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Neurodevelopmental Significance of Minor and Major Congenital Anomalies in Neonatal High Risk Children

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Key words

Congenital anomalies – Risk infants – Development

Abstract

Minor and major congenital anomalies were studied in 395 neonatal risk children and 107 normal school children at the age of nine in the context of follow-up of the risk children. The purpose of the study was to evaluate the impact of early prenatal disturbances on the long term prognosis. Minor physical anomalies (MPA) were scored by a weighted scoring system modified from that of *Waldrop* and *Halverson*. The children with minor or major congenital anomalies performed worse in a cognitive test (WISC) and in a motor performance test. The differences were significant in the neonatal risk group. There were more small for gestational age (SGA) children in the anomaly group of the neonatal risk group as a whole and in the low birthweight group than in the non-anomaly group. Hyperactivity was associated with a high MPA score in the comparison group, but not in the study group. The results are consistent with earlier reports of associations between intrauterine growth disturbance and minor physical anomalies. Our findings suggest an additive effect of prenatal insults and neonatal risk factors in the origin of neurodevelopmental disturbances.

Introduction

Congenital anomalies are signs of pathological morphogenesis. Irrespective of etiology and pathogenesis they may be divided into major and minor anomalies on the basis of their consequences to the affected individual. A major anomaly has adverse effects on either the function or the social acceptability of the individual, and therefore calls for medical intervention. Minor congenital anomalies have insignificant medical or cosmetic consequences to the patient (*Marden* et al 1964, *Smith* 1966). This definition inevitably requires subjective judgement, which is a problem in comparing various studies.

The incidence of major congenital anomalies among live born infants has been reported to be 2–3% for structural malformations and up to 3–6% when more controversial abnormalities, such as pyloric stenosis, dislocation of the hip and club foot, are included. The cumulative detection frequencies of diagnosed anomalies tend to rise up to double depending on the length of the follow-up time (*Marden* et al 1964, *Ekelund* et al 1970, *Hakosalo* 1973, *Heinonen* et al 1977, *Klemetti* 1978). Functional defects such as mental retardation, cerebral palsy or metabolic diseases have not been included in these figures.

The reported frequencies of minor congenital anomalies in children vary considerably depending on the classification of the anomalies and the scoring system used. One single minor anomaly has been observed in 9.7–37.5%, and at least two in 1.3–14.6% of children examined (*Marden* et al 1964, *Smith* and *Bostian* 1964, *Ekelund* et al 1970, *Crichton* et al 1972, *Méhes* et al 1973). The weighted minor physical anomaly (MPA) scoring system of *Waldrop* and *Halverson* (1971) has been used in several studies of children at different ages and clinical set-ups. A summary of nine earlier studies is presented in Table I.

A common prenatal etiology for minor and major anomalies has been suggested by the high incidence of major anomalies in children with multiple minor anomalies and vice versa (*Marden* et al 1964, *Méhes* et al 1973, *Smith* and *Bostian* 1964). Prenatal insults seem to be probable background factors in both minor physical anomalies and major neurological and cognitive disturbances considering the high incidence of minor anomalies in children with cerebral palsy and mental retardation of unknown origin (*Smith* and *Bostian* 1964, *Firestone* et al 1978, *Kalbe* 1978).

Associations between MPAs and neurodevelopmental and behavioural disturbances have also been reported. As early as 1902, *Still*, lecturing on defective moral control in children, stated that 15 of 19 children suffering from various behavioural problems but without general impairment of intellect showed obvious anomalies of appearance, which he called "stigmata of degeneration". In a study of behavioral disturbances, *Daryn* (1960) regarded minor malformations as signs of "organicity". *Waldrop* and her co-workers demonstrated in several studies a relationship between problem behaviour and minor anomalies especially in boys (*Waldrop* et al 1968, *Waldrop* and *Halverson* 1971, *Waldrop* and *Goering* 1971, *Halverson* and *Victor* 1976, *Waldrop* et al 1978). These findings have been confirmed by *Quinn* and *Rapoport* (1974), *Burg* et al (1980), *O'Donnel* and *Van Tuinan* (1979) and *Firestone* and *Prabhu* (1983). In a recent Swedish epidemiological study of perceptual, motor and attentional deficits a significant association was found between high minor anomaly scores and minimal brain dysfunction in six-year-old children (*Gillberg* and *Rasmussen* 1982).

