Variation and Variability: Key Words in Human Motor Development

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This article reviews developmental processes in the human brain and basic principles underlying typical and atypical motor development. The Neuronal Group Selection Theory is used as theoretical frame of reference. Evidence is accumulating that abundance in cerebral connectivity is the neural basis of human behavioral variability (ie, the ability to select, from a large repertoire of behavioral solutions, the one most appropriate for a specific situation). Indeed, typical human motor development is characterized by variation and the development of adaptive variability. Atypical motor development is characterized by a limited variation (a limited repertoire of motor strategies) and a limited ability to vary motor behavior according to the specifics of the situation (ie, limited variability). Limitations in variation are related to structural anomalies in which disturbances of cortical connectivity may play a prominent role, whereas limitations in variability are present in virtually all children with atypical motor development. The possible applications of variation and variability in diagnostics in children with or at risk for a developmental motor disorder are discussed.
Human behavior is characterized by variation; each human individual has a large repertoire of motor, cognitive, and social actions that can be arranged in virtually endless combinations. This repertoire allows for a flexible adjustment to changing conditions, including the creation of new solutions.

The wealth of distinctly human behavior is attributed to the neocortex, the part of the brain that expanded greatly during evolution.1,2 For instance, in insectivores such as the hedgehog, the neocortex occupies 10% to 20% of total brain volume, whereas this proportion has risen to about 80% in humans.3 The enlargement of the neocortex has been brought about mainly by expansion of the surface area and not so much by an increase in cortical thickness.2 The expansion of cortical surface allowed for the emergence of new areas (eg, language-related areas) and the extension of the prefrontal cortex and association areas. Interestingly, the volume of the white matter—mostly consisting of corticocortical connections—increased more during the evolutionary expansion of the cortex than the volume of gray matter.5,4

The development of the human brain is an intricate and long-lasting process, which is mirrored by a multitude of developmental changes in behavior. The latter include the changes involved in the transformation of non–goal-directed fetal motility into the accurate and goal-directed movements of an adult person, such as those involved in writing a letter or riding a bike. The aim of this article is to discuss putative mechanisms and principles underlying developmental changes in motor behavior. Particular attention is paid to the notions of “variation” and “variability.” The article starts with a short overview of the ontogeny of the human brain. Sections on theoretical considerations and typical and atypical motor development follow. The emphasis is on the early phases of development and the Neuronal Group Selection Theory (NGST) is used as a frame of reference. The NGST was chosen because it highlights that variation and variability are key elements of typical development. Variation implies the presence and expression of a broad repertoire of behaviors for a specific motor function. Variability denotes the capacity to select from the repertoire the motor strategy that fits the situation best. The article concludes with possible applications of variation and variability in diagnostics in infants with or at risk for a developmental motor disorder.

**Development of the Human Brain**

The development of the human brain, and in particular, that of the neocortical circuitries, lasts about 4 decades.5 It starts during the early phases of gestation with the proliferation of neurons. The majority of telencephalic neurons are produced in the germinal layers near the ventricles.2,6 Once neurons have been generated, they move from their place of origin to their final destination, the cortical plate.6,7 Before the cortical plate is formed, however, neurons halt in the subplate. The subplate is a temporary layer between the ventricular zones plus intermediate zone (the future white matter) and the cortical plate.8 The subplate emerges in early fetal life, is thickest at around 29 weeks postmenstrual age (PMA), and disappears gradually until it is absent at around 6 months post-term.9,10 The major proportion of its afferent and efferent connections run through the (future) periventricular white matter. The subplate mediates fetal behavior.

Neurons start to differentiate during migration and during their stay in the subplate. Neuronal differentiation includes the formation of dendrites and axons, the production of neurotransmitters and synapses, and the elaboration of the intracellular signaling machinery and complex neuronal membranes.11,12 The process of differentiation is particularly active in the few months prior to birth and the first postnatal months, but it takes many years before the adult state of differentiation is achieved.5 Besides neuronal cells, glial cells are generated. The peak of glial cell production occurs in the second half of gestation. Some of the glial cells take care of axonal myelination. Myelination takes place especially between the second trimester of gestation and the end of the first postnatal year. Thereafter, myelination continues till the age of about 40 years, when the last intracortical connections complete myelination.13

