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Evaluation of Motor Function in Young Infants by Means of the Assessment of General Movements: A Review

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Purpose: Optimal management of children with developmental disorders, such as cerebral palsy (CP), requires detection at an early age. The purpose of this paper is to review the predictive value of various forms of traditional neonatal neurological examination and that of a new form of neuromotor assessment of young infants, based on the assessment of the quality of general movements (GMs). **Summary of Key Points:** The technique of GM assessment is presented and the features of normal, mildly abnormal and definitely abnormal GMs discussed. Essential to GM assessment is the Gestalt evaluation of movement complexity and variation. The quality of GMs at two to four months postterm has been found to have the highest predictive value. The presence of definitely abnormal GMs at this age, ie, GMs devoid of complexity and variation, puts a child at very high risk for CP. **Conclusions:** This implies that definitely abnormal GMs at two to four months are an indication for early physical therapy intervention. **(Pediatr Phys Ther 2001;13:27–36)** *Key words: infant, movement, motor activity, physiology, prognosis*

INTRODUCTION

Because of improvements in obstetrical and neonatal care and an associated decrease in perinatal mortality, the number of infants who are at high risk for developmental problems is gradually increasing.[1,2] Yet, the ability to predict at early age which infant actually will develop cerebral palsy (CP), clumsiness, attention deficit hyperactivity disorder (ADHD), and/or a learning problem is rather limited. Part of the difficulty in predicting developmental outcome in early infancy can be attributed to the characteristics of the developing nervous system. The continuous developmental changes of the brain during infancy and childhood can lead to a disappearance of signs of dysfunction present at an early age. Also the reverse can occur. Children can be free from signs of dysfunction at early age, but begin to demonstrate a functional deficit with increasing age due to the age-related increase in complexity of neural functions.[3,4]

The difficulty in predicting outcome in young infants is reflected by the diverse techniques available to assess the brain at an early age. The techniques vary from clinical bedside methods requiring no equipment, such as the various forms of neurological examinations, to more or less sophisticated technical procedures, such as brain imaging (ultrasound, magnetic resonance imaging, and computer tomography) and neurophysiological tests, including electroencephalogram (EEG) recordings and visual or somatosensory-evoked potentials. The sensitivities, specificities, and capacity of these examinations and tests to predict developmental outcome are quite variable (for a review, see Ref. 5). The heterogeneity in predictive validity points to the need for advanced and more accurately described methods.

The aim of this report is to review the reliability and validity of a new neuromotor assessment of young infants, ie, the assessment of the quality of general movements (GMs). The description and discussion of GM assessment is preceded by a short survey of the validity of other neuromotor assessment techniques available for the evaluation of young infants.

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MEASURES OF PREDICTIVE VALUE

In general, the capacity of infant tests to predict neurodevelopmental disorders is expressed in the test's sensitivity, specificity and positive and negative predictive values[6] (Table 1). Sensitivity can be defined as the capacity of a test to correctly identify those who actually do have the disorder, and specificity as the capacity of the test to correctly identify those who do not have the disorder. High sensitivity results in few false-negatives, high specificity in few false positives. The positive predictive value of a test is defined as the proportion of true positives among all those who have positive results. The negative predictive value is the proportion of true negatives among all those who have negative test results.

The predictive values of a test depend on the at-risk criteria of the test. For instance, a stringent cutoff score within an infant test, that results in only infants with the most serious abnormalities being classified at risk, is associated with high specificity and low sensitivity values. Less stringent cutoff scores result in lower specificity and higher sensitivity values.[7] Predictive values also depend on the type of disorder evaluated and the age at which the follow-up evaluation is carried out. Predictive values for serious motor impairments, such as CP, are generally better than those for minor developmental disorders (see Table 2). The age of evaluation is important because children can grow into and out of a neurological deficit.[3,4]

PREDICTIVE VALUE OF EXISTING NEUROMOTOR ASSESSMENT TECHNIQUES

The first neurological examination techniques for young infants were developed in the middle of the twentieth century.[8,9] In line with neurological thinking at that time, the foci of these neurological assessments were muscle tone regulation and postural reflexes. The first examination techniques formed the basis for other, more standardized forms of neurological examination of young infants, such as the techniques developed by Saint-Anne Dargassies,[10] Prechtl,[11] Amiel-Tison and Grenier,[12] and Dubowitz and co-workers.[13,14]

Neonatal neurological assessment techniques are widely used. Little information has been provided on the reliability of these tests, but the information available suggests that reliability is fairly good.[15] Few studies furnish data on the validity of neonatal neurological assessments. Research reports that provided information permitting the calculation of sensitivity, specificity and positive and negative predictive values of neonatal neurological findings are presented in Table 2. These show considerable variation in the capacity to predict CP: sensitivity values vary from 0% to 100% and specificity values from 59% to 96%. The capacity of the two neonatal assessments techniques according to Prechtl[11] and Amiel-Tison and Grenier[12] does not differ. The capacity to predict minor neurological dysfunction is somewhat less than the capacity to predict CP: sensitivity values vary between 51% and 79% and specificity values between 54% and 80%. The data in Table 2 illustrate that sensitivity and specificity are inversely related.

TABLE 1.
Calculation of Measures of Predictive Value

	Disorder at Follow-up	
Infant Test	Present	Absent
At risk	a	b
Not at risk	c	d

Sensitivity = a/(a + c); specificity = d/(b + d); positive predictive value (PPV) = a/(a + b); negative predictive value (NPV) = d/(c + d).

In addition to the various forms of neurological examination, evaluations of infant motor behavior have been developed such as the Movement Assessment of Infants (MAI)[25] and the Alberta Infant Motor Scales (AIMS).[26] The MAI provides a detailed and systematic appraisal of motor behaviors that occur during the first year of life by assessing behavior in four areas: tone, primitive reflexes, automatic reactions, and volitional movement. The interrater and test-retest reliabilities of the MAI risk scores vary, but are generally satisfactory.[27–29] Predictive validity of the MAI has been reported especially for the MAI assessment at the age of four months (Table 3). The sensitivity of MAI risk scores of more than nine in predicting clear developmental disorders is about 70% and the accompanying specificity is about 90%. At this relatively high at-risk level, with at least 10 at-risk items present, a substantial number of children who develop minor disorders is not detected. The detection of the latter children could be improved by applying a less stringent cutoff score in the MAI. By doing so, the capacity to predict both major and minor developmental disorders increases, but is lower than the capacity to predict only major disorders. The sensitivity values of MAI risk scores of more than four in predicting major plus minor developmental disorders lie around 60%, with associated specificity values being about 80% (Table 3).

