Intrauterine growth retardation (IUGR) due to placental dysfunction is a risk factor for an impaired neurodevelopmental outcome. This is the case particularly in growth-retarded foetuses whose conditions in utero are so compromised that they have to be delivered before term. In recent years, neonatal treatment and care have improved markedly, and this has led to increased chances of survival of very immature and extremely low-birthweight infants. The optimal time to deliver IUGR infants remains difficult to assess. There is an obvious need for methods that accurately monitor foetal and neonatal condition, and it is important that the implications of these methods are evaluated for the neurodevelopmental outcomes of these infants. It is vitally important to be able to predict the outcome, whether it is normal or abnormal. A method that covers both aspects is the qualitative assessment of spontaneous motility of foetuses and newborn infants.

This review summarizes several studies on the neurodevelopmental outcome of IUGR infants. It also investigates the relationship between the development of spontaneous motility early in life and obstetrical variables indicative of the foetal condition.

**Neurodevelopmental outcome of IUGR infants**

Although several studies have reported on the neurodevelopmental outcomes of IUGR infants, the results are conflicting. When reviewing these studies, a number of points that can explain the discrepancies among them should be considered (Table I). The most important point is the heterogeneity of the group of infants with growth retardation. Growth retardation is generally determined according to birthweight, so IUGR is considered more or less equivalent to small for gestational age (SGA). However, different criteria are used to define SGA. Neonatologists variously define SGA as a birthweight below the 2.3, 5th, or 10th centile on a birthweight versus gestational age curve. Particularly when the broadest criterion is applied, many of these infants would not qualify as IUGR at all: they just happen to fall into the lower range of the normal population distribution. In addition, some infants are born small because of an (undetected) chromosomal abnormality or dysmorphic syndrome. In addition, within the group of growth-restricted infants due to placental dysfunction, the onset and severity of the growth retardation add to the heterogeneity of the SGA group.

Another problem relates to heterogeneity of developmental tests used in the studies we reviewed. Neurodevelopmental assessment can be difficult. Gross effects can be easily quantified, whereas subtle or discrete lesions are more problematic. Some sophisticated tests may detect these subtle lesions, while others may overlook them. Furthermore, follow-ups of different durations, small sample sizes, inclusion of all gestational ages, or preterm or term SGA infants only, as well as specific characteristics of the control infants, could also explain the conflicting results.

Despite this, a number of conclusions can be drawn from the studies. Tables II, III, and IV show some characteristics and the main findings of case–control studies on neurodevelopmental outcomes published during the last 20 years. Table II presents studies that included infants of all gestational ages. Table III does the same for studies that included only term infants, and Table IV presents the findings on preterm
infants only. Generally speaking, there is an increased risk for mild neurodevelopmental abnormalities with cognitive disabilities and behavioural problems, more so than motor disturbances. In addition, microcephaly at birth and a lack of catch up of head growth are related to an increased prevalence of neurodevelopmental abnormalities, especially in term infants with growth retardation. This suggests that brain sparing is a relatively favourable feature in growth retardation. However, not all studies confirm this hypothesis. A recent study related foetal growth pattern in the third trimester to the neurodevelopmental outcome at 1 year of age. Although the incidence of minor abnormalities was quite high (approximately one-third), no differences were found between foetuses that were already small and foetuses whose intrauterine growth was restricted.

The risk for major neurological abnormalities in infants with IUGR is less clear. Several studies reported a slightly increased prevalence of cerebral palsy (CP), especially in term infants with growth retardation. It is unclear if this finding also applies to preterm infants. Some studies did not find any difference, while others reported an increased risk. In comparison to term infants, the prevalence of CP is several times higher in preterm infants, a slightly increased risk caused by growth retardation may be too small to be detected.

