pushed wheelchair condition. Naturally, an active movement requires postural adjustments and cannot be compared to a passive position for the sake of monitoring spasticity. But, more important, a control group (that probably would have shown a similar trend) was not employed; the long-term changes of arm position with practice were not investigated and finally, more valid measures of actual muscle tone were not recorded. How justified is it, then, to limit the functional independence of hemiplegic patients on these grounds?

The exclusive phenomenon of spasticity in people with acquired brain trauma, dramatic as it is, may not justify the clinical attention it receives, especially when that attention means the avoidance of much-needed physical training. Recent research clearly distinguishes between spasticity and control factors with regard to their immediate influence on functional activities.[117,118] More specifically, it has been suggested that factors such as prolongation of agonist action and decreased synchrony of input to motor cells "may be as important or perhaps more important than antagonist spasticity in limiting voluntary movement."[118] This notion is supported by the strength of association between functional measures (e.g., gait speed) and measures of spasticity and muscle

strength.[117] The authors demonstrated that gait performance was correlated with measures of knee extensor muscle torque but not with measures of spasticity (angle of reversal) in stroke patients.

There is no guarantee, then, that reduction of muscle tone will improve motor control in patients with upper motorneuron syndrome. The reverse argument, however, may hold more substance. Improvement of the individual's motor control should result in a decrease in muscle tone, by way of increased synchrony of synergistic muscles, and a lesser degree of muscle coactivation, which is secondary to skill acquisition in general. The issue of strength training as a major component in reacquiring motor control ought to receive due consideration by therapists working in neurological rehabilitation. In addition, issues related to the intensity, frequency, and specific techniques of resistance training, rather than the general validity of such training, ought to become the focus of future work.

V. CONCLUSIONS

This review of kinesiological issues in neurorehabilitation in general and in post head trauma rehabilitation in particular is by no means inclusive. This article focuses on issues and theories with workable implications for professionals in the field. In order to arrive at such workable implications, freedom has been taken to extrapolate beyond the existing data or to extend certain theories to touch on pertinent issues in neurotherapy. The relatively shaky ground of existing therapeutic norms has been pointed out and alternative measures suggested. The movement sciences in general, and kinesiology in particular, appear to be both specific enough and applicable enough to contribute to the field of neurotherapy. The practical applications of theories developed within its scientific domains may require some changes in perspective but should, nevertheless, be easily accommodated by practitioners in the clinic.

The significance of kinesiological contributions to the practice of neurorehabilitation may lie in their wider scope, i.e., in their view of the human motor system as a flexible entity that requires cognitive, neurologic, and mechanical considerations. This multilevel approach to neurorehabilitation is illustrated in Figure 5, which also summarizes the major concepts put forth in this article. Existing methods of therapy ought to be reviewed in that light, and specific, objective re-