Table II List of minor physical anomalies and their weighted scores

Anomaly	Score
Eyes	
Epicanthus	
Deeply covered	2
Partly covered	1
Hypertelorism (x)	
Inner canthal distance > 34 mm	2
Inner canthal distance 32-34 mm	1
Ears	
Low set	1
Adherent lobes	
Lower edge of the ear oblique	2
Lower edge of the ear horizontal	1
Malformed	1
Asymmetrical	1
Mouth	
High arched palate	
Roof of mouth steepled	2
Roof of mouth moderately high	1
Furrowed tongue	1
Hands	
Fifth finger	
Marked clinodactyly	2
Slight clinodactyly	1
Single transverse palmar crease	1

(x) > *Feingold* and *Bossert* 1974

Table III Distribution of the neonatal risk diagnoses in the study group. The figures indicate the total number of each diagnosis

Diagnosis	MPA	Malformation	Non-anomaly	Total
	n	n	n	n
Hyperbilirubinaemia	19	8	140	167
Low birthweight	10	9	76	95
Asphyxia	6	5	66	77
Neurological symptoms	4	5	39	48
Hypoglycaemia	6	2	29	37
Respiratory problems	4	1	28	33
Diabetic mother	4	2	25	31
Only one dg	29	13	269	311
More than one dg	11	8	65	84
Total	40	21	334	395

At the age of nine the children were subjected to a neuropaediatric examination and the Test of Motor Impairment (*Stott* et al 1972) by one of the authors (E.L.) who had no knowledge of the perinatal status of the child. The Test of Motor Impairment (TMI) includes items assessing five areas of motor performance. The result is expressed as an impairment score: the higher the score, the greater the impairment of motor performance. Minor physical anomalies (MPA) were scored by the same examiner (E.L.) using a modification of the system of *Waldrop* and *Halverson* (1971); for details see Table II. The score for deviations of head circumference from normal was omitted from the original list except for cases in which the abnormality was already present at birth. Notes on the quality of the hair were also excluded, because many normal Finnish children have fine and electric hair. The assessment of hair whorls was considered very liable to subjective errors and was hence excluded. The characteristics of feet and toes (wide gap between the first and second toe and syndactyly between the second and third toe) were found to be very difficult to evaluate objectively, and were therefore omitted. Major anomalies were also registered. According to the definition of a major anomaly, those with medical or marked cosmetic significance were included. Of controversial anomalies those classified as congenital malformations in the International Classification of Diseases (WHO 1977) were included (pyloric stenosis, congenital hip dislocation and testicle retention, for instance, were included, but inguinal hernia and hydrocele of the testicle were excluded).

For analysis of the data, four groups were formed according to the characteristics of the anomalies observed:

a) *Non-anomaly group* comprising the children with no anomalies.
b) *Combined anomaly group* including all children belonging to one of the following groups (c and d)
c) *Minor physical anomaly (MPA) group* including children given a weighted anomaly score of four or more.
d) *Malformation group* comprising children with a score of less than four but having a major anomaly and children with a specific anomaly syndrome irrespective of the MPA score.

The corresponding codes for the comparison group are a, b, c and d, respectively. The distribution of the neonatal risk diagnoses in the study group is presented in Table III.

The cognitive abilities of 389 study group and 72 comparison group children were evaluated by a psychologist using the Wechsler Intelligence Scale for children (WISC), Finnish edition (1971). During the medical examination the attention span and hyperactivity of the child were assessed and scored from zero to two according to the severity of the disturbance. No laboratory analyses were made during the study. Chromosomal studies had been performed earlier in some cases, but no abnormalities were found.

In the statistical computations the BMDP (BMDP Statistical Software 1983) statistical package was used. In the analysis of continuous variables differences between the groups were at first calculated with two-way analysis of variance (ANOVA, BMDP 7D). Pairways computations with T-test (*Student* or *Welch*) were then calculated with adjustment for multiple comparisons (Bonferroni correction). Logarithmic transformation was made for not normally distributed variables. In the analysis of discrete variables the chi square test with Yates' correction and Fisher's exact probability test were used (BMDP 4F).

