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Qualitative changes in general movements and their prognostic value in preterm infants

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Abstract. Qualitative asessments of general movements have been shown to have considerable power in predicting neurological outcomes in preterm infants with brain damage. In the present study such assessments were made in 35 preterm infants without major neurological problems before term age, born between 27 and 34 weeks gestation, of whom 12 were small-for-gestational-age (SGA). Most infants maintained a normal or (mildly) abnormal quality from 35 weeks postmenstrual age through 6, 12, 18 to 24 weeks corrected age. Seven changed from initially abnormal movements to a normal quality, six of them after 12 weeks. Differences between SGA and appropriate-for-gestational-age infants became less evident with age, particularly after 12 weeks. This was not the case when comparisons were made on the basis of gestational ages below or above 32 weeks. The prediction of neurological and mental outcomes at 1 year also improved after 12 weeks, around which age a major transformation in neural functions occurs. It is concluded that assessments of movement quality are particularly successful in predicting abnormal outcomes in comparison to examinations based on muscle tone and elicited responses.

Key words: Preterm infants – General movements – Movement quality – Neurological transformation – Predictive value

Introduction

Previous studies have indicated that spontaneous (i.e. endogeneously generated) movements unrelated to overt external stimuli, appear to have a greater sensitivity to adverse conditions that elicited responses [24]. Hence, they may consitute a useful means of evaluating the neurological condition of fetuses and preterm infants. The most frequently occurring category of spontaneous movements is that of general movements (GMs) which involve all parts of the body. In healthy infants GMs are complex and fluent in appearance, with variations in speed and amplitude [12]. Assessments of the quality of GMs in preterm infants with documented brain injury have indicated a lack of variation and fluency [7]. The question remains as to their usefulness in evaluating the effect of adverse conditions such as preterm birth and intra-uterine growth retardation in infants without such obvious neurological damage.

Preterm birth, especially before 32 weeks gestation, and growth retardation are related to developmental problems at later ages [1, 14, 15]. It should be noted, however, that studies identifying 32 weeks as a critical age have included infants with serious medical complications [14, 15], which in itself may account for the poor development outcomes of these children. Moreover, neonatal morbidity appears to be increased in small-for-gestational-age (SGA) compared to appropriate-for-gestational-age (AGA) preterm newborns [11]. The higher incidence of neurological sequelae in SGA preterm infants reported in the 1st year [20], and at preschool [4] and school ages [10], could be related therefore to their increased neonatal morbidity. Thus, studies concerned with whether preterm birth before 32 weeks or a birth weight deemed to be SGA adversely influences developmental outcomes should be based on longitudinal data obtained from infants without serious medical complications or neonatal morbidities. Qualitative assessments of GMs appear to be a reliable and non-invasive means of obtaining such data.

The present study had two main aims. Firstly, to examine whether birth before 32 weeks and/or a SGA birth weight adversely affect the development of GM quality in otherwise healthy preterm infants. Secondly, whether the resultant developmental courses are predictive of neurological and mental outcomes at 1 year corrected age. In addition, it can be asked if the transformation of GMs from a writhing to a fidgety character, which occurs around 2–3 months of (corrected) age in healthy full-term [12] and low-risk preterm infants [3],

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Abbreviations: AGA = appropriate-for-gestational-age; GMs = general movements; SGA = small-for-gestational age

is impaired in those subjects with a deviant movement quality.

Subjects and methods

Subjects

Infants were carefully selected for the absence of serious medical complications. The selection criteria are given in Table 1. The selected infants were examined by a child neurologist at 35 weeks postmenstrual age according to the method of Dubowitz and Dubowitz [5] together with additional items concerning muscle power taken from Saint-Anne Dargassies [27] to exclude overt neurological abnormalities. Asphyxia was also excluded.