**Evaluation of the foetal condition in cases of IUGR**

The evaluation of the foetal condition in growth-retarded foetuses is of the utmost importance to determine the optimal time for early delivery by Caesarean section, aiming at intact survival with a minimum of neurological sequelae. As yet, it remains uncertain when the growth-retarded foetus should be delivered. Various methods are used to monitor the foetal condition, e.g. foetal heart rate patterns, foetal biophysical profile scoring, and umbilical artery waveform patterns. All these methods provide a good indication of perinatal complications, but the relevance for neurological sequelae remains unclear.

The foetal biophysical profile score consists of a combination of five items: foetal heart rate patterns, foetal breathing movements, foetal tone, foetal body movements, and amniotic fluid volume. A score of 10 is obtained if all items are normal and 0 if all are abnormal. A clear correlation exists between perinatal complications, including mortality and a low biophysical profile score, particularly if the low score was caused by abnormal foetal heart rate patterns and decreased amniotic fluid volume. However, data on the relationship with neurological outcome are lacking.

Many studies have demonstrated that umbilical artery waveform patterns are impaired in pregnancies complicated by growth retardation. A clear correlation was reported between abnormal umbilical artery waveform patterns and the risk of perinatal complications. An increased pulsatility index of the umbilical artery often precedes other signs of

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**Table I: Reasons for conflicting results in studies on neurodevelopmental follow-up in infants with SGA/IUGR**

<table>
<thead>
<tr>
<th>Reasons for conflicting results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definition of growth retardation (BW &lt;10, &lt;5, or &lt;2.3 centile)</td>
</tr>
<tr>
<td>Inclusion or exclusion of infants with congenital anomalies</td>
</tr>
<tr>
<td>Inclusion of all gestational ages versus preterm or term infants only</td>
</tr>
<tr>
<td>Number of infants lost to follow-up (may exceed 50%)</td>
</tr>
<tr>
<td>Heterogeneous developmental tests to assess outcome</td>
</tr>
<tr>
<td>Different kinds of growth retardation (early or late onset)</td>
</tr>
</tbody>
</table>

SGA, small for gestational age; IUGR, interuterine growth retardation.

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**Table II: Follow-up studies that included all gestational ages of infants with IUGR**

<table>
<thead>
<tr>
<th>Reference</th>
<th>IUGR group characteristics</th>
<th>Control characteristics</th>
<th>Age at follow-up</th>
<th>Developmental tests</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berg (1988)</td>
<td>602 All GA &lt;10th BW &gt;10th centile, N=4761</td>
<td>7 years Neurological examination</td>
<td>IUGR infants had higher frequency of developmental delays compared to controls. Microcephaly at age 2 years associated with developmental abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tenovuo et al. (1988)</td>
<td>519 All GA &lt;10th BW &gt;10th centile, term, N=3375</td>
<td>2 years Denver Developmental Screening Test</td>
<td>IUGR infants had higher frequency of developmental delays compared to controls. Microcephaly at age 2 years associated with developmental abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low et al. (1992)</td>
<td>77 All GA &lt;10th BW &gt;10th centile, high risk group, N=141</td>
<td>9–11 years Neurological exam, Bruininks–Oseretsky test, Wechsler Intelligence Scale for Children, Questionnaires (parents and teacher)</td>
<td>Overall 35% learning deficits associated with growth retardation and father’s occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fattal-Valeski et al. (1999)</td>
<td>85 All GA &lt;5th BW &gt;10th centile, N=420</td>
<td>3 years Neurodevelopmental Assessment Scale (NAS), Stanford–Binet test</td>
<td>IUGR infants had lower scores on NAS. IUGR infants with neonatal complications had lower scores than infants without complications</td>
<td></td>
<td></td>
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</tbody>
</table>

GA, gestational age; BW, birthweight.
foetal distress. Absent or reversed end-diastolic velocities in the umbilical artery are associated with a perinatal mortality risk of up to 50%\textsuperscript{43, 44}. However, only a few studies succeeded in finding a correlation between abnormal umbilical artery waveform patterns and later neurodevelopmental outcome\textsuperscript{33, 34}. Moreover, although IUGR strongly correlates with placental markers indicating impaired utero–placental blood flow, no association was demonstrated between placental pathology and the neurodevelopmental outcome in the first year of life\textsuperscript{26}.

