Reevaluating the Theoretical Model Underlying the Neurodevelopmental Theory

A Literature Review

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The purpose of this paper is to evaluate the foundations of a prevalent physical therapy technique based on the current research on motor control. The conceptual framework of the neurodevelopmental theory, as described in the writings of the Bobaths, is presented. Their explanations of central nervous system disorders and recommendations for intervention are based upon a unidirectional model of the nervous system in which postural and voluntary motion become two separate and distinct entities. Systems theory is an alternative model of nervous system structure. In systems theory, the organism is a circular network of interacting yet autonomous subsystems, rather than a vertical structure of descending controls. Relevant research that supports the systems viewpoint is discussed and applied to the theories in the neurodevelopmental approach. Thus, another model is offered for understanding the functioning of the central nervous system when it is intact and when it is in a pathological state.

Key Words: *Neural transmission, Motor control, Neurodevelopmental theory.*

Validation and acceptance of a clinical treatment require that the practice stem from an acknowledged theoretical model of the functioning organism and be supported by replicable research designs. One physical therapy technique that has popular acceptance through its empirical appropriateness is based on the neurodevelopmental theory (NDT) as devised by the Bobaths.[1-3] Their doctrine of early intervention[4,5] has been followed by clinical practitioners and has received several reports of positive results in the patient population.[6-8]

Generalized support of the doctrine by the medical field would require that the clinical observations on which this treatment is based be validated by the relevant research. The Bobaths originally substantiated the conceptual model of NDT through the theoretical approach of Jackson.[3] Jackson described the CNS as a hierarchically functioning structure in which the normal, more complex patterns of behavior (for example, righting and equilibrium reactions) are, at a higher level of organization, combinations of the same movements that make up the abnormal, phylogenetically simpler behaviors (for example, tonic neck and tonic labyrinthine reflexes).[3,9,10] Current literature on motor control and the recovery of function after brain damage suggests that the CNS does not function as a strictly descending hierarchy, but as a network of independent systems that support desired movement results through their interaction.[11,12]

Application of the approach based on NDT to the distributed control model of the CNS creates questions about the underlying assumptions of the technique. This paper is directed toward clarifying the concepts upon which NDT is based and evaluates them through an extensive review and presentation of the relevant literature. The following questions are answered:

1) How does the systems approach differ from the hierarchical model of the CNS?
2) Is normal movement production dependent upon a postural mechanism that provides for automatic responses?
3) What are the roles of tonus, reciprocal inhibition, and species-typical patterns of coordination in the production of normal and abnormal movement behaviors?

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THE NDT MODEL

The literature on motor control and its production refers to two systems of output: the open loop, or voluntary control mechanism, and the closed loop, or postural control mechanism.[13] The open-loop system commands sequences of movement that are centrally stored in the nervous system and that serve the functions of mobility in the production of isolated joint and limb motions. The closed-loop system is dependent upon afferent feedback for the elicitation of its automatic movements that serve as the principle motility or stability of the organism.[14]

In NDT, a closed-loop postural mechanism is the prerequisite for the development of normal movement behaviors. Called the normal postural reflex mechanism,[4] the closed-loop mechanism provides the dynamic background for movement in a constantly changing environment. The postural movements are automatic and arise from patterns of coordination that are common to our species.[1]

The postural reflex mechanism allows for skilled movements by modulating the level of tonus so that the organism is able to move against gravity. Three factors assumed to participate in the normal functioning of this mechanism are postural tone, reciprocal innervation of agonist and antagonist muscles, and patterns of coordination.[15] The patterns of coordination are reflected in the commonly described righting and equilibrium reactions.[16–19] With CNS lesions, malfunctioning of any one of the above factors might produce a dysfunction in the postural reflex mechanism and, consequently, a dysfunction in voluntary movement behaviors.

The Bobaths accept the hierarchical description of the lesioned CNS, that is, a system released from the inhibition of higher centers.[3, 10] In a lesioned state, the normal postural reflex mechanism would become abnormal as a result of the alteration of its three fundamental components into hypertonus, abnormal reciprocal innervation, and abnormal coordination.[15] In the abnormal postural reflex mechanism, the patterns of coordination are represented by the tonic neck reflexes, tonic labyrinthine reflexes, and associated reactions.[19, 20]

When placed within the hierarchical representation, the functional base of NDT is that the normal postural reflex mechanism is a prerequisite for normal movement.[4] In a damaged CNS, the abnormal reflex mechanism would be released from the control of the higher levels and dominate the movement so that normal sensorimotor experiences would be prevented. Input into a damaged system has been described as being shunted directly to the lower centers, thereby resulting in the production of the phylogenetically primitive movement patterns.[3] The idea of shunting is a purely descriptive conclusion and has no neurophysiologic support or validation.

