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REVIEW ARTICLE

Definition and Classification of Negative Motor Signs in Childhood

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ABSTRACT

In this report we describe the outcome of a consensus meeting that occurred at the National Institutes of Health in Bethesda, Maryland, March 12 through 14, 2005. The meeting brought together 39 specialists from multiple clinical and research disciplines including developmental pediatrics, neurology, neurosurgery, orthopedic surgery, physical therapy, occupational therapy, physical medicine and rehabilitation, neurophysiology, muscle physiology, motor control, and biomechanics. The purpose of the meeting was to establish terminology and definitions for 4 aspects of motor disorders that occur in children: weakness, reduced selective motor control, ataxia, and deficits of praxis. The purpose of the definitions is to assist communication between clinicians, select homogeneous groups of children for clinical research trials, facilitate the development of rating scales to assess improvement or deterioration with time, and eventually to better match individual children with specific therapies.

"Weakness" is defined as the inability to generate normal voluntary force in a muscle or normal voluntary torque about a joint. "Reduced selective motor control" is defined as the impaired ability to isolate the activation of muscles in a selected pattern in response to demands of a voluntary posture or movement. "Ataxia" is defined as an inability to generate a normal or expected voluntary movement trajectory that cannot be attributed to weakness or involuntary muscle activity about the affected joints. "Apraxia" is defined as an impairment in the ability to accomplish previously learned and performed complex motor actions that is not explained by ataxia, reduced selective motor control, weakness, or involuntary motor activity. "Developmental dyspraxia" is defined as a failure to have ever acquired the ability to perform age-appropriate complex motor actions that is not explained by the presence of inadequate demonstration or practice, ataxia, reduced selective motor control, weakness, or involuntary motor activity.

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Key Words
weakness, selective motor control, ataxia, apraxia, dyspraxia, developmental coordination disorder, cerebral palsy, movement disorders

Abbreviation
DCD— developmental coordination disorder

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CHILDREN WITH MOTOR disorders often have a combination of multiple symptoms and clinical signs that contribute to their disability. One general classification of motor signs distinguishes 2 basic categories: positive signs and negative signs.[1] Positive motor signs can be defined as those that lead to involuntarily increased frequency or magnitude of muscle activity, movement, or movement patterns. Examples include hypertonia, chorea, tics, and tremor. Negative motor signs describe insufficient muscle activity or insufficient control of muscle activity. Examples include weakness, impaired selective motor control, ataxia, and apraxia.

Positive motor signs are often easier to detect in the clinic, and there has been significant effort to identify and quantify such signs.[2–7] Several treatment options can decrease tone and reduce involuntary movements for some children (eg, see ref 8). Negative motor signs are often more difficult to quantify, and there are fewer effective treatments. Nevertheless, negative signs may be even more significant contributors to disability than positive signs.[9] For example, although hypertonia is a frequently measured clinical sign and an indication for medical treatment, a child with spastic diplegia may have a greater component of disability that results from lower-extremity weakness[3,4,10,11] and the inability to selectively activate specific muscles.[12] Positive and negative motor signs are often simultaneously present and may be linked rather than independent features of a motor disorder.[13]

Several different classification schemes for assessment of disability have been proposed,[14] including the National Center for Medical Rehabilitation Research classification and the International Classification of Functioning, Disability, and Health.[15] These schemes distinguish between the underlying pathophysiology or etiology of the disorder, the observable impairment including clinical signs and symptoms, and the functional consequences of the impairment that may include difficulty performing tasks or participating fully in life situations. The relationships between pathophysiology, impairment, functional consequences, activity limitations, and participation are often complex. As a first step toward understanding these complex relationships and developing new treatments, it is essential that consistent definitions of impairment be available.

The purpose of the definitions we propose is to assist communication between clinicians, select homogeneous groups of children for clinical research trials, facilitate the development of rating scales to assess improvement or deterioration with time, and eventually to better match individual children with specific therapies. The ultimate goal is to improve functional outcome and reduce disability for children with motor disorders.

Our goal is to define some of the common negative motor signs at the level of impairment without reference to etiology or functional consequences. An important reason for this decision is to permit disease to be inferred and treatment to be chosen on the basis of observable elements of the clinical examination. We do not intend to ignore the importance of the causative pathophysiology or the resulting limitations in functional abilities. Although little is known about the underlying causes of the motor disorders we discuss, we recognize that pathophysiology is an essential determinant of impairment and dysfunction; thus, we hope that those conducting research in the future will be able to study the relationships between etiologies and clinical outcomes. We also recognize that reducing the functional consequences of motor impairments is a primary goal of treatment.