The AIMS is a norm-referenced measure of infant gross motor development. Interrater and test-retest reliabilities are excellent.[26] Little is known about the predictive validity of AIMS in early infancy. To date, one study reported that the prediction of AIMS at four months was best with the cutoff score set at the 10th centile. This resulted in sensitivity for major developmental disorders of 77% and a specificity of 82%; the sensitivity and specificity values for prediction of both major and minor developmental problems were 58% and 83%, respectively.[7]

ASSESSMENT OF GENERAL MOVEMENTS

Prechtl,[34–36] a pioneer in the field of early neurological development, studied motor activity in the human fetus and newborn infant over many years. He learned to appreciate the significance of spontaneous motor behavior in early life. Prechtl and others realized that self-generated motility during early development plays an important role in survival and adaptation.[28,29] In addition, Prechtl discovered that the quality of spontaneous motility, especially the quality of GMs, reflects the condition of the nervous system of the fetus and young infant.

TABLE 2.

Predictive Validity of the Neonatal Neurological Examination in Infants Born Preterm and Full Term

Author(s)	Study group (*n*)	Neonatal Assessment*	Age (yr)	Outcome Type†	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Preterm								
Stewart et al[16] 1988	111	A-T A vs N	1	Devel disab	80	67	35	94
Allen and Capute[17] 1989	210	Mainly A-T A vs N	1–5	CP	80	69	38	94
Lanzi et al[18] 1990	71	A-T A vs N	2	CP	100	60	38	100
Den Ouden et al[19] 1990	859	Method? A+S vs N	2	Devel disab	21	96	50	86
Cioni et al[20] 1997	60	Pr. A vs N	2	CP	79	71	72	78
Weisglas-Kuperus et al[21] 1992	79	Pr. A vs S+N	3 ½	CP	89	89	50	98
		Pr. A+S vs N		MND	75	62	44	86
Hadders-Algra et al[22] 1988	80	Pr. A vs S+N	6	CP	0	85	0	91
		Pr. A+S vs N		MND	58	54	38	73
Full term								
Cioni et al[23] 1997	58	Pr. A vs N	2	CP	88	59	?	?
Hadders-Algra et al[22] 1988	372	Pr. A vs S+N	6	CP	50	95	15	99
		Pr. A+S vs N		MND	51	78	36	87
Hadders-Algra et al[24] 1988	747	Pr. A vs S+N	9	CP	67	82	10	99
		Pr. A+S vs N		MND-2	79	80	13	95

* Neonatal assessment: A-T = according to Amiel-Tison and Grenier; Pr = according to Prechtl; A vs N = neonatal findings dichotomized as abnormal vs normal; A+S vs N = neonatal findings dichotomized as abnormal plus suspect vs normal.

† CP = cerebral palsy; Devel disab = developmental disability; MND = minor neurological dysfunction; MND-2 = more serious form of MND.

TABLE 3.

Predictive Validity of MAI in Groups of Infants Considered High Risk and Born Preterm at the Corrected Age of Four Months

Author(s)	Study Group (*n*)	MAI Cutoff*	Outcome		Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
			Age (yr)	Type				
Paban and Piper[30] 1987	27	>7	1	Susp + CP	67	35	35	35
		>12		Susp + CP	22	71	29	67
Piper et al[31] 1992	75	>4	1 ½	Susp + CP	61	83	61	83
		>9		CP	67	94	60	95
Swanson et al[32] 1992	160	>9	1 ½	Susp + Abn	70	72	?	?
Darrah et al[7] 1998	164	>4	1 ½	Abn	82	75	33	96
		>9	1 ½	Abn	73	93	58	96
		>4	1 ½	Susp + Abn	64	76	43	88
		>9	1 ½	Susp + Abn	50	94	69	87

* MAI cutoff: risk scores above the cutoff level denote an at-risk score.

Abn = clear developmental disorder, including CP; Susp = suspect for developmental disorder.

Normal Development of General Movements

According to Prechtl[35] normal GMs are: gross movements involving the whole body. They may last from a few seconds to a minute. What is particular about them is the variable sequence of arm, leg, neck and trunk movements. They wax and wane in intensity, force and speed, and their onset and end are gradual. The majority of extension or flexion movements of the arms and legs is complex, with superimposed rotations and often slight changes in direction of the movements.

GMs emerge at seven to eight weeks postmenstrual age (PMA) and remain the most frequently observed movement pattern during fetal life.[37] After birth, GMs continue to be common until the age of three to four months postterm, when they gradually are replaced by goal-directed motor behavior.[38,39]

Three phases can be distinguished during normal GM development (Table 4). Before 36 to 38 weeks PMA, GMs are characterized by abundant variation. At 36 to 38 weeks, the very variable "preterm" GMs change into the forceful "writhing" GMs. Notably this transition occurs at the very same age at which fully established behavioral states develop.[43] A second transition in the form of GMs takes place at the age of six to eight weeks postterm. At this age, the writhing characteristic of the GMs disappears and is replaced by a continuous stream of tiny elegant movements, a charming dance of fidgety GMs. The change of writhing GMs into fidgety GMs is

TABLE 4.
Age-Specific Characteristics of GMs Considered Normal[38–42]

GM Type	Period of Presence (in wks PMA)	Description
Preterm	Before 36–38	Extremely variable movements, including many trunk movements.
Writhing*	From 36–38 Until 54–58	Movements with a rather forceful (writhing) aspect. In comparison with preterm GMs writhing, GMs seem to be somewhat slower and to show less participation of the trunk.
Fidgety*	From 46–52 Until 54–58	Basic motility consists of a continuous flow of small and elegant movements occurring irregularly all over the body, ie, head, trunk and limbs participate to a similar extent. The small movements can be superimposed by large and fast movements.