This selection resulted in a study group of 35 preterm infants: 12 were SGA (with birth weights below the 10th percentile) and 23 were AGA (with birth weights between the 25th and 75th percentile) according to the Amsterdam growth curves, corrected for parity and sex [13]. Gestational ages ranged from 27 to 34 weeks. The median gestational ages of the SGAs and AGAs were around 32 weeks, being 31^3 and 32^3 ($weeks^{days}$) respectively. Thus, 32 weeks gestational age was used to categorise subjects within the preterm group in order to compare the 'younger' (born at or before 32 weeks) and 'older' (born after 32 weeks) preterm infants. Clinical data are given in Table 2. All infants had Apgar scores of 7 or higher at 5 min and for the seven infants who had apnoeas none of these events were assessed as being prolonged. In all infants except four, there were no abnormal findings on ultrasound brain scans. For these four (patients 6, 8, 16, 40), the findings were considered to be of minor clinical importance. Congenital or chromosomal anomalies were excluded, as well as non-Caucasian subjects and those with small mothers (< 1.60 m.). Therefore, the SGA infants were considered to have suffered from late-occurring intra-uterine growth retardation. They had significantly lower ponderal indices (mean 2.11 ± 0.23) than their AGA counterparts (mean 2.44 ± 0.23) ($t = 4.01$, $df = 33$, $P < 0.001$). All AGA preterm infants had ponderal indices above the 10th percentile for gestational age [19]. Eight of the SGA preterm infants had values below the 10th percentile and four above. These four infants were considered to be dysproportionally growth retarded as well, on the basis of a mid-arm circumference/head circumference ratio [9] more than two standard deviations below the mean value for their gestational age. In addition there were 18 full-term infants, all of whom were AGA, with gestational ages of 38–42 weeks.

Table 1. Selection criteria

Variables	Selection criteria
Fetal factors	
Chromosomal anomalies	No
Cardiac diseases	No
Other congenital abnormalities	No
Fetal infections	No
Teratogenic factors	No
Pregnancy	Singleton
Neonatal factors	
Gestational age at birth	27–34 weeks
Birth weight status	$<P_{10}$ (SGA) or P_{25}–P_{75} (AGA)
Hypoglycaemia	<48 h
Cerebral haemorrhages	No degree III or IV
Periventricular leucomalacia	No
Other serious complications	No
Maternal factors	
Age and height	18–40 years, ≥1.60 m
Drugs, e.g. anti-epileptica	No
Alcohol consumption	<3 units per day
Menstrual cycle	Not irregular
Pregnancy duration	Based on reliable information of the mother or an early ultra-sound scan
Parental factors	
Race	Caucasian
Language at home	Dutch

Recordings

At all ages video recordings were made with infants lying supine and naked. At 35 weeks postmenstrual age the preterm infants were filmed in their cribs or incubators with an ambient temperature of 31 ± 2°C. Additional recordings were made of both preterm and full-term infants at the (corrected) ages of 6, 12, 18 and 24 weeks by a camera hidden above a purpose-built bed, surrounded by white curtains, with the temperature maintained at 27 ± 1°C. For 15 preterm and 6 full-term infants assessments were not possible at 24 weeks due either to rolling from supine to prone or crying.

Assessment of movement quality

From the recordings, GMs were selected for the assessment of the movement quality. They involve arm, leg, neck and trunk movements, with a variable sequence, speed and amplitude [25]. The quality of these movements are classified using the performance of GMs by healthy infants as a reference. In healthy infants GMs have an organized appearance which is both fluent and elegant. They have a gradual onset and offset and they wax and wane in speed and amplitude. The assessment of movement quality was based on Gestalt perception, i.e. on the global impression of the movement quality, during repeated viewings of the recordings [12]. A normal quality was assigned when an integrated impression of complexity, fluency and variability was obtained. An abnormal quality was designated when GMs were performed monotonously with almost no variation in speed and amplitude and were rigid and awkward in appearance, lacking rotations in arms and legs. Movements which were neither normal nor abnormal were classified as mildly abnormal: GMs were awkward but not rigid in appearance and lacked a consistent impression of complexity and variation. At each age classifications were made by an observer who was uninformed as to gestational age and birth weight status of each infant. The inter-judge agreement with two other observers was 87% and 93% (kappas 0.81 and 0.91). In addition, it was noted whether the GMs of each infant had changed from a writhing to a fidgety character by 12 weeks. Fidgety movements are defined as restless but smoothly rounded movements involving the whole body. They are always small in amplitude and of moderate speed [12].

Assessment of outcomes at 1 year

An age-appropriate neurological examination based on Touwen [29] was carried out at the (corrected) age of 1 year. This examination was devised to detect problems in postural control, fine motor abilities and asymmetries in posture and responses. Outcomes were classified as either good, borderline or poor, according to whether the infants were judged to have problems in none, one to two, or in all three of these domains, respectively. The examiner was unaware of the classifications of GM quality and also of the gestational age and birth weight status of each subject.