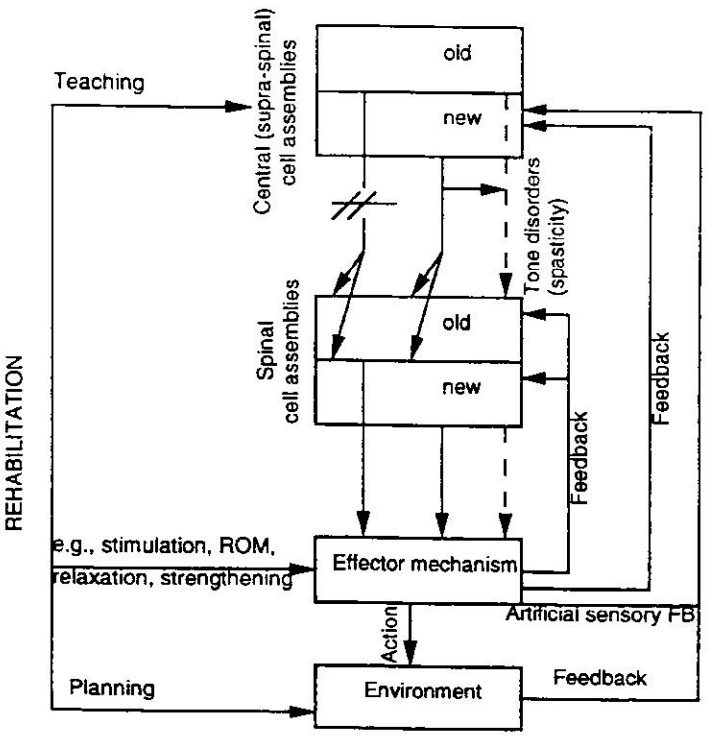

FIGURE 5. A conceptual model of multilevel neurorehabilitation. Learning of new skills is associated with the formation of new cell assemblies at the supraspinal and, subsequently, at the spinal level. Both central (teaching) and peripheral (feedback) processes contribute to the acquisition of new skills. Artificial sensory feedback helps maintain this process by feeding back information regarding muscle activity and its behavioral impact, which is otherwise scarcely available to the disabled learner. Adapting the environment to elicit motor response from the learner may also help the learner access already established spinal cell assemblies (e.g., spinal generators). Spontaneous activation of such cell assemblies may require specific conditions at the effector level, such as sufficient range of motion (ROM) and muscle strength. Finally, while peripheral manipulation at the effector level (e.g., functional electrical stimulation) may temporarily reduce muscle tone (dashed line), the central acquisition of skills associated with new cell assemblies at the brain level can lead to permanent reduction of spasticity.

search should be conducted to establish new norms as part of the new paradigm in neurorehabilitation.

REFERENCES

1. **Carr, J. H., Shepherd, R. A., Gordon, J., Gentile, A. M., and Held, J. M.,** *Movement Science: Foundations for Physical Therapy in Rehabilitation*, Aspen Publishers, Rockville, MD, 1987.
2. **Tammivaara, J. and Shepard, K. F.,** Theory: the guide to clinical practice and research, *Phys. Ther.*, 70, 578, 1990.
3. **Gordon, J.,** Assumptions underlying physical therapy intervention: theoretical and historical perspectives, in *Movement Science: Foundations for Physical Therapy in Rehabilitation*, Carr, J. H., Shepherd, R. B., Gordon, J. et al., Eds., Aspen Publishers, Rockville, MD, 1987, 2.
4. **Pylyshyn and Zenon, W.,** Some remarks on the theory-practice gap, in *Designing Interaction: Psychology at the Human-Computer Interface*, Carroll, J. M., Ed., Cambridge University Press, Cambridge, 1991, 39.
5. **Carroll, J. M.,** Introduction: the Kittle House manifesto, in *Designing Interaction: Psychology at the Human-Computer Interface*, Carroll, J. M., Ed., Cambridge University Press, Cambrdige, 1991, 4.
6. **Schmidt, R. A.,** *Motor Control and Learning: A Behavioral Emphasis*, Human Kinetics Publishers, Champaign, IL, 1982.
7. **Schmidt, R. A.,** *Motor Learning & Performance: From Principles to Practice*, Human Kinetics Publishers, Champaign, IL, 1991, 50.
8. **Magill, R. A.,** *Motor Learning: Concepts and Applications*, Wm. C. Brown, Dubuque, IA, 1980.
9. **Marteniuk, R. G.,** *Information Processing in Motor Skills*, Holt, Rinehart & Winston, New York, 1976.
10. **DeLisa, J. A., Currie, D., Gans, B., Leonard, J. A., and McPhee, M.,** Eds., *Principles and Practice of Rehabilitation Medicine*, J. G. Lippincott, Philadelphia, 1988.
11. **Goodgold, J. R.,** *Rehabilitation Medicine*, Mosby, Saint Louis, 1988.
12. **Kabat, H. and Knott, M.,** Proprioceptive facilitation techniques for treatment of paralysis, *Phys. Ther. Rev.*, 33, 53, 1953.