Brain development consists not only of creation of components, but also of an elimination of elements. About half of the created neurons die off by means of apoptosis. Apoptosis is brought about by interaction between endogenous programmed processes and chemical and electrical signals induced by experience14; it occurs in particular during midgestation.5 Similarly, axons and synapses are eliminated, the latter especially between the onset of puberty.
and early adulthood. As a result, the adult level of synaptic density in the cortex is reached first in early adulthood.5

For a long time, it had been debated whether brain development is driven by endogenous processes or by external input. Gradually, however, it became clear that genetic instruction ("nature") and environmental information ("nurture") both play an important role, albeit with different weights during different phases of development. In the early phases, the role of the genome dominates; later on, environment and experience become crucial. The importance of genetic instruction in early development is reflected by animal studies that indicated the primary cortical areas, their connections, their modular organization, and their size are largely determined by differential gene expression in the neural stem cells.1,15 Thus, the genetically specified areas attract specific inputs instead of inputs specifying the areas.2 This means that the functional topography of the brain is primarily driven by genetic instruction (ie, the fact that occipitally located neurons virtually always become involved in the processing of visual information and frontally located networks in activities such as planning and attention). A primary genetic determination, however, does not preclude variation, as each individual has his or her own sets of genes. Moreover, the primary genetic determination is only the starting point for epigenetic cascades, allowing for abundant interaction with the environment and activity-dependent processes.16–18 Note that the interaction is bidirectional: experience affects gene expression, and genes affect how the environment is experienced.18

Virtually all of the neurodevelopmental processes described above are affected by experience, including motor experience. Animal studies have demonstrated that the effect depends on the type of experience (eg, specific versus generalized motor experience), the age at exposure, the individual’s sex, and the neural area.18,19 Experience may affect, for instance, apoptosis, axon retraction, synapse elimination, and synapse formation.19–21 It may even affect the somatotopic organization of the primary motor cortex, as was indicated by the recent human study by Stoeckel et al.22 This imaging study revealed that the motor “foot” representation in individuals with congenitally compromised hand function and compensatory skillful foot use had extended beyond the classical foot area into the vicinity of the lateral hand area.

Theoretical Considerations

Various Theoretical Frameworks

Despite increasing knowledge of the developmental processes in the human brain, our understanding of the neural mechanisms underlying motor development is limited. As a result, multiple theories of motor development have been produced, all aiming to facilitate the understanding of typical and atypical motor development. During the major part of the previous century, motor development basically was regarded as an innate, maturational process.23–24 but during the century’s last 2 decades, it became increasingly clear that motor development is largely affected by experience.

Currently, 2 theoretical frameworks are most popular: dynamic systems theory25–27 and NGST.28,29 The frameworks share the opinion that motor development is a nonlinear process with phases of transition that is affected by many factors. The factors may vary from features of the child to external influences such as housing conditions, the presence of stimulating caregivers, and the presence of toys. In other words, both theories acknowledge the importance of experience and the relevance of context. The 2 theories differ, however, in their opinion on the role of genetically determined neurodevelopmental processes. Genetic factors play only a limited role in dynamic systems theory, whereas genetic endowment, epigenetic cascades, and experience play equally prominent roles in NGST.28,29 In the following paragraphs, I will use the NGST framework to discuss principles of typical and atypical motor development.

NGST and Typical Motor Development

The NGST was developed by Gerald Edelman. He described motor development as characterized by 2 phases of variability: primary and secondary.28,29 The borders of variability are determined by genetic instructions.1,15 During the phase of primary variability, motor behavior is characterized by abundant variation. The variation is brought about by explorative activity of the nervous system. The system explores all motor possibilities. The exploration generates a wealth of self-produced afferent information, which, in turn, is used for further shaping of the nervous system. The exploration reflects the continuous, dynamic interaction between genes and experience, including experience with changing body proportions. Initially, however, the afferent information is not used for adaptation of motor behavior to environmental constraints. In other words, the phase of primary variability is characterized by variation in motor behavior and the absence of the ability to adapt the various movement possibilities to the specifics of the situation (ie, by the absence of variability in sensu strictu, as defined in the introduction of the article).24