Results

Anomaly groups, minor and major anomalies

The numbers of children in the anomaly groups with associated major anomalies are presented in Table IV. In the mal-

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Table IV Anomaly groups with associated major anomalies

Major anomalies	Study group MPA (C) n = 40	Malf. (D) n = 21	Total n = 395	(%)	Comparison group MPA (c) n = 6	Malf. (d) n = 2	Total n = 107	(%)
Congenital heart defect	2	3	5	(1.3)	–	–	–	(0)
Craniofacial	1	3	4	(1.0)	–	–	–	(0)
Musculosceletal	1	4	5	(1.3)	–	1	1	(0.9)
Club foot	–	3	3	(0.8)	–	–	–	(0)
Hypospadia*	1	2	3	(1.4)	–	–	–	(0)
Testicular retention*	3	3	6	(2.9)	–	1	1	(1.9)
Congenital hip dislocation	2	5	7	(1.8)	1	–	1	(0.9)
Hypertrophic pylorus	–	1	1	(0.3)	–	–	–	(0)

* In boys, study group n = 209, comparison group n = 52

Table V Minor physical anomaly scores by group and sex

Group	n	Mean	Range	Score ⩾ 4 n	%
Study group total	395	1.5	0–8	41	10.4
female	186	1.4	0–8	13	7.0
male	209	1.7	0–8	28	13.4
Comparison group total	107	1.7	0–6	6	5.6
female	55	1.5	0–6	2	3.6
male	52	1.8	0–6	4	7.7

formation group of the study group, there was one girl with Williams syndrome, on boy with Prader-Willi syndrome and one boy with mild rubella embryopathy. The total number of major anomalies was 34 (8.6% of the total) in the study group and three (2.8%) in the comparison group. The differences in the numbers of anomalies were not statistically significant. The number of most anomalies was higher than expected in the study group. There were no significant differences in sex or social class distributions within or between the study and comparison groups. The means and ranges of the weighted anomaly score and the percentages of children scoring four or more are shown in Table V. In ANOVA neither group nor sex were significant sources of variation ($F = 2.49$ and $F = 1.91$, $df = 1$). The number of boys scoring four or more was higher ($p < 0.05$) than the number of girls in the study group. The anomaly score cutpoint was obtained from the distribution of the score in the comparison group, in which about five per cent of the group scored four or more (Table V).

Obstetrical and perinatal data

No significant differences were found between the anomaly groups and the non-anomaly group in regard to maternal age, birthweight or head circumference at birth within the study and comparison groups (ANOVA and pairways comparisons, see Table VI). The differences in the birthweight and head circumference between the study and comparison group reflect the great proportion of low birthweight infants in the study group. In Table VII the adverse obstetrical factors analysed are presented for the non-anomaly, combined anomaly and MPA groups. Various factors present during pregnancy were analysed by their occurrence during the three trimesters in different combinations. In the study group there were more first trimester urinary tract infections in the combined anomaly group and more mothers with moderate-severe toxaemia in the MPA group than in the non-anomaly group. No significant differences were found within the comparison group. Most differences between the study and comparison group are due to toxaemic manifestations known to be potentially hazardous for the fetus and newborn, and reflect the composition of the study group.

The distribution of the neonatal risk diagnoses within the non-anomaly, malformation and minor anomaly group did not show any statistically significant differences (Table III). However, the proportion of babies too small for gestational age (SGA) was significantly greater in the combined anomaly than in the non-anomaly group (15 of 61 and 39 of 334 respectively, $p < 0.01$). The number of SGA low birthweight babies was higher in the combined anomaly and MPA groups than in the non-anomaly group (9/61, 6/40 and 17/334, respectively, $p < 0.05$). Within the low birthweight group, there were proportionately more SGA babies in the MPA group than in the combined anomaly and the non-anomaly groups (6/10, 9/19 and 17/76 respectively, $p < 0.05$). In the comparison group, there were 8 SGA babies in the whole group (7.5%) and none in the anomaly groups.

Nine-year examination

The mean scores in the study and comparison groups in the Test of Motor Impairment and WISC by anomaly groups are presented in Table VI. With regard to the Test of Motor Impairment, the anomaly group was a significant source of variation in ANOVA. In pairways comparisons the differences were observed in the study group (A/B, A/D). No significant differences were found in the comparison group.