13. **Bobath, D.,** *Adult Hemiplegia: Evaluation and Treatment*, 2nd ed., William Heinemann Medical Books, London, 1978.
14. **Finch, L. and Barbeau, H.,** Hemiplegic gait: new treatment strategies, *Physiother. Can.*, 38, 36, 1986.
15. **Bohannon, R. W.,** Integrating movement science and physical therapy, Letter to the editor, *Phys. Ther.*, 71, 344, 1991.
16. **Twinstein, C. J. and Knecht, H. G.,** Movement science and its relevance to physical therapy, *Phys. Ther.*, 70, 759, 1990.
17. **Treanor, W. J., Cole, O. M., and Dabato, R.,** Selective reeducation and the use of assistive devices, *Phys. Ther. Rev.*, 34, 618, 1954.
18. **Bobath, K. and Bobath, B.,** The neuro-developmental treatment of cerebral palsy, *Phys. Ther.*, 47, 1039, 1967.
19. **Perry, C. E.,** Principles and techniques of the Brunnstrom approach to the treatment of hemiplegia, *Am. J. Phys. Med.*, 46, 789, 1967.
20. **Jackson, J. H.,** *Selected Writings of John Hughlings Jackson*, Taylor, J., Ed., Hodder & Stoughton, London, 1931.
21. **Bernstein, N.,** *The Coordination and Regulation of Movement*, Pergamon Press, London, 1967.
22. **Doman, R. J., Spitz, E. B., Zucman, E., Delacato, C. H., and Doman, G.,** Children with severe brain injuries, *JAMA*, 174, 257, 1960.
23. **Bobath, K. and Bobath, B.,** The Neurodevelopmental approach to treatment, in *Physical Therapy Services in the Developmental Disabilities*, Pearson, P. and Williams, C., Eds., Charles C Thomas, Springfield, IL, 1972.
24. **Carr, J. H. and Shepherd, R. S.,** *A Motor Relearning Programme for Stroke*, Heinemann, London, 1982.
25. **Turbull, G. I. and Wall, J. C.,** Gait re-education following stroke: the application of motor skills acquisition theory, *Physio. Prac.*, 5, 123, 1989.
26. **Carr, J. H. and Shepherd, R. A.,** A motor learning model for rehabilitation, in *Movement Science: Foundations for Physical Therapy in Rehabilitation*, Aspen Publishers, Rockville, MD, 1987, 31.
27. **Mulder, T.,** *The Learning of Motor Control Following Brain Damage: Experimental and Clinical Studies*, Swets North America, Berwyn, PA, 1985.
28. **Higgins, S.,** Motor skill acquisition, *Phys. Ther.*, 71, 123, 1991.
29. **Bach-y-Rita, P., Lazarus, J., Boyeson, M. G., Balliet, R., and Myers, T.,** Neural aspects of motor function as a basis of early and post-acute rehabilitation, in *Principles and Practice of Rehabilitation Medicine*, DeLisa, J. A., Currie, D., Gans, B., Gatens, P., Leonard, J. A., and McPhee, M., Eds., J. G. Lippincott, Philadelphia, 1988, 10.
30. **Kaplan, M. S.,** Plasticity after brain lesions: contemporary concepts, *Arch. Phys. Med. Rehabil.*, 69, 984, 1988.
31. **Bach-y-Rita, P. and Balliet, R.,** Recovery from stroke, in *Motor Deficits Following Stroke*, Duncan, P. W. and Badke, M. B., Eds., Year Book Medical Publishers, New York, 1987, 3.
32. **Bach-y-Rita, P.,** Applications of principles of brain plasticity and training to restore function, in *Principles and Practice of Restorative Neurology*, Young, R. R. and Delwaide, P. J., Eds., Butterworth & Co., London, 1989.
33. **Gentile, A. M.,** A working model of skill acquisition with application to teaching, *Quest*, 17, 3, 1972.
34. **Allard, F.,** Skill in sport, *Can. J. Psych.*, 39, 204, 1985.