THE SYSTEMS APPROACH

Contrary to the vertically designed model of the CNS as seen by the NDT theorists, research results in motor control suggest that the CNS does not operate in a strictly descending manner. The concept of descending neural inhibition of phylogenetically older behaviors by those higher on the evolutionary scale implies a localization and storage of concrete behaviors and an inefficiency and inflexibility that is not characteristic of the human system.[21]

In contrast to the unidirectional flow of information of the hierarchical model, a systems network has no higher levels with which to control the operation of the lower levels. Rather, there is a mutable relationship between the various levels so that each level will alternate between command and subordinate roles in relation to the other levels. This variation in level of controller is dependent upon the moment-to-moment demands of the external circumstances. When in control, the higher levels change the state and mode of interaction of the lower centers. The lower centers provide information about the state of the environment and the performer.[11]

Placing the CNS in a distributed control paradigm further disputes the hierarchical belief that the whole is simply a sum of its parts (that is, a constant additive representation of the movements found at ascending levels).[10] An interaction effect between the various levels contributes to the information about the whole but cannot be attributed to any one of the component processes.[12] In other words, it is important to know all the acting components and how they affect one another in order to account for the observed product of their actions.

In their "principle of least interaction," Gelfand and associates described the process of motor control as the coordinated actions of relatively autonomous subsystems.[22] The role of supraspinal influences would be to rearrange the interaction of the subsystems (or spinal neural mechanisms) so that they function in the most coordinated manner for resolution of the motor problem. Bernstein described the motor problem as the perception of an environmental situation that needs to be altered and the translation of that into an action particular to that individual.[23] Comparison of the two models uncovers the following differences in the systems model:

1) The functioning of spinal levels in the systems model is not inhibited or dominated by the commands of supraspinal levels. Spinal level output tends to be relatively consistent and invariant in its autonomous functioning, but it can undergo some modification as a result of supraspinal influences.

2) Whereas spinal levels are responsible for the basic manual work (that is, the final neural output producing a response) of movement production through their autonomous capacities, supraspinal levels provide the structure of a movement through functional synergies and reorganization of the interaction of the lower elements. The increasing complexity of these upward levels is not represented by increasing determinate combinations of movements, but by an increased variance in function as opposed to the invariability of the spinal levels. Functional variations are obtained through an integration and abstraction of environmental stimuli.

3) Habitual movements, elicited through stimulation of spinal levels, minimize the necessity for vertical interactions. This frees other sites for the analysis of input: supraspinal centers are particularly concerned with the unexpected (novel or nonequilibrium) sensory feedback from active movement. Once the spinal levels have been modified to correct any error between input and output, supraspinal areas are again free to engage in information reception during the course of a movement.

TOPICAL RESEARCH

The role of the primary control center is not the sending of direct commands, but the reorganization (tuning or priming) of the interactive system. Tuning is directed toward readying the system for an encounter with predicted or anticipated environmental demands. Gurfinkel and associates took EMG recordings of muscles in the lower extremities during the interval of a command and its response.[24] They found that there was a positive change in the amplitude of the tendon reflex responses as much as 70 msec before the start of the movement. At the same time, the systems that would engage in reciprocal inhibition of those reflexes were suppressed.

Higgins attributes to the open-loop voluntary mechanism the function of presetting (tuning), as well as resetting (error correction), the closed-loop postural mechanism, thus establishing the findings of Gurfinkel and associates as part of the control model.[13] The above results corroborate the contention of the Bobaths that tonus and reciprocal innervations are participating elements in the normal functioning of the postural mechanism[19]; however, their concept is enlarged by noting the essential contribution of the voluntary mechanism to the system's interactive dynamics.

The Bobaths considered normal tone coexistent with normal postural reflexes and abnormal tonus the result of a release of tonic reflex activity as part of the abnormal postural reflex mechanism.[17, 25] Increased tonus was considered not only a result of a loss of inhibitory influences, but also an inhibitor of normal patterns of movement.[3] Milani Comparetti believed that tone is a result, and not a determining factor, of the motor function of a damaged CNS.[8] Evidence supporting the conjecture of Milani Comparetti stems from the research of Sahrmann and Norton. From EMG recordings of active and passive flexion and extension motions of the upper extremities in hemiplegic adults and normal adults, Sahrmann and Norton found a positive correlation, on quantitative analysis, between the hyperactive stretch reflexes and the speed of a voluntary movement.[26] However, on qualitative analysis of the EMG activity, the primary cause of impairment of decreased movement speed did not appear to be the hyperactivity of the antagonist's stretch reflexes, but rather the diminished and prolonged response of the agonistic contraction. The agonist muscle was delayed in its initiation of the desired movement and then would not shut off when its action was to be terminated.