Multiple impairments may occur simultaneously, which complicates identification of individual deficits. Recognition of different patterns of coexisting impairments may define specific clinical syndromes. Our goal is to define negative clinical signs without reference to the syndromes of which they may be a part. We further believe that it is essential that the definitions allow recognition of specific signs in a clinical environment without the requirement for specialized tools or other equipment.

We have limited this discussion to 4 motor signs that we believe are significant contributors to reduced functional ability in children, namely weakness, reduced selective motor control, ataxia, and deficits of praxis. However, we acknowledge the existence of other components that may contribute as much or more to reduced function, including sensory deficits, biomechanical limitations, abnormalities of posture and balance, cognitive deficits, learning disabilities, fatigue, and decreased motivation. We have further limited our discussion to include only limb and trunk signs, and in particular we do not discuss oculomotor or oromotor signs.

CATEGORIES OF NEGATIVE MOTOR SIGNS

1. Insufficient muscle activation (weakness)
2. Inability to activate a specific pattern of muscles (reduced selective motor control)
3. Inability to activate the correct pattern of muscles during movement (ataxia)
4. Inability to activate the correct pattern of muscles to accomplish a task (apraxia and developmental dyspraxia)

A partial taxonomy of negative signs can be based on the manner in which the deficit is elicited. For example, weakness is assessed during attempts to generate force in a single joint at one point in time. Reduced selective motor control is assessed during attempts to generate a pattern of force or relaxation in multiple joints at one point in time. Ataxia is assessed during attempts to generate movement over time and space. Apraxia and developmental dyspraxia are elicited during attempts to

generate posture or movement as part of a complex multiple-component or goal-oriented task.

On the other hand, the taxonomy can be based on whether a deficit occurs independent of certain contexts. For example, we expect that when weakness is present, it is manifest whenever activation of the involved muscles is required, independent of the particular pattern, movement, or task in which the muscles participate. When reduced selective motor control is present, it is manifest whenever particular patterns of muscle activation are required, either as an isolated effort or as part of a movement or task. When ataxia is present, it is manifested during many movements involving the affected limb regardless of whether those movements occur as part of a complex task. When apraxia or dyspraxia is present, it is manifested only in the context of a complex task.

Weakness

"Weakness" is defined as the inability to generate normal voluntary force in a muscle or normal voluntary torque about a joint.

By "normal" force and torque we refer to the range of values that would be expected in that muscle or joint in unaffected children of the same age and size or in the unaffected contralateral limb of the same patient. By "voluntary" we require that the force or torque be generated in response to instruction, imitation, or other maneuvers.[16] For the purposes of quantifying weakness, this would require the child to make a near-maximal level of effort.

We include in the definition the inability to generate normal voluntary force in a single muscle to accommodate muscles that do not cross a joint (such as facial muscles), but we currently have no method to make objective measures of the force exerted by such muscles. More often, clinical testing of weakness will assess an inability to generate normal voluntary torque about a joint on the basis of net force exerted by the child and measured by the examiner at a single point on a body segment just distal or proximal to the joint (eg, see ref 17). The resultant torque would be due to all the skeletal muscles crossing that joint and is calculated as the product of the distance from the location of force measurement to the center of joint rotation and the measured force. Reporting of torque is encouraged for research and comparison between different children and examiners because a calculated torque about a joint resulting from a measured force on a body segment is independent of the location of force measurement, whereas measured force can differ on the basis of the point of measurement. For comparison between different children, normalization of force and torque by height may reduce variability of data.[18]

Weakness can occur in the presence of hypertonia, hyperkinetic disorders, or other involuntary movements.[19,20] However, weakness may be masked by the fact that a muscle with spasticity, dystonia, or rigidity may resist passive movement by the examiner.[5] Weakness may also be masked by the appearance of significant active but involuntary joint extension torque resulting from dystonia. According to our definition, in such cases the force generated involuntarily does not exclude the presence of weakness.