At a certain point in time, the nervous system starts to use the afferent
information produced by behavior and experience for selection of the motor behavior that fits the situation best: the phase of secondary or adaptive variability starts. Hitherto, the mechanisms underlying the shift from primary variability to secondary variability are not understood. The process of selection, which is characteristic of variability and thus the phase of secondary variability, is based on active trial-and-error experiences that are unique to the individual.30–32 Indeed, evidence is accumulating that self-produced sensorimotor experience plays a pivotal role in motor development.31,33–35

To determine whether a movement is most adaptive, reference values are used that most likely are function specific. The solution that is selected is specific for the situation and the infant’s stage of development. For instance, in sitting infants whose balance is perturbed, information linked to the stability of the head in space is used to select the postural adjustment in which most of the so-called direction-specific postural muscles are recruited (the en bloc pattern).36 Selection of the en bloc pattern depends on the degree of balance perturbation—the pattern is recruited more often during large perturbations than during small perturbations—and the age of the child.36,37 Children select the en bloc pattern during marked perturbations of balance especially often between the ages of 9 months and 2½ years. Thereafter, similar perturbations of balance are associated with child-specific postural adaptations in which fewer direction-specific muscles are recruited.37

The process of learning to select the most appropriate motor solution in a specific situation is based on implicit motor learning and does not involve conscious decision making. It occurs at various interdependent levels of neural organization. Animal data indicate that at the cellular level, selection is mediated by changes in synaptic strength, in which the topology of the cells58 and the presence or absence of coincident electrical activity in presynaptic and postsynaptic neurons play a role.39,40 In terms of the organization of motor control, selection occurs at the level of motor strategies and at the level of temporal and quantitative tuning of motor output.36 Recent neurophysiological data indicated that the basal ganglia might play a major role in the selection of motor strategies (ie, in motor sequence learning).32,41 whereas the cerebellum might be the key structure involved in the selection of situation-specific temporal and quantitative parameters of motor output (ie, accurate motor adaptation).42,43 The idea that frontostriatal circuitries play a role in the selection of motor strategies is supported by a recent birth cohort study by Murray et al.44 The study indicated that an earlier development of the ability to stand independently—which might be interpreted as an earlier ability to select an appropriate strategy to keep balance in upright stance—was associated with better executive functions in adulthood. The association was specific for executive functions, which are subserved by frontostriatal circuitries; other cognitive functions such as verbal and visual learning, which are more closely related to temporal cortex function, were not related to an earlier development of standing.

The transition from primary variability to secondary variability occurs at function-specific ages. For instance, in the development of sucking behavior, the phase of secondary variability starts prior to term age45; in the development of postural adjustment, it emerges after the age of 3 months46,47; and in the development of foot placement during walking, it starts between 12 and 18 months.48 The age at which adaptive behavior first can be observed depends on the method of investigation. For instance, with the application of electromyographic recordings, the first signs of adaption in postural behavior during sitting may be observed at the age of 4 months,46 but when simple behavioral observation is used, signs of adaptive sitting behavior are first detected from 6 months onward.49 Around the age of 18 months, all basic motor functions, such as sucking, reaching, grasping, postural control, and locomotion, have reached the first stages of secondary variability. Due to the ingenious interaction between self-produced motor activities with trial-and-error learning and the long-lasting developmental processes in the brain, such as dendritic refinement, myelination, and extensive synapse rearrangement,5 which furnish new neuromotor possibilities, it takes until late adolescence before the secondary neural repertoire has obtained its adult configuration. In other words, the basic, variable motor repertoire that is formed during the phase of primary variability continues to develop during the phase of secondary variability and to change throughout life.

The ongoing developmental changes in the nervous system, which are based on a never-ending interaction between experience and genetic information, allow for increasingly precise and complex motor skills, which may be regarded as refinements of the basic, variable repertoire. As a result, adult human beings are equipped with a variable movement repertoire with an efficient motor solution for each specific situation.

NGST and Atypical Motor Development

Atypical motor development may originate from genetic aberrations or adversities occurring during early development. Both etiological
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pathways may result in a structural anomaly or lesion of the developing brain or in a different setting of specific neurotransmitter systems, such as the monoaminergic systems. Accumulating evidence indicates that stressful situations in the prenatal and perinatal periods may induce lifetime changes in dopaminergic, serotonergic, or noradrenergic circuitries. The monoaminergic systems are widespread systems involved in the modulation of behavior. The 2 sequela—the lesion of the brain and the different setting of the monoaminergic systems—will be discussed as different entities, but it should be kept in mind that lesions of the immature brain often are associated with changes in specific neurotransmitter systems.