Recently, a method of recording foetal cerebral circulation was added to the diagnostic methods available for IUGR infants. It calculates the cerebro–placental ratio of blood flow which provides some measure of the degree of brain sparing. The cerebro–placental ratio was also impaired in growth-retarded foetuses, but again, no correlation was found between perinatal complications and neurodevelopmental outcome\textsuperscript{16, 61}, except, possibly, before 34 weeks' gestation\textsuperscript{3}. Impairment may be a rather late sign: in a longitudinal study, the reduction in growth velocity preceded the changes observed in foetal circulation\textsuperscript{29}.

Other efforts to evaluate the condition of the foetus aim at a more accurate assessment of the neurological condition of growth-retarded foetuses and preterm infants. This approach

### Table III: Studies on follow-up of term IUGR infants

<table>
<thead>
<tr>
<th>Reference</th>
<th>IUGR group characteristics</th>
<th>Control characteristics</th>
<th>Age at follow-up</th>
<th>Developmental tests</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low et al. (1982)\textsuperscript{35}</td>
<td>76 Term born &lt; 10th BW &gt; 25th centile, N=88</td>
<td>5 years Neurological examination, McCarthy motor scores, Wechsler Intelligence Scale for Children</td>
<td>No difference between groups</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Villar et al. (1984)\textsuperscript{73}</td>
<td>59 Term born &lt; 10th BW &gt; 10th centile, N=146</td>
<td>3 years Battery of mental tests</td>
<td>IUGR infants had lower cognitive scores. Those with adequate ponderal indices had lowest scores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berg (1989)\textsuperscript{10}</td>
<td>467 Term born &lt; 10th BW &gt; 10th centile, N=4068</td>
<td>7 years Neurological examination</td>
<td>IUGR infants with perinatal hypoxia-related factors had more neurological abnormalities but no difference between groups in absence of perinatal hypoxia-related factors. Microcephaly associated with increased risk of neurological abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paz et al. (1995)\textsuperscript{47}</td>
<td>64 Term born &lt; 3rd BW &gt; 3rd centile, N=1643</td>
<td>17 years Neurological examination, Wechsler Adult Intelligence Scale</td>
<td>IUGR infants had increased risk for lower cognitive performance and schooling achievement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pryor et al. (1995)\textsuperscript{55}</td>
<td>91 Term born &lt; 10th BW &gt; 10th centile, N=946</td>
<td>13, 15, and 18 years Wechsler Intelligence Scale for Children–Revised, Burt Reading Scores, Revised Problem Behaviour Checklist</td>
<td>IUGR infants had lower IQs at age 13 (IQ 101.2 vs 109.0). Parents of IUGR infants reported more behavioural problems at age 15y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Markestad et al. (1997)\textsuperscript{10}</td>
<td>265 Term born &lt; 15th BW &gt; 15th centile, N=529</td>
<td>13 months Bayley Scales of Infant Development</td>
<td>IUGR infants scored lower on mental scale (DQ 112 vs 116)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strauss and Dietz (1998)\textsuperscript{66}</td>
<td>2719 Term born ≤ 2500g BW &gt; 2500g, N=43 104</td>
<td>7 years Wechsler Intelligence Scale, Bender–Gestalt test (visual–motor development)</td>
<td>IUGR infants had lower IQs (90.6 vs 96.8) compared to non-IUGR group, and lower Bender–Gestalt scores, No difference between IUGR infants and non-IUGR siblings. Siblings with IUGR and large deficit in head circumference had lower scores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strauss (2000)\textsuperscript{65}</td>
<td>106 220 non-IUGR siblings, N=220</td>
<td>16 and 26 years Vocabulary/spelling test at age 16y, Questionnaires (teacher and community health officer) on school performance, Questionnaire participants at age 20y</td>
<td>IUGR infants demonstrated small deficits in academic achievement at age 16 years. No difference between groups at 26 years</td>
<td></td>
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</tr>
</tbody>
</table>