* Note: writhing and fidgety are also words used to describe pathological movements. Here the words denote age-specific details of GMs considered normal. At any GM age, the basic characteristics of GMs considered normal are 1) participation of all body parts and 2) movement complexity and variation.

more strongly related to postmenstrual age than to postnatal age, suggesting that the developmental changes in the form of normal GMs are mainly based on endogenous maturational processes, leaving only a minor role for postnatal experience.[39] The minor contribution of postnatal experience is exemplified by the fact that infants born preterm and at low-risk for developmental disorders develop fidgety GMs about one week earlier than do infants who are healthy and full-term.[44]

Little is known about the neural mechanisms underlying the changes in GM form. Possibilities include:[40] maturational changes in the properties of motoneurones,[45] regression of polyneuronal muscle innervation,[46] increasing participation of Renshaw inhibition, and between two and four months when fidgety GMs are present, decreasing excitability of motoneurons due to intra- and supraspinal reorganization.[47]

Abnormal General Movements

Electromyography (EMG) shows that GMs can be divided into three groups: normal movements, mildly abnormal GMs and definitely abnormal GMs (Table 5; Fig. 1). Normal GMs at any age are characterized by variation, complexity, and fluency.[35,42] The variation of normal GMs is expressed in muscle coordination patterns that underlie movement patterns. Muscle coordination is characterized by variation in the muscles that participate and in the timing and the quantity of muscle activation[40–42] (Fig. 2). Despite this variation, muscle activity is not random. For example, normal GMs show a pattern of antagonistic coactivation during 70% to 85% of movement time. Mildly abnormal GMs lack fluency, but show some movement complexity and variation. The lack of fluency can be expressed in two ways: movements can be jerky and abrupt, or stiff and cramped. Both expressions of the lack of fluency can be present in EMG recordings of a single GM. The EMG recordings of mildly abnormal GMs are relatively variable, but exhibit abnormalities in the temporal and quantitative scaling of phasic muscle activity (Fig. 2).

Definitely abnormal GMs lack fluency, complexity, and variation. This is reflected by the absence of variation in muscle coordination: the patterns consist either of a stereotyped synchronous activation of most participating muscles, or a stereotyped pattern of reciprocal activity[42] (Fig. 2). Thus, movement fluency is a feature that is easily disturbed. Minor dysfunctions give rise to movements that lack fluency. This also holds true for movements of adults,

TABLE 5.
Definitions of the Basic Classification of GM Quality

Parameter	GMs Considered: Normal	Mildly Abnormal	Definitely Abnormal
GM complexity = spatial variation			
The infant actively produces frequent changes in movement direction of the participating body parts. The changes in movement direction are brought about by continuously varying combinations of flexion-extension, abduction-adduction and endorotation-exorotation of the participating joints.	++	+	−
GM variation = temporal variation			
Across time, the infant produces continuously new movement patterns, ie, the infant has an apparently infinite movement repertoire.	++	+	−
GM fluency			
Presence of smooth, supple, and graceful movements. Fluency in particular points to the velocity profile of the movements, which is characterized by gradual accelerations and decelerations.	+	−	−

Complexity and variation: − = absent; + = present to a limited extent; ++ = fully present.
Fluency: − = absent; + = present.

A

B

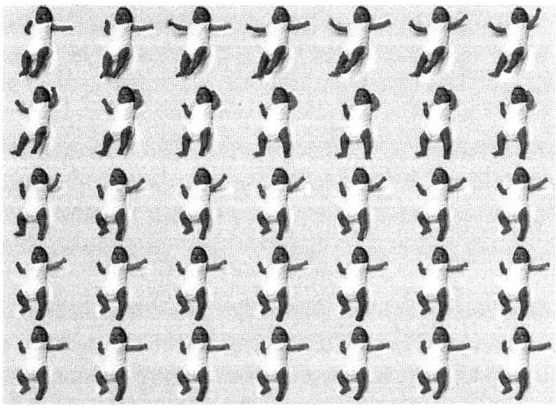

Fig. 1. Representation of video-frames with GMs of two infants each three months old. The video recordings start in the left hand upper corner and should be read as the lines in a book. The interval between the video frames is 0.24 seconds and the total duration of the displayed fragments is 8.16 seconds. The infant in panel A was born at term and shows normal fidgety GMs. The continuously varying positions of the limbs illustrate the rich spatial and temporal variation of normal movements. Movement complexity is exemplified by the movement of the left leg on the third row: the movement is not restricted to a simple flexion-extension movement, but the flexion-extension movement is combined with a simultaneously occurring abduction movement in the hip and endorotation movement of the foot. The infant in panel B was born at 28 weeks PMA. The infant shows definitely abnormal GMs. The abnormal character of the movement is reflected by the lack of temporal variation (the frames are almost identical, giving a false impression that the infant hardly moves) and the lack of movement complexity (arm and leg movements are simple and restricted to a single plane). (Video recordings were made in collaboration with the Department of Developmental and Experimental Clinical Psychology; figure used with permission of the parents and the *Nederlands Tijdschrift voor Geneeskunde*[48]).

which become shaky and tremulous with anger and fear, and sluggish with fever and flu.