The best example of atypical motor development due to an early lesion of the brain is the motor development of children with cerebral palsy (CP). Other examples are some children with developmental coordination disorder and some children with attention deficit hyperactivity disorder. It is important to note, however, that in general developmental coordination disorder and attention deficit hyperactivity disorder cannot be attributed to a lesion of the brain. In the following paragraphs, CP is used as a prototype to describe the motor sequela of a lesion occurring in the fetal or infant brain.

NGST and an Early Lesion of the Brain

According to NGST, an early lesion of the brain has 2 major consequences. First, the repertoire of motor strategies is reduced. This reduced repertoire results in less variability, children with an early lesion of the brain have problems with selection of the most appropriately adapted strategy out of the repertoire. In other words, they have a limited capacity to vary motor behavior in relation to the specifics of the situation (ie, they have a limited variability).

The deficient capacity to select has a dual origin: it is related to deficits in the processing of sensory information that are virtually always present in children with an early lesion of the brain and to the fact that the best solution may not be available due to repertoire reduction. The difficulties in selection have 2 practical consequences. First, impaired selection may give rise to the paradoxical finding that results of motor tests of children with CP often are more variable than those of children with typical development. The variable test results are brought about by prolonged periods of trial and error needed to explore the reduced repertoire due to impaired selection. This consequence means that a reduced repertoire may be associated with more variable motor output. Second, impaired selection induces the need of ten- to hundredfold more active motor experience than typically needed to find the best strategy. Consequently, children with CP need considerably more practice than their peers without CP to learn a specific motor task.

Recall that exploratory drive is a fundamental feature of the typically developing nervous system. As a result, young infants spontaneously generate a wealth of everyday motor practice. The child with CP needs much more practice. In addition, the brain lesion responsible for CP may be associated with reduced exploratory drive. This reduced exploratory drive creates a challenging situation for the child with CP and his or her environment. The need for much practice requires strong motivation, which is obtained most easily when tasks to be learned have functional significance or are enjoyable.

NGST and an Altered Setting of Monoaminergic Systems

Adversities prior to term age, such as low-risk preterm birth, intrauterine growth retardation, or psychological stress of the pregnant woman, may give rise to a different setting of the monoaminergic systems in the absence of a lesion of the brain. Most of our knowledge about the effect of stressful conditions during early life on the developing brain is based on animal data. The animal data indicate that stress during early life gives rise to changes in serotonergic and noradrenergic activity in the cerebral cortex and alterations in dopaminergic activity in the striatum and prefrontal cortex. These changes have been associated with impaired development of the maps of body representation in the primary somatosensory cortex, inappropriately developed ocular dominance columns in the visual cortex, and mild motor problems. In terms of NGST, this impaired development may reflect a situation in which the child has a typical movement repertoire but has difficulties in selecting the best strategy in a specific situation due to the deficits in processing of sensory information. In other words, the child has an impaired ability to vary motor behavior—an impaired variability—in relation to
task-specific requirements. As a result, the child often exhibits more variable behavior during motor tests and needs more practice—and thus more time—to learn new motor skills. Indeed, such mechanisms appear to play a role in the frequently encountered impaired motor development of preterm children without cerebral palsy.79,75,76

**Early Phases of Motor Development**

**Typical Motor Development**

Non–goal-directed motility. A recent, detailed ultrasound study on the emergence of fetal motility revealed that the earliest movements can be observed at the age of 7 weeks 2 days PMA.77 The first movements are slow, small, sideways bending movements of head or trunk. A few days later, these simple movements develop into movements in which 1 or 2 arms or legs also participate, but the movements continue to be slow, small, simple, and stereotyped. At the age of 9 to 10 weeks PMA, general movements (GMs) emerge (ie, movements in which all parts of the body participate). Initially, GMs show little variation in movement direction, amplitude, and speed. After a few days, however, the majority of GMs show a substantial degree of variation in speed, amplitude, participating body parts, and movement direction. Interestingly, the emergence of GMs with movement variation and complexity at 9 to 10 weeks PMA coincides with the emergence of synaptic activity in the cortical subplate.78 This coincidence and the finding that the evolution and transient nature of the subplate match that of GM development inspired the hypothesis that variable and complex GMs result from activity of the subplate modulating the basic activity of GM networks in the spinal cord and brain stem.79

Soon after the emergence of the first movements, other movements are added to the fetal repertoire, such as isolated arm and leg movements, startles, various movements of the head (rotations, anteflexion, and retroflexion), stretches, periodic breathing movements, and sucking and swallowing movements.80 The age at which the various movements develop shows considerable inter-individual variation, but at about 16 weeks PMA, all fetuses exhibit the entire fetal repertoire. The repertoire continues to be present throughout gestation.