35. **Bloom, B. S.**, Ed., *Developing Talent in Young People*, Ballantine Books, New York, 1985.
36. **Anderson, J. R.**, Acquisition of cognitive skill, *Psych. Rev.*, 89, 369, 1982.
37. **Fitts, P. M. and Posner, M. I.**, *Human Performance*, Brooks/Cole, Belmont, CA, 1967.
38. **Fitts, P. M.**, Perceptual-motor skill learning, in *Categories of Human Learning*, Melton, A. W., Ed., Academic Press, New York, 1964.
39. **Turnbull, G. I.**, Some learning theory implications in neurological physiotherapy, *Physiother. Can.*, 68, 38, 1982.
40. **Crossman, E. R. F.**, A theory of acquisition of speed skill, *Erognomics*, 2, 153, 1959.
41. **Sivenius, J., Kalevi, P., Heinonen, O. P., and Salonen, J. T.**, The significance of intensity of rehabilitation of stroke — A controlled trial, *Stroke*, 16, 928, 1985.
42. **Krebs, D. E.**, Biofeedback in neuromuscular reeducatin and gait training, in *Biofeedback, A Practitioner's Guide*, Schwartz, M. S., Ed., Guilford Press, 1987, 343.
43. **Sherwood, S. and Hewlett, H.**, The use of microcomputers in physiotherapy, *Physiother. Can.*, 70, 297, 1984.
44. **Anzai, Y. and Simon, A. H.**, A theory of learning by doing, *Psych. Rev.*, 86, 124, 1979.
45. **Cohen, N. J.**, Preserved learning capacity in amnesia: evidence for multiple memory systems, in *Neuropsychology of Memory*, Squire, L. S. and Butters, N., Eds., Gilford Press, New York, 1984.
46. **Bilodeau, E. A. and Bilodeau, I. M.**, Motor skills learning, *Ann. Rev. Psych.*, 12, 243, 1961.
47. **Newell, K. M.**, Motor learning without knowledge of results through the development of a response recognition mechanism, *J. Motor Behav.*, 8, 209, 1976.
48. **Salmoni, A. W., Schmidt, R. A., and Walter, C. B.**, Knowledge of results and motor learning: a review and critical reappraisal, *Psych. Bul.*, 95, 355, 1984.
49. **Schmidt, R. A.**, A schema theory of discrete motor skill learning, *Psych. Rev.*, 82, 225, 1975.
50. **Winstein, C. J.**, Knowledge of results and motor learning — implications for physical therapy, *Phys. Ther.*, 71, 140, 1991.
51. **Winstein, C. J. and Schmidt, R. A.**, Reduced frequency of knowledge of results enhances motor skill learning, *J. Exp. Psychol.* [Learn. Mem. Cogn.], 16, 677, 1990.
52. **Prather, D. C.**, Trial-and-error versus errorless learning: training, transfer, and stress, *Am. J. Psychol.*, 84, 377, 1971.
53. **Singer, R. N. and Pease, D.**, Effect of guided versus discovery learning strategies on learning, retention, and transfer of a serial motor task, *Res. Quart.*, 47, 788, 1976.
54. **Balliet, R., Levy, B., and Blood, K. M. T.**, Upper extremity sensory feedback therapy in chronic cerebrovascular accident patients with impaired expressive aphasia and auditory comprehension, *Arch. Phys. Med. Rehabil.*, 67, 304, 1986.
55. **Cleeland, C. S., Hodes, R. L., and Howland, E. H.**, Electromyographic feedback training, in *Encyclopedia of Medical Devices and Instrumentation*, Vol. 1, Webster, J. G., Ed., John Wiley & Sons, New York, 1988, 232.
56. **Wolf, S. L., LeCraw, D. E., and Barton, L. A.**, Comparison of motor copy and targeted biofeedback training techniques for restitution of upper extremity function among patients with neurologic disorders, *Phys. Ther.*, 69, 719, 1989.
57. **Baker, M., Regenos, E., Wolf, S. L. et al.**, Developing strategies for biofeedback: applications in neurologically handicapped patients, *Phys. Ther.*, 157, 402, 1977.
58. **De Weerdt, W. and Harrison, M. A.**, The efficacy of electromyographic feedback for stroke patients: a critical review of the main literature, *Physiother. Can.*, 72, 108, 1986.
59. **Herman, R.**, A therapeutic approach based on theories of motor control, *Int. Rehabil. Med.*, 4, 185, 1982.
60. **Sachs, D. A., Talley, E., and Boley, K.**, A comparison of feedback and reinforcement as modifiers of a functional motor response in a hemi-paretic patient, *J. Behav. Ther. Exp. Psychiatry*, 7, 171, 1976.
61. **Santee, J. L., Keister, M. E., and Kleinman, K. M.**, Incentives to enhance the effects of electromyographic feedback training in stroke patients, *Biofeed. Self-Regul.*, 5, 51, 1980.
62. **Goldberger, M. E.**, Recovery of movement after CNS lesions in monkeys, in *Plasticity and Recovery of Function in the Central Nervous System*, Stein, D. G., Rosen, J. J., and Butters, N., Eds., Academic Press, New York, 1974.
63. **Basmajian, J. V., Kukulka, C. G., Narajian, M. G., and Takabe, K.**, Biofeedback treatment of foot-drop after stroke compared with standard rehabilitation technique: effects of voluntary control and strength, *Arch. Phys. Med. Rehabil.*, 56, 231, 1975.
64. **Takebe, K., Kukulka, C. G., Naragan, M. G., and Basmajian, J. V.**, Biofeedback treatment of foot-drop after stroke compared with standard rehabilitation techniques. Part 2. Effects on nerve conduction velocity and spasticity, *Arch. Phys. Med. Rehabil.*, 57, 1, 1976.
65. **Johnson, H. E. and Garton, W. H.**, Muscle re-education in hemiplegia by use of EMG device, *Arch. Phys. Med. Rehabil.*, 54, 320, 1973.
66. **Lashley, K. S.**, The accuracy of movement in the absence of excitation from the moving organ, *Am. J. Physiol.*, 43, 169, 1917.
67. **Keele, S. W.**, Movement control in skilled motor performance, *Psych. Bull.*, 70, 387, 1968.
68. **Henry, F. M. and Rogers, D. E.**, Increased response latency for complicated movements and a "memory drum" theory of neuromotor reaction, *Res. Quart.*, 31, 448, 1960.
69. **Fentress, J. E.**, Development of grooming in mice with amputated forelimbs, *Science*, 179, 704, 1973.
70. **Taub, E. and Berman, A. J.**, Movement and learning in the absence of sensory feedback, in *The Neuropsy-*