In the analysis of variance of the WISC scores both grouping factors (study/comparison, anomaly groups) were found to be significant sources of variation (Table VI). Pairways comparisons showed significant differences within the study group and between the non-anomaly groups. Though not showing any statistically significant differences, the TMI scores expressed a trend towards inferior performance in the anomaly groups of the comparison group. Same trend can be seen in the birthweights and WISC scores.

Table VI Maternal age, birthweight, birth head circumference, Test of Motor Impairment (TMI) and WISC by anomaly groups

			Maternal age (y)	Birth weight (g)	Head circumf. (cm)	TMI	WISC	
		n	mean ± sd	mean ± sd	mean ± sd	mean ± sd	n	mean ± sd
Study group		395						
non-anomaly	(A)	334	25.7 ± 4.8	2942 ± 883	33.6 ± 2.4	6.4 ± 5.3	329	114.3 ± 13.5
comb. anomaly	(B)	61	25.8 ± 4.1	2808 ± 985	33.5 ± 2.7	10.8 ± 9.6	60	106.3 ± 18.6
MPA	(C)	40	25.4 ± 4.3	2942 ± 1016	33.9 ± 2.9	9.9 ± 9.1	40	108.5 ± 17.0
malformation	(D)	21	26.4 ± 3.8	2553 ± 889	32.7 ± 2.3	10.1 ± 10.0	20	101.9 ± 21.1
Comparison group		107						
non-anomaly	(a)	99	25.9 ± 4.7	3516 ± 446	34.9 ± 1.2	4.9 ± 3.6	66	120.8 ± 11.7
comb. anomaly	(b)	8	25.6 ± 2.5	3293 ± 545	34.9 ± 1.6	5.9 ± 3.6	6	113.5 ± 16.3
MPA	(c)	6	26.7 ± 1.9	3098 ± 462	34.7 ± 1.8	4.7 ± 3.3	4	105.3 ± 12.1
malformation	(d)	2	22.5 ± 0.7	3875 ± 318	35.5 ± 0.7	9.5 ± 0.7	2	130.0 ± 8.5
Summary of ANOVA								
Study/comparison (df = 1)	F		0.38	8.25	6.13	0.74		6.01
	p		ns	< 0.01	< 0.05	ns		< 0.05
Anomaly group (df = 2)	F		0.34	0.63	0.03	3.29		3.99
	p		ns	ns	ns	< 0.05		< 0.05
Levels of significance in T-test								
$p < 0.05$				D/d	D/d			A/C, A/D, D/d
Bonferroni				ns	ns			ns, ns, ns
$p < 0.01$						A/C, A/D		A/B
Bonferroni						A/D		A/B
$p < 0.001$				A/a	A/a	A/B		A/a
Bonferroni				A/a	A/a	A/B		A/a

Logarithmic transformation of TMI scores before calculations. A slash separates the groups compared

Hyperactivity

The mean and range of the minor physical anomaly score for non-hyperactive and hyperactive boys and girls is presented in Table VIII. In ANOVA study/comparison group and hyperactivity were found to be significant sources of variation. In pairways comparisons the MPA score of the hyperactives in the comparison group was higher than that of the non-hyperactives of the comparison group, and of the hyperactives of the study group ($p < 0.05$, not significant after Bonferroni correction). In the study group, there was no significant differences in the number of hyperactive children in the non-anomaly group A and the anomaly groups B and C (45/334, 13/61 and 8/40), while in the comparison group there were significantly more hyperactives in the anomaly groups b (4/8) and c (3/6) than in the non-anomaly group a (6/99, $p < 0.01$). There were more boys among the hyperactives in both the study and the comparison group ($p < 0.05$).

School problems

Forty-one study group children (10.4%) either attended a special class or experienced marked difficulties in school progress (started school one year later or repeated a grade); 17 of them belonged to the combined anomaly group, 10 to the minor anomaly group and 24 to the non-anomaly group $p < 0.001$ between the anomaly groups and the non-nomaly group. According to the criteria of selection, all the comparison group children attended normal school, but one of them had repeated a class.

Discussion

The results of our study point to the significance of prenatal events in the development of children. This was definitely demonstrated in the neonatal high risk group, and some associations were found in the non-risk comparison group. Earlier reports of associations of hyperactivity and MPA are supported by our results.