At the same age, the Mental Scale of the Dutch versions of the Bayley Scales of Infant Development [18] was used to obtain information on the general level of mental development. Develop-

Table 2. Clinical data of the preterm infants. PI, ponderal index. Ventilatory support: endotr, endotracheal ventilation; CPAP, continuous positive airway pressure

Subject	Sex	GA at birth weeks^{days}	Birth weight status	PI in g × 100/cm^3	Apgar 1 min/5 min	Ventilatory support (type, days)	Clinical events
SGAs							
1.	M	29^4	$<P_{2.3}$	1.74	8/10	Endotr. 4, CPAP 13	Apnoeas, pneumonia
2.	M	29^6	P_5	2.02	7/9	Endotr. 5, CPAP 27	Pneumonia, mild IRDS
3.	M	30^0	$P_{2.3}$–P_5	2.54	9/10	Endotr. 8, CPAP 1	Mild IRDS
4.	M	30^4	P_5–P_{10}	2.25	7/10	O_2 mask <1	Enterocolitis
5.	M	30^5	$P_{2.3}$–P_5	1.96	10/10	O_2 mask 3	Apnoeas
6.[a]	F	31^2	P_5–P_{10}	2.09	7/9	CPAP 1	No
8.[b]	M	31^4	$<P_{2.3}$	2.32	3/9	Endotr. 2, CPAP 1	No
11.	M	33^1	$<P_{2.3}$	2.09	9/10	CPAP 2	Apnoeas
12.	M	33^4	$<P_{2.3}$	2.31	8/10	O_2 mask 1, CPAP 2	No
13.	F	33^6	P_5	2.16	8/9	No	No
15.	F	34^0	P_5–P_{10}	2.05	8/9	No	No
38.	M	34^3	$<P_{2.3}$	1.78	5/8	O_2 mask <1	No
AGAs							
16.[c]	F	27^1	P_{75}	2.31	8/9	Endotr. 19, CPAP 16	Apnoeas, pneumonia, mild IRDS, bradycardias
17.	M	28^0	P_{25}	2.54	3/7	Endotr. 19, CPAP 8	Mild IRDS, pneumonia
18.	F	28^5	P_{25}	2.34	9/10	Endotr. 3, CPAP 7	Apnoeas
41.	F	28^5	P_{25}–P_{50}	2.35	8/10	No	No
19.	F	29^4	P_{50}–P_{75}	2.79	7/10	CPAP 1	Apnoeas
21.	F	31^1	P_{50}–P_{75}	2.39	7/10	Endotr. 2, CPAP 1	Pneumonia
40.[d]	M	31^1	P_{25}–P_{50}	2.73	–/8	CPAP 4	Mild IRDS
22.	F	31^3	P_{50}–P_{75}	2.32	8/8	CPAP 1	No
39.	M	31^3	P_{25}–P_{50}	2.25	9/10	No	No
23.	F	31^5	P_{50}–P_{75}	2.58	–/8	CPAP 1	No
24.	M	32^1	P_{50}–P_{75}	2.16	–/10	No	No
25.	F	32^3	P_{50}–P_{75}	2.96	8/9	No	Apnoeas, bradycardias
26.	F	32^3	P_{50}–P_{75}	2.61	8/8	CPAP 1	No
27.	M	33^0	P_{25}–P_{50}	2.13	9/8	Endotr. 2, CPAP 1	Pneumonia
28.	M	33^1	P_{25}–P_{50}	2.35	4/8	CPAP 1	Mild IRDS
29.	F	33^3	P_{25}–P_{50}	2.29	9/10	CPAP 1	No
30.	F	33^3	P_{50}	2.41	9/10	No	No
31.	F	33^3	P_{50}	2.30	6/10	No	No
32.	F	33^3	P_{25}	2.31	3/9	O_2 mask <1	No
34.	F	33^5	P_{50}–P_{75}	2.68	9/10	No	No
35.	F	33^6	P_{75}	2.88	9/10	CPAP 2	Mild IRDS
36.	M	34^3	P_{25}–P_{50}	2.24	9/10	No	No
37.	F	34^3	P_{25}	2.20	8/10	CPAP 1	No

Brain scan findings on ultrasound:

[a] 6. Periventricular echodensity;

[b] 8. Intraventricular haemorrhage (IVH) grade I;

[c] 16. IVH grade II;

[d] 40. IVH grade II

mental Indexes were derived for corrected ages in the case of preterm infants. These assessments were also carried out by a 'blind' examiner.