Movement complexity and variation co-vary strongly with the degree of dysfunction of the nervous system. Movement complexity and variation, which can be regarded as two forms of variation (Table 5), are the primary parameters of GM assessment. Variation is a fundamental feature of the function of the healthy young nervous system and stereotypy a principle property of early brain dysfunction. The variable expression of GMs underscores the notion that variability in neural function is the hallmark of a healthy nervous system.[50–53]

The clinically relevant trichotomy of normal, mildly abnormal, and definitely abnormal GMs is somewhat artificial. The quality of movement is a continuum with splendidly complex, variable and fluent movements at one extreme and at the other extreme very stereotyped movements, such as cramped-synchronized movements (*cf* Ref. 54). The latter movements are characterized by an abrupt *en bloc* start and stop of stiff movements devoid of complexity and variation.[42,55,56]

GM Assessment: Appraisal of Movement Quality by Gestalt Perception

The technique of GM assessment is identical during fetal life, the preterm period and during the first months after term age. Crucial to the technique is the appraisal of the quality of spontaneous movements. Changes in movement quality, not changes in movement quantity, reliably reflect pathology of the brain.[55,57] Changes in movement quality are appreciated by means of Gestalt perception by the observer.[58] This global Gestalt perception is the result of the evaluation of the complexity and variation of GMs. The assessment of GMs is based on the evaluation of the repertoire of movement patterns displayed by all parts of the body and does not pay special attention to particular behavior of specific body parts (eg, fisting). GM assessment can be illustrated by reference to Fig. 1. Infant A is portrayed in numerous different postures, reflecting the fact that the limbs, head and trunk are moved in a continuous exploration of all possible combinations of joint configuration. The infant's motility shows variation in time and in space. Infant B, who moves just as much as infant A, produced only a few different postures. This means that Infant B's motility lacks temporal variation. In addition, this latter infant's movements are not complex. The limbs show relatively simple flexion, extension or elevation movements. The third aspect of GM assessment, movement fluency, cannot be illustrated by reference to Fig. 1. People seldom have difficulties, however, in discovering deviance in the fluency of movements. The visual system has an innate sensitivity to detect a loss of movement fluency. The visual propensity for detecting abnormalities in movement fluency, such as jerkiness, tremulousness and stiffness, interferes to some extent with the assessment of the major components of the GMs, ie, movement complexity and variation.

The assessment of movement quality is facilitated substantially by video recording.[35] Video recording creates the possibility of off-line evaluation. This, in turn, promotes focused attention of the observer, which is a prerequisite for the evaluation of movement complexity and variation. In addition, the video offers the opportunity of movement replay, both at normal and at high speed. A replay at high speed is especially helpful in the evaluation of movement

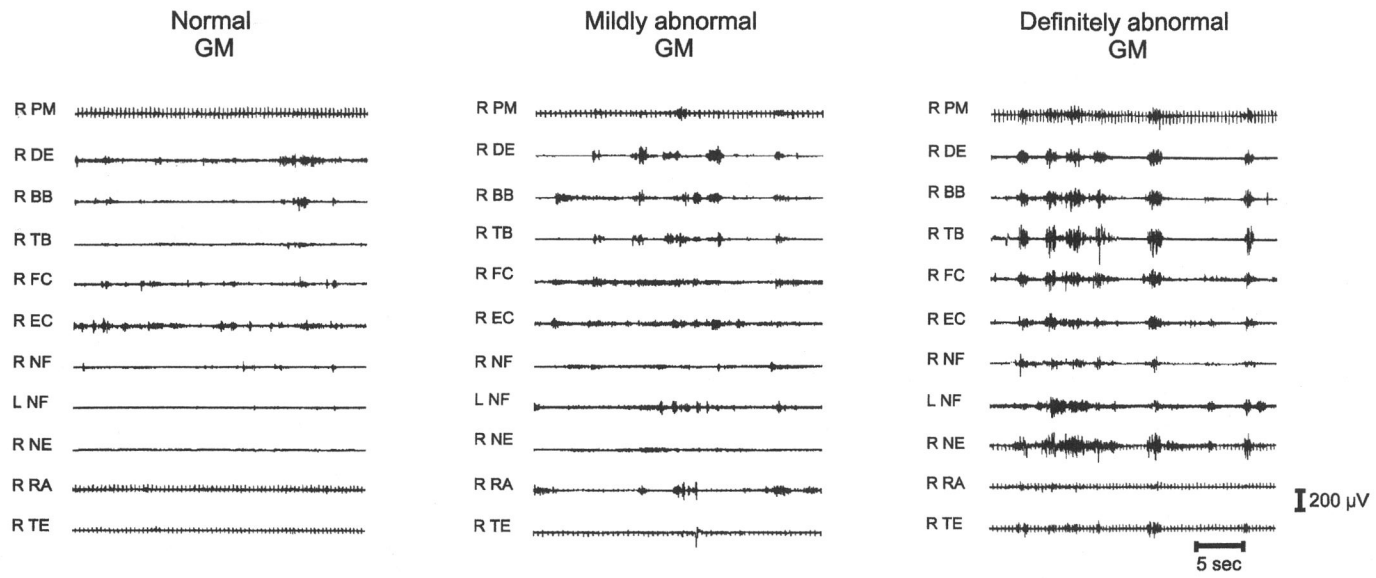

Fig. 2. EMG recordings of arm, neck, and trunk muscles during a normal, a mildly abnormal, and a definitely abnormal GM at three months of age. The normal EMG pattern shows the variable bursting pattern with small phasic bursts which is characteristic for normal fidgety GMs. The mildly abnormal pattern has conserved the variability in bursting, but the size (amplitude and duration) of the phasic bursts is disproportionally large. In the definitely abnormal pattern, the variation in bursting is absent totally: the majority of recorded muscles is activated synchronously. L = left; R = right; BB = m biceps brachii; DE = deltoid muscle; EC = mm extensor carpi; FC = mm flexor carpi; NE = neck extensor muscles; NF = neck flexor muscles; PM = m pectoralis major; RA = m rectus abdominis; TB = m triceps brachii; TE = thoracal extensor muscles.

complexity and variation. A high-speed replay produces an effect that is comparable to the effect produced by the video-frame sampling procedure of Fig. 1. An additional advantage of video recording is the possibility of data collection in the absence of the examiner.

GMs are affected by the behavioral state of the infant.[59] The optimal state for GM-analysis is active wakefulness, ie, Prechtl's state 4.[60] In this state the complexity and fluency of normal GMs are expressed best. During other behavioral states, normal GMs demonstrate features reminiscent of abnormality. During REM sleep, state 2, or during state 2-like conditions before the age of 36 to 38 weeks PMA,[43] normal GMs are in general short-lasting, occasionally jerky, and they sometimes have an abrupt and synchronous onset. REM sleep does not affect movement complexity and variation. Although GMs are assessed preferably in state 4, when a video recording only contains GMs during state 2, or state 2-like conditions, the primary parameters of GM analysis, complexity and variation, still can be evaluated.