chology of Spatially Oriented Behavior. Freedman, S. J., Ed., Dorsey Press, Homewood, IL, 1968.
71. **Adams, J. A.,** A closed-loop theory of motor learning, *J. Motor Behav.*, 3, 111, 1971.
72. **Sachs, O.,** *The Man Who Mistook His Wife for a Hat*, Harper & Row, New York, 1987.
73. **Grillner, S.,** Neurological bases of rhythmic motor acts in vertebrates, *Science*, 228, 143, 1985.
74. **Arbib, M. A.,** Perceptual structures and distributed motor control, in *Handbook of Physiology*, Vol. II, Brookhart, J. M., Mountcastle, V. B., Brooks, V. B., and Geiger, S. R., Eds., American Physiological Society, MD, 1981, 1449.
75. **Easton, T. A.,** On the normal use of reflexes, *Am. Scientist*, 60, 591, 1972.
76. **Forssberg, H., Grillner, S., and Halbertsma, J.,** The locomotion of the low spinal cat. I. Coordination within a hindlimb, *Acta Physiol. Scand.*, 108, 269, 1980.
77. **Shik, M. L. and Orlovsky, G. N.,** Neurophysiology of locomotor automation, *Physiol. Rev.*, 56, 465, 1976.
78. **Arshavshky, Yu. I., Gelfand, I. M., and Orlovsky, G. N.,** The cerebellum and control of rhythmical movements, *Trends in Neurosci.*, October, 6, 417, 1983.
79. **Winter, D. A.,** Biomechanics of normal and pathological gait: implications for understanding human locomotor control, *J. Motor Behav.*, 21, 337, 1989.
80. **Dickey, J.,** Motor compensations in gait resulting from unilateral ischemic block of the leg, Master's thesis, University of Waterloo, Waterloo, Ontario, 1988.
81. **Brown, D. A. and DeBacher, G. A.,** Bicycle ergometer and electromyographic feedback for treatment of muscle imbalance in patients with spastic hemiparesis: Suggestion from the field, *Phys. Ther.*, 67, 1715, 1987.
82. **Waagfjord, J., Levangie, P. K., and Certo, C. M. E.,** Effects of treadmill training on gait in a hemiparetic patient, *Phys. Ther.*, 70, 549, 1990.
83. **Scholz, J. P.,** Dynamic pattern theory — Some implications for therapeutics, *Phys. Ther.*, 70, 827, 1990.
84. **Corcos, D. M.,** Strategies underlying the control of disordered movement, *Phys. Ther.*, 71, 25, 1991.
85. **Fitch, H. L. and Turvey, M. T.,** On the control of activity: some remarks from an ecological point of view, in *Psychology of Motor Behavior and Sport — 1977*, Landers, D. M. and Christina, R. W., Eds., Human Kinetics, Urbana, IL, 1978, 3.
86. **Gibson, J. J.,** The theory of affordance, in *Perceiving, Acting and Knowing: Toward an Ecologcial Psychology*, Shaw, R. and Bransford, J., Eds., Erlbaum, Hillsdale, NJ, 1977.
87. **Turvey, M. T.,** Preliminaries to a theory of action with reference to movement, in *Perceiving, Acting and Knowing*, Shaw, R. and Bransford, J., Eds., Erlbaum, Hillsdale, NJ, 1977, 211.
88. **Reed, E. S.,** An outline of a theory of action systems, *J. Motor Behav.*, 14, 98, 1982.
89. **Turvey, M. T. and Kugler, P. N.,** An ecological approach to perception and action, in *Human Motor Actions: Bernstein Re-Assessed*, Whiting, H. T. A., Ed., Elsevier, Amsterdam, 1984, 373.