GA, gestational age; BW, birthweight; DQ, developmental quotient; sgtns, singletons.
Table IV: Studies on follow-up of preterm IUGR/SGA infants

<table>
<thead>
<tr>
<th>Reference</th>
<th>IUGR/SGA characteristics</th>
<th>Control characteristics</th>
<th>Age at follow-up</th>
<th>Developmental tests</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matilainen et al. (1987)\textsuperscript{41}</td>
<td>N GA/BW Centile</td>
<td>BW &gt; 2.3 centile, PT-AGA N=48, term N=36</td>
<td>4 years</td>
<td>Neurodevelopmental Scoring Test, Psychological performance tests</td>
<td>IUGR infants had more moderate or high risk scores compared to both control groups. Abnormalities consisted of complex deviations of motor, visual, and perceptual functions.</td>
</tr>
<tr>
<td>Hadders-Algra et al. (1988)\textsuperscript{57}</td>
<td>27 &lt; 37 weeks BW &gt; 10th centile, preterm AGA N=55, term AGA N=206. BW &lt; 10th centile, term GA N=166</td>
<td>6 years</td>
<td>Neurological examination, Questionnaire (parents and teacher)</td>
<td>Major and minor neurological abnormalities were more frequent in all three low-birthweight groups, especially in preterm SGA. Incidence of abnormalities: PT-SGA, major 15% minor 37% PT-AGA, major 4% minor 26% Term SGA, major 2% minor 24% Term AGA, major 1% minor 15%</td>
<td></td>
</tr>
<tr>
<td>Veelken et al. (1992)\textsuperscript{72}</td>
<td>96 ≤1500g &lt; 10th weeks BW &gt; 10th centile, AGA N=275</td>
<td>18–20 months</td>
<td>Neurological examination, Griffiths developmental test</td>
<td>Incidence of CP was decreased in IUGR group compared to AGA infants (7% vs 17.5%, due to lower GA in AGA group). IUGR infants had higher frequency of minor neurological abnormalities (30% vs 15.3%)</td>
<td></td>
</tr>
<tr>
<td>Smedler et al. (1992)\textsuperscript{64}</td>
<td>14 &lt; 37 weeks BW &gt; 10th centile, term born, matched for age, sex, socioeconomic background</td>
<td>8.7–11.2 years</td>
<td>Wechsler Intelligence Scale for Children, Bruininks–Oseretsky test, Halstead–Reitan neuropsychological battery, Southern California Tests of Sensory Integration</td>
<td>IUGR infants had lower scores on visuospatial ability, nonverbal reasoning, strategy formation, and gross motor coordination. More preterm IUGR infants had lowest scores</td>
<td></td>
</tr>
<tr>
<td>Sung et al. (1993)\textsuperscript{67}</td>
<td>27 ≤31 weeks BW &gt; 10th centile, Matched for GA, AGA-GA, N=27, Matched for BW, AGA-BW, N=27</td>
<td>3 years</td>
<td>Neurological examination, McCarthy Scales of Children’s Abilities, Peabody Picture Vocabulary Test</td>
<td>No difference for neurological outcome between IUGR group and AGA-GA matched group. Lower scores for developmental tests in IUGR group compared to AGA-GA matched group, but similar to AGA-BW matched group. Latter is due to lower GA of AGA-BW matched group</td>
<td></td>
</tr>
<tr>
<td>Wocadlo and Rieger (1994)\textsuperscript{81}</td>
<td>18 &lt; 30 weeks BW &gt; 10th centile, Matched for GA, sex, CLD and ultrasound findings, N=18</td>
<td>1 year</td>
<td>Bayley Scales of Infant Development or Griffiths Mental Development Scales</td>
<td>No difference between groups</td>
<td></td>
</tr>
<tr>
<td>McCarton et al. (1996)\textsuperscript{12}</td>
<td>129 ≤37 weeks, ≤2500g BW &gt;3rd centile, AGA N=300</td>
<td>6 years</td>
<td>Neurological examination, Wechsler Intelligence Scale for Children–Revised</td>
<td>Increased risk for neurodevelopmental impairment in IUGR infants (IQ 85.4 vs 91.4). Higher incidence of neurological abnormalities (18% vs 10%, suspicious 11% vs 2%)</td>
<td></td>
</tr>
</tbody>
</table>