Results

All full-term infants maintained a normal movement quality from 6 to 24 weeks. For 24 of the preterms the assessments of movement quality stayed largely the same from 35 weeks postmenstrual age up to the corrected age of 24 weeks (Table 3). Fourteen of them had only normal assessments (group A). Three others were classified as having mildly abnormal quality at almost all ages (group B), wherease seven others had a combination of mildly abnormal and abnormal assessments (group C). Seven preterm infants recovered from a (mildly) abnormal movement quality (group D). In all but one of these infants the recovery occurred after 12 weeks corrected age. Five of these preterm infants were SGA, of whom four were born before 32 weeks. The other two were AGA, both born after more than 32 weeks. The quality of GMs deteriorated in four children (all AGA) after a normal assessment at 35 weeks postmenstrual age, for which no clinical explanation could be found (group E).

By the age of 12 weeks all infants with a normal quality (group A) or who recovered (group D) showed fidgety GMs. Of the infants who maintained a (mildly) abnormal quality (groups B and C) or changed to such a quality (group E) only two displayed GMs with a fidgety character at this age (patients 18, 41).

The percentage of cases with a normal quality at each age (group A) was strikingly lower for the SGA infants (9%) relative to the AGA infants (57%) ($\chi^2 = 7.63$, $P < 0.01$). However, 17% of the preterm AGA infants, but none of the SGA infants, became abnormal after a normal first assessment (group E). On the other hand, a larger percentage of SGA infants (42%) recovered compared to AGA infants (9%) ($\chi^2 = 5.36$, $P < 0.05$). Thus, differences between SGA and AGA preterm infants became less evident with age. This was not the case when the preterm infants were grouped on the basis of pregnancy durations of 32–34 weeks as opposed to less than 32 weeks, a conclusion based on the following findings. The percentages of infants who maintained a normal quality was slightly larger in the former (47%) than in the latter (35%) group. Furthermore, the percentages of infants who deteriorated were comparable in both groups (13% and 10%, respectively), as were the percentages of recoveries (20% in both groups). Thus, differences between 'younger' and 'older' preterm infants appear to remain fairly constant.

All full-term children had a normal course of GMs and a good neurological outcome at 1 year. Thus, the diagnostic value of the assessments of movement quality was examined only for the preterm infants. The specificities, sensitivities and predictive values of the assessments at different ages are given in Table 4. Only preterm infants with complete data at all ages, except 24 weeks, were considered ($n = 27$). The specificity for

Table 3. GM quality at 35 weeks postmenstrual age up to 24 weeks corrected age: N, normal; MA, mildly abnormal; A, abnormal; Nr, number; S, SGA; ≤, born at or before 32 weeks; BM, score for the Bayley mental scale at 1 year corrected age; NE, neurological outcome at 1 year: G, good, B, borderline, P, poor. For borderline cases ^{A}, ^{P} and ^{F} indicate asymmetry and problems with postural control and fine motor abilities, respectively