The nine-year follow-up percentage in the study group may be considered satisfactory though not optimal. The drop-outs were mainly caused by migration and parental refusals. The most severely handicapped children, often institutionalized, have deliberately been excluded from the follow-up, because the main interest of the original study design (*Michelsson* et al 1981) was focussed on the minor handicap group. The follow-up group included children from all social classes and risk groups in proportions comparable to those of the original group. Our study design was not an epidemiological one, and

Table VII Adverse obstetrical factors in the non-anomaly, combined anomaly and minor physical anomaly (MPA) groups of the study and comparison groups

		Study group			Comparison group		
		Non-anomaly (A) n	Comb. anomaly (B) n	MPA (C) n	Non-anomaly (a) n	Comb. anomaly (b) n	MPA (c) n
	Group total	334	61	40	99	8	6
Unmarried mother		29	2	0	10	1	1
Previous abortions > 2		18	4	3	4	0	0
Previous infertility		1	0	0	1	0	0
Premature contractions		44*	9	6	5*	2	2
Bleeding during	I trimester	16	3	1	4	0	0
	II–III trimester	22	8	4	5	0	0
Anaemia < 110 g/l		11	3	3	6	0	0
Hypertension	> 140/90	93	22	15	26	1	0
	> 160/110	42*	8	7	5*	0	0
Toxaemia moderate/severe		32*	10	9*	2	0	0
Chest x-ray < 20 gest. weeks		18*	1	1	0*	0	0
Smoking 5–10 cig/day		27	4	3	10	2	2
> 10 cig/day		32	5	2	8	1	1
Viral infection	I trim.	2	1	1	0	1	1
	II–III trim.	28	2	1	8	1	1
Urinary tract infection	I trim.	10*	6*	4	1	0	0
	II–III trim.	45*	6	2	4*	1	1
Drugs							
sulfa	I trim.	8	3	2	0	0	0
	II–III trim.	32	4	2	5	1	1
nitrofurantoin	I trim.	2	3	1	0	0	0
	II–III trim.	12	3	0	0	0	0
analgetics	I trim.	2	1	0	3	0	0
	II–III trim.	11	6	3	0	0	0
antihypertensives							
	I trim.	1	0	0	0	0	0
	II–III trim.	59***	10	7	2***	0	0
Placental calcifications		57	10	7	24	3	1
infarction		7	1	1	0	0	0
partial ablation		5	2	0	0	0	0
Umbilical cord < 30 cm		3	2	2	0	0	0
1 artery		1	0	0	0	0	0
abnormal insertion		3	0	0	5	0	0

* $p < 0.05$, *** $p < 0.001$ between the two numbers of events on the same line

hence does not warrant conclusions about the incidence of congenital anomalies or neurodevelopmental disturbances.

The methods of scoring minor physical anomalies have been criticized by *Krouse* and *Kaufmann* (1982). The main points of their criticism were taken into consideration in our study. By scoring only those anomalies in the original list of *Waldrop* and *Halverson* (1971) that can be objectively evaluated and measured, we have tried to minimize the pitfalls of

Table VIII Mean and range of the MPA score in the hyperactive and non-hyperactive children by sex

	Study group						Comparison group					
	Hyperactive			Non-hyperactive			Hyperactive			Non-hyperactive		
	n	mean	range	n	mean	range	n	mean	range	n	mean	range
Total	58	1.6	0–8	337	1.5	0–8	10	2.9	1–6	97	1.5	0–6
Girls	20	1.9	0–8	166	1.4	0–6	1	3.0		54	1.5	0–6
Boys	38	1.5	0–8	171	1.7	0–8	9	2.9	1–6	43	1.6	0–5

Summary of ANOVA: Study/comparison group $F = 7.22$, $p < 0.01$, hyperactivity $F = 5.70$, $p < 0.05$, sex $F = 1.91$, ns

subjective errors. In spite of the reduced number of anomalies scored, significant differences were found. The scores in the Test of Motor Impairment in our comparison group were inferior to those previously reported (*Stott* et al 1972, *Drillien* et al 1980). The opposite is true for the WISC scores. These findings will be discussed in separate communications.