In some cases, there may be reduced torque about a joint resulting from obligate co-contraction of muscles that are generating normal levels of force.[21] In other cases, the child may have voluntary control of the degree of co-contraction, but he or she may not be able to modulate the relative contribution of agonist and antagonist muscles and, therefore, may not be able to modulate the resulting joint torque.[22] Therefore, a child may be unable to generate normal voluntary torque about a joint despite muscles that are, individually, generating large voluntary forces. This situation can occur in dystonia or with a deficit of selective motor control,[23] but it may be difficult to confirm during clinical examination without the use of specialized equipment. According to the definition shown above, it is a form of weakness because there is an inability to generate normal voluntary torque about a joint. (In such cases, it is important to realize that individual muscles may generate normal levels of force, a fact that may determine the choice of medical or surgical interventions.)

When weakness is present it will normally be manifest in many different postures, movements, or tasks. In some cases, however, the weakness may only be evident for certain joint angles, speeds of movement, or postures of other joints.[24] Ability to generate force may deteriorate as a result of early fatigue of muscles, reduced endurance, or an inability to generate a sufficiently rapid increase in force,[25] and in some cases it may be important to measure both the ability to maintain force over time as well as the ability to generate a brief rapid force. In such cases, we encourage the use of more descriptive terms to specify the conditions under which reduced force or torque was observed. It may be important to document other factors such as time of day, level of alertness, and degree of motivation.

In the context of significant contractures or other limitations of range of motion, it may not be possible to identify weakness.[26,27] In addition, if it is not possible to establish the level of effort exerted by the child then it may not be possible to measure weakness. For example, it may not be possible to test very young children or those with cognitive deficits or psychiatric disorders unless a task can be devised in which it is likely that they are making a significant effort to drive the tested muscles. In very young children and infants, weakness is one possible cause of hypotonia, and hypotonia may be the only clinically evident sign of weakness in a young or uncooperative child.[28]

There are many possible causes of weakness, and weakness may be caused by dysfunction in many different parts of the neuraxis including the cerebrum, corticospinal and bulbospinal tracts, spinal cord, lower motor neuron, neuromuscular junction, or contractile elements of muscle. Such dysfunction could lead to decreased descending drive to the spinal cord, decreased drive to muscles, or decreased muscle force in response to neural drive. In the context of chronic weakness there may be changes in passive and active muscle properties that either perpetuate or partially counteract the weakness.[29–32]

Reduced Selective Motor Control

"Reduced selective motor control" is defined as the impaired ability to isolate the activation of muscles in a selected pattern in response to demands of a voluntary posture or movement.

By "muscles in a selected pattern" we refer to the simultaneous activity level of each muscle in a group of muscles in which certain muscles may be activated while others are relaxed. By "expected or desired" we refer to the pattern that would be expected to be observed in unaffected children during the elicited posture or movement. By "impaired ability" we indicate that the expected pattern is not completely achieved, either because of excessive activation of muscles that would be expected to be relaxed or inability to activate muscles that would be expected to be active.[12] By "voluntary" we again require that the pattern of muscle activity be generated in response to instruction, imitation, preparation for movement, or other maneuvers.

When reduced selective motor control involves decreased muscle activation, then particular muscles may be unable to generate full force when they are a part of the abnormal pattern of activity.[33] We distinguish this situation from weakness if the muscles are able to generate full force in other contexts. When weakness is present, it may not be possible to determine if a deficit of selective motor control exists, because the pattern of activation will be abnormal simply because of the inability to activate the weak muscle(s). However, if weakness is mild then it is possible that reduced selective motor control could be detected if the abnormality in the pattern of activation is out of proportion to what would be expected if it were solely caused by pattern-invariant weakness in 1 or more muscles.

We allow use of the term "reduced selective motor control" whenever the definition is met independent of the cause. For example, a task-specific dystonia may lead to a deficit of selective motor control as a result of involuntary activation of patterns of muscles during attempts at a particular task.[5] A child with congenital mirror movements will exhibit a bilateral reduction in selective motor control, with obligate activation of corresponding muscles on both sides of the body.[34]

Certain patterns of reduced selective motor control are frequently recognized between different children. For example, there may be activation of the knee and hip flexor muscles during a simultaneous attempt at ankle dorsiflexion, or there may be reduced activation of elbow extensors during shoulder abduction.[12,33] These recognizable patterns are sometimes referred to as "obligate synergies," reflecting the assumption of the existence of muscle synergies that lead to simultaneous activation of groups of related muscles. Just as patterns are recognized across different children, we expect that an abnormal pattern of muscle activation in a single child will be reproducible over time.