Nr	35	6	12	18	24	BM	NE
A: Normal GMs							
13^{S}	N	N	N	N	N	69	B^{AP}
$17^{\leq}$	N	N	N	N	–	82	B^{PF}
$19^{\leq}$	N	N	N	N	–	112	G
$24^{\leq}$	N	N	N	N	–	111	G
$25^{\leq}$	N	N	N	N	–	118	B^{AP}
$26^{\leq}$	N	N	N	N	–	101	G
27	N	N	N	N	–	107	G
29	N	N	N	–	N	94	G
30	N	N	N	N	N	101	G
34	N	N	N	N	–	127	G
36	N	N	N	N	N	115	B^{PF}
37	N	N	N	N	–	125	G
$39^{\leq}$	–	N	N	N	N	93	G
$40^{\leq}$	–	N	N	N	N	100	G
B: Mildly abnormal GMs							
$6^{S\leq}$	MA	MA	MA	MA	–	83	B^{A}
$21^{\leq}$	MA	MA	N	MA	–	127	B^{P}
38^{S}	–	MA	MA	MA	MA	85	B^{AF}
C: Abnormal/mildly abnormal GMs							
$2^{S\leq}$	A	A	MA	MA	MA	91	P
$4^{S\leq}$	MA	A	A	–	A	66	P
12^{S}	MA	MA	–	A	A	98	B^{PF}
15^{S}	MA	–	MA	A	MA	69	B^{AP}
$16^{\leq}$	A	A	A	A	–	64	P
$22^{\leq}$	A	A	MA	MA	–	102	P
$41^{\leq}$	–	A	MA	MA	MA	101	P
D: Early (mildly) abnormal, later normal GMs							
$1^{S\leq}$	MA	N	N	N	–	83	G
$3^{S\leq}$	A	A	MA	MA	N	127	B^{A}
$5^{S\leq}$	MA	MA	MA	N	N	84	G
$8^{S\leq}$	A	A	MA	N	N	97	B^{P}
11^{S}	MA	MA	MA	N	N	93	G
32	MA	A	MA	N	N	106	G
35	MA	A	MA	N	N	125	G
E: Early normal, later (mildly) abnormal GMs							
$18^{\leq}$	N	MA	MA	MA	MA	100	B^{P}
$23^{\leq}$	N	N	MA	A	–	–	P
28	N	MA	A	A	A	77	B^{P}
31	N	A	A	A	A	69	P

Table 4. The specificity, sensitivity and predictive values from 35 weeks postmenstrual age to 18 weeks corrected age

	35 weeks	6 weeks	12 weeks	18 weeks
Specificity for good outcome	0.58	0.67	0.67	1.00
Sensitivity for borderline outcome	0.40	0.60	0.50	0.50
Sensitivity for poor outcome	0.60	0.80	1.00	1.00
Predictive value				
Of normal movement quality	0.47	0.62	0.62	0.71
Of mildly abnormal quality	0.29	0.67	0.64	1.00
Of abnormal movement quality	1.00	0.75	1.00	1.00

good outcome increased with age to 1.00 by 18 weeks. Borderline outcomes appeared to be less strongly related to previous assessments of movement quality. The sensitivity for poor outcomes, calculated as the proportion of infants with a poor outcome who were previously assessed as being (mildly) abnormal in movement quality increased with age to 1.00 by 12 weeks. The predictive value of a normal movement quality was not very high, increasing to 71% at 18 weeks. However, mildly abnormal movements correctly predicted borderline or poor outcomes in a rising proportion of the preterms, so that by 18 weeks the predictive value was 100%. Abnormal movements had predictive values of 100% at all ages except 6 weeks.

The predictive value of an abnormal movement quality at 18 weeks, calculated for only poor outcomes (thus excluding borderline cases), was still 75%. The prevalence of poor outcomes in the group was 19%, which means that the chance of correctly predicting a poor outcome increases from 19% to 75% when the movement quality is known to be abnormal at the age of 18 weeks.

Outcomes were also compared with the trends in qualitative assessments as opposed to assessments at a single age (Table 3). The most striking finding was the similarity between the outcomes of the infants with only normal assessments (group A: ten good, four borderline) and those who changed to a normal quality (group D: five good, two borderline). Likewise, the outcomes of the preterm infants in whom the GM quality deteriorated (group: two borderline, two poor) were comparable to those of infants with consistent abnormal or mildly abnormal qualities (groups B and C: five borderline, 5 poor).

One preterm infant (patient 16) developed overt cerebral palsy (viz. quadriplegia) within the 1st year, but additional clinical data have since indicated a mild case of hemiplegia (patient 21) and another of mild diplegia (patient 31). The quadriplegia was preceded by abnormal GM qualities and the hemiplegia by mildly abnormal GMs at all ages except 12 weeks. The child with a mild diplegia was assessed as having normal GMs at the first recording only (at 35 weeks postmenstrual age), with abnormal qualities thereafter.

The Bayley Developmental Indexes at 1 year indicated moderate to severe developmental retardation in six infants (patients 4, 13, 15, 16, 28, 31, with indexes ranging from 64 to 77). Five of them had been previously identified as maintaining or changing to (mildly) abnormal GMs and none of them had good neurological outcomes. Full data are given in Table 3.