A possible conclusion to be drawn from the findings of Sahrmann and Norton is that limitations of movement in upper motor neuron syndromes are a result of decreased convergence of excitatory and inhibitory neural input to the anterior horn of the spinal cord, and thus to the alpha motoneuron. This would implicate both ascending and descending, excitatory as well as inhibitory, pathways in the production of hypertonus; hypertonus could not then be a result of a release phenomenon from the descending inhibition of higher centers. Further support arises from an EMG study on the neck, trunk, and arm musculature of cerebral palsied children.[27] On eliciting head control by pulling the children to a sitting position from the supine position, these investigators found a decreased response in the agonistic flexor muscles rather than any marked increase in the antagonistic extensors. The findings point to a temporal dysfunction in reciprocal innervation as a cause for voluntary movement disorders.

Bizzi investigated the control properties of coordinated movements through a head-positioning task in vestibulectomized monkeys and through an arm-positioning task in monkeys whose peripheral feedback (that is, dorsal roots and vision) was eliminated.[28] He hypothesized that the equilibrium points of the interplay between agonist and antagonist for every possible final position are coded in the CNS. Each equilibrium point represents a terminal location and is dependent upon the length-tension relations of the muscles around a joint. This is called the mass-spring theory of motor control. According to Bizzi, relationships between the joint musculature are coded spatially (for location) in the CNS and are representative of the levels of muscle stiffness.

A function of the active, contracting muscle for resistance to perturbations is the production of stiffness.[29] The initial stiffness of any muscle depends on its degree of activation prior to the movement in question. The slow conduction of the fusiform neuron axons causes the muscle's response to be one step behind the new task demand. Thus, initial stiffness of a muscle is a preset function.[30] The fusimotor loop maintains stiffness at an almost-constant level, thereby creating a useful tool for coding of positions.

Equilibrium points of the muscular interplay result from an interaction of the following factors: 1) centrally patterned commands to alpha and gamma motoneurons, 2) length-tension curves of agonist and antagonist muscles (stiffness), 3) passive elastic forces of the muscles, and 4) external loads from the environment. The establishment of equilibrium points was based on the biomechanical aspect of muscle stiffness. For these points to be accurate, a graded reciprocal innervation would be imperative at the level of the motoneuron. Combinations of the above four factors also seem to be representative of the interaction of the open-loop (descending, preprogrammed commands) and the closed-loop (feedback-dependent commands) control mechanisms that are postulated to produce adaptive movements.[13] Thus, reciprocal innervation, cited by the Bobaths as critical in the production of normal movement, is shown to be a participant in the interaction of the postural and voluntary mechanisms.

If the higher centers of the CNS are coding movement coordinations in terms of location and a balance of forces,[28, 31] where is the actual initiation of neuromotor impulses taking place? The impulse-timing theory of motor control suggests that there exist in the spinal cord "pattern generators" that innervate the relevant musculature. These generators would control the order of the muscular contractions, their force, and the timing of the contractions. Grillner[32] and Wetzel and Stuart[33] have found support for the existence of some internal program controlling the duration and intensity of the muscles involved in the locomotion patterns of deafferented animals. This program is believed to be held in the pattern generators and thus is a centrally stored command sent to the lower-level subsystems, which then select the specific muscles to be involved in the actuation of the program.[22, 34] The subsystems are composed of antagonists acting on the same joint (biomechanical synergies), postural reactions coordinating different sensory inputs (reflexes), and temporary connections that are made in order to match the requirements of a task.

The inclusion of reflexes in the normal output operations of the system implies that the dichotomy of normal and abnormal reflexes might not be valid. Rather, each of the reflexes that appear in the course of human development might be a necessary component of efficient and adaptive motor programs. Easton spoke of reflexes as coordinative structures; a group of muscles that automatically work together in response to certain stimuli compose each structure.[35] Easton extended the activation of these structures to include internal as well as peripheral stimuli, thus implicating them in the production of all normal motor functions. The coordinative structures of Easton are the wired-in motor responses that form the basic language of the motor programs of both control mechanisms. These include the stretch, crossed extension, tonic neck, tonic labyrinthine, and righting reflexes.

The appearance of reflexes, such as the tonic neck reflex during functional movements and when the subject is under stress, has been supported through research.[36–38] The identification of specific reflexes in the normal neonate has a history of controversy.[18] The possibility exists that the intact nervous system never exhibits a purely reflexive response. Instead, synergistic reactions (reflexes that have the appearance of components or elements of motor acts) are used. The tuning mechanisms (reflexes that bias the musculature according to intention) could modify the synergistic reactions in order to produce the variations in the motor programs.[35, 36]