Reduced selective motor control may be manifested by abnormal postures or unusual movement patterns. For example, in the presence of obligatory synergistic muscle activation that permits elbow extension only during shoulder adduction, it may be necessary to adduct the shoulder or press downward against a table to generate elbow extension during a reaching task.[24,33]

Certain abnormal muscle activation patterns or synergies are recognized as occurring in the context of lesions of the descending spinal tracts, and these patterns have been called the "upper motor neuron" pattern.[35,36] Although the origin of this pattern is not completely known, research suggests the involvement of spinal and brainstem mechanisms in the generation of these synergies. Synergies could be manifest by a limited ability to regulate individual muscle activity or reflex thresholds.[22] A reduction of corticospinal drive may lead to an increased dependence on brainstem pathways, which branch extensively compared with the relatively focused projections of the corticospinal tract.[37] The increased reliance on descending subcortical pathways may give rise to the appearance of synergistic activation of multiple muscles during voluntary action. On the other hand, evidence of cortical reorganization after central nervous system injury[38,39] and the presence of cortical neurons reflecting the activation of multiple muscle groups suggest a cortical origin of muscle synergies.[40]

Ataxia

"Ataxia" is defined as an inability to generate a normal or expected voluntary movement trajectory that cannot be attributed to weakness or involuntary muscle activity about the affected joints.

As in the previous definitions, we use "normal" to emphasize that function is to be compared against expected and age-appropriate performance or the performance of an unaffected contralateral limb. "Voluntary" indicates that the movement must be performed in response to instruction, imitation, or other motivation. A "trajectory" is a series of positions or joint angles over time, and we use this term to indicate that either the timing or the spatial pattern of muscle activity could be affected. In some cases, this will lead to decreased accu-

racy, through failure to either achieve a desired trajectory or contact an intended target. We exclude weakness as a cause of abnormal trajectories, but we note that many other motor disorders could lead to abnormal trajectories or reduced accuracy. We exclude involuntary muscle activity to eliminate hyperkinetic or hypertonic disorders such as spasticity, dystonia, chorea, myoclonus, or tremor from the definition of ataxia, although such movement disorders may coexist with ataxia.

Ataxia may be present in multiple parts of the body, leading to disorders of gait, limb, or trunk control.[41,42] It also may occur only during certain types of movement. For example, there may be a greater abnormality for multijoint movements or greater deficits of rhythmic compared with nonrhythmic movements.[43,44] However, the deficits are not specific to particular tasks or goals, which distinguishes ataxia from apraxia and developmental dyspraxia.

Specific deficits that may be seen as components of ataxia include dysmetria (inaccurate motion to a target either undershooting [hypometria] or overshooting [hypermetria]), dyssynergia (decomposition of multijoint movements), and dysdiadochokinesia (lack of rhythmicity or excessive difficulty performing rhythmic tasks).[45,46] Increased movement variability and intention tremor can occur, and it is not known whether these are primary deficits or compensatory responses.[47,48] Although ataxia is associated with increased movement velocity or variability, we classify it as a negative sign because disability is more closely linked to the failure to compensate for interjoint dynamics during rapid multijoint movements.[49–51] For example, failure to adjust trunk musculature before or during reaching may lead to postural instability and poor reaching,[52] and failure to account for the effect of shoulder movement on elbow torque may lead to inaccurate control of the elbow.[53,54] Problems with the control of interjoint dynamics are not limited to ataxic syndromes but have been suggested as underlying the deficits in trajectory formation in adults with hemiparesis.[33,55] We also note that even single-joint movements may involve abnormal timing, rhythmicity, or magnitude of muscle activation.[54,56,57]

Ataxia is often observed in association with injury to the cerebellum or to its inflow or outflow tracts.[58] It also can be seen in the context of peripheral sensory loss, particularly when that sensory loss affects the large fibers that carry proprioceptive information. We do not know if late-acquired cerebellar dysfunction or sensory loss manifests differently from congenital or early-acquired dysfunction. The cerebellum has many functions during development,[59] and it would be reasonable to expect different manifestations of deficits to occur at different developmental stages.

Apraxia and Developmental Dyspraxia

Deficits in praxis can take 2 forms: apraxia and developmental dyspraxia.

"Apraxia" is defined as an impairment in the ability to accomplish previously learned and performed complex motor actions that is not explained by ataxia, reduced selective motor control, weakness, or involuntary motor activity.