Discussion

Qualitative assessments of GMs stayed largely the same from 35 weeks postmenstrual age to 24 weeks corrected age in most preterm infants. Fourteen of them retained a normal movement quality. Most of the infants who started off with abnormal or mildly abnormal GMs persisted with such qualities up to 24 weeks, while a smaller number changed to a normal quality. This improvement occurred in most cases after the age of 12 weeks. It is around this age that a major transformation in neural functions has been reported [21]. A relation might exist between this transformation and our findings on changes in movement quality around the age of 12 weeks. Until this age the movement repertoire changes little from that observed during fetal life [21]. At around 12 weeks the fetal repertoire is replaced by movements better adapted to the extra-uterine environment. As suggested by Prechtl [22, 23], the demise of transitory fetal neural mechanisms may explain the disappearance of some early neurological dysfunctions at approximately the same age. An age-related change in GM quality from abnormal to normal has already been documented by Ferrari et al. [7] in some preterm infants with brain lesions around 5–12 weeks. In our study a similar change was also found in preterm infants without serious brain lesions or serious medical and neurological problems. In this context it should be noted, however, that grade I and II cerebral haemorrhages, which are generally thought not to be serious, should be treated with caution, since three of the four infants with such brain scan findings showed at least some sequelae at 1 year.

The specificities, sensitivities and predictive values improved from the age of 3 months onwards. Such improvements indicate that the values obtained are age-dependent, which strongly suggests that reliable predictions for later problems in preterm infants without gross abnormalities may only be possible after this age. Data obtained from other forms of neurological assessment also demonstrate that prediction improves at 3–4 months [6, 26]. Such findings do not necessarily imply that assessments prior to the age of 3 months are of no value. They could be important for the initial detection of infants at risk for later problems and for whom close follow up is needed. Decisions on therapeutic intervention, however, might be postponed until after this age.

It is important to recognize that the size of the values for specificity and sensitivity depend on the nature of the groups involved. In some studies the early assessments were carried out in two extreme groups of infants, such as low-risk preterm infants and those with documented brain lesions [7]. As pointed out by others [8], the use of such divergent groups might inflate the specificity and sensitivity. They suggest, therefore, that the prediction of later outcomes in such studies might be more appropriately expressed in terms of relative risk.

Neurological outcomes are often only defined in terms of the presence or absence of cerebral palsy [2, 6, 16]. It should be stressed that in our study poor outcomes did not necessarily involve such gross pathology. Only three preterm infants developed cerebral palsy. Nevertheless, in children who do not become afflicted with cerebral palsy, developmental sequelae may still be detected [17]. One year is, of course, quite young for definite statements on outcome, but serious abnormalities can be expected to be manifest by that age.

Neurological examinations based on the assessment of muscle tone and elicited responses appear to provide satisfactory predictions for normal outcomes at 1 [28] and 3 years [26]. Abnormal outcomes at both ages, however, are less accurately predicted. This crucial aspect of prediction seems to be addressed more adequately by assessments of movement quality. Results from this study indicate that assessments of GM quality provide a sensitive means for the early detection and prognosis not only of gross pathology, but also of more differentiated forms of neurological dysfunctioning and early signs of mental retardation.