GA, gestational age; BW, birthweight; AGA, appropriate for gestational age; PT, preterm infants; CLD, chronic lung disease.
leads to a better understanding of CNS functioning in preterm infants with growth retardation, and is necessary to evaluate interventions. One of the new methods is the qualitative assessment of general movements (GMs) by videotape9. It proves to be a sensitive method for assessing the integrity of the CNS in early life. This method is non-intrusive, takes little time to carry out, and high interrater reliability scores are obtained19. Assessment can be repeated in the same individual so that a document of an individual’s developmental course can be compiled. An added advantage is that the same qualitative criteria apply to both foetuses and preterm babies, as well as to infants during their first months of life.

**Quantitative analysis of movement patterns**

Prechtl’s classification of spontaneous movement patterns led to a classification of the entire movement repertoire of the foetus and newborn infant55, 54, 79. The quantitative aspects of the movement patterns that were investigated included the emergence of rest–activity cycles as well as the rate of occurrence of several movement patterns. Both aspects were investigated under normal and compromised circumstances.

**REST–ACTIVITY CYCLES AND BEHAVIOURAL STATES**

During the second half of pregnancy, many movement patterns increasingly occur in clusters. This leads to rest–activity cycles that are clearly recognizable from about the 30th week of gestation onwards. In healthy preterm infants the rest–activity cycles can be identified at equivalent postmenstrual ages18, 53. The rest–activity cycles precede the emergence of true behavioural states. In healthy foetuses, behavioural states are present from 36 to 38 weeks of gestation onwards45, 77. In the way they are organized they are fully comparable to the behavioural states of term neonates. A number of researchers have demonstrated that the emergence of behavioural states is delayed in growth-retarded foetuses4, 76. It is unknown if this is also the case in infants with growth retardation. The significance of these findings for the neurodevelopmental outcome remains unclear, because a delay in the development of behavioural states does not have a specific predictive value for the later outcome50.

**RATE OF OCCURRENCE OF DISTINCT MOVEMENT PATTERNS**

A large range of intra- and interindividual variability exists in the rate of occurrence of all movement patterns throughout pregnancy59, 78, 80. This is also the case in healthy preterm infants18, 53, 54. The large variability in the quantitative data of spontaneous motility in uncompromised foetuses and in healthy preterm infants makes quantitative assessment an insensitive indicator of compromising conditions of the nervous system, a fact which has been demonstrated repeatedly. In growth-retarded foetuses a reduction in the quantity of general movements is a late sign of foetal deterioration6, 7, since a reduction below the lower limit of normal occurs only in preterminal or terminal foetuses58, 65. In several studies on preterm infants, no difference in the quantity (frequency and duration) of various movement patterns was found between a high-risk group with brain lesions and subsequent disability, and a low-risk normally developing group20, 25, 30, 54. In preterm infants with growth retardation, the rate of occurrence of various movement patterns did not differ from that found in low-risk preterm infants14, with the exception of the duration of GMs during the first week after birth, which was significantly lower in the infants with growth retardation.