At birth, be it term or preterm, only minor changes in the motor repertoire occur. Breathing movements become continuous instead of periodic, the Moro reaction can be elicited for the first time, and the infant, who is now hampered by the forces of gravity, is no longer able to anteflex the head in a supine position.81 General movements continue to be the most frequently observed motor pattern.

Between 2 and 4 months postterm, infant behavior changes drastically. The infant is able to use smiles and pleasure vocalizations in social interaction, the head can be stabilized on the trunk, and a steady visual fixation and brisk visual orienting reactions have been developed.81 Simultaneously, GM activity is about to disappear and to be replaced gradually by goal-directed activity of arms and legs. Interestingly, the final phase of GMs, which occurs at 2 to 4 months postterm, is characterized by a specific movement property: the “fidgety” nature of GMs. The fidgety character denotes the presence of a continuous stream of tiny, elegant movements occurring irregularly all over the body.58 Functional neuroimaging studies suggest that increasing activity in the basal ganglia, the cerebellum, and the parietal, temporal, and occipital cortices plays a prominent role in the behavioral transition at 2 to 4 months.82

**Goal-directed motility.** The development of goal-directed behavior during infancy is characterized by intraindividual and interindividual variation.55,57,83,84 The variation occurs, for instance, as variation in the emergence of a function, variation in the performance of a function (Figs. 1 and 2), variation in the duration of specific developmental phases, and variation in the disappearance of infantile reactions, such as the Moro reaction. The variation in development includes the co-occurrence of different developmental phases. For instance, infants of a certain age can alternate belly crawling with crawling on hands and knees.85,86 Infants with typical development also may exhibit a temporary regression—an “inconsistency”—in the development of a specific function.85 As long as the regression is restricted to a single function, it can be regarded as another expression of developmental variation. Large variation in the attainment of milestones in goal-directed motor behavior (Fig. 3) implies that the assessment of milestones has less clinical value than previously was thought.86 Slow development of a single function usually has no clinical significance, but the finding of a general delay is clinically relevant.

Infancy is the period of transition from primary variability to secondary variability (ie, from motor behavior that cannot be adapted to task-specific conditions to adaptive motor behavior). This transition occurs at function-specific ages. A recent observational study indicated that the transition in sitting behavior occurs between 6 and 10 months, that in abdominal progression occurs between 8 and 15 months, that in reaching movements occurs between 6 and 12 months, and that in grasping occurs between 15 and 18
months. These findings mean that after the transition, people observing the infants could notice a change in their motor behavior.

A major accomplishment during infancy is the development of postural control, resulting in the ability to stand and walk without support. Postural control is aimed primarily at the maintenance of a vertical posture of head and trunk against the forces of gravity because a vertical orientation of the proximal parts of the body provides an optimal condition for vision- and goal-directed motility. In the control of posture, 2 functional levels can be distinguished. The basic level of control deals with the directional specificity of the adjustments: when the body sways forward, primarily the dorsal muscles are recruited; when the body sways backward, primarily the ventral muscles are activated. A study by Hedberg et al indicated that 1-month-old infants have direction-specific adjustments, which suggests that the basic level of postural control has an innate origin. Young infants show a variable repertoire of direction-specific adjustments from which, from the age of 4 months onward, they learn to select by means of active trial and error the adjustment that fits the situation best.

Meanwhile, the infant learns to sit independently. With increasing age, the means to adapt postural activity become increasingly refined. A major developmental change is the emergence of anticipatory postural activity between 12 to 14 months, an ability that strongly promotes the development of independent walking.