"Developmental dyspraxia" is defined as a failure to have ever acquired the ability to perform age-appropriate complex motor actions that is not explained by the presence of inadequate demonstration or practice, ataxia, reduced selective motor control, weakness, or involuntary motor activity.

Praxis refers to the ability to perform complex learned motor actions. In children, it is essential to determine if a task has been previously learned and performed to understand the origin of a deficit of praxis. Therefore, we define 2 separate entities in children: "apraxia" is an acquired disorder that leads to the loss of a learned skill, whereas "developmental dyspraxia" is the failure to have acquired a skill that a child would ordinarily be expected to exhibit at that age.

The essential distinction between apraxia and developmental dyspraxia is whether the child ever learned and competently performed the motor acts at some time in the past. For apraxia, the skill must have been lost as a result of an injury or disorder that occurs after the time of skill acquisition. For developmental dyspraxia, the skill must never have been acquired despite attempts, so there must be evidence of impairment of learning or performance of a novel task or group of tasks. Therefore, both the history of the disorder and the current manifestation of the impairment contribute to the definition of disorders of praxis in children.

By "an impairment in the ability" we mean that the motor acts are performed in a manner that is awkward, slow, or fails to accomplish the desired goal. By "complex motor actions" we refer to actions that may have multiple components and are associated with goal-oriented task performance, tool use, or gestures. Such actions include 1 or more specific skills (eg, pantomimed brushing teeth, throwing or kicking a ball, jumping rope), gestures (eg, OK sign), postures (eg, thumb to thumb), and sequences that are commonly used in a clinical examination (eg, picking up and then using a tool), as well as more naturalistic actions that reflect tasks of behavioral relevance to the child. We exclude simple movements (eg, reaching to a target) and nonpurposeful rhythmic movements (eg, finger-tapping). We note that whether a movement is goal oriented may depend on the context in which it is elicited (for example, touching the tips of the thumb and the index finger may be a meaningless movement when elicited by demonstration and a more goal-oriented movement when elicited by a request to make the "OK" sign). By "age-

appropriate" we intend to compare both the specific task and its quality of performance to what would be expected of children at a similar age with no known motor impairment. For developmental dyspraxia, we specifically exclude the possibility that the child is simply unfamiliar with the action because of inadequate demonstration or practice, or that the child is unable to understand the instructions. Assessment of impairments of praxis requires that the examiner ascertain (1) age, (2) familiarity with the skill or gesture, (3) adequate demonstration or explanation of the task, (4) appropriate understanding of the demonstration or explanation, (5) adequate muscle force, selective voluntary control, balance, endurance, and flexibility to perform the task, and (6) motivation to perform the requested action.

For both types of praxis deficits we specifically exclude other motor disorders that, by themselves, may explain the poor quality of performance or inability to accomplish a task. We also note that there may be a subgroup of children for whom a generalized inability to perform motor skills may be sufficiently mild to not interfere with successful task performance of basic ontological skills yet still leads to deficient age-appropriate skill acquisition and poor quality of performance. These children are often described as being clumsy. Such children are not specifically either excluded or included, and we recognize a need for additional research in this area.

Apraxia in adults has been divided into subgroups by Liepmann[60] and others. Subgroups include ideational, ideomotor, and limb-kinetic apraxias, although the use of these terms varies among different authors.[61,62] The intent of these subgroups has been to identify deficits that occur at various stages of the movement-selection, motor-planning, and movement-execution process, although it is not known whether the process can, in fact, be divided into these discrete stages.[63] We do not discourage the use of these terms when children meet criteria according to the adult definitions and, in fact, suggest that each definable type, if found in isolation, may be remediated by a different method specific to it. Sometimes, if more than 1 type is found in the same child, combined remediation specific to each observed type is useful.[64] Whereas most literature on dyspraxia in children has focused on ideomotor type, May-Benson[65] has described ideational problems. We emphasize a need for additional research to determine which, if any, of these distinctions are meaningful in children of different ages and whether these distinctions or others specific to children will aid in the selection of appropriate interventions.

The causes of developmental dyspraxia are unknown, but this disorder may be associated with early mild global cortical injury in some children with mild cerebral palsy or other static disorders.[66,67] In adults, the lesion in apraxia is often located in the left frontal or parietal cortex and is often associated with aphasia. We hypothesize that developmental dyspraxia in childhood could be associated with maturational processes in similar locations, but this has not been tested yet. Functional imaging suggests a role for parietal association areas, premotor cortex, and supplementary motor area in the planning and execution of complex movement sequences in adults.[44,68–73] Such noninvasive methods may eventually help to refine our understanding of praxis disorders in children. We note that a lesion that leads to a loss of skill in apraxia may impair learning of new skills; thus, the same lesion also may be a cause of developmental dyspraxia.