90. **Kelso, J. A. S.,** Ed., *Human Motor Behavior: An Introduction*, Erlbaum, Hillsdale, NJ, 1982.
91. **Kugler, P. N. and Turvey, M. T.,** *Information, Natural Law, and the Self-Assembly of Rhythmic Movement*, Erlbaum, Hillsdale, NJ, 1986.
92. **Brunnstrom, S.,** *Movement Therapy in Hemiplegia*, Harper & Row, New York, 1970.
93. **Mulder, T.,** A process-oriented model of human motor behavior: toward a theory-based rehabilitation approach, *Phys. Ther.*, 71, 157, 1991.
94. **O'Sullivan, S. B.,** Stroke, in *Physical Rehabilitation Assessment & Treatment*, 2nd ed., O'Sullivan, S. B. and Schmitz, T. J., Eds., F. A. Davis Co., Philadelphia, 1988, 335.
95. **Garbe, G.,** Critical observation of selected exercises of power training in relation to prevention of postural damage and physical handicaps, *Rehabil.* (Stuttgart), 28, 123, 1989.
96. **Bohannon, R. W.,** Is the measurement of muscle strength appropriate in patients with brain lesion? A special communication, *Phys. Ther.*, 69, 225, 1989.
97. **Bohannon, R. W.,** Correlation of lower limb strengths and other variables with standing performance in stroke patients, *Physiother. Can.*, 41, 198, 1989.
98. **Bohannon, R. W.,** Relevance of muscle strength to gait performance in patients with neurologic disability, *J. Neuro. Rehab.*, 3, 97, 1989.
99. **Rothstein, J. M., Riddle, D. L., and Finucane, S. D.,** Commentary, *Phys. Ther.*, 69, 230, 1989.
100. **Milner-Brown, H. S., Mellenthin, M., and Miller, R. G.,** Quantifying human muscle strength, endurance and fatigue, *Arch. Phys. Med. Rehabil.*, 67, 530, 1986.
101. **Milner-Brown, H. S. and Miller, R. G.,** Muscle strengthening through high-resistance weight training in patients with neuromuscular disorders, *Arch. Phys. Med. Rehabil.*, 69, 14, 1988.
102. **Milner-Brown, H. S. and Miller, R. G.,** Muscle strengthening through electric stimulation combined with low-resistance weights in patients with neuromuscular disorders, *Arch. Phys. Med. Rehabil.*, 69, 20, 1988.
103. **McCartney, N., Moroz, D., Garner, S. H., and McComas, A. J.,** The effects of strength training in patients with selected neuromuscular disorders, *Med. Sci. Sport Exerc.*, 20, 362, 1988.
104. **Horvat, M.,** Effects of a progressive resistance training program on an individual with spastic cerebral palsy, *Amer. Corr. Ther. J.*, 41, 7, 1987.
105. **McCubbin, J. A. and Shasby, G. B.,** Effects of isokinetic exercise on adolescents with cerebral palsy, *Adapt. Phys. Activ. Quart.*, 2, 56, 1985.
106. **Cardenas, D. D. and Clawson, D. R.,** Management of Lower extremity strength and function in traumatically brain-injured patients, *J. Head Trauma Rehabil.*, 5, 43, 1990.
107. **Cohadon, F.,** The importance of rehabilitation programmes in the prevention and alleviation of head injury sequelae, *Prog. Neurol. Surg.*, 10, 344, 1981.