In contrast, GMs during crying have a reduced complexity and variation. Moreover, GMs during crying are abrupt, jerky, and tremulous. GMs during crying should be excluded from the analysis. Although crying behavior can be stopped by a pacifier, nonnutritive sucking largely modifies the character of the GMs. Sucking induces a physiological motor stereotype during which small amplitude movements are made with the arms and hips in flexion and the knees in extension.[59,61] Thus, GMs accompanying nonnutritive sucking also should be excluded from analysis. The optimal conditions for the evaluation of GMs are listed in Table 6.

The basic principles of GM assessment can be learned in two days. Thereafter, it requires further practice of about 100 GM recordings to become a skilled observer. Various studies reported that the intra- and interobserver agreement of GM assessment of skilled observers is high with kappa values varying between 0.8 and 1.0,[42,49,62–64] implying an excellent interrater and test-retest reliability.

Validity of GM Assessment

Various pre-, peri- and neonatal adversities, such as maternal diabetes,[65] premature rupture of the membranes,[66] intrauterine growth retardation,[63,67–70] preterm birth,[54,55,63,71–73] perinatal asphyxia,[56,74] and neonatal hyperbilirubinemia,[75] can give rise to mildly and definitely abnormal GMs. Definitely abnormal GMs are specifically but not exclusively related to discernible lesions of the brain.[42,55,56,73] The latter is supported by the finding of strikingly stereotyped movements in anencephalic fetuses.[76] It has also been demonstrated that movement quality is not a fixed phenomenon. Quality can change in various ways: movement quality can be transiently affected by illness,[71,77] and movement abnormalities can vanish or become more distinct with increasing age. Recently, it was demonstrated that the changes in the quality of GMs mainly occur in the transitional periods when normal GMs change form, ie, between 36 and 38 weeks PMA and between six and eight weeks postterm.[49] During the three GM phases (Table 4), movement quality is relatively stable.

The predictive validity of GM quality varies with the age at which the GMs are evaluated and with the type of outcome being predicted (Table 7). The best prediction

TABLE 6.

Optimal Conditions for GM Assessment

Requirement	Age (in weeks PMA)	Conditions
1. Video recording	Until term age	Selection of three best GMs out of a 1-hour recording; best means spontaneously generated, supine position, state 4, longest duration.
	After term age	Recording of 5–10 minutes in state 4.
2. Behavioral state	Any age	Not during crying Not during nonnutritive sucking. Not during interaction with adult or toy.
	Before 36–38 weeks	Preferably analysis of GMs in state-4-like condition. In case infant sleeps: note state-like configuration and be aware that normal GMs in state-2-like conditions are occasionally abrupt or sometimes have a synchronized onset.
	After 36–38 weeks	Restrict analysis to GMs in state 4.
3. Position	Any age	Support surface: flat and moderately soft. Start in supine position. In case infant roles to side: return it into supine position; when infant persists in rolling to side, leave the infant in lateral position. Note: lateral position hampers evaluation of movement complexity and variation.
4. Clothes	Until term age	No clothes, with or without small diaper.
	After term age	In underwear; when infant does not tolerate undressing, start video recording with infant dressed.
5. Environment	Any age	Neutral temperature; avoid high levels of noise and very bright light.

TABLE 7.

Predictive Validity of GM Assessment

		GM-Classification*		Outcome		Sensitivity	Specificity	PPV	NPV
Author(s)	Study Groups	Type	Age	Age (yr)	Type	(%)	(%)	(%)	(%)
Ferrari et al[55] 1990	PT	N vs A	PT	1–2	Abn	100	59	70	100
	(n = 43)		FID			100	92	90	100
Prechtl et al[56] 1993	FT-asph	N vs A	WRI	2	Abn	100	46	65	100
	(n = 26)		FID			85	85	85	85
Bos et al[73] 1998	PT	N vs A	FID	2	Abn	57	95	80	86
	(n = 27)								
Prechtl et al[78] 1997	PT+FT-asph	N+MA vs DA	FID	2	CP	88	99	98	93
	(n=110)								
Geerdink and Hopkins[79] 1993	PT	N+MA vs DA	PT	1	Abn	50	92	60	88
	(n = 35)		WRI		Abn	86	85	60	96
			FID		Abn	43	96	75	87
		N vs MA+DA	FID		MND	86	79	60	94
Hadders-Algra and Groothuis[49] 1999	PT+FT	N+MA vs DA	PT	4–9	CP	25	77	25	77
	(n = 52)		WRI		CP	100	89	62	100
			FID		CP	88	100	100	98
		N vs MA	FID		MND	85	58	46	90
			FID		ADHD	79	57	46	85

* GM-classification: PT = at preterm GM phase, WRI = at writhing GM phase, FID = at fidgety GM phase. N vs A = GM data dichotomized as normal vs abnormal; N+MA vs DA = GM data dichotomized as normal + mildly abnormal vs definitely abnormal; N vs MA+DA = GM data dichotomized as normal vs mildly + definitely abnormal.

Abn = clear developmental disorder, including CP; ADHD = attention deficit hyperactivity disorder; FT = full term; FT-asph = full-term infants with asphyxia; MND = minor neurological dysfunction; PT = preterm.

can be obtained through a longitudinal series of GM assessments. Infants, who persistently show definitely abnormal GMs, even during the transformational phases at 36 to 38 weeks PMA and six to eight weeks postterm, have a high risk (70%–85%) for the development of CP.[55,56] Due to developmental changes in the quality of GMs, the prediction of a single GM assessment improves with increasing age. Thus, prediction is best at two to four months, the age of fidgety GMs. Definitely abnormal GMs at two to four months, which implies a total absence of fidgety movements, predict CP with an accuracy of 85% to 98%.[49,78] Ongoing studies suggest that infants with definitely abnormal GMs at two to four months who do not develop CP, usually show other developmental problems, such as minor neurologic dysfunction (MND), attention deficit hyperactivity disorder (ADHD), or cognitive problems. Mildly abnormal GMs at two to four months are related to the development of MND, ADHD, and aggressive behavior,[49] but the capacity to predict these minor problems is modest, due to the

presence of a relatively large number of false-positives, resulting in moderate specificity.