We have not been able to find in the literature any report on minor and major congenital anomalies in the context of a prospective study of potential etiological factors in neurodevelopmental disturbances. The significance of prenatal disturbances has been demonstrated, however, in studies on children with cerebral palsy (*Hagberg* et al 1975a and b, *Kalbe* 1978), minimal brain dysfunction (*Gillberg* and *Rasmussen* 1982) and low birthweight (*Drillien* et al 1980, *Hertzig* 1981). On the other hand, a combination of high MPA and pre-perinatal complications has been found to increase the risk of hyperactivity (*Firestone* and *Prabhu* 1983). Learning-disabled children with a history of obstetrical complications have been reported to have more MPA than those without (*Steg* and *Rapoport* 1975). In both these studies the anamnestic data were obtained from the parents.

The aim of our study was to clarify the role of intrauterine disturbances in the origin of neurodevelopmental dysfunctions using minor and major physical anomalies as potential markers. The incidence figures of the associated major anomalies (Table IV) in the study group were somewhat higher than those generally presented. The significance of this observation is difficult to confirm, but it seems to demonstrate an increased risk of perinatal morbidity, even though the anomaly is not life-threatening. In spite of the lack of statistical significance, there was a reduction in birthweight of the babies in the anomaly groups compared with those of the non-anomaly group. The number of babies small for gestational age was significantly higher in the combined anomaly group of the study group, although SGA babies were only included in the study series if they belonged to a risk group. The proportion of SGA low birthweight infants was greater in the anomaly groups, and in the low birthweight group SGA babies were overrepresented in the MPA group. These findings are in good agreement with the hypothesis put forward and discussed by *Spiers* (1982) that growth retardation and low birthweight are factors predisposing to congenital anomalies, although the mechanisms involved are not understood. In the comparison group there were no SGA babies in the anomaly group, probably because of the small number of children.

At the age of nine years the mean test scores in the Test of Motor Impairment and WISC were consistently lower in the anomaly groups compared to the non-anomaly group of the study group. A similar trend was observed in the comparison group. The comparison group children had almost consistently better scores than the study group children. These findings suggest an additive effect of prenatal insults and neonatal risk factors in the etiology of the disturbances.

The implications of adverse obstetrical events in the context of increased MPA have been discussed in several earlier studies (*Marden* et al 1964, *Quinn* and *Rapoport* 1974, *Berkowitz* et al 1983). A positive correlation is also suggested by the MPA scores in the studies reviewed in Table I. In the studies of *Waldrop* et al (1968) and *Waldrop* and *Halverson* (1971) children with perinatal risk factors had higher MPA scores than non-risk children. Our results bear some evidence of this, too. Our study was not biassed by retrospective data collection, because obstetrical anamnestic data were collected before the children were born.

The concept of early and late MPA (*Cummings* 1982) is interesting when the vulnerable growth spurt period of the brain is considered (*Dobbing* 1974). It would be reasonable to think that it is especially the second-third trimester insults that cause minor neurodevelopmental disturbances and that the same insults would be able to cause minor morphological anomalies. In our study we did not, however, make any distinction between early and late MPA. On the other hand, we found no difference in the occurrence of adverse obstetrical events between the early and late phases of pregnancy in the anomaly groups.

The association of minor physical anomalies and hyperactivity observed in earlier investigations (*Waldrop* et al 1968, *Waldrop* and *Halverson* 1971, *Halverson* and *Victor* 1976, *Waldrop* et al 1978, *Quinn* and *Rapoport* 1974, *Burg* et al 1978, *O'Donnel* and *Van Tuinan* 1979, *Firestone* and *Prabhu* 1983) was corroborated in this study, although only in the comparison group (Table VIII). The lack of a significant association in the study group is presumably accounted for the multiplicity of other possible etiological factors in poor concentration capacity and hyperactivity.

In our study it remains unclarified whether the results of the non-risk anomaly and non-anomaly groups would have differed more if children from special classes had been included in the comparison group. It seems probable, but an epidemiological study design would be required to confirm the assumption.

The involvement of prenatal disturbances seems to be of great importance in the etiology of neurodevelopmental disturbances. A prenatal insult renders the baby more liable and vulnerable to perinatal disturbances, and the effects of pre- and perinatal unfavourable events are likely to be potentiated by each other. The signs of prenatal insults expressed as minor and major congenital anomalies and retarded intrauterine growth are important hallmarks that should be carefully evaluated when examining children with suspected neurodevelopmental disturbances. The possible impact of intrauterine insults should be taken into consideration in struggling for a comprehensive etiological diagnosis for these children, although we often are lacking in the knowledge of the exact nature of the insults.

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