Few correlations were found between the quantity of movement patterns after birth and obstetrical variables indicative of an impaired foetal condition. Only a reduction in foetal heart-rate variation was found to correlate slightly with an increased incidence of startles and twitches in the first week of life14.

Therefore, it is clear that quantitative changes in motility are unsuitable markers of neurological dysfunction in foetuses and newborn infants. This is in striking contrast to the qualitative aspects of motility, which change dramatically in the case of brain damage or malformation25, 24, 54, 75.

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**Table IV: Continued**

<table>
<thead>
<tr>
<th>Reference</th>
<th>IUGR/NGA characteristics</th>
<th>Control characteristics</th>
<th>Age at follow-up</th>
<th>Developmental tests</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amin et al. (1997)3</td>
<td>N GA/BW Centile &lt;1250g</td>
<td>&lt;2.3</td>
<td>3 years</td>
<td>Neurological examination, Stanford–Binet Intelligence Scale</td>
<td>No differences between groups. Incidence of major neurodevelopmental abnormalities 15.4% vs 16.1% (GA-matched) and 16.4% (BW-matched). Persistence of microcephaly (in all groups) associated with adverse neurodevelopmental outcome</td>
</tr>
<tr>
<td>Kok et al. (1998)31</td>
<td>124 ≤32 weeks &lt;10th</td>
<td>25th and &lt;75th centile</td>
<td>5 and 9 years</td>
<td>Neurological examination 9y: Questionnaire to parents (school performance)</td>
<td>At 5y: IUGR infants had more gross motor dysfunction and MND (50% vs 56%); incidence of CP was identical. At 9y: &gt;70–75% of IUGR infants needed special education vs 50% of control infants</td>
</tr>
</tbody>
</table>

GA, gestational age; BW, birthweight; AGA, appropriate for gestational age; MND, minor neurological dysfunction.
Qualitative analysis of general movements

Of all the endogenously generated movement patterns in foetuses and infants, GMs occur most frequently and they are very complex. They can be observed in foetuses as young as 9 weeks’ postmenstrual age20, and continue in a similar pattern after birth until about the end of the second month postterm. Normal GMs are characterized by a large variability in speed, amplitude, force, and intensity. The sequence of arm, leg, head, and trunk movements is complex, with rotations superimposed on flexion and extension, making the normal general movement look fluent and elegant39. In cases of brain abnormalities, such as cystic periventricular leukomalacia or large intracranial haemorrhages, the quality of the GMs is impaired: GMs appear monotonous with reduced complexity, variability, and fluency25. Three main types of abnormal GMs are described: (1) ‘Poor repertoire GMs’, when the sequence of successive movement components is monotonous, and arm, leg, trunk and head movements do not occur in the normal rich and complex sequence; (2) ‘Crammed–synchronized GMs’, when GMs appear rigid and stiff, lack the normal smooth and fluent character, and all limb and trunk muscles contract and relax almost simultaneously; (3) ‘Chaotic GMs’, when the movements of all limbs are of large amplitude and occur in a chaotic order without any fluency or smoothness19, 52.

At about the end of the second month postterm, during the so-called major neural transformation48, the GMs acquire a fidgety character. Fidgety GMs are circular movements of small amplitude, moderate speed, and variable acceleration of neck, trunk, and limbs in all directions52. They may be seen as early as 6 weeks postterm but usually occur around 9 weeks and remain present until 15 to 20 weeks. In preterm infants, this change in characteristics of spontaneous movements can be observed at about the same time if the infant’s age is corrected for preterm birth. Abnormal movements at this age range are the complete absence of fidgety movements (however, other movements can occur), and abnormal fidgety movements, which resemble normal fidgety movements, but with exaggerated speed, amplitude and jerkiness52. The quality of fidgety movements has a high predictive power for the neurological outcome. Not only are abnormal or absent fidgety movements indicative of a poor outcome, but normal fidgety movements are an excellent predictor for a normal neurological outcome22, 23.