Significance and Prospects of GM Assessment

The assessment of the quality of GMs, involving a Gestalt evaluation of the spatial and temporal variation of spontaneous motility, is a sensitive tool proposed to evaluate brain function in young infants. It is a tool complementary to the traditional neurological examination. The combination of GM assessment and neurological examination allows for early detection of virtually all infants with CP, whereas an assessment limited to a neurological examination occasionally misses an infant with CP.[20,23,42,49]

The quality of GMs at two to four months has substantial predictive value. European experience has taught us that GM assessment at two to four months can be integrated easily into clinical practice, as it only requires a video recording of spontaneous motor behavior for about five minutes (Table 6) and another five minutes of video analysis.

The presence of definitely abnormal GMs at fidgety age puts a child at such a high risk for CP that it warrants physical therapy intervention. It is unlikely that the intervention will prevent the development of CP, but animal data[80,81] suggest that early intervention could improve later functional abilities. Of course, this is an issue begging for further exploration and research, as at present the body of literature on intervention in young infants has neglected the long-term effect of intervention on motor development. A positive effect of early intervention on cognitive and social development of infants with environmental disadvantage and infants biologically at risk because of preterm birth has been demonstrated unambiguously.[82–84] Early sensorimotor intervention might have a similar positive effect on motor development. The clinical implications of mildly abnormal GMs at two to four months are less clear. It could be that mildly abnormal GMs indicate a nonoptimally wired brain, putting the infant at risk for problems like MND, ADHD, and aggressive behavior. The risk needs to be determined by future investigations of the general population.

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REFERENCES

1. Bhushan V, Paneth N, Keily JL. Impact of improved survival of very low birth weight infants on recent secular trends in the prevalence of cerebral palsy. *Pediatrics* 1993;91:1094–1100.
2. Ornstein M, Ohlsson A, Edmonds J, et al. Neonatal follow-up of very low birthweight infants to school-age: a critical overview. *Acta Paediatr.* 1991;80:741–748.
3. Vohr BR, Garcia Coll CT. Neurodevelopmental and school performance of very low-birthweight infants: a seven-year longitudinal study. *Pediatrics* 1985;76:345–350.
4. Hadders-Algra M, Touwen BCL. Perinatal events and soft neurological signs in neurobehavioral outcome studies. *Dev Neuropsychol.* 2001; In press.
5. Hadders-Algra M. The assessment of general movements is a valuable technique for detecting brain dysfunction in young infants. A review. *Acta Paediatr.* 1996;(Suppl 416):39–43.
6. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research. Principles and Quantitative Methods.* New York: Van Nostrand Rienhold Co; 1982.
7. Darrah J, Piper M, Watt M-J. Assessment of gross motor activity of at-risk infants: predictive validity of the Alberta Infant Motor Scale. *Dev Med Child Neurol.* 1998;40:485–491.
8. Peiper A. *Cerebral Function in Infancy and Childhood.* 3rd ed. New York: Consultants Bureau; 1963.
9. André-Thomas, Saint-Anne Dargassies S. *Études neurologiques sur le nouveau-né et la jeune nourisson.* Paris: Masson; 1952.
10. Saint-Anne Dargassies S. *Le développement neurologique du nouveau-né à terme et prématuré.* Paris: Masson; 1974.
11. Prechtl HFR. *The Neurological Examination of the Full-Term Newborn Infant.* 2nd Ed. London: Spastics International Medical Publishers, Heinemann; 1977. Clinics in Developmental Medicine, No. 63.
12. Amiel-Tison C, Grenier A. Neurological assessment during the first year of life. New York: Oxford University Press; 1986.
13. Dubowitz LMS, Dubowitz V. *The Neurological Assessment of the Preterm and Full-Term Newborn Infant.* London: Heinemann; 1981. Clinics in Developmental Medicine, No. 79.
14. Dubowitz LMS, Dubowitz V, Mercuri E. *The Neurological Assessment of the Preterm and Full-Term Newborn Infant.* 2nd Ed. Cambridge: MacKeith Press; 1999. Clinics in Developmental Medicine, No. 148.
15. Harris SR, Brady DK. Infant neuromotor assessment instruments: a review. *Phys Occup Ther Pediatr.* 1986;6:121–153.
16. Stewart A, Hope PL, Hamilton P, et al. Prediction in very preterm infants of satisfactory neurodevelopmental progress at 12 months. *Dev Med Child Neurol.* 1988;30:53–63.
17. Allen MC, Capute AJ. Neonatal examination as a predictor of neuromotor outcome in premature infants. *Pediatrics* 1989;83:498–506.
18. Lanzi G, Fazzi E, Gerardo A, et al. Early predictors of neurodevelopmental outcome at 12 to 36 months in very low-birth-weight infants. *Brain Dev.* 1990;12:482–487.
19. Den Ouden L, Verloove-Vanhorick SP, van Zeben-van der AA DM, et al. Neonatal neurological dysfunction in a cohort of very preterm and/or very low birth-weight infants—relation to other perinatal factors and outcome at 2 years. *Neuropediatrics* 1990;21:66–71.
20. Cioni G, Ferrari F, Einspieler C, et al. Comparison between observation of spontaneous movements and neurological examination in preterm infants. *J Pediatr.* 1997;130:704–711.
21. Weisglas-Kuperus N, Baerts W, Fetter WPF, et al. Neonatal cerebral ultrasound, neonatal neurology and perinatal conditions as predictors of neurodevelopmental outcome in very low birthweight infants. *Early Hum Dev.* 1992;31:131–148.
22. Hadders-Algra M, Huisjes HJ, Touwen BCL. Preterm or small-for-gestational-age infants. Neurological and behavioral development at the age of 6 years. *Eur J Pediatr.* 1988;147:460–467.
23. Cioni G, Prechtl HFR, Ferrari F, et al. Which better predicts later outcome in full-term infants: quality of general movements of neurological examination. *Early Hum Dev.* 1997;50:71–85.
24. Hadders-Algra M, Huisjes HJ, Touwen BCL. Perinatal correlates of major and minor neurological dysfunction at school age: a multivariate analysis. *Dev Med Child Neurol.* 1988;30:472–481.
25. Chandler LS, Andrews MS, Swanson MW. *Movement Assessment of Infants: A Manual.* Rolling Bay, Wash; 1980.
26. Piper MC, Darrah J. *Motor Assessment of the Developing Infant.* Philadelphia: WB Saunders; 1994.
27. Harris SR, Haley SM, Tada WL, et al. Reliability of observational measurements of the Movement Assessment of Infants. *Phys Ther.* 1984;64:471–475.
28. Swanson MW, Bennett FC, Shy KK, et al. Identification of neurodevelopmental abnormality at four and eight months by the Movement Assessment of Infants. *Dev Med Child Neurol.* 1992;34:321–337.
29. Brander R, Kramer J, Cancsak M, et al. Inter-rater and test-retest reliabilities of the Movement Assessment of Infants. *Pediatr Phys Ther.* 1993;5:9–15.