For this article we sought to define signs independently of their causes, but we note that developmental dyspraxia is likely to be strongly associated with a disorder of motor learning. In fact, because our definition requires poor performance despite demonstration and adequate practice, it requires a decreased ability to learn simply by observation and practice. Therefore, developmental dyspraxia most probably arises from a motor learning disability or a performance deficit that affects learning, and it reflects impaired ability to acquire new skills. The relationship between developmental dyspraxia and other developmental disorders (eg, attention-deficit disorder, dyslexia, and learning disabilities) is unknown but raises the intriguing possibility of a family of related disorders of higher cognitive development that may share a common pathophysiology.

For more than 15 years, researchers and clinicians have been working to provide definitions and investigate treatment for a group of related disorders that collectively have been termed "developmental coordination disorder" (DCD). The London consensus meeting in 1994 defined DCD as "an impairment of both functional performance and quality of movement that is not explicable by age, intellect, or other diagnosable conditions."[74] DCD includes deficits in motor planning and execution; therefore, the definition closely mirrors our definition of the impairment of developmental dyspraxia.[75,76] We expect that developmental dyspraxia as we have defined it will commonly be seen in DCD and may be one of its cardinal features. However, we emphasize that developmental dyspraxia is distinct from DCD and may or may not be present in individuals with DCD. Thus, the diagnosis of DCD is not required to use the term "developmental dyspraxia." Conversely, a child may have developmental dyspraxia yet not meet other criteria for a diagnosis of DCD.

Other Negative Signs in Childhood

Deficits of sensory function including tactile, kinesthetic, or proprioceptive sensation may be a cause of poor motor performance.[77–80] Sensory information is needed to determine the starting position of a limb before movement, and this information is essential for accurate movement planning. Sensory information and attention

are needed to correct for errors during movement and to determine errors in the outcome of movement to drive motor learning and improve performance.[81] It is possible, therefore, that a sensory deficit is a cause of developmental dyspraxia by preventing skill acquisition or refinement. Deficits of higher-order sensory function (sometimes called sensory motor integration deficits) may impair the ability to determine spatial relationships between objects and, therefore, could interfere with tool use, bimanual coordination, or task-planning.[82]

Another important contributor to negative motor signs is neglect. Neglect of a limb may lead to inadequate effort and, thus, could be a cause of weakness. Neglect may also lead to inadequate practice or self-observation and thereby slow learning of complex tasks. A particularly important form is "learned nonuse" or "developmental disregard," which occurs in association with unilateral or asymmetric motor deficits, because this syndrome may be amenable to treatment using constraint-induced movement therapy.[83]

We note that disorders of posture and balance, oculomotor control, vision, endurance, motivation, attention, other nonmotor learning disabilities, or other cognitive deficits may all be causes of negative motor signs. There are additional signs, including bradykinesia and hypotonia, that could be classified as negative signs but have not yet been adequately studied in children.

CONCLUSIONS

Our purpose in establishing these consensus definitions was to distinguish different clinical signs from each other yet unify the opinions of experts from multiple fields. Our intention was to establish clinically useful definitions that have sufficient sensitivity to capture the full range of each impairment but are sufficiently specific so as not to include children who are better classified otherwise. We believe that these definitions allow for the simultaneous presence of more than 1 impairment, which is frequently the case in children with motor disorders. We have worked to ensure that our definitions remain at the level of impairment independent of pathophysiology, functional ability, activity, or participation.

Definitions, by their very nature, are expected to change over time as a result of changing clinical practice and new research results. We fully expect and, indeed, hope that improvements and refinements of these definitions will be made over the coming years. Our attempt here was to create a starting point for future discussion and research to ensure that clinicians and researchers are in agreement in their current use of terminology.

The next step in this process is the creation and validation of rating scales or other quantitative instruments that are based on these definitions. Such instruments allow for quantitative comparison between children and inclusion of homogeneous groups of children in research trials. In addition, the results of studies of validation of rating scales provide important data for modification of our definitions. We hope that through a continuing process of defining, measuring, and testing these impairments, it will be possible to make significant progress toward the evaluation of new treatments for children with motor disabilities.

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