Relationship between quality of GMs, brain ultrasound findings, and neurodevelopmental outcome in IUGR

Several studies have investigated the quality of general movements in foetuses and infants with IUGR. Movement quality was found to be impaired in IUGR foetuses6, 52. Similar findings were reported in cross-sectional studies in preterm25 and term52 SGA infants. The developmental course of the quality of general movements in preterm IUGR infants has also been elucidated. A clear relationship exists between specific developmental trajectories of GM quality and the neurological outcome at 2 years of age15. In contrast, the neurological outcome is not correlated to brain ultrasound findings, obstetrical variables indicative of foetal distress, the degree of growth retardation, or the extent of brain sparing. Thus, the assessment of the quality of GMs in preterm SGA infants is a sensitive method with a high predictive value for the later outcome, as it is in preterm infants who are appropriate-for-gestational age (AGA)1, 11, 25.

It appears that most IUGR infants have an abnormal quality of GMs during their preterm period, but the longitudinal approach reveals that the quality of GMs normalizes in the majority of the infants, at or after term age15. Some infants have consistently abnormal GMs, and this feature is predictive for an abnormal outcome. The quality of fidgety movements, in particular, is predictive of the final outcome. It must be noted that cramped–synchronized GMs, as an abnormal type of movement predictive for CP, occur rather late in infants with growth retardation, as compared to AGA infants23. Interestingly, a large proportion of infants with growth retardation who have abnormal GMs have normal findings on brain ultrasound scans12. This suggests that the chronically-reduced foetal supply of oxygen and nutrients may lead to a longer lasting, but often transient, brain dysfunction, which is not necessarily caused by haemorrhagic or hypoxic–ischaemic lesions detectable on ultrasound scans.

The qualitative assessment of GMs has proved its worth mainly in relation to the prediction of motor disorders. Future studies will need to elucidate whether the quality of GMs is also predictive of the cognitive disabilities and behavioural problems that are found more often in IUGR infants.

Relationship between quality of GMs and obstetric variables

Detailed analysis reveals several types of abnormal GMs in growth-retarded foetuses and infants. In two studies, a large proportion of foetuses and infants had ‘slow motion’ GMs15, 62. Obstetrical variables indicative of a compromised foetal condition were not related to this movement quality. It was suggested that a reduced amount of amniotic fluid directly affects the speed and amplitude of GMs62. Surprisingly, slow motion GMs also occurred in infants whose mothers had not received corticosteroids before birth15. The reason for this effect remains unclear. It could be due to a decreased incidence of neonatal respiratory morbidity, or direct neurological or metabolic effects that alter the character of the movements12.

‘Chaotic’ GMs are also frequently observed in infants with growth retardation, but have not been reported in growth-retarded foetuses. In term SGA infants, the qualitative assessment of movement patterns reveals an increased incidence of jerky and tremulous movements32. In preterm SGA infants, the presence of this abnormal movement type is related to late foetal heart-rate decelerations and ischaemic alterations of the placenta15. This indicates that acute foetal deterioration, superimposed on chronic placental insufficiency, might be responsible for the occurrence of this movement type. The chaotic GMs in the latter study were not related to detectable brain lesions.

Conclusion

Recent studies have shown that the qualitative assessment of GMs is a powerful diagnostic method to evaluate brain dysfunction in preterm and term infants. Many infants with growth retardation have transiently abnormal GMs, indicating the importance of obtaining multiple observations. The quality of fidgety movements, in particular, is predictive for the neurodevelopmental outcome. It has been suggested that GM abnormalities at a young age are related to lesions of neural subsystems whose role in motor control ceases after 2 to 3 months17. These abnormalities may disappear if the new, posttransformation set of neural functions is not impaired. The assessment of developmental trajectories of GM quality
helps to select, at an early age, those infants who are at risk for developmental deficits and to provide surveillance and intervention on time.

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References