30. Paban M, Piper MC. Early predictors of one year neurodevelopmental outcome for 'at risk' infants. *Phys Occup Ther Pediatr.* 1987;7:17–34.
31. Piper MC, Pinnell LE, Darrah J, et al. Early developmental screening: sensitivity and specificity of chronological and adjusted scores. *J Dev Behav Pediatr.* 1992;13:95–101.
32. Swanson MW, Bennett FC, Shy KK, et al. Identification of neurodevelopmental abnormality at four and eight months by the Movement Assessment of Infants. *Dev Med Child Neurol.* 1992;34:321–337.
33. Oppenheim RW. Ontogenetic adaptations and retrogressive processes in the development of the nervous system and behavior: a neuroembryological perspective. In: Connolly KJ, Prechtl HFR, eds. *Maturation and Development: Biological and Psychological Perspectives*: London: Heinemann; 1981:73–109. Clinics in Developmental Medicine, No. 77–78.
34. Prechtl HFR. The study of neural development as a perspective of clinical problems. In: Connolly KJ, Prechtl HFR, eds. *Maturation and Development: Biological and Psychological Perspectives.* London: Heinemann; 1981:189–215. Clinics in Developmental Medicine, No. 77–78.
35. Prechtl HFR. Qualitative changes of spontaneous movements in fetus and preterm infant are a marker of neurological dysfunction. *Early Hum Dev.* 1990;23:151–158.
36. Prechtl HFR. State of the art of a new functional assessment of the young nervous system. An early predictor of cerebral palsy. *Early Hum Dev.* 1997;50:1–11.
37. De Vries JIP, Visser GHA, Prechtl HFR. The emergence of fetal behavior. I. Qualitative aspects. *Early Hum Dev.* 1982;7:301–322.
38. Hopkins B, Prechtl HFR. A qualitative approach to the development of movements during early infancy. In: Prechtl HFR, ed. *Continuity of Neural Functions Form Prenatal to Postnatal Life.* Oxford: Blackwell Scientific Publications; 1984:179–197. Clinics in Developmental Medicine, No. 94.
39. Hadders-Algra M, Prechtl HFR. Developmental course of general movements in early infancy. I: Descriptive analysis of change in form. *Early Hum Dev.* 1992;28:201–214.
40. Hadders-Algra M, Van Eykern LA, Klip-van den Nieuwendijk AWJ, et al. Developmental course of general movements in early infancy. II. EMG correlates. *Early Hum Dev.* 1992;28:231–252.
41. Hadders-Algra M, Prechtl HFR. EMG correlates of general movements in healthy preterm infants. *J Physiol.* 1993;459:330P.
42. Hadders-Algra M, Klip-Van den Nieuwendijk AWJ, Martijn A, et al. Assessment of general movements: towards a better understanding of a sensitive method to evaluate brain function in young infants. *Dev Med Child Neurol.* 1997;39:88–98.
43. Nijhuis JG, Prechtl HFR, Martin CB, et al. Are there behavioral states in the human fetus? *Early Hum Dev.* 1982;6:177–195.
44. Cioni G, Prechtl HFR. Preterm and early postterm motor behavior in low-risk premature infants. *Early Hum Dev.* 1990;23:159–192.
45. Fulton BP, Walton K. Electrophysiological properties of neonatal rat motoneurones studied in vitro. *J Physiol.* 1986;370:651–678.
46. Gramsbergen A, Ijkema-Paassen J, Nikkels PGJ, et al. Regression of polyneural innervation in the human psoas muscle. *Early Hum Dev.* 1997;49:49–61.
47. Chugani HT, Phelps ME, Maziotta JC. 18-FDG Positron emission tomography in human brain. Functional development. *Ann Neurol.* 1987;22:487–497.
48. Hadders-Algra M. De beoordeling van spontane motoriek van jonge baby's: een doeltreffende methode voor de opsporing van hersenfunctiestoornissen. *Ned Tijdschr Geneeskd* 1997;141:816–820.
49. Hadders-Algra M, Groothuis AMC. Quality of general movements in infancy is related to the development of neurological dysfunction, attention deficit hyperactivity disorder and aggressive behavior. *Dev Med Child Neurol.* 1999;41:381–391.
50. Touwen BCL. Variability and stereotypy in normal and deviant development. In: Apley J, ed. *Care of the Handicapped Child.* London: Heinemann; 1978:99–110. Clinics in Developmental Medicine, No. 67.
51. Touwen BCL. Variability and stereotypy of spontaneous motility as a predictor of neurological development of preterm infants. *Dev Med Child Neurol.* 1990;32:501–508.
52. Touwen BCL. How normal is variable, or now variable is normal? *Early Hum Dev.* 1993;34:1–12.
53. Hadders-Algra M. The Neuronal Group Selection Theory: an attractive framework to explain variation in normal motor development. *Dev Med Child Neurol.* 2000;42:566–572.
54. Kakebeeke TH, Von Siebenthal K, Largo RH. Differences in movement quality at term among preterm and term infants. *Biol Neonate.* 1997;71:367–378.
55. Ferrari F, Cioni G, Prechtl HFR. Qualitative changes of general movements in preterm infants with brain lesions. *Early Hum Dev.* 1990;23: 193–231.
56. Prechtl HFR, Ferrari F, Cioni G. Predictive value of general movements in asphyxiated fullterm infants. *Early Hum Dev.* 1993;35:91–120.
57. Prechtl HFR, Nolte R. Motor behavior of preterm infants. In: Prechtl HFR, ed. *Continuity of Neural Functions From Prenatal to Postnatal Life.* Oxford: Blackwell Scientific Publications; 1984:79–92. Clinics in Developmental Medicine, No. 94.
58. Lorenz KZ. Gestaltwahrnehmung als Quelle wissenschaftlicher Erkenntnis. In: Lorenz K, ed. *Über tierisches en menschliches Verhalten.* München: Piper; 1971:255–300.
59. Hadders-Algra M, Nakae Y, Van Eykern LA, et al. The effect of behavioral state on general movements in healthy full-term newborns. A polymyographic study. *Early Hum Dev.* 1993;35:63–79.
60. Prechtl HFR. The behavioral state of the infant—a review. *Brain Res.* 1974;76:185–212.
61. Casaer P. *Postural Behavior in Newborn Infants.* London: Heinemann; 1979. Clinics in Developmental Medicine, No. 72.
62. Van Kranen-Mastenbroek V, Van Oostenbrugge R, Palmans L, et al. Inter- and intraobserver agreement in the assessment of the quality of spontaneous movements in the newborn. *Brain Dev.* 1992;14:289–293.
63. Geerdink JJ, Hopkins B. Effects of birthweight status and gestational age on the quality of general movements in preterm newborns. *Biol Neonate.* 1993;63:215–224.
64. Einspieler C, Prechtl HFR, Ferrari F, et al. The qualitative assessment of general movements in preterm, term and young infants—review of methodology. *Early Hum Dev.* 1997;50:47–60.
65. Kainer F, Prechtl HFR, Engele H, er al. Assessment of the quality of general movements in fetuses and infants of women with type-1 diabetes mellitus. *Early Hum Dev.* 1997;50:13–25.
66. Sival DA, Visser GHA, Prechtl HFR. Does reduction of amniotic fluid affect fetal movements? *Early Hum Dev.* 1990;23:233–246.
67. Bekedam DJ, Visser GHA, De Vries JJ, et al. Motor behavior in the growth-retarded fetus. *Early Hum Dev.* 1985;12:155–165.
68. Sival DA, Visser GHA, Prechtl HFR. The effect of intrauterine growth retardation on the quality of general movements in the human fetus. *Early Hum Dev.* 1992;28:119–132.
69. Van Kranen-Mastenbroek VH, Kingma H, Caberg HB, et al. Quality of spontaneous general movements in full-term small-for-gestational-age and appropriate-for-gestational-age newborn infants. *Neuropediatrics* 1994;25:145–153.
70. Bos AF, Van Loon AJ, Hadders-Algra M, et al. Spontaneous motility in preterm, small for gestational age infants. II. Qualitative aspects. *Early Hum Dev.* 1997;50:131–147.
71. Albers S, Jorch G. Prognostic significance of spontaneous motility in very immature preterm infants under intensive care treatment. *Biol Neonate.* 1994;66:182–187.
72. Bos AF, Martijn A, Van Asperen RM, et al. Qualitative assessment of general movements in high risk preterm infants with chronic lung disease requiring dexamethasone therapy. *J Pediatr.* 1998;132:300–306.
73. Bos AF, Martijn A, Okken A, et al. Quality of general movements in preterm infants with transient periventricular echodensities. *Acta Paediatr.* 1998;87:328–335.