Successful reaching is preceded by various forms of prereaching activity. For instance, Von Hofsten demonstrated that newborn infants move their hands closer to a nearby object when they visually fixate on it, than when they do not pay visual attention to the object. Reaching
results in actual grasping of an object from about 4 months onward. At this age, reaching movements have an irregular and fragmented trajectory consisting of multiple movement units. These characteristics underline the probing nature of early reaches and the heavy reliance of the first reaching movements on feedback control mechanisms. During the following months, the reaching movement becomes increasingly fluent and straight, and the orientation of the hand becomes increasingly adapted to the object. After their first birthday, infants increasingly use the pincer grasp to pick up tiny objects. This change in behavior implies that corticomotoneuronal pathways are being involved increasingly in fine motor control.

Figure 2.
Variation in motor behavior in a sitting position at 11 months postterm. The figure consists of frames selected from a videorecording of about 3 minutes. Figure produced with permission of the parents.
At birth, the infant—like the fetus—shows locomotor-like behavior in the form of neonatal stepping movements. These movements probably are generated by spinal pattern generators analogous to the locomotion in the hind limbs of kittens after a transection of the thoracic cord and the locomotor-like activity in people with a spinal cord injury. The infant stepping movements are rather primitive in character and differ from the flexible plantigrade gait of adulthood. This non-goal-directed neonatal stepping is characterized by a lack of segment-specific movements, implying that the legs tend to flex and extend as a single unit; by the absence of a heel-strike; by a variable muscle activation with a high degree of antagonistic coactivation; and by short-latency bursts of electromyographic activity at the foot contact due to segmental reflex activity. In the absence of specific training, the stepping movements can no longer be elicited after the age of 2 to 3 months.

A period of locomotor silence follows, which is succeeded in the third quarter of the first postnatal year by goal-directed progression in the form of crawling and supported locomotion. When neonatal stepping is trained daily, the stepping response can be elicited until it is replaced by supported locomotion. This progression is perhaps not so surprising in light of the fact that the locomotor pattern of supported locomotion is reminiscent of that of neonatal stepping—both lacking the determinants of plantigrade gait. In addition, the milestone transition into independent walking is not associated with a major change in specific locomotor activity. This finding indicates that the emergence of independent locomotion is not primarily induced by changes in the locomotor networks. Presumably, the development of independent walking is largely dependent on the development of postural control, which, in turn, is dependent in particular on developmental changes in the subcortical-cortical circuitry.

Motor development beyond infancy is characterized by a gradual increase in agility, adaptability, and the ability to make complex movement sequences. It is the phase of secondary variability, during which maturational processes in continuous interaction with changing body proportions and experience produce highly adaptive secondary neuronal repertoires. The creation of secondary repertoires is associated with extensive synapse rearrangement, which is the net result of synapse formation and synapse elimination. It is facilitated by increasingly shorter processing times, which can be attributed, in part, to ongoing myelination.

**Atypical Motor Development**

Developmental changes in the young brain have a large impact on the expression of atypical motor behavior. It may happen that a lesion of the developing brain results in neuromotor dysfunction in infancy but is followed by a typical developmental outcome. The reverse may also occur (ie, an apparently typical development in the early phases of infancy may be followed by the development of CP). In infancy, atypical motor development may be expressed by a delay in the achievement of milestones (which may be related to impaired selection), by mild or major deviations in muscle tone (velocity-dependent resistance to stretch), by a persistence of infantile reactions (eg, the Moro reaction), and by a reduced variation in motor behavior. The latter sign may be the most specific expression of an early lesion of the brain, whereas the other signs may be the result of a lesion of the brain but also may be related to other types of adversities during early development, such as low-risk preterm birth. Reduced variation in motor behavior is well expressed in the quality of GMs: abnormal GMs are characterized by limited variation and limited complexity. Interestingly, definitely abnormal GMs are related to white matter pathology and not to abnormalities of the brain’s gray matter.
when GMs have disappeared, atypical motor development is characterized by reduced variation (Figs. 4 and 5). Well-known stereotypies are fisting of the hands, extension of the legs, clawing of the toes, dominant asymmetrical tonic neck reflex posturing, hyperextension of the neck and trunk, or stereotyped asymmetries.\textsuperscript{106} It has been postulated that the degree to which movement variation is reduced may reflect the extent to which cortical connectivity is impaired.\textsuperscript{79,107}