108. **Sullivan, S. J., Richer, E., and Laurent, F.,** The role of and possibilities for physical conditioning programmes in the rehabilitation of traumatically brain-injured persons, *Brain Injury*, 4, 407, 1990.
109. **Sale, D. G., MacDougall, J. D., Upton, A. R. M., and McComas, A. J.,** Effect of strength training upon motoneuron excitability in man, *Med. Sci. Sport Exerc.*, 15, 57, 1983.
110. **Milner-Brown, H. S., Stein, R. B., and Lee, R. G.,** Synchronization of human motor units: possible roles of exercise and supra-spinal reflexes, *Electroencephalogr. Clin. Neurophysiol.*, 38, 245, 1975.
111. **Condon, S. N. and Hutton, R. S.,** Soleus muscle electromyographic activity and ankle dorsiflexion range of motion during four stretching procedures, *Phys. Ther.*, 67, 24, 1987.
112. **Etnyre, B. R. and Abraham, L. E.,** Gains in range of ankle dorsiflexion using three popular stretching techniques, *Am. J. Phys. Med.*, 65, 189, 1986.
113. **Basmajian, J. V.,** Ed., *Therapeutic Exercise*, 3rd ed., Williams & Wilkins, Baltimore, 1978.
114. **Young, R. R. and Wiegner, A. W.,** Spasticity, *Clin. Orth. Rel. Res.*, 219, 50, 1987.
115. **Landau, W. M.,** Spasticity: What is it? What is it not? in *Spasticity: Disordered Motor Control*, Feldman, R. G., Young, R. R., and Koella, W. P., Eds., Yearbook Medical Publishers, Chicago, 1980, 17.
116. **Lance, J. W.,** Symposium synopsis, in *Spasticity: Disordered Motor Control*, Feldman, R. G., Young, R. R., and Koella, W. P., Eds., Yearbook Medical Publishers, Chicago, 1980, 485.
117. **Bohannon, R. W. and Andrews, A. W.,** Correlation of knee extensor muscle torque and spasticity with gait speed in patients with stroke, *Arch. Phys. Med. Rehabil.*, 71, 330, 1990.
118. **Nwaobi, O. M.,** Voluntary movement impairment in upper motor neuron lesions: is spasticity the main cause? *Occup. Ther. J.*, 3, 131, 1983.
119. **Ashby, P., Mailis, A., and Hunter, J.,** The evaluation of "spasticity", *Can. J. Neurol. Sci.*, 14, 497, 1987.
120. **Wyke, B.,** Neurological mechanisms in spasticity: a brief review of some current concepts, *Physiother.*, 62, 316, 1976.
121. **Ashby, P. and Verrier, N.,** Neurophysiologic changes in hemiplegia: possible explanation of the initial disparity between muscle tone and tendon reflexes, *Neurology*, 26, 1145, 1976.
122. **Kerrigan, D. C., Gronley, J., and Perry, J.,** Stiff-legged gait in spastic paresis: a study of quadriceps and hamstrings muscle activity, *Am. J. Phys. Med. Rehabil.*, 70, 294, 1991.
123. **Stefanovska, A., Rebersek, S., Bajd, T., and Vodovnik, L.,** Effects of electrical stimulation of spasticity, *Crit. Rev. Phys. Rehab. Med.*, 3, 59, 1991.
124. **Bishop, B.,** Spasticity: its physiology and management, Part 1., neurophysiology of spasticity: classical concepts, *Phys. Ther.*, 57, 371, 1977.
125. **Rushworth, G.,** Some pathophysiological aspects of spasticity and the search for rational and successful therapy, *Conf. Proc., Third Congress of Int. Rehabil. Med. Assoc.*, Basel, July, 1978.
126. **Hudgson, P.,** Clinical features of spastic states, *Physiother. Can.*, 62, 323, 1976.
127. **Nathan, P. W.,** Factors affecting spasticity, *Int. Rehab. Med.*, 2, 27, 1980.
128. **Dietz, V., Ketelsen, U.-P., Berger, W., and Quintern, J.,** Motor unit involvement in spastic paresis, *J. Neurol. Sci.*, 75, 89, 1986.
129. **Gauthier-Smith, P. C.,** Clinical management of spastic states, *Physiother. Can.*, 62, 326, 1976.
130. **Spira, R.,** Contribution of the H reflex to the study of spasticity in adolescents, *Physiother. Can.*, 62, 401, 1976.
131. **Weintraub, A. H. and Opat, C. A.,** Motor and sensory dysfunction in the brain-injured adult, *Phys. Med. Rehabil.*, 3, 59, 1989.
132. **Katz, R. T., Rovai, G. P., Brait, C., and Rymer, Z.,** Objective quantification of spastic hypertonia: correlation with clinical findings, *Arch. Phys. Med. Rehabil.*, 73, 339, 1992.
133. **Price, R.,** Mechanical spasticity evaluation techniques, *Crit. Rev. Phys. Rehab. Med.*, 2, 65, 1990.
134. **Cornall, C.,** Self-propelling wheelchairs: the effects on spasticity in hemiplegic patients, *Physiother. Theor. Pract.*, 7, 13, 1991.