74. Van Hall M. *Assessment of Spontaneous Motor Behavior in Relation to Umbilical Artery pH in Full-Term Infants*. University Maastricht, The Netherlands: PhD Thesis 1999.
75. Soorani-Lunsing RJ, Woltil HA, Hadders-Algra M. Are moderate degrees of hyperbilirubinaemia in healthy term neonates really safe for the brain? *Pediatr Res.* 2001, In press.
76. Visser GHA, Laurini RN, de Vries JIP, et al. Abnormal motor behavior in anecephalic fetuses. *Early Hum Dev.* 1985;12:173–182.
77. Bos AF, Van Asperen RM, De Leeuw DM, et al. The influence of septicaemia on spontaneous motility in preterm infants. *Early Hum Dev.* 1997;50:61–70.
78. Prechtl HFR, Einspieler C, Cioni G, et al. An early marker of developing neurological handicap after perinatal brain lesions. *Lancet.* 1997;339:1361–1363.
79. Geerdink JJ, Hopkins B. Qualitative changes in general movements and their prognostic value in preterm infants. *Eur J Paediatr.* 1993; 152:357–367.
80. Kolb B, Whishaw IQ. Plasticity in the neocortex: mechanisms underlying recovery form early brain damage. *Prog Neurobiol.* 1989;276:235–276.
81. Villablance JR, Hovda DA. Developmental neuroplasticity in a model of cerebral hemispherectomy and stroke. *Neuroscience* 2000;95:625–637.
82. Infant Health and Development Program. Enhancing the outcomes of low-birth-weight, premature infants. *JAMA.* 1990;263:3035–3042.
83. Achenbach TM, Howell CT, Aoki MF, et al. Nine-year outcome of the Vermont Intervention Program for low birth weight infants. *Pediatrics.* 1993;91:45–55.
84. Majnemer A. Benefits of early intervention for children with developmental disabilities. *Semin Pediatr Neurol.* 1998;5:62–69.