Atypical motor development is associated with postural dysfunction. Children with a severe lesion of the brain who develop severe bilateral spastic CP or severe athetosis and function at Gross Motor Function Classification System level V\textsuperscript{108} presumably lack the basic level of postural control.\textsuperscript{109} In infants with less-severe forms of CP, the basic level of postural organization is more or less intact. However, their postural development is hampered—and, therefore, delayed—by a limited repertoire of postural adjustments and a deficient capacity to adapt posture to the specifics of the situation.\textsuperscript{109}

Diagnostic Application of Variation and Variability
Gradually, European clinicians working in the field of developmental neurology realized that variation and variability may assist in the evaluation of motor development. However, before addressing the value of these parameters, some general reflections on the significance of the infant neuromotor assessment are necessary. Evaluation of neuromotor function in early life has 2 goals. First and foremost, it aims at assessing the infant’s current capacities and limitations, as the assessment offers

Figure 4.
Reduced variation in motor behavior in a supine position at 2 months postterm. The figure consists of frames selected from a videorecording of about 3 minutes. Figure produced with permission of the parents.
the basis for therapeutic guidance. Second, an assessment at an early age may assist in the prediction of the infant's developmental prospects. However, as indicated in the preceding paragraphs, the developmental characteristics of the brain preclude precise prediction. Prediction of developmental outcome is best when multiple sources of information are used, such as the infant's history, the results of neuroimaging, and neurophysiological assessments in combination with a neurological, developmental, and neuromotor assessment. Prediction also is largely facilitated when longitudinal series of assessments are used.

The various instruments available to evaluate the infant's neuromotor and...
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developmental status have specific aims, advantages, and disadvantages. Most instruments provide information on the child’s function in terms of age-adequate performance versus underachievement or delay. Examples are the Bayley Scales of Infant Development and the Albert Infant Motor Scales. Neurological assessments pair information on motor performance with specific details on sensorimotor function in terms of muscle tone and reflexes. The growing awareness that the quality of motor behavior may assist in the evaluation of the child’s neuromotor condition inspired the development of 3 new assessment methods; the Test of Infant Motor Performance (TIMP), the GM method, and the Infant Motor Profile (IMP). The TIMP and the GM method are applicable till the age of 4 months postterm, whereas the IMP is designed for infants aged 3 to 18 months postterm. In contrast to the other 2 instruments, the TIMP does not use variation or variability as explicit parameters of movement quality. The TIMP has good reliability, and the limited data available suggest that it is a valuable instrument in the prediction of CP. The 2 methods using the concepts of variation and variability are discussed below. Variation (ie, the evaluation of the size of the repertoire) is a parameter that may be applied in the phase of primary and secondary variability. Variability (ie, the ability to make an adaptive selection) is particularly relevant from the emergence of secondary variability onward.

The examination of the quality of GMs is a reliable assessment based on the evaluation of movement variation, and ability to select (variability). The other 3 domains evaluate more traditional aspects of motor behavior: symmetry, fluency, and performance. The reliability and construct validity of the IMP are good, and its predictive and evaluative power is promising.

Concluding Remarks
Accumulating evidence indicates that abundance in cerebral connectivity is the neural basis of human behavioral variability (ie, the ability to select from a large repertoire of behavioral solutions the one most appropriate for a specific situation). Indeed, typical human motor development is characterized by variation and the development of adaptive variability, and atypical motor development is characterized by limitations in variation and variability. Limitations in variation are based on structural anomalies, in which disturbances of cortical connectivity may play a prominent role, whereas limitations in variability are present in virtually all children with atypical motor development. In infants with reduced variation, early intervention may aim to enlarge the limited movement repertoire, but animal data indicate that this aim is difficult to achieve. This situation implies that—despite intervention—repertoire reduction most likely will remain a feature of the motor behavior of the child with an early lesion of the brain. In such a situation, equipment may offer a proper means to facilitate functional activity and participation.

The limited variability of children with atypical motor development is based on the limited ability to select a strategy out of the movement repertoire due to deficiencies in the processing of sensory information brought about by self-produced actions. This fact suggests that children with limited variability may profit from ample, variable, self-produced, trial-and-error activities.
References


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57 Touwen BC. How normal is variable, or now variable is normal? *Early Hum Dev*. 1993;34:1–12.


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