Andre Thomas delineated the differences between reflexes and reactions. The reflex is characterized by predictability, constancy, and a fixed latency that produces a stereotypical response. Reactions are not as predictable and are dependent upon the circumstances that exist during their presentation.[39] Bobath claimed that the behaviors identified as reflexes in the Hirt and Waterland studies were too variable and did not dominate the motor behavior.[20] The difficulty with this criticism lies in the level of analysis. Assumptions would be better made on EMG evidence of fixed latencies and predictable muscular interplay than on variable movement behaviors. Measures of EMG were taken by Higgins, and the emergence of the tonic neck reflex was confirmed.[36]

Whether the tonic neck reflex can be observed in its sterotypical form during movement behavior, or whether it serves merely to bias the system for efficient response to a variety of environmental conditions, the evidence strongly indicates that synergic reactions are active in the production of volitional as well as automatic movements. That a dynamic continuum between reflexes and reactions exists is substantiated by the finding of differences in the temporal factors in the motor output of passive and active movements.[36] The inclusion of intention produces a bias in the system by the voluntary control mechanism so that the response time of a centrally initiated action is shortened. As Gurfinkel and associates suggest, there is a profound reorganization of the spinal segmental apparatus preceding the beginning of volitional movement.[24] Thus, reflex action in the formation of a movement is not abnormal but rather essential to the efficient production of movement behavior.

DISCUSSION

The Bobaths had formulated a theoretical model of neurodevelopmental disabilities that explained their clinical observations and experiences and was based upon an accepted interpretation of the CNS' structure-function interaction.[4,15,16,19] Recognizing the CNS as a systems network requires that there be an alteration in previous explanations of the causes of neuromotor dysfunction. Paucity of movement and production of stereotypical movements cannot be referred to as release phenomena, inasmuch as the role of higher mechanisms is not one of inhibition or control. The hypothesis that release of a lower-level abnormal postural reflex mechanism creates the observed motor dysfunction has no justification in a model that accords a functional role to all of its components.

In neurodevelopmental disabilities, the area of lesion is suspected to be at supraspinal levels; thus, the experiments of Bizzi[28] and Broadbent[11] have special significance. Assume that the region of equilibrium point mapping was damaged in one or more of its areas. The ability to formulate the interim locations for movement organization would be grossly curtailed. If the points were not completely lost, but blocked or altered by the reactions of tissue to injury (for example, edema or scar tissue), then the normal interplay of muscular forces would be altered. In either event, the individual would probably not experience or centrally code a variety of movement alternatives.

Tabary and associates state that the observed movements in a normal infant, which imitate the head-righting reactions, tend to be ephemeral and fluctuate in space between the flexed and hyperextended positions.[40] Their interpretation of this action was that the neonate was actually in the process of coding the normal postural circumstances. Without the feedback arising from the oscillations as a continuous code, behavior might rely upon the motor discharge that was coded for terminal positions. These terminal positions would be governed by the lower-level central programs inherent in that system and the gravitational forces acting upon the system. The result would be stereotypical behavior patterns identified by the Bobaths as abnormal reflexes. Patterns that normally prime the system for an intended motion would now dominate and become the movement.

Bobath attributed the production of normal and abnormal movement behaviors to the operation of three factors: tone, reciprocal innervation, and patterns of coordination.[15] After gathering recent evidence, disruption of the first (tone) and third (patterns) factors seem to be symptomatic results, whereas the second factor (reciprocal innervation) seems to be a cause of abnormal motor behavior when it has been disrupted. Bizzi distinctly tied his central coding of movement to the biomechanical as well as the central structures of the organism (that is, passive elastic forces, length-tension curves, and neural impulses to alpha and gamma motoneurons).[28] The tuning mechanism within the CNS might be even more fundamental—at a lower level than the observable spinal reflex patterns.[41] These basic patterns—or synergies providing the postural background for the movement—would involve a definite, restricted set of muscles, controlled as a unit, thus implicating the biomechanical structure of the species in the formation of neural programs.

CONCLUSIONS

It must be remembered that both NDT and systems theory are suggestions about the structure of the CNS. From this, assumptions about how the organism operates have been made. The treatment technique arising from NDT concentrates on influencing the postural mechanism. It approaches the central mechanisms, however, through altering the observed response behaviors rather than attempting to recognize and influence the stimuli that activate those responses. Evaluating the NDT treatment approach on the basis of a systems schema raises some pertinent questions that might be addressed through clinical research. First, which of the applied stimuli are actually controlling the observed response? If it is the stimuli to the autonomous subsystems, then the peripheral proprioceptive and tactile inputs might have the strongest effect. If descending control exists, verbal stimuli and goal setting will alter movement behaviors. The inclusion of a reflex in a movement behavior must be measured quantitatively to determine if it serves to ease or limit the performance. Finally, the synergic patterns need to be identified in order to apply and measure the effect of peripheral tuning (for example, vibration and tapping). Adoption of the systems theory and its approach to research could alter the treatment emphasis in physical therapy, clarify the ability to influence the patient, and aid in the evaluation of the patient's potential.

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