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References

1. Allen MC (1984) Developmental outcome and follow-up of the SGA infant. Semin Perinatol 8:123–156
2. Blair E, Stanley F (1990) Intrauterine growth and spastic cerebral palsy. I. Association with birth weight for gestational age. Am J Obstet Gynecol 162:229–237
3. Cioni G, Prechtl HFR (1990) Preterm and early postterm motor behaviour in low-risk premature infants. Early Hum Dev 23: 159–191
4. Commey JOO, Fitzhardinge PM (1979) Handicap in the preterm small-for-gestational age infant. J Pediatr 94:779–786
5. Dubowitz L, Dubowitz V (1981) The neurological assessment of the preterm and fullterm newborn infant. Clinics in developmental medicine, vol 79. Spastics Int Med Publ
6. Ellenberg JH, Nelson KB (1981) Early recognition of infants at high risk for cerebal palsy: Examination at age four months. Dev Med Child Neurol 23:705–716
7. Ferrari F, Cioni G, Prechtl HFR (1990) Qualitative changes of general movements in preterm infants with brain lesions. Early Hum Dev 23:193–231
8. Frankenburg WK, Chen J, Thornton SM (1988) Common pitfalls in the evaluation of developmental screening tests. J Pediatr 113:1110–1113
9. Georgieff MK, Sasanow SR, Mammel MC, Pereira GR (1986) Mid-arm circumference/head circumference ratios for identification of symptomatic LGA, AGA and SGA newborn infants. J Pediatr 109:316–321
10. Hadders-Algra M, Huisjes HJ, Touwen BCL (1988) Preterm or small-for-gestational-age infants. Eur J Pediatr 147:460–467
11. Heinonen K, Matilainen R, Koski H, Launiali K (1985) Intrauterine growth retardation (IUGR) in preterm infants. J Perinat Med 13:171–178
12. Hopkins B, Prechtl HFR (1984) A qualitative approach to the development of movements during early infancy. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal life. Clinics in develomental medicine, vol. 94. Blackwell, Oxford, pp 179–197
13. Kloosterman GH (1970) On intrauterine growth. Int J Gynaec Obstet 8:895–912
14. Largo RH, Pfister D, Molinari L, Kundu S, Lipp A, Duc G (1989) Significance of prenatal, perinatal and postnatal factors in the development of AGA preterm infants at five to seven years. Dev Med Child Neurol 31:440–456
15. Low JA, Galbraith RS, Muir DW, Broekhoven LH, Wilkinson JW, Karchmar EJ (1985) The contribution of fetal-newborn complications to motor and cognitive deficits. Dev Med Child Neurol 27:578–587
16. Luthy DA, Shy KK, Strickland D, Wilson J, Bennett FC, Brown ZA, Benedetti TJ (1987) Status of infants at birth and risk for adverse neonatal events and long-term sequelae: A study in low birthweight infants. Am J Obstet Gynecol 157: 676–679
17. Marlow N, Roberts BL, Cooke RWI (1989) Motor skills in extremely low birthweight children at the age of 6 years. Arch Dis Child 64:839–847
18. Meulen BF van der, Smrkovsky M (1984) Bayley developmental scales. Kinderstudies, Groningen (in Dutch)
19. Miller HC, Hassanein K (1971) Diagnosis of impaired fetal growth in newborn infants. Pediatr 48:511–522
20. Pena IC, Teberg AJ, Finello KM (1988) The premature small-for-gestational-age infant during the first year of life: Comparison by birth weight and gestational age. J Pediatr 113:1066–1073
21. Prechtl HFR (1984) Continuity and change in early neural development. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal life. Clinics in developmental medicine, vol. 94. Blackwell, Oxford, pp 1–15
22. Prechtl HFR (1984) Epilogue. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal life. Clinics in developmental medicine vol. 94. Blackwell, Oxford, pp 245–247
23. Prechtl HFR (1986) Frühe Schäden – späte Folgen; Neuere Erkenntnise aus Nachuntersuchungen von Kindern. In: Schmidt MJ, Droemann S (eds) Langzeitverlauf Kinder und Jugendpsychiatrischer Erkrankungen. Ferdinand Enke Verlag, Stuttgart, pp 15–21
24. Prechtl HFR (1990) Qualitative changes of spontaneous movements in fetus and preterm infant are a marker of neurological dysfunction. Early Hum Dev 23:151–158
25. Prechtl HFR, Nolte R (1984) Motor behaviour in preterm infants. In: Prechtl HFR (ed) Continuity of neural functions from prenatal to postnatal Life. Clinics in developmental medicine vol. 94. Blackwell, Oxford, pp 79–92
26. Ross G, Lipper E, Auld PAM (1986) Early predictors of neurodevelopmental outcome of very low-birthweight infants at three years. Dev Med Child Neurol 28:171–179
27. Saint-Anne Dargassies S (1977) Neurological development in the fullterm and premature infant. Elsevier, Amsterdam
28. Stewart A, Hope PL, Hamilton P, Costello AM de-L, Baudin J, Bradford B, Amiel-Tison C, Reynolds EOR (1988) Prediction in very preterm infants of satisfactory neurodevelopmental progress at 12 months. Dev Med Child Neurol 30:53–63
29. Touwen BCL (1976) Neurological development in infancy. London, Heinemann